MEDICAL ENTOMOLOGY

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MEDICAL ENTOMOLOGY

With Special Reference to the Health and Well-being of Man and Animals

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I gratefully and proudly dedicate the fourth edition of *Medical Entomology* to my former graduate students of the University of California, more than fifty of whom served during World War II in many parts of the world in various capacities in the mammoth struggle to protect our fighting forces against the ravages of arthropod-borne tropical diseases, particularly malaria.

PREFACE TO THE FOURTH EDITION

THE ORIGINAL MANUSCRIPT for this book was prepared about forty years ago, and the first edition, entitled *Medical and Veterinary Entomology,* appeared in 1915. The book was most generously received from the beginning and has been widely and increasingly used in each succeeding edition. It took considerable perseverance to complete another revision for a fourth edition. With the encouragement of long-time users of the work and the assistance of colleagues, graduate students, and others this has now been accomplished.

The science of medical entomology has been fully accepted as one of the important fundamentals for a complete knowledge and understanding of public health operations as well as for an understanding of the health of domestic animals; it is essential to a complete understanding of the epidemiology of disease. Medical entomology is now taught in the entomological curricula of most colleges and universities, as well as in curricula of public health, preventive medicine, and tropical medicine.

The general plan of the book remains essentially unchanged; but owing to the rapid expansion of knowledge pertaining to arthropods as vectors of infections of man and animals and because of changes in control technique due to the discovery of new insecticide, such as DDT, a complete revision of nearly all chapters became necessary. Stress continues to be placed on biology (life history and ecology) as fundamental to rational control and as basic to sound epidemiological procedures.

While it is recognized that the advent of DDT did measurably change arthropod-control concepts, the use of this and other new insecticides precipitated new problems or new phases of old ones, particularly as these newer insecticides affect the public health either directly or indirectly and to some degree also the "balance" in nature. Instead of requiring less knowledge concerning the ecology of living things (so-called "shot-gun" methods of application have too largely prevailed), the wise and successful use of these insecticides requires much more fundamental knowledge of the ecology of the offending organisms as well as of others living in the same niche. Hazards to the health of man and his domestic animals must be well considered. Those in charge of training military personnel for vector control operations (to check malaria, tsutsugamushi disease, etc.) under military conditions must recognize, as do public health officials, that sole reliance on the techniques of chemical control (use of insecticides) and the neglect of knowledge of vectors can lead only to failure in the long run.

Among the many persons to whom I am indebted for assistance I wish to thank Professor E. O. Essig, my successor as head of the Division of Entomology and Parasitology, University of California, for placing clerical and other assistance at my disposal. Thanks are due Professors W. M. Hoskins, M. A. Stewart, R. Craig, and Dean Furman for technical assistance, and to Dr. W. C. Reeves, H. F. Gray, Willis Doetschmann, and Richard Coleman for critical reading of certain chapters. I wish to thank Arve Dahl and Richard Peters of the Bureau of Vector Control of the California State Department of Public Health for many courtesies extended during the preparation of this edition. My thanks go particularly to Mrs. K. E. Frick for the task of typing the manuscript. To my wife for long-continued encouragement I owe more than words can express.

W. B. H.

Berkeley, California
April, 1949

PREFACE TO THE FIRST EDITION

MUCH of the matter contained in the following pages was prepared for the press more than six years ago, but owing to the rapid advances made in the field of parasitology, particularly concerning insects, the writer has withheld it until this time, when, after considerable revision and addition, it has seemed expedient to publish the same. The manuscript has been in almost constant use for a period of six years in teaching classes in parasitology, both in the University of California and in the San Francisco Veterinary College. It has been the aim to include herewith a large part of the writer's original work, some of which has until now remained unpublished, as well as the published observations of many other investigators in this field, all of which has gone to build up the foundation of the new science of *medical entomology.*

This book is not intended to be a comprehensive treatise touching all the investigations in the field of medical entomology, but rather an attempt to systematize the subject and to assist in securing for it a place among the applied biological sciences. However, a discussion is included of all of the more important diseases and irritations of man and of the domesticated animals in which insects and arachnids are concerned, either as carriers or as causative organisms.

Owing to the immense literature on insects as relating to disease, much of which is widely scattered, the student in this field must spend considerable time in searching for the desired information and, what is more important, the information is not readily accessible to the physician, the veterinarian, the health officer, and the sanitarian. It is therefore to be hoped that this book will not only prove useful as a text, but also as a handbook for all individuals who are professionally interested in the health and well-being of man and beast, as affected by insects and arachnids.

In the second place detailed accounts of experiments are included here and there, so that the investigator might employ the methods described in either the repetition of the work or in carrying on further investigations along the lines suggested.

Although many special papers have been consulted in the preparation of this work, a bibliography is not included herewith, inasmuch as this information is obtainable in much more complete form in the bibliographical works of other writers. Reference to special papers is usually made in footnote form, but where certain facts have long been accepted as common knowledge, reference is ordinarily omitted.

Sources from which assistance has been drawn are too numerous to enumerate adequately, but to all who have contributed toward the preparation of this work I wish to express my sincere appreciation and thanks, but most particularly to my advanced students in parasitology, who have contributed much valuable data, and to my colleagues, Professor C. W. Woodworth, Dr. Edwin C. Van Dyke, Dr. W. A. Sawyer, and Dr. S. B. Freeborn, and to my wife, Lillie M. Herms, for generous cooperation and kindly criticism.

Unless otherwise credited the illustrations are from photographs and drawings made by the author and various assistants. Thanks are due particularly to Dr. William Colby Rucker for the use of flea drawings, to Professor Herbert Osborn for permission to reproduce certain drawings of biting and sucking lice, to Dr. Bruce Mayne for photographs of *Tabanas striatus,* and stomachs of infected *Anopheles* mosquitoes, to Professor J. S. Hine for photographs of certain other tabanids, and to Mr. W. C. Matthews, scientific illustrator, for valuable assistance in the preparation of many of the figures.

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CHAPTER I

INTRODUCTION

Historical. In the King James version of the Old Testament (Exodus 8: 24) we read, " . . . and there came a grievous swarm of flies into the house of Pharoh, and into his servants' houses, and into all the land of Egypt: the land was corrupted by reason of the swarm of flies." The Douay Version reads, "and the land was corrupted by this kind of flies." Whether the term "flies" as used in this passage is coextensive with the modern use of the word may be questioned, but it is interesting to contemplate the implications. As early as 1577 Mercurialis[1]* expressed the belief that flies carried the virus of plague from those ill or dead of the disease to the food of the well. Although we now know that this is not the usual mode of plague transmission, the principal role that flies play as vectors of disease was correctly interpreted; i.e., they are food contaminators.

In 1587 Gabriel Soares de Souza[2] stated that flies suck poisons from sores (*Framboesia tropica*) and leave them in skin abrasions on healthy individuals, thus infecting many persons. In 1769 Edward Bancroft[3] advanced a similar theory, but it was not until 1907 that Castellani[4] demonstrated experimentally that flies do transmit *Treponema pertenue* Castellani, the causal agent of yaws. The role of the housefly as a vector of disease was, however, not fully appreciated until 1898 when, as the result of investigations made during the Spanish-American War, Veeder[5] wrote:

> I have made cultures of bacteria from fly tracks and from the excrement of flies and there seems to be not the slightest difficulty in so doing. Indeed the evidence of every sort is so clear that I have reached the conclusion that the conveyance of infection in the manner indicated is the chief factor in decimating the army. Certainly so far as is known to the writer, nothing adequate has been said about it in current discussions.

Although popular beliefs in many parts of the world had for some time connected mosquitoes with various tropical fevers, no well-formulated ideas were advanced until 1848, when Josiah Nott[6] of New Orleans published his belief that mosquitoes gave rise to both malaria and yellow

* Superior figures in the text refer to Bibliography at the end of each chapter.

fever. In 1854 Beauperthuy,[7] a French physician in the West Indies, formulated an excellent theory that mosquitoes were responsible for the transmission of yellow fever, believing, however, that the unknown disease factor was carried by the insect from certain decomposing matter and in turn introduced by it into the human body.

While considerable was known by early naturalists and physicians concerning the larger intestinal parasites, such as roundworms and tapeworms, little information relating to microörganisms was available until after the development of the microscope by Anton van Leeuwenhoek (1695),[8] who found that his "material contained many tiny animals which moved about in a most amusing fashion; the largest of these showed the liveliest and most active motion, moving through the water or saliva as a fish of prey darts through the sea." This discovery led to the study of hitherto invisible organisms and eventually to the formulation of the "germ theory" by Pasteur[9] in 1877.

Although according to Howard[10] no standard medical treatise mentioned any specific disease as insect-borne prior to 1871, Raimbert[11] showed in 1869 by experiment (inoculation of proboscides, wings, etc., of nonbiting muscids into guinea pigs) that anthrax (*Bacillus anthracis* Cohn) could be disseminated by flies, which as early as 1776 was believed by Montfils[12] to be the case. The first discovery of primary importance in the field of medical entomology, however, was made in 1878 by Patrick Manson[13] who, working in China, observed the development of *Wuchereria* (= *Filaria*) *bancrofti* (Cobbold) in the body of a mosquito, *Culex quinquefasciatus* Say (= *Culex fatigans* Wied.), and eventually, together with Bancroft, Low, and others, proved the mosquito to be the intermediary host and vector of the causal organism of filariasis.

The discovery by Laveran[14] in 1880 of the causal organism of malaria (*Plasmodium malariae*) living parasitically in the red blood cells of man, marks an epoch in protozoölogy. Taking rank with Laveran's discovery of the malaria parasite is the discovery by Theobald Smith in 1889 (Smith and Kilbourne, 1893[15] of the causal protozoön organism (*Babesia bigemina*) of Texas cattle fever, also living parasitically within the red blood corpuscles of the host. Associated with Smith in the investigation of the disease was F. L. Kilbourne, and together in 1893 they made the second great fundamental discovery in the field of medical entomology, namely that the cattle tick, *Boöphilus annulatus* (Say) = (*B. bovis* Riley), is the necessary intermediate host of the causal agent of the disease. This knowledge, combined with Manson's discovery concerning mosquitoes and filariasis, established a new basis for the control and prevention of disease in both man and domestic animals.

In quick succession there followed a series of famous discoveries. In 1895 Bruce[16] investigated nagana, the fatal tsetse-fly disease of Africa

(Zululand) and established the fact that the infection is conveyed from animal to animal through the agency of *Glossina morsitans* Westwood.

In 1897 Ronald Ross[17] announced that he had found the zygotes of the malaria parasite in two "dapple-wingled mosquitoes" (anophelines) which had been bred from the larva and fed on a patient whose blood contained crescents. In the discovery that mosquitoes carry malaria there are linked the names of Ross; Manson;[18] MacCallum;[19] Bastianelli, Bignami, and Grassi;[20] Koch;[21] and Sambon and Low,[22] the last two having (1900) demonstrated beyond a doubt the fact of transmission.

One of the world's outstanding achievements in the field of experimental medicine is that of the United States Army Yellow Fever Commission, consisting of Reed,[23] Carrol, Lazear, and Agramonte, which in 1900 on the island of Cuba proved conclusively that yellow fever is carried by a mosquito, *Aedes aegypti* (Linn.) [then known as *Culex fasciatus* Fabr. and later as *Stegomyia fasciata* (Fabr.)]. Carlos Finlay,[24] a Cuban physician, had as early as 1880 propounded the theory and conducted experiments in an attempt to prove it; hence he, too, amply deserves recognition and great praise.

These two discoveries concerning malaria and yellow fever gave great impetus to the subject of mosquito control, although L. O. Howard had already demonstrated the value of kerosene in his experiments in the Catskill Mountains in 1892. Howard's pioneer book entitled *Mosquitoes: How They Live; How They Carry Disease; How They Are Classified; How They May Be Destroyed,* appeared in 1901.

During almost a third of a century following these fundamental discoveries little advance was made in knowledge concerning the transmission of malaria and yellow fever, and the complete solution of the problem of control of both diseases seemed to be within reach—that is, simply mosquito control. However, in 1937 malaria was again referred to as a mysterious disease by Hackett[25] in his treatise *Malaria in Europe* vis.: " . . . under close examination malaria became only more intricate and impenetrable, more protean in its character, more diverse in its local manifestations." The expression "anophelism without malaria" came into use, and malariologists became more interested, as Hackett points out, in the anophelines which did not transmit malaria than in those that did. The discovery by Falleroni[26] in 1926 that *Anopheles maculipennis* Meigen, an important vector of malaria, was in reality separable into races on differences in the egg pattern, led Hackett, Martini, and Missiroli[27] (1932) to the discovery that the races of this species differ markedly in their vector relationship to malaria, thus opening new avenues of research. Now what appeared to be a clear-cut situation in 1898 became once more a malaria "puzzle."

Furthermore, the apparently well-solved problem of yellow fever control through the control of *Aedes aegypti* (Linn.) was again completely thrown open for further investigation by the discovery of Stokes, Bauer, and Hudson[28] in 1927 that experimental animals (monkeys) can be infected with yellow fever. Now, because of the availability of experimental animals, more than a dozen species of mosquito instead of just one species are known to have the ability to transmit the infection from monkey to monkey by the bite.

In 1932 there was first observed in the Valle de Chanaan, Espirito Santo, Brazil, a type of yellow fever designated as jungle yellow fever (Soper, 1936),[29] differing from the known type, transmitted usually by *Aedes aegypti* (Linn.), only in that it occurs under conditions suggesting that infection takes place away from urban habitations, that man may not be an essential factor in the continuity of infection, indeed "man may be but an accident in the course of an epizoötic in the lower animals, or it may even be due to the persistence of the virus in invertebrate vectors for long periods of time."

In 1898 Simond[30] succeeded in transmitting plague from a sick rat to a healthy rat through the agency of infected fleas. This discovery was at first discredited, but the experiments were successfully repeated by Verjbitski[31] in 1903 and Liston[32] in 1904.

The designation sylvatic (selvatic) plague has come into use particularly since 1928 (Ricardo Jorge[33]) to specify plague of wild rodents in which fleas play an important role as invertebrate reservoirs as well as vectors.

At this juncture of our historical review of the subject it is appropriate to call attention to the first comprehensive treatise dealing with arthropods as carriers of disease, namely the work of the late Professor George H. F. Nuttall[34] entitled, *On the Role of Insects, Arachnids and Myriapods as Carriers in the Spread of Bacterial and Parasitic Diseases of Man and Animals. A Critical and Historical Study.* Every student of medical entomology should be familiar with this publication. The following quotation from that work is significant:

> Whilst hygienists have given much attention to the study of pathogenic organisms in air, water, soil and food, their behavior under different chemical and physical conditions, as also to the possibility of their direct or indirect transmission from diseased to healthy individuals; relatively little attention has been paid to one of the means by which infectious diseases are spread, to the rôle played especially by insects, which may serve either as carriers or intermediary hosts of disease-agents. The most thorough work in this direction has been done by parasitologists. Very few of the works on hygiene even mention the rôle of insects as carriers of infection, and those that do, generally speak vaguely on the subject.

Nuttall deserves to be called the father of medical entomology.

In 1901 Forde[35] observed certain parasites in the blood of persons suffering from Gambian sleeping sickness, which Dutton[36,37] recognized as trypanosomes and named *Trypanosoma gambiense;* and in 1903 Bruce and Nabarro[38] showed that *Glossina palpalis* (Robineau-Desvoidy) was the carrier, thus adding another tsetse-fly disease to the list. Stephens and Fantham[39] in 1910 described *Trypanosoma rhodesiense* as the causal organism of Rhodesian sleeping sickness, and Kinghorn and Yorke[40] in 1912 proved *Glossina morsitans* Westwood to be the responsible vector.

Graham,[41] while working in Syria in 1902, found that dengue, or breakbone fever, a widely distributed disease particularly of warm climates, though frequently occurring elsewhere, is mosquito-borne. He, and later Ashburn and Craig,[42] reported that possibly several species of mosquitoes, notably *Aedes aegypti* (Linn.), are able to transmit the infection. A closely related disease is pappataci fever, also known as "three-day fever" and "sand fly fever," transmitted by *Phlebotomus papatasii* Scopoli, as proved by Doerr, Franz, and Taussig[43] in 1909.

In 1903 Marchoux and Salimbeni[44] proved that fowl spirochetosis caused by *Spirochaeta gallinarum* Blanchard is tick-borne and that *Argas persicus* (Oken), the common fowl tick, is a vector. Another tick-borne disease came to light when Dutton and Todd[45] and Ross and Milne[46] in 1904 discovered that African relapsing fever is carried by the tick *Ornithodoros moubata* (Murray), the causal organism being *Borrelia* (= *Spirochaeta*) *recurrentis* Lebert = (*Spirochaeta duttoni* Novy and Knapp). Furthermore, in 1906 Ricketts,[47] working in Montana (U.S.A.), proved conclusively that another tick, which he believed to be *Dermacenter occidentalis* Neum., but now known to be *Dermacentor andersoni* Stiles [*Dermacentor venustus* (Banks)], is the principal vector of Rocky Mountain spotted fever. Wolbach[48] (1919) named the causal organism *Dermacentroxenus rickettsi.*

Although lice have for centuries been associated with filth and disease, apparently little thought was given these insects as possible carriers of infection, even though Melnikoff[49] had shown in 1869 that the biting dog louse, *Trichodectes canis* DeGeer, was an intermediate host of the double-pored dog tapeworm, *Dipylidium caninum* (Linn.), which also occasionally occurs in humans. Aubert[50] (1879), according to Nuttall, considered that pediculi were spreaders of impetigo, and the cause of prurigo, pityriasis, etc. Also, in experiments conducted by Dewèvre[51] in 1892, lice were shown to carry the specific microörganisms mechanically on their front legs, and infection was thus accidentally transmitted to healthy persons. Furthermore, Flügge[52] in 1891 and Tictin[53] in 1897 both supposed that disease might be carried by vermin and conducted experiments with bedbugs. In 1907 Mackie[54] working in India found that relapsing fever was transmitted by the body louse, *Pediculus humanus*

Linn., in whose body the causal organism, *Borrelia* (= *Spirochaeta*) *recurrentis* Lebert (*S. carteri* Manson), multiplies.

Nicolle, Comte, and Conseil,[55] working in Tunis in 1909, and Ricketts and Wilder[56] working independently in Mexico in 1910, proved experimentally that the body louse (*Pediculus humanus* Linn.) is a carrier of typhus fever, the causal organism of which, *Rickettsia prowazeki*, was described and named by Da Rocha-Lima[57] in 1916.

Members of the insect family Reduviidae (conenose bugs or kissing bugs) have been long known for their fierce bites and bloodthirstiness, but it was apparently not until 1909 that insects of this group were experimentally proved to be disease carriers. In that year Chagas,[58] who had already described the causal organism [*Schizotrypanum* (= *Trypanosoma*) *cruzi*,] of Chagas' disease, also known as "Brazilian trypanosomiasis," demonstrated that this disease is carried by the conenose bug *Mestor megistus* (Burm.) [*Triatoma megista* (Burm.) = *Panstrongylus megistus* (Burm.)]. In 1933 Kofoid and Donat[59] showed that the trypanosome of the conenose bug *Triatoma protracta* (Uhler) in California is identical with that found in *Mestor megistus* (Burm.).

Flies of the family Tabanidae (horseflies, gadflies, earflies, deer flies, etc.) were looked upon with suspicion as early as 1776, but apparently no satisfactory evidence against them was forthcoming until 1913, when Mitzmain[60] (Mayne), working in the Philippine Islands, demonstrated transmission of surra of the carabao by *Tabanus striatus* Fabr., which he regarded as the principal carrier. Strong evidence against tabanid flies of the genus *Chrysops* as intermediary hosts of *Loa* (= *Filaria*) *loa* (Cobbold) was advanced by Leiper,[61] also in 1913.

Bloodsucking gnats belonging to the dipteron family Simuliidae are a terrible scourge to both man and beast in many parts of the world and have long been under suspicion as vectors of disease. In 1926 Blacklock[62] reported *Simulium damnosum* Theob. as the vector of the filarial worm *Onchocerca volvulus* (Leuckart), the causal agent of onchocercosis. In 1934 O'Roke[63] reported *Simulium venustum* Say to be the vector of a disease of ducks caused by *Leucocytozoön anatis* Wickware.

Tularemia, also known as Pahvant Valley plague (Utah, U.S.A.) or deer fly fever, was shown by Francis and Mayne[64] in 1921 to be carried from rodent to rodent by the tabanid fly *Chrysops discalis* Williston, and presumably from rodent to man in the same manner. The causal organism of this disease, *Pasteurella* (= *Bacterium*) *tularensis*, was described in 1911 by McCoy and Chapin as the cause of a plague-like disease of California ground squirrels. Though transmitted in nature by the deer fly and several other species of arthropods, particularly the tick *Dermacentor andersoni* Stiles, which is involved hereditarily, the infection is most commonly contracted by handling infected rabbits.

In 1933 Kelser[65] announced that he had succeeded in transmitting the virus of equine encephalomyelitis from inoculated guinea pigs to a horse by the bite of the mosquito *Aedes aegypti* (Linn.).

The discovery of the insecticidal value of the compound dichloro-diphenyl-trichloroethane (DDT) represents the beginning of a new era in the prevention of insect-borne diseases as pertains to both plants and animals, particularly of such a devastating disease of man as malaria. This compound was first synthesized by Othmar Zeidler[66] in 1874 at Strasbourg, Germany. Zeidler apparently was ignorant of its insecticidal properties. It was not until about 1939 that Paul Müller, (Läuger *et al.*[67]) then a member of the scientific staff of Geigy Company in Basle, Switzerland, discovered its remarkable insecticidal value. For this discovery Müller was awarded the Nobel Prize in Medicine for 1948.

Both historically and for future investigation the relation which the protozoön subfamily Herpetomoninae of Castellani and Chalmers bears to insects and their relation to animal and plant diseases, is one of interest to parasitologists. Numerous insects are known to harbor *Leptomonas* (inclusive of *Herpetomonas*), *Crithidia, Leishmania,* and other genera, some of doubtful classification, but the problem of segregating those which are zoö- and phytopathogenic from those which are merely entomoparasitic in exceedingly difficult and fraught with snares and pitfalls. Apparently the earliest discovery in this connection concerning plants was made by Lafont[68] in 1910 when he demonstrated that *Leptomonas davidi* Lafont, the cause of "flagellosis" in three species of Euphorbiaceae, required as its intermediary host the bug, *Nysius euphorbiae* Horvath.

BIBLIOGRAPHY

1. Mercurialis (Hieronymus), 1577 (?). *De pestes in universum, praesertim vero de Veneta et Patavina. Item de morbis cutaneis, et omnibus humani corporis excrementis.* (Not available to the author.)

2. de Souza, Gabriel Soares, 1587. *Tratado descriptivo do Brazil em 1587, obra de Gabriel de Souza.* Rio de Janeiro: Typographia universal de Laemmert, 120 pp. (Cited by França, C., in *Tr. Roy. Soc. Trop. Med. & Hyg.,* 15:58–60.)

3. Bancroft, Edward, 1769. *An Essay on the Natural History of Guiana in So. America.* London: T. Becker and P. A. De Houdt, 402 pp.

4. Castellani, Aldo, 1907. "Experimental investigation on *Framboesia tropica* (Yaws)," *J. Hyg.,* 7:558–59.

5. Veeder, M. A., 1898. "Files as spreaders of sickness in camps," *Med. Rec.,* 54:429–30.

6. Nott, Josiah C., 1848. "On the origin of yellow fever," *New Orleans M. & S. J.,* 4:563–601.

7. Beauperthuy, L. D., 1854. "Transmission of yellow fever and other diseases by mosquito," *Gazeta oficial de Cumanà,* Año. 4, no. 57, May 23. (Cited by Howard, Dyer, and Knab.)

8. Leeuwenhoek, Anton van, 1695. *Arcana naturae detecta ope microscopiorum.* Kronevelt: Delphis Batavorum, 568 pp. (Cited by Howard, Dyer, and Knab.) *See also The Select Works of Antony van Leeuwenhoek,* translated by Samuel Hoole, London, 1798.

9. Pasteur et Joubert, 1877. "Chimie Physiologique: Etude sur la maladie charbonneuse," *Compt. rend. Acad. d. sc.*, 84:900–906.

10. Howard, L. O., 1921. "Sketch History of Medical Entomology," in *A Half Century of Public Health,* M. P. Ravenel. New York: Amer. Pub. Health Assn., pp. 412–38.

11. Raimbert, A., 1869. "Recherches experimentales sur le transmission du charbon par les mouches," *Compt. rend. Acad. d. sc.*, 69:805–12. (Cited by Nuttall.)

12. Montfils, A. J., 1776. "D'une maladie fréquente connue en Bourgogne sous le nom de Puce maligne," *J. de méd.*, 45:500. (Cited by Nuttall.)

13. Manson, Patrick, 1878. "On the development of *Filaria senguinus hominis,* and on the mosquito considered as a nurse," *J. Linn. Soc., Zool.*, London, 14:304–11.

14. Laveran, A., 1880. "Note sur un nouveau parasite trouvé dans de sang de plusieurs malades atteints de fievre palustre," *Bull. Acad. de méd.*, Paris, 9:1235.

15. Smith, Theobald, and Kilbourne, F. L., 1893. *Investigations into the Nature, Causation, and Prevention of Texas or Southern Cattle Fever.* Washington, D. C.: U. S. Dept. Agric. in Bur. Animal Indust. Bull., no. 1. 301 pp.

16. Bruce, David, 1895. *Tsetse-Fly Disease or Nagana in Zululand: Preliminary report.* Durban: Bennett and Davis. (Cited by Nuttall.)

17. Ross, R., 1897. "On some peculiar pigmented cells found in two mosquitoes fed on malarial blood," *Brit. M. J.*, 2:1786–88.

18. Manson, P., 1898. "Surgeon-Major Ronald Rose: Recent investigations on mosquito-malaria theory," *Brit. M. J.*, 1:1575–77.

19. MacCallum, W. C., 1898. "On the haematozoan infections of birds," *J. Exper. Med.*, 3:117–36.

20. Bastianelli, G.; Bignami, A. E.; Grassi, B.; 1898. "Coltivazione delle semilune malariche dell' uomo nell' *Anopheles claviger* Fabr: Note preliminare." *Atti della Reale Accad. del Lincei,* Nov. 28, p. 313. (Cited by Nuttall.)

21. Koch, R., 1899. "Ueber die Entwicklung der Malariaparasiten," *Ztschr. f. Hyg. u. Infektionskr.*, 32:1–24.

22. Sambon, L. W., and Low, G., 1900. "The malaria experiments in the Campagna," *Brit. M. J.*, 2:1679–82.

23. Reed, Walter, 1900. "The etiology of yellow fever," *Philadelphia Med. J.*, 6:790–96.

24. Finlay, Carlos J., 1881 *et seq. Trabajos selectos.* Havana: República de Cuba, Secretaría de Sanidad y Beneficencia, 1912. xxxiv + 657 pp.

25. Hackett, L. W., 1937. *Malaria in Europe: An ecological study.* Oxford University Press, London: Humphrey Milford. xvi + 336 pp.

26. Falleroni, D., 1926. "Fauna anofelica italiana e suo habitat' (paludi, risaie, canali). Metodi di lotta contro la malaria," *Riv. di Malariol.*, 5:553–93.

27. Hackett, L. W.; Martini, E.; and Missiroli, A.; 1932. "The races of *A. maculipennis,*" *Am. J. Hyg.*, 16:137–62.

28. Stokes, Adrian; Bauer, J. H.; and Hudson, N. Paul; 1928. "The transmission of yellow fever to *Macacus rhesus:* Preliminary note," *J.A.M.A.*, 90: 253–54.

29. Soper, Fred L., 1936. "Jungle yellow fever: A new epidemiological entity in South America," *Rev. de hyg. e saude pub.*, 10:107–44.

30. Simond, P. L., 1898. "La propagation de la peste," *Ann. Inst. Pasteur,* 12:625.

31. Verjbitski, D. T., 1908. "The part played by insects in the epidemiology of plague," *J. Hyg.*, 8:162–208.

32. Liston, W. G., 1905. "Plague rats and fleas," *J. Bombay Nat. Hist. Soc.*, 16:253–73.

33. Jorge, Ricardo, 1928. *Les faunes regionales des rongeurs et des puces dans leurs rapports avec la peste.* Paris: Masson et Cie. 306 pp.

34. Nuttall, G. H. F., 1899. "On the role of insects, arachnids, and myriapods as carriers in the spread of bacterial and parasitic disease of man and animals. A critical and historical study." *Johns Hopkins Hospital Reports,* 8:1–154.

35. Forde, R. M., 1902. "Some clinical notes on a European patient in whose blood a Typanosoma was observed," *J. Trop. Med.*, 5:261.

36. Dutton, J. E., 1902. "Trypanosoma in man," *Brit. M. J.*, 1:42.

37. ———, 1902. "Note on a Trypanosoma occurring in the blood of man," *Brit. M. J.*, 2:881–884.

38. Bruce, D., and Nabarro, D., 1903. *Progress Report on Sleeping Sickness in Uganda.* "Rept. Sleeping Sickness Comm., Roy. Soc. London," no. 1.

39. Stephens, J. W. W., and Fantham, H. B., 1910. "On the peculiar morphology of a trypanosome from a case of sleeping sickness and the possibility of its being a new species (*T. rhodesiense*)," *Proc. Roy. Soc. London,* ser. B, 83:28–33.

40. Kinghorn, A., and Yorke, W., 1912. "On the transmission of human trypanosomes by *Glossina morsitans* Westw., and on the occurrence of human trypanosomes in game," *Ann. Trop. Med.*, 6:1–23.

41. Graham, H., 1902. "Dengue: A study of its mode of propagation and pathology," *Med. Rec.*, 61:204–7.

42. Ashburn, P. M., and Craig, C. F., 1907. "Experimental investigations regarding the etiology of dengue fever," *J. Infect. Dis.*, 4:440–75.

43. Doerr, R.; Franz, K.; and Taussig, S.; 1909. *Das Pappatacifieber.* Leipzig u. Wien.: Franz Deutiche. 166 pp.

44. Marchoux, E., and Salimbeni, A., 1903. "La spirillose des poules," *Ann. Inst. Pasteur,* 17:569–80.

45. Dutton, J. E., and Todd, J. L., 1905. *The Nature of Human Tick Fever in the Eastern Part of the Congo Free State, with Notes on the Distribution and Bionomics of the Tick.* Liverpool School Trop. Med. in Memoir no. 17. 18 pp.

46. Ross, P. H., and Milne, A. D., 1904. "Tick fever," *Brit. M. J.*, 2:1453–54.

47. Ricketts, H. T., 1906. "The transmission of Rocky Mountain spotted fever by the bite of the wood tick (*Dermacentor occidentalis*)," *J.A.M.A.*, 57:358.

48. Wolbach, S. B., 1919. "Studies on Rocky Mountain spotted fever." *J. Med. Research,* 41:1–193.

49. Melnikoff, H., 1869. "Ueber die Jugendzustände der *Taenia cucmerina,*" *Arch. f. Naturgesch.,* 25:62–69.

50. Aubert, 1879. "Les pous et les écoles: Un point d'hygiène scolaire," Reviewed in *Ann. de Dermat. et Syph.,* 1880, 2 ser., 1:292–93.

51. Dewèvre, 1892. "Note sur le rôle des pediculi dans la propagation de l'impetigo," *Compt. rend. Soc. de biol.,* 4:232–34.

52. Flügge, C., 1891. Grundriss der Hygiene. Leipsig: veit & Co. x + 560 pp. (Cited by Nuttall.)

53. Tictin, J., 1897. "Zur Lehre vom Rückfalltyphus," *Centralbl. f. Bakt.,* 1 Abt., 21:179–86.

54. Mackie, F. P., 1907. "The part played by *Pediculus corporis* in the transmission of relapsing fever," *Brit. M. J.,* 2:1706–9.

55. Nicolle, Charles; Comte, C.; et Conseil, E.; 1909. "Transmission experimentals du typhus exanthimatique par le pou du corps," *Compt. rend. Acad. d. sc.,* 149:486–89.

56. Ricketts, H. T., and Wilder, R. M., 1910. "The transmission of the typhus fever of Mexico (tarbardillo) by means of the louse (*Pediculus vestimenti*)," *J.A.M.A.,* 54:1304–7.

57. Da Rocha-Lima, H., 1916. "Untersuchungen über Fleckfieber," *München. Med. Wchnschr.,* 63:1381–84.

58. Chagas, C., 1909. "Ueber eine neue Trypanosomiasis des Menschen," *Mem. Inst. Oswaldo Cruz,* 1:159–218.

59. Kofoid, Charles A., and Donat, F., 1933. "Experimenntal infection with *Tryanosoma cruzi* from intestine of conenose bug *Triatoma protracta,*" *Proc. Soc. Exper. Biol. & Med.,* 30:489–91.

60. Mitzmain, M. B., 1913. "The mechanical transmission of surra by *Tabanus striatus* Fabr," *Philippine J. Sc.,* 8 (ser. B):223–29.

61. Leiper, Robert R., 1913. "Metamorphosis of *Filaria loa,*" *Lancet,* 1:51.

62. Blacklock, D. B., 1926. "Development of *Onchocerca volvulus* in *Simulium dammosum,*" *Ann. Trop. Med.,* 20:1–48 and 203–18.

63. O'Roke, Earl C., 1934. *A Malaria-like Disease of Ducks Caused by* Leucocytozoon anatis *Wickware.* Ann Arbor: University of Michigan Press.

64. Francis, Edward, and Mayne, Bruce, 1921. "Experimental transmission of tularaemia by flies of the species *Chrysops discalis,*" U. S. Public Health Service, *Pub. Health Rep.,* 36:1738–46.

65. Kelser, R. A., 1933. "Mosquitoes as vectors of the virus of equine encephalomyelitis," *J. Am. Vet. M. A.,* 82, n.s. 35:767–71.

66. Zeidler, Othmar, 1874. "Verbindungen von Chloral mit Brom und Chlorbenzol," *Ber. d. Deutsch. Chem. Gesellsch.,* 7:1180.

67. Läuger, P.; Martin, H.; and Muller, P.; 1944. "Über Konstitution und toxische Wirkung von natürlichen und neuen synthetischen insektentötenden Stoffen," *Helvet. chimica acta,* 27:892–928.

68. Lafont, A., 1910. "Sur la présence d'un Leptomonas parasite de la classe des Flagellés dans le latex de trois Euphorbiacées," *Ann. Inst. Pasteur* 24:205–9.

CHAPTER II

SCOPE AND METHOD

Scope. The preceding brief outline of the historical development of knowledge pertaining to the relationship of arthropods to diseases, parasitic infestations, and various disorders of man and his domesticated animals will in itself give the student some understanding of the scope of medical entomology. The name, medical entomology, as a designation for this field of knowledge apparently did not come into use until about 1909.[1] Even at that late date there were those in high places who ridiculed the importance of arthropods as vectors of disease in spite of important entomological discoveries relating to the transmission of plague, malaria, and yellow fever. Today medical entomology is not only recognized as a science in its own right, but also takes equal rank with some of the older sciences contributing to the fields of public health, tropical medicine, preventive medicine, and veterinary medicine.

Medical entomology may be defined as the science which deals with the relation of insects, arachnids, and other arthropods to parasitic infestations and diseases of man and beast: they may be causal agents, as in scabies; or vectors, as in the malarias. The science is directly concerned with the biology and control of the offending arthropods. It contributes to the conservation of the public health and the health and well-being of animals.

Training. The medical entomologist must be thoroughly trained in general zoölogy; he must be particularly well trained in entomology and arachnology; and his training must include protozoölogy, helminthology, and bacteriology. He must be a parasitologist in the truest and widest sense. He must be well versed in field ecology. Many insect-borne diseases of man and his domesticated animals are maintained in nature in wild mammalian and other reservoir animals; hence the medical entomologist must have a thorough understanding of vertebrate zoölogy and also a wider knowledge of invertebrates (particularly fresh-water forms) than is offered in the usual courses in zoölogy and entomology. Familiarity with aquatic organisms is also helpful in determining public health implications when these organisms are found in domestic water supplies. Familiarity with insects attacking stored and dried foods, stored grain, and foods in general is valuable. A knowledge of house-invading animals is essential. Entomological training must emphasize immature stages in the developmental cycle. Although mycology is not usually considered essential, the author has derived much benefit from a course in that field and recommends it to students of medical entomology. Much direct benefit will be derived from many of the courses offered in medical, veterinary, and public health curricula, such as general anatomy, general physiology, epidemiology, pathology, histology, toxicology, hematology, and coprology. If available, training in public health administration should be acquired. A substantial knowledge of chemistry is essential. In order to solve certain field problems involving arthropods, for example, mosquitoes and flies, knowledge of such phases of engineering as pertain to drainage and sewage disposal is useful. Familiarity with these last-named subjects will enable the medical entomologist to cooperate intelligently with other medical investigators and with engineers or, on occasion, to make intelligent use of the professional services of physicians, veterinarians, engineers, epidemiologists, and other experts and specialists in the solution of complicated problems. For a more extended consideration of the preparation required for a career as a medical entomologist the student is referred to an article by the author in the *Journal of Economic Entomology* for February, 1943.[2]

Objectives. The aim of medical entomology is the pursuit of knowledge concerning disease vectors and particularly concerning the control and prevention of arthropod-borne diseases. There are many notable examples of the service rendered by workers in this fertile field, such as the control of malaria and yellow fever in Cuba and the Panama Canal Zone,[3] and the campaign against rats and fleas in San Francisco in 1907.[4] The latter resulted in the eradication of plague from that city, thus averting the spread of this terrible scourge to the surrounding area. The eradication during 1939–1940 of *Anopheles gambiae* Giles in Brazil after its devastating introduction from tropical Africa is an excellent recent example.[5] The benefit derived by animal industry is well illustrated in the control of the tick vector [*Boöphilus annulatus* (Say)] of Texas cattle fever in the southern United States.

Method. In common with researchers in other related fields, the medical entomologist must employ the experimental method to advance his science.[6] The experimental method is described by Thomas Hunt Morgan (1907)[7] in *Experimental Zoölogy* (by permission of The Macmillan Company, publishers) as "the most important tool of research that scientists employ. . . . The essence of the experimental method consists in requiring that every suggestion (or hypothesis) be put to the test of experiment before it is admitted to a scientific status. . . . It is the method of attacking problems that is the chief characteristic of experimental work. . . . We demand in the case of a problem in experimental science that the conditions under which an event takes place be discovered, and that, if possible, we reproduce artificially the result by controlling the conditions. In fact the control of natural phenomena is the goal of experimental work." Ivy (1948)[8] states, "It has been through the experimental method of controlled or conditioned observation, and *only* through this method, that scientists have discovered and will continue to discover the most intimate secrets of Nature."

Since discovery of disease vectors and development of methods for the control of arthropod vectors and reservoir animals are aims of medical entomology, it is highly important that the experimental method be employed. An examination of the literature pertaining to arthropods and disease shows that much of the work in this field is still largely in the descriptive stage. Knowledge of taxonomy, morphology, anatomy, histology, and physiology of arthropods is fundamental to sound work in the field of medical entomology, but such knowledge alone, even of insects of public health importance, does not *per se* qualify one as a medical entomologist. Referring to the importance of fundamental knowledge, Morgan (*loc. cit.*) states: "The carrying out of an experiment implies the formulation of a working hypothesis, and this usually presupposes some knowledge of the possible conditions that control the phenomena. The experimental work becomes more explicit and accurate the more we know beforehand of the possible conditions that may enter into the result. . . . "

In the experimental study of insect vectors one would ordinarily first determine by experiment whether or not laboratory animals are susceptible to a certain disease. If they are, further experimentation is greatly simplified. After long years of slow progress, rapid strides were made in our knowledge of yellow fever when it was discovered that laboratory animals such as monkeys and white mice could be used in experimentation.[9,10]

Well does the author remember the experiment completed December 16, 1909, which proved *Diamanus montanus* (Baker) = (*Ceratophyllus acutus* Baker), the ground-squirrel flea, to be a transmitter of plague from ground squirrel to ground squirrel. The cage used by the United States Public Health Service in San Francisco for the experimental animals was an ordinary galvanized iron garbage can 33 inches high by 19 inches in diameter, suitably screened and smeared at the rim with "tanglefoot" to prevent the escape of the fleas. Every possible precaution was taken to obviate danger. McCoy[11] describes the experiment as follows:

A ground squirrel was inoculated subcutaneously with a broth culture of *Bacillus pestis* derived from a human case of plague. This animal died on the fifth day. Three days before its death, one hundred fleas *(Ceratophyllus acutus)* were put in the cage with it. While yet warm the dead rodent was removed from the cage, and twenty-seven live fleas were taken from its body. Two of these were crushed, and staining of the resulting smears showed an abundance of pest-like bacilli in each. The twenty-five fleas remaining were put in a clean cage with a healthy squirrel. This animal died of subacute plague ten days later. . . . This experiment is conclusive in showing that *C. acutus* may convey plague from a sick to a healthy squirrel. It should be stated that all of the squirrels were kept in quarantine for at least a month prior to their being used for the experiment. In fact, all of these squirrels were obtained in a region in which no plague squirrels have ever been found.

Although human malaria was not known to be transmissible to laboratory animals, knowledge of the vector was hastened because a related malaria occurs in birds. Discoveries by MacCallum (*loc. cit.*) on bird malaria furnished the key to the famous discoveries of Ross, Grassi, Bignami, and others. Naturally induced (mosquito-transmitted) as well as blood-induced malaria in the routine therapy of neurosyphilitics has enabled investigators to make progress, heretofore impossible, in the understanding of the behavior of *Plasmodium vivax* and other human malaria plasmodia (Young, 1944).[12] Also the discovery of a malaria infection in monkeys caused by *Plasmodium knowlesi* opened the way for much experimental work applicable to human malaria.[13]

Faust[14] has aptly stated: "In most experimental work with human parasitic infections laboratory animals can be utilized for all practical experimental tests, thus obviating the need for any potential risk by human volunteers. . . . Yet in certain crucial types of experimentation it has been found highly desirable to know if human host-parasite relationships are directly parallel to those of susceptible animals." Less dangerous diseases such as tick-borne relapsing fever have been given human tests (see Chapter XX). Ivy (1948), *loc. cit.*) states: "In the medical sciences, the only method which can clearly reveal and establish the cause, prevention, and treatment of disease is the method of controlled experimentation on animals and volunteer human subjects."

In experiments in the field of medical entomology an unusual amount of care is required to prevent the escape of infected insects. Also much skill and patience are needed to rear an adequate supply of living insects in the laboratory for experimental use and to secure normal feeding responses on the part of imprisoned insects.

Importance of ecology. The importance of ecological knowledge in the investigation of insect-borne diseases has been long stressed by the author. As early as 1909 (*loc. cit.*) he pointed out that "It is essential that the student become familiar with the habits and habitat of the insect in the field, its life history under normal and unusual conditions." Ecology is variously defined, but few of the later proposed definitions define it as well as did Haeckel (1869) when he described it as the "relation of the animal to its organic as well as its inorganic environment." Chapman[15] said, . . . "he [Haeckel] considered oekologie to include the general economy of the household of nature." In many instances man will need to learn how to live in the same world with these now threatening members of that "household." Pearse[16] in a paper on the ecology of parasites points out that "Man has succeeded by changing the environment or *by changing his own characteristics* as a habitat, in ridding himself of many of his parasites."

In dealing with the importance of ecology in relation to malaria, Richard P. Strong[17] has stated

> In addition to these effects of the immediate environment upon the human host, ecological studies must often consider its effects upon the intermediate hosts in instances where they exist. Here, also, climate plays an important rôle, not only in the character of the vertebrate fauna which the region harbors, but especially of the invertebrate fauna. Also, at temperatures below a certain degree, the parasites in the insects which transmit them may be unable to multiply or the insects satisfactorily to breed or even exist, as, for example, the parasites and insects concerned in the transmission of sleeping sickness and of malaria.
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> . . . .
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> The epidemic of malaria with its high mortality which has recently been raging in Ceylon, India, is a striking example of the effect that climatic conditions and environment may exert upon a disease. This epidemic has occurred in what has been hitherto regarded as the most healthy and prosperous portion of the island, the southwestern part, in which there has usually been a high annual rainfall and where there has been evidence that the percentage of the population infected with malarial parasites has been but small, and hence the population relatively non-immune to the disease. This year the prevailing rains which are brought so regularly by the southwest monsoon failed to supply the usual amount of water, resulting in a prolonged drought. Then came a few heavy rains and drought again. Thus conditions arose greatly favoring the breeding of the mosquito. *Anopheles culicifacies,* which transmits the disease in this region, as many shallow pools were formed along the river beds and streams. Through these innumerable temporary breeding places, more perfect conditions for the production of mosquitoes could probably not have been devised. The outbreak of malaria was followed by failure of the crops, also due particularly to the lack of rain. Thus the people became further impoverished and the general state of their health reduced, and within five months there were 113,811 deaths, of which 66,704 were estimated to be due to malaria.

The complexity of ecological factors as pertains to medical entomology is well shown in malaria, in which three animal species are involved, namely, *man* the victim, the vector *mosquito,* and the causal *plasmodium*—each species having characteristic ecological requirements. The complexity becomes even greater when a reservoir animal enters the epidemiological picture, as in endemic (tick-borne) relapsing fever in California. In the cycle of this disease *man* is again involved, then there is a *tick* vector, a *spirochete* as the pathogen, and a fourth species, a *chipmunk,* as a reservoir. Meyer[18] has clearly portrayed the complexity of an ecological approach to the study of plague in his De Lamar lecture, entitled "The Ecology of Plague."

The student of medical entomology will do well to sturdy Uvarov's[19] *Insects and Climate,* Martini's[20] *Wege der Seuchen,* and Buxton's[21] "The Effect of Climatic Conditions upon Populations of Insects." The latter author (p. 326) remarks, " . . . the geographical spread of human diseases and the seasonal occurrence of certain epidemics appear to be directly due to alteration in the numbers of insects which are the essential vectors of these diseases. Our ultimate objective is to know the numbers of particular sorts of insects which are capable of infecting us with the organisms which they carry."

Control of insect-borne diseases. The control of insect-borne diseases involves not only the control of the responsible insect vectors, often very difficult or even at times impossible, but depends also upon the control of the fomites from which the arthropod receives its infection.

In the simplest form of insect transmission, i.e., by mechanical contamination of food and drink, the source of infection may be found in human excreta or other dangerous animal wastes, in which case the possibility of spread by insects may be largely overcome by correcting the defect in sanitation. Properly constructed fly-tight privies and septic tanks would largely prevent the spread of typhoid fever and related filth diseases by flies in rural areas. Rodent control is intended to destroy the natural reservoirs of plague, as the flea is usually only an agent of transmission. The control of plague through flea control alone does not satisfy all requirements.

The handling of persons with infectious diseases is a matter of great importance, as for example the screening of yellow fever patients against the mosquito vector during the critical infectious stage of the disease. Furthermore the proper screening of malaria patients against mosquitoes is a factor in malaria control. Searching out and adequately treating carriers, while beset with many difficulties, should not be overlooked in a program for control of insect-borne disease. There are situations where vector control is not economically possible; for example, there are highly malarial areas where mosquito control cannot be economically practiced, yet certain agricultural procedures or construction work must go on. In such cases suppressive (prophylactic) treatment is indicated.

In the face of universal rapid transportation by airplane and other means, including automobile, and in the light of wartime experience, health authorities of states and nations should be thoroughly familiar with the vector potentialities of arthropods within their respective geographical boundaries (Herms, 1947).[22] Vigilance at the point of departure and proper quarantine measures at the destination should be maintained to avoid the spread of arthropod-borne diseases.[23]

Vector control. In the control of disease-transmitting arthropods the most vulnerable point in the life history is usually sought and the most effective control means are then employed. This involves an intimate knowledge of biology. The more familiar one is with the life history, habits, and ecology of the vector, the better equipped one will be to cope with the problems of control.

Control measures may be only of a temporary nature for purposes of immediate relief and hence must be repeated; or they may be intended to have permanent effect. Temporary control measures involve holding a nuisance in check for a short time, a few hours or a few days, and require constant repetition: for example, the use of aerosols to kill adult mosquitoes and flies, the use of mosquito repellents, or even the application of oil to mosquito-breeding pools. Permanent control, on the other hand, involves correction of breeding places by mechanical or other means in order to prevent vector breeding: for example, draining or filling unnecessary ponds and pools of standing water in which mosquitoes may breed; the correction of irrigation defects, particularly errors in drainage; the disposal of manures and organic wastes in such a manner as to prevent breeding of houseflies, e.g., close packing or immediate spreading of manure. Permanent control measures are usually more economical in the long run.

Species sanitation implies that control measures aimed at disease vectors must fit the particular offending species. The importance of this procedure is particularly applicable in vector-control operations where closely related species or subspecies occur in the same general area but only one is of public health importance. By concentrating appropriate efforts on the proven vector species or subspecies, for example, in malaria control operations in the presence of two or more anopheline species, good results may be economically obtained.

Naturalistic control implies a planned change in the natural habitat of an offending species, such as a disease vector, so as to make it impossible for the species to continue breeding in effective numbers. For example, *Anopheles albimanus* Wied., a potent vector of malaria in the Caribbean area, breeds abundantly in certain coastal lagoons having a salinity between 15 and 25 per cent. By simply connecting these lagoons with the sea so as to facilitate tidal action and thus increase the salinity

2. ——, 1943. "Preparation for a career as a medical entomologist," *J. Econ. Entomol.*, 36:18–22.

3. Le Prince, Joseph A., and Orenstein, A. J. 1916. *Mosquito Control in Panama: The eradication of malaria and yellow fever in Cuba and Panama.* New York: G. P. Putnam's Sons. 355 pp.

4. Todd, F. M., 1909. *Eradicating Plague from San Francisco: Report of Citizens Health Committee.* San Francisco: C. A. Murdock Co. Press. 313 pp.

5. Soper, Fred L., and Wilson, D. Bruce, 1943. *Anopheles gambiae in Brazil 1930 to 1949.* New York: The Rockefeller Foundation. xviii + 262 pp.

6. Herms, William B., 1929. "The experimental method as applied to entomological investigations," *J. Econ. Entomol.*, 22:45–59.

7. Morgan, Thomas Hunt, 1907. *Experimental Zoology.* New York: The Macmillan Company. xiii + 454 pp.

8. Ivy, A. C., 1948. "The history and ethics of the use of human subjects in medical experiments," *Science,* 108:1–5.

9. Stokes, Adrian; Bauer, J. H.; and Hudson, N. Paul; 1928. "The transmission of yellow fever to *Macacus rhesus.* Preliminary note," J.A.M.A., 90:253–54.

10. Theiler, Max, 1930. "Studies on the action of yellow fever virus in mice," *Ann. Trop. Med.*, 24:249–72.

11. McCoy, G. W., 1911. "Studies upon plague in ground squirrels. Part iv: Insect transmission in relation to plague among ground squirrels." Washington, D. C.; Govt. Print. Office, in Pub. Health Bull., no. 43, pp. 41–51.

12. Young, M. D., 1944. "Studies on the periodicity of induced *Plasmodium vivax,*" *J. Nat. Malaria Soc.*, 3:237–40.

13. Knowles, R., and Das Gupta, B. M., 1932. "A study of monkey malaria and its experimental transmission to man," *Indian Med. Gaz.* 67:301–20.

14. Faust, Ernest C., 1933. "The use of the experimental method in the study of human parasitic infections," *Scient. Monthly,* 37:139–46.

15. Chapman, R. N., 1931. *Animal Ecology,* New York: McGraw-Hill Book Co., Inc. 464 pp.

16. Pearse, A. S., 1926. "The ecology of parasites," *Ecology,* 7:113–19.

17. Strong, Richard P., 1935. "The importance of ecology in relation to disease," *Science,* 82:307–17.

18. Meyer, K. F., 1942. "The ecology of plague," *Medicine,* 21:143–74.

19. Uvarov, B. P., 1931. "Insects and climate." *Tr. Entomolog. Soc. London,* vol. 79, part 1, 247 pp.

20. Martini, E., 1936. *Wege der Seuchen, Lebensgemeinschaft, Kultur, Boden und Klima als Grundlagen von Epidemien.* Stuttgart: Ferdinand Enke. 109 pp.

21. Buxton, P. A., 1933. "The effect of climatic conditions upon populations of insects," *Tr. Roy. Soc. Trop. Med. & Hyg.*, 26:325–64.

22. Herms, William B., 1947. "Vector potentialities with respect to the spread of insect-borne diseases of man in California," *California Med.*, 67:95–99.

23. Herms, W. M., 1946. "Wartime aviation quarantine: Pests and their control," J. *Pest Control Indust.*, 14:24–25.
24. Hunter, W. D., 1913. "American interest in medical entomology *J. Econ. Entomol.*, 6, 27–39.
25. Fernald, H. T., 1926. *Applied Entomology*. New York: McGraw-Hill Book Co., Inc., xiv + 395 pp.
26. Carter, H. R., 1919. "The malaria problem of the South," U. S. Public Health Service, *Pub. Health Rep.*, 34:1927–35.
27. Herms, William B., 1919. "What shall we do with our information concerning malaria in California?" Calif. State Bd. of Health, *Month. Bull.*, 15:181–89.

CHAPTER III

PARASITES AND PARASITISM

Symbiosis and parasitism. Biologists are not agreed as to the definition of *symbiosis,* but for our purpose it may be regarded as a condition of conjoint life existing between different organisms, and in its most perfect form the associated organisms or symbionts "are completely adapted to a life in common," while on the other hand a poor adaptation to a symbiotic existence may lead to serious pathological reactions and even "to the death of the organism that is invaded." This interpretation of symbiosis by Nuttall[1] differs from the usual definition, which denotes a condition of conjoint life that is more or less beneficial to the associated organisms.

Nuttall (*loc. cit.*) well said " . . . it is difficult to imagine that symbiosis originated otherwise than through a preliminary stage of parasitism on the part of one or the other of the associated organisms, the conflict between them in the course of time ending in mutual adaptation." When the symbiotic relationship is of benefit to both organisms (i.e., reciprocal) it may be termed *mutualism.* For example, the tiny staphylinid beetles *Xenodusa cava* (Lec.) and *Xenodusa montana* (Csy.) secrete a fluid which ants, *Formica rufa* Linn. and other species, suck from glandular hairs; and in return for this favor the ants feed the beetles, which are said to be unable to feed themselves and hence perish if unassisted by the ants. Also the corn root aphid, *Aphis maidiradicis* Forbes, is cared for in a most solicitous fashion, from egg to adult, by several species of ants, *Lasius* sp., which feed on the so-called honeydew secreted by the aphids.

When only one of the two organisms is benefited by the symbiosis, the relationship is known as *commensalism.* Thus a minute species of cockroach, *Attaphila fungicola* Wheeler, is said by Wheeler[2] (p. 397) (reprinted by permission of Columbia University Press) to lick the surfaces, feeding on oily secretions of *Atta* ants, which tolerate the little roaches, "without the slightest signs of hostility." No harm, of course, results to the ants, and evidently no benefit is derived by them.

Parasitism. The definition of parasitism is well within the meaning of the term symbiosis; however, the parasitologist instead of using the term symbionts as applied to the associated species employs the terms *host,* the physically larger of these, and *parasite,* the smaller in size, assuming that the latter lives at the expense of the former. This conception of the relationship of the associated species has led to a commonly used definition, viz.: parasitism is the state of one organism, the parasite, gaining nourishment and other advantage, such as shelter, at the expense of another living organism, the host. However, the latter (the host) must not be destroyed as the result of this association before at least the growth period of the former is completed, otherwise the result would be disastrous to the parasite as well as the host. The life of a true parasite is closely tuned to the life cycle and habits of the host. Essentially "*a parasite is an organism which lives at the expense of its host, giving nothing of value in return*" (Stunkard, 1929[3]). In spite of the fact that the life of a parasite is commonly regarded as an easy one, it is rigorously circumscribed and full of dangers. It is sometimes referred to as a form of "hopeless specialization," since it leads eventually to extinction, unless conceivably the parasite were able to work back gradually from parasitism to a free-living condition. On the other hand if parasitism is but the beginning of an ill-adapted symbiotic life, then conceivably it might gradually end through progression into a condition of mutual adaptation. Indeed Nuttall (*loc. cit.*) has gone so far as to point out that " . . . it is difficult to imagine that symbiosis [in the restricted sense, that is, mutual adaptation] originated otherwise than through a preliminary stage of parasitism on the part of one or other of the associated organisms."

Swellengrebel[4] describes a heavy malaria parasite incidence in the blood of isolated bush Negroes in the interior of Dutch Guiana, yet with no malarial fever (disease) resulting from this infection. He reports that this high and constant malarial infection (mostly *falciparum*) leaves not only the adult natives unaffected but does little harm even to the youngest children. Swellengrebel himself suffered an infection while among these bushmen after stopping "quinine prophylaxis," and in due time (11 days) became ill with *falciparum* malaria. The parasites, therefore, had lost none of their potential virulence when introduced in the usual manner by mosquitoes into the body of a nonbushman.

Swellengrebel argues that this phenomenon in the case of these isolated bush Negroes is the outcome of an interaction, a struggle, between host and parasite in which "both have had to sacrifice something," "a nicely adjusted equilibrium" having been achieved. "It is an adaptation of the host to a greatly subdued parasite" which does no harm to its host and consequently none to itself; it behaves as an "*efficient parasite.*" Swellengrebel points out that the title of his paper might perhaps better have been "The Efficient Host."

There are many species of insects which are parasitic on other insects, and some of these are very useful in holding certain agricultural pests in check. Such *"parasitoid"* insects, as they are better termed, are often reared in insectaries in enormous numbers to be liberated at a proper time for control purposes. This practice is known as "biological control." While only of indirect concern to the medical entomologist, it is nevertheless a subject which will interest him in insect-control operations. The "parasitism" of "parasitoids" results in the slow death of the parasitized host insects. Predator insects kill their prey quickly, e.g., ladybird beetles of the family Coccinellidae; also the Chrysopidae, commonly known as aphid lions. These latter predators capture their prey and literally eat it; while still others, such as the kissing bugs of the family Reduviidae, suck the vital juices; but in all cases the prey is killed quickly.

An interesting form of *social parasitism* occurs in social insects such as ants and hornets, where one species lives in the colony of another species which is "deluded" into feeding both the adults and progeny of the invaders. It has been suggested that this form of parasitism exists among human societies as well. The extreme case, as suggested by Root[5] would appear to exist among certain wasps that construct no cells of their own and do not hunt, but provide for the future of their progeny by placing their eggs in the cells which have been constructed and stocked with food by some other species of wasp, and in some cases the egg or larva which originally inhabited the cell is bundled out to starve, or is devoured.

Parasitism in its various forms is widespread in nature among many groups of animals from the Protozoa to the Chordata, but as Stunkard (*loc. cit.*) has so well stated, " . . . few clearly apprehend that it comprises one of the most distinctive categories of animal existence, and constitutes a discrete and characteristic phenomenon. Careful and critical study of large numbers of parasites belonging to various classes of animals, has demonstrated that parasitism has certain basic and fundamental characteristics, no matter in what group of animals the parasitic habit may have been developed. The same general tendencies are manifest, similar attributes and relationships appear, and similar consequences inevitably follow the adoption of the parasitic habit."

Classes of parasites. Animal parasites are known as *zoöparasites*, while parasitic organisms belonging to the plant kingdom are known as *phytoparasites*. Parasites which live either temporarily or permanently on the outside of the body are termed *ectoparasites*, and animals of this group are known as ectozoa, e.g., the lice. Parasites which live within the body or its cavities are called *endoparasites*, the animals being called entozoa, e.g., tongue worms. *Obligatory parasites* are forced to remain during their entire life in or on the body of the host, e.g., both biting and suckling lice. *Facultative parasites* are able to exist as free-living organisms, and they can also live as parasites, e.g., the larvae of various species of blowflies and flesh flies. Adult female mosquitoes are example of *intermittent parasites* which prey upon the host at times only and are free-living during intervals between meals.

Many other forms of parasitism are recognized, such as *transitory* parasitism, which is said to refer to cases in which the organism is parasitic during only a part of its life history, e.g., botflies. When two or more hosts are required for the development of an obligatory parasite, that host in which the parasite reaches sexual maturity is termed the *primary* or *definitive* host, while the other, or others if more than one species is required, should be referred to as the *secondary* host or hosts. This is illustrated in the life history of the malaria *Plasmodium*, which reaches sexual maturity in the body of certain anopheline mosquitoes, the primary or definitive host, while the human being is the secondary host. The lung fluke, *Paragonimus westermani* (Kerbert), requires both a melaniid snail (*Melania libertina*, etc.) and a fresh-water crab or crayfish (*Astacus japonicus* W. de Haan, etc.) as secondary hosts, while man (and certain other mammals) is the definitive or primary host.

Effects of parasitism on host and parasite. Parasites are adapted to their mode of life in two general respects: physiologically, in that they require a certain host species or group of species as food; and morphologically, in that their structure is modified to this end. These adaptations become the more intense and exclusive as this mode of life progresses, i.e., the further back one goes in the history of a parasitic species, the more nearly like its free-living relations it becomes. This accommodation is obvious in those species in which the young are free-living and resemble the young of related wholly free-living species. The extreme case of the cirriped "louse," *Sacculina carcini* (Rathke), is illustrative of this recapitulation. The parasitic Cirripedes, fish "lice," show striking structural adaptations to a parasitic mode of living: thus the first pair of antennae may become well-formed hooks for holding fast and the first pair of maxillipedes become suckers; the mandibles are transformed into piercing organs, and the mouth becomes a sucking tube.

With the introduction of easily assimilable substances into the digestive tract of the parasite, the digestive organs gradually become modified; and with the entire body in direct contact with "predigested food," as is the case with tapeworms and other intestinal parasites also, to a certain extent, with fleshfly larvae, the outer covering of the parasite becomes modified to allow direct absorption, and thus the digestive tract becomes wholly unnecessary and may be entirely lost. However, modification of the digestive tract is not restricted to parasites by any means.

The modification or loss of wings in wholly parasitic insects, such as the Mallophaga, Anoplura, and certain Hippoboscidae, is commonly said to be the result of the parasitic mode of life; however, this phenomenon is again not characteristic of parasitic forms alone, as all will readily concede. There are numerous examples of wing reduction and extreme modification of appendages in nonparasitic forms.

Keilin and Nuttall[6] report that while *Pediculus humanus capitis* DeGeer, the head louse of man, and *Pediculus humanus corporis* DeGeer, the body louse, can be distinguished on morphological grounds, "typical *capitis* lose all their distinctive morphological characters when raised experimentally on man under conditions which are favourable for the propagation of *corporis*, and they acquire all the morphological characters of *corporis* after four or more generations" (Nuttall[7]). Adaptations of each of these races to the host are apparently quickly acquired. Typical *capitis* is more active at a lower temperature than is *corporis* owing, no doubt, to the exposed environment on the scalp of the host, while the latter is protected among the clothing. *Capitis* is smaller in size and has stouter legs, hence can climb hairs more rapidly than can *corporis*. Under a normal environment *corporis* imbibes larger meals at longer intervals in conformance with the resting habits of the host, while *capitis* with continuous opportunity for feeding takes a small amount of food at short intervals. Furthermore, Nuttall (*loc. cit.*) remarks that, "The effect of darkness no doubt is responsible for *corporis* possessing longer and slimmer antennae and legs than *capitis*. The latter is more exposed to light upon the head than is *corporis* beneath the clothing in most instances. It is, of course, well known that arthropods inhabiting dark places have longer antennae and legs than those living exposed to light." Adaptation to the skin color of the host appears to be common. Lice collected from the heads of the brown-skinned Gilbertese during the author's investigations among these people on Fanning Island[8] were strikingly sooty in color. Rearing lice in pill boxes of different colors inside, as carried on by Nuttall,[9] shows that change of color is rather easily accomplished.

Nuttall's conclusions based on the observations cited above are very interesting, namely, "There is little doubt in my mind that *capitis* is being converted into *corporis* today in nature, and that the latter, when man has become hairless, will constitute a species whose birth we are witnessing."

The wider adaptability of parasites, i.e., adaptation to different host species as compared with different parts of the body of the same host, as in *Pediculus humanus* Linn., is shown in the different races of *Sarcoptes scabiei* (Linn.), the itch or mange mite of humans, swine, horses, and other animals. While there appear to be specific differences in pigmentation, cuticular markings, and chaetotaxy, they are not constant, though some insist on the validity of these characters. Transfer from host to host of different species can be more or less readily accomplished. Other examples could be cited to illustrate this adaptability, such as certain polymorphic mammalian tryanosomes, namely, *T. brucei* Plimmer and Bradford, *T. gambiense*, and *T. rhodesiense*, as cited by Duke,[10] who refers to these as physiological variants of a single species.

The relation, both as to behavior and structure, between the parasite and the host becomes more perfect as the symbiosis grows more intimate. The true parasite and its host represent a type of machine with all its parts functioning coördinately, hence it is difficult to discuss the behavior of the former without also dwelling on the behavior of the latter. The stage in this relationship when the former cannot exist without the latter is certainly reached in many instances, but one may well wonder whether the reverse condition is ever actually achieved through the agency of parasitism.

In the case of insects which suck the blood of human beings, one is impressed with the large degree of tolerance that is manifested toward those species which are wholly or largely dependent on man; no doubt immunity is an important factor, and in turn one wonders just how far the parasite has gone in making its blood lust less offensive. Thus several extreme cases will illustrate what is meant. Gilbert Islanders with whom the author spent some time during the summer of 1924 will reluctantly give a few head lice on request, but prefer keeping them for festive reasons. To an old timer the body louse is not so offensive as it is to a tenderfoot.

The bite of *Anopheles maculipennis* Meigen is generally benign, while the bite of *Aedes dorsalis* (Meigen), a common salt-marsh species, is almost always viciously irritating. The former species has become closely associated with man and is a potent vector of malaria, the latter is a "wild" species and not a disease vector in nature. The bite of *Triatoma protracta* (Uhler) is very painful to most persons, and one is inclined to suggest that this species must moderate its bite before it can become a successful disease vector. Besides adaptations of mouth parts for piercing and bloodsucking, and moderation of venoms to lessen pain in the host, there is also the factor which prevents blood coagulation. This factor is particularly well developed in the bloodsucking helminths such as leeches and hookworms. Much needs to be done to explain the specific symptoms caused by arthropod salivary fluids following insect and tick bites, such as urticaria following the bite of *Triatoma protracta* (Uhler) and paralysis following the bite of *Dermacentor andersoni* Stiles.

The study of parasitism has contributed much during the past few years to the field of pathology and clinical medicine. Disturbances resulting from parasitism are usually of a specific nature, and hitherto unknown causes of certain symptoms are now often readily attributable to specific parasite infestations. The student of medical entomology will certainly consult *Parasitism and Disease* by Theobald Smith[11] (Princeton University Press).

Origin of parasitism. Parasitism is one of the ways in which organisms acquire food and is only one of the avenues, though an important one, that bring the arthropod into relation with man and other animals as potential pathogenic agents. Scavenger insects with indiscriminate feeding habits, for example, scavenger flies and cockroaches, which may feed on excrement, often become food contaminators and consequently may be important factors in the dissemination of filth diseases such as typhoid fever. Also *predaceous* arthropods, such as the black widow spider and the conenose bugs, may attack man and inject venoms. While the female black widow spider probably bites human beings only in self-defense or in defense of her cocoon, certain reduviid bugs, such as *Triatoma,* are said actually to suck the blood of sleeping persons, though many other warm-blooded animals may also serve as hosts. It is but a short step from sucking blood by tapping the body of a bedbug which has fed on a person and tapping the body of the person directly. This procedure is followed at least in some instances by *Triatoma protracta* (Uhler). Various species of bloodsucking arthropods secure blood meals by tapping the abdomens of their blood-engorged associates.

Parasites may be restricted more or less completely to particular species of host animals (host specific); hence, the parasite must have developed its parasitic habit after the host came into existence, i.e., parasitism must be a recently acquired habit on the part of a one-time free-living organism. This becomes more apparent by a study of the life history of the parasite; the earlier stages commonly point to a primitively free-living existence. Perhaps the ancestors of a given group of present-day parasites were attracted to waste food, offal, and exudations of certain animals. When the search for food became simplified, they began living as messmates, or commensalists, or as scavengers; thus the association between the two species became closer and eventually the parasitic habit was established. This is borne out by a study of the nearest allies of a given parasite, in which the gradation from the free-living animal to the parasite may be traced. The very close structural similarity between the free-living, wingless book louse, *Troctes divinatoria* (Müll.) (a member of the order Psocoptera, family Psocidae), and a common hen louse, *Menopon gallinae* Linn. (= *M. pallidum* Nitzsch) (a member of the order Mallophaga), leads us to believe that the parasitic Mallophaga have been derived from the primitive Psocidae. Knowing the habits of the book louse, we can easily imagine how the practice of parasitism might eventually have become established, i.e., from the eating of feathers, scales, and excretions *off* the animal to the eating of the same *on* the animal as a host.

Degrees of parasitism may also be illustrated by examples from the biting lice, Mallophaga, in which there are species having the power to run freely and live for a considerable length of time off the host, e.g., *Menopon gallinae* Linn. (= *M. pallidum* Nitzsch), the common hen louse, while other related species have become entirely sessile, as in the extreme case of the worm-like louse, *Menopon titan* Piaget, inhabiting the gular pouch of the pelican. Among the fleas there are also good examples of gradation in habit and structure: the human flea, *Pulex irritans* Linn., has developed remarkable springing power and is comparatively free to move from place to place, while the mature female sticktight flea, *Tunga penetrans* (Linn.), is quite sessile, holding fast to one spot much like a tick.

The medical entomologist is continually pressed with question concerning the origin of the present intimate relationship existing between insects and causal organisms of diseases of higher animals. It is interesting to know that Galli-Valerio[12] found that *Herpetomonas pyrrhocoris* Zotta et Galli-Valerio lives part of the time in flagellate form in the rotting blossoms of the meadow saffron, *Colchicum autumnale*, where it is picked up apparently by the bug in the spring and is deposited in nonflagellate form again in the blossom during the autumn by means of the insect's excreta. This is believed to be a very primitive degree of the adaptation of a saprophytic protozoön to an internal parasitic life in an insect. The relationship existing between *Leptomonas davidi*, already referred to, living in the latex of Euphorbia and carried by the bug *Nysius euphorbiae* Horv., represents a step in advance.

Systematic position of animal parasites. Though parasitic animals are found in other phyla, those of economic importance affecting man and beast are included almost exclusively in the following groups:

a. Protozoa: unicellular animals; e.g., *Endamoeba histolytica* (Schaudinn), causing amoebic dysentery; *Plasmodium vivax* (Grassi and Feletti), causing malaria; *Trypanosoma gambiense* Dutton, causing African sleeping sickness.

b. Nemathelminthes: bilateral, unsegmented worms of cylindrical form; e.g., *Trichinella spiralis* (Owen), causing trichinosis; *Ascaris lumbricoides* Linn., roundworm of man; *Ancylostoma duodenale* (Dubini), a hookworm of man. Development is usually direct (Fig. 1).

c. Platyhelminthes: bilateral worms; flattened dorsoventrally; no anal opening. Usually requiring an intermediate host.

1. *Cestoda:* scolex with separable segments called proglottids; e.g., *Taenia solium* Linn., the pork tapeworm of man; *Taenia*

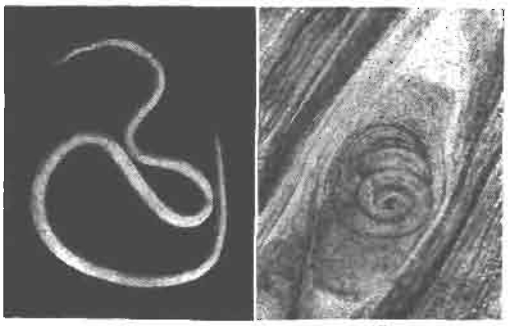

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FIG. 1. Examples of parasitic roundworms (Phylum Nemathelminthes, Class Nematoda). (*A*) roundworm of swine (*Ascaris lumbricoides*); (*B*) *Trichinella spiralis*, greatly enlarged.

FIG. 2. Examples of parasitic flatworms (Phylum Platyhelminthes, Class Cestoda). (*Left*) a poultry tapeworm (*Choanotaenia infundibulum*, ×1), and (*right*) a common tapeworm of sheep (*Moniezia expansa*, greatly reduced).

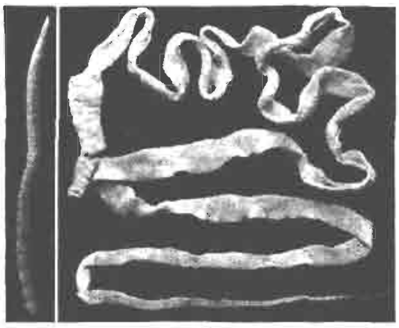

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FIG. 3. Example of parasitic flatworms (Phylum Platyhelminthes, Class Trematoda). A liver fluke of sheep (*Fasciola hepatica*). ×1.

FIG. 4. Example of segmented cylindrical worms (Phylum Annelida, Class Chaetopoda). Earthworm (*Lumbricus* sp., ×.5) nonparasitic, but may serve as an intermediary host for certain poultry tapeworms.

FIG. 5. Example of segmented cylindrical worms (Phylum Annelida, Class Hirudinea). Leech (*Hirudo medicinalis*). ×.5.

saginata Goeze, the beef tapeworm of man; *Dipylidium caninum* (Linn.), a common tapeworm of the dog (Fig. 2).

2. *Trematoda:* alimentary canal branched; mouth in a sucker; e.g., *Fasciola hepatica* Linn., the liver fluke of sheep (Fig. 3).

d. Annelida: bilaterally symmetrical, segmented or annulated worms.

1. *Chaetopoda:* locomoter chaetae; segmentation extending to internal organs; e.g., *Lumbricus terrestris* Linn., a common earthworm (nonparasitic) (Fig. 4).
2. *Hirudinea:* flattened; sucker at each end of body; arrangement of internal organs does not correspond to external segmentation; e.g., *Hirudo medicinalis* Linn., the medicinal leech (Fig. 5).

e. Arthropoda: segmented body with paired jointed appendages; chitinous exoskeleton; bilaterally symmetrical; heart dorsal; ventral nerve cord.

1. *Crustacea:* head and thorax often united to form a cephalothorax; numerous paired, biramous appendages; two pairs of antennae; respiration usually branchial; habitat usually aquatic; e.g., shrimp, crayfish, and the sow bug (the latter terrestrial). These examples are nonparasitic (Fig. 6).
2. *Onychophora* (*Protracheata*): vermiform and externally unsegmented; numerous paired, imperfectly segmented legs; one pair of antennae; tracheal respiration; habitat terrestrial; e.g., *Peripatus* (nonparasitic) (Fig. 7).
3. *Chilopoda:* body flattened dorsoventrally; one pair of jointed legs on each segment except first two and last one; one pair of antennae; eyes simple; genital pore posteriorly located as in insects; habitat terrestrial; e.g., centipedes (nonparasitic) (Fig. 8a).
4. *Diplopoda:* cylindrical in form; two pairs of jointed legs on each segment except first two and last one; simple eyes; one pair of antennae; genital pore anteriorly located; habitat terrestrial; e.g., millipedes (nonparasitic) (Fig. 8b).
5. *Symphyla:* very small; body flattened dorsoventrally; one pair of jointed legs per segment and not more than 12 pairs in all; spinnerets present at posterior end; eyeless; one pair of antennae; genital pore anterior; e.g., garden centipedes (nonparasitic).
6. *Pauropoda:* minute; body flattened; nine, seldom ten, pairs of jointed legs; antennae branched; eyes generally absent; e.g., pauropods (nonparasitic).
7. *Insecta* or *Hexapoda:* body divided into three pairs (head, thorax, and abdomen); three pairs of walking appendages on

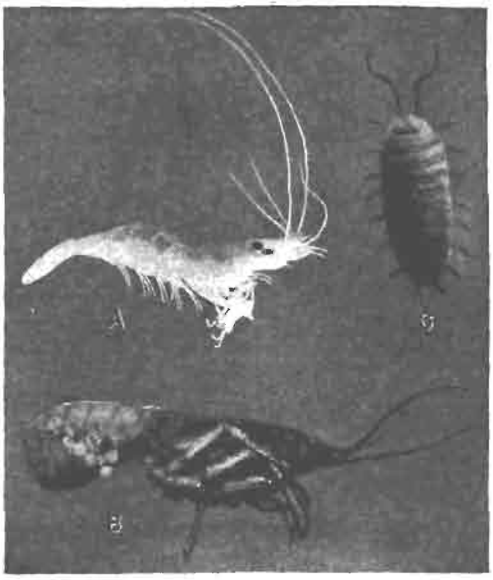

FIG. 6. Examples of the Phylum Arthropoda, Class Crustacea. (*A*) shrimp, ×1.2; (*B*) crayfish, ×.6; (*C*) sow-bug, ×2. (All three examples are nonparasitic.)

thorax; two pairs of wings on thorax (may be reduced or absent); one pair of antennae; compound eyes; usually three simple eyes; tracheated respiratory system; e.g., *Triatoma protracta* (Uhler) (conenose bug); *Cimex lectularius* Linn. (bedbug); *Anopheles maculipennis* Meig. (malaria mosquito) (Fig. 9).

8. *Arachnida:* head and thorax fused to form cephalothorax; four pairs of walking appendages on cephalothorax (larvae may be

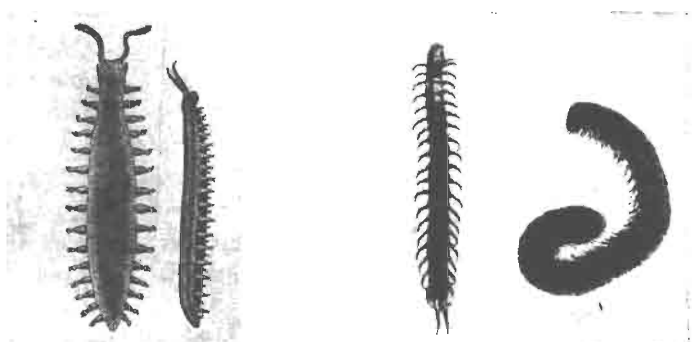

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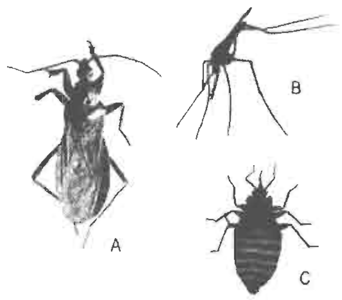
FIG. 7. Example of Phylum Arthropoda, Class Onychopora. Peripatus. ×.5. (After Folsom.)

FIG. 8. Examples of the Phylum Arthropoda: (*left*) Class Chilopoda, a centipede, ×.5; (*right*) Class Diplopoda, a millipede, ×.7.

hexapod); wingless; no antennae; eyes simple, when present; e.g., *Latrodectus mactans* (Fabr.), a poisonous spider; *Hadrurus hirsutus* (Wood), scorpion; *Boöphilus annulatus* (Say), the



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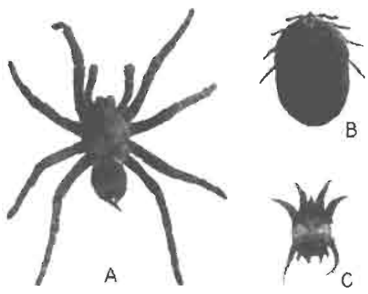

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FIG. 9. Examples of the Class Insecta. (*A*) a reduviid (conenose), ×1; (*B*) a mosquito (*Anopheles*), ×2; and (*C*) bedbug (*Cimex*), ×2.5.

FIG. 10. Examples of the Phylum Arthropoda, Class Arachnida. (*A*) a spider, ×.5; (*B*) a tick, ×1.3; and (*C*) a mite, ×30.

Texas fever tick; *Dermanyssus gallinae* (DeGeer), the poultry mite; *Psoroptes communis* var. *ovis* (Hering), the sheep scab mite (Fig. 10).

BIBLIOGRAPHY

1. Nuttall, George H. F., 1923. *Symbiosis in Animals and Plants. Section 1. Physiology.* Liverpool: British Association. 18 pp.

2. Wheeler, W. M., 1926. *Ants: Their Structure, Development and Behavior.* New York: Columbia Univ. Press. xxv + 663 pp.

3. Stunkard, Horace W., 1929. "Parasitism as a biological phenomenon," *Scient. Monthly,* 28:349–62.

4. Swellengrebel, N. H., 1940. "The efficient parasite," *Science,* 92:465–69.

5. Root, Francis M., 1924. "Parasitism among the insects," *Scient. Monthly,* 9:479–95.

6. Keilin, D., and Nuttall, G. H. F., 1919. "Hermaphroditism and other abnormalities in *Pediculus humanus,*" *Parasitology,* 11:279–328 (7 plates).

7. Nuttall, G. H. F., 1919. "The systematic position, synonymy, and iconography of *Pediculus humanus* and *Phthirus pubis,*" *Parasitology,* 11:329–46.

8. Herms, W. B., 1925. "Entomological observations on Fanning and Washington Islands, together with general biological notes," *Pan-Pacific Entomologist,* 2:49–54.

9. Nuttall, G. H. F., 1919. "The biology of *Pediculus humanus,*" *Parasitology,* 11:201–20 (1 plate).

10. Duke, H. L., 1923. "Further inquiries into the zoölogical status of the polymorphic mammalian trypanosomes of Africa," *Parasitology*, 15:358–95.

11. Smith, Theobald, 1934. *Parasitism and Disease*. Princeton Univ. Press. xiii + 196 pp.

12. Galli-Valerio, B., 1920. "Le cycle evolutif probable de' l'*Herpetomonas pyrrhocoris*, Zotta & Galli-Valerio," *Schweiz. med. Wchnschr.*, 1:401–2. (Cited in *Rev. Applied Entomol. Med. & Vet.*, 1922, 10 (ser. B, part 1):8.

CHAPTER IV

HOW ARTHROPODS CAUSE AND CARRY DISEASE

Arthropod and pathogen. When one considers the fact that man and his domesticated animals are daily closely associated with scores of species of insects* and their kin, the wonder is that there are not more arthropodan infestations and insect-borne diseases. Many of the one-time free-living arthropods have in time become parasitic; some now burrow into the skin, as do certain mites (acariasis); others have invaded the alimentary tract, as do larvae of botflies (myiasis); still others have become bloodsuckers, as are bedbugs, sucking lice, horseflies, mosquitoes, etc. Bloodsuckers, by virtue of their bloodsucking habit, may readily become vectors of pathogenic blood-inhabiting microörganisms. It has been well said that no bloodsucking arthropod can be trusted; eventually many more species will prove to be vectors of disease.

The medical entomologist must acquaint himself with the detailed biology of the disease-producing microörganisms as well as with that of the vectors. In order to know where and how the arthropod acquires the pathogenic agent and how it, the vector, becomes infectious, the behavior of such organisms must be studied; their habitat in the body of the diseased host or reservoir must be known; the gateways of escape must be ascertained; their longevity and virulence when removed from the host as well as many other pertinent factors must be determined. After having acquired the pathogenic organism, its course within the body of the arthropod must be studied in order to know how escape is effected and how it reaches the body of the next host. A knowledge of the feeding habits and mouth parts of arthropods is essential. (See mechanism of infection, p. 36.)

Bubonic plague, for example, is a bacillary disease caused by *Pasteurella pestis*, of which the rat, among other animals, is an important host. Man readily succumbs to the infection. While the pathogenic organisms may pass from host to host in several ways, it has been found

* Z. P. Metcalf estimates the number of species of insects alone, described for the whole world between 1758 (Linnaeus) and 1940, at 1,500,000. (*Entomological News*, 1940, 51:219–22.)

that rat fleas are the most important vectors. The bacilli are found in great abundance in the buboes which are situated largely in the axillary and inguinal regions of the infected rat, and it has been observed that these regions are favored by fleas, which, because of their bloodsucking habits, imbibe the highly infectious fluids. If the rat dies of the plague, the fleas leave the body and seek another host; this interval in the change of hosts raises the question of environmental resistance and longevity of the bacilli. Do all species of rat fleas lend themselves equally well as hosts? Can the bacilli resist the digestive fluids of the flea, and if so, how long do they remain infective in the body of the flea? In the meantime what course do these organisms take within the body of the insect, and how do they make their escape? How does the flea find another host, and what animals will it attack? How long can the flea live without food? How are the plague bacilli introduced into the body of the next victim? Will the flea remain infective throughout the rest of its normal life? What is the length of life of a flea, and does this vary with the species?

Malaria of man, another example, is caused by certain species of plasmodia which live under far more restricted conditions than do the plague bacilli. In the human being these invade the red corpuscles part of the time and so far as is known do not live in the blood of other warm-blooded animals. They require certain mosquitoes belonging to the genus *Anopheles* as definitive hosts. Although many other bloodsucking insects imbibe parasitized blood, these do not offer the necessary environment within the alimentary canal for the completion of the life cycle of the parasite.

Chain of infection. For each arthropod-borne disease there is a chain of events which leads to the successful transfer of an infection by an arthropod vector from one animal to another. This chain consists of several links, each more or less vulnerable and hence subject to rupture, which could prevent successful transfer of the infection. The *first* link in this chain is the developmental cycle of the pathogen within the body of an appropriate host. This link may be broken by medicinal treatment, as in malaria. The *second* link in the infection chain involves the vector, its access to infection, its feeding habits, its manner of ingesting food, the structure of its mouth parts, etc.; the *third* link represents the progress of development of the pathogen (also more or less complicated) within the body, stomach, or other organs of the vector; the *fourth* link represents the manner in which the now infectious microörganism leaves the body of the now infectious vector and thereupon enters the body of the next susceptible animal, thus resulting in infection—specifically it is the *mechanism of infection,* e.g., (1) simple *contamination* by means of germ-laden houseflies or cockroaches coming in contact with food, milk, etc.; (2) simple mechanical *inoculation* of anthrax bacilli by means of contaminated horsefly mouth parts, or inoculation of sexually mature malaria plasmodia by an *Anopheles* mosquito; (3) *regurgitation* of plague bacilli upon the skin by fleas while biting after a proventricular blockage; (4) *fecal* contamination where trypanosomes of Chagas' disease are deposited on the skin when the conenose bug bites; (5) *skin contamination with crushed tissues* of infectious arthropods, e.g., crushed infected ticks may result in spotted fever infection.

The *terminal* link in the infection chain is the successful completion of a cycle similar to that of the first link but in the body of another host animal after the intervention of an arthropod vector. Other links in the chain of infection as well as other variations in the mechanism of infection will occur to the student as he progresses in the field of medical entomology.

Pathological conditions caused by arthropods. The ways in which arthropods relate to the public health and the health and well-being of animals are almost as numerous as the species of responsible arthropods themselves. Each species, however similar to its next of kin, has certain characteristics which affect the host differently. It is nevertheless possible to classify the relationships of arthropods to health, putting them into two divisions, each of which is subject to further segregation. The two major relationships may be designated as (A) pathological conditions caused directly by arthropods, and (B) arthropods as vectors of pathogenic organisms. The first major division (A) includes such categories as (1) entomophobia, (2) accidental injury to sense organs. (3) envenomization, (4) dermatosis, (5) myiasis, (6) allergy. The second major division (B) may be subdivided into (1) accidental carriers, and (2) obligatory vectors involving some degree of development (cyclical or propagative) within the arthropod.

As our subject develops in the remaining chapters of this book, the student will classify the way or ways in which the particular groups of arthropods fit into the scheme presented in this chapter.

Entomophobia. Insects and spiders, even though they may be wholly innocuous, frequently cause man acute annoyance and worry which may eventually lead to a nervous disorder with sensory hallucinations.[1] This class of mental disturbances may perhaps deserve a specific designation such as entomophobia.

The author has observed two or three cases of this nature each year for many years. The cases are usually referred to him by physicians, primarily because the patient is assumed to have confidence in one who has knowledge of the control of tormenting insects. The patient usually imagines that insects of very minute size persist in threatening him and biting him, usually at particular sites of the body, and that they achieve this contact in the most cunning ways in spite of every precaution.

Accidental injury to sense organs. Various species of insects, like other minute objects, may accidentally enter the eye; this is most likely to be true of small flying insects. Some of these, notably several species of rove beetles, Staphylinidae, cause extreme pain because of an irritating secretion. Many species of insects discharge odoriferous fluid or vapor, and in some instances the fluid is so forcibly ejected that it may be thrown some distance from the insect. Stewart (1937)[2] records the case of a phasmid walking stick, *Anisomorpha buprestoides* (Stoll), squirting fluid a distance of two feet and striking a person in the eye. "The pain in the left eye was immediately excruciating; being reported to be as severe as if it had been caused by molten lead. Quick, thorough drenching with cool water allayed the burning agony to a dull, aching pain. The pain eased considerably within the course of a few hours. Upon awaking the next morning the entire cornea was almost a brilliant scarlet in color and the eye was so sensitive to light and pressure for the next forty-eight hours that the patient was incapacitated for work. Vision was impaired for about five days. The cornea gradually cleared of congestion and vision improved so that at the end of this time the eye was perfectly normal again. No subsequent ill effects were experienced." Stewart remarks that, "In Texas there is a rather common belief that devil's horses will spit in human eyes."

Injury to the human eye is often caused by the spiny larva of the sheep botfly or head maggot fly, *Oestrus ovis* Linn. This fly deposits living young in the nostrils of sheep, and persons working in the field with sheep are occasionally "struck" in the eye by the fly and one or more larvae may be deposited. This indicates that the fly may "strike" sheep similarly at times.

Insects commonly enter the ears of sleeping persons and may cause much pain before they are removed. Thus in the author's experience a carpet beetle larva (*Dermestes* sp.) was taken from the ear of an elderly man, and a hyaline grass bug, *Corizus hyalinus* (Fabr.), family Coreidae, from the ear of a child, having caused much pain in each case. The spinose ear tick, *Otobius megnini* (Dugès) also invades the ears of man and many animals.

Envenomization. Venoms of insects cause reactions which are remarkably characteristics. The effects may be classified under the following headings: (1) hemolytic, (2) hemorrhagic, (3) neurotoxic, (4) vesicating. The venoms are introduced in the following ways: (1) by the bite, as of conenose bugs and black widow spiders; (2) by the sting, as of bees, wasps, scorpions; (3) by urticating hairs, as with the browntail moth; and (4) by contact, as with vesicating fluids from blister beetles. (See Chapter XXII.)

Dermatosis. Various skin irritations are caused by arthropods, either by bites or by skin invasions. Some of these irritations could be appropriately classified as envenomizations. Skin irritations commonly result from the bites of such insects as mosquitoes, fleas, lice, and bedbugs. Various species of burrowing mites cause skin irritations known as *acariasis*. Among the latter are the itch mites, *Sarcoptes scabiei* (Linn.); the scab mites, *Psoroptes communis* Hering; the follicle mites, *Demodex folliculorum* Simon; the chigger mites, *Eutrombicula alfreddugési* (Oudemans), and other trombiculids.

Myiasis. An invasion of organs and tissues of man and beast by maggots, the larvae of Diptera, is termed *myiasis*. The invading maggots may be specific myiasis-producing forms, i.e., obligatory sarcobionts, which invade cutaneous tissues, as does *Dermatobia hominis* (Linn.) in man and *Hypoderma bovis* (DeGeer), the warble fly of cattle; invading the gastric and intestinal tract are the botflies of horses, *Gasterophilus intestinalis* (DeGeer); and invading the nasal and frontal sinuses are the head maggots of sheep, *Oestrus ovis* Linn. The invading maggots may be necrobionts or facultative sarcobionts, in which case *traumatic dermal myiasis* may result, as with infestations of screwworms, *Callitroga* (= *Cochliomyia*) *americana* C. & P. Accidental myiasis may be the result of fly larvae in food, or the result of flies attracted by anal, vaginal, or nasal discharges. The larvae of blowflies, bluebottle flies, and greenbottle flies commonly occur in accidental intestinal myiasis as well as in traumatic myiasis.

Allergy caused by insects. The condition of being specifically hypersensitive to certain insect proteins is a fairly common and widespread phenomenon among persons working habitually with bees or collections of dead insects, or exposed for longer periods to pulverized insect parts, scales of butterflies, moths, and caddis flies. Residents on the shores of lakes where cast skins of mayflies abound, are subject to attacks of "asthma" as the result of allergens (Figley, 1929).[3]

Mechanical (simple) carriers of infection. Many species of insects may accidentally contribute to the transmission of various filth diseases; however, when insects habitually and alternately feed and/or breed in excrement and then feed on human food prepared for the table, they may be classified as food contaminators and a real menace to the public health, e.g., flies, particularly the common housefly, and cockroaches. The causal organisms of filth diseases, such as typhoid fever, cholera, and amoebiasis, may adhere to the mouth parts and feet of these insects and may be deposited on human food, with resulting infection to the consumer. The mechanical transmission of yaws (a spirochete infection) and of certain eye infections (so-called pinkeye) is similarly effected by flies, particularly *Hippelates*. The eggs of helminth parasites, notably pinworms [*Enterobius vermicularis* (Linn.)] may also be so disseminated. Not only do pathogenic bacteria, protozoa, and helminthic ova cling to the mouth parts, feet, wings, and other parts of the insect body, but they may also be swallowed by the insect and pass uninjured through its alimentary canal and be deposited on food with the insects' feces or be regurgitated with similar effect.

It has been amply proved that coprophagous fly larvae (maggots), which feed on and develop in human excrement, may transfer bacteria taken up in this stage through the pupal stage to the mature flies. In this manner the infection of anthrax (*Bacillus anthracis*) may be disseminated by flesh flies, bred in carcasses of animals dead of this disease. This is a strong argument in favor of the incineration of dead animals.

Another purely mechanical method of disease transmission is by means of *contaminated piercing* mouth parts, in which these organs in the act of feeding become contaminated with blood-inhabiting pathogenic organisms, and *simple inoculation* may result from the next feeding. Here again, as in the aforementioned cases, the pathogenic organisms undergo no interim developmental change. Insects that belong to this class of simple carriers generally have strong, piercing mouth parts, capable of drawing considerable blood and are intermittent feeders, flying readily and quickly from one host to another, e.g., horseflies (Tabanidae), which may be vectors of anthrax in this manner.

Cyclico-propagative transmission. Of the several ways in which so-called biological transmission by arthropods is effected, the *cyclico-propagative* type is the one most likely to be used to illustrate insect transmission of disease; however, Huff[4] has pointed out that there are actually at least two other ways in which this occurs, namely, *cyclico-developmental* and *propagative*. In the *cyclico-propagative* type of transmission the causal organisms "undergo cyclical change and multiply" in the body of the arthropod, as in the transmission of malaria plasmodia by anopheline mosquitoes and in the transmission of *Babesia bigemina* of Texas cattle fever by the Texas fever tick, *Boöphilus annulatus.*

Cyclico-developmental transmission. When the causal organisms "undergo cyclical change but do not multiply" in the body of the arthropod, transmission may be classified as *cyclico-developmental,* as in the mosquito transmission of the worm *Wuchereria bancrofti,* causal organism of filariasis.

Propagative transmission. When "the organisms undergo no cyclical change, but multiply" in the body of the vector, transmission is said to be *propagative* only. The transmission of bubonic plague is probably propagative, since it is known that the causal organism, *Pasteurella pestis,* can multiply in the fore-gut of the flea. It is also very probable that the transmission of relapsing fever by ticks falls into this category.

Fecal contamination. The feces of several species of disease-transmitting arthropods are known to be infectious after the insect has fed on infectious animals, for example, the feces of fleas fed on plague-infected rodents, also the feces of conenose bugs, *Triatoma,* after feeding on animals infected with Brazilian trypanosomiasis, Chagas' disease. The feces of infective yellow fever mosquitoes are said to be highly dangerous. The Rockefeller Foundation (Annual Report, 1930) states that a dilution of between one ten-millionth and one-billionth of one cubic centimeter of yellow fever virus has frequently proved fatal to monkeys and that infection through the unbroken skin is readily accomplished.

Hereditary transmission. Transmission of infection through the arthropodan egg was discovered by Smith and Kilbourne in 1893 in their investigation of Texas cattle fever. When the cattle tick, *Boöphilus annulatus* (Say), reaches maturity on the body of the animal, it drops to the ground, where the female deposits her eggs and dies. The young ticks on emerging from the eggs are infective and transmit the causal protozoön, *Babesia bigemina,* to susceptible cattle. The term "hereditary transmission" is not correctly applied to the transmission of this disease, since no hereditary or genetic mechanism is involved; however, it is now widely used and may be permissible under the circumstances. Substitutes such as congenital contamination, ovarian contamination, or transovarian transmission have been suggested.

Other infections transmitted transovarianly are Rocky Mountain spotted fever, caused by *Rickettsia rickettsi* (Wolbach) and transmitted by ticks, e.g., *Dermacentor andersoni* Stiles; tularemia caused by *Pasteurella* (= *Bacterium*) *tularensis* and transmitted by the tick *Dermacentor andersoni* Stiles; relapsing fever, caused by *Borrelia* (*Spirochaeta*) *recurrentis* and transmitted by several species of ticks belonging to the genus *Ornithodoros.* Ticks play an important role in the hereditary transmission of infections involving protozoa, rickettsiae, bacteria, and spirochetes. Hereditary transmission of virus diseases is not so clear, although sand fly fever, or Pappataci fever, has been produced by the bites of *Phlebotomus papatasii* Scopoli bred from infected parents (Whittingham 1922, according to Hinman).[5]

Arthropods as intermediate hosts of helminths. Numerous arthropods serve as intermediate hosts of many species of parasitic worms. There are about 150 known species of worms parasitic in vertebrates which have arthropods as intermediate hosts and for which the primary hosts are known. About 275 species of arthropods are known to serve in this capacity; of these nearly 200 are insects, about 75 are crustaceans, and the remainder are arachnids and myriapods. No doubt hundreds of such life histories are still to be ascertained. Hall[6] states that only 1 per cent of the life histories of the known tapeworms have been worked out.

The reasons for the frequent host-parasite relationships between arthropods and helminths are no doubt mainly the following: (1) the arthropods are an exceedingly large group of animals; (2) they constitute an important food supply for fish, amphibians, birds, and some mammals; (3) herbivorous animals are sure to ingest arthropods which are habitually present on plants; (4) many species of insects feed on and breed in manures, and are thus exposed to infection from eggs and larvae of worms parasitic in hosts responsible for the manure; (5) many arthropods are transient or permanent ectoparasites feeding on blood in which there may be parasitic worms.

Monoxenous worms such as *Ascaris* and *Enterobius* during the course of their life histories transfer from one host animal to a similar host animal without the intervention of an intermediate host. "The heteroxenous worms have life histories in which in most cases the worms pass from mature stages in one host animal to larval stages in a host animal of a different sort, the intermediate host, and then return to a host animal of the first sort or a more or less closely related species and develop in this animal to maturity. In some instances two intermediate hosts are utilized in sequence for larval stages." (Hall, *loc. cit.*)

As already pointed out, knowledge concerning arthropods serving as intermediate hosts for the Cestoda (tapeworms) is quite fragmentary. Among the better-known instances are the double-pored dog tapeworm, *Dipylidium caninum* (Linn.), which requires either the biting dog louse, *Trichodectes canis* DeGeer, or a flea, *Ctenocephalides canis* (Curtis), as intermediate host. *Choanotaenia infundibulum* (Bloch), a fowl tapeworm, utilizes the housefly, *Musca domestica* Linn. (See appropriate chapters.) Stunkard[7] has shown that *Moniezia expansa* (Rudolphi), an important tapeworm of sheep and other herbivores, uses a species of oribatid mite, *Galumna* sp., as intermediate host. Eggs of the tapeworm were fed to these mites and in time the onchospheres were recovered in large numbers from the body cavity of the mites. More recently Stunkard[8] (1944) reported a similar developmental pattern in oribatid mites for anoplocephaline cestodes of rabbits and for the monkey-human species.

Numerous trematodes (flukes) of fish, amphibians, reptiles, and other insectivorous animals use arthropods as intermediate hosts. Since many of the vertebrate hosts are aquatic or semiaquatic, so most of the arthropods are also aquatic, such as dragonflies (Odonata), caseworms (Trichoptera), mayflies (Ephemerida), and stoneflies (Plecoptera). Among these flukes are the poultry fluke, *Prosthogonimus pellucidus* (v. Linstow), particularly of ducks, which use the larvae of the dragonfly, *Libellula quadrimaculata* Linn., as intermediate host. The important lung fluke of man, *Paragonimus westermani* (Kerbert), requires as its second intermediate host (the first is a melaniid snail) a crustacean, *Astacus* spp., crayfish.

Among the Nematoda (threadworms) are numerous species that use arthropods as intermediate hosts; These include the *Gongylonema* worms (Spiruridae), such as *G. pulchrum* Molin, which causes an infection of humans (also of the pig, sheep, ox, etc.) known as gongylonemiasis. These worms occur as larvae in such insects as cockroaches (Blattidae), meal worms (Tenebrionidae), and a few other forms. The mature worms, extremely slender (0.5 mm diameter), reach a length of 145 mm in the female. In the vertebrate hosts the worms are found in burrows of the mucosa and submucosa of the mouth, tongue, and esophagus. The eggs are evacuated with the fecal material of the host and do not develop until ingested by an insect. The eggs hatch in the digestive tract of the insect and soon penetrate the intestinal wall, coming to rest as encapsulated larvae in the body cavity. There they remain until the insect is ingested intact or in fragments by an appropriate vertebrate host. In this host the larvae are freed and migrate along the digestive tract to the oral cavity, where they mature. Sambon[9,10] based his deductions concerning cancer on a study of *Gongylonema* worms. (See Chapter VII.)

Other nematodes which require arthropods as intermediate hosts are certain species belonging to the family Filariidae, such as *Wuchereria bancrofti* and *Onchocerca volvulus*, the former requiring mosquitoes and the latter black gnats (see later chapters).

The thornheaded worms (*Acanthocephala*) use beetles (Scarabaeidae) mainly as intermediate hosts, e.g., the thornheaded worm of swine, *Macracanthorhynchus hirudinaceus* (Pallas).

The famous guinea worm of the Nile Valley and equatorial Africa, *Dracunculus medinensis* (Linn.), a worm which as an adult female may measure from 70 to 120 cm in length, requires Crustacea belonging to the genus *Cyclops* as intermediate hosts.

Reservoir animals. Reservoir animals play an important role in the natural distribution of insect-borne diseases. Since true reservoir animals suffer little or no ill effect from certain microörganisms pathogenic to man their presence may go unnoticed, as is the case with rabbit reservoirs of Rocky Mountain spotted fever; however, rat epizoötics are commonly the forerunners of human plague epidemics. The human being may himself be a reservoir of certain insect-borne infections, even plague. Since there are numerous vertebrates which serve as disease reservoirs, it behooves the medical entomologist to acquaint himself thoroughly with the broad field of vertebrate zoölogy, particularly the ecological aspects, and with the parasitic ectozoa of wild mammals. Greater attention must be given to the problems of forest sanitation in recreation areas.

There are at least seventy species of mammals which may serve as possible reservoirs of plague, according to Stallybrass.[11] The rickettsial infections are notable for their wild-animal reservoirs: Rocky Mountain spotted fever with its rabbits, badgers, woodchucks, field mice, and others; typhus fever with rats; Japanese flood fever with voles. The spirochete infections are equally noteworthy in that the relapsing-fever reservoirs are evidently fairly numerous; thus young porcupines, as well as armadillos and opossums, have been listed, and in California certain squirrels and chipmunks. Trypanosome infections are well represented by sleeping sickness with its numerous big-game reservoirs, and Chagas' disease with its armadillos, opossums, dogs, and cats.

Hudson[12] calls attention to the fact that the animal reservoir host plays a prime role in tropical medicine. In tropical regions of the earth as well as in the less highly organized countries of the temperate zone there is a striking increase in the number of diseases drawn from animal reservoirs. Here huge numbers of human victims are "encompassed by an intricate web of animal and insect life, in a physical environment of extraordinary complexity and variability."

Recently a remarkable suggestion has been made by Maldonado[13] that certain plants may act as reservoirs of the causal organism of verruga, a disease of man, and that the species of *Phlebotomus* flies that transmit the disease may feed on the latex. It is also suggested that this would explain why these sand flies are so abundant during the rains (January to April), when this particular plant growth (*Jatropa basiacantha* and *Orthopterygium huancui*) is most luxuriant.

BIBLIOGRAPHY

1. Smith, Roger C., 1934. "Hallucinations of insect infestation causing annoyance to man," *Bull. Brooklyn Entomolog. Soc.*, 29:208–12.

2. Stewart, Morris A., 1937. "Phasmid injury to the human eye," *Canad. Entomolgist*, 69:84–86.

3. Figley, K. D., 1929. "Asthma due to the may-fly," *Am. J. M. Sc.*, **178**: 338–45.

4. Huff, Clay G., 1931. "A proposed classification of disease transmission by arthopods," *Science*, 74:456–57.

5. Hinman, E. Harold, 1933. "Hereditary transmission of infections through arthropods," *Am. J. Trop. Med.*, 13:415–23.

6. Hall, M. C., 1929. *Arthropods As Intermediate Hosts of Helminths*, "Smithsonian Misc. Collect., vol. 81, no. 15, Washington D. C.: Smithsonian Inst. (Publ. no. 3024). 77 pp.

7. Stunkard, H. W., 1937. "The life cycle of *Moniezia expansa*," *Science*, **86**: 312.

8. ———, 1944. "How do tapeworms of herbivorous animals complete their life cycles?" *Tr. New York Acad. Sc.*, ser. 2, 6:108–21.

9. Sambon, L. W., 1925. "Researches on the epidemiology of cancer made in Iceland and Italy, July–Oct., 1924." *J. Trop. Med.* 28:39–71.

10. ———, 1926. "Observations and researches on the epidemiology of cancer made in Holland and Italy, May–Sept., 1925," *J. Trop. Med.*, 29:233–87.

11. Stallybrass, C. O., 1931. *The Principles of Epidemiology and the Process of Infection.* London: C. Routledge & Son. 696 pp. (1 plate, 1 chart).

12. Hudson, E. H., 1944. "The role of the reservoir host in tropical disease," *Am. J. Trop. Med.*, 24:125–30.

13. Maldonado, A., 1931. "Rôle probable de quelques plantes caracteristique de la region verruqueuse sur l'etiologie de la verruga du Perou," *Bull. Soc. path. exot.*, 24:27–28.

CHAPTER V

STRUCTURE, DEVELOPMENT, AND CLASSIFICATION OF INSECTS AND ARACHNIDS

The Insecta (**Hexapoda**) constitute the largest class in numbers of species (see footnote, p. 35) in the Phylum Arthropoda, which in turn comprises a greater number of species than all of the animal kingdom combined. As members of the Phylum Arthropoda, insects share the following arthropodan characteristics: segmented body with paired, jointed appendages; bilateral symmetry; dorsal heart; ventral nerve cord; and chitinous exoskeleton. Insects have the body divided into three more or less distinct parts, the *head*, the *thorax*, and the *abdomen*. There were primitively 20 or 21 segments, but owing to the specialization of the head and posterior terminal segments, there are now only about 12 recognizable ones. The *head* of the adult insect bears a pair of antennae, the mouth parts, and the eyes; the *thorax* bears the locomotor appendages, usually two pairs of wings, (these may be reduced or absent), and three pairs of jointed legs; the *abdomen* bears no appendages except the terminalia. Respiration is effected by means of a complex system of microscopic tracheal tubules opening through the body wall and carrying air directly to all parts of the body of the insect. Immature insects belonging to many of the orders differ markedly from the mature forms, e.g., maggots of flies, but all possess tracheal tubules.

External anatomy. In order to familiarize himself with the external anatomy of insects, especially with the parts upon which classification is mainly based, the student should study carefully some large hard-bodied insect, such as the horsefly (Fig. 11) or the cockroach. He should give sufficient time to this exercise in the laboratory to become thoroughly informed.

Wings. The earliest systems of insect classification were based on wing characters, which together with the mouth parts and metamorphosis afford a basis for modern classification. The venation of insect wings is so markedly characteristic for most species that even a part of a wing is often all that is necessary for determination. The winged insects are

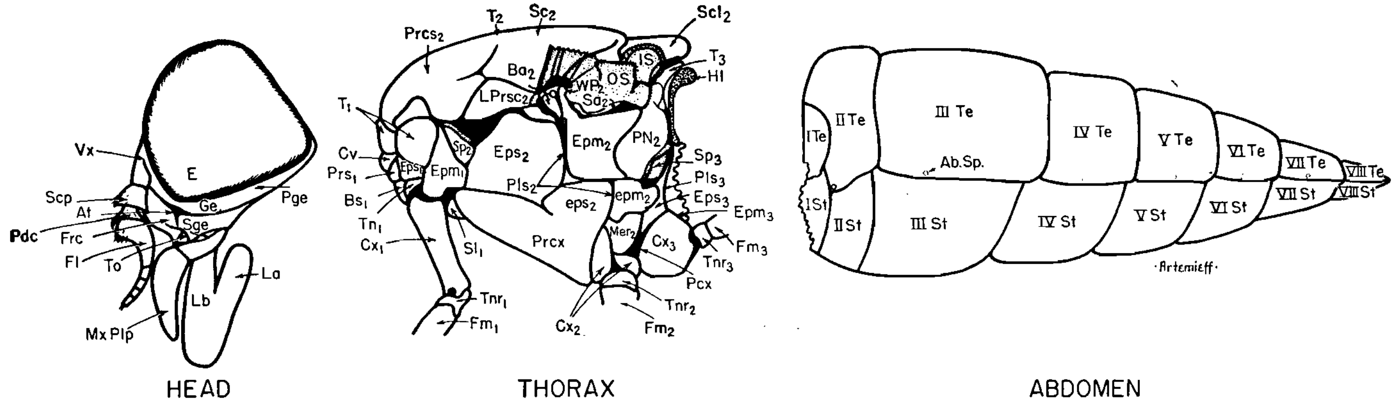


FIG. 11. Showing external morphology of a horsefly, *Tabanus punctifer*. Explanation of abbreviations: *Ab. Sp.*, abdominal spiracle; *At.*, anterior tentorial pit; *Ba.*, basalare; *Bs.*, basisternum; *Cv.*, cervical sclerite; *Cx.*, coxa; *E.*, eye; *Epm.* (*epm.₂*), epimeron (*Epm.₁* = propleuron in part; *Epm.₂* = pteropleuron; *epm.₂* = hypopleuron in part); *Eps.* (*eps.₂*) = episternum (*Eps.₁* = propleuron in part; *Eps.₂* = mesopleuron; *eps.₂* = sternopleuron in part); *Fl.*, flagellum of antenna; *Fm.*, femur; *Fr.c.*, frontoclypeus; *Ge.*, gena (cheek); *Hl.*, haltere; *I.S.*, inner squama; *La.*, labella; *Lb.*, labium; *L. Prsc.*, lobe of prescutum (notopleuron); *Mer.*, meron (hypopleuron in part); *Mx. Plp.*, maxillary palpus; *O.S.*, outer squama; *Pex.*, postcoxal bridge (postcoxale); *Pdc.*, pedicle of antenna; *Pge.*, postgena (occiput in part); *Pls.*, pleural suture (sterno-, mesopleural, mesepisternal sutures); *P. N.*, postnotum; *Prex.*, precoxal bridge (precoxale; sternopleuron in part); *Prs.*, presternum; *Prsc.*, prescutum; *Sa.*, subalare; *Sc.*, scutum; *Scl.*, scutellum; *Scp.*, scape of antenna; *Sge.* subgena (cheek in part); *Sl.*, sternellum; *Sp.*, spiracle; *St.*, sternite; *To.*, torma; *Vx.*, vertex; *W. P.*, wing process.

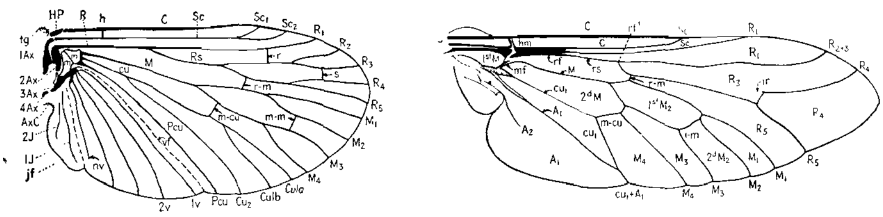
usually referred to as the *Pterygota,* and may be either *Exopterygota* (wings developing externally), e.g., cockroaches; or *Endopterygota* (wings developing internally), e.g., beetles (see under orders). There are typically two pairs of wings present, situated on the mesothorax and metathorax, though in many parasitic insects, such as the bedbugs, lice, fleas, certain louse flies, etc., the wings are absent. The wingless insects just mentioned should, of course, not be included with the *Apterygota,* a term designating a group of primitively wingless (apterous) forms. The above-mentioned parasitic, wingless insects belong to several different orders, as will be seen later.

In form the wing presents a more or less triangular appearance. The three sides are called margins: the costal margin is cephalic or anterior, the anal margin is posterior or caudad, and the outer margin (apical) is between these. The three angles connecting the margins are humeral (at the base), apical (apex of wing) and anal (between the apical and anal margins). Generally the fore and hind wings differ considerably in size; the fore wing in some groups, such as the mayflies, many butterflies and moths, and the bees and wasps, is larger than the hind wing; while in the grasshoppers, cockroaches, beetles, etc., the fore wing is narrow and serves largely as a cover (tegmina) to the hind wing, which folds fan-like. In the dragonflies, termites, and ant lions, the fore and hind wings are nearly equal. In the flies, the hind pair of wings is replaced by club-shaped organs known as *halteres,* leaving consequently only one pair of wings, hence the name Diptera (two-winged). In the calypterate Diptera (muscoid flies) there are present large lobes (squamae, also called "alulae") at the junction of the wings and the thorax.

There are some differences in the structure of the wings within the order, though for each order a certain general pattern prevails; e.g., the Neuroptera have thin membranous wings, often quite filmy; however, the wings of Diptera and many Hemiptera have the same texture throughout but possess fewer and differently arranged veins. The Diptera can, of course, be readily distinguished, as stated above, by the presence of but a single pair of wings. The winged Hemiptera-Heteroptera have the front wings thickened at the base, while the apical portion is membranous. The winged Homoptera have two pairs of wings of even texture.

The *venation* of the insect wing is an important element in classification, on account of the great variety of arrangement and the reliability of this character for the identification of the family and genus. The veins are hollow, rib-like structures which give strength to the wing. The areas of membrane between the veins are called cells; they are said to be open if the membranous area extends to the wing margin. By a careful study of the evidence, a fundamental type of wing venation has been constructed by Comstock and Needham and revised by Tillyard. The accompanying figure (Fig. 12A) illustrating this type will be useful in determining the identity of the principal veins. Fig. 12B illustrates Tillyard's revision of the R-C-N (Redtenbacher, Comstock, and Needham) system of nomenclature, which is used in this book.

Metamorphosis. In order to achieve the size and development of the parent, the young insect undergoes greater or less change in size, form, and structure. This series of changes is termed *metamorphosis*. The least



A PRIMITIVE WING TILLYARD B TABANUS

FIG. 12. (*A*) diagram of wing venation after the R-C-N nomenclature (Tillyard revision) with associated sclerites. *A*, anal veins = *V*, vannal veins except the first; *Ax*, axillary sclerites (1, 2, 3, 4); *AxC*, axillary cord; *C*, costa; *Cu*, cubitus; *h*, humeral cross-vein = *hm*; *HP*, humeral plate; *i-m*, inter-medial cross-vein; *ir*, interradial cross-vein (often spoken of as a spur on the third vein); *J*, jugal veins; *jf*, jugal fold; *M*, media; *mf*, medial form; *m*, *m'*, median plates; *m-m*, median cross-vein = *im*, intermedial cross-vein; *m-cu*, mediosubital cross-vein; *Pcu*, postcubitus (first anal); *R*, radius; *r*, radial cross-vein; *rf*, radial fork; *rf'*, 1st radial fork; *r-m*, radio-medial cross-vein; *Rs*, radial sector; *S*, sectorial cross-vein; *Sc*, subcosta; *tg*, rudiment of tegula; *V*, vannal veins; *vf*, vannal fold.

(*B*) wing of *Tabanus* labeled (in parenthesis) according to terminology of the Dipterists. Veins: *C* (costal); *Sc* (auxillary); R_1 (1st longitudinal); R_{2+3} (2nd longitudinal); R_4, R_5 (3rd longitudinal); M_1, M_2 (4th longitudinal); M_3, M_4Cu (5th longitudinal); *A* (6th longitudinal). Cells: *C* (costal); *Sc* (subcostal); R_1 (marginal); R_3 (1st submarginal); R_4 (2nd submarginal); R_5 (1st posterior); M_1 (2nd posterior); 2^dM_2 (3rd posterior); M_3 (4th posterior): M_4 (5th posterior); 1st M_2 (discal cell); *R* (1st basal); 2^dM (2nd basal); cu_1 (anal cell); A_1 (axillary).

change is found in the Apterygota (silverfish and springtails), which are primitively wingless insects, and hence the newly emerged young individual is externally unlike the parent only in size: this type of metamorphosis is termed *primitive* (Fig. 13).

A greater degree of metamorphosis occurs in the grasshopper. There is not only a great difference in size, but the absence of wings in the young is at once apparent. In order to reach the winged condition, the young individual casts its skin at intervals, and with each *ecdysis* achieves longer wings until, after a certain number of molts, the fully developed wings appear. The following stages may be recognized: (1) *egg*, (2) *nymph,* (3) *imago* or sexually mature adult. This type of metamorphosis is called *simple* or *incomplete,* and the orders comprising these are known as the *Heterometabola* (Fig. 14).

The greatest difference between the newly hatched young and the parents occurs in such forms as the housefly (Fig. 15) and the butterfly. In these forms the newly hatched insect has no resemblance whatsoever

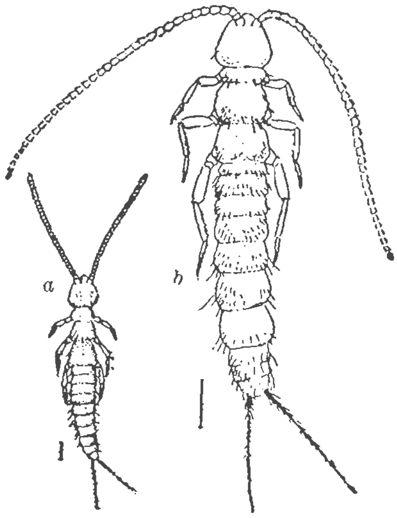


FIG. 13. Illustrating primitive metamorphosis. (*a*) young of a Thysanuran insect (Campodea), (*b*) adult of the same. (After Kellogg.)

to the adult, but looks more like a segmented worm. However, the internal anatomy and certain other features are distinctly insectan. The fact that the young are mandibulate and the adults haustellate in Diptera and Lepidoptera offers much interesting ground for ecological discussion. In order to attain the winged condition of the adult, the wingless, worm-

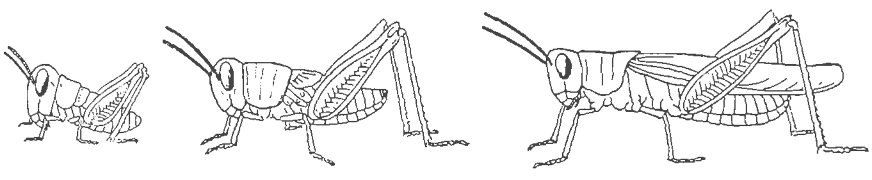


FIG. 14. Illustrating simple metamorphosis. (*A*) young wingless grasshopper; (*B*) grasshopper showing wing pads after the first molt; (*C*) adult of the same. (Redrawn after Packard.)

like form must undergo many profound changes, and a new stage is interjected, the *pupa,* or resting stage, in which this transformation is accomplished. The newly hatched young insect emerging from the egg is called the *larva,* hence the following stages: (1) *egg,* (2) *larva,* (3) *pupa,* and (4) *imago.* This type is termed *complex* or *complete* metamorphosis, and the orders comprising these are known as the *Holometabola.*

Importance of knowing internal anatomy. It is important that the student familiarize himself with the internal anatomy of insects,[1] giving special attention to the digestive system and its accessory structures, such as the salivary glands. Two cases will illustrate reasons for this:

1. The simplest condition in which the internal organs of insects are involved in disease transmission is in the case of the common housefly, in which pathogenic organisms may be sucked up with infectious dejecta from those ill with cholera or typhoid fever and passed out with the feces of the fly, which may be deposited on human food, either in their original virulent condition or more or less attenuated. Regurgitation on the part of the insect may be equally effective.

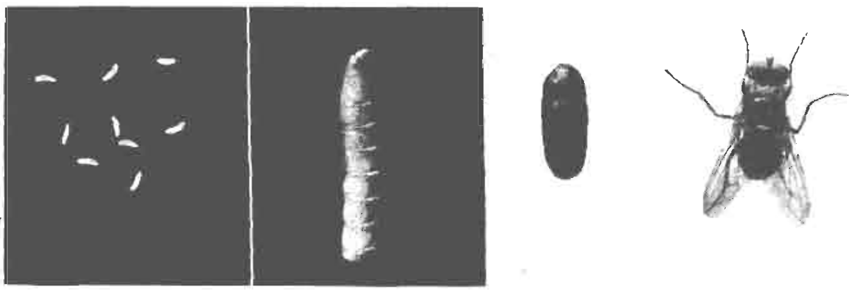


FIG. 15. Illustrating complex metamorphosis in the life cycle of the common housefly: egg, larva, pupa, adult.

2. A more complicated situation exists in the case of the *Anopheles* mosquito, which sucks up pathogenic organisms (plasmodia) with its meal of human blood, whereupon these parasites undergo vital sexual changes within the body of the insect, eventually finding lodgment in the salivary glands before final introduction by the "bite" into the next human victim. The insect in this case is the essential natural vector.

Digestive system. There are three distinct regions to the insect intestine (Fig. 16); namely, (1) the *fore-gut,* consisting of the mouth, pharynx, esophagus, crop, and proventriculus; (2) the *mid-gut,* comprising the stomach; and (3) the *hind-gut,* consisting of the ileum, colon, rectum, and anus. The crop presents merely a widened portion of the esophagus in the more generalized forms and serves as a food receptacle. In the more specialized groups, such as the Diptera and Lepidoptera, the crop is expanded into a capacious pocket or pouch. In such forms as the cockroach and grasshopper the proventriculus consists of a highly muscular dilation provided internally with chitinous teeth for grinding or straining food. The stomach is a simple sac into which open the *gastric*

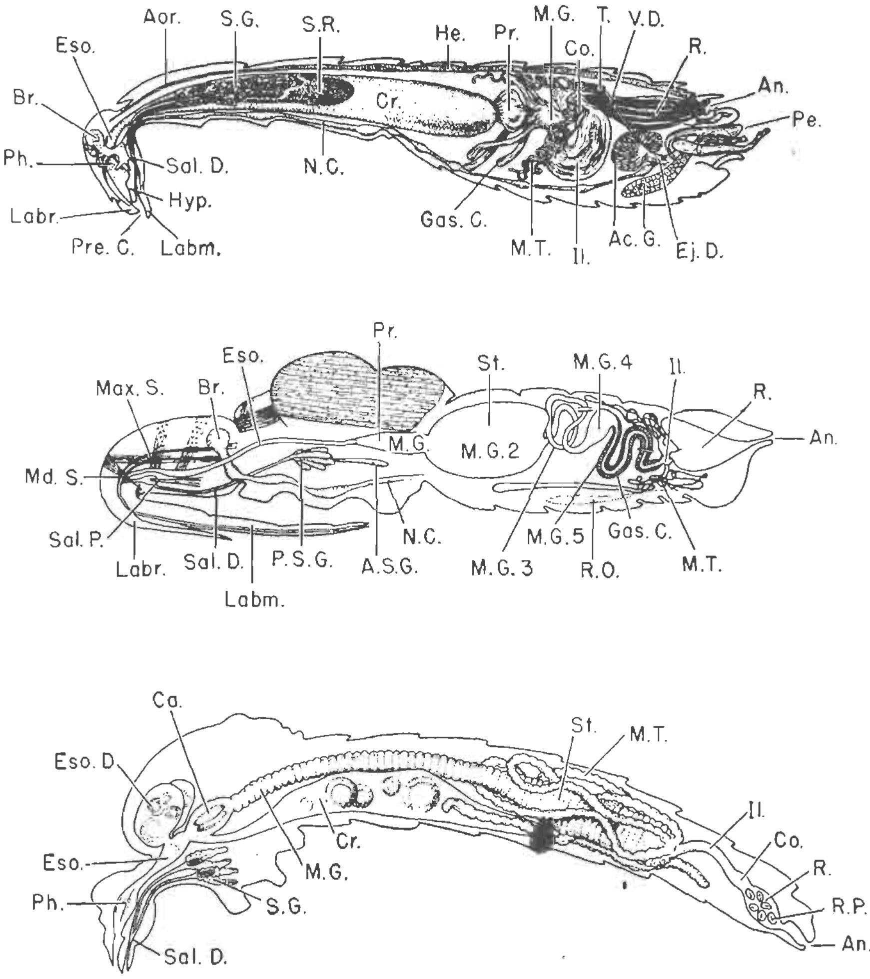


FIG. 16. Showing digestive tract of cockroach, Order Orthoptera (*top*); of conenose bug, Order Hemiptera (*middle*); and of anopheline mosquito, Order Diptera (*bottom*). Explanation of abbreviations: *Ac.G.*, accessory glands; *An.*, anus; *Aor.*, aorta; *A.S.G.*, accessory salivary gland; *Br.*, brain; *Ca.*, cardia; *Co.*, colon; *Cr.*, crop; *Ej.D.*, ejaculatory duct; *Eso.*, esophagus; *Eso.D.*, esophageal diverticula; *Gas.C.*, gastric caeca; *He.*, heart; *Hyp.*, hypopharynx; *Il.*, ilium; *Labm.*, labium; *Labr.*, labrum; *Max.S.*, maxillary stylet; *Md.S.*, mandibular stylet; *M.G.*, mid-gut; *M.T.*, Malpighian tubules; *N.C.*, nerve cord; *Pe.*, penis; *Ph.*, pharynx; *Pr.*, proventriculus; *Pre.C.*, preoral cavity; *P.S.G.*, principal salivary gland; *R.*, rectum; *R.O.*, reproductive organs; *R.P.*, rectal papilla; *Sal.D.*, salivary duct; *S.G.*, salivary gland; *Sal.P.*, salivary pump; *S.R.*, salivary reservoir; *St.*, stomach; *T.*, testis; *V.D.*, vas deferens. (Top figure adapted after Miall; middle figure adapted after Elson; and lower figure, Herms.)

caeca, generally few in number, which give rise to certain digestive fluids. At both ends of the stomach are located valves which control the flow of the food. There is much variation in the length and the degree of convolution of the hind intestine, but usually the three regions mentioned, namely, ileum, colon, and rectum, can be located. Emptying into the ileum are the excretory or *Malpighian tubules,* varying in number and length in the several groups of insects.

The *salivary system* consists of a pair of salivary glands which may be lobed; they are situated within the head, often extending into the thorax. Usually each gland empties into a *salivary* duct, the two ducts discharging into a common duct which opens into the mouth at the base of the labium. In many species of insects there is present a pair of *salivary reservoirs;* these may be located near the opening of the common duct and then present a compound condition, or may be situated on either side of the esophagus at the end of a long slender duct.

Insect larvae. When insect larvae, parasitic or accidental, are encountered in the body of man or beast, confusion may arise because of the worm-like appearance of the invaders, which may be incorrectly classified as worms, for example, muscoid fly larvae. These larvae are short and plump, and have 11 or 12 well-marked segments. Microscopic examination of fragments will reveal tracheal tubules, which are not present in worms. The student of medical entomology must be thoroughly familiar with the immature stages of arthropods.

Although the larvae of insects belonging to the Order Diptera (flies and mosquitoes) are characteristically legless and usually have an unchitinized, eyeless head, the variations within the order are considerable. *Maggots* of muscoid flies, for example, are smooth, with the body tapering to the inconspicuous head, which bears hook-like mouth parts; while the larvae (*wrigglers*) of mosquitoes have a chitinous, freely moving, conspicuous head with faceted eyes, and both the head and body bear many hairs, spines, and bristles. The larvae of fleas (Order Siphonaptera) are also legless; the chitinous head is well developed; and each of the thoracic and abdominal segments is well armed with a band of bristles. The larvae of beetles (Order Coleoptera), commonly called *grubs,* have three pairs of legs on the thorax only; the head is well developed; the body may be hairy, spiny, or naked. The larvae of moths and butterflies (Order Lepidoptera) have three pairs of thoracic legs and two to five abdominal prolegs; the head is prominent, and the mouth parts are usually well developed and mandibulate; they are commonly called *caterpillars* and are hairy, spiny, or naked. The larvae of ants, bees, and wasps (Order Hymenoptera) are usually called *grubs;* they are usually without legs (apodous); the head is more or less well developed; the body is usually fairly smooth and gourd-shaped.

Insect classification. The medical entomologist must be equipped with a good knowledge of the basic principles of classification so that he may be able to place the insect at hand correctly in at least its proper Order and family; in the case of insects of importance in medical entomology he should be able to run the specimen down to the species with the aid of a key. To determine the *order* to which an insect belongs one need usually know only the venation and structure of the wings, if present, and the type of mouth parts. This knowledge will enable the student to place at least 90 per cent of the commoner insects in their proper *orders*. Unfortunately, the parasitic forms have undergone many modifications, such as reduction or loss of wings and great alteration in form, but generally the mouth parts will serve as a ready means of crude identification. Before passing on to a list of the orders of insects, the usual basis for classification may be pointed out, viz.:

1. Wings: (a) presence or absence, (b) form, (c) structure.
2. Mouth parts: (a) biting (mandibulate), (b) sucking (haustellate).
3. Metamorphosis: (a) primitive, (b) simple (incomplete), (c) complex (complete).

The orders of insects. The following list of orders of the class Insecta includes those generally recognized by present-day entomologists. The Comstocks in 1901 listed 19 orders of insects. The present number, 33 (Essig, 1942[2]) has been primarily achieved by splitting up the original Orthoptera and Neuroptera.

Subclass I. Apterygota (Ap'ter-y-go'ta), apterous (primitively wingless) insects.

1. Order Protura (Pro-tu'ra), (*protos,* first; *oura,* tail), proturans; (because of certain affinities with the Chilopoda and Symphyla, these insects are sometimes placed in a separate class, Myrientomata Berlese); biting mouth parts; primitive metamorphosis.
2. Order Thysanura (Thy'sa-nu'ra), (*thysanos,* a tassel; *oura,* tail); silverfish, bristletails; biting mouth parts; primitive metamorphosis.
3. Order Aptera (Ap'ter-a), (*á-*, without; *ptera,* wings); campodeids; biting mouth parts; primitive metamorphosis.
4. Order Collembola (Col-lem'bo-la), (*colla,* glue; *embolos,* wedge or peg); springtails, snowfleas; biting mouth parts; primitive metamorphosis.

Subclass II. Pterygota (Pter'y-go'ta), winged insects.

Division I. Exopterygota (Exo-pter'y-go'ta).

(Heterometabola: Insects with simple metamorphosis.)

5. Order Orthoptera (Or-thop'ter-a), (*orthos,* straight; *ptera,* wings); grasshoppers, locusts, katydids, crickets, mole crickets; biting mouth parts; simple metamorphosis.

6. Order Blattaria (Blat-tar'ia), (*blatta*, insect that shuns the light); cockroaches; biting mouth parts; simple metamorphosis.
7. Order Dermaptera (Der-map'ter-a), (*derma*, skin; *ptera*, wings); earwigs; biting mouth parts; simple metamorphosis.
8. Order Grylloblattodea (Gryl'lo-blat-to-de'a), (*gryllus*, a cricket; *blatta*, a cockroach); grylloblattids; biting mouth parts; simple metamorphosis.
9. Order Phasmida (Phas'mi-da), (*phasma*, an apparition); walking sticks, leaf insects; biting mouth parts; simple metamorphosis.
10. Order Mantodea (Man-to'de-a), (*mantis*, a prophet; *eidos*, form, like); preying mantids, praying mantids, soothsayers; biting mouth parts; simple metamorphosis.
11. Order Diploglossata (Dip'lo-glos'sa-ta), (*diplos*, double; *glosso*, tongue); hemimerids; biting mouth parts; simple metamorphosis.
12. Order Plecoptera (Ple-cop'ter-a), (*plektos*, to weave; *ptera*, wings); stoneflies, salmonflies, perlids; biting mouth parts; simple metamorphosis.
13. Order Isoptera (I-sop'ter-a), (*isos*, equal; *ptera*, wings); termites, white ants; biting mouth parts; simple metamorphosis.
14. Order Zoraptera (Zor-ap'te-ra), (*zoros*, pure; *ptera*, wings); zorapterans; biting mouth parts; simple metamorphosis.
15. Order Embioptera (Em'bi-op'-ter-a), (*embios*, lively; *ptera*, wings); embiids, webspinners; biting mouth parts; simple metamorphosis.
16. Order Corrodentia (Cor'ro-den'ti-a), (*corrodens*, gnawing); book lice, bark lice, dust lice, psocids; biting mouth parts; simple metamorphosis.
17. Order Mallophaga (Mal-loph'a-ga), (*mallos*, a lock of wool; *phagein*, to eat); bird lice, biting bird lice; biting mouth parts; simple metamorphosis.
18. Order Ephemerida (Eph'e-mer'i-da), (*ephemeros*, living but a day); mayflies, dayflies, ephemerids; vestigial mouth parts; simple metamorphosis.
19. Order Odonata (O-don'a-ta), (*odontos*, a tooth); damsel flies, dragonflies, mosquito hawks, devil's darning needles, snakedoctors; biting mouth parts; simple metamorphosis.
20. Order Thysanoptera (Thy'sa-nop'ter-a), (*thysanos*, fringe; *ptera*, wings); thrips; rasping-sucking mouth parts; simple metamorphosis.
21. Order Anoplura (An'o-plu'ra), (*anoplos*, unarmed, *oura*, tail) true lice, sucking lice; piercing-sucking mouth parts; simple metamorphosis.
22. Order Hemiptera (He-mip'ter-a), (*hemi*, half; *ptera*, wings); bugs, cicadas, treehoppers, leafhoppers, aphids, scale insects, etc.; piercing-sucking mouth parts; simple metamorphosis.

Division II. Endopterygota (En-do-pter'y-go'ta).
(Holometabola—Insects with complex metamorphosis).

23. Order Megaloptera (Meg'a-lop'ter-a), (*megalos*, great; *ptera*, wings); dobsonflies, alderflies, sialids; biting mouth parts; complex metamorphosis.
24. Order Neuroptera (Neu-rop'ter-a), (*neura*, nerves; *ptera*, wings); lace-

wings, nerve-winged insects; biting mouth parts; complex metamorphosis.

25. Order Raphidiodea (Ra-phid'i-o'de-a), (*raphis,* needle; referring to ovipositor of female); snakeflies, serpent flies; biting mouth parts; complex metamorphosis.
26. Order Mecoptera (Me-cop'ter-a), (*mecos,* length, long; *ptera,* wings); scorpionflies; biting mouth parts; complex metamorphosis.
27. Order Trichoptera (Tri-chop'ter-a), (*thrix,* hair; *ptera,* wings); caddis flies, caseflies, water moths; biting mouth parts, complex metamorphosis.
28. Order Coleoptera (Col'e-op'ter-a), (*coleos,* sheath; *ptera,* wings); beetles, weevils, biting mouth parts; complex metamorphosis.
29. Order Strepsiptera (Strep-sip'ter-a), (*strepsi,* twist; *ptera,* wings); stylops, twisted wings; biting mouth parts; complex metamorphosis.
30. Order Diptera (Dip'ter-a), (*dis,* two; *ptera,* wings); flies, gnats, mosquitoes; sucking mouth parts; complex metamorphosis.
31. Order Siphonaptera (Si'pho-nap'ter-a), (*siphon,* a tube; *aptera,* wingless); fleas, chigoes; piercing-sucking mouth parts; complex metamorphosis. *(See Snodgrass.)*
32. Order Hymenoptera (Hy'men-op'ter-a), (*hymen,* a membrane; *ptera,* wings); bees, wasps, ants, sawflies, horntails, etc.; lapping-sucking mouth parts; complex metamorphosis.
33. Order Lepidoptera (Lep'i-dop'ter-a), (*lepidos,* scale; *ptera,* wing); moths, butterflies; sucking mouth parts; complex metamorphosis.

The Arachnida. The class Arachnida includes the ticks, mites, spiders, scorpions, and related forms. Among the species of arachnids are some of the most important parasites and vectors of diseases of man and beast, such as the ticks which carry spotted fever and relapsing fever of man,

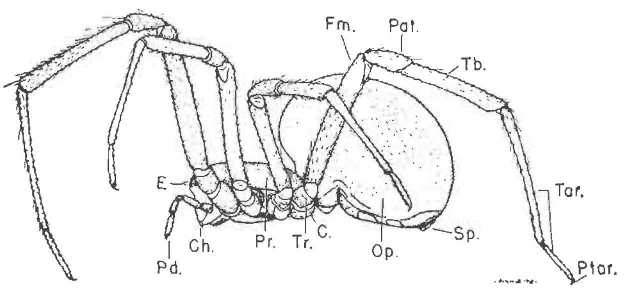


FIG. 17. Showing external morphology of a spider. *C,* coxa; *Ch.,* chelicera; *E,* eyes; *Fm.,* femur; *Op.,* opisthosoma; *Pat.,* patella; *Pd.,* pedipalp; *Pr.,* prosoma; *Ptar.,* pretarsus; *Sp.,* spinnerets; *Tar.,* tarsus; *Tb.,* tibia; *Tr.,* trochanter.

and Texas cattle fever and bovine anaplasmosis. Parasitic mites cause acariasis, often serious, such as mange, scabies, and various forms of itch, and may, like the ticks, serve as vectors of disease, particularly Tsutsugamushi disease.

The more important arachnids lack distinct segmentation of the body, e.g., ticks, mites, and spiders; while scorpions, pseudoscorpions, and a few others are clearly segmented. The body is divided into two parts (Fig. 17): first the *cephalothorax* (prosoma) composed of combined head and thorax, and second, the *abdomen* (opisthosoma). In the ticks and mites there is a strong fusion of the cephalothorax and the abdomen so that the body becomes sac-like in form.

Adult arachnids with few exceptions have four pairs of legs, though the larvae of ticks and nearly all mites have but three pairs. In spiders there is a pair of *pedipalpi*, which in scorpions, whip scorpions, and pseudoscorpions are strongly chelate. All arachnids are devoid of wings and antennae. Eyes, when present, are simple; compound eyes are wanting. The mouth parts usually consist of a pair of piercing *chelicerae, pedipalpi*, and in the Acarina a *hypostome*. The respiratory system of many arachnids, particularly ticks and mites, is tracheal as in insects,

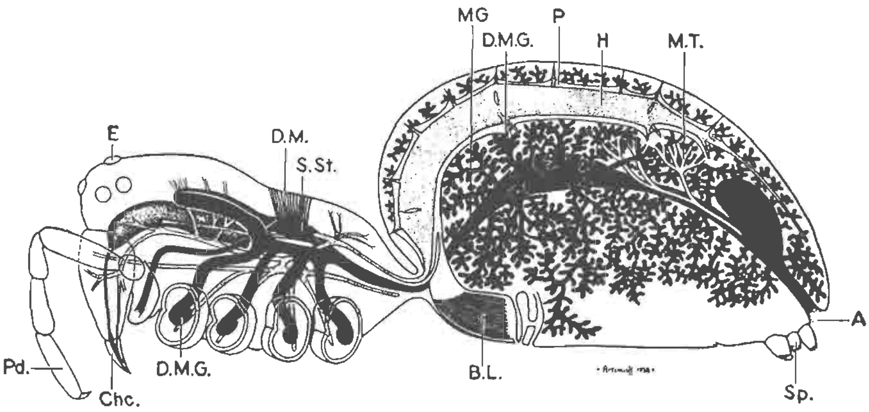


FIG. 18. Showing internal anatomy of a spider. *A*, anus; *B.L.*, book lung; *Chc.*, chelicera; *D.M.*, dorsal muscle; *D.M.G.*, diverticula of mid-gut; *E.*, eye; *H.*, heart; *M.G.*, mid-gut; *M.T.*, Malpighian tubule; *P.*, pericardium; *Pd.*, pedipalp; *Sp.*, spinneret; *S.St.*, sucking stomach. (Redrawn and adapted after various authors.)

except that there is usually but one pair of spiracles. In spiders the respiratory organ is a combination of lung books and tracheae. There is frequently a strong sexual dimorphism in the arachnids; the males are commonly smaller than the females. (See later chapters dealing with ticks, mites, and spiders.)

In general the arachnids are predatory and perhaps most of them are nocturnal, although there are some species of spiders and scorpions which are largely diurnal.

Arachnid development. Arachnida deposit eggs in all the orders except the scorpions and some mites (*Pediculoides*), which are viviparous. Eggs are usually numerous, particularly in the ticks, which may deposit as many as 18,000 per female. The newly hatched individuals have the general form of the adults, although the number of legs may vary, e.g., newly hatched ticks and mites usually have three pairs of legs. Metamorphosis is simple, as in cockroaches and grasshoppers. Molting takes place as in insects, the various stages being termed instars as in the Insecta. The longevity of many arachnids is remarkable: ticks have been known to live for as many as six to seven years, and some species are able to endure starvation for several years.

Internal anatomy. The digestive tract of arachnids (Fig. 18) is characterized by various types of diverticulae and branched tubules. The diverticulae, which diverge from the tract between the sucking organ of the pharynx and the mesenteron, range, according to Savory[4] from two short simple sacs directed forward in the cephalothorax to a condition of five pairs, four of which extend laterally, reaching the bases of the legs, and enter the coxae for a short distance; also there is a very complex type which branch and divide and become very large. Leading from the mesenteron is a complex system of branched tubules which occupy most of the abdomen and function partly as a digestive gland and partly as a reservoir. The Arachnida are thus enabled to store large quantities of food and to undergo long periods of fasting, an adaptation which particularly favors the parasitic forms.

The excretory organs of the arachnids are *Malpighian tubules,* which empty into the gut, and *coxal glands* which empty excretory products into tubules and discharge to the exterior from openings which vary in relations to the coxae with the several orders.

Arachnid classification.* Excluding the marine arachnid order *Xiphosura,* king-crabs or horseshoe crabs, the following orders, all terrestrial, may be considered in a classification for the purposes of this work, namely, (1) *Scorpionida,* scorpions; (2) *Araneida* (*Araneae*), spiders; (3) *Pedipalpida,* whip scorpions; (4) *Pseudoscorpionida* (*Chelonethida*), false scorpions; (5) *Solpugida* (*Solifugae*), sun spiders or wind scorpions; (6) *Phalangida* (*Opiliones*), harvestmen or harvest spiders; (7) *Acarina* (*Acari*), mites and ticks.

Among the more important gross characters used to separate the terrestrial arachnids into orders are the following: *segmentation* of the body; presence or absence of *pedicle;* presence or absence of *telson; chelicerae,* large or small; *pedipalpi,* chelate or unchelate; location and form of *spiracles.*

* Some authors include among the Arachnida the Pycnogonida (Pantopoda) sea-spiders, the Tardigrada (water-bears), and the Pentastomida (Linguatulida) tongue worms.

Key to the Terrestrial Orders of the Class Arachnida†

1. Abdomen distinctly segmented 2
   Abdomen not segmented 7
2. Abdomen with tail-like prolongation 3
   Abdomen without tail-like prolongation 4
3. Tail stoutly armed with a sting at end .... *Scorpionida* (Scorpions)
   Tail slender, without sting .... *Pedipalpida* (Whip scorpions)
4. Palpi chelate .... *Pseudoscorpionida* (False or Pseudoscorpions)
   Palpi not chelate 5
5. Abdomen constricted at base and narrowly joined to the cephalothorax .... *Pedipalpida*
   Abdomen not constricted at base and broadly joined to cephalothorax 6
6. Legs very long and slender, body hairless, whole body fused together .... *Phalangida* (Harvest spiders)
   Legs moderate; body hairy, appearing 3-segmented .... *Solpugida* (Solpugids, Vinegarroons)
7. Abdomen constricted at base and joined to cephalothorax by a narrow stalk .... *Araneida* (True spiders)
   Abdomen fused with cephalothorax .... *Acarina* (Ticks and Mites)

BIBLIOGRAPHY

1. Snodgrass, R. E., 1935. *Principles of Insect Morphology*. New York: McGraw-Hill Book Co., Inc. 667 pp.

2. Essig, E. O., 1942. *College Entomology*. New York: The Macmillan Co., vii + 900 pp. (308 figs).

3. Snodgrass, R. E., 1946. *The Skeletal Anatomy of Fleas*. "Smithsonian Misc. Collect.," vol. 104, no. 18, Washington, D. C.: Smithsonian Inst. (Publ. no. 3815). 89 pp. + 21 plates.

4. Savory, Theodore H., 1935. *The Arachnida*. London: Edward Arnold & Co. 218 pp.

† Adapted after various authors.

CHAPTER VI

INSECT AND ARACHNID MOUTH PARTS

Importance of mouth parts. No doubt all insects possessing mouth parts capable of piercing the skin of man and beast may be regarded with some suspicion by the medical entomologist as being potential vectors of blood-inhabiting microparasites, even though the bloodsucking habit may not normally prevail. The nonpiercing insects manifestly cannot be responsible for infections introduced directly into the circulation, except through previously injured surfaces. Thus the housefly although possessing nonpiercing mouth parts, is recognized as one of the vectors of *Trypanosoma hippicum* Darling, a blood-inhabiting parasite and the causal organism of murrina of horses and mules. It carries the parasites on its proboscis, which becomes contaminated while it is feeding from bleeding wounds caused by stable flies (*Stomoxys*) or other means, and conveys the organisms thence to open wounds on healthy animals.

The student of arthropods in relation to disease must have a thorough knowledge of their mouth parts and feeding habits for at least two reasons: first, that he may know how the pathogenic organisms are acquired as well as transmitted and, secondly, that he may know, if occasion requires, what control measures may be best employed. The medical entomologist will soon find that a knowledge of the feeding habits of the adult insect alone is inadequate and that he must be familiar with the feeding habits of the insect in *all* stages of its life cycle. The larvae of many of the sucking insects have mandibulate (biting-chewing) mouth parts. The biting mouth parts of the worm-like flea larva enable it to ingest particles of excrement or other matter in which eggs of the double-pored dog tapeworm occur, and it may thus become an intermediate host of this worm, passing the infection on to the adult flea which, if *ingested* by a suitable host, becomes the agent of infection. Insects with complete metamorphosis commonly change from chewing mouth parts in the larval stage to the sucking type in the adult. Insects with simple metamorphosis, like the cockroaches, have biting mouth parts in all active stages of development. Snodgrass[1] describes the feeding apparatus of biting and sucking insects affecting man and animals in great detail.

Classification of mouth parts. All insect mouth parts, however highly specialized, may be traced to the simple primitive type still existing with some modification in the cockroach. Insect mouth parts are commonly divided into two general classes: (1) *mandibulate,* i.e., biting and chewing, as in the cockroaches, grasshoppers, and beetles; and (2) *haustellate,* i.e., sucking, as in the bugs, flies, bees, butterflies, and moths. This classification is far from satisfactory for use in the field of medical entomology. For example, the common housefly, *Musca domestica* Linn., and the stable fly, *Stomoxys calcitrans* (Linn.), both possess haustellate mouth parts and belong to the same family of insects, Muscidae, hence are systematically closely related, yet are quite unrelated in their manner of disease transmission. By virtue of its efficient piercing stylets the stable fly has the power to pierce the skin and suck blood, thus enabling it to become a direct infector; whereas the housefly, because of the structure of its proboscis and its inability to suck blood by piercing the skin, is only indirectly responsible for infection, i.e., it is more particularly a food contaminator.

Obviously insects could be grouped on the basis of mouth parts and feeding habits, into two simple divisions: (1) piercing as in mosquitoes, and (2) nonpiercing, as in cockroaches. This, however, leaves much to be desired in the light of medical entomology, hence the following grouping is suggested.

1. *Orthopteron type:* generalized mouth parts consisting of opposable jaws used in biting and chewing; upper and lower lips easily recognized, as in the cockroach and grasshopper.
2. *Thysanopteron type:* mouth parts representing a transitional type, minute in size; approaching the biting form, more particularly rasping, but functioning as suctorial organs; as in the thrips; the right mandible is greatly reduced or possibly even absent, causing a peculiar asymmetry shown in the drawing (Fig. 20).
3. *Hemipteron type:* mouth parts consisting of piercing-sucking organs, comprising four stylets closely ensheathed within the elongated labium, forming a proboscis, as in the conenose bug and bedbug.
4. *Anopluran type:* mouth parts piercing-sucking, in a sac concealed within the head but evertible when functioning. Three stylets, i.e., one consisting of the pair of united maxillae, the other two consisting of the hypopharynx and labium; mandibles vestigial, e.g., sucking lice.
5. *Dipteron type:* suctorial organs, piercing in some, nonpiercing in others; no single representative is available to illustrate the entire group of Diptera, hence the following subtypes may be recognized.
   a. First subtype: mosquito; mouth parts consisting of six stylets, loosely ensheathed within the labium.
   b. Second subtype: horsefly; mouth parts consisting of six short blade-like structures used for piercing and cutting, all loosely ensheathed within the labium.

   c. Third subtype: stable fly; piercing stylets reduced to two in number, closely ensheathed within the labium.
   d. Fourth subtype: housefly; mouth parts consisting of a muscular proboscis, not suited for piercing; stylets rudimentary.
   e. Fifth subtype: the louse fly; mouth parts closely related to those of the third subtype.
6. *Siphonapteron type:* piercing-sucking, fully exposed mouth parts consisting of a pair of broad maxillary lobes bearing long palpi; two (paired) stylets, a pair of maxillary lacinae (lacinial blades), chief cutting organs commonly regarded as "mandibles"; a slender labium with parallel palpi, and a median unpaired stylet, the "epipharynx," the labrum is difficult to interpret (Snodgrass), e.g., fleas.
7. *Hymenopteron type:* mouth parts consisting of suctorial, lapping organs, mandibles specialized for portage and combat, as in the bee, wasp, and ant.
8. *Lepidopteron type:* mouth parts consisting of a suctorial coiled tube, as in the butterfly.

MORPHOLOGY OF MOUTH PARTS

Orthopteron type. To illustrate the orthopteron type of mouth structure either the grasshopper or the cockroach may be used. This type, the mandibulate or biting-chewing, is the generalized or primitive form and will serve as a basis for later comparisons and derivations. It is not directly of importance in medical entomology except as it furnishes a basis for a better understanding of the haustellate or sucking types.

If the head of a cockroach (Fig. 19) is viewed from the side and again from the front, the relative position of the separate parts will be better understood. Separating the individual parts, the following structures will be observed. In front, low down on the head, hangs the *labrum* or lip, easily lifted as one would raise a hinged lid, the hinge line being at the lower edge of the sclerite or plate, known as the *clypeus.*

The labrum functions as does the upper lip in higher animals, i.e., it draws the food toward the mandibles. In this the labrum is greatly aided by a rough structure called the *epipharynx*, which forms the inner lining of the labrum and clypeus. Because of the close association of these two structures they are often referred to as a double organ, the *labrum-epipharynx.* Removing the labrum, a pair of heavy, opposable jaws, the *mandibles*, is exposed. These are biting structures *par excellence.* They are toothed and movable laterally. Dislodging the mandibles brings into view the pair of maxillae, or accessory jaws. These organs are known as *first maxillae.* They are composite structures separable into *cardo, stipes, lacinia, galea,* and *palpus*, which should be carefully observed, inasmuch as they undergo great modification in the remaining types of mouth parts. The two supporting sclerites of the maxillae are

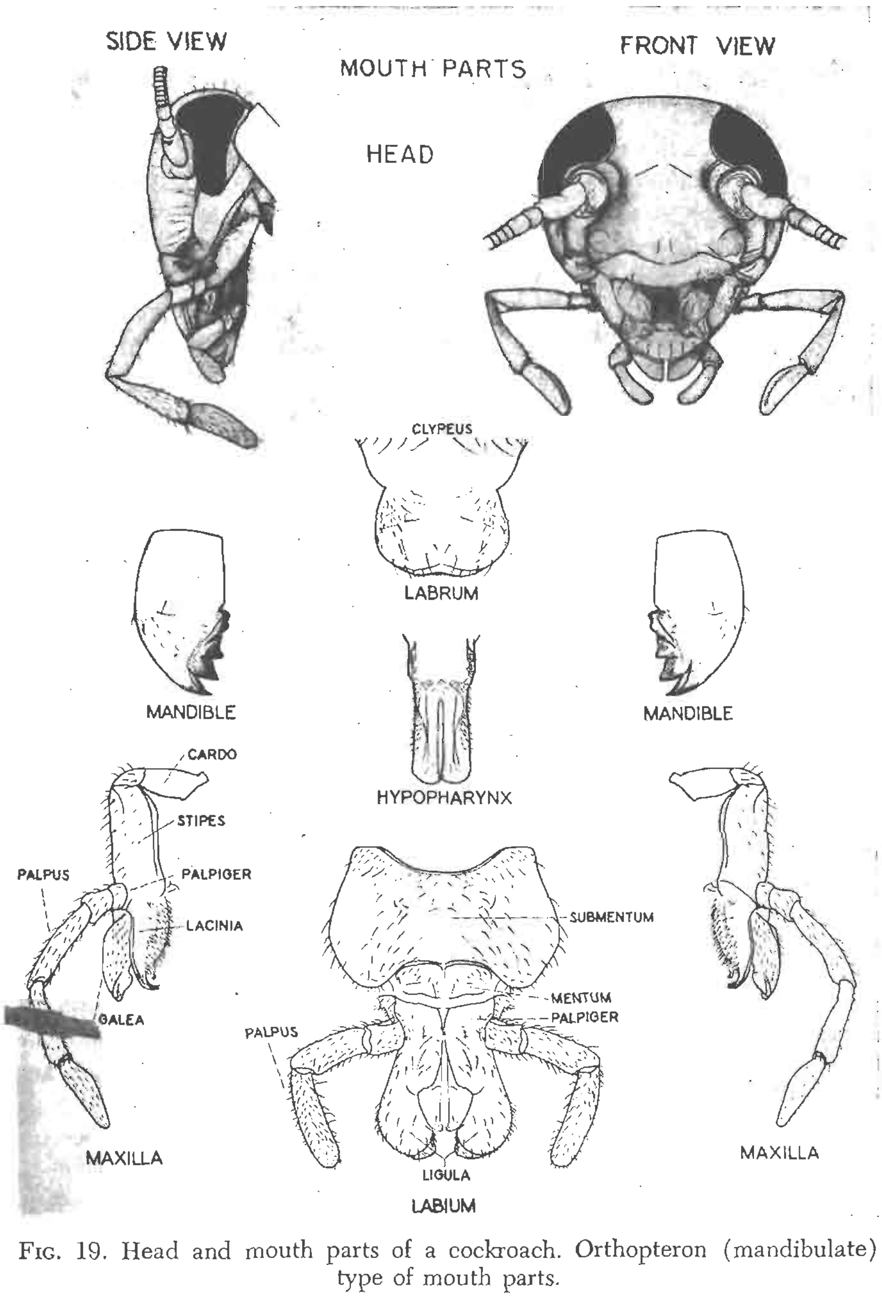


FIG. 19. Head and mouth parts of a cockroach. Orthopteron (mandibulate) type of mouth parts.

cardo (basal) and *stipes* (the second), while the distal lobes are (1) the *maxillary palpus* (a jointed structure), (2) the *galea* (median and fleshy), (3) the *lacinia* (inner and toothed), capable of aiding in comminuting food.

Underneath the maxillae and forming the floor of the mouth lies the lower lip or *labium,* a double structure frequently called the *second maxilla.* On the same plan as the maxillae, the labium consists of a basal sclerite, the *submentum,* followed by the *mentum,* upon which rest the *labial palpi* (a pair of outer, jointed structures to the right and left), and the *ligula* (a pair of strap-like plates which together correspond to the upper lip.) The labium is also subject to much modification in insects.

The fleshy organ still remaining in the mouth cavity after the parts just described have been removed is the tongue or *hypopharynx,* an organ of taste, comparable in a measure to the tongue of higher animals.

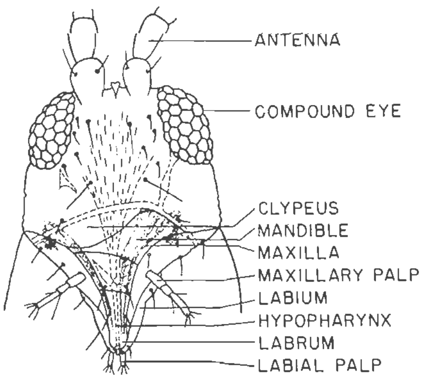


FIG. 20. Head and mouth parts of a thrips. Thysanopteron type. (Redrawn after Borden.)

The mandibles are most useful landmarks, since they are almost universally present in insects, though in various degrees of development, from the strong mandibles of certain beetles (Lucanidae) to the vestigial structures of fleas (Siphonaptera). In the Hymenoptera, even though the order is haustellate, the mandibles are nevertheless present and important structures, serving, however, in the honeybee as wax implements and organs of defense, and in ants as organs of portage and combat. In Hemiptera and many Diptera the mandibles are converted into piercing organs while the maxillae are also greatly changed in form.

Thysanopteron type. This type (Fig. 20) is interesting phylogenetically as a connecting link between the biting-chewing and piercing-sucking mouth parts. It is in the very minute thrips, order Thysanoptera, that we find this transitional type of mouth parts, biting in general structure but sucking in function. According to various authors the right mandible is reduced, and by others it is said to be entirely wanting, making the head and mouth parts asymmetrical; the left mandible, maxillae, and hypopharynx are elongate, suggesting the stylet of the piercing type, and adapted to move in and out through a circular opening at the apex

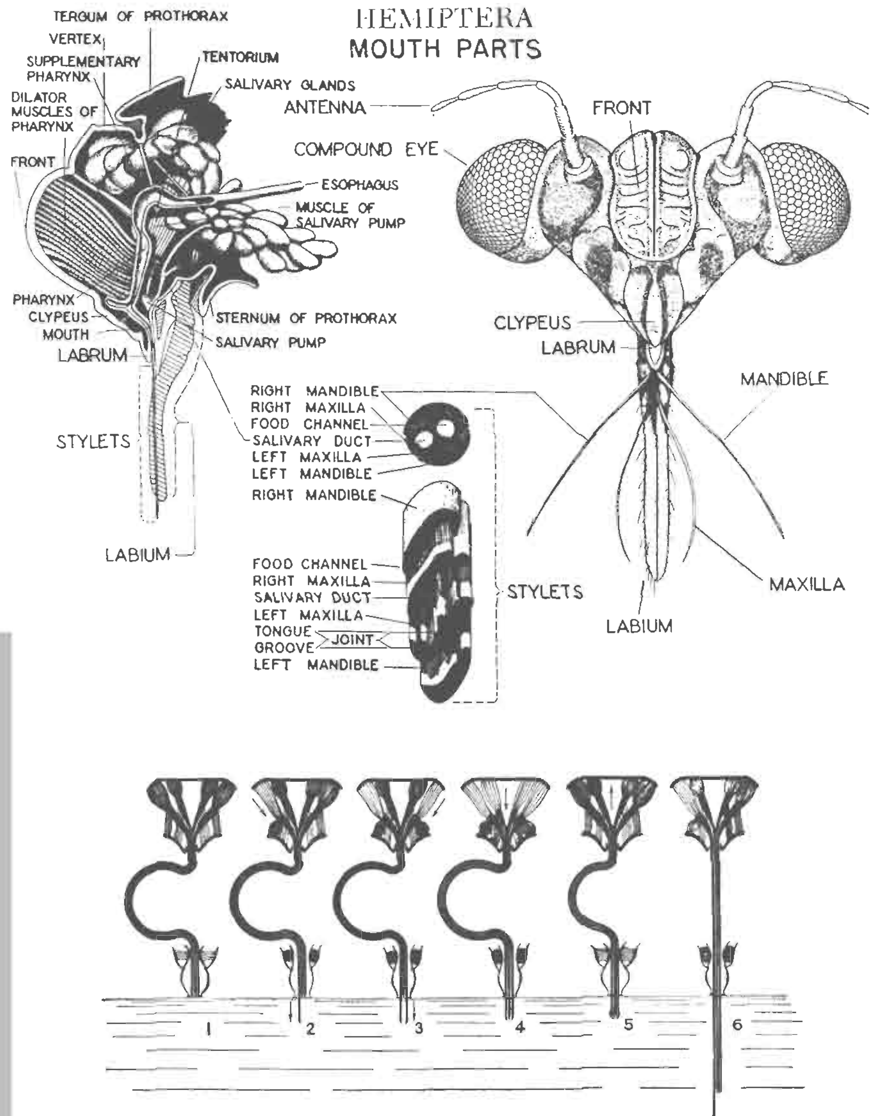


FIG. 21. Hemipteron type of mouth parts. (Adapted after various authors.)

of the head. No food channel is formed, but the sap from plants is lapped up as it exudes from the abraded surface.

Hemipteron type. A very different sort of organ from those above described is found in the order Hemiptera (Fig. 21). Here the cylindrical labium forms a prominent beak-like proboscis which is usually three- or four- (rarely one- or two-) jointed and telescopic. It is devoid of palpi. The proboscis encloses a pair of *mandibles,* often with terminal

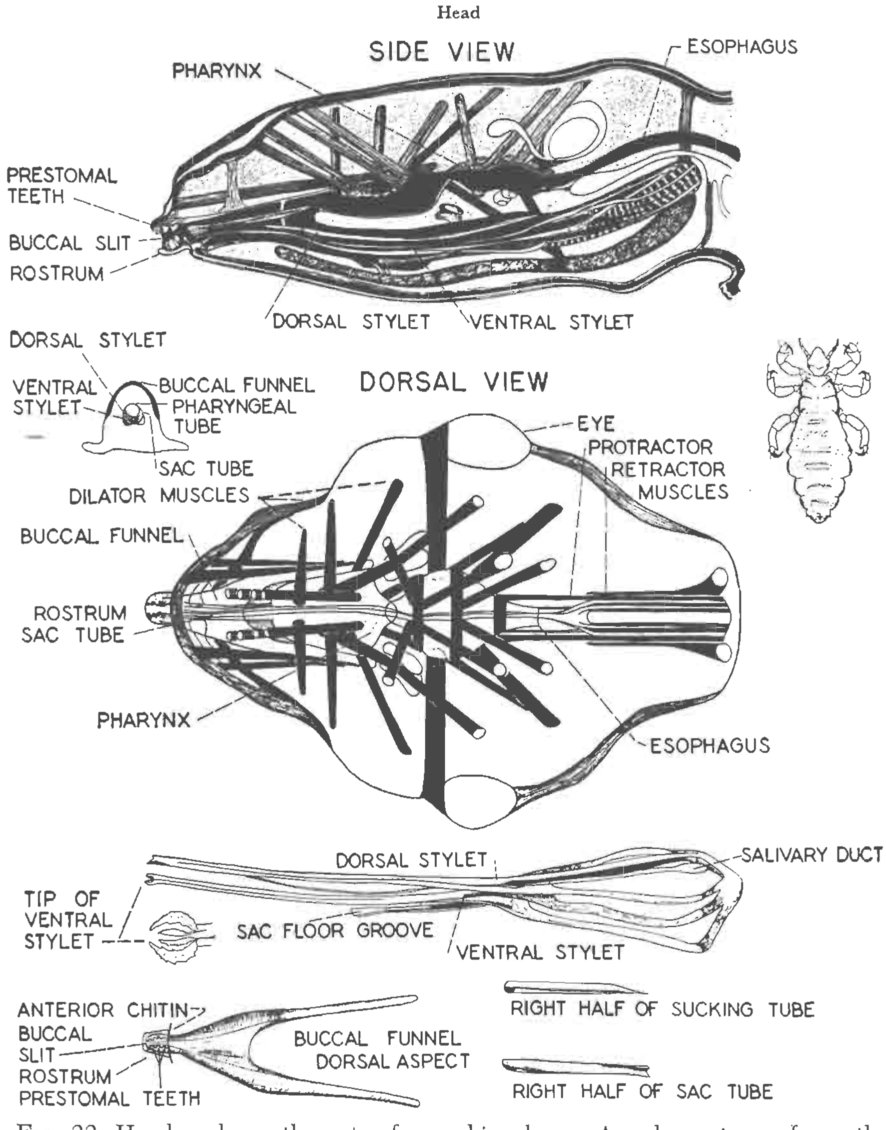


FIG. 22. Head and mouth parts of a sucking louse. Anopluron type of mouth parts. (Redrawn and adapted after various authors.)

barbs, and a pair of *maxillae*, all four are stylet-like and efficient piercing organs, the maxillae operating as a unit and the mandibles functioning separately. The maxillae are closely apposed, forming the food and are narrower and provided with conspicuous *palpi*. The *hypopharynx* and *labrum-epipharynx* are both lancet-like. In the male these piercing parts are very weakly developed.

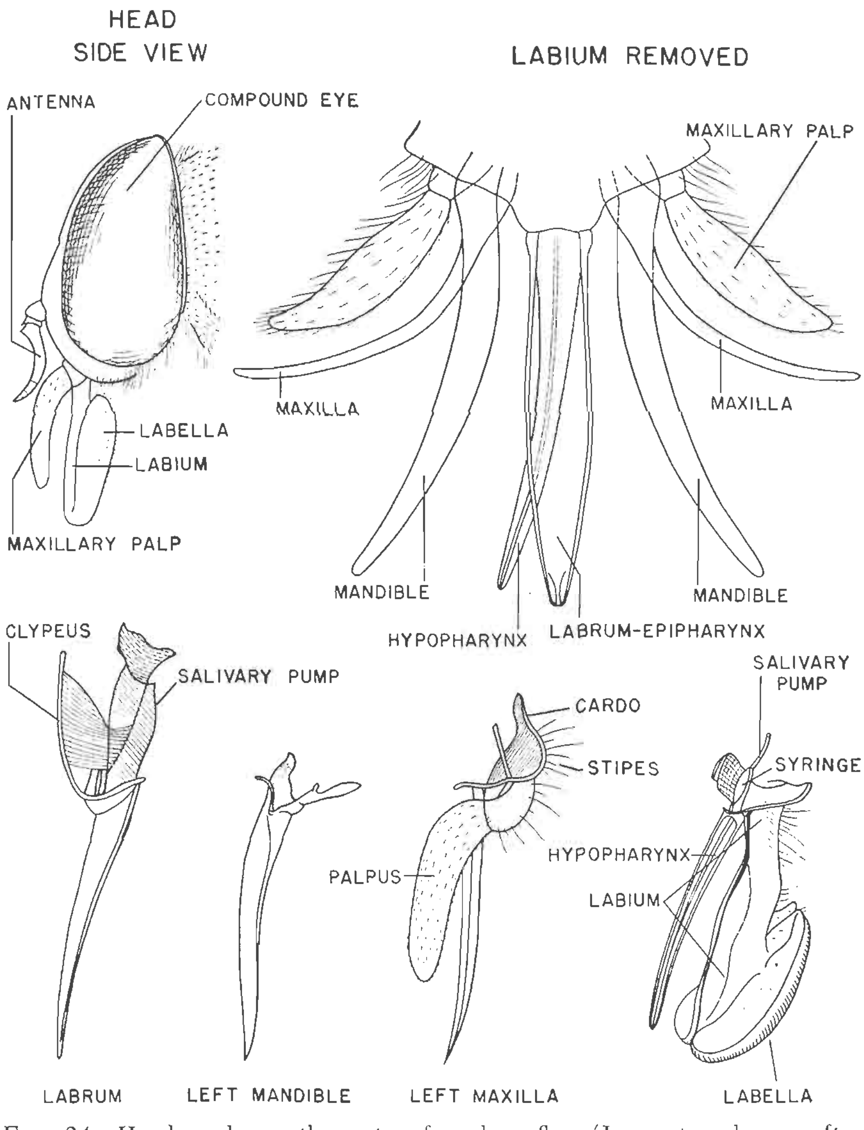


FIG. 24. Head and mouth parts of a horsefly. (In part redrawn after Snodgrass.)

(c) *Third subtype, the stable fly.* This subtype (Fig. 25) is represented by a group of flies in which the mouth parts are distinctly specialized for piercing, and show, together with the next subtype, to what extent these structures may become modified within the same family of insects.

The proboscis at rest is carried at the position of a bayonet at charge, and is therefore provided with a prominent muscular elbow or knee. This conspicuous organ (the proboscis) is the *labium* terminating in the *labella*, which are provided with a complex series of rasping denticles. Within the folds of the labium and easily removable through the upper groove lie two stylets: the *labrum*, the uppermost and heavier, and the *hypopharynx*, a lower and weaker one, the two forming a sucking tube

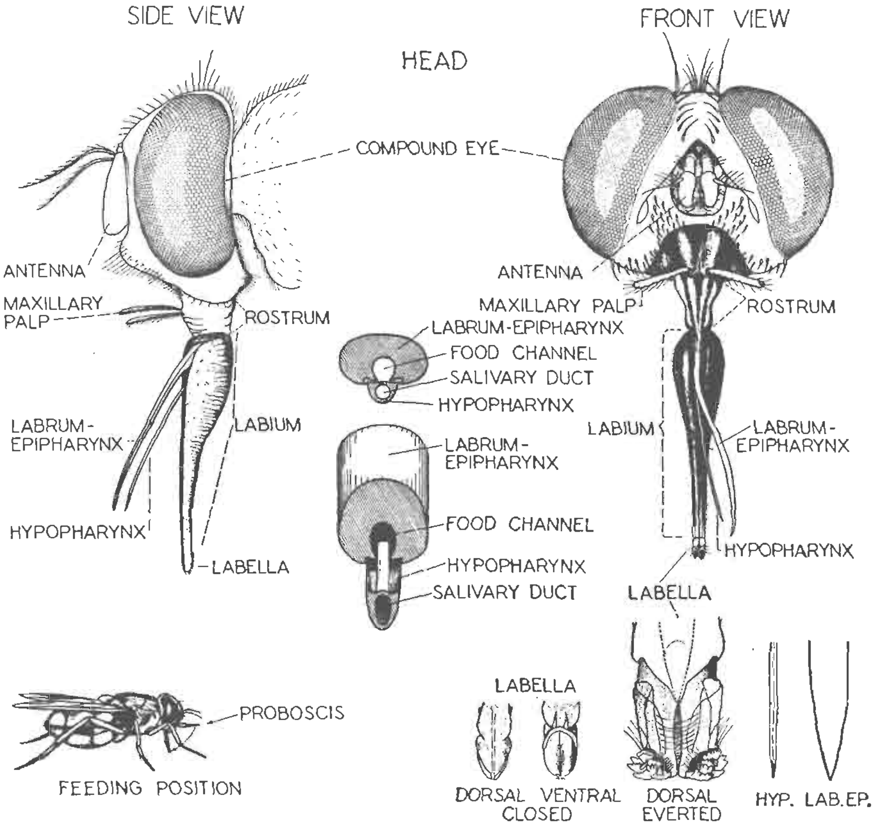


FIG. 25. Head and mouth parts of the stablefly. (Redrawn and adapted after various authors.)

supported within the folds of the labium. The maxillary palpi are located at the proximal end of the proboscis.

(d) *Fourth subtype, the housefly.* Here (Fig. 26) the prominent fleshy proboscis consists mainly of the *labium*, which terminates in a pair of corrugated rasping organs, the *labella*, and is attached in elbow-like form to the elongated head. The entire structure is highly muscular and may be either protruded in feeding or partially withdrawn while at rest. Lying on top of the grooved labium is the inconspicuous spade-like

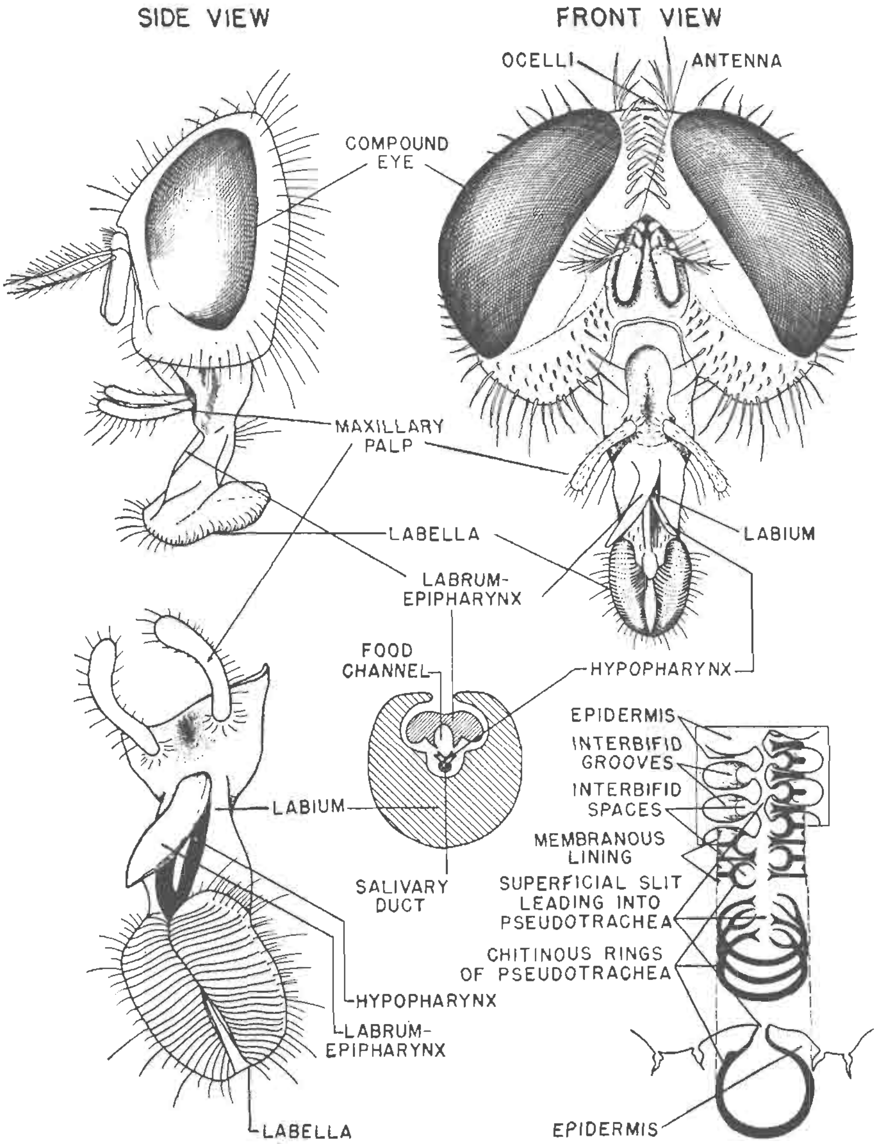


FIG. 26. Head and mouth parts of the common housefly. Lower right-hand figure shows detailed cross section of a pseudotrachea in the labella. (Redrawn and adapted after various authors.)

labrum, which forms, with the hypopharynx, a sucking tube, supported by the labium, which latter also encloses the salivary canal. By an examination of the labrum it will be seen that this forms a sort of convex covering to the concaved hypopharynx, thus giving rise to a food tube. The maxillae have evidently become fused with the fleshy elbow of the proboscis, and only the prominent *maxillary palpi* remain.

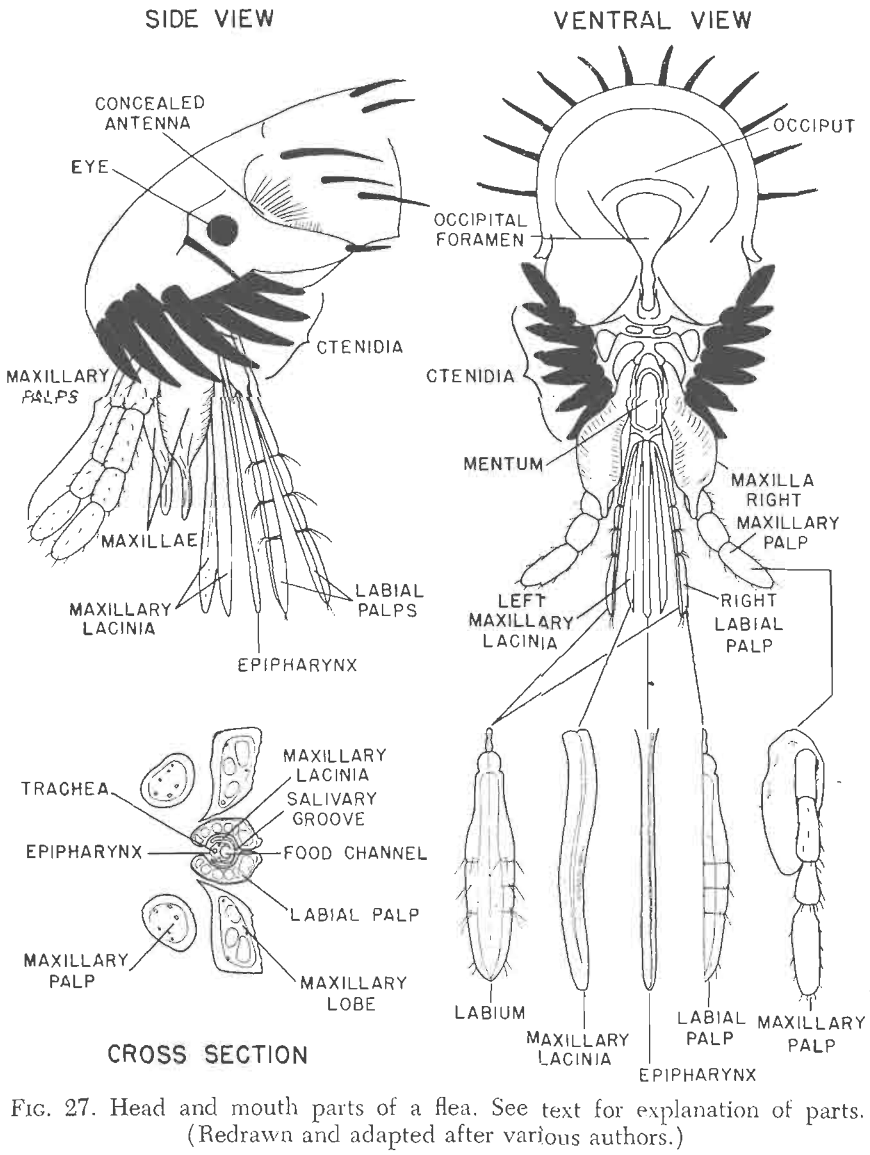


FIG. 27. Head and mouth parts of a flea. See text for explanation of parts. (Redrawn and adapted after various authors.)

(e) *Fifth subtype, the louse fly.* The louse flies, members of the family Hippoboscidae, have mouth parts closely related to those of the third subtype, the stable fly; the characteristic tubular or cylindrical haustellum is adapted for penetration into the skin of the host. The *labrum- epipharynx* is stylet-shaped; its proximal portion is strongly chitinized and rigid, whereas the distal end is membranous and very flexible.[2] The hypopharynx in the two common species, *Pseudolynchia canariensis* (Macq.) = [*Lynchia maura* (Bigot)] and *Melophagus*

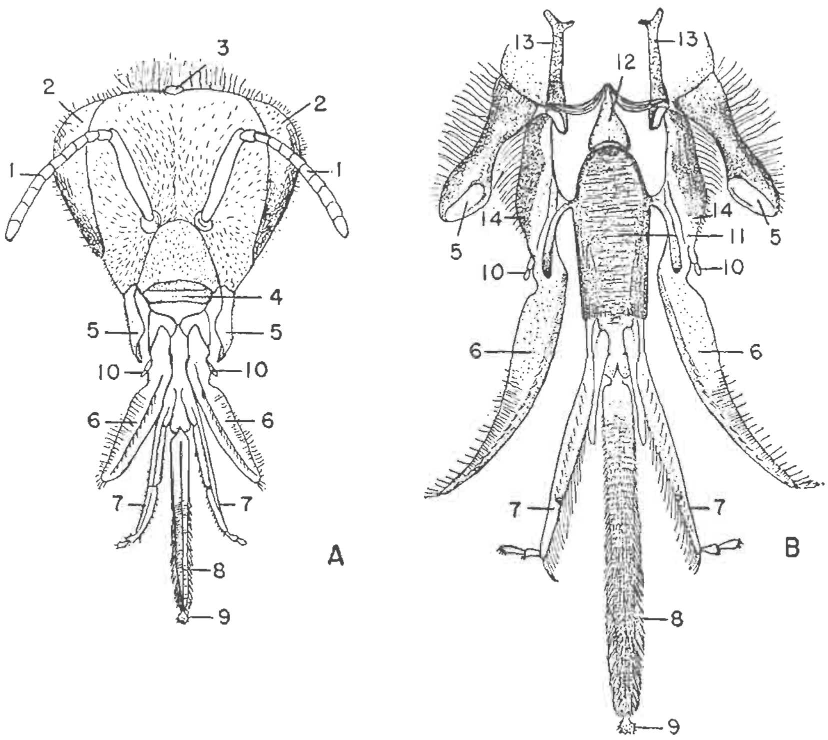


FIG. 28. Head and mouth parts of the honeybee (*Apis mellifica*). Both types of mouth parts well developed, but the mandibles are used chiefly for portage and modeling. (Hymenopteron type.) (*A*) front view of the head showing (1) antennae, (2) compound eyes, (3) simple eye, (4) labrum, (5) mandibles, (6) maxillae (galea), (7) labium (palpi only), (8) labium (glossa), (9) bouton, (10) maxillary palpus. (*B*) mouth parts removed to show (5) mandibles, (6) maxillae (lacinia), (7) labium (palpi only), (8) labium (glossa), (9) bouton, (10) maxillary palpus, (11) prementum, (12) mentum, (13) cardo, (14) stipes.

ovinus Linn., is nearly as long as the combined haustellum and labellum and is a very slender and hyaline mouth part.

Siphonapteron type. The mouth parts of the Siphonaptera (fleas) (Fig. 27), though typically of a piercing-sucking type, are peculiar to this order of insects. The broad *maxillary lobes* bearing long palpi are conspicuous landmarks (Fig. 27); the other organ (slender) bearing long parallel palpi is the *labium*. The principal blade-like piercing organs are a pair of independently moveable structures commonly referred to as mandibles but are said to be maxillary laciniae by Snodgrass (*loc. cit.*) and others. The mandibles are believed to be rudimentary in fleas. The median stylet (unpaired) is said to be the *epipharynx,* not the labrum of many authors, the labrum being difficult to demonstrate. The *epipharynx* is said to be closely embraced by the lacinial blades. The three stylets (a pair of maxillary laciniae and the epipharynx) are held in the channel of the labium. The labrum is rudimentary, and the existence of a hypopharynx is not demonstrable. The wound is made by the protraction and retraction of the maxillary laciniae. As soon as the blood begins to flow, it is drawn up into the pharynx by the action of both the cibarial and the pharyngeal pumps.

Hymenopteron type. In this type the two general classes of mouth structures, the *mandibulate* and *haustellate,* find full development in the same species, though the mandibles are not involved in the feeding process. The honeybee (Fig. 28) serves as a representative species. The *labrum* is narrow and quite simple; the *mandibles* are easily distinguish-

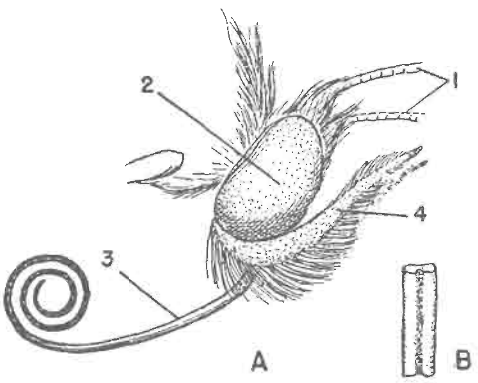


FIG. 29. Head and mouth parts of a butterfly (*Vanessa* sp.). (*A*) side view: suctorial, coiled tube, Lepidopteron type. (1) antennae; (2) compound eye; (3) proboscis, consisting only of galeae; (4) labial palpus. (The labrum is not visible in side view.) (*B*) section of proboscis showing double nature.

able and are useful wax implements. In ants the mandibles are highly efficient organs of portage and weapons of defense. The *maxillae* form the lateral conspicuous wings of the suctorial parts; the *lacinia* and *galea* are fused, and the *maxillary palpi* are minute. The *labium* is represented by the long structures to the right and left of the middle tube, which is probably the *hypopharynx.* The hypopharynx terminates in the spoon-like *labellum* or bouton which completes the lapping character of the subtype.

Lepidopteron type. This type, represented by the commoner butterflies and moths, is typically a coiled, sucking tube capable of great elongation. Taking the cabbage butterfly, *Pieris rapae* (Linn.), as an example (Fig. 29) the *labrum* is seen to be greatly reduced, and the mandibles are absent. (These may be weakly present in the lower

Lepidoptera.) The maxillae are apparently represented only by the *galeae,* which by close approximation of their inner grooved surfaces form the long, coiled proboscis. The double structure of the proboscis can be easily demonstrated by manipulation. The labium is represented by the *labial palpi.*

Orders of Insects Arranged According to Type of Mouth Parts

I. *Orthopteron type:** Biting and chewing (mandibulate) mouth parts. Orders Orthoptera, Blattaria, Dermaptera, Grylloblattodea, Phasmida, Mantodea, Coleoptera, Isoptera, Odonata, Diploglossata, Plecoptera, Zoraptera, Embioptera, Corrodentia, Mallophaga, Ephemerida (vestigial mouth parts), Megaloptera, Neuroptera, Raphidiodea, Mecoptera, Trichoptera, Strepsiptera, Protura, Thysanura, Collembola, Aptera.

II. *Thysanopteron type:* Rasping-sucking; biting in structure, but sucking in function; right mandible reduced or wanting; represents a transitional form between biting and sucking mouth parts.
Order Thysanoptera.

III. *Hemipteron type:* Elongated (beak-like), 3- or 4-segmented proboscis, snugly enclosing stylet-like mandibles and maxillae; piercing and suctorial.
Order Hemiptera (including Homoptera).

IV. *Anopluran type:* Piercing-sucking mouth parts lie in sac concealed in head when not in use; mandibles vestigial; maxillae, hypopharynx, and labium form functional stylets; circlet of evertible oral teeth provides means of attachment when feeding.
Order Anoplura.

V. *Dipteron types:* Unsegmented proboscis, which may or may not contain piercing stylets (see previous description).
Order Diptera.

VI. *Siphonapteron type:* Piercing-sucking mouth parts; principal blade-like, piercing organs consisting of a pair of independently moveable maxillary laciniae, which together with stylet-like epipharynx are held within the channel of the labium.
Order Siphonaptera.

VII. *Hymenopteron type:* For feeding purposes the mouth parts are of a nonpiercing, lapping type; mandibles present, but used for combat and portage.
Order Hymenoptera.

VIII. *Lepidopteron type:* Greatly elongated coiled sucking tube; nonpiercing.
Order Lepidoptera.

Arachnid mouth parts. In his recent excellent study of the feeding organs of Arachnida, Snodgrass[3] points out that arachnids come from

* Orthopteron is used here merely to indicate a biting-chewing type which varies considerably within this grouping of the orders.

an ancestral line that never acquired organs for mastication and even today have no true jaws, hence are forced to subsist on liquids. A liquid

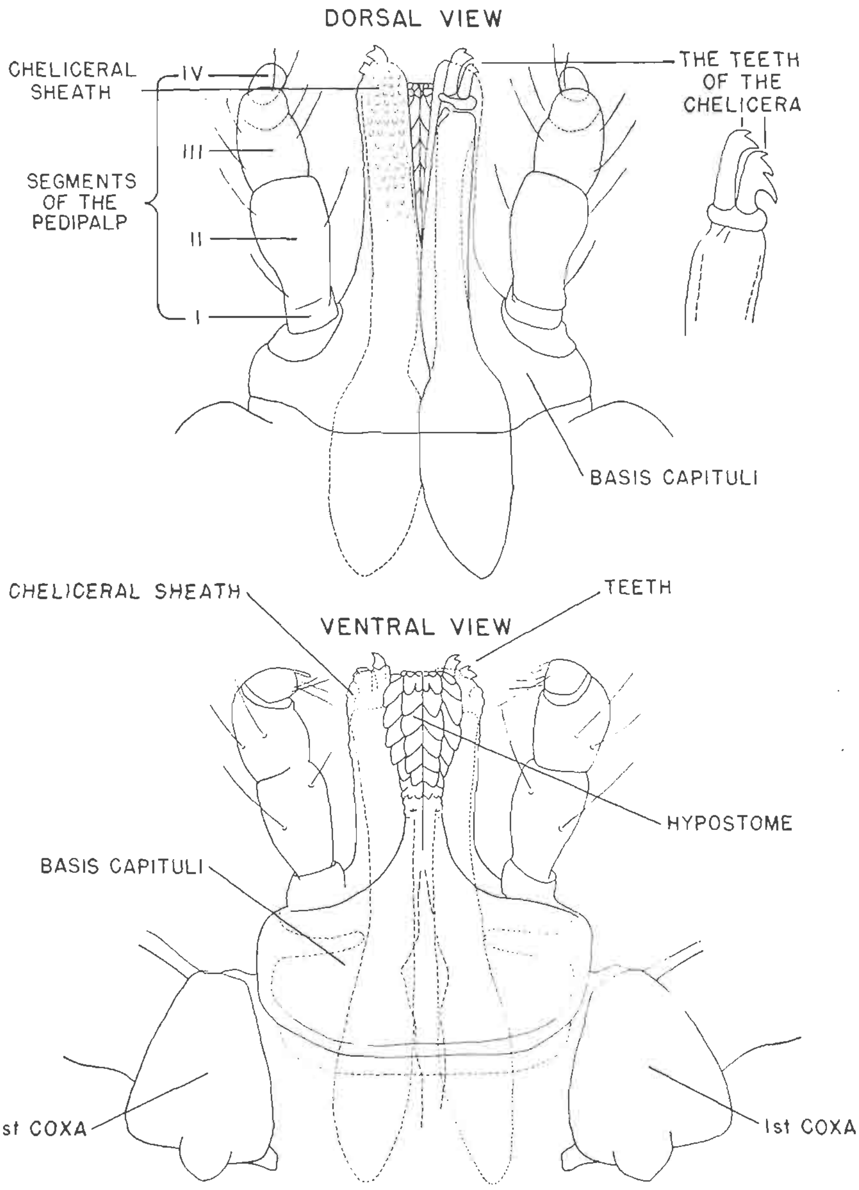


FIG. 30. Tick, capitulum; showing mouth parts of a tick (Arachnida, Acarina).

diet requires an ingestion pump, and with all arachnids a highly developed sucking apparatus constitutes the essential part of the feeding mechanism. The *chelicerae* (a pair) are the first postoral appendages of the arachnid (see Chapter XX), and while functioning more or less as "jaws" they are not homologous with the mandibles of insects; they are used for grasping, holding, tearing, crushing, or piercing. The leg-like *pedipalps* are the second postoral appendages of the Arachnida and are the homologues of the mandibles of mandibulate arthropods (Snodgrass). These organs are modified in various ways, functioning as organs of prehension, protection, and, in male spiders, as sperm-carrying organs. In the scorpions the pedipalps are chelate and serve for catching, holding, and crushing the prey.

According to Snodgrass (*loc. cit.*) the only features of the acarine (ticks and mites) mouth parts that cannot be homologized with structures generally present in other Arachnida are the variously developed appendicular lobes or processes often associated with the distal part of the hypostome (Fig. 30). Also among the trombidiform mites the chelicerae become progressively adapted for piercing by a transformation of the moveable digits into hooks or stylets.

BIBLIOGRAPHY

1. Snodgrass, R. E., 1944. *The Feeding Apparatus of Biting and Sucking Insects Affecting Man and Animals.* "Smithsonian Misc. Collect.," vol. 104, no. 7, (Publ. no. 3773). Washington, D. C.: Smithsonian Inst. 113 pp.

2. Jobling, B., 1926. "A comparative study of the structure of the head and mouth parts in the Hippoboscidae (Diptera Pupipara)," *Parasitology*, 18:319–49.

3. Snodgrass, R. E., 1948. *The Feeding Organs of Arachnida, Including Mites and Ticks.* "Smithsonian Misc. Collect.," vol. 110, no. 10. Washington, D. C.: Smithsonian Inst. (Publ. no. 3944). 93 pp. (29 figs).

CHAPTER VII

COCKROACHES AND BEETLES

A. COCKROACHES

Order Blattaria

Cockroaches of today are direct descendants of the huge ancient cockroaches (Paleoblatta), some reaching about six inches in length, which together with enoromous dragonflies (Protodonata) "had conquered the world"[1] some 250,000,000 years ago. Though much smaller in size but still cosmopolitan, modern cockroaches have changed but little in general structure. They are usually flattened dorsoventrally with smooth (sometimes pilose) tough integument, varying in color from chestnut-brown to black in the more pestiferous house-invading species, but commonly green, or orange and other colors, in tropical species. The head is decidedly flexed backward and downward when at rest. The prominent antennae are filiform and many segmented. There are two pairs of wings in most species; in some species the wings are vestigial, in others the wings are well developed in the males and short in the females, e.g., *Blatta orientalis* Linn. The outer pair of narrow wings (tegmina) is thick and leathery, while the inner pair is membranous and folds fan-like. While most cockroaches possess the power of flight, they are typically runners (cursorial) and can move swiftly by means of their long well-developed legs. They are highly gregarious and primarily nocturnal. but most cockroaches may be seen occasionally singly during the day. Metamorphosis is simple.

Feeding habits. The mouth parts of cockroaches are of the generalized biting-chewing type (Orthopteron). They feed on a great variety of foods (omnivorous), with preference for starchy and sugary materials. They will sip milk, nibble at cheese, pastry, meats, grain products, sugar, sweet chocolate—in fact, practically no edible material available for human consumption is exempt from attack by these vile insects which just as freely feed on book bindings, the sized inner lining of shoes, dead insects, their own cast-off skins and dead and crippled kin, fresh and dried blood, excrement, and sputum. They feed principally at night, hence we live in ignorance as to their disgusting and dangerous feeding habits.

Furthermore, roaches habitually disgorge portions of their partly digested food at intervals and drop their feces wherever they go. They also discharge a nauseous secretion from both the mouth and from glands opening on the body, imparting a persistent and typical "cockroach" odor to food and dishes with which they come in contact.

Species of sanitary importance. There are about 2,250 species of cockroaches belonging to some 250 genera heretofore placed in the Family Blattidae of the Order Orthoptera, now generally put into the Order Blattaria. Cockroaches occur throughout the world but are chiefly tropical. World-wide distribution of certain species has been effected by maritime trading; holds of vessels, the galleys, and crew's sleeping quar-

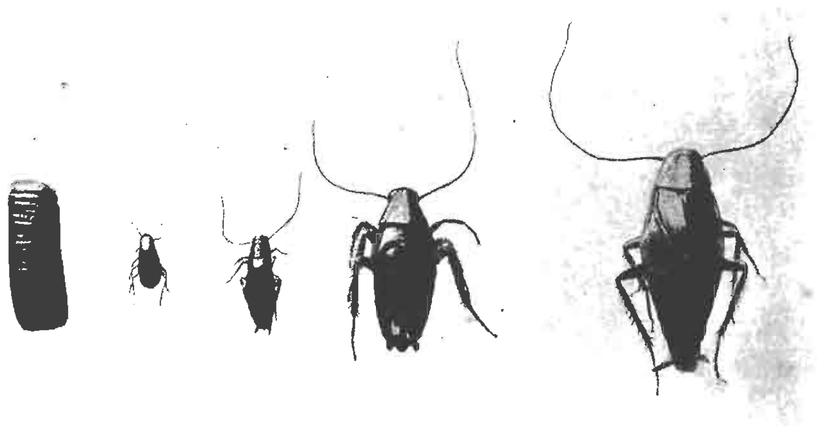


Fig. 31. The German cockroach, *Blatella germanica,* in various stages of development. The adult female is shown with egg case or oötheca in normal position protruding from the terminal abdominal segment. ×2.

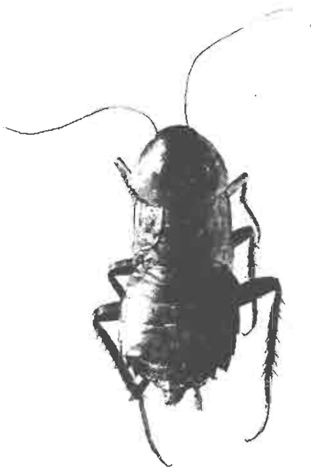
ters are often overrun with cockroaches. Among the better known species invading the household, restaurants, hotel kitchens, etc., particularly in neotropical regions, are described below:

Blatella germanica (Linn.), known as the *German cockroach, water bug,* or croton bug, belongs to the Family Phyllodromiidae. It is the best known and probably the most widely distributed species. It is a small species, native to Europe, measuring from 14 to 16 mm in length, and is pale yellowish-brown in color with two dark brown longitudinal stripes on the pronotum. Both sexes are fully winged. The female carries the egg capsule partly protruding from the tip of the abdomen (Fig. 31) until hatching time.

Blatta orientalis (Linn.), known as the *Oriental cockroach* and also as the *"black beetle,"* is very much darker than the German roach. It is dark brown to black in color, whence its common name "black beetle"

(Fig. 32). It is about 25 mm in length; the wings of the female are rudimentary, and those of the male do not quite reach the tip of the abdomen. Enormous colonies of the Oriental cockroach are often found in damp basements where food is available. This species belongs to the Family Blattidae.

Periplaneta americana (Linn.), commonly called the *American cockroach* (Fig. 33), is a very large (30–40 mm in length) chestnut-brown species, native to Mexico and Central America, but now widely distributed over the earth. Both sexes have long wings, which are frequently used in flying short distances. This species also belongs to the Family Blattidae.



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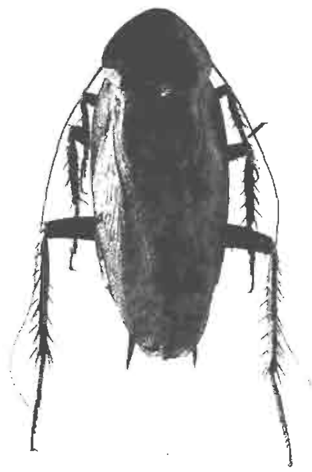

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FIG. 32. The Oriental roach (female), *Blatta orientalis*. ×1.3.
FIG. 33. The American cockroach, *Periplaneta americana*. ×1.3.

Periplaneta australasiae (Fabr.), while commonly called the *Australian roach*, though probably not indigenous to Australia, is, like *americana*, a cosmopolitan species. It is reddish-brown in color resembling *americana*, but has a strong straw-colored streak extending about one-third of the way down the outer margins of the wing covers (tegmina) as well as a yellow area around the margin of the pronotum forming a double dark area on the dorsum. The wings are well developed in both sexes. This species, too, belongs to the Family Blattidae.

Supella supellectilium Serv., known as the *brown-banded* or *tropical cockroach*, resembles the German cockroach in appearance but is smaller (10–12 mm) and has two yellow cross bands, one at the base of the wings and the other about $\frac{1}{16}$ of an inch farther back. The wings do not quite reach the tip of the abdomen; adults fly readily when disturbed. The species is a decidedly gregarious one. Unlike the German cockroach, which confines its activities to the kitchen or around water and heat pipes, the tropical cockroach hides in cupboards and pantries, invades all rooms of the house, and seems to prefer "high locations, such as shelves in closets, behind pictures and picture moldings, and the like." This species, like the German cockroach, belongs to the Family Phyllodromiidae. It is rapidly becoming cosmopolitan.

Leucophaea surinamensis (Linn.), the *dusty-tail roach*, or *Surinam* cockroach, is a dark brown to blackish tropical species measuring from 14 to 20 mm in length. It is a burrowing species, burrowing under piles of debris, leaves, etc. It, too, belongs to the Family Phyllodromiidae.

FIG. 34. Egg cases (oöthecae) of cockroaches: (*left*) Oriental roach, (*right*) croton bug. ×3.

Panchlora cubensis Sauss. is the green Cuban roach which is frequently brought to the ports of many lands in bunches of bananas from Central America.

Tokuichi Shiraki[2] lists 68 species of cockroaches for Japan, many of these newly described.

Life history. The eggs of cockroaches are assembled in leathery bean-shaped capsules, oötheca (Fig. 34), within the body of the female and are thus carried for some time, often weeks, usually partly protruding from her abdomen until just before hatching, when they are dropped. There are several tropical species belonging to the Family Panchloridae which are ovoviviparous, i.e., give birth to living young. The number of eggs within each capsule varies with the species. For *Periplaneta americana* the number is usually 16, and occasionally 18 to 24 (Gould and Deay)[3]; the normal number for *Blatta orientalis* is also 16, while the number per capsule for *Blattella germanica* varies between 30 and 40 with a maximum of 48; and for *Supella supellectilium* the average is 18.

The latter species drops the egg capsule after a day or two and attaches it to some bit of debris. The Oriental roach may do this too. Many egg capsules are produced during the lifetime of the female cockroach, e.g., as many as 90 by the American cockroach, 18 by the Oriental, and but 4 to 6 by the German (Gould and Deay, *loc. cit.*).

The length of the incubation period varies with temperature and humidity. At a constant temperature of about 86° F and a relative humidity of about 70 per cent, Gould and Deay (*loc. cit.*) found the incubation period for the American cockroach to be 31.8 days, at room temperature it averaged 52.9 days. For the Oriental cockroach the same authors found the incubation period at room temperature to vary from 42 to 81 days; for the German cockroach under similar conditions (room temperature averaging 76° F) it was 28.4 days. The eggs of this latter species hatched in 16 days at a temperature of 88° F or higher. The brown-banded roach required 90 days to hatch at room temperature of 73° F and 49 days at 82° F.

On hatching, the young cockroaches are almost white and quite wingless; the skin is cast on emergence, with a second molt usually in three or four weeks, followed by other molts at intervals of several weeks until maturity is reached. The American roach may have as many as 13 molts, with wing pads appearing in the third or fourth molt; maturity being attained in from 285 to 642 days; however, Gould and Deay report as high as 971 days under similar conditions for one specimen. Rau[4] reports the developmental period for the Oriental roach as one year. For the smaller species, such as the German cockroach, the developmental period is much shorter; an average of about two months (50 to 60 days according to Seamans and Woodruff[5]; and 90 to 95 days at room temperature according to Gould and Deay). There are normally six molts, but there may be seven under adverse conditions. This permits the development of two or more generations a year for the German cockroach.

The longevity of the American cockroach is reported by Gould and Deay to range from 102 to 588 days under room conditions, and the *complete* life span of three females of this species is reported to have been 783, 793, and 913 days respectively. The mean length of life of female German cockroaches is reported by these authors to be 200 days, with a maximum of 303 days.

Cockroaches as vectors. Herms and Nelson[6] by means of a simple bacteriological experiment showed that cockroaches (*Blatella germanica*) can acquire specific bacteria by crawling over cultures and then deposit the bacteria on food, e.g., by crawling over sugar. They also showed that these roaches carried a minimum of 13,370 bacteria. There were more bacteria on the hind pair of legs than on the other two pairs of legs, probably due to the fact that the insect's hind legs are more constantly in contact with surfaces and that the forelegs are very frequently brushed clean by means of the antennae. The number of bacteria carried by a fly ranges from 550 to 6,600,000 according to Esten and Mason,[7] hence, potentially at least, the fly is a more effective carrier of microörganisms.

Longfellow[8] has shown that *Escherichia coli* (Migula), *Proteus vulgaris* Hauser, *Staphylococcus aureus* Rosenbach, *S. citreus* (Migula), and a bacillus of the subtilis type are carried on the legs of roaches and that the same organisms are found in the feces, passing uninjured through the alimentary tract. However, the most important results have been obtained by Barber,[9] experimenting with the roach *Periplaneta americana* (Linn.). He fed dejections of cholera patients to roaches and found that a single insect would frequently ingest as much as 0.2 cc. In eight cases cholera vibrios were recovered from the insect's feces and in one case as long as 79 hours after feeding. After the vibrios were discharged by the roach on such moist materials as fresh beef, lettuce, etc., they remained mobile for at least 16 hours, and no loss of virulence was observed when the vibrios remained in the insect's intestine as long as 29 hours. Barber also found that the roaches disgorged portions of their meal at intervals after feeding and that such disgorged material contained cholera vibrios.

Cockroaches as intermediate hosts of nematode parasites. It was very early known that cockroaches may become infected with *Spirura rytipleurites* (Deslgch.) of the rat by feeding on rat feces, and that other rats may become infected in turn by feeding on infected roaches. Galeb[10] (1878) reports that he discovered numerous parasites in the adipose tissue of the roach, *Blatta orientalis* Linn., which were identical with nematodes found in the rat, *Rattus r. norvegicus.* He also observed hair of the rat in the alimentary canal of the roach. On feeding rats (*Rattus r. rattus*) on infected roaches and examining them after the expiration of eight days, he found the parasites in the folds of the mucous membrane of the rats' stomachs. Several nematodes (three females and one male) had already developed sexual organs.

According to Fibiger[12] much evidence is at hand to prove that certain nematode parasites (Spiroptera) of the rat produce true malignant tumors. Eggs of these nematodes were found in the rat feces but no embryos, and he reports that none developed for six months and also that rats could not be infected with the eggs, the roach being a necessary intermediate host. Two species of roaches were used, namely, *Blatta orientalis* Linn. and *Periplaneta americana* (Linn.). Ransom and Hall[11] found that although *Aphodius* beetles are the normal carriers, the croton bug, *Blatella germanica* (Linn.), might also serve as an intermediate host for *Gongylonema pulchrum* Molin, a nematode parasite of sheep and cattle. Referring to the discovery of Fibiger relative to sarcoma in rats cited above and based on his (Sambon's) epidemiological studies on human cancer Sambon (1925, *loc. cit.*, Chapter IV) concluded:

> Similar to the role of the filaria in elephantiasis is, no doubt, that of gongylonema in cancer. Like the filaria, so the gongylonema is not the direct essential cause of the disease it gives rise to, but it probably is, in certain localities, an etiological factor of considerable importance. The gongylonema, known to be a cancer-producing agent in the rat, seems an equally likely factor in the incidence of cancer among human beings in certain regions, such as the Romagna and the Trentino. Here the disease affects principally the upper portion of the alimentary tract and more especially the buccal cavity, the tongue, the oesophagus, the larynx, and the cardia (situations invaded by these forms) and here cockroaches, meal beetles, and cellar beetles (certain beetles such as *Tenebrio* and *Blaps* of the family Tenebrionidae are suitable intermediary hosts of these worms) are an ever present vermin in the old houses in which such cancer cases occur. These observations so clearly designative of a relationship between worm and neoplasm, are strengthened by the fact that gongylonema is present and common in the majority of the domestic animals of these regions.

Although Sambon dwells particularly on Gongylonema he points out that other helminths bear a similar relationship, each according to the organ which is usually invaded and in which irritation is set up, thus inviting the cancer factor. It is pointed out that cockroaches, either intact or in fragments, are only too frequently found in bread and other food which may be insufficiently baked to kill the encysted Gongylonema. Cockroaches appear to lend themselves to many species of parasitic nematode worms (see Chitwood[18]).

Cockroach control. Cockroach infestations are generally an indication of a breakdown in good housekeeping practices, whether in the home, a restaurant, a hotel, or a grocery store. There are many ways in which these insects (in any stage of development) may be brought in to start an infestation, e.g., in boxes of groceries, in sacks of produce from warehouses, in freight shipments. They may come in under their own power at night from neighboring foci; they may follow water pipes, sewers, etc. Many homes and nearly all restaurants, grocery stores, etc., have a few cockroaches now and then. Kill the individual intruders at once in any convenient way; do not let them gain a foothold by providing them with food, because of poor garbage disposal or other poor housekeeping practices. See to it that there are no convenient hiding places in cracks in walls, etc.; use plastic wood to fill up such cracks. Dark, moist, cluttered-up situations favor cockroaches; open, clean and neatly kept spaces, permitting plenty of daylight are not inviting to these darkness-loving pests.

The abatement of infestations generally calls for the application of an appropriate insecticide. The use of *sodium fluoride* is one of the most effective methods for the control of roaches, of all species. It is, however, toxic to man and other animals and hence must be used with caution. As an insecticide it is usually given a bluish color so that it will not be confused with flour, soda, etc. *Do not store this insecticide in the kitchen or pantry, and do not dust sodium fluoride in cupboards or where food can be contaminated with it. The container should be labeled "Poison."* For use against cockroaches a mixture is made of sodium fluoride (25 per cent by volume) and tested pyrethrum powder (75 per cent). This mixture is applied by means of a dust gun to all cracks and crevices and places where cockroaches may be in hiding or where they congregate. Applying the powder in the evening is advised, and the application should be repeated in a week or two. The insects come in contact with the dust when running over the treated surfaces, and afterward they clean themselves and swallow the particles adhering to their feet and bodies; thus the sodium fluoride acts as a stomach poison. Various other materials may be used as substitutes for this insecticide, such as finely powdered *borax* and *boric acid,* which may be dusted safely in cupboards and kitchens. This powder is effective only as long as it is dry; it kills very slowly.

DDT is useful in the control of all the common house-invading cockroaches, but *Blatella germanica,* the croton bug, requires somewhat special treatment. Using the same methods of application as for sodium fluoride, force a 10 per cent DDT powder (in 90 per cent powdered pyrophyllite) into cracks and crevices and over surfaces where cockroaches crawl. Against the German roach precede the dusting with a 5 per cent DDT-xylene spray. Apply a 5 per cent oil solution of DDT spray to the undersides of shelves, sinks, drawers, and tables. *Do not allow DDT to fall on dishes, food, or cooking utensils.* Outside areas, dooryards, the vicinity of garbage cans, and other places where cockroaches lurk should be dusted with DDT.

Chlordane (empirical formula, $C_{10}H_6Cl_8$) ranks high as a cockroach poison when used as a 2 to 5 per cent dust. Its "knock-down" powers, in common with some other newer insecticides is low, hence formulations should contain pyrethrum or one of the thiocyanates. In oil formulations used as sprays, 2 per cent chlordane is recommended. Precautionary measures with reference to food, dishes, cooking utensils, etc., must be exercised as for DDT.

B. BEETLES

Order Coleoptera

The Order Coleoptera consists of over 250,000 described species of beetles, comprising 40 per cent of all known members of the Class Insecta. Very few of the families of beetles concern the medical entomologist directly, but because of their abundance and successful invasion of all sorts of environments it is certain that contact will be made with members of this Order of insects sooner or later. Familiarity with the Order is important.

Characteristics. Beetles are readily distinguishable from all other insects. Their integument is leathery; their mouth parts are strongly mandibulate, i.e., biting-chewing. Although wings may be absent in some species, there is usually a pair of wings present; the forward pair, elytra, not used in flight are horny, and when at rest meet in a straight line down the dorsum; the hind wings are membranous and functional, often folding both horizontally and vertically. Metamorphosis is complex (egg, larva, pupa, imago), with hypermetamorphosis in certain species. The larvae are commonly known as "grubs" and may usually be recognized by the presence of three pairs of fairly well-developed legs; however, the larvae of weevils are apodous.

Scavenger beetles. All scavenger beetles, of which there are several families, are potentially of some public health importance since their habit of feeding as larvae or adults on dead animals, hides, or other animal matter may accidentally bring them in contact with pathogenic organisms. They may carry infection in at least two ways, either mechanically on their legs, mouth parts, or body, or in their excreta after feeding on infectious material. Destruction of carcasses by incineration is urged.

Nuttall (*loc. cit.*, p. 4) reports that, as early as 1894, "Proust in examining goatskins taken from anthracic animals, found quantities of living *Dermestes vulpinus* Fabr. upon them. He found virulent anthrax bacilli in their excrements, as also in the eggs and in the larvae. It is evident from this that these insects which feed on the skins permit the anthrax spores to pass uninjured through their alimentary tract. Heim (1894) also had occasion to examine some skins which were suspected of having caused anthrax in three persons engaged in handling the leather. He found larvae of *Attagenus pellio* Linn., *Anthrenus museorum* Linn. (both Dermestidae), and *Ptinus;* also fully developed insects of the latter species on the skins. All these insects had virulent anthrax bacilli (spores) on their surface and in their excreta, from which Heim concludes they might spread disease. He says the excreta are very light and easily scattered by the slightest current of air. Heim does not believe the bacilli multiply in the bodies of these insects, but that the latter may be dangerous through their scattering the spores about."

Among the families of scavenger beetles are the following:

1. *Family Staphylinidae,* commonly known as rove beetles, includes many species that feed on carrion, dung, and decaying animal matter; they are

characterized by the abbreviated condition of the wing covers (elytra), which leaves much of the abdomen exposed and gives these beetles a larval or worm-like appearance; the abdomen is flexible and is often curled up and thrown forward dorsally; the long functional wings are folded up and concealed beneath the short wing covers when not in use. The scavenger species are commonly seen on turning over carrion, hides, heaps of bones, etc. Among these are members of two representative genera, *Creophilus* and *Staphylinus* (Fig. 35).

2. *Family Silphidae,* commonly known as carrion beetles, burying beetles, or sexton beetles, are as a rule attracted to dead animals which they undermine and, in the case of small ones, may even bury. They deposit their eggs on the dead animals, and the larvae feed on the decomposing flesh. Two genera, *Silpha* and *Necrophorus* (Fig. 36) illustrate the family.

35A 35B 36A 36B

FIG. 35. Rove beetles (Staphylinidae); (*A*) *Creophilus,* (*B*) *Staphylinus.* ×1.5.

FIG. 36. Sexton beetles (Silphidae); (*A*) *Silpha americana,* (*B*) *Necrophorus* sp. ×1.5.

3. *Family Dermestidae,* hide beetles, larder beetles, museum pests, dermestids, are small oval or elongate beetles, often mottled, grayish, or brownish in color. The hairy larvae as well as the adults feed on dead animals, museum specimens, wool, cured meats, cheese, and many other animal as well as vegetable products (cereals). *Dermestes lardarius* Linn. is known as the larder beetle; *Dermestes vulpinus* Fabr. is commonly used to clean dried flesh from bones for museum use; *Anthrenus scrophulariae* (Linn.) is known as the carpet beetle, a serious household pest. Larvae of the carpet beetle may enter the ears of persons and cause much discomfort (Fig. 37).

Beetles as intermediate hosts of helminths. Many species of beetles serve as intermediate hosts of helminthic parasites of man and of wild and domesticated animals. This common relationship is no doubt due to the variety of feeding habits of beetles which enables them to ingest fecal matter in which eggs of intestinal parasites of animals commonly occur; thus many cereal and omnivorous feeders, as well as coprophagous beetles may readily lend themselves as intermediate hosts. As suggested previously (see cockroaches) the infective stage of nematode worms of the genus *Gongylonema* belonging to the family Spiruridae commonly occur in dung beetles belonging to the beetle family Scarabaeidae, such as members of the genus *Aphodius;* also in meal worms, belonging to the family Tenebrionidae such as *Tenebrio molitor* Linn. *Gongylonema pulchrum* Molin parasitizes many species of mammals, such as goats, swine, and occasionally man. It invades tissues of the oral cavity and esophagus, causing *gongylonemiasis.* Adult females measure about 145 mm in length and are very slender. Eggs are evacuated in feces and lie dormant until ingested by an appropriate insect in whose gut the larva hatches and comes to lie encapsulated in the body cavity until the insect is ingested by an appropriate vertebrate host.

May beetles or cockchafers (family Scarabaeidae) are known to be intermediate hosts both in the larval and adult stages of the thorn-headed worm, *Macracanthorhynchus hirudinaceus* (Pallas) [= *Gigantorhynchus gigas* (Bloch)], a parasite of swine also said to occur in man in rare instances. This nematode worm in its adult stage measures from 20 to 30 cm in length and about 3 to 5 mm in thickness, and inhabits the small intestine of its host. The eggs are deposited in the intestine and pass out with the feces, which may be swallowed by larvae of cockchafers. These are often extremely abundant among the rootlets of grass in heavily sodded pastures, and swine with free range are fond of these grubs, in search of which they diligently root up the soil with their snouts. Thus every opportunity is given for the grubs to become infected and in turn the swine.

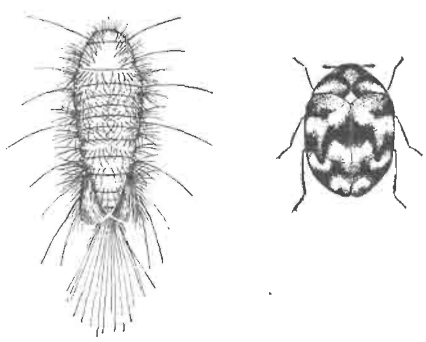


FIG. 37. The varied carpet beetle, *Anthrenus verbasci;* showing larva and adult.

After the ova have been ingested the larvae hatch in a few days within the intestine of the insect and proceed to burrow through the intestinal wall and into the muscles, where they are said to encyst themselves. In Europe the intermediate insect host is commonly *Melolontha melolontha* (Linn.) or *Cetonia aurata* (Linn.). May beetles of the genus *Lachnosterna* (according to Stiles, *Lachnosterna arcuata* Smith, and others) are probably all more or less concerned. The life of nearly all May beetles is quite long, the larval stage alone often requiring nearly three years. In districts infested with the thorn-headed worm a systematic crusade against cockchafers would be the logical means of control, together with the treatment of swine.

Numerous species of beetles have been proved to be intermediate hosts of the fowl tapeworm, *Raillietina cesticillus* (Molin). The species listed by Reid, Ackert, and Case[14] belong to the following families, Scarabaeidae, two species; Tenebrionidae, one species; Carabidae, subfamily Harpalinae, 26 species, to which they added 12 not previously reported, giving a total of 38 species in this family. The beetles belonging to the genus *Amara* proved to be particularly favorable hosts although the largest number of cysticercoids were produced by a species of *Pterostichus*, a total of 626 by one beetle which had been fed on four proglottids.

The adult tapeworms, which measure from 10 to 12 cm in length, are generally attached to the lumen of the upper third of the chicken intestine. The terminal gravid proglottids which break off pass from the body of the host with the feces and continue to be motile for a short time after evacuation. If the proglottids are eaten by appropriate species of beetles the hexacanth embryos develop, penetrate the intestinal wall of the host, and become mature cysticercoids in the body cavity of the beetle in from 14 to 21 days or longer, usually in 14 to 16 days during the hot part of the summer. If the beetles are now eaten by chickens the mature ovoid yellowish-brown cysticercoids, which measure from 363μ to 521μ in length by 199μ to 398μ in width, according to the abovementioned investigators, are freed in the duodenum of the birds and in about two weeks adult tapeworms are produced. The onchosphere stage of *R. cesticillus* can be distinguished from onchospheres of other species of fowl tapeworms by two funnel-like structures in the membranes which surround the hexacanth embryo, (Reid, Ackert, and Case).

Canthariasis is a term used to designate the rare accidental beetle (larva or adult) infestations of organs of the body, e.g., infestations of the alimentary canal by larvae of the churchyard beetle, *Blaps mortisaga* Linn., as the result of superstitiously drinking foul graveyard water in which these beetles may occur. The ingestion of meal- and flour-infesting insects such as the meal worm, *Tenebrio molitor* Linn., has also been known to occur.

Vesicating beetles. Many beetles possess a vesicating substance in their body tissues which produces blisters when coming in contact with the skin. The family Meloidae, commonly referred to as the "blister beetles," contains the species which are the important source of cantharidin used medicinally. Among these species are the Spanish fly, *Lytta vesicatoria* (Linn.) the telini fly, *Mylabris cichorii* (Linn.), and *Epicauta hirticornis* (Haag-Rutenberg) of India. At least two species of the family *Oedemeridae, Sessinia collaris* (Sharp) and S. *decolor* Fairm., known as coconut beetles by the Gilbertese, cause severe blistering if the skin is contacted. (See Chapter XXII.)

Rose chafers poisonous to poultry. The rose chafer, *Macrodactylus subspinosus* (Fab.), family Scarabaeidae, was found by Lamson[15] to be poisonous to chicken, ducklings, goslings, and young turkeys. He reported that chickens fed in quantity on rose chafers showed the following symptoms after from four to five hours: "Drowsy appearance, with wings dropping, the eyes closed, and a slight shaking of the body. This drowsiness increased, leg weakness developed, until the chickens no longer stood, but slept resting on their feet and legs, and later died. Death occurred usually from five to twenty-four hours after they had fed upon the rose chafers. Convulsions occurred in about five per cent of the deaths. Chickens that survived the twenty-four-hour period after eating rose chafers seldom died from the poison, though they did not seem altogether normal for several days afterward."

Sundry annoying beetles. Aside from any economic or other injury for which beetles may be responsible, the sheer abundance of certain species at times causes annoyance. For example, while trying to do microscopic work or write reports by lamp light the author, stripped to the waist during warm evenings in the tropics, was greatly disturbed by the presence of hordes of the so-called copra "bug" (red-legged ham beetle), *Necrobia rufipes* Fabr., family *Corynetidae*.[16] These small greenish-blue beetles, 3-5 mm long, originated in vast numbers in copra stored in neighboring sheds. The larvae of this species may also enter the ears of persons.

Tiny (3 mm long) saw-toothed grain beetles, *Oryzoephilus surinamensis* (Linn.), belonging to the family *Cucujidae*, may invade bed chambers in great numbers and crawl over the bodies of occupants and nibble the skin. An infestation of this kind was traced to the bathroom and thence out of the house through the yard and into an old barn where, under the stalls, grain from the manger had collected, affording a breeding ground for the beetles. Extreme dryness had apparently driven the insects to the bathroom for moisture and the attack on occupants of the adjoining bedchamber was merely incidental.

Minute species of *Staphylinidae* (such as *Atheta occidentalis*) Bernhaeur, a blackish species 3 mm long, are often encountered on the wing in the late autumn and may accidentally enter the eyes, causing a severe burning sensation, and temporarily blinding the victim. Such a mishap to a person driving a motor car might lead to a serious accident. These minute species breed in cow dung and decomposing plant refuse.

The so-called tule beetle, or stink beetle [*Platynus maculicollis* (Dej.)], of California is normally a beneficial predator, but when its natural habitat in the marshes becomes dry in summer it commonly leaves in search of moisture and may invade homes in the neighborhood. Heavy early winter rains and cold weather may likewise cause invasions. The nauseous odor of this beetle is almost intolerable. It belongs to the family Carabidae which contains many other species possessing vile odors.

Beetles as parasites. Belonging to the family Platypsyllidae, indeed the only representative of this family, is the coleopterous parasite of the beaver, *Platypsyllus castoris* Ritsema (1869). This is a permanent, obligate parasite in all its stages. The eggs are deposited on the skin of the beaver among dense hairs. It occurs on these animals in both Europe and North America.

Another family of Coleoptera, the Leptinidae, includes beetles which are parasitic on beavers, and certain other rodents. The three known species are *Leptinus testaceus* Muller, parasitic on mice and shrews in Europe and North America; *Leptinillus validus* (Horn) found on North American beavers; and *Leptinillus aplodontiae* Ferris, taken on Aplodontia, a genus of rodents known as mountain beavers, peculiar to the Pacific coast (Ferris).[17]

*Key to the Families of Coleoptera Referred to in this Chapter**

1. Antennae variable in structure but never lamellate 2
Antennae with a club composed of from three to seven leaf-like segments (lamellae); legs usually fossorial; tarsi five-segmented. (May beetles, June beetles, Cockchafers) .... *Scarabaeidae*
2. Anterior and intermediate tarsi five-segmented; posterior tarsi four-segmented 3
All of the tarsi either three-, four-, or five-segmented 4
3. Head as wide as prothorax; anterior coxal cavities open behind; body not heavily chitinized (Blister beetles) .... *Meloidae*
Head narrower than prothorax; anterior coxal cavities closed behind; body usually heavily chitinized (Darkling ground beetles) ....
.... *Tenebrionidae*
4. Elytra long, covering most of abdomen; abdominal tergites mostly membranous 5
Elytra short, exposing most of abdomen; abdominal tergites mostly chitinous 8
5. Fourth segment of tarsus distinct, free; antennae clavate, capitate or moniliform 6
Fourth segment of tarsus very small, fused with fifth segment; antennae usually filiform; body often brightly colored (Leaf beetles) ....
.... *Chrysomelidae*

* Prepared by Dr. E. Gorton Linsley.

6. Abdomen with five visible sternites 7
   Abdomen with six visible sternites; tibial spurs large; antennae gradually thickened or clavate (Carrion beetles, Burying beetles) .... *Silphidae*
7. Anterior coxae conical, prominent; antennae moniliform; flat, nonscaly beetles (Flat beetles, Grain beetles) .... *Cucujidae*
   Anterior coxae globular, not prominent; antennae capitate or clavate; convex, scaly beetles (Leather beetles, skin beetles) .... *Dermestidae*
8. Wingless; eyes wanting or abortive 9
   Winged; the wings folding under the short elytra; eyes usually well developed; tarsi from three- to five-segmented .... *Staphylinidae*
9. Anterior coxae globular (Beaver beetles) .... *Platypsyllidae*
   Anterior coxae flat (Rodent beetles) .... *Leptinidae*

BIBLIOGRAPHY

1. Cockerell, T. D. A., 1927. "The carboniferous insects of Maryland," *Ann. & Magazine of Nat. History*, 19:385.

2. Shiraki, Tokuichi, 1931. "Orthoptera of the Japanese Empire. Part II Blattidae, Insects Matsumurana," Taihoku Imperial Univ. Entomolog. Lab. Contrib. no. 29, 5 (Part 4):171–209.

3. Gould, George E., and Deay, H. O., 1940. "The biology of six species of cockroaches which inhabit buildings." Lafayette: Purdue Univ., in Agric. Exper. Sta., Bull. no. 451, 31 pp.

4. Rau, P., 1924. "Biology of the roach," *Tr. Acad. Sc. St. Louis,* 25:57–79.

5. Seamans, Lois, and Woodruff, Laurence C., 1939. "Some factors influencing the number of molts of the German roach," *J. Kansas Entomolog. Soc.*, 12:73–76.

6. Herms, W. B., and Nelson, Y., 1913. "The croton bug *(Ectobia germanica)* as a factor in bacterial dissemination," *Am. J. Pub. Health,* 3:929–34.

7. Esten, W. N., and Mason, C. J., 1908. "Sources of bacteria in milk," *Storrs Agric. Exper. Sta., Bull.* no. 51, pp. 94–98.

8. Longfellow, R. C., 1913. The common house roach as a carrier of disease. *Am. J. Pub. Health,* 3:58–61.

9. Barber, M. A., 1914. "Cockroaches and ants as carriers of the vibrios of Asiatic cholera," *Philippine J. Sc.*, sec. B, 9:1–4.

10. Galeb, Osman, 1878. "Observations et expériences sur les migrations du *Filaria rytipleurites,* parasite des blattes et des rats," *Compt. rend. Acad. d. sc.*, 87:75–77.

11. Ransom, B. H., and Hall, M. C., 1915. "The life history of *Gongylonema scutatum*," *J. Parasitol.*, 2:80–86.

12. Fibiger, Johannes, 1913. "Ueber eine durch Nematoden (Spiroptera sp. n.) hervorgerufene papillomatöse und carcinomatöse Geschwulstbildung im magen der Ratte," *Berl. klin. Wchnschr.*, 50:289–98.

13. Chitwood, B. G., 1932. "A synposis of the nematodes parasitic in insects of the family Blattidae," *Ztschr. fpkarasitenkunde,* 5:14–50.

14. Reid, W. M.; Ackert, J. E.; and Case, A. A; 1938. "Studies on the life history and biology of the fowl tapeworm, *Raillietina cesticillus* (Molin)," *Tr. Am. Micr. Soc.*, 57:65–76.

15. Lamson, G. H., Jr., 1922. "The rose chafer as a cause of death of chickens." *Storrs Agric. Exper. Sta. Bull.* no. 110, pp. 117–34.

16. Herms, William B., 1926. "Diocalandra taitensis (Guerin) and other coconut pests of Fanning and Washington Islands," *Philippine J. Sc.*, 30:243–71.

17. Ferris, G. F., 1918. "An apparently new species of Leptinillus," *Canad. Entomologist*, 50:125–28.

CHAPTER VIII

THE BUGS

BEDBUGS, CONENOSES, AND OTHER BUGS

Order Hemiptera

Order Hemiptera. The Hemiptera comprise all the insects, about 48,000 species, included in the Heteroptera and the Homoptera. The order Hemiptera is divided into two suborders: (1) *Heteroptera,* in which the fore pair of wings (hemelytra) is usually thickened at the base and the distal overlapping portion is membranous, and (2) *Homoptera,* in which the fore pair of wings is usually of about the same thickness (membranous) throughout.

The suborder Homoptera includes such important phytophagous families as the Aphididae (plant lice), Cicadidae (cicadas or harvest flies), Cicadellidae (leafhoppers, sharpshooters), Membracidae (tree-hoppers) and many others of great agricultural importance, particularly many important vectors of plant diseases. These families include plant-feeding insects with piercing mouth parts, such as leafhoppers and tree-hoppers, many of which have been reported as biting and sucking blood from human beings. Usinger[1] attributes this rather rare phenomenon of bloodsucking in the normally phytophagous groups of the Hemiptera to three influences, namely, the "stimulus of artificial light or other unusual conditions of the environment, the attractive qualities of exposed liquids, mainly perspiration, and hunger." He remarks further that this change "from plant feeding to bloodsucking, is not such a profound one as would at first be supposed. This is evidenced by a comparison of the composition of plant juices and blood and by the various plant-feeding groups, some members of which have adapted themselves to a predaceous habit or have shown their ability occasionally to suck the blood of mammals."

The Hemiptera-Heteroptera are the true bugs and are characterized by a jointed suctorial proboscis attached anteriorly, which, when not in use, is commonly flexed under the head. The winged members of this order normally have the wing covers, hemelytra (fore pair of wings), thickened at the base and the distal overlapping portion more or less membranous. The true bugs are separated into two divisions, (1) the

Gymnocerata, in which the antennae are conspicuous and capable of being moved freely in front of the head, e.g., *Cimex lectularius* Linn., the common bedbug; *Anasa tristis* (DeGeer), the squash bug; and *Triatoma protracta* (Uhler), a conenose; and (2) the **Cryptocerata,** in which the antennae are concealed in small concavities (foveae) and are closely pressed to the under side of the head, e.g., *Lethocerus americanus* (Leidy), the giant water bug. Metamorphosis is simple.

A. THE BEDBUGS

Family Cimicidae

Family Cimicidae. The family Cimicidae (Acanthiidae) includes the bedbugs, swallow bugs, and the poultry bug, characterized by a very short, broad head, broadly attached to the prothorax, an oval body, well-developed compound eyes, absence of ocelli, four-jointed conspicuous antennae, three jointed proboscis lying in a groove beneath the head and thorax, very short pad-like hemelytra. The bodies are broad and flat, enabling the bugs to creep into narrow crevices. A nasty pungent odor is attached to the group as a whole with few exceptions. They are night-prowling and bloodsucking in habit, some feeding on birds and bats and others on human beings. Peculiar to these bugs is the organ of Berlese located in the IV or V abdominal segments. The presence or absence of this organ and its particular location when present provides a character useful in identification of species.

The three principal genera of the family Cimicidae[2] are: (1) *Cimex*, e.g., *C. lectularius L.*, the cosmopolitan bedbug; *C. hemipterus* (Fabr.) [*C. rotundatus* (Sign.)], tropical and subtropical, also known as the Indian bedbug; C. *boueti* Joyeux,[3] the tropical bedbug of Africa and South America, all of which resemble each other very closely as do the following: *C. columbarius* Jenyns, parasitic on pigeons in Europe, *C. pipistrelli* Jenyns, parasitic on bats in Europe, and *C. pilosellus* (Horv.) parasitic on bats in North America; (2) *Œciacus*, in which the body is clothed with long silky hairs, the filiform third and fourth joints of the antennae only a little thinner than the first and second, e.g., *Œciacus hirundinus* (Jenyns), the European barn-swallow bug and *O. vicarius* Horv., the corresponding American species; (3) *Haematosiphon*, in which the rostrum is long, reaching to the posterior coxae, of which *Haematosiphon inodora* (Duges) is the only known species. This species infests poultry in the southwestern United States and in Mexico and is often a serious household pest. Its native hosts are the California condor and great horned owl (Usinger[4]). Although there are less than 40 described species in the family, a more intensive study will no doubt result in the discovery of additional species and changes in genera, as suggested by the work of List.[5]

The common bedbug. The adult of the common bedbug, *Cimex lectularius* Linn. (Fig. 38), measures from 4 to 5 mm in length and 3 mm in breadth, it is obovate in form and much flattened. The adult is reddish brown in color, whereas the young are yellowish white. Among the local names applied to bedbugs are "chinches," "chintzes," "red coats," "mahogany flats," "wall louse," "bedbugs," or simply "bugs."

Bedbugs, like lice, have been the constant companions of man for centuries; the earliest writings on natural history (Pliny and Aristotle) mention them. Bedbugs occasionally gain a foothold among laboratory animals such as white rats and guinea pigs, upon which they feed readily. They are nocturnal in their feeding habits, hiding in crevices during the

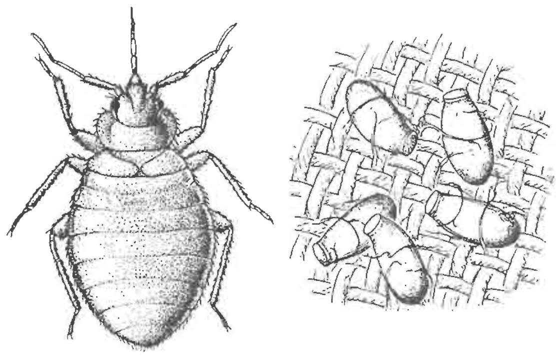


Fig. 38. The common bedbug, *Cimex lectularius*. Eggs shown at right.

day. At night they are very active, crawling out of their hiding places, often traveling considerable distances to attack their victims. This is especially true where iron bedsteads are used, as these do not provide convenient hiding places for the bugs. Ordinarily where the old-fashioned wooden bedsteads are used the bugs stay closer to their point of attack. Mattresses in any case may afford harborage. Bedbugs are gregarious, hence often great assemblages may be found in some convenient crevice or beneath some nearby loose wallpaper, where the eggs are deposited and the tarry black excrement collects.

The females deposit eggs in batches of from 10 to 50, totaling from 200 to 500, spread out in a yellowish patch. The eggs are large and yellowish white in color. Oviposition occurs at intervals during a period of from two to three months, apparently limited to the spring and summer months, notwithstanding the fact that the insects are commonly favored by warm rooms during the winter. The young hatch in from 7 to 30 days (usually about ten days), according to temperature, which also affects their later growth. The time required for development from egg to maturity is given as from 45 days to 11 months, although the time is greatly influenced by temperature; there may be three or more generations a year under average condition. Ordinarily they require six to eight weeks to reach maturity. Bedbugs are evidently sensitive to high temperatures; even a temperature of 100° F with fairly high humidity will kill many of them. Activity ceases at about 60° F. At 60° F to 65° F bedbugs have lived for 136 days without food. Normally fed individuals may survive from 54 to 316 days under ordinary room-temperature conditions. The presence or absence of food influences longevity; without food adults may survive from 17 to 42 days (Marlatt[6]). Bedbugs molt five times, and the minute wing pads characteristic of the adult insect make their appearance with the last molt. Ordinarily but one meal is taken between each molt and one before egg deposition; an average period of eight days is required between moltings. Metamorphosis is simple.

Methods of distribution. Bedbugs, lice, or other organisms cannot originate spontaneously in filth as is believed by many; infestations are traceable to introduced eggs, young, or adults. Thus the introduction of one impregnated female might furnish the nucleus for a well-developed colony in a few months. Hence the best-regulated household is not exempt from invasion, though cleanliness is the best preventive against the multiplication of any household pest.

Public conveyances are commonly means for the dissemination of bedbugs. Furthermore, migration from house to house by way of water pipes, walls, and the like is not at all unlikely when infested houses are vacated and the food supply is cut off. The insects are also easily carried in clothing, traveling bags, suit cases, etc.

Bedbug bites. Persons "bitten" by bedbugs respond differently; in some the bite produces marked swellings and considerable irritation, while in others not the slightest inconvenience is caused. (This is true also in the case of flea bites and mosquito bites.) The bite, so-called, of the bedbug is produced by piercing organs of the hemipteron type already described. It is probable that puncture by these stylets, unattended by contamination or specific poisons, would produce little pain. The welts and local inflammation are unquestionably caused by a specific poison secreted by the salivary glands of the insect and introduced in the act of feeding. The bedbug is able to engorge itself completely with blood in from three to five minutes. Although persons are usually bitten at night while in bed because of the normal nocturnal habits of the bedbugs, the insects will bite freely in subdued light by day.

The fact that bedbugs are obliged to feed at least five times either upon the same or a different host in order to reach maturity, has placed these insects under grave suspicion as potential vectors of disease.

Disease transmission. In consequence of statements made by various authors that bedbugs are capable of transmitting plague and other septicemic infections, Nuttall (1899, *loc. cit.*, p. 4) carried on a series of experiments with these insects. Mice were used as the experimental animals because they are very susceptible to the infections in question. He allowed the bugs to bite mice which had just died or were dying of anthrax, plague, and mouse septicemia and then transferred them to healthy mice. His experiments with anthrax are particularly instructive. Mice inoculated with anthrax died in from 18 to 24 hours, after which they were placed in glass-covered dishes and hungry bugs were introduced. As soon as the bugs had sucked a little blood they were removed to test tubes by means of a small camel's hair brush and transferred to a shaved spot on healthy mice by inverting the tubes. Eight mice bitten by 124 infected bugs all remained healthy. Variations of this experiment gave similar results. It was found that the anthrax bacilli died in the stomach of the insect in 48 to 96 hours at 13° to 17° C, and in 24 to 28 hours at 37°, and that the feces from the bugs contained living bacilli during the first 24 hours after feeding. In view of these experiments it may be concluded that infection through the *bite* of a bedbug either does not occur or is exceptional. Infection might occur if recently infected bugs were crushed while feeding and the punctured parts were thereupon scratched. The feces of such bugs may also be infectious. Nuttall[7] showed that spirochetes survive in the bodies of these bugs for a period of six days at a temperature of 12° C and for a much shorter period (six hours) at 20° to 24° C. He did succeed in transmitting the infection to a mouse, in one instance, by transferring 35 bugs from an infected mouse to an uninfected mouse. Patton in 1907[8] reported experiments with *Cimex hemipterus* (Fabr.) [= *C. rotundatus* (Sign.)] in which he was able to trace the development of the Leishman-Donovan body of kala-azar through all its intermediate stages up to the formation of the mature flagellates. Francis and Lake[9] reported transmission of tularemia to mice by means of *Cimex lectularius;* however, Davis[10] points out that during the hour-long interrupted feeding the tails of the mice became soiled with bug feces and that in his tests bedbugs failed to transmit *P. tularense* when a "clean" method of feeding was used, i.e., when fecal contamination of the host was eliminated.

The bedbug would appear to be relatively unimportant as a disease vector, as indicated by the experiments cited above as well as many others performed by various investigators. In spite of the fact that bedbugs can experimentally transmit the pathogenic agents of plague, relapsing fever, leprosy, kala-azar, and tularemia (?), there is no convincing evidence that the bedbug is a vector of these or any other diseases at present known. It is of course possible, as shown in laboratory tests, that occasional transmissions may be effected.

Bedbug control. With the advent of "DDT" the older methods of bedbug control have been largely discarded; however, the use of hydrocyanic acid gas for house fumigation in some types of pest-control operations will continue to be a sound practice in the hands of professional operators.[11] Where live steam under pressure is available this may be employed with good results in bunkhouses and the like.

DDT in bedbug control. A bedbug infestation in dwellings, barracks, etc., can be successfully eradicated by the thorough application of a 5 per cent DDT spray applied to beds, mattresses, wall crevices, and other infested situations. A single correct treatment will "bug proof" for six months or longer. A 5 per cent DDT spray (xylene-Triton X-100) is recommended; it is applied as a spray until misty-wet to all parts of mattresses (standing on edge), bedsprings, and bedsteads. In heavy infestations the walls around the beds, particularly cracks along baseboards, surfaces back of loose wall paper, and all other harborages must also be sprayed. Chicken coops infested with the chicken bedbug can also be freed of these pests by using DDT spray. Not only should food be protected against the spray, but *spraying must not be done near an open flame.*

*Key to North American Cimicidae**

1. Head longer than broad, clypeus narrowed anteriorly. Labrum much longer than broad. Legs very long, the femora extending beyond sides of body for a distance greater than width of pronotum. Organ of Berlese absent. *Primicimex cavernis* Barber
   Head broader than long, clypeus broadened anteriorly. Labrum short, about as broad as long. Legs relatively short; the femora not much longer than width of pronotum and only briefly projecting beyond sides of body. Organ of Berlese present 2
2. Middle and hind coxae subcontiguous, the metasternum compressed. Large bristles of body dentate only at their tips. Organ of Berlese dorsal 3
   Middle and hind coxae rather widely separated, the metasternum broad and plate-like, widening posteriorly. Large bristles of body dentate on convex side. Organ of Berlese ventral 6
3. Rostrum long, reaching hind margin of mesosternum. Posterior abdominal tergites in female strongly sinuate, organ of Berlese located at middle of fifth segment. New Mexico, Texas, Oklahoma, California, Mexico. Hosts: California condor, Great horned owl, domestic chicken. *Haematosiphon inodora* Duges.
   Rostrum shorter, not reaching mesosternum. Posterior abdominal sutures only feebly sinuate 4
4. Pronotum strongly narrowed posteriorly. Posteroventral portion of head and adjoining prosternum strongly convex. California, Nebraska. Host: White-throated swift .... *Synxenoderus comosus* List
   Pronotum not strongly narrowed posteriorly. Head beneath and prosternum not strongly convex 5

* Prepared by Dr. Robert L. Usinger.

5. Body clothed with long bristles, sides of abdomen with some bristles twice as long as width of an eye. Colorado, Mexico. Hosts: Graybreasted martin and other swallows .... *Hesperocimex coloradensis* List
Body with very short, inconspicuous bristles, those of sides of abdominal segments much shorter than width of an eye. Eastern and central United States. Host: Chimney swift........*Cimexopsis nyctalis* List
6. Pubescence long and sericeous. Last two antennal segments subequal in length..........*Oeciacus vicarius* Horvath
Pubescence short, stiff. Third antennal segment longer than fourth. Genus *Cimex* Linn.......... 7
7. Sides of pronotum not widely dilated and not reflexed, fringed with sparse, nearly straight hairs. Hemelytra with apical margins distinctly rounded. Tropicopolitan. Host: Man. *Cimex hemipterus* (Fabr.)
Sides of pronotum widely dilated, broader than width of an eye, and densely fringed with backward-curved hairs. Apical margins of hemelytra nearly straight, rounded towards inner anngles 8
8. Length of contiguous portions of hemelytra shorter than scutellum. Second antennal segment slightly shorter than third. Fringing hairs of pronotal margin shorter than width of an eye. Widely distributed in temperate regions. Host: Man..........*Cimex lectularius* Linn.
Length of contiguous portions of hemelytra longer than scutellum. Second and third antennal segments equal in length. Fringing hairs of pronotum longer than width of an eye.......... 9
9. Marginal hairs relatively long. Tergum strigate. Eastern North America. Host: Bats.......... *Cimex adjunuctus* Barber
Marginal hairs relatively short. Tergum not transversely strigate. Western North America. Host: Bats..........*Cimex pilosellus* Horvath

B. THE CONENOSES

Family Reduviidae

Family Reduviidae. The Reduviidae are typical examples of the heteropterous Hemiptera. They are commonly known as conenoses, kissing bugs, and assassin bugs. There are over 2,500 species divided among 15 subfamilies, of which the **Harpactocorinae** is the largest, containing more than a third of all the species, and next in size is the subfamily **Reduviinae.** A very large percentage of the reduviids are predaceous and feed on insects, many of which are harmful; hence the family is in the main beneficial. A number of the species when handled carelessly defend themselves by biting, and a very few have developed a definite habit of sucking blood. The subfamily **Triatominae** comprises those members of the family which feed exclusively on blood of vertebrates. This subfamily is predominantly American.

In addition to the characteristic wings already described, the head of these insects is more or less elongated or cone-shaped, giving rise to the name conenose; the head has remarkably free movement; the eyes are conspicuous; the ocelli, if present, are located behind the compound eyes; the sturdy, three-jointed proboscis can be thrust forward, but in repose lies beneath the head; the piercing stylets can be extended far beyond the tip of the proboscis; the long, slender, four- or five-segmented antennae are situated in front of the eyes or on the border of the head; the prothorax is strongly developed; most of the species are able to fly well.

Life history. The rather large, usually smooth, more or less barrel-shaped eggs of reduviids (often with stellate or fringed caps) are generally deposited in situations where the adults occur, i.e., the ground-inhabiting forms deposit their eggs on the ground; arboreal forms lay their eggs on leaves and stems; and house-inhabiting forms in dusty

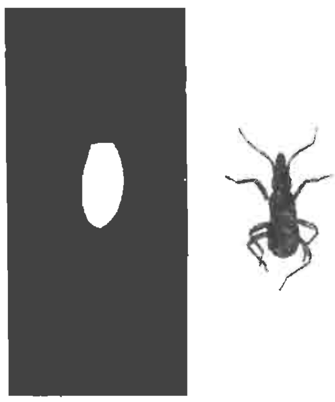


FIG. 39. Egg (*left*) and larva (*right*) of a conenose bug, *Triatoma protracta.* ×4.4

corners. The eggs of many of the species are illustrated by Readio,[12] who has also published an excellent account of the biology of the family.[13]

The eggs are commonly deposited singly, but sometimes in small clusters, the total number per female varying considerably from a few dozen to upward of 600. The incubation period varies from 8 to 10 days to nearly a month, depending upon the species and temperature. The newly hatched nymphs are wingless (Fig. 39). The usual number of nymphal instars is five, although Readio states that *Melanolestes picipes* (Herrich-Schaeffer) passed through only four. Some species overwinter in the egg stage, others as adults, and still others as nymphs. In most cases there appears to be but one generation a year. The length of the life cycle of *Triatoma rubrofasciata* (DeGeer) was found by Neiva to cover 210 days and for *Mestor megistus* (Burm.) 260 days. Usinger[14] reports a two-year life cycle for *Triatoma longipes* Barber.

The alimentary canal of Triatoma protracta (Uhl.). The alimentary canal of *Triatoma protracta* (Uhl.) according to Elson[15] is, as in other

Hemiptera, divided into three regions, the fore-, the mid-, and the hind-intestine (Fig. 16). The fore-intestine comprises the pharyngeal duct, the pharynx, and the esophagus, and measures 4.3 mm in length. The *pharyngeal duct* consists of a delicate tube which connects the pharynx with the suction canal of the maxillae. It is a very short duct and difficult to distinguish from the pharynx. The *pharynx* may be considered as a highly specialized portion of the alimentary canal. It is a boat-shaped organ, which measures on an average about 2 mm in length and .3 mm in diameter in the adult. The *esophagus* is a delicate duct, measuring about 2 mm in length and .1 mm in diameter. Before reaching the proventriculus the esophagus expands and forms the *proventricular vestibule.* The *mid-intestine* comprises the main portion of the alimentary tract and consists of the *proventriculus,* the *ventriculus* or stomach proper, and an elongated section. The *stomach* is a large sac, which when filled with blood, occupies the greater portion of the body cavity and crowds the other organs to the sides and behind. When empty it is wrinkled, but when replete with blood it is smooth and pear-shaped. The mid-intestine-3 is a much-coiled and narrowed portion of the mid-intestine; mid-intestine-4 is a short dilated portion. The hind-intestine is the shortest portion of the alimentary canal and comprises the *rectum.* This is a large muscular sac, capable of considerable distension and usually containing fecal material. It is pear-shaped with the broadest part 2.5 mm in diameter and the length 3 mm. At the termination of the mid-intestine there appears a circular whitish zone, the *rectal gland.* The *ampullae,* or enlarged bases of the *Malpighian tubules* (four in number and of equal size), form a rosette at this point. The Malpighian tubules are about twice the length of the insect, and it is difficult to unravel them.

The *salivary apparatus* consists of two pairs of glands, the principal and the accessory. The principal glands measure from 1 to 1.5 mm in length and .8 mm in diameter and are situated in the mesothorax, one on either side of the proventriculus. The spherical *accessory glands,* about .5 mm in diameter, are located in the metathorax directly posterior to the principal glands, with which they are connected by a duct.

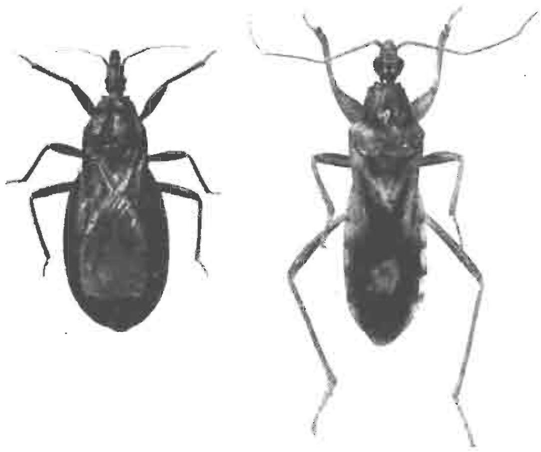
Conenose bites. Many of the species of conenoses inflict a painful bite when handled carelessly. Most notorious perhaps of all is the "kissing bug," *Reduvius personatus* (Linn.), a widely distributed species (belonging to the subfamily Reduviinae) particularly active in the middle western and eastern United States. As early as 1899 Howard,[16] quoting Le Conte, writes as follows:

> This species is remarkable for the intense pain caused by its bite. I do not know whether it ever willingly plunges its rostrum into any person, but when caught or unskillfully handled it always stings (pierces). In this case the pain is almost equal to that of the bite of a snake, and the swelling and

irritation which result from it will sometimes last for a week. In very weak and irritable constitutions it may even prove fatal.

The wheel bug, *Arilus cristatus* (Linn.), belonging to the subfamily Harpactorinae, also has a bad reputation as a biter. Reporting on the bite of this species, Hall[17] states:

> The finger became reddened and felt hot to the touch. In the course of a few days, growths resembling papillomas developed at the sites of the punctures, the largest of these projecting as a small hornlike structure. Both of these growths persisted for months, the largest slowly disappearing between six and nine months after the infliction of the bite. The injured finger remained warmer than the other fingers during this period, and, according to the patient's statement, still feels warmer than the other fingers, a year later. The development of pronounced cutaneous growths after a bite appears indicative of the action of some toxin as a stimulant irritant.



A B

FIG. 40. Members of the family Reduviidae. (*A*) *Triatoma protracta,* (*B*) *Rasahus thoracicus.*

The symptoms produced by the bites of *Triatoma protracta* (Uhler) (Fig. 40A), a widely distributed Pacific coast species commonly known as the China bedbug or cross bug and normally occuring in the nests of wood rats, *Neotoma* spp., are described as follows by reporting physicians: "In a few minutes after a bite the patient develops nausea, flushed face, palpitation of the heart, rapid breathing, rapid pulse, followed by profuse urticaria all over the body. The symptoms vary with individuals in their intensity." A blood examination made on a man about four hours after he was bitten on the left arm showed a leucocyte count of 11,000 (polymorphonuclears, 66 per cent; lymphocytes, 28 per cent; monocytes, 3 per cent; eosinophils, 3 per cent). This man's symptoms a few minutes after the bite were large hive-like swellings in the regions of the glands; intense itching and redness, especially in the hands; pulse rate, normally 60, increased to 140.

Inquiries concerning these insects in California are most frequent during May and June. They often attack sleeping individuals during the night. Tests made in the laboratory indicate that some persons experience no ill effects from the bite of this species (Wood[18]).

The bloodsucking conenose or "Mexican bedbug," *Triatoma sanguisuga* (Lec.), inflicts a very severe bite, which, because of the uniform character of the resulting symptoms, supports the view that a specific venom is injected with the bite. The bite is said to result in "a burning pain, intense itching and much swelling . . . with red blotches and welts all over the body and limbs." The effects of the bite may last for months; however, they usually disappear within a few days.

The "two spotted corsairs," *Rasahus biguttatus* (Say) and *Rasahus thoracicus* Stål (Fig. 40B), belonging to the subfamily Piratinae, the former common in the southern United States, Cuba, and South America, and giving way to the latter in the Northwest and California, are the cause of many complaints. Howard (1899, *loc. cit.*) cites the following paper concerning "so-called spider bites," published by Dr. A. Davidson in the *Therapeutic Gazette* of February 15, 1897, in which that author arrives at the conclusion that almost all of the so-called "spider bites" met with in southern California are produced by no spider at all but by *Rasahus thoracicus* Stål. The symptoms which he describes are as follows:

> Next day the injured part shows a local cellulitis with a dark central spot; around this spot there frequently appears a bulbous vesicle about the size of a ten-cent piece and filled with a dark grumous fluid; a smaller ulcer forms underneath the vesicle, the necrotic area being generally limited to the central part, while the surrounding tissues are more or less swollen and somewhat painful. In a few days with rest and proper care the swelling subsides, and in a week all traces of the cellulitis are usually gone. In some of the cases no vesicle forms at the point of injury, the formation probably depending on the constitutional vitality of the individual or the amount of poison introduced.

The use of warm compresses of magnesium sulphate is recommended for the bites of conenoses.

Chagas' disease (**American trypanosomiasis also known as Brazilian trypanosomiasis**) was first described in 1909 by Chagas (*loc. cit.*, p. 6) from Brazil. He named the causal organism *Schizotrypanum cruzi* (now more generally known as *Trypanosoma cruzi*), a spindle-shaped trypanosome, with a single flagellum and a characteristic undulating membrane. The trypanosome occurs sparsely in human blood, hence diagnosis of the infection by recovery of the organism from the blood is very difficult. The diagnostic method of Brumpt[19] to which he gave the name *"le xénodiagnostic"* is now widely used; he described the method as follows: "Je donne le nom de *xénodiagnostico* (zenos, hôte) au diagnostic pouvant être fait au moyen de l'hôte agent vecteur habituel de la maladie parasitaire, ou à l'aide d'un hôte vicariant pouvant assurer la culture et l'evoluation du parasite. C'est en somme une culture naturelle du parasite chez des hôtes favorables." Essentially this refers to the use of appropriate noninfected (clean) *Triatoma* bugs, which are allowed to feed upon a person suspected of having the disease, and after incubation in the body of the insect the trypanosomes, if present, may be recovered very easily from the digestive tract of the bug either by dissection or by microscopic sampling of feces taken from the rectum by means of a slender pipette.

The most apparent symptom of Chagas' disease in most cases is the unilateral swelling of the eyelid and face known as the sign of Romaña. The disease is most prevalent in children in whom it causes a high, long-continuous fever, facial edema, adenitis, and anemia. Infection may last many years. Symptoms of the chronic stage are commonly cardiac, because of the cardiotropism of the causal organisms; death is frequently due to chronic myocarditis and is commonly sudden. The *Leishmania* form of the trypanosome invades and destroys endothelial and other cells notably cardiac and striated muscle. In a recent contribution, Romaña[20] (1947) re-emphasizes the nervous forms of Chagas' disease, based on a study of cases of chronic encephalopathy; the patients presented "a syndrome of psychic states and of spastic paralysis" which agrees with the classical descriptions of Chagas and Villela. The disease occurs in man throughout most of South and Central America from Argentina to Mexico. In reservoir animals the infection occurs also in the southwestern United States.

Transmission of the infection. Chagas (*loc. cit.*, Chapter I) reported successful transmission of the infection through the agency of *Triatoma* (= *Conorhinus*) *megistus* (Burm.), but believed it was effected by means of the bite of the insect. Brumpt[21] (1912), using *Rhodnius prolixus* Stål, disproved the salivary theory of transmission by demonstrating that the infectious stage of the life cycle of the trypanosome is completed in the hind-gut of the insect and that the infection reaches the victim in the feces of the bug, which almost invariably defecates on the skin of its victim while in the act of sucking blood. From the soiled skin trypanosomes are readily transferred by fingers or otherwise to the highly receptive conjunctiva of the eyes or the mucosa of the mouth or nose, and infection takes place. Inoculation may also be effected by ***rubbing in***

the organisms through the excoriated skin, e.g., by scratching. The incubation period in man is said to be from 10 to 12 days.

Although bugs may receive their infection from man, it is no doubt usually received from reservoir animals. On ingestion, the metacyclic trypanosomes lose the flagella, change to oval "Leishmania forms," and multiply, giving rise to "crithidial forms" and the final infectious metacyclic stage in the hind-gut of the insect. This life cycle requires from 6 to 15 days. The percentage of *Triatoma* found infected in nature is startling; thus studies of this nature by various workers in widely separated areas cited by Usinger (*loc. cit.*) showed that 43 per cent of 4,181 bugs were infected.

The following species of conenose bugs, among others, have been found naturally infected: *Rhodnius prolixus* Stål in Mexico and Venezuela; *Panstrongylus geniculatus* (Latr.) in Brazil and Panama; *Triatoma*

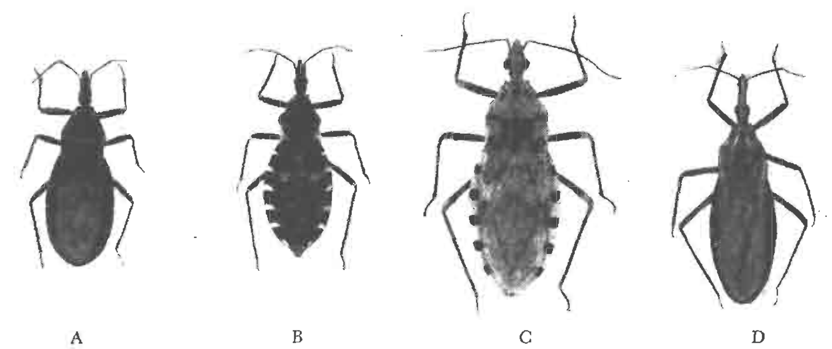


FIG. 41. Examples of Reduviidae: (*A*) *Triatoma protracta*, (*B*) *Triatoma sanguisuga*, (*C*) *Panstrongylus geniculatus*, and (*D*) *Rhodnius pallescens*.

dimidiata (Latr.) in Mexico, Panama,[22] Guatemala, and San Salvador; *Triatoma rubrofasciata* (DeGeer) in Brazil; *Triatoma rubida* (Uhl.), *T. hegneri* Mazzotti, *T. barberi* Usinger, and *T. phyllosoma* (Burm.), all in Mexico; *Triatoma sordida* (Stål) and *T. delpontei* Romaña y Abalos, in Argentina; *Triatoma flavida* (Neiva) in Cuba; *Triatoma sanguisuga* (Le Conte), *T. ambigua* Neiva, *T. gerstaeckeri* (Stål), *T. heidemanni* Neiva, and *T. protracta woodi* Usinger in Texas; *T. longipes* Barber, *T. protracta* (Uhl.) and *T. rubida uhleri* Neiva, in Arizona; in California, S. F. Wood[23] found a high natural infection in *Triatoma protracta* (Uhl.), namely 25 per cent of 816 bugs examined (Fig. 41).

The infection occurs in natural reservoir animals such as armadillos, opossums, house mice, rats (Norway), bats (several species), cats, dogs, squirrels (*Sciurus*), and wood rats.

Kofoid and Donat (1933[24]) reported on the occurrence of the infection in the wood rat, *Neotoma fuscipes macrotis* Thomas, in San Diego County, California, with *Triatoma protracta* (Uhler) the vector. F. D. Wood,[25] reporting more fully later (1934), states that the bloodsucking bug, *Triatoma protracta* (Uhler), and the wood rat, *Neotoma fuscipes macrotis* Thomas, are natural carriers of *Trypanosoma cruzi* in southern California. She was able experimentally to infect the following animals with the trypanosome: albino rats, albino mice, rhesus monkeys, a puppy, an opossum, the dusky-footed wood rat, and five species of white-footed mice. S. F. Wood[26] (1943) reports naturally infected conenose bugs from Arizona. Packchanian[27] reports successful experimental infection of a human being in Texas, using *Triatoma heidemanni* Neiva as the vector.

It is of interest to note that Morishita (1935)[28] has conducted experiments with *Trypanosoma conorhini* Donovan, which is regarded as non-pathogenic; it resembles *T. cruzi* in its insectan phase, occurring commonly in the gut of *Triatoma rubrofasciata* (DeGeer) in Formosa. The natural vertebrate host has not yet been discovered.

Several species of bedbugs are capable of transmitting the infection experimentally, as well as many species of ticks, among these *Amblyomma cajennense* (Fabr.), *Rhipicephalus sanguineus* (Latr.), *Ornithodoros moubata* (Murray), and *Ornithodoros savignyi* (Audouin). *Ornithodoros turicata* (Duges) has been proved to be an experimental vector of the Brazilian strain by Wheeler.[29]

Control. The medical and social importance of Chagas' disease is being widely recognized with the growing knowledge of the disease and particularly with the advance in the knowledge of its vectors. There appears to be no effective etiologic treatment, and there seems to be no spontaneous cure. Measures to prevent the spread of the infection should be aimed at the suppression of the insect vectors and limitation of reservoir animals. The widespread distribution and common occurrence of both vectors and reservoirs add tremendously to the difficulty of the problem. Educational measures that will reach the general public in all infested areas are of paramount importance.

It is generally agreed that one of the major approaches to the problem is improvement in housing conditions in endemic areas, so as to reduce and exclude reservoir animals (both wild and domestic) and to prevent entrance and multiplication of the responsible insects.

Disinfestation of sleeping quarters, dwellings, and outhouses by the use of one or more of the newer insecticides, such as benzene hexachloride, chlordane, and special formulations of DDT has been recommended. Romaña and Abalos[30] consider the first named as the preferred insecticide, to be used on roofs, walls, furniture, henhouses, corrals, etc. at the rate of 500 mg "Gammexane" per square meter, giving six months' protection.

*Key to Some Predaceous Reduviidae Likely to Be of Medical Importance**

1. Hemelytra with a quadrangular or discoidal cell at the base of the membrane. Subfamily Harpactocorinae 2
   Hemelytra without a quadrangular or discoidal cell at the base of the membrane 4
2. Sides of mesosternum without a tubercle or fold in front. Basal segment of beak about as long as front portion of head. Tribe Zelini .... *Zelus socius* Uhler
   Zelus exsanguis (Stål)
   Sides of mesosternum with a tubercle or fold in front of the hind angles of the prosternum. First segment of the beak longer than front portion of head. Tribe Harpactocorini 3
3. Front femora but little, if at all, thickened, unarmed, a little granulated. Pronotum produced posteriorly over scutellum and with a high, median, tuberculate ridge. Form robust .... *Arilus cristatus* (Linn)
   Front femora thickened, spinose, densely granulated. Pronotum not produced or elevated as above. Form slender .... *Sinea diadema* (Fabr.)
4. Pronotum constructed behind the middle. Front coxae with outer sides flat or concave. Subfamily Piratinae 5
   Pronotum constricted at or before the middle. Front coxae not flattened, their outer sides convex. Subfamily Reduviinae 6
5. Apical portion of anterior tibia angulately dilated beneath, the spongy fossa being preceded by a small prominence. Hemelytra entirely black .... *Melanolestes picipes* (H. S.)
   Tibia not dilated, the spongy fossa elongate. Corium and membrane of hemelytra each marked with a yellow spot .... *Rasahus thoracicus* (Stål
   Rasahus biguttatus (Say)
6. Antennae inserted on top of head between margins close to eyes. Antenniferous tubercles not projecting from sides of head. Beak stout, distinctly curved. Front of head turned downward. Tribe Reduviini
   Reduvius personatus (Linn.)
   Antennae inserted in lateral or dorsolateral margins of head. Antenniferous tubercles projecting slightly from sides of head. Beak slender and relatively straight. Head strongly produced anteriorly. Tribe Triatomini 7
7. Head very long and slender with antennae inserted near the apex .... *Rhodnius prolixus* Stål
   Head moderately long with the antennae inserted at or behind anteocular region .... *Triatoma sanguisuga* (Lec.)
   Triatoma protracta (Uhl.)

* Prepared by Dr. Robert L. Usinger for the purposes of this book.

C. SOME OTHER BUGS

Family Polyctenidae

This family includes the relatively little known *bat bugs,* which have led a very uncertain systematic existence since 1874, when Westwood founded the family Polyctenidae to receive these insects as aberrant Anoplura. Later they were placed with the Hippoboscidae, or louse flies (Waterhouse, 1879). The number of known species is quite small (less than a score); all are bloodsucking ectoparasites of bats. They have a four-jointed rostrum (three-jointed in a single species from Africa), tarsi three-jointed and antennae four-jointed, eyes wanting, hemelytra short; the body commonly bears ctenidia (combs). They are viviparous, the young being born at an advanced stage of development.

The two species known from the United States have recently been described by Ferris and Usinger. One of these, *Hesperoctenes hermsi,* was taken on the free-tailed bat, *Tadarida macrotis* (Gray), in the Chisos Mountains, Texas, at an elevation of 6,200 feet, by Mr. A. E. Borell. The other, *Hesperoctenes eumops,* has been taken at several localities in southern California on another free-tailed bat, *Eumops perotis californicus* (Merriam).

*Key to the Principal Families of Hemiptera-Heteroptera of North America which contain predaceous species**

1. Antennae at least as long as the head; either free or, in the Phymatidae, fitting in a groove beneath the lateral margins of the pronotum. Suborder Gymnocerata 6
   Antennae shorter than the head and nearly or quite concealed in a cavity beneath the eyes. Suborder Cryptocerata 2
2. Ocelli present. Shore-frequenting insects. (Toad bugs) .... Family Gelastocoridae
   Ocelli absent. Aquatic forms 3
3. Hind tarsi with indistinct, setiform claws (except in the minute, 3 mm or less, Pleine). Swim with ventral side upward (Back swimmers) .... Family Notonectidae
   Hind tarsi with distinct claws 4
4. Membrane of the hemelytra without veins. Abdomen without caudal appendages (Water creepers) .... Family Naucoridae
   Membrane of the hemelytra with distinct veins. Abdomen with caudal appendages 5
5. Caudal appendages of abdomen long and slender. Tarsi one-segmented (Water scorpions) .... Family Nepidae
   Caudal appendages of abdomen short, flat, and retractile. Tarsi two-segmented (Giant water bugs) .... Family Belostomatidae

* Prepared by Dr. Robert L. Usinger for the purposes of this book.

6. Apex of last tarsal segment more or less split. Claws of at least the front tarsi inserted before the apex. Superfamily Gerroidea 7
Last segment of tarsi entire, with claws of all the legs inserted at apex 8
7. Hind femora much surpassing apex of abdomen. Middle and hind coxae approximate, distant from front ones (Water striders) .... Family Gerridae
Hind femora scarcely surpassing tip of abdomen. Middle coxae (except in *Rhagovelia*) equally distant from front and hind ones. (Broad-shouldered water striders) .... Family Veliidae
8. Head very long, at least as long as the three thoracic segments combined. Body linear. Legs and antennae very long and slender (Marsh-treaders) .... Family Hydrometridae
Head shorter than pronotum and scutellum together 9
9. Antennae five-segmented. (Only members of the subfamily Asopinae are predaceous. These have nonraptorial front legs, three-segmented tarsi, and the first segment of the beak short, thick, and free) (Stink-bugs) .... Family Pentatomidae
Antennae four-segmented (except in one genus of the Hebridae and the genus *Pagasa* of the Nabidae, where they are five-segmented, and in the Ectrichodine genus *Rhiginia* of the Reduviidae, where they are eight-segmented. These exceptions do not have the combination of characters listed under Asopinae) 10
10. Beak three-segmented 11
Beak four-segmented 16
11. Front legs more or less raptorial. Head cylindrical. Prosternum with a median longitudinal stridulatory groove which receives the tip of the beak 12
Front legs not raptorial. Head rarely cylindrical. Prosternum without a median longitudinal stridulatory groove between the front coxae 13
12. Front legs with greatly thickened femora (Ambush bugs) .... Family Phymatidae
Front femora somewhat thickened but much less than half as wide as long (Assassin bugs) .... Family Reduviidae
13. Hemelytra with a cuneus (e.g. apical triangular region of corium separated by a fracture) (Flower bugs) .... Family Anthocoridae
Hemelytra without a cuneus 14
14. Ocelli absent. Hemelytra reduced to small pads, without any trace of a membrane. Body oval in form (Bedbugs, bat bugs, etc.) .... Family Cimicidae
Ocelli present 15
15. Membrane, when present, without veins; Clavus of same texture as membrane. Body linear (Water treaders) .... Family Mesoveliidae
Membrane with distinct, looped veins. Clavus and corium alike in texture. Body suboval (Shore bugs) .... Family Saldidae
16. Hemelytra with a cuneus (Leaf bugs) .... Family Miridae
Hemelytra without a cuneus **17**

17. Body densely clothed with a very short, velvety pile. Membrane without veins (Velvet water bugs) .... Family Hebridae
Body not clothed with a velvet pile 18
18. Hemelytra reduced to small pads. Ctenidia present at least on under side of head and often on antennae, head, pronotum, and elsewhere (Bat bugs) .... Family Polyctenidae
Hemelytra usually well developed. Without ctenidia 19
19. Front legs raptorial. Beak with basal segment stout and very short; 20
Front legs not raptorial. Beak long and slender, the first segment long, held between the bucculae at rest 21
20. Hemelytra entirely membranous (Gnat bugs) .. Family Enicocephalidae
Hemelytra with the basal portions thickened to form a corium distinct from the apical membrane (Damsel bugs) .... Family Nabidae
21. Membrane with numerous veins which more or less run together (Squash bug, etc.) .... Family Coreidae
Membrane with only four or five simple veins usually arising from the base (Chinch bug, etc.) .... Family Lygaeidae

BIBLIOGRAPHY

1. Usinger, Robert L., 1934. "Bloodsucking among phytophagous Hemiptera," *Canad. Entomologist,* 66:97–100.

2. Horvath, G., 1912. "Revision of the American Cimicidae," *Ann. Musei Nationalis Hungarici,* 10:257–62.

3. Joyeux, Charles, 1913. "Biologie de *Cimex boueti,*" *Arch.* de. *parasit.,* 16:140–46.

4. Usinger, Robert L. 1947. "Native hosts of the Mexican chicken bug, *Haematosiphon inodora* (Duges)," *Pan-Pacific Entomologist,* 23:140.

5. List, George M., 1925. "Three new genera and three new species of Cimicidae from North America," *Proc. Biol. Soc. Washington,* 38:103–10.

6. Marlatt, C. L., 1946. "Bedbugs and their control," *Pest Control and Sanitation,* 1:8–11.

7. Nuttall, G. H. F., 1913. "The Herter Lectures. I. Spirochaetosis." *Parasitology,* 5:262–74.

8. Patton, W. S., 1907. *The development of the Leishman-Donovan parasite in Cimex rotundatus.* Govt. of India: "Scientific Mem." (by Officers of the Med. and San. Depts.), n.s. no. 31. 25 pp.

9. Francis, Edward, and Lake, G. C., 1922. "Transmission of tularaemia by the bedbug, *Cimex lectularius,*" U. S. Public Health Service, *Pub. Health Rep.,* 37:83–95.

10. Davis, Gordon E., 1943. "Further attempts to trasmit *Pasteurella tularensis* by the bedbug *(Cimex lectularius),*" *J. Parasitol.,* 29:395–96.

11. Wakeland, Claude, 1935. *Fumigation for the Control of Household Insects.* Moscow: Univ. Idaho, College Agric., Extension Circular no. 50.

12. Readio, P. A., 1926. "Studies on the eggs of some Reduviidae (Heteroptera)," Lawrence: Univ. Kansas, in Sc. Bull., vol. 16, pp. 157–79.

13. ——, 1927. "Studies on the biology of the Reduviidae of America north of Mexico." Lawrence: Univ. Kansas, in Sc. Bull., vol. 17. 289 pp.

14. Usinger, Robert L., 1944. *The Triatominae of North and Central America and the West Indies and Their Public Health Significance.* Washington. D. C.: Govt. Print. Office, in Public Health Bull., no. 288. iv + 83 pp.

15. Elson, J. A., 1933. *A Morphological and Histological Study of the Mechanism of Suction, Salivary Apparatus and Alimentary Canal of Hemiptera, with Special Reference to Triatoma protracta (Uhler).* Doctorate thesis, Univ. Calif.

16. Howard, L. O., 1899. *The Insects to Which the Name "Kissing Bugs" Became Applied During the Summer of 1899.* Washington, D. C.: Dept. Agric., in Div. Entomol. Bull., no. 22.

17. Hall, Maurice C., 1924. "Lesions due to the bite of the wheel-bug, *Arilus cristatus* (Hemiptera; Reduviidae)," *Arch. Int. Med.,* 33:513–15.

18. Wood, Sherwin W., 1942. "Reactions of man to the feeding of reduviid bugs," *J. Parasitol.,* 28:43–49.

19. Brumpt, E., 1914. "Le xénodiagnostic application au diagnostic de quelques infectious parasitaires et en particulier à la Trypanosome de Chagas," *Bull. Soc. path. exot.,* 7:706–10.

20. Romaña, C., 1947. "Miocarditis cronica equizo trepanosica," *An. d. Inst. med. regional,* Univ. Nacional de Tucuman, Argentina, 2:1–39.

21. Brumpt, E., 1912. "La *Trypanosoma cruzi* evolue chez *Conorhinus megistus, Cimex lectularius, Cimex boueti* et *Ornithodoros moubata,* cycle evolutiv de ce parasite," *Bull. Soc. path. exot.,* 5:360.

22. Rozeboom, L. E., 1936. "*Triatoma dimidiata Latr.,* found naturally infected with *Trypanosoma cruzi* Chagas in Panama," *Am. J. Trop. Med.,* 16:481–84.

23. Wood, S. F., 1942. "Observations on vectors of Chagas' disease in the United States. I. California," *Bull. Calif. Acad. Sc.,* 41 (pt. 2):61–69.

24. Kofoid, Charles A., and Donat, Fae, 1933. "South American trypanosomiasis of the human type—occurrence in mammals in the United States," *Calif. & West. Med.,* 38:245–49.

25. Wood, Fae Donat, 1934. "Natural and experimental infection of *Triatoma protracta* (Uhler) and mammals in California with American human trypanosomiasis," *Am. J. Trop. Med.,* 14:497–517.

26. Wood, S. E., 1943. "Observations on vectors of Chagas' disease in the United States. II. Arizona," *Am. J. Trop. Med.,* 23:315–20.

27. Packchanian, A., 1943. "Infectivity of the Texas strain of *Trypanosoma cruzi* to man," *Am. J. Trop. Med.,* 23:309–14.

28. Morishita, Kaoru, 1935. "An experimental study on the life history and biology of *Trypanosoma conorhini* (Donovan), occurring in the alimentary tract of *Triatoma rubrofasciata* (de Geer) in Formosa," *Japanese J. Zoöl.,* 6:459–546.

29. Wheeler, Charles M., 1938. "Experimental infection of *Ornithodoros turicata* (Duges) with a Brazilian strain of *Trypanosoma cruzi* Chagas," *Proc. Soc. Exper. Biol. & Med.,* 38:191–93.

30. Romaña, C., and Abalos, J. W., 1948. "Accion del "Gammexane" sobre los Triatomideos. "Control" domiciliario," *An. d. Inst. med. regional,* Universidad Nacional de Tucuman, Argentina, 2:95–106.

CHAPTER IX

THE LICE

A. THE SUCKING LICE

Order Anoplura

General characteristics. The sucking lice comprise the order Anoplura. Some believe them so closely related to the biting lice that both groups are placed in the same order, viz., Anoplura, with the Mallophaga (biting lice) reduced to a suborder and the sucking lice placed in the suborder Siphunculata. There is little to support placing these parasites in a suborder (Parasita) of the Hemiptera. For the purposes of this work the lice are classified as members of two orders, Anoplura (sucking) and Mallophaga (biting), resembling each other in many respects, but differing radically in their feeding habits, the former being bloodsuckers while the latter feed on scales, secretions, and detritus of the skin. Wings are absent in both groups, and metamorphosis is simple. The body is flattened; the legs are in part adapted for clinging to hairs and feathers. The Anoplura have a protrusible proboscis at the tip of the head; the Mallophaga have a pair of tiny mandibles situated on the under side of the head. The biting lice are, as a rule, much more active than the sucking lice.

The Anoplura parasitize mammals, while the Mallophaga are parasites of both mammals and birds and in all cases are normally host specific, permanent ectoparasites, very rarely except accidentally or experimentally transferable to a different species. The United States Bureau of Entomology is successfully rearing the human body louse on selected rabbits. The entire life cycle, egg to egg, is normally spent on one host.

Classification of Anoplura. Students technically concerned with the sucking lice will need to consult the classical works of Ferris, particularly his monograph appearing in eight parts, 1919 to 1935, in Publications of Stanford University Press.[1] The order comprises some 30 genera and 500 species (Essig); these are arranged according to various authors in four families: (1) **Echinophthiriidae,** with body thickly covered with short stout spines and scales, antennae four- or five-jointed, spiracles small; occurring exclusively on marine mammals, e.g., *Antarctophthirus* *trichechi* (Boh.) on the Pacific walrus, and *Echinophthirus phocae* (Lucas) on the seal; (2) **Haematopinidae,** body spines or hairs in rows, never with scales, tibiae with thumb-like process opposing the claw, eyes lacking, antennae five-segmented, e.g., *Haematopinus suis* (Linn.) on swine, and *Linognathus vituli* (Linn.) on cattle; (3) **Haematopinoididae,** lice of gophers in North America; (4) **Pediculidae,** the most important family of all from the public health standpoint, since it includes body lice, head lice, and pubic lice of man and other primates. **Haematomyzidae,** heretofore included as a family of the Anoplura, has been placed by Ferris[2] in the Mallophagan suborder, Rhynchophthirina; it includes but the single species *Haematomyzus elephantis* Piaget of the elephant.

The lice of man* and other primates. The family Pediculidae the only eye-possessing lice, includes the three genera, *Pedicinus, Pediculus,* and *Phthirus.* The genus *Pedicinus* belongs exclusively, according to Ferris, to the *Cynomorpha* monkeys, and includes eight known species, *Pedicinus eurygaster* (Burm.) on *Macacus; Pedicinus longiceps* Piaget on *Pithecus; Pedicinus albidus* (Rudow) on *Macaca* (Barbary ape); *Pedicinus hamadryas* Mjöberg on *Hamadryas; Pedicinus patas* (Fahrenholtz) on *Cercopithecus patas; Pedicinus ancoratus* Ferris on *Presbytis; Pedicinus pictus* Ferris on *Colobus caudatus; Pedicinus obtusus* (Rudow) on *Semnopithicus maurus.*

The genus *Pediculus,* according to Ferris, includes only three species: (1) *Pediculus humanus* Linn., the head louse and body louse of man, commonly referred to in medical literature as two distinct species, namely, *Pediculus capitus,* head louse, and *Pediculus vestimenti,* body louse; (2) *Pediculus mjöbergi* Ferris of *Ateles* apes; and (3) *Pediculus schaffi* Fahrenholz, of the chimpanzee.

The genus *Phthirus* (also spelled *Phthirius*) includes the so-called crab lice, *Phthirus pubis* (Linn.) of man; and *Phthirus gorillae* Ewing, of the gorilla.

SUCKING LICE OF MEN

The human head louse, *Pediculus humanus capitis* DeGeer (Fig. 42), is gray in color, but is said to vary according to the color of the hair and color of the host (Murray[3]). The male averages 2 mm in length and the female 3 mm. This species occurs on the head, about the ears and occiput, but from reliable observations made by a number of observers it may in heavy infestations establish itself on other hairy parts of the body. In severe infestations the hair may become literally matted with eggs

* For extensive list of references (961) see "Bibliography on Lice and Man, with particular reference to wartime conditions," by Mary E. Grinnell and Ina L. Hawes, U. S. Dept. Agric. Washington, D. C. *Bibliographical Bull.* no. 1, July, 1943.

(nits), parasites, and exudate from the pustules which originate from the louse bite. The term *plica palonica* is applied (Stiles) to the fetid mass, forming a sort of carapace (*trichoma*), in which fungus may develop, and beneath which myriads of lice may be found.

The number of eggs deposited by the female ranges from 50 to 150. These are glued to the hair and hatch in from 5 to 10 days, an average of 7 days. Development is very rapid (incomplete metamorphosis with three molts), three weeks usually covering the entire cycle from egg to egg. Lice are easily disseminated by physical contact and otherwise, hence slight infestations may occur under the best of sanitary conditions, particularly among school children. However, the continued presence of lice on head or body is inexcusable since eradication is simple (see lousicides, p. 124). The mere use of soap and water in washing the head and hair is ineffective in destroying lice present in the hair.

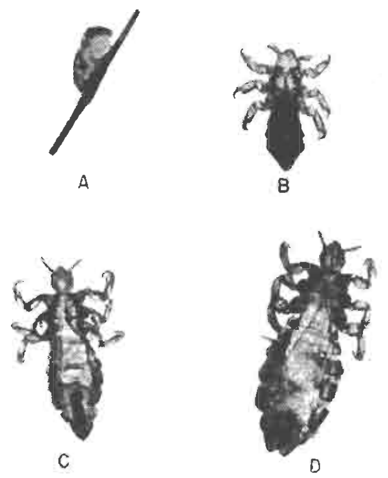


FIG. 42. Life cycle of the head louse, *Pediculus humanus capitis*. (*A*) egg attached to hair, (*B*) nymph, (*C*) male, and (*D*) female.

The pubic louse, *Phthirus pubis* (Linn.) (= *Phthirus inguinalis* Redi), also called crab louse (Fig. 43), is easily recognized by its crab-like appearance. It measures from 1.5 to 2 mm in length, is nearly as broad as long, and is grayish white in color. It infests the pubic regions particularly but also the armpits and more rarely other parts of the body, such as the mustache, beard, eyelashes, and eyebrows. The writer has seen soldiers infested with this species of louse from the ankles to the eyebrows. These lice are remarkably stationary in their habits, often remaining attached for days at one point with mouth parts inserted into the skin. The pruritus caused by the bites of these parasites is very intense, and a discoloration of the skin usually results if infestation continues over a longer period of time. The term *phthiriasis* may be employed to designate infestations of pubic lice, although the term *pubic pediculosis* is also used.

The female louse deposits its eggs on the coarser hairs of the body where the parasites occur. The number of eggs deposited per female is apparently quite small, although Nuttall[4] states that he would not be surprised to learn that 50 or more eggs may be layed. The incubation period seems to be from six to eight days. After three molts the adult stage was reached in Nuttall's experiments in from 15 to 17 days, and the egg-to-egg period was 22 to 27 days. Pubic louse infestations are very common among adults. Spread is usually by physical contact with infested individuals or use of infested toilet seats, blankets, etc.

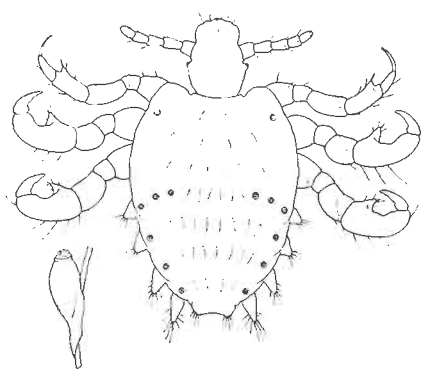


FIG. 43. The pubic louse, *Phthirus pubis*. Egg attached to hair.

The body louse, *Pediculus humanus corporis* DeGeer (= *Pediculus vestimenti* Nitzch) (Fig. 44), is the common clothing louse which during World War I became known as the "cootie," also called the "grayback." During World War II it became known as "mechanized dandruff" and "seam squirrel." Parasitologists now quite generally agree that there is no specific difference between it and the head louse; both are regarded as racial forms of *Pediculus humanus* Linn. Evidence concerning this matter is presented by Nuttall,[5] whose admirable treatises on lice and their relation to disease and control should be read by all students interested in the subject. Patrick A. Buxton's *The Louse*[6] is an important contribution.

Body lice infest the clothing where it comes in close contact with the body rather continuously, e.g., the fork of the trousers, the armpits, the waist line, neck, and shoulders. In his inspection of troops at delousing stations during World War I, the author usually found lice on the underclothing, but the nits were generally found in the seams of the breeches at the crotch if present at all. After all clothing was removed lice were occasionally found on the body. Eggs are undoubtedly by preference deposited on fibers in the seams of clothing as already mentioned. Nuttall and others have satisfactorily proved that the body louse may at times attach its eggs to the coarser hairs of the body.

Nuttall states that a female body louse may lay from 275 to 300 eggs, the average number laid per day being about 10 for 20 to 30 days. The shortest incubation period varies from 5 to 7 days when eggs are kept near the body at between 35° and 37° C. Hatching, according to Leeson[7] (1941), does not occur when the temperature reaches 23° C or below, nor at 38° C or higher. At 24° C the incubation period is 17 to 21 days; at 29° C it is from 9 to 11 days; at 35° C it is 5 to 7 days; at 38° C the

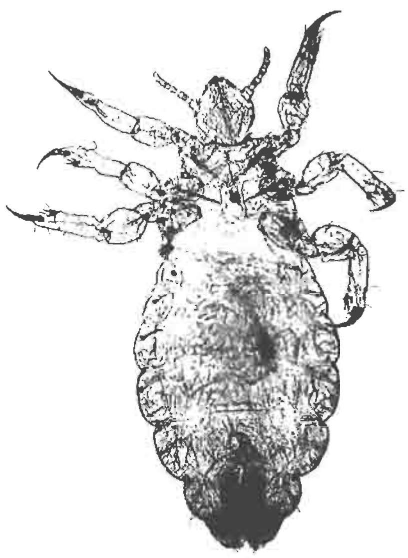


FIG. 44. Human body louse, *Pediculus humanus corporis.* ×15.

eggs will not hatch. The effective zone for the egg stage is apparently from about 23° to 38° C.

The young lice begin sucking blood at once on hatching from the eggs and throughout their development feed frequently both day and night, particularly when the host is quiet. Maturity is reached in 16 to 18 days from the time the eggs are deposited. There are three molts. Females begin laying eggs a day or two after reaching maturity. The egg-to-egg cycle averages about three weeks. Unfed lice soon die; probably 10 days would cover the longest period of survival without food. However, if fed, lice may live from 30 to 40 days. Moist fecal matter in masses or spiral threads is extruded as the louse feeds; the feces drys quickly in the air. Death may lurk in the excrement of a louse.

The following summary of temperature influences on lice given in a lecture on the "Life History of the Human Louse," by Pierce,[8] June 17, 1918, while varying somewhat from Leeson's (*loc. cit.*) results, is worth repeating:

> In the absence of definite humidity data we may roughly describe the zones of climatic influence on the lice as follows. The zone of minimum fatal temperatures for eggs in below 20° C. (68° F.) and for adults lies below zero centigrade (32° F.). The zone of dormancy in adults extends from about –10° to 5° C. (14° to 41° F.). The zone of sluggish movement without reproductive activity and with practically no digestive processes extends from 5° to 20° C. (41° to 68° F.). Digestion ceases at 12° C. The zone of optimum activity lies between 20° to 40° C. (68° to 104° F.) with the optimum about 30° C. Practically all egg hatching occurs within this zone, as does all oviposition, practically all assimilation of food, and all normal activity. From 40° to 44° C. the lice are wildly active; this zone represents one of exhaustion in which death of eggs occurs. Above 44° C. (112° F.) lies the zone of maximum fatal temperatures.

Dissemination of body lice. Lice live normally on the surface of the the body or in clothing being worn. Lice do not voluntarily leave unless the body grows cold in death or becomes hot with high fever. Even then they cannot travel far but are easily dislodged and may fall to the ground; they will quickly invade a new host if there is one close enough. Proof that lice are uncommon in blankets used by heavily infested troops was obtained by Peacock[9] who reports 0.8 per blanket and 31.4 per man including clothing. Louse infestation is mainly the result of contact with lousy persons or their infested clothing. As many as 10,428 lice and 10,253 nits have been reported on one shirt; in heavily infested populations one may readily collect 400 to 500 lice from one person.

Pediculosis. The presence of lice on any part of the body may be referred to as *pediculosis.* That louse bites may produce certain systemic disturbances seems to be indicated in a report made by Moore:[10]

> I started feeding about 700 to 800 twice a day. Almost immediately a general tired feeling was noticed in the calf of the legs and along the shin bones, while on the soles of the feet and underneath the toes this tired feeling was so intense as often to prevent sleep until late in the night. An irritable and pessimistic state of mind developed. An illness resulted with symptoms very similar to grip and a rash similar to German measles was present, particularly over the shoulders and abdomen.

The skin of persons who continuously harbor lice becomes hardened and deeply pigmented, a condition designated as *vagabond's disease* or *morbus errorum.* To relieve itching due to pediculosis a lotion made of 1 per cent spirits of thymol may be used, or a mixture of 5 per cent benzocaine, 2 per cent methyl salicylate, and 0.5 per cent salicylic acid made up with 70 per cent alcohol.

Favus and impetigo. Experiments conducted by Dewèvre (1892, *loc. cit.,* p. 5) and others prove that lice may carry the causal organisms of both favus (*Achorion schoenleini* Lebert) and tropical impetigo (*Diplococcus pemphigi contagiosi* Wherry). Nuttall (1899, *loc. cit.,* p. 4) states the Dewèvre "removed ten pediculi from a child suffering from impetigo and placed them on a healthy infant, which a few days later developed impetigo. The experiment was repeated several times with the same results. In a second series of experiments, he took scrapings from under the nails of children that had impetigo and placing them on artificially scratched places, reproduced the disease. Lastly he took pediculi from a child that was not affected with impetigo and placed them on a child that had the disease; removing them after twenty minutes, he replaced them on a healthy child. The latter acquired the disease, as did 50 per cent of the children so experimented with. He claims the specific microörganism adheres to the front legs especially, also to the hairs of the insect, and the latter carries them as bees do pollen. In the last set of experiments, he only allowed the pediculi to remain half an hour on the healthy head, but this was sufficient to produce infection."

Epidemic relapsing fever has occurred in many parts of the world and may be regarded as at least having been cosmopolitan in distribution. Widespread epidemics no longer occur. Great epidemics did occur, however, during World War I, notably in Rumania and later (1921) in Africa. There were frequent epidemics of this disease in Europe during the 18th and 19th centuries, and it was in the 1868 epidemic in Berlin that Obermeier observed "myriads of living and actively motile spirilla in the blood of relapsing fever patients during the febrile attack." In 1873 during another epidemic he applied Ehrenberg's nomenclature and called these organisms "*Spirochaeta.*" To honor the discoverer of the organism in the blood of relapsing fever patients Cohn gave the name *Spirochaeta obermeieri.* The designation *Spirochaeta recurrentis* (Lebert) has gained wide acceptance; however, because of priority one should now use *Borrelia recurrentis* (Lebert).

Although lice had long previously been under suspicion it was not until 1907 that Mackie[11] in India secured evidence that relapsing fever is a louse-borne disease. He noted that the spirochetes multiplied within the gut of the lice and that they could be found in the ovary, testis, and the Malpighian tubules of the insects. Mackie believed that infection might result from the insect's regurgitating the contents of its gut into the wound in the act of feeding. Later (1912) Nicolle, Blaizot, and Conseil[12] failed to transmit the spirochetes through the bites of infected lice and found that the only reliable successful experiments involved the injection or subcutaneous inoculation of an extract of infected lice. On the basis of experiments in which men and monkeys were exposed to hundreds of bites, they came to the conclusion that infection results from spirochetes which collect under the fingernails and on the finger tips of an individual after he has crushed lice on his skin, and which, in scratching, he inoculates into the excoriated skin.

Nicolle and his colleagues also found that the spirochetes disappear from and later reappear in the louse. Only a few remain in the insect's intestine up to 5 or 6 hours after infection, and none after 24 hours; but they reappear in the insect in from 8 to 12 days and are then present in the general body cavity, none being found in the alimentary canal. The authors believed that the spirochetes were transmitted to the offspring of infected lice. Chung and Feng[13] (1936) state that transovarian transmission of *Borrelia recurrentis* does not occur in lice. They also state that the salivary glands and Malpighian tubules of infected lice do not contain the spirochetes, and that the feces of infected lice are not infectious. The gastric juice of lice is detrimental to the *Borrelia;* only about 1 to 5 per cent or less of the ingested spirochetes gain access to the tissue and coelomic cavity where multiplication takes place, multiplication being by transverse division.

Man is the reservoir of the louse-borne infection (man-to-louse-to-man). There must be a human carrier in a lousy population. In tick-borne or endemic relapsing fever, endemicity is maintained in reservoir animals (rodents, etc.) Nuttall has reported that a single infective louse crushed upon the excoriated skin has produced relapsing fever. The incubation period in the human is from 6 to 10 days. The onset of the disease is sudden, with headache, chills and fever, and generalized pains. The fever remains high for four to six days and subsides abruptly, with an afebrile period of four to eight days followed by a relapse. There may be several relapses. Mortality is usually low but may vary from 2 to 50 per cent, or even higher in crowded poverty-stricken and louse-infested populations. Microscopic (darkfield) examination of blood smears taken during febrile periods will readily reveal *Borrelia.* Mouse inoculation with the patient's blood will produce the organisms in the blood of the mouse in from 24 to 48 hours. (For tick-borne or endemic relapsing fever consult Chapter XX.)

Typhus fever. Classical or old world typhus fever, known also as tabardillo (Mexico), Brill's disease (United States), jail fever, or war fever, is a disease of ancient origin and wide distribution, chiefly in Europe, north Africa, Asia, and higher altitudes of Mexico and Central and South America. The causal organism is *Rickettsia prowazeki* Da Rocha-Lima. Wherever human beings are concentrated in close quarters, especially in times of war and famine, this disease may become rampant. It is chiefly a disease of winter and spring, with mortality from 15 to 75 per cent. The disease is characterized by a high fever continuing about two weeks, backache, intense headache, bronchial disturbances, mental confusion, stupor, a congested face (designated also as a "besotted expression"), and on the fifth or sixth day by a brick-red macular eruption on chest and abdomen, which later spreads to other parts of the body, even to hands, feet, and face. This mottling led to the belief that tabardillo of Mexico was identical with spotted fever of Montana, a fact that was proved to be erroneous by Ricketts and Wilder.[14] Ricketts contracted typhus fever during the course of this investigation and died of the disease. Laboratory tests for typhus consist of complement fixation with typhus antigen, becoming positive in the second week; and Weil-Felix reaction (agglutination of *B. proteus* strain OX-19).

Transmission by lice. That the louse (*Pediculus humanus* Linn.) is probably the sole agent in the transmission of typhus fever from man to man was shown by Nicolle, *et al.*,[15] (1909, working in Tunis) and Ricketts and Wilder[16] (1910, working in Mexico). The latter found that *Pithecus* (*Macacus*) *rhesus* (Desmarest) can be infected with tabardillo (Mexican typhus) invariably by the injection of virulent blood from man taken on the eighth to tenth day of fever, that the monkey may pass through an attack of typhus so mild that it cannot be recognized clinically, and that immunity results. Typhus was transmitted to the monkey by the bite of the louse in two experiments, the lice in one instance deriving their infection from man and in another from the monkey. Another monkey was infected through the introduction of the feces and abdominal contents of infected lice into small incisions. There is a tendency to disregard the head louse (*P. humanus capitis*) as a vector of typhus; however, it must be remembered that Goldberger and Anderson[17] (1912) did succeed in transmitting typhus to a monkey by cutaneous injection of a saline suspension of crushed head lice.

Nuttall[18] points out that if lice (*Pediculus humanus corporis*) are crushed 9 or 10 days after an infective feed, or if their feces are collected three to six days after they have fed on infective blood, their contents and feces respectively are capable of producing infection if placed upon excoriated skin. It is now believed that the usual channel of infection is through fecal contamination. The Rickettsiae may remain alive and virulent in louse feces kept dry at room temperature for more than 60 days, thus infection may be acquired through the respiratory passages by inhalation of minute particles of louse excrement. It is generally believed that the normal channel is through scratches or abrasions and through conjunctival contact by fingers contaminated with louse feces.

The typhus fever patient is infectious for the louse, generally speaking, from early in the disease to about the tenth day, though later infections are possible. The Rickettsiae multiply enormously in the mid-gut of the louse; the epithelial cells become so distended after a few days that they rupture and enormous numbers of Rickettsiae appear in the insect's feces. It is believed that infected lice may die in a week or 10 days because of the enormous multiplication of these organisms. It is believed that few lice become infected while feeding on a typhus fever patient, probably less than half. If a louse survives the infection it remains infective for the rest of its life.

Murine typhus fever (flea-borne) is maintained in nature in rats, and transmission to man is effected through the agency of rat fleas. The causal organism is *Rickettsia mooseri* Montiero (sometimes designated as *Rickettsia prowazeki mooseri*). This disease is clinically similar to louse-borne typhus except that its course is milder and the rash is less profuse. The mortality is low, perhaps about 3 per cent. It is widely distributed in many parts of the world, particularly in warmer climates, in contrast to epidemic typhus which occurs in cooler climates favoring lousiness. Both may occur in the same areas. The use of the terms "endemic" and "epidemic" to distinguish the "flea-borne" from the "louse-borne" strain of typhus is open to criticism, since both must have an endemicity to survive and both may be epidemic at times. Furthermore it is now generally believed that "flea-borne" murine typhus may become "louse-borne" and epidemic, with high mortality. (The relation of fleas to murine typhus is discussed in Chapter XIX.)

Trench fever, also known as five-day fever, Volhynia fever, shank fever, His-Wernerische Krankheit, and Febris quintana, is a nonfatal disease characterized by sudden onset of fever, headache, dizziness, pains in the muscles and bones, particularly in the legs, with especial tenderness of the shins, and lasting 24 to 48 hours or longer, followed at intervals of about five days by other attacks of fever of less and less severity. While this disease was of considerable importance during World War I, it did not make its appearance during World War II.

The causal organism of trench fever is believed to be *Rickettsia quintana,* so named by Da Rocha-Lima[19] in 1916, confirming the earlier work of Toepfer.[20] This organism is found in the stomach lumen of the louse, while *Rickettsia prowazeki* of typhus invades the epithelial cells.

Two commissions, one British consisting of Byam, Carroll, *et al.,* and the other American, consisting of Strong, Swift, *et al.,* carried out very thorough investigations, during World War I; the summary, in part, of the findings of the British commission follows:

1. The whole blood from febrile trench fever cases, up to the 51st day of disease, when injected intravenously, is capable of reproducing the disease. The incubation period in such infectious varies greatly—from 5 to 20 days.

2. The virus as contained in the circulating blood is destroyed by the addition of distilled water in large quantities.

3. The bites alone of infective lice do not produce trench fever.

4. The excreta of infective lice when applied to a broken surface of skin do readily reproduce trench fever. The incubation period of such infections is remarkably constant and averages 8 days.

5. The excreta passed by lice fed on trench fever patients are *not* infective till the expiration of not less than 5 days from the commencement of the feeding on trench fever blood, thus indicating a developmental cycle in the louse or a period during which the organism multiplies.

6. Once lice are infective they remain so till at least the 23rd day from date of their infection.

7. The virus of trench fever, as contained in infected louse excreta, is capable of withstanding drying at room temperature, exposure to sunlight, keeping for not less than 16 days, and heating to 60° C. for 20 minutes.

8. 80° C. for 10 minutes destroyed the virus, which is therefore not a spore-bearing organism.

9. The bodies of infected lice when crushed upon the broken skin are capable of producing trench fever. When lice become so infective remains to be determined.

9a. Active trench fever blood equivalent to the content of 11 lice does not produce trench fever when rubbed into the broken skin.

10. Infection probably does not take place by the mouth or by inhalation.

11. The excreta of lice are not normally capable of producing trench fever. [Note: Some reports indicate that feces may be highly infectious for as long as 4 months.]

12. Trench fever infected lice do not transmit the disease to their offspring.

13. There is a possibility of some attacks of trench fever being afebrile throughout.

14. The percentage of individuals naturally immune to trench fever is exceedingly small.

15. Old age is no bar to infection.

16. Such immunity as results from an attack of trench fever is not permanent, and may only persist for so long as the individual shows evidence of the disease.

17. Even as late as the 79th day of disease a patient's blood may remain infective and be capable of infecting lice fed on such a patient while febrile.

Delousing methods practiced over a long period of years prior to and including the early part of World War II were cumbersome and usually expensive. The results were as a rule not wholly satisfactory in that the recently deloused individual was immediately subject to reinfestation if exposed to lousy persons. Among the methods available during the period of World War I were the following:

Fumigation of clothing, persons to be properly bathed while this was being done; required specially constructed sealed chambers, stationary or portable; fumigants, even when vacuum was used did not as a rule kill louse eggs;

among gases used were hydrocyanic acid gas, carbon tetrachloride,[21] and chlorpicrin.

Laundry methods, washing of rough cotton goods[22] at 180° F. for 30 minutes destroyed lice and their eggs; woolen goods[23] required a different treatment to avoid shrinkage.

Dry heat, temperature of 60° C. (140° F.) for 20 minutes killed both lice and eggs; required special types of heat chambers in order to circulate heat to insure equal distribution among the loosely hung garments.

Ironing with a hot iron.

Steam sterilization, insured disinfestation as well as disinfection; disadvantageous in that shrinkage and wrinkling resulted; required special expensive equipment such as steam sterilizers, delousing stations, etc; vacuum of from 12 to 15 inches for about 5 minutes, followed by 15 to 20 pounds steam pressure for 15 minutes, then allowed steam to escape and create 15 inches of vacuum, broke vacuum and unloaded contents when zero was reached. Serbian barrels were also used to generate steam.

Storage of garments, to kill lice by starvation; it was assumed that dry storage for about three weeks would prove effective.

Louse powder, N.C.I., consisting of commercial naphthalene 96 per cent, creosote 2 per cent, and iodoform 2 per cent, dusted in underclothing once a week.

Following World War I and up to 1941 little attention was paid to the development of new methods of louse control. Control methods fell back into the old groove of temporary relief, in spite of the fact that insect toxicology had advanced enormously as applied to agricultural pests. There was added, however, a new fumigant for delousing, namely *methyl bromide,* a very dangerous gas which destroys all stages of the louse. It was employed in two ways for delousing clothing, (1) fumigation of quantities of clothing in large gas-tight vaults; 30 minutes' exposure in a 330 cubic-foot vault with three pounds of the gas; lower temperatures required either more of the gas or a longer exposure; (2) fumigation of single outfits of clothing in individual gas-tight bags—each bag, 56 by 30 inches, was equipped inside in a pocket with an ampule containing about 20 cc of methyl bromide; after clothing was placed inside the bag and the bag closed, the ampule was crushed, liberating the gas; after 45 minutes the bag was opened and the clothing shaken out and aired. The use of this gas (methyl bromide) and practically all other aforementioned methods of delousing finally gave way to lousicidal powders in wartime practice.

Lousicides. Great credit is due to the group of workers stationed at the Orlando, Florida, laboratory of the United States Bureau of Entomology and Plant Quarantine who developed the "*MYL*" *insecticide powder,* which according to Miscellaneous Publication No. 606, United

States Department of Agriculture, August 1946, is composed of the following ingredients: 0.2 per cent of pyrethrins, 2 per cent of N-isobutylundecylenamine, 2 per cent of 2,4-dinitroanisole, and 0.25 per cent of Phenol S (isopropyl cresols, by-product of thymole manufacture) in pyrophyllite (all per cents by weight). It is effective against all species of human lice and not only repels them but kills them and their eggs. The powder is dusted lightly into the seams of the underclothing or on the infested parts of the body at weekly intervals.

Then came the Swiss louse powder, Neocid, consisting of 5 per cent mixture of DDT (dichloro-diphenyl-trichloroethane) in talc. With the successful field tests made with the three lousicide powders—MYL, DDT, and the British AL-63 (containing rotenone)—there was opened a new era in the control of louse-borne diseases. The Germans, according to Bushland[24] (1948), developed chloromethyl p-chlorophenyl sulfone ("Lauseto neu"), which was highly toxic to lice and an excellent ovicide, but not nearly so persistent in residual effect as DDT. The arresting of small typhus epidemics in native villages of Egypt, Algeria, and Mexico proved that the louse powders are practical. The enormity of this discovery can only be measured in the light of the great loss of life due to typhus during 1917–1921 (World War I) when, according to the Statistical Bulletin of the Metropolitan Life Insurance Company (November, 1941), Russia alone is said to have lost 2,500,000 to 3,000,000 of her people; millions more died as the epidemic swept through the Balkans, Poland, and Germany.

Typhus fever was again threatening in the early years of World War II. During 1942 there were some 3,000 cases in Egypt and about 80,000 cases in the rest of North Africa. When Allied forces landed in Italy in September, 1943, a typhus epidemic was threatening Naples, a city with a population of nearly a million persons, where there was congestion, insanitary living conditions, food scarcity, undernourishment, and confusion. The United States of America Typhus Commission was charged with responsibility for control of the disease on January 3, 1944 (Wheeler[25]); however, actual dusting operations were instituted on December 15, 1943, by a dusting team previously trained by a member of the Health Commission of the Rockefeller Foundation. Wheeler, a member of the United States Typhus Commission describes the measures used in the epoch-making campaign to control typhus in Italy. Although both MYL and DDT powders were used in the beginning of the campaign, it was essentially a DDT program, a mixture of 10 per cent DDT in talc being used.

Procedure. The procedure used in applying louse powders is described by Wheeler (*loc. cit.*) as follows:

It consisted essentially of forcefully blowing powder, by hand dusters or power dusters between the layers of clothing worn by the individual and between the innermost layer of clothing and the skin of the body. This was accomplished by a uniform technique, inserting the nozzle of the duster up the sleeves, down the neck (both front and back), around the waistline and into the crotch area of clothing. Hair and any cap or hat were dusted thoroughly. An infested person properly dusted is no longer a menace to others and will remain so for a period of at least two weeks, at the end of which time he should be redusted. Approximately 1 to 1½ oz. of powder per person is sufficient to insure the thorough dusting of all clothing worn.

The total number of dustings were reported as 3,265,786. By February 20, 1944, the typhus fever epidemic in Naples appeared to have been definitely broken, and upon direction of Brigadier General L. A. Fox, the majority of the United States Typhus Commission members were called back to headquarters in Cairo.

In the use of *louse powders under military conditions,* dusting with power dust guns should follow a certain routine to avoid missing some parts of the clothing being worn. Also in spite of the fact that few lice occur in blankets, bedding, extra clothing, and mattresses, these must be dusted to prevent reinfestation. If the treatment is done by the individual himself with a "sifter can" it is necessary to remove all the clothing from the body. From 1½ to 2 oz of the powder is needed on an average per person; however, under field conditions as much as 4 oz may be needed. Lice are usually killed in 24 hours, but the eggs are not killed; however, because of the lasting qualities of DDT all the young lice will be killed eventually on hatching. Where troops or others are working in intimate contact with infested populations, semimonthly dusting is recommended to prevent reinfestation.

Classical typhus has been the scourge of armies throughout recorded history. During World War I military hospitals in the Balkan countries were crowded with typhus patients, thousands being admitted daily during 1915. In striking contrast there were only 64 cases of louse-borne typhus in the American Army during all of World War II (Philip).

Treatment for head lice. Among the many and various methods employed in the eradication of head lice, from the primitive use of fingers, still a practice among natives as many of us have observed, and the use of a fine-toothed comb dipped in kerosene, which some of us have experienced, to the most modern use of DDT louse powder, only a few need be mentioned here:

1. A towel wet with 10 per cent solution tincture of larkspur wound turban-like around the head for several hours, repeat in about a week.
2. Washing the hair and scalp with 2 per cent solution of creolin; repeated in about a week.

3. A wash made of a mixture of 5 level tablespoonfuls of derris or cube powder, 2 level tablespoonfuls of neutral soap, and one quart of warm water, applied thoroughly to the hair, mopped up with a towel without drying the hair, and allowed to remain for at least eight hours; repeat in about ten days if necessary.
4. An organic thiocyanate lotion recommended by Busvine and Buxton[26] (1942), Lethane 284 Special: 12.5 per cent N-butyl carbitol thiocyanate, 37.5 per cent beta-thiocyanoethyl laurate; 50 per cent refined paraffin (Busvine and Buxton diluted the paraffin to 25 per cent); apply a little of the fluid to several parts of the scalp, exposed by parting the hair, then massage with the fingers.
5. Dusting the scalp with derris or cube powder containing 3 per cent rotenone; part the hair little by little and bring the powder in contact with the scalp with the fingers; leave the powder in the hair for 48 hours before washing.
6. Dusting the hair and scalp with DDT or MYL louse powder previously described, rubbing the powder in with the hands; an additional treatment is suggested in a week to 10 days later; the head should not be washed for at least 24 hours after each treatment.
7. NBIN formula, recommended by the United States Bureau of Entomology and Plant Quarantine:* benzyl benzoate 68 parts by weight, DDT 6 parts, benzocaine (or ethyl p-amino-benzoate) 12 parts, and Tween 80 (sorbitan monooleate, polyoxyalkylene derivative) 14 parts; dilute in 5 volumes of water and apply to scalp and hair; one treatment will kill all lice and nits.

Treatment for crab lice. Usually the treatments effective against head lice can also be used against crab lice, *Phthirus pubis.* The liquid or powder must be applied to the pubic and anal regions of the body, under the arms and wherever the body is hairy; in particularly hairy persons the lousicide must be applied from neck to foot, perhaps also to the eyebrows and beard. Whatever material is used it must be well distributed and must reach the skin. DDT, MYL, and NBIN (already described) as well as 5 per cent rotenone in 10 parts of petrolatum, are all effective if used as directed.

SUCKING LICE OF MAMMALS

Anoplura affecting domesticated mammals. The important sucking lice of domesticated ungulate mammals belong to the family Haematopinidae and are arranged mainly under three genera, *Haematopinus, Linognathus,* and *Solenopotes. Swine* have one species of louse, *Haematopinus suis* (Linn.) = (*H. urius* Nitzsch) (Fig. 45). This is the largest species of the entire group, measuring as much as 5 to 6 mm in length; it is cosmopolitan in distribution. The members of this species occur chiefly in the folds of the skin on the neck, at the base of and inside the ears, along the belly, and on the inner sides of the legs. According to

* Publication 606 on DDT.

Florence[27] hog lice feed readily on man but will not feed on guinea pigs. The same author summarizes, as follows, the life history of these lice at 35° C, when they were kept continuously next to the body in vials: incubation period of eggs (Fig. 46), 13 to 15 days, first molt after five to six days, second molt after four days, third and last molt after four to five days, sexual maturity after three days, a total of 29 to 33 days from egg to egg.

Cattle lice belonging to the Anoplura number five species, namely, (1) *Linognathus vituli* (Linn.), commonly known as the long-nosed ox louse or "blue louse," measuring about 2 mm in length and distinguished

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FIG. 45. Hog louse, *Haematopinus suis*. ×7.

FIG. 46. Nits (eggs) of the hog louse attached to the hair of the host. One of the eggs has hatched. × 10.

from the next species by its long nose and slender body; cosmopolitan in distribution; (2) *Haematopinus eurysternus* (Nitzsch), the cosmopolitan short-nosed ox louse, somewhat larger (3.5 to 4.75 mm in length) than the former and much broader in proportion; (3) *Haematopinus tuberculatus* (Burm.), the buffalo louse, infests cattle in parts of Australia; common on cattle and carabao in India; absent in North America; measures from 3.5 to 5.5 mm; resembles *H. eurysternus* closely but differs in that the number of setae at the margin of the abdominal segments caudad of the paratergal plates is usually eight or more (may be five or six); (4) *Haematopinus quadripertusus* Fahr., the tail louse; (5) *Solenopotes capillatus* Enderl. has been redescribed by Bishopp[28] and shown to have a wide distribution in the United States as well as in many other parts of the world. It is known as the little blue cattle louse. It measures from 1.2 to 1.5 mm in length and in general appearance resembles the short-nosed ox louse.

All species except the tail louse show a tendency to attach on the head, neck, and shoulders of the host, where the eggs are glued to hairs. In heavy infestations many parts of the animal are infested. The eggs of the short-nosed ox louse are usually whitish in color, while those of the long-nosed louse are dark blue, and those of the *Solenopotes* pale yellowish. The incubation period averages about 12 days with a range of from 8 to 15 days. The life cycle from egg to egg requires from 20 to 30 days. There are three molts at about four-day intervals. Lamson[29] reports that the short-nosed ox louse lays from 35 to 50 eggs over a period of from 10 to 15 days. Anyone concerned particularly with cattle lice will certainly consult Bulletin 832, Cornell University Agricultural Experiment Station (Matthysse[30]).

Horses, mules, and asses are frequently infested with one species of sucking louse, *Haematopinus asini* (Linn.) = [*H. macrocephalus* (Burm.)], which measures from 2.5 to 3.5 mm in length. It resembles the hog louse except that the head is relatively longer and more robust. The lice usually congregate at the base of the mane and forelock and the root of the tail. Hall[31] (*loc. cit.*) reports that 22 out of 38 horses examined were infested with this species. The same author reports the incubation period for the egg to range from 10 to 19 days (eggs kept in Petri dishes at from 21° to 31° C). It is quite probable that this species requires about the same time as the hog louse for the completion of its life cycle, i.e., from 29 to 33 days from egg to egg.

Sheep in some parts of the United States are affected by the so-called foot louse, *Linognathus pedalis* (Osborn). The author has observed this species on sheep from California and Nevada, and Osborn has reported it from Iowa. It occurs on the legs, especially in the region of the dew claws, but in heavy infestations it may actually invade the wool above the knee. Deaths have been reported as due to this louse. The species measures about 2 mm in length; and as it is the only sucking louse likely to attack sheep in the manner described above, its identity can be easily established. *Goats* often suffer heavy infestations of *Linognathus stenopsis* (Burm.).

Sucking lice on dogs and rabbits. Dogs are commonly heavily infested with *Linognathus piliferus* (Burm.), and *rabbits* harbor *Haemodipsus ventricosus* (Denny).

Control of sucking lice of mammals. Poorly fed animals, crowded pens, and dark, insanitary quarters are factors in the multiplication of lice, but the parasites may gain a foothold in spite of plenty of food, clean quarters, and adequate space; hence the stock farmer living in climates permitting this should not neglect to install a good dipping vat as a part of his equipment. Although lice will not breed away from their host, they may drop off with hair and may remain alive for probably not over five days; and the same animals after dipping or other animals of the same species, introduced into quarters before the dropped lice have died, may become reinfested. Furthermore, the great majority of the ordinary dips do not destroy the eggs present at the time of treatment; hence a second dipping is usually necessary after the young lice have hatched. This second dipping should be properly timed and in most cases should be done 16 to 18 days after the first. Dipping procedures are described by Babcock and Laake,[32] who recommended for cattle-louse control two dippings in a sulfur-rotenone dip, containing one pound of cube (5 per cent rotenone) and 10 pounds of wettable sulfur in 100 gallons of water. Application of dusts containing 10 per cent derris or cube (0.5 per cent or more of actual rotenone) is highly recommended by Matthysse (*loc. cit.*) for the control of all species of cattle lice. Experiments with DDT indicate that it is not as effective as rotenone; however, a 0.3 per cent DDT emulsion dip has been reported effective with one treatment (Furman).

Furman[33] (1947) reports complete eradication of *Haematopinus eurysternus* and *Linognathus vituli* with a single dip in a 0.3 per cent suspension of benzene hexachloride (approximately equivalent to 0.036 per cent gamma isomer). This material, which is now being used widely, should not be applied to lactating dairy animals. Furman and Hoskins[34] (1948) have shown that benzene hexachloride appears in the milk for several days following application. It is also known that this material may occur in the body fat of treated animals as long as 30 days after treatment where a concentration approximately double that used for louse control.

B. THE BITING LICE

Order Mallophaga

Classification. The biting lice (*Mallophaga*), of which there were about 1,400 described species[35] in 1916, and about 2,500 in 1937 according to Muesebeck, may be divided into two suborders: (1) **Amblycera** with short, clavate or capitate, four-segmented antennae, concealed in shallow cavities on the under side of the head; four-segmented maxillary palpi, mandibles horizontal; and (2) **Ischnocera,** with short, slender three- or five-segmented, exposed antennae, no palpi, mandibles vertical.

The **Amblycera** are divided into six families among them (1) **Gyropidae,** having tarsi with one claw, infesting mammals only, e.g., *Gyropus ovalis* Nitzsch and *Gliricola porcelli* (Linn.), both of the guinea pig; (2) **Menoponidae,** having tarsi with two claws, infesting birds mainly, e.g., *Menopon gallinae* Linn. (= *Menopon pallidum* Nitzsch), the common hen louse, and *Trinoton luridum* Nitzsch of ducks; (3) **Boöpiidae,** a small family of biting lice on kangaroos and wallabies, confined to Australia, e.g., *Heterodoxus longitarsus* (= *H. armiferus* Paine), may also occur on dogs. The **Ischnocera** are divided into four families, among them (1) **Trichodectidae,** with antennae three-segmented, tarsi with one claw, infesting mammals only, e.g., *Bovicola bovis* (Linn.) [= *Trichodectes bovis* (Linn.)] [(= *Trichodectes scalaris* Nitzsch)] of cattle; and (2) **Philopteridae,** with antennae five-segmented, tarsi with two claws, infesting birds mainly, e.g., *Goniodes stylifer* Nitzsch, the large turkey louse, and *Lipeurus caponis* (Linn.), the wing louse of chickens.

Injury done by biting lice. The injury done by the biting lice is largely restricted to poultry, although some trouble may result when mammals are badly infested. The injury is largely due to irritation or to itching caused by the creeping insects and their incessant gnawing at the skin. This irritation causes the host to become exceedingly restless, thereby affecting its feeding habits and proper digestion; egg production in fowls

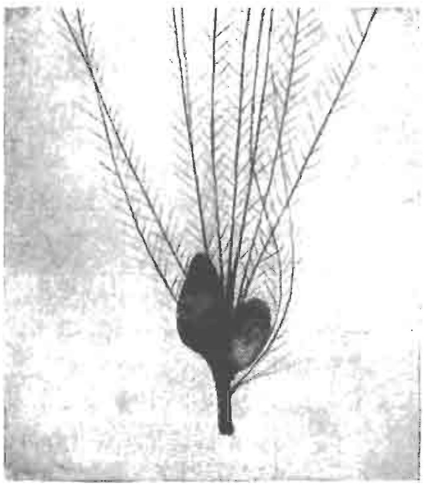


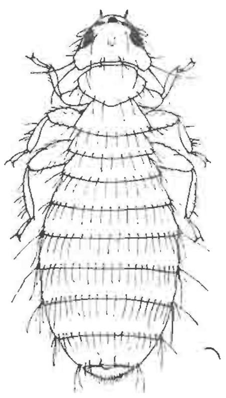
FIG. 47. Eggs of biting lice (Mallophaga) on feathers of a bird.

is greatly reduced and development retarded. A lousy flock of poultry is not a good investment. When lice are abundant uncleanliness and overcrowded conditions usually exist.

The life history of a biting louse. One of the most complete life history studies of a biting louse of birds was made by Martin[36] on the pigeon louse, *Columbicola columbae* (Linn.) (= *Lipeurus baculus* Nitzsch), family Philopteridae. She found that as many as sixty white, opaque eggs of this louse are attached to a single feather (Fig. 47). The incubation period at 37° C was normally from three to five days; at 33° C it ran from 9 to 14 days. At the latter temperature the nymphs always died in from one to six days. There are three instars, each requiring on an average slightly less than seven days, making a total of between 24 and 25 days for the complete life cycle. At 37° C the adults live usually from 30 to 40 days, the longest time being 51 days. Temperature is the chief factor in determining the length of the life cycle as well as the survival of the young.

The life cycle of *Bovicola bovis* (Linn.), the red louse of cattle (family Trichodectidae) is described by Matthysse (*loc. cit.*) as follows: at 95° F and 75 per cent humidity, the egg stage averages 5 to 9 days, first instar 6 to 10 days, second instar 4 to 10 days, third instar 4 to 9 days, preoviposition period 3 to 4 days, cycle egg to egg 22 to 42 days; maximum adult longevity was 42 days. The egg-to-egg cycle under normal conditions would probably average 29 to 30 days.

Lice infesting domestic fowls. More than forty species of lice are said to occur on domestic fowls of which seven species are commonly found on chickens. Losses due to poultry lice are most evident among the young birds, but heavy infestations on older fowls result in loss of



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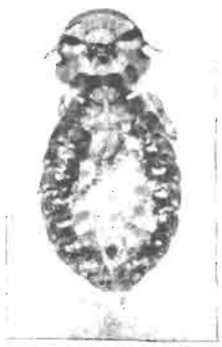

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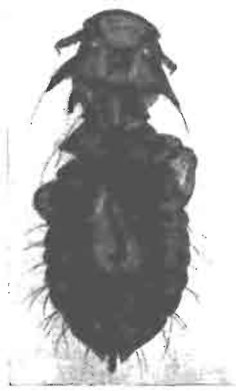

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FIG. 48. The common shaft louse of poultry, *Menopon gallinae*.
FIG. 49. The large hen louse, *Goniodes gigas*. ×10.
FIG. 50. A turkey louse, *Goniodes stylifer*. ×14.

weight, lowered egg production, and lowered vitality. Although other maladies may present similar symptoms, infested fowls are droopy, lower the wings, present an unkempt and ruffled appearance, and suffer from diarrhea. The commoner lice of chickens are (1) the "body louse," *Eomenacanthus stramineus* (Nitzsch) (= *Menopon biseriatum* Piaget), a rapidly running species occurring on all parts of the fowl; it is light yellow in color and about 2 mm in length; it lays its eggs in large clusters, particularly on the small feathers below the vent; the egg stage requires about a week and maturity is reached in about two weeks thereafter; (2) the "shaft louse," *Menopon gallinae* Linn. (= *Menopon pallidum* Nitzsch) (Fig. 48), which resembles the body louse very closely but is smaller in size, and occurs mainly on the shafts of the feathers; it is said to gain its nourishment from barbs and scales of the feathers and is therefore not so irritating as the body louse; according to Bishopp and Wood[37] it does not occur on young chickens and deposits its eggs singly, at the base of the feathers between the main shaft and the after shaft; the life history appears to require a somewhat longer time than that of the body louse; (3) the "head louse" *Cuclutogaster* (= *Lipeurus*) *heterographus* Nitzsch, a dark grayish species about 2 mm in length, infesting the head and neck of young chickens to which it is most injurious; it deposits its eggs singly on the down or small feathers about the head and requires about the same time for complete development as the first species mentioned; (4) the large hen louse or "blue bug," *Goniodes gigas* Tasch. (Fig. 49), about 3 mm in length; broad with rounded head, and smoky gray in color; it is generally distributed over the body and easily recognized; *Eomenacanthus stramineus* Nitzsch (= *Menopon stramineum* Nitzsch) is also referred to as the large poultry louse, infesting turkeys and other fowl as well as chickens; (5) the "wing louse," *Lipeurus caponis* (Linn.) (*Lipeurus variabilis* Nitzsch), also known as the "variable louse," a long slender species about 2 mm in length; the margins of the head are black, the head is large and rounded, and the general appearance is sufficiently distinct to separate it from all the other species; (6) the "fluff louse" *Goniocotes hologaster* Nitzsch, a very small and broad species about 1 mm in length, pale in color and seldom abundant; see Ancona[38] for morphology of this species; (7) the "brown louse" *Goniodes dissimilis* Nitzsch, reported for the southern United States by Bishopp and Wood; it is described by them as somewhat smaller than the large hen louse, reddish brown in color, and found on the feathers of the body.

Turkeys are commonly infested with the large (3 mm long) *Goniodes stylifer* Nitzsch (Fig. 50), which has the posterior angles of the head extended backward into long projections terminating in stylets or bristles. Another louse found on turkeys is *Lipeurus polytrapezius* Nitzsch, a long slender species measuring from 3 to 3.5 mm in length. *Eomenacanthus stramineus* Nitzsch (2.0 to 2.5 mm) occurs on turkeys also. *Ducks* and *geese* harbor a rather small-sized species, *Docophorus icterodes* Nitzsch, measuring about 1 mm in length; the head of this species is curiously expanded and rounded in front and is a darkish red; the thorax is also red with darker bands. Another species infesting ducks and geese is *Lipeurus squalidus* Nitzsch (Fig. 51), which is about 4 mm in length, with the head longer than broad; it is very slender and is light yellowish in color. Another long species infesting ducks (4 mm) is *Trinoton luridum* Nitzsch, dark grayish in color with triangular head about as long as broad. None of these species appears to become abundant enough to be of any great consequence. The common lice of the swan are *Docophorus cygni* Denny, about 1 mm in length; "in color the head, thorax and legs are bright reddish brown while the abdomen is white in the center and dark brown at the sides, the brown occupying hard-like portions at the side of each segment"; and the extremely large and common *Ornithobius bucephalus* Piaget (4 mm long). The latter is conspicuous because of its size; the body is white and quite transparent.

Pigeons are often abundantly infested with *Columbicola columbae* (Linn.) (= *Lipeurus baculus* Nitzsch), a very slender species measuring about 2 mm in length; *Goniodes damicornis* Nitzsch, a broad brownish species about 2 mm long; and *Goniocotes bidentatus* Scopoli, about 1 mm in length, whitish in color with a head rounded in front. Guinea fowls are said to harbor *Goniodes numidianus* Denny, *Lipeurus numidae*

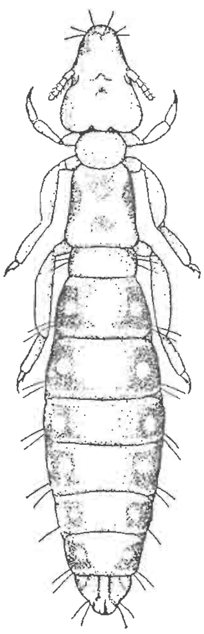

FIG. 51. A duck louse, *Lipeurus squalidus*. ×19. (Redrawn after Osborn.)

Denny, and *Menopon numidae* Gieb.; pea fowls, *Goniocotes rectangulatus* Nitzsch, *Goniodes falcicornis* Nitzsch, and *Menopon phaeostomum* Nitzsch; pheasants, *Goniocotes chrysocephalus* Gieb., *Goniodes colchicus* Denny, *Lipeurus heterographus* Nitzsch, and *Menopon fulvomaculatum* Denny.

Control of poultry lice. No remedy has given such uniformly satisfactory results in the control of the lice of all kinds on domesticated birds as has sodium fluoride (NaF), apparently first used against these parasites by Bishopp and Wood in 1917. Sodium fluoride can be obtained in two forms, a white powder or commercial form (90 to 98 per cent pure) and in fine crystals or chemically pure form. For louse control the former more finely powdered form is preferable. It retains its efficiency almost indefinitely if kept in a dry place in stoppered bottles or cans. One application generally will destroy all lice present. It may be applied in three ways, viz., the pinch method, dusting, and dipping.

The *pinch method* consists of placing on the skin of each fowl approximately ten "pinches" (amount held between thumb and forefinger) of the commercial sodium fluoride, distributed on the breast, each thigh, below the vent, on each side of the back, on the neck, on the head, and finally one sprinkled on the underside of each outspread wing. The birds, when treated, should be held over a shallow pan or newspaper in order that the excess of the chemical may be saved.

For *dusting,* the powdered sodium fluoride is sometimes mixed with three or four times its bulk of flour or talc. It is applied with a large shaker, and the feathers of the bird are ruffled as this is being done. This procedure is not as economical of material or as efficient as the pinch method, and the excess of chemical in the air is irritating to birds and operators.

Dipping in sodium fluoride solution is rapidly becoming a standard method of treatment among a large group of producers that have overcome the poultrymen's prejudice against wetting their birds. In warmer climates, such as that of California, birds may be dipped safely in almost every month of the year by choosing a warm day with little wind and completing the operations an hour or two before sundown in order that the fowls may dry thoroughly before roosting for the night. The solution should be prepared in a wooden container, avoiding contact with galvanized iron. The ordinary wooden washtub is excellent for this purpose. One ounce of the commercial sodium fluoride or two-thirds of an ounce of the chemically pure crystals should be dissolved in each gallon of tepid water. The best method for dipping the birds is to hold them with the left hand by both wings. They are then placed feet foremost in the warm dip and submerged until only the head remains above the surface. They should be held in this position from 20 to 25 seconds while the feathers are being ruffled to permit penetration of the liquid. Just before removal the head should be ducked under the surface. The birds should be held above the dip for two or three seconds to allow them to drip before releasing them. One hundred birds will use up approximately 5 gallons of dip; material should be available at the start of operations to keep the dip replenished on this basis.

The dipping method kills all lice immediately, but where the chemical is applied as a powder three or four days pass before elimination is complete. If the birds are caught and handed to the operator from 100 to 125 birds an hour can be treated by dipping or dusting and approximately 60 to 75 per hour by the "pinch" method.

Other methods. The very fact that poultry wallow in dust whenever it is available indicates a means of partly controlling the bird lice. Special box wallows, conveniently placed, broad and deep enough so that there will be room for several birds at a time, should be partly filled with fine road dust or ashes with the addition of a quantity of tobacco dust in the proportion of about six parts of the former to one of the latter. It is quite desirable to add a few handfuls of sulphur. The finer the dust the better, since it is believed by some that the dust particles enter and clog up the breathing pores of the lice. However, it is more probable that the agitation caused by the birds wallowing in the dust dislodges many of the lice and they are thus lost in the shuffle.

Powdered derris root applied as a dust, or as a dip, one-fourth ounce to a gallon of water, is said to be effective. This method is described by Wells, Bishopp, and Laake.[39]

The application of 40 per cent nicotine (Black Leaf 40) to the roosts in a thin layer with a brush or swab before the birds go to roost gives excellent results. The treatment should be repeated at an interval of 10 days

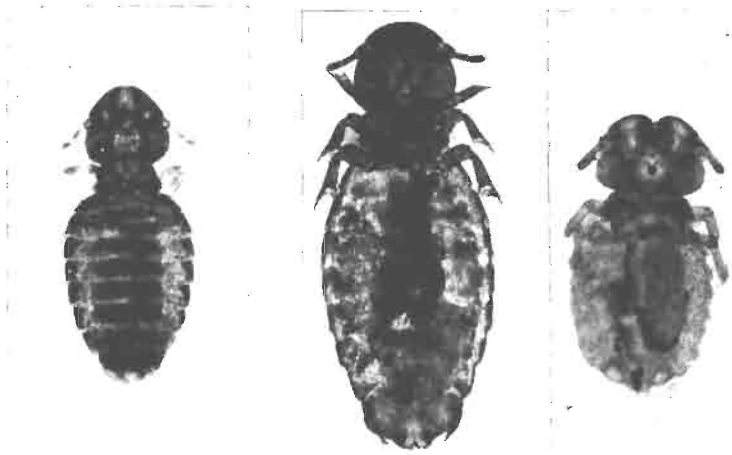

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FIG. 52. The biting ox louse, *Bovicola bovis.* ×26.
FIG. 53. Biting louse of the Angora goat, *Trichodectes hermsi.* ×22.
FIG. 54. The biting dog louse, *Trichodectes canis.* ×35.

to kill the lice which have hatched from eggs on the birds. It is recommended that the application be made when the weather is calm. Apparently this treatment acts as a fumigant. Telford[40] (1945) tested the effects of 37 louse powders on several species of chicken lice and found that practically complete control was given by DDT, 0.5 to 4.0 per cent; derris (with 5 per cent rotenone) 10 per cent; sodium fluoride, 33 per cent, cryolite, 30 per cent, and micronized wettable sulfur.

Biting lice of domesticated mammals. The biting lice of domesticated mammals are for the most part rather easily identified by their presence on a given host, as commonly not more than one species of Mallophaga is found on each species of mammalian host. *Cattle* are often heavily infested on the withers, root of tail, neck, and shoulders with *Bovicola bovis* (Linn.) (= *Trichodectes scalaris* Nitzsch) (Fig. 52), a little red louse about 1½ mm in length, definitely marked with transverse bars (ladder-like) on the abdominal segments. The biting lice are most numerous on the animals during dry, cold weather when the hair is long. Although they do not irritate the cattle as much as suckling lice do, the following noteworthy observation is made by Imes:[41]

When present in large numbers, however, they often form colonies or groups around the base of the tail, over the withers, and on other parts of the animal, and produce lesions resembling those of scab. These lesions vary in size from that of a 25-cent piece to 4 or 5 inches in diameter. The skin over these areas appears to be raised, and ringworm may be suspected; but when the lesion is manipulated the scarf skin falls off, exposing the lice grouped on the raw tissues beneath. Under such conditions the irritation is very great and the damage to the animal may be fully equal to that caused by scab.

Horses, mules, and *asses,* but horses more particularly, when poorly or irregularly groomed may suffer from two species of biting lice, *Trichodectes equi* Linn. (= *Trichodectes parumpilosus* Piaget) and *Trichodectes pilosus* Giebel; the latter according to Kellogg and Ferris[42] has not been reported from North America, although it is reported by Hall (*loc. cit.*) on horses in Michigan. *Trichodectes equi* Linn. is described thus by Osborn:[43]

. The head is decidely rounded in front, the antennae inserted well back, so that the head forms a full semicircle in front of the base of the antennae. The abdomen is more slender and tapering than in *bovis (scalaris)*. . . . The color is much as in the allied species, the head, thorax and legs being a bright reddish brown, or chestnut, and the abdomen of a dusky yellowish color, with about eight transverse dusky bands occupying the central or anterior portions of the segments and extending from the middle line a little more than halfway to the margin. They are hardly as conspicuous as in *bovis (scalaris)*.

T. pilosus Gieb. is a smaller species and the antennae are inserted well forward, almost on a line with the anterior border. Hall states that these "lice give rise to itching, and the results from this are often surprisingly unpleasant. A barn full of horses may become a pandemonium as a result of lice. The itching animals attempt to relieve the itching by rubbing and biting, other animals start to kick, presently the kicking becomes general and there is a resultant clamor and din, with a substantial element of danger to the horses and attendants." The life history is about the same as for the biting cattle lice.

Sheep may at times show severe infestations of *Trichodectes ovis*

Linn. (= *T. sphaerocephalus* Nitzsch). This species is about 1.5 mm in length, the head being somewhat rounded, as long as broad, and reddish in color; the abdomen is whitish. Because of extensive dipping operations against scab this louse has seldom had an opportunity to thrive, but we have observed that when scab disappeared and consequently no dipping was done, the biting lice appeared in troublesome numbers.

Goats are very commonly enormously infested with biting lice. Several species from goats have been described, about which there is still some confusion, but the common species is *Bovicola caprae* (Gurlt) (= *T. climax* Nitzsch). The author has found Angora goats to be heavily infested with *Trichodectes hermsi* Kellogg and Nakayama (Fig. 53),

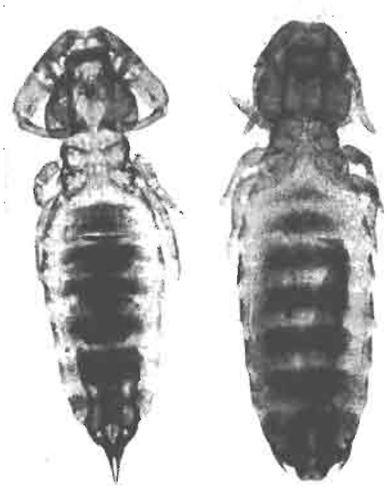


Fig. 55. A biting louse of deer, *Trichodectes tibialis*. (*Left*) male, (*right*) female. × 31.

which resembles and is probably a synonym of *T. penicillatus* Piaget, which in turn is said to be a synonym of *T. limbatus* Gerv., of *T. crassipes* Rudow, and of *T. major* Piaget. The irritation produced by the lice, particularly on Angora goats, causes the animals to rub or bite themselves so much that the mohair is matted and pulled out, resulting in considerable loss. The individual hairs are weakened by the gnawing of the lice. Sulfur dips (10 pounds wettable sulfur to 100 gallons of water) are highly recommended. Two dips, with an 11-day interval between, are recommended.

Dogs, particularly puppies, may suffer much irritation from a small biting louse, *Trichodectes canis* De Geer (= *T. latus* Nitzsch) (Fig. 54). It is a broad, short species, measuring about 1 mm in length. The author took biting lice from a dog in Berkeley, California, many years ago, which were described as *Heterodoxus armiferus* by Paine.[44] As this genus is said to be restricted to the kangaroo the incident was interesting. The later species is now considered to be a synonym of *Heterodoxus longitarsus* Piaget, which today is frequently found on dogs in California.

Cats may become heavily infested with *Felicola subrostratus* (Nitzsch) (*Trichodectes subrostratus* Nitzsch). *Guinea pigs* commonly harbor two species, *Gyropus ovalis* Nitzsch and *Gliricola porcelli* (Linné) (= *Gyropus gracilis* Nitzsch). The llama harbors *Trichodectes breviceps* Rudow. *Trichodectes tibialis* Piaget (Fig. 55) is very abundant on California deer.

Control of biting lice on mammals. As a general rule lousicides effective against sucking lice are also effective against biting lice, although the reverse does not always hold. Sodium fluoride has proved effective in the destruction of the biting lice on cattle, horses, goats, sheep, dogs (dangerous to cats), and guinea pigs. Bishopp states that "a high degree of effectiveness (90 to 100 per cent destruction) may be obtained by applying the sodium fluoride with a dust gun to the flock in a pen as the goats are driven through a chute. It does not seem to be necessary to drive the dust into the mohair especially and only a small amount—about one-third ounce per head—is necessary." Because of the toxicity of sodium fluoride its use on dairy cattle is *not recommended* not only because of possible danger to the animals but also because of milk contamination. Furman (1947, *loc. cit.*) found that *Bovicola bovis* infestations could be eliminated by a single dip in 0.15 per cent benzene hexachloride. A persistent odor is a factor to be considered if this insecticide is used.

The following suggestion by Lamson (*loc. cit.*) for the treatment of cattle lice in general is noteworthy, though perhaps of little importance:

> Of the many different measures for the control of lice on *dairy cows* and *young stock*, *raw* linseed oil gave the best results from the standpoint of economy of material and labor of application, killing all the lice but not injuring the skin and at the same time not making it necessary to thoroughly drench the cow. It has no poisonous properties. At the same time it is a logical remedy, as the lack of oiliness in the skin of the cow is a fundamental reason for her being lousy. Linseed oil can be put on at the time taken for grooming or cleaning the cows, thus doing two things in one application. . . . From four to five cows can be treated with a pint . . . treatment should be repeated at least five times. Raw linseed oil can be best applied with a brush having bristles of unequal length. Do not rub the skin too vigorously when applying the oil. Do not allow the animals that have been treated to go out in the strong sunlight until at least twelve hours after applying the oil. Do not exercise the animals after the treatment. Do not use the boiled or refined linseed oil.

Lice and taeniasis. *Dipylidium caninum* (Linn.), the double-pored dog tapeworm, is a common parasite of the dog and is occasionally found in humans, especially children. It measures from 10 to 14 inches in length, has long seed-like proglottids and an armored scolex; as its larval host it has the biting dog louse, *Trichodectes canis* DeGeer, the dog flea, *Ctenocephalides canis* (Curtis), or the human flea, *Pulex irritans* Linn. The cysticercoid stage has been experimentally produced in the louse by placing ripe crushed proglottids of the tapeworm on the skin of a dog infested with lice.

As has already been explained, the biting lice subsist on epidermal scales, skin exudations, and other matter on the skin of the animal. This habit makes it comparatively easy for the louse to become infected by swallowing "eggs." The dog, on the other hand, readily infects itself by devouring the lice (or fleas) which irritate its skin.

Persons, particularly children, while fondling louse-infested (or flea-infested) dogs may easily become infected by accidentally swallowing lice (or fleas) which contain bladder worms (larval tapeworms). This is more readily accomplished if the person is eating while fondling dogs.

BIBLIOGRAPHY

1. Ferris, G. F., 1919–35. *Contributions toward a Monograph of the Sucking Lice,* parts 1–8. Univ. ser. "Leland Stanford Junior Univ. Publ.," no. 41. and Univ. ser. "Stanford Univ. Publ. Biol. Sc.," vol. 2. nos. 2–8.

2. Ferris, G. F., 1934. "A summary of the sucking lice (Anoplura)," *Entomological News,* 45:70–74, and 85–88.

3. Murray, Andrew, 1860. "On the pediculi infesting the different races of man," *Tr. Roy. Soc. Edinburgh,* 22:567. (Cited by Osborn.)

4. Nuttall, G. H. F., 1918. "The biology of *Phthirus pubis,*" *Parasitology,* 10:383–405.

5. ———, 1917. "The biology of *Pediculus humanus,*" *Parasitology,* 10:80–185.

6. Buxton, P. A., 1939. *The Louse.* London: Edward Arnold & Co. viii + 164 pp.

7. Leeson, H. S., 1941. "The effect of temperature upon the hatching of the eggs of *Pediculus humanus corporis* DeGeer," *Parasitology,* 33:243.

8. Pierce, W. Dwight, 1918. *The Life History of the Human Louse.* Washington, D. C.: U. S. Dept. Agric., Bur. Entomol., in Proceedings of the Class Formed to Study the Entomology of Disease, Hygiene, and Sanitation. (Mimeograph copy received from author.)

9. Peacock, A. D., 1916. "Structure of the mouth parts and mechanism of feeding in *Pediculus humanus,*" *Parasitology,* 11:98–117 (1 plate).

10. Moore, W., 1918. "An interesting reaction to louse bites," *J.A.M.A.,* 71:1481–82.

11. Mackie, F. P., 1907. "The part played by *Pediculus corporis* in the transmission of relapsing fever," *Brit. M. J.,* 2:1706–09.

12. Nicolle, C. N.; Blaizot, L.; and Conseil, E.; 1913. "Etiologie de la Fièvre récurrente. Son mode de transmission par les poux," *Ann. Inst. Pasteur*, **27**:204–25.

13. Chung, Huei-Lan, and Feng, Lan-Chou, 1936. "Studies on the development of *Spirochaeta recurrentis* in body louse," *Chinese M.J.*, **50**:1181–84.

14. Ricketts, H. T., and Wilder, R. M., 1910. "Further investigations regarding the etiology of tabardillo, Mexican typhus fever," *J.A.M.A.*, **55**:309–11.

15. Nicolle, Charles; Comte, C.; et Conseil, E.; 1909. "Transmission expérimentale du typhus exanthématique par le pou du corps," *Compt. rend. Acad. d. sc.*, **149**:486–89.

16. Ricketts, H. T., and Wilder, R. M., 1910. "The transmission of the typhus fever of Mexico (tabardillo) by means of the louse *(Pediculus vestimenti)*," *J.A.M.A.*, **54**:1304–7.

17. Goldberger, J., and Anderson, J. F., 1912. "The transmission of typhus fever, with special reference to transmission by the head louse *(Pediculus capitis)*," U. S. Public Health Service, *Pub. Health Rep.*, **27**:297–307.

18. Nuttall, G. H. F., 1917. "Lice and disease," *Parasitology*, **10**:43–79.

19. Da Rocha-Lima, H., 1916. "Untersuchungen über Fleckfieber," *München. med. Wchnschr.*, **63**:1381–83.

20. Toepfer, H., 1916. "Zur Ursache and Uebertragung des Wolhynischn Fiebers," *München. med. Wchnschr.*, **63**:1495–96.

21. Foster, M. H., 1918. "Preliminary report on carbon tetrachloride vapor as a delousing agent," U. S. Public Health Service, *Pub. Health Rep.*, **33**:1823–27.

22. Moore, W., and Hirschfelder, A. D., 1919. *An Investigation of the Louse Problem*. Minneapolis: Univ. Minnesota Press, in Research Publ., vol. 8. 86 pp.

23. Moore, William, 1918. "The effect of laundering upon lice *(Pediculus corporis)* and their eggs," *J. Parasitol.*, **5**:61–68.

24. Bushland, R. C., 1948. "Insecticides for the control of lice attacking man and animals." *Proc. 4th Internat. Cong. Trop Med. & Malaria, Abstracts.* Washington, D. C.

25. Wheeler, Charles M., 1946. "Control of typhus in Italy 1943–1944 by use of DDT," *Am.J.Pub.Health*, **36**:119–29.

26. Busvine, J. R., and Buxton, P. A., 1942. "A new method of controlling the head louse," *Brit.M.J.*, **1**:464–66.

27. Florence, Laura, 1921. *The Hog Louse, Haematopinus suis Linné: Its biology, anatomy, and histology*. Ithaca: Cornell Univ. in Agric. Exper. Sta. Memoir, No. 51, pp. 641–743.

28. Bishopp, F. C., 1921. "*Solenopotes capillatus*, a sucking louse of cattle not heretofore known in the United States," *J. Agric. Research*, **21**: 797–801.

29. Lamson, G. H., Jr., 1918., 1918. "Cattle lice and their control," *Storrs Agric. Exp. Sta., Bull.*, No. 97, pp. 397–414.

30. Matthysse, John G., 1946. *Cattle Lice, Their Biology and Control.* Ithaca: Cornell Univ., in Agric. Exper. Sta. Bull., no. 832, 67 pp.

31. Hall, M. C., 1917. "Notes in regard to horse lice, *Trichodectes* and *Haematopinus*," *J. Am. Vet. M. A.*, **51**, n. s. 4:494–504.

32. Babcock, O. G., and Laake, E. W., 1942 *The Control of Cattle Lice.* Washington, D. C.: Dept. Agric., in Bur. Entomol. & Plant Quar. Circular, E 447: 1–2 revised (Mimeograph ser.)

33. Furman, Deane P., 1947. "Benzene hexachloride to control cattle lice," *J. Econ. Entomol.*, **40**:672–75.

34. ———, and Hoskins, W. M., 1948. "Benzene hexachloride in cream from cows' milk," *J. Econ. Entomol.*, **41**:106.

35. Harrison, Launcelot, 1916. "The genera and species of Mallophaga," *Parasitology,* **9**:1–156.

36. Martin, Margaret, 1934. "Life history and habits of the pigeon louse, *Columbicola columbae* (Linn.)," *Canad. Entomologist,* **66**:6–16.

37. Bishopp, F. C., and Wood, H. P., 1917. *Mites and Lice of Poultry.* Washington, D. C.: Dept. of Agric., in Farmers' Bull., no. 801, 27 pp.

38. Ancona H. L., 1935. "Contribucion al conocimiento de los piojos de los animales de Mexico, III *Goniocotes hologaster* Nitzsch," *An.Inst.Biol.,* Mexico, **6**:119–28.

39. Wells, R. W.: Bishopp, F. C., and Laake, E. W.; 1922. "Derris as a promising insecticide," *J. Econ. Entomol.* **15**:90–5.

40. Telford, Horace S., 1945. "New insecticide for chicken lice control," *J.Econ. Entomol.,* **38**:573–76.

41. Imes, Marion, 1925. *Cattle Lice and How to Eradicate Them.* Washington, D. C.: Dept. Agric., in Farmers' Bull., no. 909. 24 pp. (Revised).

42. Kellogg, V. L., and Ferris, G. F., 1915. *The Anoplura and Mallophaga of North America.* Univ. ser. "Leland Stanford Junior Univ. Publ." 74 pp. + 8 plates.

43. Osborn, Herbert, 1896. *Insects Affecting Domestic Animals.* Washington, D. C.: Dept. Agric., in Div. Entomol. Bull., no. 5 (n. s.) 302 pp.

44. Paine, J. H., 1912. "The Mallophagan genus *Heterodoxus* Le Souëf and Bullen," *Entomological News,* **23**:359–62.

CHAPTER X

GNATS (Exclusive of Mosquitoes)

Simuliid Gnats, Phlebotomus Flies, Hippelates Flies

(FAMILIES SIMULIIDAE, PSYCHODIDAE, CHLOROPIDAE)

Order Piptera

Order Diptera. The several families of gnats discussed in this chapter *are members of the Order Diptera, "the flies," which comprises some* 75,000 described species belonging to some 140 families (see Curran, 1934).[1] Many species of insects belonging to this order are involved in the transmission of important diseases of man and animals; hence the medical entomologist must be thoroughly familiar with the Diptera.

As the name implies, all of the winged members of the Order have only one pair of wings; the posterior pair is represented in nearly all species by a pair of short (often minute) knobbed organs known as balancers or halteres. In certain families, e.g., muscoid flies, there is present a pair of minute wing-like structures at the juncture of the wings with the body; these are known as squamae or allulae. Conspicuous compound eyes are present, and most species possess three simple eyes (ocelli). The metamorphosis is typically complete, consisting of the four stages—egg, larva, pupa, and imago (adult). Some species are viviparous, notably the tsetse flies. The mouth parts, as previously described, are subject to great variation although all are suctorial; many species are provided with very effective piercing stylets which enables them to "bite" fiercely and to suck blood.

Much attention must be given the larval stages because the larvae frequently invade the tissues and organs of the body of man and animals, causing myiasis (see Chapter XVII); also a wider knowledge of aquatic larvae (of which there are many species) is important in pursuing work with mosquitoes and other gnats, as well as in the study of the biology of water supplies (see Johannsen[2]).

The Diptera have a wide range of breeding habits. There are very few habitats suitable for animal life which have not been invaded by the flies. The petroleum fly, *Psilopa petrolei* Coq., as its name implies, actually passes its larval life in crude oil.

Classification of the Diptera. In the classification of Diptera, knowledge of wing venation is important (Fig. 56). The great diversity of antennal structure provides a useful series of characters, as does the arrangement of spines (chaetotaxy) on the body of certain species, such as the blowflies (see Walton[3]). The terminalia are important taxonomic structures in many Diptera.

The Diptera are usually separated into two major groups (1) **Orthorrhapha**, referring to the species in which the winged insect escapes

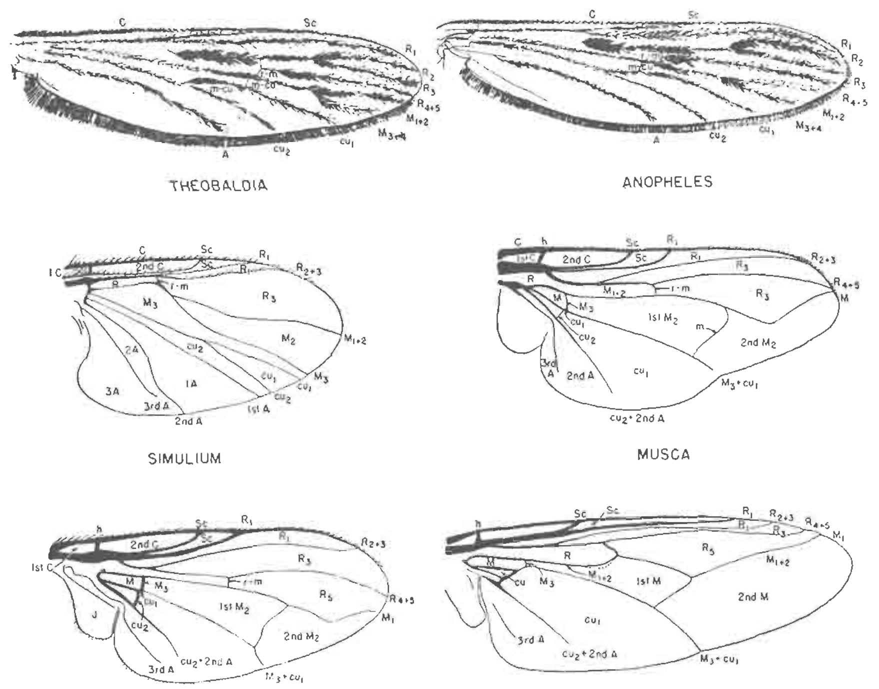


FIG. 56. Wings of Diptera. For explanation of venation, see Figure 12.

from the puparium through a T-shaped anterodorsal split, as horseflies, buffalo gnats, and mosquitoes; and (2) **Cyclorrhapha,** in which the insect escapes from the puparium through a circular opening; in fact it pushes off the anterior cap by means of pressure exerted by the bladder-like ptilinum on the front of the head of the insect, e.g., houseflies and blowflies.

For the purposes of this book the Diptera are divided into three suborders. (1) **Nematocera,** in which the antennae are filiform and many-jointed, as in mosquitoes; (2) **Brachycera,** in which the antennae are short, not filamentous, generally three-segmented, variously formed, as in horseflies; (3) **Cyclorrhapha** as described above, and with antennae, brachycerous, generally three-segmented, and frequently bearing an arista on the terminal joint; ptilinum usually present, as in houseflies and blowflies.

SUBORDER I. NEMATOCERA

Larvae have well-developed, exserted head and horizontally biting mandibles; pupa free. Antennae of imago are many-jointed, longer than the head and thorax, and the majority of the joints are usually alike; arista is lacking. Palpi are usually four- or five-jointed, pendulous. Discal cell is generally absent; cubital cell when present, widely open.

SUBORDER II. BRACHYCERA

Larvae have incomplete head, usually retractile, and with vertically biting mandibles; pupa free. Antennae of adult are shorter than thorax, very variable, generally three-segmented with last elongate; arista or style, when present, is terminal. Palpi are porrect, one- or two-segmented. Discal call is almost always present, cubital cell is contracted before wing margin or closed.

SUBORDER III. CYCLORRHAPHA

Larvae have vestigial head; pupa coarctate; antennae of adult are three-segmented with arista usually dorsal in position. Palpi are one-segmented. Discal cell is almost always present. Cubital cell is contracted or closed. Head with frontal lunule and usually with ptilinum.

*Some Families of the Order Diptera**

Suborder Nematocera

A. Mesonotum with an entire V-shaped suture........(Crane Flies) Tipulidae

AA. Mesonotal suture transverse, not V-shaped.

- B. Costa continued around the margin of the wings, though weaker behind the apex.
  - C. Wings short and broad, folded roof-like over the body when at rest, usually pointed
    (Moth Flies, Sand Flies, Owl Midges) Psychodidae
  - CC. Wings long, or if broad, the apex is very broadly rounded, always lying flat over the back when at rest.
    - D. Apical veins strongly arched............(Dixa Midges) Dixidae
    - DD. Veins straight or nearly so.
      - E. Proboscis not elongate, extending but little beyond the clypeus; wings with scales (when present) confined mostly to the fringe............................Chaoboridae
      - EE. Proboscis elongate, extending far beyond the clypeus; wings with the veins and margins with scales
        (Mosquitoes) Culicidae

* Classification adapted after Curran (*loc. cit.*).

BB. Costa ending at or near the apex of the wing.
C. Wings very broad, the posterior veins weak and poorly developed.................... (Black Flies, Buffalo Gnats) Simuliidae
CC. Wings narrow and long, the posterior veins stronger.
D. Wings lying flat over the back when at rest; metanotum short and without a longitudinal groove; femora sometimes swollen............ (Biting Midges) Ceratopogonidae
DD. Wings lying roof-like over the back when at rest; metanotum long and with a median longitudinal groove; legs long and slender................ (Midges) Chironomidae

Suborder Brachycera

A. Third antennal joint annulated; arista absent
(Horseflies, Deer Flies) Tabanidae
AA. Third antennal joint not annulated; but if annulated, with extremely long flagellum with terminal arista; squamae vestigial; normally shaped flies.. (Snipe Flies) Rhagionidae (Leptidae)

Suborder Cyclorrhapha

A. Anal cell closed very close to the wing margin; a spurious vein running obliquely between the third and fourth longitudinal veins
(Flower Flies) Syrphidae
AA. Anal cell usually shorter; no spurious vein.
B. Second antennal segment with a longitudinal seam along the upper outer edge extending almost the whole length; posterior calli definitely formed by a depression extending from behind the base of the wings to above the base of the scutellum
(Calypteratae: Muscoidea)
C. Metascutellum developed, appearing as a strong convexity below the scutellum; hypopleura (meron of Snodgrass) with strong bristles................................ (Tachina Flies) Tachinidae
CC. Metascutellum weak or absent, or if developed there is only hair on the hypopleura.
D. Oral opening and mouth parts very small; hypopleura with abundant long hair.
E. Scutellum extending far beyond the base of the metanotum; metascutellum never developed
(Robust Botflies) Cuterebridae (Oestridae)
EE. Scutellum very short; metascutellum usually strongly developed; palpi usually large
(Botflies) Oestridae
DD. Oral opening normal; hypopleura with a row of bristles or only short sparse hair.
E. Hypopleura with a row of bristles.
F. Apical cell strongly narrowed apically
(Blowflies) Metopiidae*

* Includes the Sarcophagidae, part of the Tachinidae and Muscidae of Williston's Manual. (Curran.)

FF. Apical cell not at all narrowed apically
Muscidae*

EE. Hypopleura with fine, short hair or bare....Muscidae

BB. Second antennal segment rarely with a well-developed dorsal seam; the posterior calli not differentiated (except in *Gasterophilus*): squamae small (Acalyptratae).

C. Mouth parts vestigial, sunken in a very small oral pit
(Horse Botflies) Gasterophilidae (Oestridae)

CC. Mouth parts well developed; the oral opening large. Ocellar triangle large; fifth vein with a distinct curvature near the middle of the discal cell
(Frit Flies) Chloropidae (Oscinidae)

FAMILY SIMULIIDAE

(Buffalo Gnats—Black Flies)

Characteristics. The family Simuliidae, consisting of over 600 species,[4,5] includes insects commonly known as buffalo gnats, black flies, and turkey gnats. They are small (1 to 5 mm long) bloodsucking flies,

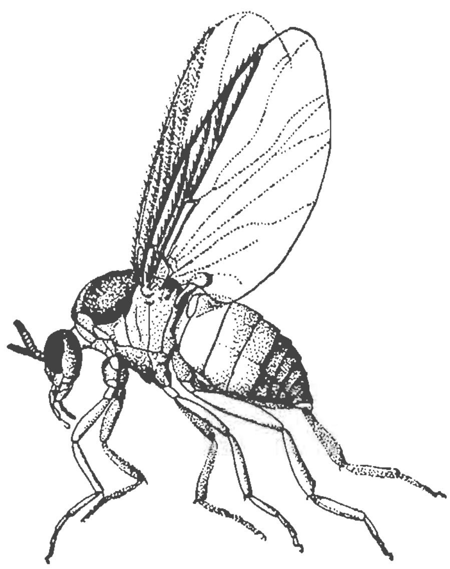


FIG. 57. A buffalo gnat, *Eusimulium pecuarum*. (Redrawn after (Garman.)

with mouth parts blade-like and piercing in the female but more or less rudimentary in the male. They are stout bodied and usually almost black in color. The thorax presents a strong development of the scutum and reduction of prescutum resulting in a prominent hump Fig. 57). The antennae are ten- to eleven-jointed; the eyes of the female are distinctly separated (in the male they are usually close together and prominent,

* Includes the Scatophagidae (Cordyluridae), Anthomyidae, and those Muscidae (of the Williston Manual) lacking hypopleural bristles. (Curran.)

i.e., holoptic); ocelli absent; palpi four-jointed; wings broad and iridescent, with distinct alulae, the venation is characterized by a strong development of the costal veins (Fig. 56). Buffalo gnats often occur in enormous swarms during late spring and early summer in hilly sections where swiftly flowing streams provide well-aerated water for larval development. They are particularly abundant in the north temperate and subarctic zones.

Although running water, such as that of shallow mountain creeks is favored as a breeding place, the gnats may also breed in roadside ditches of more slowly moving water. The gnats may be found in abundance a mile or two from water, probably in search of food.

Life history. Eggs to the number of 350 to 450 per female are deposited in masses at the water surface of aquatic plants, logs, and water-splashed rocks. Comstock says he has often watched the gnats hovering over the brink of a fall where there was a thin sheet of swiftly flowing water, and has seen them dart into the water, and out again. At such times he has always found the surface of the rock more or less thickly coated with eggs, and has no doubt that an egg is fastened to the rock each time a fly darts into the water. The shiny eggs are at first creamy white, changing to almost black.

The time required for hatching is from 5 to 30 days, depending on temperature and motion of water. In running water at a temperature of 20° to 22° C the incubation period is four to five days.[6] The newly emerged larva attaches itself by means of a caudal sucker to any submerged object, such as a stone or log, and is kept from being washed away by a silken thread. Movement from place to place is achieved by shifting anchorage. In some favorable location, such as the riffles on the downstream side of an old log partially damming a little stream, there may be thousands of these tiny black spindle-shaped larvae. The larvae as well as the pupae are provided with gill filaments and usually remain submerged or partly so. The larval period of some species is said to require but three to five weeks. The food of the larvae consists of small crustacea, protozoa, and algae. The larval period for *Simulium ornatum* Meigen is given by Smart[7] as 7 to 10 weeks when temperatures in the stream ranged between 9° to 15.5° C. At the end of the larval period the organism spins a rough reddish-brown basket-like cocoon in which pupation takes place. These cocoons are firmly attached to shallowly covered objects such as rocks.

The pupal period is quite short in some species, requiring not over five or six days; while in other species it lasts three to four weeks. Temperature influences this stage, i.e., cooler weather retards the emergence of adults. Smart gives the pupal period for S. *ornatum* Meig. as 3.75 days at a constant temperature of 21° C. In some species there is continual breeding from early spring to late autumn with overlapping generations; in others there is evidently one sudden brood coming fairly early in the spring with stragglers following. They overwinter in either the larval or egg stage. The life history, egg to adult, ranges from 60 days to 15 weeks and over, depending on species and climatic conditions.

Larvae. The light brown to black larvae are cylindrical, twelve-segmented, slightly thinner in the mid-region, and when fully grown are from 10 to 15 mm in length (Fig. 58A). The posterior end of the body is provided with a toothed disk-like sucker, composed of two modified parapodia. The anterior proleg is also modified into a prehensile toothed disk. By means of these organs the larvae move from place to place with a looping motion. They attach themselves to rocks or other supports in

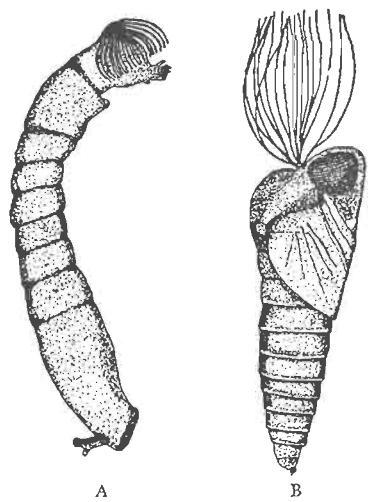


FIG. 58. (*A*) larva and (*B*) pupa of *Simulium;* latter removed from cone-shaped cocoon. (Redrawn after Lugger from Washburn.)

the water by means of the posterior sucker, the hooks of which they insert into the network of silken threads produced by secretions from the salivary glands with which they they covered the substratum. The larvae may hang from threads produced in similar fashion or travel along their length.

Although the larvae are provided with a well-developed tracheal system, and nine pairs of spiracles may be observed, these are not *open*, and respiration is carried on by means of gills, recognized as branched retractile structures located dorsally on the last abdominal segment. The fan-shaped filamentous structures located on the head are for the purpose of creating a current, by means of which food is drawn into the mouth.

Pupae. When the larvae are ready to pupate, each spins a crude pocket-like cocoon open at the upper end. The pupae are provided with respiratory filaments attached anteriorly to the dorsal portion of the thorax. The filaments are often quite numerous and because of their constancy in number in a given species may be of diagnostic value (Fig. 58B).

Classification. The family Simuliidae is divided into four genera according to Dyar and Shannon,[8] viz.:—the three genera with the radius setose on its entire length: (1) *Parasimulium,* with the radius joining the costa at the middle of the costal vein, the radial sector forked, antennae ten-jointed; (2) *Prosimulium,* with the radius joining the costal vein far beyond its middle, the radial sector with a long fork, the second hind tarsus without dorsal incision, the front usually broad; (3) *Eusimulium,* with the radius also joining the costal vein far beyond its middle, but with the radial sector simple, the hind basitarsus produced or not produced apically, the second hind tarsus with or without dorsal incision and less than twice the width of the basitarus, and the front narrowed; (4) *Simulium,* the radius bare between the stem vein and the base of the radial sector, the radial sector simple, the hind basitarsus produced apically, the second hind tarsus with dorsal incision and less than twice the width of the basitarsus. Smart (1945, *loc. cit.*) recognizes six genera, viz., *Parasimulium, Prosimulium, Cnephia, Austrosimulium, Gigantodax,* and *Simulium.*

Common species. Only one species is given under the genus *Parasimulium* by Dyar and Shannon, namely *P. furcatum* Malloch from Humboldt County, California. *Prosimulium fulvum* Coq. is a widely distributed species in the mountainous regions of the west and along the Pacific coast from Alaska to California; *P. hirtipes* (Fries) is said to be confined to the region east of the Mississippi and north of the Carolinas. It rarely attacks man, according to Dyar and Shannon, though it is known on occasion to bite rather severely; it is not considered an important pest of livestock.

Eusimulium pecuarum (Riley) [= *Cnephia pecuarum* (Riley) according to Smart] is known as the southern buffalo gnat and is a great scourge of livestock as well as of man in the Mississippi Valley. During the height of the gnat season in the early spring, work on plantations is often greatly handicapped because of the annoyance to work animals. *E. minus* D. and S. [= *Cnephia minus* (D. and S.)] is a widely distributed western and Pacific coast species with the type locality indicated as Yosemite. It resembles the buffalo gnat but is smaller and darker.

Simulium pictipes Hagen occurs in the eastern United States. It is said to be an inoffensive species. *Simulium vittata* Zetterstedt is widespread throughout North America and is a common species in Europe. It attacks man and livestock freely. *Simulium occidentale* Townsend and *Simulium meridionale* Riley, known as "turkey gnats," are also common and widespread species in North America but particularly in the southern states, where the gnats appear in late spring following the buffalo gnat. They attack poultry, biting the combs and wattles, and are said to cause symptoms similar to "cholera," hence the name "cholera gnat." *Simulium venustum* Say is the black fly. It is one of the most annoying and widespread species. It torments fishermen and campers in New England and Canada. The gnats occur in the greatest numbers during June and July.

The bite. There is perhaps no other insect of equal size that can inflict so painful a bite as can the buffalo gnat. It is a day biter and is rarely found indoors. The mouth parts are of the dipteron type (similar to those of the horsefly), consisting of six blade-like lancets.

Human beings as well as domesticated animals are viciously attacked. The eyes, ears, nostrils, wrists, and all exposed parts of the body of man are subject to attack. The extreme pain, intense itching, and the resultant local swelling, and occasional severe complications, indicate the presence of an active venom. Losses due to the bite of this fly are considerable and are estimated variously by stockmen. Myriads of these gnats appear after the spring floods of the Mississippi River and its tributaries. Horses, mules, and cattle are often killed in a few hours by the venomous bites and loss of blood. This sudden appearance of the gnats is explained by the large accumulation of eggs that have been washed into the area during floods. These eggs do not hatch until the next flood causes movement of the water, for it is in flowing and well-aerated water that the larvae hatch and develop rapidly.

Simulium columbaschensis Fabr. is the famous Columbacz gnat of middle and southern Europe. Patton and Evans[9] (p. 193), citing Ciurea and Dinulescu, report that in 1923 two immense swarms of this fly invaded southwest Roumania in May, June, and July, causing the death of 16,474 domestic animals, including cattle, horses, pigs, sheep, and goats. Large numbers of deer, foxes, and hares, as well as other wild animals, were killed at the same time, according to these authors.

Relation to disease. Owing to the vicious intermittent bloodsucking habits of the buffalo gnats, it has long been suspected that they might play a role in the transmission of disease.

With the startling report of Dr. Louis W. Sambon[10] in 1910 ascribing the transmission of pellagra to a buffalo gnat, the study of the Simuliidae gained new impetus. The gnats, however, have no relation to this disease. Goldberger[11] states (1914) that pellagra is neither infectious nor contagious, that it is essentially of dietary origin, and that the disease does not develop in those who consume a mixed, well-balanced, and varied diet. The most important disease transmitted by Simuliid flies is ***onchocerciasis*** (onchocercosis), a disease of natives of certain parts of

Africa, Mexico, and South and Central America. It is caused by filarial worms, *Onchocerca volvulus* (Leuckart) (= *O. caecutiens* Brumpt), which require black gnats as intermediate hosts. The female worm measures from 35 to 50 cm in length, the male 20 to 40 mm. These worms occur in conspicuous subcutaneous nodular tumors located primarily on the trunk, shoulders, and head of infected persons. Several adult worms and numerous larvae (produced viviparously) usually occur in each tumor. Serious involvements of the eye occur, often resulting in complete blindness evidently due to migration of the larvae. Strong[12] points out that from a clinical standpoint the association of ocular disturbances (such as photophobia) with the disease is emphasized by the high percentage of failing vision and blindness in a locality where at least 95 per cent of the populations are infected with the parasite and have demonstrable nodules. The student should particularly consult a treatise on ocular onchocerciasis by Hisette.[13]

Blacklock,[14] working in Sierra Leone, has shown that when the microfilariae are taken up with the bite of *Simulium* flies, they migrate from the fly's stomach, finding lodgment in the thoracic muscles, where further development takes place, and then travel to the head and finally to the labial structures of the fly, where escape is made when the fly bites, and infection of the human being is accomplished. Development in the gnat requires about 10 to 14 days. The time required for development in the human from inoculation by the bite to maturity of the worm in the skin nodule is said to be about four months. The species of fly observed in these experiments was *Simulium damnosum* Theob., a widely distributed black gnat of tropical Africa. Bequaert[15] points out that of 57 species of Simuliidae described from the Ethiopian region, five only are definitely reported as biting people, but all must be looked upon with suspicion as possible carriers of onchocerciasis, although *S. damnosum* Theob. and *S. neavei* Roubaud are the only ones positively incriminated.

Strong[16] investigated the disease in Guatemala, where he reports it is characterized by the formation of nodular tumors (containing adult filariae) situated on the scalp, although they may occur on the trunk and in the pelvic region. Vargas states that three species of black gnats are reported to be vectors of the infection in Guatemala and Mexico (in the states of Oaxaca and Chiapas, Mexico, according to Dampf[17] and Mazzotti[18]). *Simulium ochraceum* Walker is said by Vargas (verbal communication) to be the principal vector in these areas; others of less importance being *Simulium callidum* Dyar and Shannon (= *Simulium mooseri* Dampf) and *Simulium metallicum* Bellardi. Vargas has recently reported the presence of onchocerciasis in Venezuela, the vector being unknown.

Bovine onchocerciasis. It has been pointed out by Steward[19] that bovine onchocerciasis is of considerable economic importance in Australia, that the "worm nodules" due to *Onchocerca gibsoni* Cleland and Johnston cause losses to the state of Queensland estimated at £500,000 per annum. The work done by Steward in England with *Onchocerca gutturosa* Neumann proved that this latter parasite is transmitted by *Simulium ornatum* Meigen. He showed that the filariae are conveyed from the skin of the cow with the bite of the fly, thence to the mid-gut of the insect, where development commences. Ten days after ingestion they reach the "sausage" stage in the thoracic muscles, and by the nineteenth to twenty-second day they migrate forward to the head ready for emergence from the proboscis when the gnat feeds again.

Leucocytozoön infections of poultry. The name Leucocytozoön was given to certain Sporozoa found in the blood of birds by Danilewsky in 1890, and in 1895 Theobald Smith discovered a *Leucocytozoön* in the blood of turkeys; this parasite was named *Leucocytozoön smithi* by Volkmar. In 1932 Skidmore,[20] working in Nebraska, reported the successful transmission of this parasite by *Simulium occidentale* Town. In 1938 Johnson *et al.*[21] reported transmission through the agency of *Simulium nigroparvum* (Twinn). Johnson and his associates state that when the organism is taken into the stomach of the fly gametes are formed, macrogametes being clearly observable as well as the zygote.

An important infection of both domestic and wild ducks, caused by the protozoön parasite, *Leucocytozoön simondi* Mathis and Leger, 1910 (= *L. anatis* Wickware 1915), occurs in Michigan according to O'Roke[22] who proved that the disease is transmitted by the black fly, *Simulium venustum* Say. Other vectors are *S. occidentale* Town and *S. nigroparvum* Twinn. The development of the organism within the body of the gnat is cyclico-propagative, resembling closely the life cycle of the plasmodium of malaria in anopheline mosquitoes. O'Roke states that the asexual cycle in the duck requires 10 days and the sexual cycle in the gnat not to exceed five days, with field evidence that it may be as short as two days or less.

Black-gnat control. Knowing the breeding habits of black gnats, it will be appreciated that larval control is a difficult task. The writer has repeatedly recommended that, in the vicinity of communities, streams in which these insects are breeding should be kept as free as possible from debris, including submerged roots and dipping branches of overhanging trees. Although this can be done, prevailing winds may nevertheless bring swarms of gnats from a distance; but the removal of debris from streams lessens the opportunity for them to disposit their eggs. Old logs lying crosswise of a small stream are a particular menace because shallow waterfalls are thus usually produced and afford ideal breeding places for the gnats. The fact that the larvae tend to congregate in masses in certain parts of streams in which they are breeding makes it possible to remove them in quantities when they are once located.

The newer repellents such as indalone, Rutgers 612, and dimethylphthalate are useful but require frequent application according to Vargas.[23] Experimental work in larval control now being carried out by several investigators indicates that a good measure of success can be achieved by the use of either DDT or TDE as applied in tests for the control of chaoborid gnats. Romaña and Abalos report successful control of larvae in streams by the use of gammexane in wettable mixtures at the rate of 700–1,000 mg per 1/sec of water. *Ann. d Inst. med. Regional,* Univ. Nacional de Tucuman, Argentina, Dec., 1948.

R. D. Glasgow, New York State entomologist, reports economical and successful control of black flies (adults) by applying a DDT aerosol fog by means of a helicopter. About one quart of a 15 per cent solution of DDT in a petroleum solvent was used per acre.

FAMILY PSYCHODIDAE

(Moth Flies–Phlebotomus Flies)

Family Psychodidae. The family consists of several hundred species and includes the tiny gnats known as owl midges, moth flies, and *Phlebotomus* flies (sand flies). The ovate, usually pointed wings and the body are densely covered with hairs, whence the name moth flies. In the *Psychoda* flies the wings when at rest lie roof-like over the abdomen. Because of the faint transverse venation the wings appear to have only longitudinal veins. The antennae are usually fairly long and from twelve- to sixteen-segmented.

The family may be divided into two subfamilies, (1) Psychodinae, known as moth flies or owl midges, which are not bloodsuckers and whose wings are held roof-like over the body; and (2) Phlebotominae, the sand flies, of which the females are bloodsuckers; they are never aquatic.

Psychoda flies. Several species of *Psychoda* are commonly found in great numbers about sewage disposal plants, cesspools, and washbasins in bathrooms where the larvae may develop in sink drains in spite of hot water and soap. A common Pacific coast species is *Psychoda pacifica* Kincaid, brown in color, measuring from 2 to 2.3 mm in length, and moth-like. Although the flies of this genus are not bloodsuckers, they may breed in such numbers in the filter beds of sewage disposal plants, as the author has personally observed, as to constitute a real annoyance to neighboring households. The corresponding eastern species, *Psychoda alternata* Say is known as the "sprinkling sewage filter fly." These gnats may become annoying in the house, where they originate in the surface of the gelatinous material in sink and bathroom drain traps. Their life cycle is quite short, ranging from 8 to 24 days depending on temperature, the average is about two weeks (see Mallis and Pence[24]).

The adult gnats may be controlled by spraying their resting places with a 5 per cent DDT emulsion or suspension at the rate of 1 qt to 250 sq ft of area. Caution must be exercised in the use of DDT emulsions for the control of the larvae because of damage of filter growth by the solvent. Simpson[25] states that suspensions with a wetting agent eliminates adverse effects. He recommends addition of a quantity of DDT at one time equal to 1 p.p.m., based on daily flow, and retarding filter operation ½ to 1 hour for contact time and repeating the operation every two weeks.

Phlebotomus (= Flebotomus) flies. The genus *Phlebotomus* (= *Flebotomus*) of the family Psychodidae comprises many species of small-sized hairy gnats or midges measuring from 1.5 to about 4 mm in length. They are commonly known as sand flies. They differ from the *Psychoda* flies, their nearest relatives, in that the wings are held upward and outward so that the costal margins form angles of about 60 degrees with each other and with the body. The second longitudinal vein branches at a considerable distance from the base of the wing. The females alone have piercing mouth parts and are bloodsuckers; some species are said to feed on cold-blooded animals such as lizards and snakes;[26,27] others feed on field mice; no doubt most of them feed on a variety of warm-blooded animals including man. The males suck moisture from any available source and are even said to suck up sweat from humans. These gnats are active only at night when there is little or no wind, seeking protection by day in shelters both out of doors in crevices, caves, and among vegetation, and within buildings where they hide in dark corners. Their noiseless flight, which is commonly described as weak, is usually in short so-called "hops" when they are disturbed; however, in longer flights their progress is slow and steady and can be followed with the eye. Townsend[28] points out that "deep canyons, free from wind and dimly lighted, are especially adapted to them."

Life history of sand flies. Hertig,[27] who has had wide experience with *Phlebotomus* flies, points out that "the breeding-places of sand flies are notoriously hard to find and have been demonstrated in the case of only a few species. They are typically under stones, in masonry cracks, in stables, poultry houses, etc., in situations combining darkness, humidity, and a supply of organic matter which serves as food for the larvae. In no case is the breeding-place aquatic." To situations mentioned above may be added hollow trees and animal burrows. The eggs are deposited in small batches. The incubation period is said to be from 9 to 12 days (Whittingham and Rook).[26] The minute whitish larvae have long anal spines; the mouth parts are strongly mandibulate; they feed on organic debris, excrement of lizards, etc.; larval stage with four instars requires from four to six weeks; the pupa, which is naked, requires about 10 days for development. The females usually lay eggs in five to seven days after a blood meal. The egg-to-egg cycle requires from 7 to 10 weeks; however, Hertig (*loc. cit.*) points out that where there are cold winters *Phlebotomus* flies are subject to a diapause in the fourth larval instar which may last from several weeks to nearly a year. This, Hertig states, was not the case in Peruvian sand flies (Fig. 59).

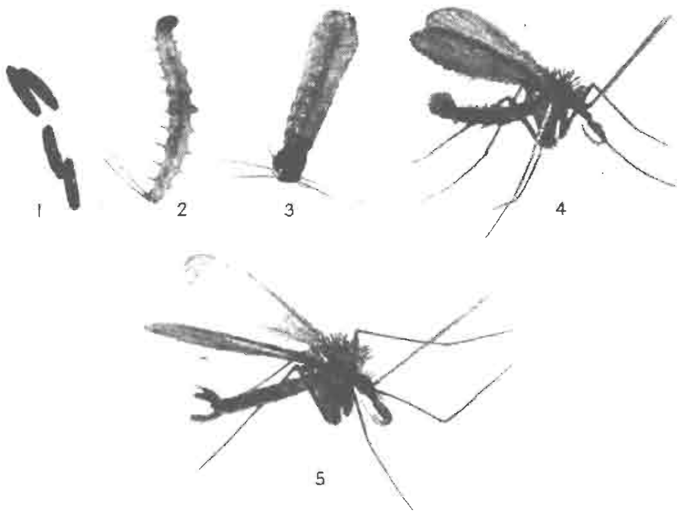


FIG. 59. Life cycle of *Phlebotomus verrucarum*. (1) eggs, (2) larva, (3) pupa (*P. noguchii*), (4) adult female, (5) adult male. (Adapted after Hertig.)

Carrión's disease (bartonellosis) is known also under the names of its two chief clinical forms, namely *Oroya* fever, a severe anemia, and usually fatal, and *verruga peruana,* benign, and recognized by a characteristic cutaneous nodular eruption. "The disease is endemic in a narrow strip along the Pacific slope and in certain other parts of the Peruvian Andes between latitudes 6 and 13 degrees south and at altitudes usually between 800 and 3,000 meters," affecting parts of Peru, Colombia (Patiño-Camargo[29]), Bolivia, Chile, and Ecuador. The etiological agent is *Bartonella bacilliformis* Noguchi, a minute rod-like or coccoid organism, "which occurs in or on the red cells and intracellularly in a number of organs, notably in the endothelial cells of lymph glands."

The transmission of verruga was attributed to *Phlebotomus* flies by Townsend[30,31] in 1913. He described the vector as *Phlebotomus verru-*

carum and attributed a human infection to this species. The author was one of those who examined a British sailor presumably successfully infected experimentally (see Hertig, *loc. cit.*, p. 155). Although there was some evidence of old cutaneous lesions no definite diagnosis of verruga was made. Although the "Verruga Expedition Report"[32] does not consider Townsend's evidence as conclusive, the latter again presented his arguments in favor of the *Phlebotomus* fly theory[33] (1915). Based on careful field studies in the "verruga zone" Noguchi and Shannon, *et. al.*,[34] substantially confirmed Townsend's contention that *Phlebotomus verrucarum* is a vector of verruga and added another species *Phlebotomus noguchii* Shannon as a probable vector. Hertig (*loc. cit.*) indicates some of the taxonomic difficulties encountered in these investigations and cities the experimental work of Battistini 1927–1931, whose publications were not available to the author, in which positive transmission to a monkey (positive in 18 days) was made through the agency (by bites) of wild sand flies of a mixed lot of *P. verrucarum* and *P. noguchii.* In 1939 Hertig in a personal communication to the author stated that he had discovered a clue to the mechanism of infection in that the verruga organism was recovered in pure culture from the extreme tip of the proboscis, the piercing stylets themselves being thoroughly infected. In the communication, Hertig stated that this infection was apparently not acquired from a blood meal. Transmission of verruga to monkeys under these circumstances was reported. The source of the proboscis infections is unknown; however, the fact that the proboscides of male sand flies, which are not bloodsuckers, are also thus infected, points to liquid sources other than the usual blood meal of the females.

Sand fly fever, also known as pappataci fever, three-day fever, Mediterranean dengue, or summer influenza, is a seasonal (hot, dry periods) febrile *virus* disease of short duration occurring in the *Phlebotomus* infested regions of the Mediterranean, South China, parts of India, Ceylon, and parts of South America. It is a nonfatal infection. Commonly a large percentage of a ship's company on shore leave, even for one night only during the proper season, may be stricken with sand fly fever within two or three days after leaving port. The incubation period in the insect is from 7 to 10 days. There is some evidence that the infection may be transmitted transovarianly, hence the insect may be itself a reservoir of the infection. *Phlebotomus papatasii* Scopoli in the Mediterranean region becomes infective six to eight days after an infecting blood meal; the virus is present in man's blood for 24 hours prior to onset and for the first 24 hours of the disease, hence the infective period for the sand fly is limited to this length of time. After a person has been bitten by an infected fly the incubation period is usually from three to six days; thus in the Canal Zone the disease is known as Panama six-day fever; in the

Dutch East Indies it is known as van der Scheer five-day fever, and in some parts of India as seven-day fever. *Phlebotomus minutus* Rond. *P. perniciosus* Newst., *P. papatasii* Scopoli, and no doubt other species are vectors of the virus.

Leishmaniasis is caused by various species of parasitic protozoa belonging to the genus *Leishmania,* round or oval intracellular bodies, which develop flagellate herpetomonad stages in the gut of insects. Leishmania infections are variously known. *Kala-azar,* dumdum fever, or tropical splenomegaly is a visceral leishmaniasis, traceable to *Leishmania donovani* (Laveran et Mesnil) which localizes in the reticulo-endothelial cells. It is a widespread disease, occurring in all countries on the shores of the Mediterranean, south Russia, India, China, Manchuria, northern (Mediterranean) and equatorial Africa, eastern Brazil and other parts of South America. There is progressive enlargement of the spleen and later of the liver. As the disease progresses the skin becomes grayish in color whence the name "black disease." It is regarded as frequently fatal, death resulting within a few weeks in acute infections and in from two to three years in chronic cases. A case of exogenous origin at the Marine Hospital, Baltimore, Md., is reported by Lewis and Spicknall.[35] Various species of bloodsucking arthropods have been suspected as being vectors. The low susceptibility of laboratory animals made progress difficult, but with the discovery that hamsters were highly susceptible to the infection rapid progress was made. Patton and Hindle[36] (1927), as well as Young and Hertig[37] and Napier, Knowles, and Smith before them (1925), observed that the Leishmania bodies underwent development in the intestinal track of *Phlebotomus* flies after being ingested. Flagellation is said to take place in the mid-intestine. The cycle in the gnat is completed in from four to five days. Evidence of infection by the insect is very meager, though Shortt and Smith, *et al.*[38] (1931), report successful transmission by the bite of *Phlebotomus argentipes* Annandale and Brunetti, believed to be the Indian vector. Other species, notably *Phlebotomus chinensis* Patton and Hindle in China, and *P. sergenti* Parrot in northern Africa, are believed to be vectors.

Transmission by the bite of the fly is doubted by Southwell and Kirshner.[39] These investigators point out that the bite of the sand fly causes an irritation and the person bitten scratches the bite, thus crushing and killing the infected insect. The leptomonads, the infective forms, do not invade the mouth parts in either *P. argentipes* A. and B., or *P. chinensis* P. and H. Thus infection by the bite is believed to be unlikely, though infection as the result of crushing infected flies on the skin appears to be possible. Recently Gupta,[40] 1948, reporting on researches in India states that sand flies kept alive on raisins after an infective meal, showed the phenomenon of "blocking," and kala-azar could be transmitted almost invariably to hamsters and human volunteers by the "bite" of the blocked sand fly.

Oriental sore. Oriental sore, also known as Bagdad or Delhi boil, is a cutaneous leishmaniasis caused by *Leishmania tropica* (Wright); it has a wide distribution in Mediterranean areas, Palestine, Arabia, Asia Minor, Iraq, India, French Congo, and other parts of the world. It is not necessarily coextensive with kala-azar. In oriental sore the leishmanias inhabit the skin and do not invade the viscera. In a series of papers by Adler and Theodor,[41] evidence is advanced to incriminate *Phlebotomus papatasii* Scopoli. These workers found a cyclical development of the *Leishmania* in the fly requiring from 8 to 21 days. Infection of a human was accomplished by rubbing the infected mid-gut of the insect into the scarified skin, and flies were reinfected successfully from the sore thus produced. Infection by the bite did not occur. Adler[42] (1948) reports that transmission by the bite of *P. papatasii* has been achieved. He reports that *P. sergenti* Parrott is the most effective vector: The infection is apparently perpetuated by sand flies inhabiting the burrows of various species of gerbils, and this explains outbreaks among groups of human beings passing through uninhabited regions.

American mucocutaneous leishmaniasis (naso-oral), also known as espundia or uta, is widely distributed in South and Central America. The causal agent is *Leishmania brasiliensis* Vianna. The horrible disfiguring effects of this infection are shown by Goldman,[43] who points out that it is the mucosal involvement which is so characteristic of American cutaneous leishmaniasis. Once the infection gets into the mucocutaneous junction, it destroys all types of tissues including cartilage and bone. Goldman states that "prevention of cutaneous leishmaniasis in the endemic areas appears to be a well nigh hopeless affair. It is likely the incidence could be reduced considerably by definite knowledge of vectors, animal reservoir, and transmission. If the foreigner in the endemic area wishes some simple practical advice, the use of modern repellents, insecticides, adequate fine-mesh screening, and the other measures for the maintenance of careful hygiene of the skin in the tropics, and early attention to indolent lesions on exposed parts of the body may be suggested." Various species of *Phlebotomus* flies are looked upon with suspicion based on experimental evidence, among these are *P. intermedius* Lutz and Neiva, *P. pessoai* Coutinho, and *P. longipalpus* Lutz and Neiva.

Species of Phlebotomus. According to Addis[44] (1945) there are six species of sand flies in the United States, none which have received much attention; namely (1) *Phlebotomus vexator* Coq. and (2) *P. limai* Fonseca, both widely distributed in the southern states; (3) *P. diabolicus* Hall from Texas, reported to feed on humans and experimentally on

Syrian hamsters; (4) *P. texanus* Dampf, also from Texas; (5) *P. stewarti* Mangabeira and Galindo, from California, collected in wild rodent burrows in arid fields; (6) *P. anthophorus* Addis, another Texas species, but found also in Mexico. Addis reports that this species feeds only in the morning, has never been observed after sundown, has a definite preference for rabbit blood, but will feed on hamsters in the laboratory; it is apparently not annoying to man.

Investigators concerned with the numerous species of South American *Phlebotomus* flies will need to consult the many excellent contributions of Mangabeira, particularly his 1942 contribution,[45] which contains a valuable bibliography.

Phlebotomus control. Marett[46] suggests, as means of controlling Phlebotomus, the facing of rock walls, removal of heaps of stones, blocking of holes affording shelter for the gnats, encouragement of gardening (cultivation of ground), planting of embankments with native aromatic plants, etc. As sand flies are highly susceptible to pyrethrum sprays, painting screens lightly each evening with a mixture of one part pyrethrum extract concentrate (20 to 1) and 20 parts of light lubricating oil has been recommended. Because of the minute size of these gnats ordinary mesh screens do not exclude them; therefore, a 0.0334-inch mesh is recommended. Repellents such as "612" and dimethylphthalate applied to skin and clothing give quite a bit of protection. Hertig[47] states that the habits and life history of sand flies render them peculiarly vulnerable to DDT. They make short flights with relatively long pauses on entering or leaving any shelter, breeding place, or structure—a procedure repeated nearly every night of their adult life. Any surface capable of retaining residual DDT, and on which sand flies must alight, may become a lethal barrier. House spraying gives virtually complete protection indoors. Hertig states that, in Peru, house spraying, combined with treatment of outdoor shelters such as stone walls, reduced the local sand fly population to negligible numbers, an effect which persisted up to a year and a half. The short flight range limits infiltration, and the long life cycle delays recovery of a depleted sand fly population.

FAMILY CERATOPOGONIDAE

(Biting Midges—Punkies)

Characteristics. The Ceratopogonidae are very small (0.6–5.0 mm in length), slender, bloodsucking gnats (males do not bite) resembling the nonbiting midges belonging to the family Chironomidae, to which family they are commonly ascribed. In their biting habits they resemble the black flies (Simuliidae) and are frequently mistaken for them. Among the 20 or more genera comprising the family, three will serve the purpose of this section, namely *Culicoides* (Fig. 60), *Ceratopogon*, and *Leptoconops*, commonly known as "punkies," "no-see-ums," "sand flies," or "black gnats." The wings, which are narrow, with few veins and no scales, may be clear or hairy and are folded flat over the abdomen when at rest; alulae slender. The larvae are aquatic or semiaquatic, and are found in fresh, brackish, or salt water; they may live in moist earth or in tree holes. A key to the North American *Culicoides* numbering thirty species is given by Root and Hoffman.[48]

Culicoides canithorax Hoffm., *C. melleus* Coq., and *C. dovei* Hall constitute a serious economic problem in the summer resort areas of the Atlantic coast, particularly about fresh-water inlets and tide-water pools

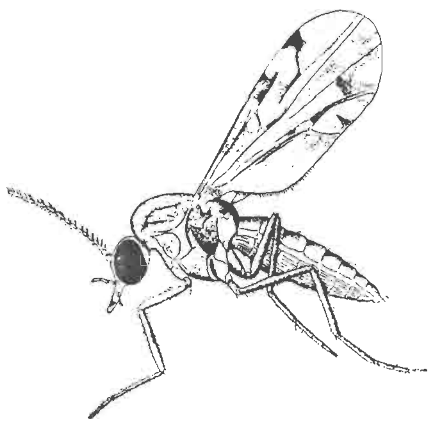


FIG. 60. Female *Culicoides* sp. (After Dampf.)

where these midges are most numerous. Dove, Hall, and Hull[49] report that the larvae are found in decaying humus of the densely shaded areas at the edges of the grass marshes of the upper Atlantic coast. The period required for development appears to last from 6 to 12 months according to these authors. The larvae and pupae of *Culicoides guttipennis* Coq. have been taken from tree holes of the live oak in Mississippi (Hinman).[50] *Culicoides diabolicus* Hoffman is a fiercely biting species in Mexico.

Ceratopogon stellifer Coq. is reported to be a severe biter in Arizona and New Mexico. *Leptoconops torrens* Townsend and *Holocopops kerteszi* Kieff. constitue a serious pest in California. The former is known as the "valley black gnat" and the latter as the "Bodega black gnat." Both species bite viciously, feeding on man, domestic animals, and birds. The bite usually produces a transient swelling, which may become vesicular, rupture, and produce a open lesion which may exude moisture for weeks. Itching resulting from the bite is intense. The following account of the two species is from a recent publication (1948) by Smith and Lowe.[51] The valley black gnat passes the larval stage in clay or clay-adobe soils situated along the western side of the Sacramento Valley and in isolated deposits in the San Joaquin Valley. The Bodega black gnat passes the larval stage in damp sand with some organic matter, at or just above high-tide level in the mouths of fresh water streams which enter Bodega and Tomales bays (California). A study of the morphology of the larvae showed that the "Leptoconops group" is widely divergent from typical ceratopogonids and warrants the acceptance of the subfamily name Leptoconopinae (Enderlein 1936).

Adults of the Bodega black gnat occur from mid-April until early October, in a continuous flow from the soil. Females may feed as many as four times. Males form large swarms, dancing in the lee of windbreaks. Eggs are laid on the surface of damp sand where the salt concentration is about 640 p.p.m. The larval stage lasts for 8 to 10 months. Pupation occurs in the sand; pupae wriggle to the surface and stand vertically before the adult emerges. The pupal period is 8 days. Adult females captured in the field lived a maximum of 11 days, with blood meals. Males do not feed; they lived a maximum of 4 days after capture in the field.

Adults of the valley black gnat occur for 4 to 6 weeks, beginning usually in the middle of May. Females feed only once; males do not feed. Unfed gnats lived only 6 hours in captivity; with a blood meal, females lived a maximum of 5 days. The larvae occur in clay-adobe soils at a depth of 15 to 30 inches. Egress and entrance is dependent upon the drying and cracking of the soil. The larval period is at least two years in length. Larvae spend the summers in immobile estivation. If the soil does not crack on schedule, the mature larvae enter a diapause. Some evidence is given to indicate that larvae may diapuase for at least three years. Larvae are found in summer in soil with a moisture content of 17 to 20 per cent, a salt concentration of 400 p.p.m., a pH of 9.6, and a temperature of 65° to 68° F. Methods of identifying breeding grounds consist of trapping adults as they emerge from the soil, and washing larvae from the soil.

Culicoides austeni Carter, Ingram, and Macfie has been reported by Sharp[52] as an intermediate host of *Acanthocheilonema perstans* (Manson) [*Dipetalonema perstans* (Manson)]. The embryos of this worm are found in the peripheral circulation both by day and by night. Sharp has observed that diurnal periodicity is the more common. In the vast majority of cases it is said to be nonpathogenic. It is primarily equatorial and African in distribution, though it occurs also in British Guiana and in New Guinea. Sharp has shown that the microfilariae undergo metamorphosis in the body of *Culicoides austeni* Carter, Ingram, and Macfie, increasing to three times their original length before they appear in the proboscis of the insect. The cycle in the fly requires seven to nine days. Sharp states that it is probable that *Culicoides grahami* Austen will also prove to be a natural carrier. Buckley[53] found that *Culicoides furens* Poey transmits the filarial worm, *Mansonella ozzardi* (Manson), in the Antilles, and Dampf[54] points out that this species of gnat is widely distributed along the coast of the Gulf of Mexico and the Caribbean Sea.

Control. Because of the variety of breeding places involved, it becomes necessary first of all to determine these for the species giving trouble. Some species breed in salt marshes, in which case dikes, tide gates and other salt-marsh control devices may be employed. Other species breed in mud and plant debris along the margins of fresh-water streams or ponds, and for the control of these removal of vegetation and

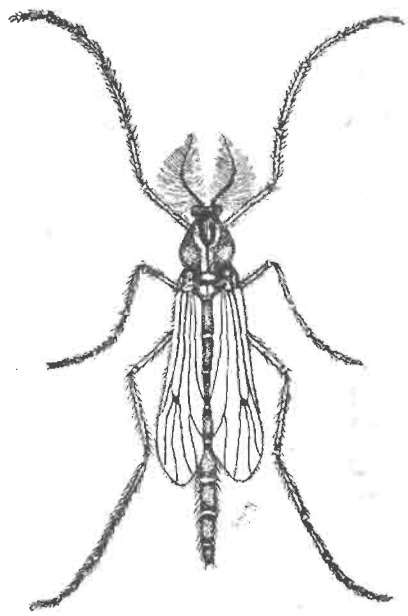


FIG. 61. A male midge (Chironomidae), commonly mistaken for a mosquito. ×12. (After Osborn.)

channelization are suggested, or filling in low ground may be helpful. Still other species breed in holes in shade trees, in which case holes should be treated with DDT or creosote, or otherwise made unfavorable for breeding.

Punkies may be excluded from the house, according to the United States Bureau of Entomology and Plant Quarantine, by applying a mixture of 1 part pyrethrum extract concentrate (20 to 1) and 20 parts lubricating oil (S.A.E. 5) to window screens by means of a brush or rag. This mixture, it is reported, will exclude the gnats from the house for 24 to 48 hours. Some of the newer insecticides would, no doubt, prove useful.

FAMILY CHIRONOMIDAE

(Midges)

Family Chironomidae. Although the midges are commonly mistaken for mosquitoes they bear little resemblance to them on closer examination. In the midges the proboscis is short and not adapted for piercing, the palpi are three- or four-jointed, the wings are bare or haired. The antennae are plumose in the males and sparsely haired in the female (Fig. 61). Midges are widely distributed and may often be extremely abundant in the vicinity of standing water, since the larvae are aquatic. Occasionally great swarms of these insects hover in the air toward evening and produce a distinct humming sound. They are attracted to light in great numbers. The family is a very large one, comprising nearly 2,000 species.

Many of the larvae are red in color, hence the name "blood worm." The larvae are worm-like (Fig. 62) and move by creeping or looping;

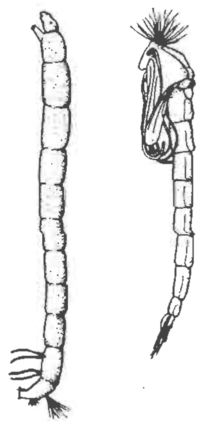


Fig. 62. Larva (*left*) and pupa (*right*) of a chironomid gnat (midge). (Larva redrawn after Needham and pupa redrawn after Grünberg.)

they have a closed (apneustic) respiratory system, hence need not come to the surface for air as do mosquito larvae. Most species are bottom feeders and scavengers in habit. While occurring most abundantly in shallow shore water with vegetation such as reeds and tule, they have been taken at great depths from the bottom of lakes; some species breed in swiftly flowing water.

Burrill[55] in a very interesting paper on the swarming of midges states that under the conditions observed they swarm an hour or two in the early morning sunlight, then mostly stop flying and rest on such objects as grass, the underside of tree leaves, tree trunks, and porch screens. They may fly throughout a cloudy day. He also observed a late summer swarm of *Chironomus plumosus* Burrill fly until after midnight. (See also Gibson,[56] 1945.)

FAMILY DIXIDAE

(Dixa Midges)

Dixa midges are usually placed in the family Culicidae (mosquitoes) and are designated as a subfamily, Dixinae; however, for the purposes of this work they are separated from the mosquitoes. They resemble mosquitoes in wing venation but are almost devoid of hairs and scales; and the proboscis, although somewhat projecting, is not fitted for piercing. This family is mentioned here particularly because the larvae are frequently mistaken for those of *Anopheles,* being commonly found in similar situations and also because the adults resemble and are related to the true mosquitoes. Dixa larvae are usually seen at the surface of water among vegetation and debris, moving in a horizontal U-shaped position. The student is referred to O. A. Johannsen's[57] "North American Dixidae," and H. G. Dyar and R. C. Shannon's "Some New Species of American Dixa, Meigen."

FAMILY CHAOBORIDAE

(Chaoborid Gnats)

Family Chaoboridae. This family is usually regarded as a subfamily (Chaoborinae) of the Culicidae, mosquitoes, but for the purposes of

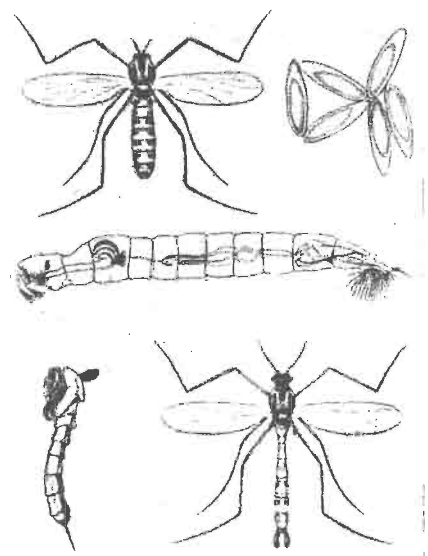


FIG. 63. A chaoborid gnat, *Chaoborus astictopus,* showing life cycle. (*Top left*) female gnat. (*top right*) eggs, (*lower left*) pupa, (*lower right*) male gnat, (*middle figure*) larva.

this book the less commonly recognized family rank is used. The members of this family are not bloodsuckers, and the gill-breathing larvae, which live in deep water, are almost transparent and are seen with some difficulty, except when in motion, even in fairly clear water, hence their name "phantom larvae." The tiny lead-colored cigar-shaped eggs are deposited in great numbers on the surface of still water, such as ponds, lagoons, lakes, etc. The eggs soon sink to the bottom. The incubation period is less than 24 hours. The larvae grow slowly during the summer, reaching approximately full growth by winter, remaining thus through the winter, and pupating in the early spring. The pupal stage requires about two weeks. The pupae quickly come to the surface, where the gnats literally "pop" out of the pupal skins, balance on the water momentarily, and then fly shoreward. *Chaoborus astictopus* D. and S. (= *C. lacustris* Freeborn) (Fig. 63) on which the foregoing life history is based is a distinct nuisance along the shores of Clear Lake, California.[59] Lindquist[60] (1948) in an informal statement reported tests made with TDE (dichloro-diphenyl-dichloroethane) which is less toxic to fish than DDT, an important matter in the control of aquatic insects in large bodies of water. He sprayed a small lake with an emulsion of TDE, and some 85 per cent of the chaoborid larvae were killed. The dosage was about one part of active ingredient to 45 million parts of water. The material disseminated through the water to a depth of about 20 feet.

FAMILY CHLOROPIDAE

(Hippelates Flies–Eye Gnats)

Hippelates flies. These flies are members of the family Chloropidae (Oscinidae), commonly known as frit flies. Unlike the gnats discussed above in this chapter, all Nematocera, the Chloropidae have short aristate antennae. The members of the genus *Hippelates* are as a rule very small flies (1.5 to 2.5 mm in length); they are frequently called "eye gnats" or "eye flies" because they have a liking for lachrymal secretions, also sebaceous secretions, pus, and blood. They may be distinguished by the presence of a distinct curved, shining black apical or subapical spur on the hind tibiae. They are extraordinarily persistent and if brushed away will quickly return to continue engorging themselves. They are nonbiting; however, the labellum is provided with spines which apparently act as cutting instruments capable of producing minute multiple incisions, likely to assist pathogenic organisms carried by the insects in gaining a foothold (Graham-Smith[61]). The flies are easily mistaken for the pomace fly, *Drosophila*. The larvae of most of the Chloropidae live in grass and other plants (stem maggots); however, those of the genus *Hippelates* develop in a wide variety of material such as decaying vegetable and animal matter.

Relation to conjunctivitis. *Siphunculina funicola* de Meyere is known as the "eye-fly" of India, Ceylon, and Java and is believed to be responsible for the spreading of conjunctivitis in these countries. Roy 1928[62] gives a chart which shows that the seasonal prevalence of this fly in Assam coincides closely with epidemic conjunctivitis.

Hippelates flies have long been looked upon with suspicion in certain parts of the southern United States as possible vectors of a form of conjunctivitis commonly known as "sore eye," "pinkeye," etc. At a meeting of the Entomological Society of Washington, held October 11, 1894, E. A. Schwarz[63] presented notes on *Hippelates pusio* Loew in the southern states. He stated that it was particularly abundant in Florida and annoying to man and animals, and that it is attracted to eyes and to the natural openings of the body as well as to infected wounds. In an article in *Insect Life* (1895), Schwarz[64] throws much suspicion on Hippelates flies as vectors of "sore eye."

For a number of years, at least since 1912, there have been numerous cases of catarrhal conjunctivitis apparently of the follicular type in the Coachella Valley of California where a veritable pest of Hippelates flies flourishes in season. Nowhere else in California are there such enormous numbers of these flies and nowhere else in the state do as many cases of so-called pinkeye exist. Several papers dealing with this subject were published following investigations in the Coachella Valley.[65-67]

At the time of the Coachella investigations the flies were identified as *Hippelates flavipes* Loew. In a letter dated May 19, 1927, J. M. Aldrich writes, "The species which you sent from Coachella Valley, California, is *pusio* Loew. It is the same species which was identified as *flavipes* Loew by Malloch, Proc. U.S.N.M., 46, 1913, p. 245. His variety *pusio* on page 246 is a different form. The true *flavipes* of Loew, is the one described by Malloch on page 243, as *nitidifrons* new species, as I have ascertained by examining the types in the Museum of Comparative Zoology at Cambridge, Mass. The earlier literature is somewhat uncertain since the time of the original description, but probably your species is the same one that has been referred to as *flavipes* when mentioning its annoying habits." In the same letter Aldrich also writes, "In the Proceedings of the California Academy of Sciences, Vol. 4, 619, Townsend described *Oscinis collusor* from Lower California which he said was reported to cause irritation of eyes of travelers and the 'mal de ojo' of natives. I examined his types in the Academy shortly before their destruction by fire in the spring of 1906 and found that they belonged to the genus *Hippelates*, and it is quite probable that the species is *pusio*." The writer has traced the Coachella Valley species through to the Mexican border at Mexicali.

The literature dealing with the Oscinidae as vectors of conjunctivitis has been reviewed with great care by Graham-Smith.[68] This review indicates a paucity of experimental evidence but a large amount of circumstantial evidence involving flies as spreaders of conjunctivitis in Egypt, the West Indies, India, Ceylon, Java, and the United States.

Relation to yaws. As pointed out in the first chapter, flies have for many years been suspected as vectors of yaws (framboesia tropica), and some experimental evidence has been advanced from time to time; however, the evidence collected by Kumm[69] (1935) in Jamaica with *Hippelates pallipes* Loew is most convincing. Kumm, as well as others, has shown that it is relatively easy to demonstrate motile *Treponema pertenue* Castellani in the "vomit drops" of eye gnats after they have fed on infectious lesions of yaws. He found, however, that the spirochetes were presumably digested in the mid-gut and hind-gut of the gnats very soon after they were ingested, none being seen after an interval of two days. There was no evidence of cyclical development.

The gnats receive the infection most readily by feeding on available primary lesions which exude fresh infected serum with large numbers of spirochetes. Inoculation is effected mechanically, i.e., the unchanged spirochetes are deposited in "vomit drops"[70] when infected gnats feed on exuding serum from wounds, excoriated areas, or susceptible surfaces. The manner in which the gnats receive the infection and their general feeding habits are well described by Kumm, Turner, and Peat.[71]

Bovine mastitis. Sanders[72] reports investigations at the Florida Agricultural Experiment Station which incriminate *Hippelates* flies (species not given) as well as the housefly, *Musca domestica*, as vectors of bovine mastitis. *Hippelates* flies were seen to hover around the natural openings of calves, yearlings, pregnant heifers, and lactating cows. They fed on lachrymal fluid, fatty body secretions, milk droplets accidentally spilled, and on secretion at the tip of the teats of animals in herds where mastitis has prevailed. Exposure tests were made with flies feeding alternately on infected material and the teat orifice; also the teat orifice was exposed to flies taken directly from premises where mastitis prevailed. "Mastitis developed in each of the experimental animals by the exposure technique employed."

Life history of Hippelates pusio Loew. This species has a wide distribution in the southern United States where the winters are mild. The adult flies are present throughout the year in the Coachella Valley (California) and are particularly annoying during two periods, i.e., March, April, May, and August, September, October. During June, July, and early August the gnats are not abundant on account of extreme heat, when the daily temperatures range well above 100° F. During the peak months the adults are noticeable early in the morning and late in the afternoon and then in deep shade, such as densely planted shrubbery, in date gardens, and in the shade of the house. The fluted, distinctly curved eggs are about .5 mm in length (Fig. 64). They are deposited on decaying organic matter of wide range. The incubation period is about three days. The larvae feed on a great variety of decaying organic matter including excrement, provided the material is rather loose and well aerated. According to Burgess (verbal communication) the larvae will not develop naturally in closely compacted soil or putrid material, neither will they breed naturally in excrement unless it is mixed with loose earth. The larval stage under optimum conditions requires about 11 days. The larvae may remain in this stage during the winter. Pupation takes place

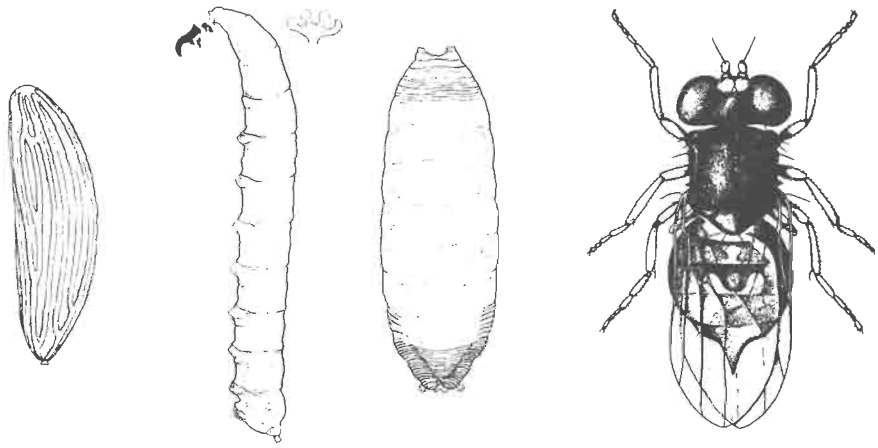


Fig. 64. *Hippelates pusio*. Egg; larva, showing cephalo-pharyngeal skeleton and anterior spiracular process; pupa; adult fly. (After Herms and Burgess, except adult fly which is redrawn after D. G. Hall.)

close to the surface of the material in which the larvae develop. The pupal stage requires about six days, giving a total of about 21 days from egg to adult fly. Except for overwintering adults, the first flies emerge from the pupae of the overwintering larvae during late February and early March, when the first great wave of the insects appears, as noted above. Experiments performed by Hall[73] show that the larval stage averaged about 11.4 days on human excrement, 8.7 days on dog manure, and about 17 days on decaying oranges. Burgess[71] points out that the majority of Hippelates gnats are bred in soil that is (1) light and friable (well drained), (2) freshly plowed (i.e., plowed not over three weeks before), and (3) contains abundant humus or vegetable matter (cover crops, manure).

Classification. The *Hippelates* flies possess a stout and distinct tibial spur, which, with the following characters, will assist in the identification of this genus: cephalic bristles short, weak, and not conspicuous; ocellars very short, erect, and convergent or cruciate; fronto-orbital hairs minute, slightly reclinate; no interhumeral bristles present (Sabrosky[75]). The color of the body in the *pusio* group is black with a yellowish trace at the base of the wings; the thorax is shiny black. Sabrosky places the following species in the *pusio* group: *H. pusio* Loew, *H. flavipes* Loew, *H. pallipes* (Loew), *H. robertsoni* Sabr., *H. bishoppi* Sabr., *H. bicolor* Coq., and *H. collusor* Tns. The *plebejus* group, in which the thorax is more or less pollinose, with at least prealar and prescutellar areas of pollen, comprises the following species: *H. plebejus* Loew, *H. dorsalis* Loew, *H. nobilis* Loew, and *H. proboscideus* Will. The *dissidens* group, according to Sabrosky, comprises smaller dark pollinose species, with legs chiefly black, proboscis short and fleshy, hind tibial spur small. The following species belong to this group: *H. dissidens* (Tucker), *H. hermsi* Sabr., *H. microcentrus* Coq., *H. montanus* Sabr., *H. convexus* Loew, and *H. particeps* Becker.

Control of Hippelates flies is difficult and involves a combination of measures, such as *trapping*, with the use of finely chopped liver as bait; *sanitation*, i.e., removal of garbage, manure piles, refuse heaps, and decaying vegetable matter; also *cultural methods* such as light disking.

Tests made with DDT ground treatment in the form of water emulsion sprays and dusts proved unsatisfactory, and thermal aerosols gave only temporary relief. Other insecticides, such as chlordane, $C_{10}H_6Cl_8$, are being tested.

Much more information concerning the breeding habits and behavior of Hippelates flies is needed to provide a sound basis for permanent control.

BIBLIOGRAPHY

1. Curran, C. H., 1934. *The Families and Genera of North American Diptera.* New York: The Ballou Press. 512 pp.

2. Johannsen, O. A., 1933–37. *Aquatic Diptera,* parts 1–3. Ithaca: Cornell Univ., in Agric. Exper. Sta. Memoirs, nos. 164, 177, and 205.

3. Walton, W. R., 1909. "An illustrated glossary of chaetotaxy and anatomical terms used in describing Diptera," *Entomological News,* 20:307–19.

4. Smart, John, 1945. "The classification of the Simuliidae (Diptera)," *Tr. Roy. Entomolog. Soc.,* London, 95:463–532.

5. Vargas, L.; Palacios, A. Martinez; and Najera, A. Diaz; 1946. "Simulidos de Mexico; Datos sobre sistematica y morphologia. Descripcion de nuevos subgeneros y especias," *Rev. d. Inst. Salub. y Enferm. Trop.,* (Mexico). 7:101–92 (25 plates).

6. Wu, Yi Fang, 1931. "A contribution to the biology of *Simulium* (*Diptera*)." Papers of Michigan Acad. Sc., Arts & Letters, 13:543–99.

7. Smart, John, 1934. "On the biology of the black fly, *Simulium ornatum* Mg. (Diptera, Simuliidae)," *Proc. Roy. Physical Soc.,* 22:217–38.

8. Dyar, Harrison G., and Shannon, Raymond C., 1927. "The North American two-winged flies of the family Simuliidae," *Proc. U. S. Nat. Museum*, 69:1–54 (Publ. no. 2636).

9. Patton, W. S., and Evans, A. M., 1929. *Insects, Ticks, Mites and Venomous Animals of Medical and Veterinary Importance. Part 1.* Croydon: H. R. Grubb Ltd. x + 786 pp.

10. Sambon, L. W., 1910. "Progress report of investigations of pellagra," *J. Trop. Med.*, 13:271–87, and 305–19.

11. Goldberger, J., 1914. "The cause and prevention of pellagra," U. S. Public Health Service, *Pub. Health Rep.*, 29:2354–57.

12. Strong, R. P., 1938. "Onchocerciasis in Africa and Central America." *Am. J. Trop. Med.* 18 (Supp.): 1–57.

13. Hissette, Jean, 1938. "Ocular Onchocerciasis," *Am. J. Trop. Med.*, 18 (Supp.): 58–90.

14. Blacklock. D. B., 1926. "The development of *Onchocerca volvulus* in *Simulium damnosum*," *Ann. Trop. Med. & Parasitol.*, 20:1–48 (4 plates).

15. Bequaert, J. C., 1938. "The black-flies, or Simuliidae, of the Belgian Congo," *Am. J. Trop. Med.*, 18 (Supp.): 116–36.

16. Strong, Richard P., 1931. "Onchoceriasis in Guatemala," *Science*, 73:593–94.

17. Dampf, Alfonso, 1931. "Los simulidos transmisores de la Onchocerciasis en los Estados de Oaxaca y Chiapas," *Medicina, México.* 40:753–61.

18. Mazzotti, Luis, 1948. "The epidemiological aspects of Onchocerciasis in Mexico," *Proc. 4th Internat. Cong. Trop. Med. and Malaria (Abstracts)*, Washington D. C.

19. Steward, J. S., 1937. "The occurrence of *Onchocerca gutturosa* Neumann in cattle in England, with an account of its life history and development in *Simulium ornatum* Meig.," *Parasitology*, 29:212–18 (1 plate).

20. Skidmore, L. V., 1932. "*Leucocytozoön smithi* infection in turkeys and its transmission by *Simulium occidentale* Townsend," *Zentralbl. f. Bakt.*, 125:329–35.

21. Johnson, E. P.; Underhill, G. W.; Cox, J. A.; and Threlkeld, W. L.; 1938. "A blood protozoön of turkeys transmitted by *Simulium nigroparvum* (Twinn)," *Am. J. Hyg.*, 27:649–65 (3 plates).

22. O'Roke, Earl C., 1934. *A Malaria-like Disease of Ducks Caused by Leucocytozoön anatis Wickware.* Ann Arbor: Univ. Michigan in School of Forestry and Conservation Bull., no. 4. 44 pages + 5 plates.

23. Vargas, Luis, 1948. "Nota sobre biología y control de los Simúlidos." *Proc. 4th. Internat. Con. Trop. Med. and Malaria (Abstracts)*, Washington, D. C.

24. Mallis, Arnold, and Pence, R. J., 1941. "The Pacific drain fly in homes," *J. Econ. Entomol.*, 34:586–87.

25. Simpson, R. W., 1948. "DDT emulsion may damage filter growths," *Sewage Works Engineering*, 19:23–24.

26. Whittingham, H. E., and Rook, A. F., 1923. "Observations on the life history and bionomics of *Phlebotomus papatasii*," *Brit. M. J.*, 2:1144–51 (2 plates).

27. Hertig, Marshall, 1942. "Phlebotomus annd Carrión's disease," *Am. J. Trop. Med.*, 22 (Supp): 1–81.

28. Townsend, Charles H. T., 1913. "A Phlebotomus, the practically certain carrier of verruga," *Science*, 38:194–95.

29. Patiño-Camargo, Luis, 1939. "Bartonellosis en Colombia," *Rev. Méd. de Bogotá*, 7:303–25.

30. Townsend, Charles H. T., 1913. "The transmission of Verruga by *Phlebotomus*," *J.A.M.A.* 61:1717–18.

31. ———, 1913. "Human case of Verruga directly traceable to *Phlebotomus verrucarum*," *Entomological News*, 25:40.

32. Strong, R. P.; Tyzzer, E. E.; Brues, Charles T.; Sellards, A. W.; and Gastiaburu, J. C.; 1913. "Verruga peruviana, Oroyo fever, and uta: Preliminary report of the first expedition to South America from the Department of Tropical Medicine of Harvard University," *J.A.M.A.*, 61:1713–16.

33. Townsend, Charles H. T., 1915. "Two years' investigation in Peru of verruga and its insect transmission," *Am. J. Trop. Dis. & Preventive Med.*, 3:16–32.

34. Noguchi, H.; Shannon, R. C.; Tilden, E. B.; and Tyler, J. R.; 1929. Etiology of Oroyo fever: The insect vectors of Carrion's disease. *J. Exper. Med.*, 49:993–1008 (3 plates).

35. Lewis, J. L., Jr., and Spicknall, C. G., 1948. "Visceral leishmaniasis (kala-azar), *J. Trop. Med.*, 28:551–54.

36. Patton W. S., and Hindle, E., 1927. "The development of Chinese Leishmania in *Phlebotomus major* var. *chinensis* and *P. sergenti*," *Proc. Roy. Soc.*, London, *ser. B.* 101:369–90.

37. Young, C. W., and Hertig, M., 1927. "The development of flagellates in Chinese sand flies *(Phlebotomus)* fed on hamsters infected with *Leishmania donovani*," *Proc. Soc. Exp. Biol. & Med.*, 23:611–15.

38. Shortt, H. E.; Smith, R. O. A.; Swaminath, C. S.; and Kishman, K. V.; 1931. "Transmission of Indian kala-azar by the bite of *Phelbotomus argentipes*." *Indian J. M. Research*, 18:373–75.

39. Southwell, T., and Kirshner, A., 1938. "On the transmission of leishmaniasis," *Ann. Trop. Med. & Parasitol*, 32:95–102.

40. Gupta, P. C. Sen, 1948. "Researches on kala-azar in India, 1938–48," *Proc. 4th Internat. Cong. Trop. Med. & Malaria (Abstracts)*, Washington, D. C.

41. Adler, S., and Theodor, O., 1929. "Attempts to transmit *Leishmania tropica* by bite: The transmission of *L. tropica* by *Phlebotomus sergenti*," *Ann. Trop. Med. & Parasitol.*, 23:1–18 (1 plate).

42. Adler, S., 1948. "Cutaneous leishmaniasis," *Proc. 4th. Internat. Cong. Trop. Med. & Malaria (Abstracts)*, Washington D. C.

43. Goldman, Lèon, 1947. "Types of American cutaneous leishmaniasis—Dermatological aspects," *Am. J. Trop. Med.*, 27:561–84.

44. Addis, C. J., 1945. "*Phlebotomus (Dampfomyia) anthophorus*, n. sp., and *Phlebotomus diabolicus* Hall from Texas." *J. Parasitol.*, 31:119–27.

45. Mangabeira, O., Filho, 1942. "Contribuição ao estudo dos Flebotomus: Descriçao dos machos de 24 novas espécies," *Mem. Inst. Oswaldo Cruz.*, 37:111–218 (148 figuras).

46. Marett, Capt. P. J., 1913. The Phlebotomus flies of the Maltese Islands. *Roy. Army Med. Corps J.*, 20:162–71. (*Abstracts in Rev. Applied Entomol.*, 1 (ser. B):27–29.)

47. Hertig, Marshall, 1948. "Sand flies of the genus *Phlebotomus*—A review of their habits, disease relationships and control," *Proc. 4th. Internat. Cong. Trop. Med. & Malaria (Abstracts)*, Washington, D. C.

48. Root, F. M., and Hoffman, W. A., 1937. "The North American species of *Culicoides*," *Am. J. Hyg.*, 25:150–76.

49. Dove, W. E.; Hall, D. G.; and Hull, J. B.; 1932. "The salt marsh sand fly problem," *Ann. Entomolog. Soc. Amer.*, 25:505–22 (3 plates).

50. Hinman, E. H., 1932. "Notes on Louisiana Culicoides (Diptera Ceratopogonoidae)," *Am. J. Hyg.*, 15:773–76.

51. Smith, Leslie M., and Lowe, Homer, 1948. "The black gnats of California," *Hilgardia* (Calif. Agric. Exper. Sta.), 18:157–83.

52. Sharp. N. A. D., 1928. "Filaria perstans: Its development in *Culicoides austeni*," *Tr. Roy. Soc. Trop. Med. & Hyg.*, 21:371–96.

53. Buckley, J. J. C., 1933. "A note on the development of *Filaria ozzardi* in *Culicoides furens* Poey," *J. Helminthol.*, 11:257–58.

54. Dampf, Alfonso, 1936. "Los Ceratopogónidoes o Jejenes (Insecta, Diptera, Fam. Ceratopogonidae) como Transmisores de Filarias," *Medicina, México*, 16:227–33.

55. Burrill, Afred C., 1913. "Notes on Lake Michigan swarms of chironomids; quantitative notes on spring insects." *Bull. Wisconsin Nat. Hist. Soc.*, 11:52–69.

56. Gibson, N. H. E., 1945. "On the mating swarms of certain Chironomidae (Diptera)," *Tr. Roy. Entomolog. Soc. London*, 95:263–94.

57. Johannsen, O. A., 1923. "North American Dixidae," *Psyche*, 30:52–58.

58. Dyar, H. G., and Shannon, R. C., 1924. "Some new species of American Dixa," *Insec. Insci. Mens.*, 12:193–201.

59. Herms, W. B., 1937. *The Clear Lake Gnat.* Berkeley: Univ. Calif., in *Agric. Exper. Sta. Bull.*, no. 607. 22 pp.

60. Lindquist, A. W., 1948. "Remarks." *Proc. Calif. Mosq. Control Assoc.*, Feb. 1948, p. 22.

61. Graham-Smith, G. S., 1930. "The Oscinidae (Diptera) as vectors of conjunctivitis, and the anatomy of their mouth parts," *Parasitology.*, 22:457–67.

62. Roy, D. N., 1928. "Report on investigation into aetiology and prevention of Naga sore in Assam," *Indian Med. Gaz.*, 63:673–87.

63. Schwarz, E. A., 1894. "Notes on *Hippelates pusio* in the southern states," *Proc. Entomolog. Soc. Washington*, 3:178–80.

64. ———, 1895. "The Hippelates plague in Florida." *Insect Life*, 7:374–79.

65. Herms, W. B., 1926. "Hippelates flies and certain other pests of the Coachella Valley, California," *J. Econ. Entomol.*, 19:692–95.

66. ———, 1928. "The Coachella Valley (California) Hippelates fly project," *J. Econ. Entomol.*, 21:690–93.

67. ———, and Burgess, R. W., 1930. "A description of the immature

stages of *Hippelates pusi* Loew and a brief account of its life history," *J. Econ. Entomol.*, 23:600–603.

68. Graham-Smith, G. S., 1930. "The Oscinidae (Diptera) as vectors of conjunctivitis, and the anatomy of their mouth parts," *Parasitology*, 22:457–67 (1 plate).

69. Kumm, Henry W., 1935. "The natural infection of *Hippelates pallipes* Loew with spirochaetes of yaws," *Tr.Roy.Soc. Trop. Med. & Hyg.*, 29:265–72.

70. ———, and Turner, T. B., 1936. "The transmission of yaws from man to rabbits by an insect vector, *Hippelates pallipes* Loew," *Am.J.Trop.Med.*, 16:245–62 (5 plates).

71. ———; Turner, T. B.; and Peat, A. A.; 1935. "The duration of motility of the spirochaetes of yaws in a small West Indian fly, *Hippelates pallipes* Loew," *Am. J. Trop. Med.*, 15:209–23.

72. Sanders, D. A., 1940. "*Musca domestica* and *Hippelates* flies, vectors of bovine mastitis," *Science*, 92:286.

73. Hall, David G., 1932. "Some studies on the breeding media, development, and stages of the eye gnat, *Hippelates pusio* Loew," *Am.J.Hyg.*, 16: 854–64.

74. Burgess, R. W., 1935. *The eye gnat in the Coachella Valley, California*, Washington, D. C.: Dept. Agric., in Bur. Entomol. & Plant Quar. Circular E—335 (Mimeograph ser.).

75. Sabrosky, Curtis W., 1941. "The *Hippelates* flies or eye gnats: Preliminary notes," *Canad. Entomologist*, 73:23–27.

CHAPTER XI

MOSQUITOES

ORDER DIPTERA, FAMILY CULICIDAE

Classification and Biology

Importance. Among the numerous species of bloodsucking arthropods that annoy man and other warm-blooded animals, both wild and domestic, mosquitoes stand out most prominently—their number is legion, and they are an almost constant annoyance; there are some that bite by night and others that bite by day. Great swarms may be produced even in small quantities of water. They breed in practically all sorts of water, fresh and salt, foul or potable; water in tin cans, broken gourds, hoof prints, tree holes, reservoirs of impounded water; and vast stretches of salt marsh. Great areas of seacoast are at times made uninhabitable by salt-marsh mosquitoes, and agriculture may be retarded. Real estate values suffer where the mosquito pest prevails, and losses resulting from lowered industrial efficiency are frequently considerable because of mosquito annoyance. Economic losses due to mosquitoes would alone, no doubt, amply justify the great sums now spent on mosquito abatement, yet these losses are minor compared with the prodigious damage done to the public health by mosquitoes as vectors of disease.

Mosquitoes are the sole vectors of the malarias, yellow fever, and dengue, and they participate very importantly in the transmission of filariasis and the encephalitides. It is reported that in an ordinary year in India alone at least a hundred million persons suffer from, and a million succumb to, the direct ravages of malaria. The indirect effect in lowered vitality and susceptibility to other diseases accounts for another million deaths. Considerable progress in spite of severe economic handicaps is being made to alleviate this serious situation.

Family Culicidae. Although some authors, Edwards[1] and others, include the dixa midges and the chaoborid gnats (see Chapter X) as well as the true mosquitoes in the Family Culicidae, only the latter are so classified for the purposes of this book. Excepting the huge sturdily built American gallinippers, *Psorophora ciliata* (Fab.) (body length 9 mm and wing spread 13 mm), and a few other species, mosquitoes are small and fragile, ranging in body length from 3 to 6 mm. Abundant scales are present on the wing veins and fringing the margins; the body, head, and legs are scaly.

Mosquitoes are distinguished by their characteristic wing venation (Fig. 65), as follows: subcosta (Sc) long, reaching the costa; radius four-branched; R_{2+3} forked; R_{4+5} simple; no cross-vein connection of R_1 and R_2; media (M) two-branched; cross-veins r-m and m-cu both present; Ax absent or very faint; cubitus (Cu) forked; An, long and reaching wing-margin; characteristic scales clothing the wings, and more or less abundant on the head and body (often scant or wanting); characteristics of the thorax, such as the absence of a definite suture between the prescutum and scutum, completely divided pronotum (Edwards). The true mosquitoes have elongate mouth parts formed for piercing, and for bloodsucking in the females, though not all mosquitoes are bloodsuckers, and males are normally not bloodsuckers. The antennae are long and filamentous with 14 or 15 segments, hairs in whorls, plumose in the males of most species; males of *Opifex* and *Deinocerites* do not have plumose antennae.

There are about 1,600 described species of mosquitoes in the world; about 120 in North America.[2] These ubiquitous insects occur at elevations of 14,000 feet in Kashmir and as low as 3,760 feet below sea level in gold mines of south India (Russell *et al.*[3]). For practical purposes mosquitoes may be divided into four tribes: (1) **Megarhinini**, basal half of proboscis rigid and distal portion flexible, the adults are flower-feeding and the larvae are predaceous, e.g., *Megarhinus inornatus* Walker; (2) **Culicini**, in which the palpi of the female are less than half as long as the proboscis, scutellum trilobed, pulvilli present, eggs laid in rafts, e.g., *Culex pipiens* Linn.; (3) **Aedini**, generally included with the Culicini, in which the palpi and scutellum are as in the Culicini, but the abdomen of the female is pointed, postspiracular bristles are present, pulvilli absent or hair-like, eggs are laid singly, e.g., *Aedes aegypti* (Linn.); (4) **Anophelini**, in which the palpi of both sexes are as long or nearly as long as the proboscis, scutellum rounded, without lobes, eggs laid singly, e.g., *Anopheles maculipennis* Meigen (Fig. 65).

The *larvae* of mosquitoes are without exception aquatic and are distinguished from all other dipterous larvae by the possession of a complete head capsule and the presence of only one pair of functional spiracles, situated dorsally on the eighth abdominal segment. The medical entomologist must be particularly familiar with larval characters in order that he may make accurate identifications in the conduct of surveys and inspections and in answering complaints (Fig. 65).

Male terminalia. The most critical differences used in the classification of adult mosquitoes are to be found in the male terminalia[4] (Fig. 66).

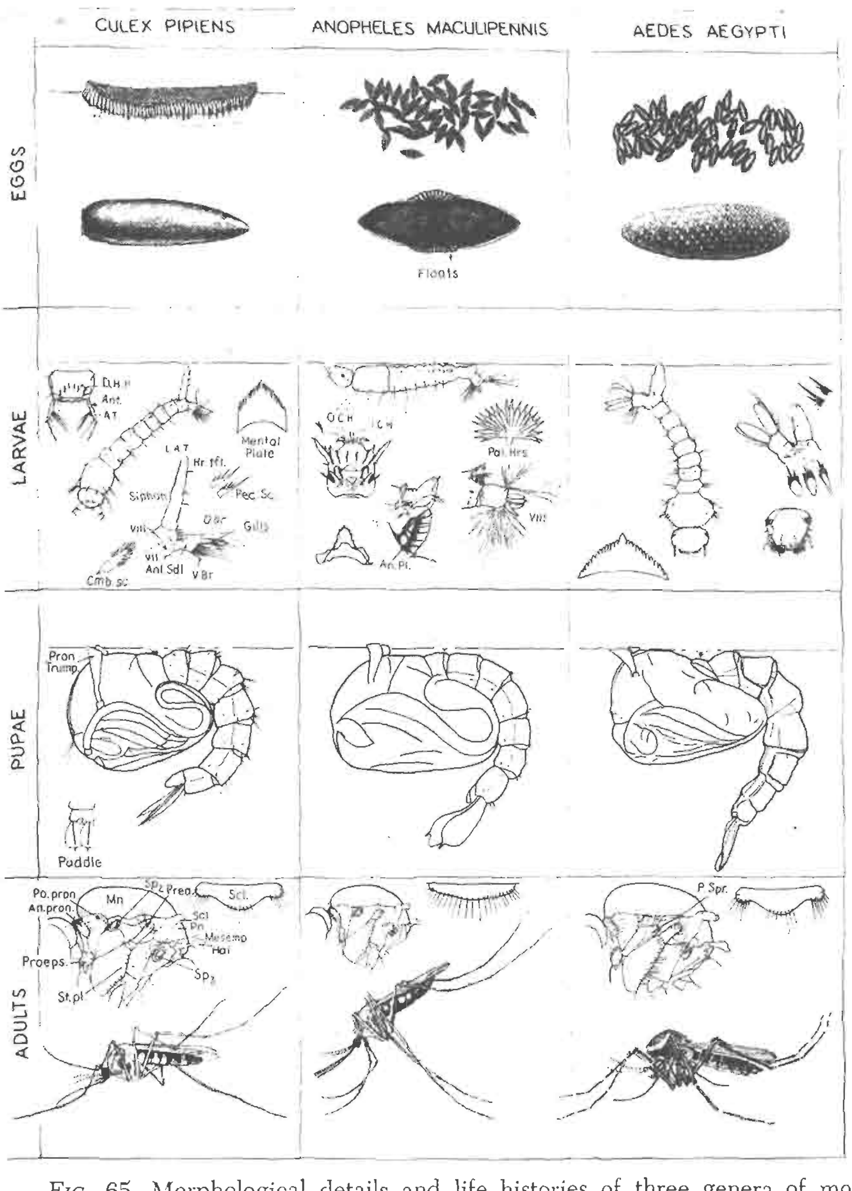


FIG. 65. Morphological details and life histories of three genera of mosquitoes: *Culex, Anopheles,* and *Aedes*. Explanation of abbreviations: *An. Pl.*, anal plate; *An. pron.*, anterior pronotal setae; *Anl. Sdl.*, anal saddle (dorsal plate); *Ant.*, antenna; *A.T.*, antennal tuft; *Cmb. sc.*, comb scale; *D. Br.*, dorsal brush; *D.H.H.*, dorsal head hairs; *Hal.*, haltere; *Hr. tft.*, siphon hair tuft; *I.C.H.*, inner clypeal hair; *L.A.T.*, lateral abdominal tuft; *Mesemp.*, mesepimeral setae; *Mn.*, mesonotum (tergum); *O.C.H.*, outer clypeal hair; *Pal. Hrs.*, palmate or float hairs (tuft); *Pec. Sc.*, pecten scale; *Pn.*, postnotum; *Po. pron.*, posterior pronotal setae; *Prea.*, prealer setae; *Proeps.*, proepisternal setae; *P. Spr.*, postspiracular setae; *Resp. Trump.*, respiratory trumpet; *Scl.*, scutellum; *Sp.*, spiracle; *St. pl.*, sternopleural setae; *V. Br.*, ventral brush. (Adapted after various authors.)

It is of importance to know that the terminal portion of the abdomen of the adult male begins to rotate on its axis within a few hours after emergence from the pupa, and a rotation of 180 degrees is completed in

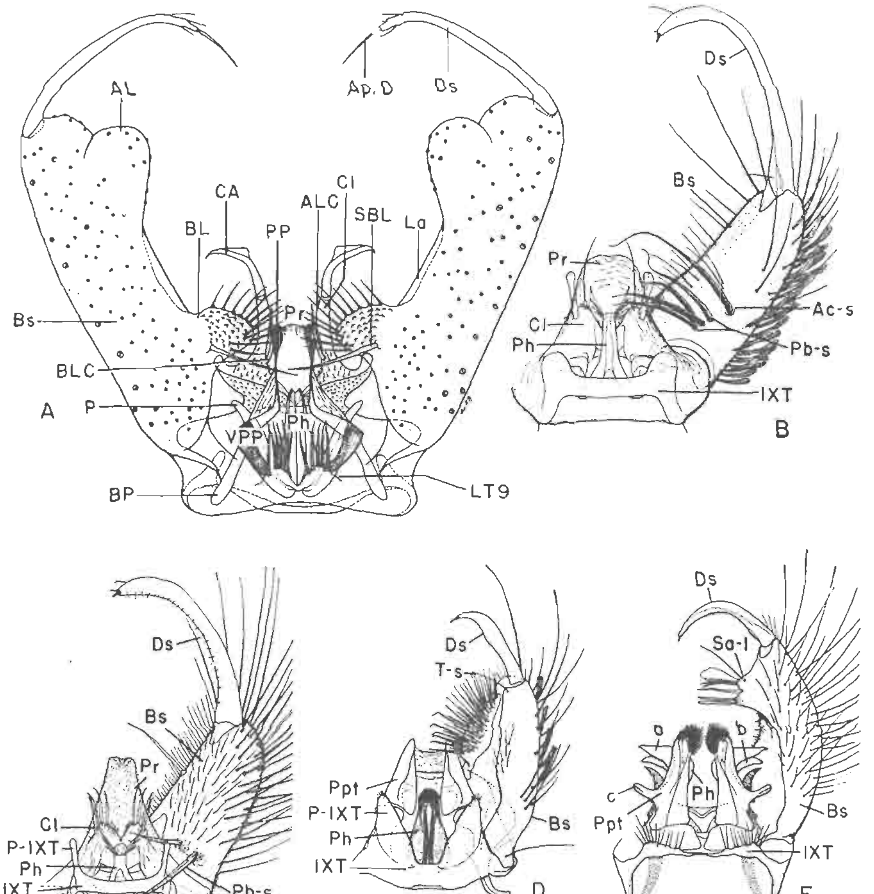


FIG. 66. Male terminalia of (*A*) *Aedes squamiger*, (*B*) *Anopheles gambiae*, (*C*) *Anopheles freeborni*, (*D*) *Aedes aegypti*, and (*E*) *Culex quinquefasciatus*. AL, apical lobe; ALC, apical lobe of claspette; Ap. D, appendage of diststyle; Bs, basistyle; BL, basal lobe; BLC, basal lobe of claspette; BP, basal plate; CL, claspette; CA, claspette appendage; Ds, diststyle; La, lacuna; LT9 (= P − IXT), lobe of 9th tergite (= process of 9th tergite); P, paramere; Ph, phallosome; Ppt, paraproct; Pr, proctiger; SBL, spine of basal lobe; VPP, ventral arm of paraproct, IXT = 9th tergite. (*B* to *E* above after Ross and Roberts.)

from 12 to 24 hours; this portion of the abdomen remains upside down for the rest of the mosquito's life. The terminalia may be prepared for microscopic study by first clipping off the tip of the male abdomen with fine scissors and then dropping it into 70 per cent alcohol for a few moments, after which it is transferred to 20 per cent KOH for twenty minutes, thence to a glass slide where the excess KOH is removed by blotting the edge of the liquid; next a drop of glacial acetic acid is put on the specimen, removing the excess by blotting; finally a drop of chloral gum is placed on it and it is then covered with a cover-slip. After drying, the specimen is ready to be sealed. Microscopic identification is made by comparison with drawings of the terminalia of various species in such publications as the *Mosquito Atlas* by Ross and Roberts.[5] A description of the technique for dissecting male terminalia of mosquitoes is given by Komp.[6]

Life history of mosquitoes. All mosquitoes pass through the several stages of a complex metamorphosis—*egg, larva, pupa, and adult* (Fig. 65). Descriptions of the developmental stages of mosquitoes were published as early as 1665 by Robert Hooke (Russell *et al., loc. cit.*). The larvae are commonly known as *wrigglers,* and the pupae as *tumblers.* Water in which to pass the larval and pupal stages is essential. Mosquitoes of some species may deposit their eggs on moist earth or on the dry edges of water-holding tree holes, and the larvae of some species may exist for several hours under relatively dry conditions. Howard[7] states that "in no case, however, were we able to revive larvae in mud from which water had been drawn off for more than 48 hours, and after 24 hours only a small proportion of the larvae revived." Eggs, on the contrary, in some species may survive long periods of desiccation, notably those of the yellow fever mosquito, which will hatch after being dry for a period of six months. According to Dyar,[8] the eggs of *Psorophora,* with their spinose protecting coat, are able to withstand desiccation on the dry ground for months or years, hatching with the advent of water. Similarly the eggs of various salt-marsh *Aedes* species may survive desiccation for several years.

Mosquito eggs are deposited either singly or in rafts (Fig. 65) on the surface of quiet pools of water and by some species along the margins, and even in fairly dry situations where pools may be formed later by rains or tidal action. The incubation period varies greatly with the species and temperature, from 16 to 24 hours in many of the Culicini; in the case of the snow mosquitoes, boreal Aedini, eggs laid during the summer and autumn remain buried under the snow through the winter and hatch with the melting snow in spring; thus one brood is produced annually.

The larvae of the various many-brooded species, most commonly observed in rain barrels, watering troughs, and similar situations, hang suspended diagonally from the surface by means of a prominent breathing siphon with head downward as in the Culicini. *Aedes* larvae behave similarly. The larvae of the tribe Anophelini lie horizontally just beneath the surface of the water, suspended particularly by means of palmate hairs (Fig. 65). The mandibulate larvae of practically all species secure their food by browsing on microörganisms, both plants and animals. It is not difficult to observe the feeding habits of the larvae as they squirm about while breathing at the surface or wriggle down to the bottom or along the sides nibbling food. Anopheline larvae are adapted for feeding at the surface, as is indicated by the palmate hairs by means of which they maintain a horizontal position and by their ability to rotate the head through 180° while feeding against the surface film, which is laden with bacteria and other microörganisms (Christophers and Puri).[9] The larvae grow rapidly during the warm summer, molting four times, the last molt resulting in the pupa. An average of seven days is required for the larval stage in several of our commoner Culicini under optimum conditions. The larval stage of the Anophelini requires a somewhat longer time.

With the fourth molt the pupa or "tumbler" appears. In this non-feeding stage there is a pair of breathing "trumpets" situated dorsally on the cephalothorax. The pupa is remarkably active and sensitive to disturbances of the water, letting go suddenly and darting with a tumbling motion to deeper water and after a few moments rising with little motion to the surface, where the breathing trumpets break the surface film and contact with the air is re-established. The pupal stage is quite short, usually from two to three days.

In a series of experiments to determine the effect of various quantities of a given larval food on the development of a common species of fresh-water mosquito in California, namely *Culiseta incidens* (Thom.), the largest percentage of emergence was obtained when 2.5 grams of yeast were supplied per liter of distilled water. The egg rafts were first placed in the water, each raft in a separate container, hatching in about 2½ days. The newly hatched larvae were thoroughly mixed and transferred in lots of 100 to battery jars containing one liter of water with a measured quantity of Fleischmann's yeast; pH readings were taken before and after the yeast was added, and in all cases the pH readings were 6.6; daily pH readings were taken thereafter until all the mosquitoes had emerged, the pH remaining the same until pupation; the average minimum room temperature was 19.2° C and the maximum 24.4° C. By far the largest percentage of adults, 88 per cent, was produced in the jar with 2.5 grams of yeast, with a sex ratio of 5.1 males to 4.9 females. The first molt took place on the fourth day, the second molt on the sixth day, the third on the ninth day, and the fourth on the twelfth day, when the first pupa appeared; the first adult mosquito, a male, emerged on the eighteenth day, giving a total of about 21 days for the complete life history, including the incubation period of the eggs. The complete record of the experiment is published elsewhere.[10] When the temperature was maintained at 24° ± 1° C the life history was shortened to 12½ days—egg stage 24 hours, larval stage 10 days, and pupal stage 36 hours.

Food habits of adult mosquitoes. The mouth parts of male mosquitoes are not suited to piercing; hence males are not bloodsuckers. Their nourishment is normally derived from nectar and plant juices and other liquids. With the exception of a few species, such as the plant-feeding Megarhinini and the *Harpagomyia,* which feed on regurgitated stomach contents offered by ants (*Cremastogaster*), all female mosquitoes are able to pierce the skin of many kinds of animals and feed on blood. No doubt vast numbers never have the opportunity to feed on blood. The great majority of species are *zoöphilous,* i.e., feed in nature on animals other than man; some feed only on the blood of reptiles and amphibians, e.g., *Culex apicalis* Adams. Species which feed on man by preference are said to be *anthropophilous.* The feeding habits of mosquitoes may be determined by applying the precipitin test to wild-caught engorged females.

Flight habits of mosquitoes. Although most of the domestic species remain fairly close to their point of origin, i.e., within a distance of a few city blocks or half a mile, there are many species, particularly among the Aedini, which may travel many miles. In searching for breeding places of the common Culicini under urban conditions the point of origin will usually be found not far from the points of complaint.

Salt-marsh mosquitoes are often a source of great annoyance far from their breeding places, and a knowledge of their migratory habits is important in mosquito control. *Aedes sollicitans* (Walker), an important salt-marsh species of the Atlantic seaboard, is known to migrate at least 40 miles. The migrations of *Aedes squamiger* (Coquillett) have been traced on the Pacific coast for a distance of 50 to 75 miles.[11] Males are seldom found far from their point of origin, although the males of *Mansonia perturbans* (Walker) are said to accompany the migrating females.

The flight range of *Aedes vexans* (Meig.) and *A. aldrichi* D. & K. has been studied by Stage, Gjullin, and Yates[12] by using a stain, 1.5 per cent aqueous solution of methylene blue or eosine, applied with a hand compressed-air sprayer to newly emerged adult mosquitoes resting on vegetation near their breeding places. Mosquitoes were collected in this area at regular intervals until no more could be found. The collected specimens were killed and each tested with one or two drops of solvent made of 3 parts glycerin, 3 parts 95 per cent alcohol, and 1 part chloroform. The following results are recorded: (1) Both species and both sexes were dispersed in all directions, with and against general wind currents, for a distance of about two miles. (2) Males moved away from the breeding areas more slowly than females. (3) Females of both species traveled one-half mile across part of the Columbia River (Oregon) within 24 hours after being stained. (4) One *Aedes vexans* (Meig.) female was recovered 46 days after being stained, three miles distant and across the Columbia River. (5) One *Aedes* sp. male was taken at a point 5 miles distant 24 days after being stained. This was the greatest distance for any positive flight record obtained. (6) The pests were abundant to a distance of 15 miles from the breeding grounds and then diminished rapidly until at about 30 miles only one female was taken during a 10-minute search.

In conducting control operations for many years involving *Anopheles quadrimaculatus* Say it has been assumed that the usual maximum flight of this species is about one mile, and this procedure is in the main sound; however, Eyles, Sabrosky, and Russell[13] recaptured marked individuals 2.0 to 2.7 miles from the point of release and a single individual at 3.63 miles. It is pointed out, however, that there was no domestic blood supply sources between the flooded swamp and the stations of recapture. The flight range of *Anopheles freeborni* Aitken during dispersal and hibernation flight is, of course, much greater, being as much as 25 miles.

Using the fluorescent dye, Rhodamine B, to mark the mosquitoes, Reeves[14] and associates released over 20,000 *Culex tarsalis* Coq.; the maximum range of recovery was 2.5 miles for females and 1.1 miles for males. The majority of recoveries were made within one mile. On the basis of these studies it is recommended that control measures for *Culex tarsalis* be carried out in a zone not less than one and one-half miles beyond the human or animal hosts to be protected.

Longevity of mosquitoes. Male mosquitoes usually remain alive for but six or seven days, although *Anopheles pseudopunctipennis franciscanus* Theob. males have been kept alive in our laboratories for over a month, and Mayne[15] was able to keep an *A. punctipennis* Say male alive for 89 days and a female of the same species for 231 days; females with ample food may live for four or five months, particularly under hibernating conditions. During their period of greatest activity it is likely that the average lifetime of the females is not far from 30 days.

The staining experiment by Stage, *et. al.* (*loc. cit.*), produced important data relative to longevity. Thus six *Aedes aldrichi* D. & K. females were taken 52 days after staining, one female of the same species 85 days after staining, one *Aedes vexans* (Meig.) female after 55 days; also, under especially favorable conditions, one 94-day-old *A. aldrichi* D. & K. male was taken, and females of both *Aedes aldrichi* D. & K. and *A. vexans* (Meig.) were collected from 104 to 113 days after staining. The latter species is said to have the greater maximum longevity by approximately 15 to 20 days.

Freeborn[16] has found that increased humidity has a protective influence on the longevity of *Anopheles freeborni* Aitken kept at constant temperature; but at a constant of 80° F no amount of relative humidity can protect them for the full life span of a month. A relative humidity of 55 per cent ensures the normal life span at 70° F. Freeborn points out that 55 per cent humidity involves a saturation deficiency of 3.6 grains per cubic foot, by which can be expressed the drying power of the air in the absence of wind currents. A deficiency of 3.8 grains at 76° F was tolerated for only three weeks instead of more than four. It is pointed out that the lethal effect may be caused by either a fatal

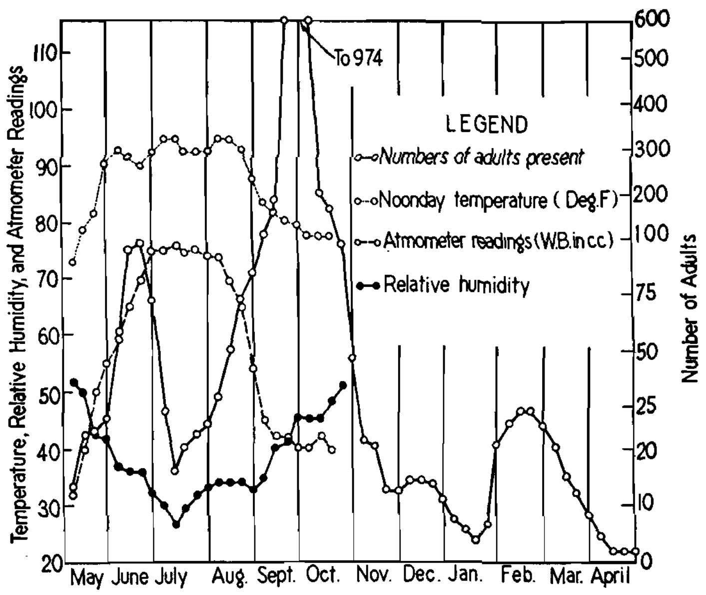


FIG. 67. Weekly catches of adult *Anopheles maculipennis* with the existing noonday temperatures in degrees Fahrenheit, the noonday relative humidity in percentage, and the weekly average of 24-hour records of the white ball atmometer in cubic centimeters. (After Freeborn.)

temperature or by desiccation of the insect's body. With an increase of temperature or a decrease in relative humidity the saturation deficiency increases and the demand on the insect's moisture content becomes greater (Fig. 67). The ability of a particular species to retain adsorbed water in the presence of existing saturation deficiencies undoubtedly explains the variability of resistance of the different species to desiccation, according to Freeborn. The length of a life of *A. freeborni* as well as other vectors of malaria has important bearing on their ability to transmit the infection, i.e., if the mosquito dies before the malaria parasite is fully developed, the latter must also perish.

Internal anatomy of mosquitoes. To be prepared to study the relation of mosquitoes to such diseases as malaria and filariasis, the student must be familiar with their internal anatomy. The attention of the student is called particularly to the excellent treatise on the "Structure and Biology of Anophelines" by Nuttall and Shipley[17] (1903).

The alimentary canal of the mosquito is separable into three regions, the *fore-*, *mid-*, and *hind-gut,* each of which may be arbitrarily subdivided into more or less distinct divisions (Fig. 16, p. 52). Thus the fore-gut consists of the sucking tube of the *proboscis,* the *pharynx,* including the pumping organ, and the *esophagus* with its *diverticula* (three in number and generally known as food reservoirs). The mid-gut consists of a narrower anterior portion (false proventriculus) and a wider posterior portion (*stomach*) occupying the thorax and much of the abdomen, and limited posteriorly by the origin of the five *Malpighian tubules,* which indicate the beginning of the hind-gut. The hind-gut is bent on itself several times and consists of the narrow, longer *ileum,* the *colon,* and what is arbitrarily termed the *rectum,* indicated by the presence of *rectal papillae.*

The *salivary system* consists of two sets of salivary glands (right and left), three glands to each set. These organs are situated ventrally in the thorax near the neck. Each set of glands empties into a duct which combines with the opposite one to form the common salivary duct. This common duct empties its contents into the pharynx through the salivary receptacle close to the base of the proboscis.

The *reproductive system* of the female mosquito occupies the posterior portion of the abdomen and comprises a pair of *ovaries* joined by a pair of oviducts terminating in the *vagina,* which opens ventrally in a depression of the ninth sternite; *spermathecae* are present (one to three, depending on the species). The spermathecae of an impregnated female contain myriads of spermatozoa, and the ovaries when mature occupy the larger part of the abdomen.

TRIBE MEGARHININI

Characteristics. The members of the tribe Megarhinini occur in tropical as well as temperate climates and are usually highly colored; they are day fliers; both sexes are flower-feeders and do not suck blood. The basal half of the proboscis is stout and rigid, while the distal portion is flexible, which accounts for the curious hook-like position of the proboscis when at rest. The palpi vary in length from one-fourth the length of the proboscis to nearly the same length. The huge larvae are predaceous and cannibalistic. The mouth parts are particularly adapted for capturing prey.

The eggs are deposited singly, and Edwards states that most species breed in small confined collections of water, such as may occur in bamboo stems, tree holes, pitcher plants, and the like.

Edwards lists 52 species, of which two species are said to occur in North America, *Megarhinus rutilus* Coq. and *M. septentrionalis* Dyar and Knab. The giant species *Megarhinus inornatus* Walker was introduced from New Britain into the Hawaiian Islands[18] for purposes of mosquito control, without practical result.

TRIBE CULICINI

Characters. All members of the tribe Culicini have the scutellum trilobed with each lobe bearing bristles, but with areas between lobes without bristles (Edwards). The abdomen is blunt and completely clothed with broad scales, which nearly always lie flat; the pulvilli are broad and distinct; postspiracular bristles are absent; the larvae have a prominent siphon with well-developed pecten (Fig. 65), and usually numerous hair tufts on the siphon. The eggs are usually deposited in tight raft-like masses on the surface of the water.

The Tribe Culicini, exclusive of the Aedini, which are separated from the Culicini for the purposes of this book, includes some 700 species distributed among more than 20 genera, of which the genus *Culex* alone contains nearly 400 known species.

Culex pipiens Linnaeus. Two subspecies are generally recognized, viz., *C. pipiens pallens* Coq. and *C. pipiens molestus* Forskål. This is the common house mosquito or rain-barrel mosquito of many temperate parts of the world. The thorax, abdomen, and proboscis are brown; the latter is darker toward the tip. The basal white bands on the abdomen join lateral basal triangular patches. This mosquito, a domestic species, lays its eggs in rafts on water, in rain barrels, tanks, cisterns, catch basins, and other small collections of water. Where breeding places are favorable it may occur in enormous numbers. It invades houses freely. Because of its vicious bites and high-pitched, tantalizing hum continued late into the night, it may be a terrific pest. Although greatly influenced by temperature the life history requires but about 10 days under warm summer conditions, egg stage 18 to 24 hours, larva about 7 days, and the pupa about 2 days.

Woke[19] fed 38 *Culex pipiens* Linn. on man, and these mosquitoes deposited 29 egg masses, totaling 2,118 eggs, or an average of 73.0 eggs per mass. At the same time 39 females fed on a canary deposited 22 egg masses, totaling 4,473 eggs, or an average of 203.3 eggs per mass. Over twice as many eggs per mass or per milligram of blood ingested were produced by mosquitoes fed on canary blood as were produced by mosquitoes fed on the blood of man.

Culex quinquefasciatus Say [= *Culex fatigans* (Wied.)] is one of the most widespread household pest mosquitoes of warmer climates. In North America it is said to extend to 39° N. latitude. It is an important vector of *Wuchereria bancrofti*. It is a severe nocturnal biter. It breeds in all sorts of artificial water containers, but also in ground pools. It shows a preference for polluted water. The adult females are medium- to small-sized, resembling *Culex pipiens* very closely.

Culex tarsalis Coq. (Fig. 68) is an abundant and widespread species of the semiarid regions of North America; however, it occurs as far east as Illinois and southerly to western Florida. It has been taken at elevations of 9,000 feet. It is an important vector of the virus of western

FIG. 68. *Culex tarsalis*, an important vector of the virus of western equine and St. Louis encephalitis. (Photograph by R. Craig.)

equine and St. Louis encephalitis. It is a fairly large and robust species; generally dark brown to black in color; the black abdomen has broad, segmental, basal bands of yellowish-white scales; each segment of the ventor with V-shaped marking of black scales, the apex of the V anteriorly; femora are black with a dotted white line along both sides, knees white; tibiae black, also with white line, bases and apices white; tarsi black; hind tarsi with apical and basal white bands on all segments, last tarsal segment white; proboscis black, with a sharply outlined white ring just before the middle. It breeds in all sorts of ground pools, roadside ditches, pools in and around corrals, artificial containers, etc.

Culiseta incidens (Thomson), like other members of this genus, partakes of characters which would place it in either of the tribes Culicini or Aedini, but for practical purposes, based largely on breeding habits, the members of this genus are placed in the tribe Culicini. In this genus, (*Culiseta*), which includes seven North American species, the postspiracular bristles (Fig. 65) are absent and in the females of at least

Pacific coast species the anterior and posterior cross-veins tend to lie in one line, except in *C. morsitans* Theo., in which they are well separated, i.e., by more than half their length. In *Culiseta incidens* (Thom.), a western species (west of the Rocky Mountains), the wings are spotted. It breeds throughout the year, where temperature permits, in all sorts of permanent pools and is a common domestic species. It lends itself particularly well to laboratory experimentation. The life history of this species is described earlier in this chapter.

Culiseta inornata (Williston) is found throughout the United States and southern Canada. Its wings are broad and clear, the cross-veins are scaled, and the very short black palpi have white scales at the tip. It breeds throughout the year under favorable temperature conditions wherever pools are present in wooded areas.

TRIBE AEDINI

Characteristics. Ordinarily the *Aedes* mosquitoes are included in the tribe Culicini, but because of their remarkable breeding habits and other striking characteristics the author has taken the liberty to set apart the *Aedes*, of which there are about 600 species, as a separate tribe, the Aedini. Nearly half of all the species of mosquitoes in North America belong to the genus *Aedes*.

Like the Culicini the Aedini have a trilobed scutellum. In most of the species, as Edwards points out, the claws are toothed in the female, postspiracular bristles are present, the pulvilli are absent or hair-like, and the female abdomen tends to be more pointed and the cerci longer than in other groups. The larvae have short siphons bearing one pair of posteroventral hair tufts, and nearly always a distinct pecten (Fig. 65). The eggs are deposited singly on the surface of the water, on mud, or even in situations where there may be little moisture but where submergence may follow. The females of all species bite, many of them viciously. Many species are diurnal in biting habits, most of them biting toward evening.

Salt-marsh mosquitoes. *Aedes dorsalis* (Meigen), a fierce day biter, is widely distributed throughout the northern half of the United States, Canada, Europe, and Asia. In general the body is straw-colored (tan), varying from almost white to dark brown; the thorax has three longitudinal bright brown stripes; the hind tarsi have white bands at bases and apices of all segments, the last one wholly white. Although the species breeds freely and abundantly in fresh water, such as flood water, rice fields, and drainage from irrigation, it is nevertheless the commonest salt-marsh mosquito of the Pacific coast north of Monterey. It is here a distinctly brackish-water breeder, generally breeding in pools reached only by the monthly "rip" tides. There are thus several monthly broods, the first appearing as early as March. The eggs are deposited singly, most of them in the mud along the edge of receding pools; they may remain unhatched for many months in situations from which water is excluded. Development after hatching is rapid, and emergence of the adult mosquitoes may be within eight or nine days.

Aedes squamiger (Coquillett) is restricted to the Californian seacoast from San Francisco Bay to San Diego, breeding in salt-marsh pools that are fed by fresh water from the winter rains. Like *A. dorsalis* (Meig.) it is a fierce day biter, rather worse toward dusk, and travels great distances. It is known as the gray salt-marsh mosquito. The vestiture is characteristically composed of large scales. The proboscis is uniformly black scaled. Its egg-laying habits are similar to those of *A. dorsalis* (Meig.); there is, however, but a single brood of *A. squamiger* in the San Francisco Bay region coming as early as February.

Aedes taeniorhynchus (Wiedemann) is a typical salt-marsh breeder distributed along the coastal area of the United States northward to Connecticut on the east coast and to Santa Barbara on the west coast. It is the brown salt-marsh mosquito; its proboscis is distinctly white-banded. It is a fierce day biter, and its egg-laying habits are similar to those of *A. dorsalis* (Meig.) and *A. squamiger* (Coq.). There are monthly broods throughout the summer. Development is exceedingly rapid, the larval stage may require but four days, the adults emerging in from 8 to 10 days.

Aedes sollicitans (Walker), the pestiferous salt-marsh mosquito of the Atlantic coast, breeds from Maine to Florida and thence west along the Gulf of Mexico to Texas. There are many broods, and in its southern range breeding may be continuous.

The numbers of larvae appearing in certain pools is almost unbelievable. Smith[20] states:

> I have found pools so crowded that an estimate of 100 wrigglers in an area of one square inch was scarcely equal to the fact. Half that number is a common occurrence. This means over 7,000 in an area of one square foot, and it needs an area of less than 150 square feet—a pool roughly ten by fifteen feet—to produce 1,000,000 mosquitoes at one hatching. If these figures seem incredible they can be easily verified anywhere along shore by anyone who will put on a pair of gum boots and will hunt out a few breeding places.

Flood-water Aedes. *Aedes vexans* (Meigen) is a typical flood-water mosquito having practically a world-wide holarctic distribution. It is one of the fiercest day biters and exceedingly abundant; it is truly a vexatious mosquito. It breeds in greatest numbers along the edges of rivers subject to overflow, and like other *Aedes* species lays its eggs along the muddy edges of receding pools, where they may hatch the same season when water due to intermittent flooding or freshets reaches them, or they carry over. There may thus be several broods where flooding occurs as a result of melting mountain snows or thunderstorms, or there may be only one brood where there is a single spring flood. The species is a rapid breeder and migrates many miles. It varies in color from brown to gray; the tarsi are basally narrowly banded; the wings are uniformly brown.

Aedes dorsalis (Meigen), already referred to as a salt-marsh breeder, is also a prolific breeder in open flood-water pools, particularly in irrigated pastures after flooding and in drainage pools due to excess water. Where early flood waters occur along wooded river courses the single-brooded *Aedes vexans* (Meig.), dominates, but as the season advances *Aedes dorsalis* (Meig.), a many-brooded species, supersedes it in these areas, but breeds particularly in open pastures where the temperature of the water is high. In water of a temperature of 99.5° F we found enormous numbers of larvae completing their cycle in about six days.

Tree-hole mosquitoes. Although the habit of breeding in water-holding tree holes occurs in various species belonging to genera other than *Aedes*, e.g., *Anopheles barberi* Coquillett, there are a number of typical tree-hole breeders in the Aedini, notably *Aedes varipalpus* (Coq.) a Pacific coast species; *Aedes triseriatus* (Say) of the eastern United States; *Aedes luteocephalus* Newstead, Ethiopian; *Aedes simpsoni* Theobald, Ethiopian, *Aedes seoulensis* Yamada, Chinese; and others.

Aedes varipalpus (Coquillett) has bright white markings on the legs at both bases and apices of the tarsal segments and many white or silvery scales distributed over the body so as to give the vestiture a silver mottled appearance. It is one of our smallest mosquitoes but a fierce biter. This Pacific coast species deposits its eggs on the sides of tree holes, notably holes in live oaks (*Quercus agrifolia*), also California laurel (*Umbellularia californica*) and valley oaks (*Quercus lobata*). Freeborn[21] states that the eggs "hatch whenever they are wet by the rising waters. There is some evidence that the eggs may drop off after a period of desiccation or, as an alternative method, the larvae may hatch without the intervention of actual wetting and fall into the water below. The straw-colored larvae with bright brown heads and enormously developed gills swim about their secluded medium with snake-like movements, but spend most of their time with their heads and thoraces buried in the silty deposit at the bottom of the tree holes. . . . The developmental period is extremely long, lasting from one to seven months. Although there is an intermingling of the broods, there are two pronounced peaks, one in the early summer and another in the fall. The fall adults deposit eggs which produce the larvae that overwinter."

Feng,[22] in his report on the tree-hole mosquitoes of Peiping, China, states that the pH of water in which all species bred varied from 7.8 to 8.4. He also states that there was no specific relationship between the species of the tree and the species of the mosquitoes, several species being frequently found breeding in the hole of the same tree.

Boreal Aedes or snow mosquitoes. An interesting group of *Aedes* consists of the so-called snow mosquitoes which appear in the early spring in the high mountains and northern ranges of distribution, breeding in the pools left by the melting snow (Fig. 69). These *Aedes* have but one generation and appear in enormous swarms in the higher elevations and

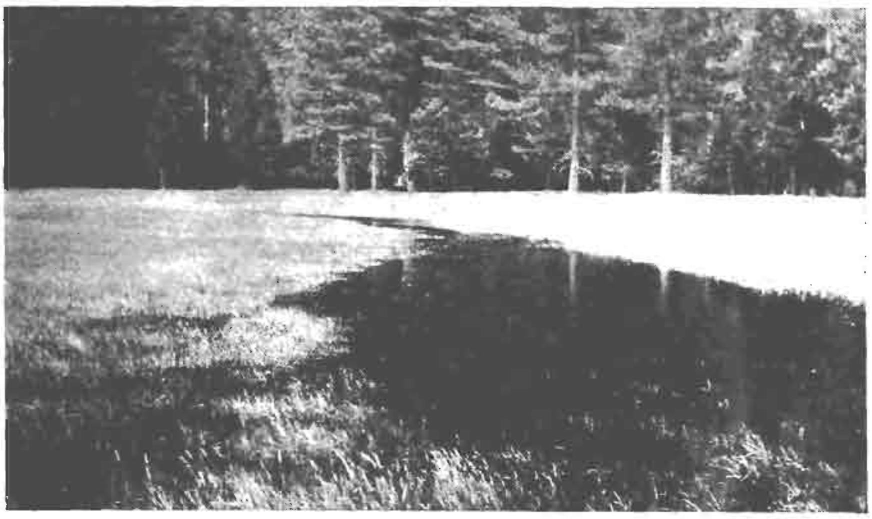


FIG. 69. Typical breeding place for snow mosquitoes on floor of Yosemite Valley. (Photograph by H. F. Gray.)

northern ranges much to the dismay of the huntsman and alpine traveler. These mosquitoes have been collected in many localities in the Sierra Nevada by the writer. The following quotations are taken from an interesting account of the mosquitoes by Dyar.[23]

At an altitude of 6,000 feet, pupae were abundant May 25 and by the first week in June the breeding was complete; even the pools that still contained water or had only just thawed out were empty. Adults appeared by the first of June, and by the 15th the woods were filled with them in all directions.

Speaking especially of the Fallen Leaf Lake region (vicinity of Lake Tahoe) a region in the heart of the Sierras to the north of the high peaks and on the eastern side of the divide, *A. (communis) tahoensis* Dyar is the commonest and earliest species, found everywhere, both in the hills and the pines in level country. It breeds in the earliest pools of clear water held in rocky land, its home being in the mountains, but it soon spreads everywhere. It was common in the early pools at the head of Fallen Leaf Lake, being the only species present. It also bred in wave-pools at the lower end of the lake. Dispersal of the adults was in general downward, they being abundant in the pines at Tallac on June 17, though no breeding places were near. *A. cataphylla* Dyar is less abundant and less widely dispersed at Fallen Leaf. It was commonest at the foot of the trail to Angora Lakes at the head of the lake, rare at the outlet of the lake and absent at Tallac on Lake Tahoe. *A. hexodontus* Dyar breeds in early pools, but especially those of a marshy character, larvae being taken from hoofprints of cattle in the edge of a marsh. The adults were well distributed and toward the end of June replaced *A. (communis) tahoensis* Dyar as the dominant species. *A. ventrovittis* Dyar is a rare species, taken only at one place near the outlet of Fallen Leaf Lake and then in small numbers. It is presumably a marsh breeder, though the larvae were not found. *A. palustris* breeds in open grassy marshes, not in large numbers. Dispersal was general, adults being taken everywhere, although seldom commonly. *A. increpitus* Dyar is the slowest breeder of any of the early species, the larvae lingering after all the others are gone, frequently in the same pools. They were abundant at the outlet of Fallen Leaf Lake with a downward dispersal, the adults being common at Tallac, about 2 miles from the breeding places, while only found a quarter of a mile up the lake and many days later.

The seasonal appearance of these mosquitoes varies with the altitude in the ratio of about a month in time to 1,000 feet of elevation. At Yosemite, at about 5,000 feet, all the species were about a month earlier than at Lake Tahoe, at 6,000 feet, while at Summit, at 7,000 feet, they were still another month later, larvae and pupae of *tahoensis* and *hexodontus* being taken there on July 2, 1916, about the same stage that they were taken at Fallen Leaf on June 1, 1916.

At an elevation of about 10,000 feet the author encountered a veritable plague of *Aedes ventrovittis* Dyar and *A. communis tahoensis* Dyar along the shores of Young Lakes in the Sierra Nevada Mountains July 22 to 27, 1936. Larvae and pupae were still present in small pools of snow water along the shores of the lakes. Apparently only certain pools were infested.

In northern Alaska the spring thaw is followed by a sudden emergence of swarms of mosquitoes. Like other boreal mosquitoes they overwinter in the egg stage and hatch as soon as the ice thaws from around the eggs in the spring. There is but one generation a year. Jachowski and Schultz,[24,25] who studied *Aedes punctor* (Kirby), *A. communis* (De Geer), and *A. nearcticus* Dyar, classify the sites from which they collected larvae as grassy sloughs, mossy pools ("tundra pools"), frost ditches, and willow-alder pools. The eggs hatch almost immediately after the spring thaw, and if a freeze occurs after this, the larvae will freeze into the ice and thaw out again when it warms up. The larval period was observed to be about 28 days and the pupal period from 3 to 5 days.

During chilly night hours mosquitoes were observed to gather in large numbers about the open doors of heated quarters or hovered in the warm draft from chimneys. Blood meals, if necessary, are readily available from herds of caribou, large populations of rodents, and nesting ducks, geese, and other birds.

Of 18 species of mosquitoes found in central Alaska according to Jenkins[26] the following species are the most abundant: *Aedes punctor* (Kirby), *A. communis* (DeGeer), *A. pionips* Dyar, *Culiseta alaskaensis*

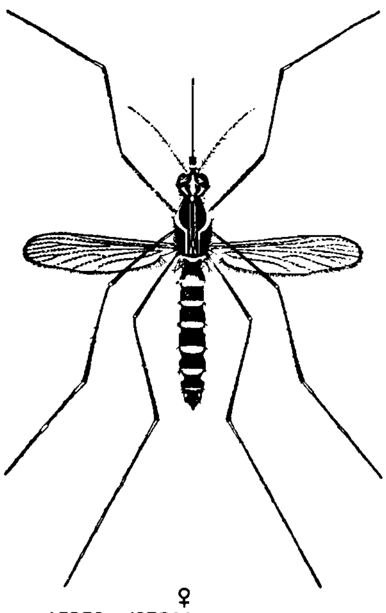


FIG. 70. *Aedes aegypti,* an important vector of the viruses of yellow fever and dengue. (Adapted after Soper, Wilson, *et al.*)

(Lud.), and *C. impatiens* (Walk.); *A. flavescens* (Muell.), locally abundant in the coastal marshes.

Aedes aegypti (Linn.) (Fig. 70), the yellow fever mosquito, will long be known under the name of *Stegomyia fasciata* (Fabr.) although it was known for several years under the names of *Aedes calopus* (Meig.) and *Aedes argenteus* (Poiret). It is not only the most important vector of yellow fever but likewise of dengue fever. This species is widely distributed within the limits of 40° N and 40° S latitude, but it is highly susceptible to temperature variations. According to Hindle[27] it soon dies in the open air at a temperature of 7° to 8° C, succumbing in a few seconds to an exposure of 0° C, and 37° C is rapidly fatal. Furthermore, it does not thrive in dry hot climates. The adult insect (Fig. 70) is beautifully marked with silvery-white or yellowish-white bands and stripes on a nearly black background, whence the name "tiger mosquito."

It has a "lyre-like" pattern dorsally on its thorax, i.e., two outer curved yellowish-white lines and two median parallel lines. The legs are conspicuously banded, and the last joint of the hind leg is entirely white. The head is covered with broad flat scales with only a single row of upright forked scales.

The yellow fever mosquito is a typical domestic species seldom found far from man's habitations. Many observers believe it to be a day-flying and day-feeding species, but this habit apparently is restricted to the younger individuals up to six or seven days after emergence, or rather until a meal of blood is secured, when the insect becomes nocturnal. Howard observes that "it prefers the blood of white races to that of dark races, and attacks young, vigorous persons of fine skin and good color in preference to anemic or aged people."

The eggs of the yellow fever mosquito are deposited singly on the surface of the water, usually in containers at or near the waterline; they are dark in color, and each egg is surrounded by air cells (Fig. 65). Comparatively few eggs are deposited at one laying, and while there may be two layings, possibly more, the total averages about 140 (144 according to Woke) when fed on man. Woke[28] found that this species produced greater numbers of eggs when fed on the frog or turtle.

Unlike the eggs of most species these can withstand desiccation to a very marked degree, some authors declaring that this is possible for several months. Ordinarily the eggs hatch in about 48 hours.

The larvae are quite robust, the breathing siphon is comparatively short and heavy and black (Fig. 65), and their position in the water is almost vertical, considerably more so than that of other culicine species. The larval stage is ordinarily passed in about 9 or 10 days under average conditions and in from 5 to 7 days under warmer conditions.

The pupae have broadly triangular breathing trumpets. Only about 36 hours is spent in the pupal stage.

According to Howard the shortest period of development from egg to imago observed by Reed and Carroll in Cuba was nine and one-half days, i.e., egg stage, two days; larval stage, six days; pupal stage, 36 hours. From this very short period the time ranges from 11 to 18 days according to the same author.

The yellow fever mosquito breeds by preference in artificial containers of rain water. (It is known, however, at times to breed naturally in brackish water.) Rain-water barrels, tanks, cisterns, tin cans, urns, etc., provide suitable breeding places (wooden containers seem to be preferred); water collected among the leaves of certain members of the Agave family, also water collected in banana palms may produce many mosquitoes.

Woke has shown that *Aedes aegypti* (Linn.) fed on frog blood and turtle blood produced viable eggs, and that the larvae developed normally and produced normal adults.

Although *Aedes aegypti* (Linn.) is called the yellow fever mosquito and is undoubtedly the most important vector of this disease under natural conditions because of its domestic breeding habits, there are nevertheless a dozen other species which are able to transmit the disease (see p. 239).

Other genera. The genus *Psorophora,* which includes only nine North American species, is distinguished by the presence of both prespiracular and postspiracular bristles and by the second marginal cell of the wing, which is more than half as long as its petiole. The larvae of some species are predaceous, feeding on other mosquito larvae and other small aquatic animals in temporary ground pools.

Psorophora columbiae (Dyar and Knab) is widely distributed in the eastern United States but ranges from parts of South America through Cuba and Mexico to Canada. It is strikingly speckled in appearance and is a fierce biter. In 1932 this species is reported to have caused great loss to livestock in the Everglades section of Florida. The *United States Insect Pest Survey Bulletin* (Vol. 12, No. 10, p. 428) describes the plague: " . . . by evening of that day the buzzing was as loud as that of a swarm of bees. During the night livestock could be heard running and thrashing in the underbrush, and on the morning of September 6, dead animals were found throughout the section. The recorded mortality was 80 head of cattle, 3 horses, 1 mule, 67 hogs, 20 chickens, and 2 dogs. Post-mortem examinations showed no mosquitoes in the respiratory apparatus, indicating that the animals died either from loss of blood, nervous exhaustion, or the effects of some toxin." The milk supply was also greatly reduced during the four days of the infestation.

The genus *Mansonia* is characterized in large measure by the scales of the wings, which are very broad by comparison with those of other species of mosquitoes. The larvae have the air tube sharply pointed, enabling them to pierce the stems or roots of aquatic plants from which they obtain air and to which they remain attached (submerged) throughout larval development.

Mansonia perturbans Walker is a small species (5 mm) which is widely distributed throughout North America. It has severe biting habits and evidently travels some distance from its breeding place. The biology of this and other species of the genus is fascinating indeed.

The genus *Orthopodomyia* includes only two species from North America. The adults are described by Matheson as "rather gaily ornamented and easily recognized by their coloring." The larvae are found in tree holes, also in broken bamboo and leaf axils of certain plants. *Orthopodomyia signifera* (Coquillett) extends along the eastern seaboard of the United States and westerly from Florida into southern California, thence northerly to near Sacramento.

TRIBE ANOPHELINI

Characteristics. The following characters are generally employed to characterize the tribe Anophelini: palpi of both sexes are usually about

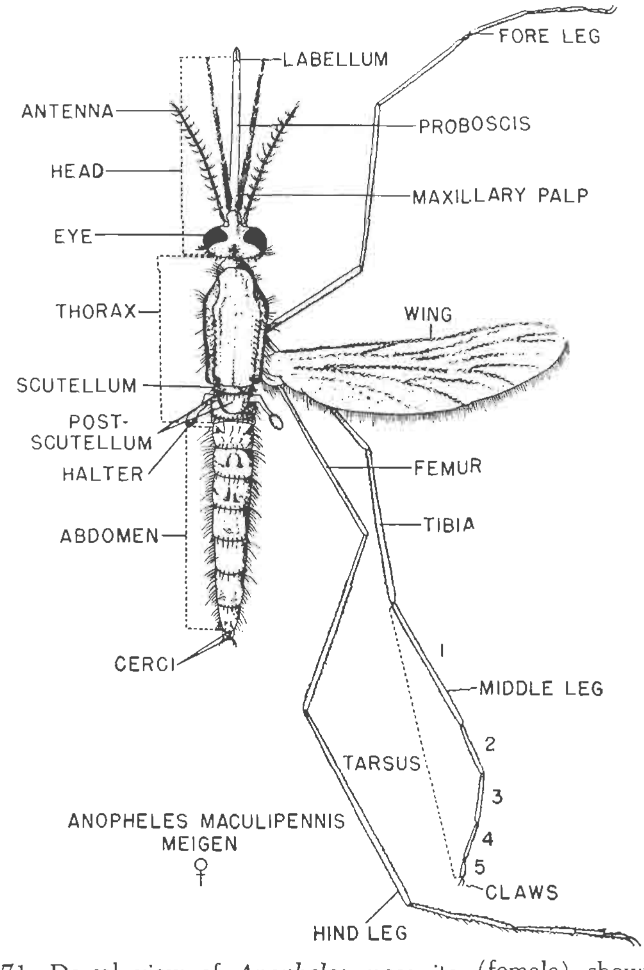


FIG. 71. Dorsal view of *Anopheles* mosquito (female) showing certain structural details useful in classification. (Modified after Essig in *College Entomology*, The Macmillan Company, New York, 1942.)

as long as the proboscis; the scutellum (Figs. 65 and 71) is evenly rounded (except in *Chagasia* where a slightly trilobed condition occurs); mandibles and maxillae of the females are well developed and toothed; legs are very long and slender, there are no distinct tibial bristles and no pulvilli; the abdomen is without scales, or at least with the sternites largely bare (Edwards); the wings usually have distinct markings.

The tribe Anophelini has been divided into numerous genera such as *Myzorhynchus, Arribalzagia, Argyritarsis, Neomyzomyia, Myzomyia,* and more than thirty others. Edwards, as well as other culicidologists, reduced the number of genera to three: *Chagasia,* with the scutellum slightly trilobed; *Bironella,* with scutellum evenly rounded, wing with stem of median (M) fork wavy; and *Anopheles,* with scutellum evenly rounded, wing with stem of median (M) fork straight. There are three species of *Chagasia,* all of tropical America. The genus *Bironella* includes two species, both of New Guinea. The genus *Anopheles* includes

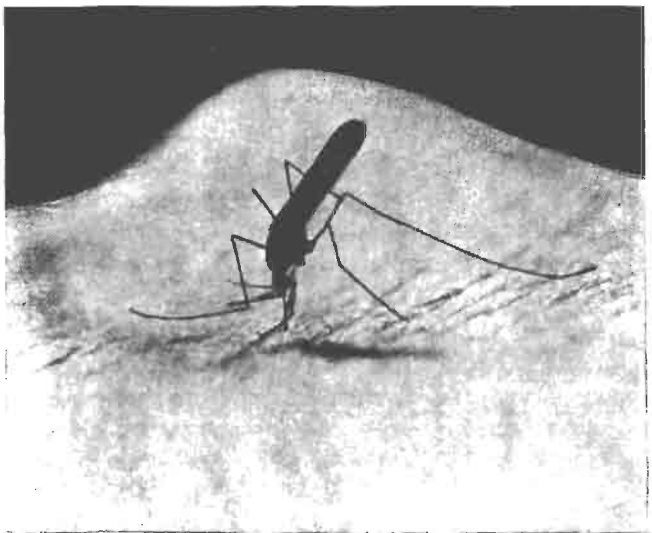


FIG. 72. *Anopheles freeborni* in the act of sucking blood from the author's hand.

over 300 species and subspecies, (Russell *et al.,* 1943, *loc. cit*), of which about 90 occur in the Americas, 14 in North America.

The common species rest with the proboscis, head, and abdomen nearly in a straight line and when resting have the appearance of a splinter lifted at an angle from a given surface (Fig. 72). In exceptional cases, as in *Anopheles culicifacies* Giles of India, the resting position is *Culex*-like. Hoffman[29] states that *A. grabhamii* Theobald rests with its body almost at a right angle to the vertical surface. *A. hyrcanus sinensis* Wied. takes a similar position. The hum of anophelines is distinctly low-pitched and almost inaudible unless they are close to the ear or in a bottle. Most of our common anophelines are not strong fliers and usually take to cover even in a moderate breeze. In California, *Anopheles freeborni* Aitken previously recorded here as *Anopheles maculipennis* Meigen, engages in an annual dispersal flight of overwintering females about mid-February, during which time the mosquitoes may invade much territory, traveling more than 25 miles. During this flight the first eggs are deposited. In the main this flight favors the spread of the species. While on the flight the mosquitoes bite by day even in broad sunlight. *Freeborni* also engages in a prehibernation flight during late October and November. The males having died soon after mating, the overwintering forms are females only.

Although some individuals of a few species may overwinter in the larval stage buried in mud and debris at the bottom of certain pools, the usual method of overwintering is in the adult (females only) stage.

Mating and oviposition. Fertilization of the females takes place almost immediately upon emergence. The males emerge first and may be seen dancing over or near the breeding places in small swarms apparently awaiting the appearance of the females; when these dart into the dancing swarm mating occurs. This type of mating requiring wide spaces is known as *eurygamous,* while those forms such as *Anopheles sacharovi* Favr. (= *elutus* Edwards) which mate in confinement in a small space are known as *stenogamous.* Overwintering females are fertilized by the last brood of males during the autumn, and the eggs are deposited soon after the spring dispersal flight. In certain localities at least, there is a period when the species exists only in the larval stage, all the adults having died after egg deposition. There is probably only a single laying at this time.

Under laboratory conditions the great majority of eggs are deposited between sunset and eleven o'clock in the evening (vicinity of Chico, California, May to August), although our records show layings later at night and a few during late afternoons of highly overcast humid days. The average number of eggs deposited by *Anopheles freeborni* Aitken is slightly in excess of 200, with 385 as the maximum for one laying.[30] The same average and somewhat smaller total number for one laying was observed for *Anopheles punctipennis* (Say), a maximum of 321, with an average of 203.[31] During 1937 the largest number in one batch for this species was 352, observed by Aitken. One *A. punctipennis* (Say) female deposited 500 eggs in our laboratory in four layings from Mar. 2 to 22, 1938. For *Anopheles pseudopunctipennis franciscanus* McCracken, formerly recorded in California as *Anopheles pseudopunctipennis* Theobold, a maximum of 283 was observed, with an average of 151. At least three batches of eggs may be laid during the lifetime of a female. It is of interest to note that in one of our observations a female *A. freeborni* deposited 174 eggs in 19 minutes, an egg every six to seven seconds with intervening periods of rest. During the entire operation the female resting on the surface of the water remained motionless except for the monotonous jerking of the abdomen when the egg was released. The eggs fell in a heap beneath the insect, pearly white in color, toppling over and forming geometrical patterns and becoming deep brownish black in about 45 minutes.

Egg characters. The characters of anopheline eggs used in classification are: presence or absence of floats, position and length of the float, presence or absence of frill, and pattern. (Fig. 73) Christophers and Barraud[32] classify anopheline eggs as of four types:

1. Eggs probably of primitive type with full-float surrounding egg,
2. Eggs with terminal frill (*pseudopunctipennis franciscanus* of Herms and Freeborn),
3. Whale-back eggs with floats separated from dorsal surface,
4. Various types of boat-like eggs with floats touching margin of dorsal surface.

The egg of the Californian *A. freeborni* Aitken (Fig. 72) is fusiform, slightly rounded at each end and tapering to the extent that one is slightly broader than the other (Herms and Frost).[31] The upper surface is flattened with a slight longitudinal concavity, while the lower surface is broadly convex, the convexity becoming more pronounced at the broad end of the egg. The upper surface is granular, bordered by a laterally striated frill 16μ in width, except at the floats, while the lower surface shows, under proper light, a silvery reticulation. Medianly placed are two roughly oval lateral floats, each divided in a majority of cases into 12 scalloped compartments. The larger part of the area covered by these floats is on the lateral faces of the egg, but they project dorsally over the margins, which are described as "gunwales" rather aptly by one author who likens the egg to a boat. The eggs range in length from 596μ to 656μ. The floats vary in length from 122μ to 224μ.

Falleroni, Martini, Hackett, and Missiroli (see Hackett[33] and Hackett and Lewis[34]) have shown that *Anopheles maculipennis* Meigen actually comprises seven races in Europe, which can be distinguished only by egg patterns, namely var. *melanoön* Hackett, var. *messeae* Falleroni, var. *typicus* Meigen, var. *atroparvus* van Thiel, var. *labranchiae* Falleroni, and var. *subalpinus* Hackett and Lewis; another, var. *elutus* (= *A. sacharovi* Favr.) has solid leaden-colored eggs (Fig. 73).

The eggs of *A. punctipennis* (Say) resemble those of the Californian *A. freeborni* Aitken with these exceptions: in the *punctipennis* form the "frill" extends along the margins of the egg without interruption at the site of the floats which are located on the upper portion of the ventral surface, and extending farther along the sides of the egg are a greater number of compartments, ranging from 16 to 22, which do not converge in fan-wise fashion as in *freeborni*.

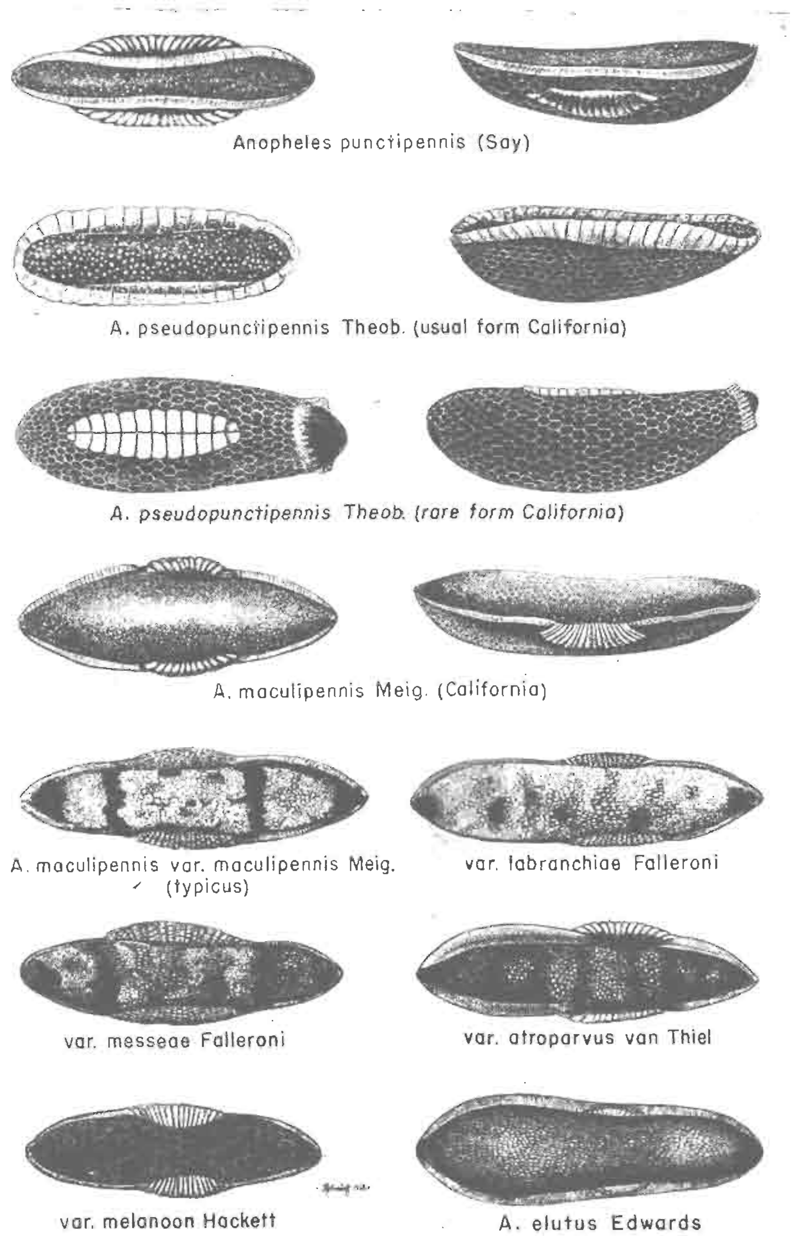


FIG. 73. Eggs of certain anopheline mosquitoes, including racial forms. California *A. maculipennis* Meig. should read *A. freeborni* Aitken, and *A. pseudopunctipennis* should read *A. pseudopunctipennis franciscanus* McCracken. (European races of *A. maculipennis* redrawn after Hackett.)

The eggs of *Anopheles pseudopunctipennis franciscanus* McCracken occur in at least two different forms in California. Herms and Freeborn[30] described the eggs as having the upper surface nearly flat, showing little concavity longitudinally although the lower surface shows a marked convexity. Both ends of the egg are rounded, one being considerably broader than the other. The floats are represented by a fusiform closely appressed area, approximately 270μ long, lying on the dorsal side of the blunt end. This area is divided medianly by a line which is assumed to be the line of contact of the two floats that have been forced up from

FIG. 74. Typical breeding place of California *Anopheles freeborni* in sunlit weed-grown ditch with slowly moving water.

the sides. Lateral lines mark off each longitudinal half of the area into 12 sections representing the original compartments of the lateral floats. This area is so appressed that its position is not distinguishable from a lateral view. Near the narrow end of the egg the membranous covering flares out from the body of the egg to form a translucent, striated collar which completely encircles the end, with the exception of a triangular incision down the dorsal median line in a manner which reminds one of an oversized dress collar (Fig. 72). The egg hangs at an angle in the water, supported by surface tension on the "collar." The eggs ranged in length from 512μ to 528μ. Rozeboom[56] states that the eggs of *A. pseudopunctipennis* Theobald in Panama resemble the eggs of the California species as described by Herms and Freeborn, except that in the Panamanian form the floats are large and have many float ridges; the collar-like frill being identical. The Panamanian eggs ranged in length from 480μ to 573μ, the mean being 520μ.

A second form of *A. pseudopunctipennis* egg, probably var. *boydi* Vargas was described by Herms and Frost (*loc. cit.*) as follows: floats are not only present but completely encircle the egg, which lies flat upon

FIG. 75. Breeding place for Californian *Anopheles freeborni* in overflow from river. Larvae particularly along margins and in hoofprints at edge. (Photograph by L. L. Williams, Jr.)

the surface of the water with the floats extended in nearly every instance (Fig. 73). The floats average from 45 to 47 in number, and from 15μ to 25μ in width, being wider along the sides of the egg and narrowed at the ends. The length of the eggs over all ranged from 510μ to 544μ.

Breeding habits. The breeding habits of anophelines differ considerably for even very closely related species, e.g., the American *Anopheles freeborni* Aitken and *A. quadrimaculatus* Say, both four-spotted anophelines separable with accuracy as adults only on differences in male terminalia, have widely different breeding requirements, the former, at least in California, breeding largely in open sunlit shallow seepage water (Figs. 74 and 75) and the latter in impounded water with floating debris and aquatic vegetation. The European races of *A. maculipennis*

Meigen already referred to emphasize the need of accurate knowledge as to breeding habits.

The following example illustrates the very great importance of knowing the breeding habits in the conduct of malaria control operations. Williams[36] points out that in the Federated Malay States *Anopheles umbrosus* Theobald is the vector of malaria in the coastal plain, breed-

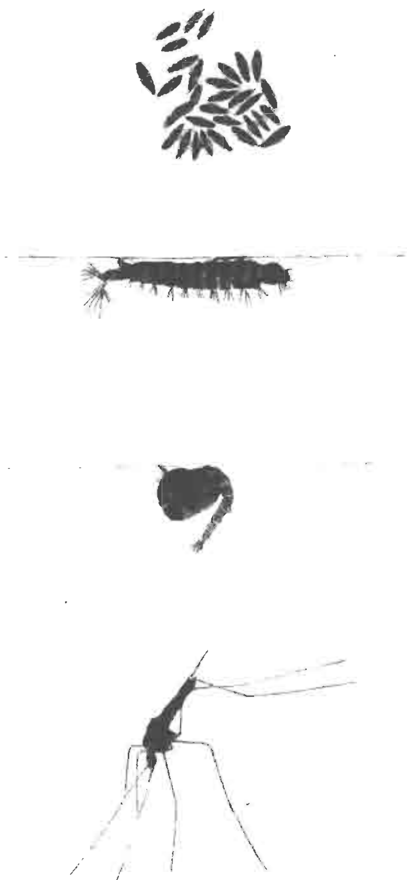


FIG. 76. Showing life history of a mosquito, *Anopheles maculipennis*.

ing in practically stagnant water densely shaded by mangrove. Its production is controlled, as Williams points out, by clearing the swamps and letting in the brilliant sunshine, or by cutting ditches and confining the water to definite channels. The same type of work when practiced on high inland plateaus increases the malaria rate, because here the vector is *Anopheles maculatus* Theobald, which prefers the quiet edges of trickling streams in the open sunshine. *Anopheles minimus* Theobald, the principal vector of malaria in the Philippines, breeds in small flowing streams in the foothills (Russell[37]). Several species of *Anopheles* (though unimportant as vectors) are tree-hole breeders, viz.: *A. plumbeus* Stephens (European) and *A. barberi* Coq. (American). *A. bellator* D. & K., an important Caribbean vector, breeds in collections of water among bromeliads.

An unusual situation is reported for *Anopheles sergenti* (Theobald), a north African and Palestinian species, the larvae of which inhabit small pools and springs among stones at the edge of the lake (Tiberias). The larvae are often under the stones and not easily found (Buxton[38]).

Life history of anophelines. Although there is much variation in the life histories of the species of *Anopheles* mosquitoes (Fig. 76) as well as considerable variation within the species due to temperature and other factors, the length of time required for development from egg to adult is generally longer than in other genera, except in genera where the egg stage may be greatly prolonged, as in *Aedes*.

Incubation period. In a series of tests in which about 20,000 eggs of *Anopheles freeborni* Aitken[39] were used the incubation period at room temperature of 70° F ± 3° was about 72 hours. Under field conditions the incubation period ranged from two to four days, with an average of 2.5 days, under which conditions the incubation period of *Anopheles punctipennis* (Say) ranged from two to six days with an average of 3.2 days.

The eggs of *A. freeborni* removed from the water and dried at temperatures of 74° F and 65° F remained viable after a period of desiccation not over 72 hours. No hatching was obtained from eggs of *A. punctipennis* (Say) after 24 hours' desiccation.

Larval period. Hatching generally took place during the evening and night in the experiments cited. With yeast as food in distilled water at pH of 6.6 to 7.6 the larvae of *A. freeborni* reached the pupal stage in 15 to 16 days.

Pupal stage. The pupal stage requires about three days. Thus the entire life history from egg to adult in *A. freeborni* under experimental conditions requires about 21 to 22 days; the same is true for *A. punctipennis* (Say) and *A. punctipennis franciscanus* McC. Under field conditions this period may be considerably prolonged. Adult mosquitoes reared in the laboratory did not begin oviposition until 13 to 15 days after receiving a blood meal.

The life cycle of *Anopheles albimanus* Wiedemann, the important vector of malaria of the Panama Canal Zone, has been carefully studied by Rozeboom.[40] With room temperature between 27° and 32° C and water temperature for larvae from 21° to 27° C, and eggs and pupae at 27° to 30° C, the entire cycle (egg to adult) required from 18 to 24 days, an average of three weeks. A period of seven days, or a little over, was necessary for the development of the ovaries, an average of 435 eggs being deposited; the incubation period was 40 to 48 hours; the larval stage required from 6 to 22 days, usually 8 to 13 days in hay infusion water; the pupal stage took 30 to 33 hours; the longest observed adult life of a female was 31 days, and of a male, 27 days.

Anopheles quadrimaculatus Say occurs in the United States from Mexico to Canada throughout the Mississippi Valley and east to the Atlantic. It is the chief vector of malaria in the eastern, central, and southern United States. The wings have four distinct spots. According to Williams (*loc. cit.*):

> It breeds almost wholly in still water that is relatively clean. It requires some sunshine, never being found in dense shade. However, it requires some darkness, for it is never found in waters which are wholly unshaded, unless they have a type of flotage which casts narrow strips of shade where the larvae may lie during a portion of the daylight hours. . . . An ideal breeding place for *A. quadrimaculatus* Say is in freshly impounded water which floods a basin containing underbrush and which is sparsely covered with trees. Such a body of water quickly gathers flotage of dead and dying land vegetation, twigs and leaves, among which algae soon appear. Such flotage not only offers the requisite amount of shade, but an abundant food supply. Such an impounding will not acquire a large quantity of natural enemies, such as top minnows and aquatic insects, for a number of years and seldom acquires enough entirely to prevent production of the mosquito.
>
> The normal detritus passing down a narrow stream will clog the interstices of a fallen tree or branch and create a dam. These natural impounded waters are excellent breeding places for *quadrimaculatus*. Swamps covered by a growth of virgin timber, on the other hand, are not good breeding places. Such swamps are almost invariably covered with such a dense timber growth that sunlight can reach the surface of the water only in those small areas where an opening has been made by the fall of a dead tree. Swamps of this description have a small seeding of *quadrimaculatus*, but not enough to propagate malaria. When the lumberman enters, cutting out the large trees, leaving the small ones, the branches and the tree tops, he changes a safe water surface into one almost ideal for *quadrimaculatus* production. He has let in the sunshine without removing all of the shade, and he has left behind waste which not only creates fine flotage, but large portions of which tend to clog the channel which traverses the average swamp, thus making a series of ponds.

The brood peaks of this species in southwestern Georgia according to Boyd[41] are from 20 to 30 days apart, and there are from 8 to 10 annual broods, the first appearing from 20 to 30 days after the last frost, and the last brood, the tenth, if there is favorable weather, in

December. January and February, he states, are the only months when no broods emerge. An excellent description of the techniques used in large-scale rearing of *Anopheles quadrimaculatus* Say is given by Greenwald, Cochran, and Scharff.[42]

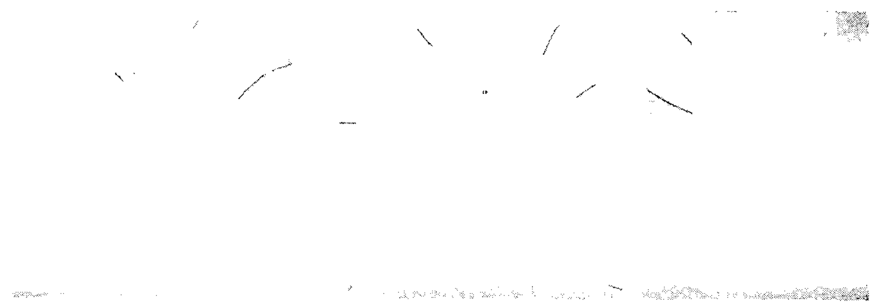
Biting habits. In determining the relative importance of various species of mosquitoes as vectors of diseases, a knowledge of their biting habits is essential. They may be preponderantly *zoöphilous,* feeding on animals other than man, or preponderantly *anthropophilous* i.e., feeding on man; they are seldom exclusively either. That a species is a strong vector of any given infection is probably not always due to an innate capacity but more probably due to a larger degree of domesticity. A close and constant contact between the vectors of a disease and the human victims is essential to extensive transmission of the disease. There are some species, such as *Anopheles minimus flavirostris* (Ludlow), an important vector of malaria in the Philippine Islands, and *A. albimanus* Wied., a vector in the Caribbean area, which do not remain indoors during the day after feeding indoors at night, an important factor in making malaria surveys.

It is often pointed out that the fact that certain species of mosquitoes predominate indoors or are the sole entrants must be weighed against the production and proximity of these and other species under comparison. Carter, Le Prince, and Griffitts[43] have observed that *Anopheles quadrimaculatus* Say is much more often found in residences than *A. punctipennis* (Say). Mayne (*Mitzmain*)[44] in a table comparing the collections of *A. punctipennis* (Say) and *A. quadrimaculatus* Say, totaling 1,377 specimens, compares the numbers of each species taken inside dwellings, under dwellings, in privies, vacated buildings, cattle sheds, horse and mule sheds, fowl roosts, and wagon and tool sheds. In commenting on his results he states:

> It is indicated that in the three sources of direct human influence, namely, inside dwellings, under dwellings, and in privies, the last produced the greatest number of specimens of *A. punctipennis.* This species comprised less than one-third (30 per cent) of the catch in houses, while under dwellings 62 per cent of the mosquitoes collected proved to be *A. punctipennis.* . . . It has been observed by service officers, conducting malarial surveys, that although *A. punctipenis* rarely bites while inside a dwelling, it is found to attack persons seated on the porch or gallery of the house, after which these mosquitoes seek rest, presumably under the house in preference to the interior.

During the summer of 1920, from May 12 to July 13, daily collections of mosquitoes were made by the author and his associates at Vina (Northern Sacramento Valley, California), one series being indoors and the other outdoors. The indoor collections were made regularly in the same buildings—a cowshed, washhouse, showerbath, storehouse, and dwelling, whereas the outdoor collections were made under a short wooden bridge within 10 to 12 feet of an aggregation of shacks occupied by Chinese and Negroes. The indoor series taken in an area where control measures were in progress does not represent a large number of mosquitoes, but the fact remains that of 77 anophelines collected, 50 were *Anopheles freeborni* Aitken (including only one male) and 27 were *A. punctipennis* (Say) (including two males), or practically twice as many *A. freeborni*. On the other hand, the outdoor series represented a total of 343 anophelines of which 102 were *A. freeborni* (42 males and 60 females) and 241 were *A. punctipennis* (130 males and 111 females) or something over twice as many *A. punctipennis*.

These collections bear out very well the general observations that



A B C

FIG. 77. Californian Anopheles: (*A*) *A. freeborni*, (*B*) *A. punctipennis*, and (*C*) *A. pseudopunctipennis franciscanus*.

A. freeborni Aitken like *A. quadrimaculatus* Say readily invades houses and is consequently of greater importance as a malaria vector, while *A. punctipennis* (Say) is chiefly an outdoor biter, porch biter, etc., and consequently probably of less importance as a vector. During the entire period of two months the well-screened cottage which was occupied by our party was not invaded a single time by *A. punctipennis* (Say), while *A. freeborni* Aitken was a common visitor. *Anopheles pseudopunctipennis franciscanus* McC. is considered to be a field mosquito in California and is apparently of no consequence as a vector. Methods used to study the feeding habits of mosquitoes are well described by Reeves and Hammon.[45] In this study they used the precipitin test in which mosquital blood smears were tested with the antisera of domestic rabbits against the sera of the horse, cow, sheep, dog, man, and chicken. In Mexico, Central America, and southerly, *Anopheles pseudopunctipennis pseudopunctipennis* Theo. invades human habitations and is an important malaria vector.

Some North American anophelines. *Anopheles freeborni* Aitken (Fig. 77A), *Anopheles occidentalis* (Dyar and Knab), and *Anopheles aztecus*

Hoffman constitute for the present American *maculipennis* group according to Aitken.[46] *Freeborni* is referred to as the brown, unicolorous form of the irrigated western regions, west of the Rocky Mountains, extending northerly into British Columbia and southerly into parts of Arizona and New Mexico, the western tip of Texas, and Baja California (Mexico). *Occidentalis* is referred to as the "silver" or "bronze-tipped" melanic form of the cool western seacoasts, into Alaska, and across the continent through southern Canada and the northern areas of the United States to Connecticut; Vargas contends that the above-indicated easterly range of *occidentalis* is taken over by *earli*. *Aztecus* is the large, long-winged, linear scaled species of the Mexican plateau, at elevations of 5,000 to 7,000 feet and over.

Anopheles quadrimaculatus Say is the common malaria vector of the eastern United States, extending from southern New Hampshire and southern Ontario westerly to Minnesota and south to Vera Cruz, Mexico. It is particularly abundant in the southeastern United States. The wing scales are all dark, some forming four blackish spots; palpi and hind tarsi are black; femora have small white spots at tip, "knee spots."

Anopheles punctipennis (Say) (Fig 77B) is said to be the most widespread anopheline in North America, ranging from southern Canada to the Mexican plateau. The wings have black and yellow scales, the latter forming two spots on the costal margin, one of which is long and situated beyond the middle, the second smaller and near the apex, giving a mottled appearance to the wings. The proboscis is black, and the palpi are unbanded. The general appearance of the body is dark brown. This species breeds in clear, cool, shaded pools. The females seldom enter dwellings but invade unscreened porches and bite in the open.

Anopheles pseudopunctipennis Theobald is a widespread plains and highland species, extending from Argentina along the Andean region of South America, through Central America, Mexico, and the south central United States. It resembles *A. punctipennis* (Say) somewhat, but is easily separated by the white banding of the palpi. The tip of each palpus is white. It breeds primarily in sunlit pools along the courses of receding streams, the larvae being exceedingly abundant in the mats of green algae.

Anopheles pseudopunctipennis franciscanus McCracken (Fig. 77C), together with the var. *boydi* Vargas, is very abundant in many parts of California where it was previously recorded as *Anopheles pseudopunctipennis* Theobald; it also occurs in Nevada, Utah, Arizona, New Mexico, western Texas, and Baja California. Its breeding habits resemble those of *pseudopunctipennis*. It differs from the latter in that the tips of the palpi are black. It rarely enters human habitations.

Anopheles crucians Wiedemann, *Anopheles bradleyi* King, and *Anopheles georgianus* King resemble each other very closely and constitute the "*crucians*" complex. The palpi of all three species are banded with white, and the last segment is entirely white. Separation of the species can be made only in the larval stage. *Crucians* has been found from Massachusetts southwesterly to Kansas, Oklahoma, and Texas. It breeds in ponds, lake margins, swamps, and pools of both intermittent and permanent character; it breeds extensively in acid waters as in the cypress swamps of Florida and Georgia (Matheson). The females bite freely during the day. *Bradleyi* occurs along the Atlantic and Gulf coasts from Maryland to Vera Cruz, Mexico. It normally breeds in saline waters. *Georgianus* occurs widely in fresh water in the Gulf states. It can be distinguished from the other two members of the complex with difficulty.

Anopheles barberi Coquillett occurs in the eastern United States from New York southward. It breeds commonly in water collected in tree holes, but occasionally in artificial containers.

Anopheles walkeri Theobald is a widely distributed species in the eastern United States to the Gulf of Mexico and north to Minnesota. The presence of white "knee" spots and the golden color of the halters distinguish it from *A. atropos* D. and K.

Anopheles atropos Dyar and Knab is strictly a salt-water mosquito confined to the shores of the Gulf of Mexico from Florida to Louisiana. It is small and very dark with unspotted wings. It lacks white knee spots. Within its range it is said to outnumber *A. crucians* Wied. three to one and breeds by preference in permanent salt pools or in shallow water on muck or alluvial marshes. It is reported to be a tormenting pest, biting in direct sunlight as well as by night.

Anopheles albimanus Wiedemann, is considered to be the most important anopheline species in the Caribbean region. The hind tarsi are conspicuously white-banded; the apical segment of the palpi is entirely white. It develops freely in brackish or salt water. This species has been reported from several points in the extreme southern parts of the United States, e.g., Key West, Florida, and the Rio Grande Valley (Brownsville), Texas.

Key to Culicid Tribes and Genera of the United States

(Prepared by T. H. G. Aitken after various authors)

1. Abdomen without scales, or at least with the sternites largely bare. Scutellum never trilobed; crescent-shaped with the marginal setae evenly distributed. Palpi (males and females) as long or almost as long as the proboscis. Tribe Anophelini, Genus *Anopheles* Meigen 1818
Abdomen with both tegites and sternites completely clothed with

scales. Scutellum trilobed (except Megarhinini–spurious vein extends toward base of wing in upper basal cell from angle of R_{4+5}). Palpi (females) much shorter than the proboscis 2

2. Proboscis rigid, outer half more slender and bent backward ....Tribe Megarhinini, Genus *Megarhinus* Robineau-Desvoidy 1827
Proboscis more flexible, of uniform thickness (unless swollen at tip), outer half not bent backward ....Tribe Culicini 3
3. Postnotum with setae ....Genus *Wyeomyia* Theobald 1901
Postnotum without setae 4
4. Wing membrane without microtrichia. Cell R_2 shorter than its stem; anal vein ends about opposite base of cubital fork ....Genus *Uranotaenia* Lynch-Arribalzaga 1891
Wing membrane with microtrichia. Cell R_2 longer than its petiole. Anal vein extending well beyond fork of cubitus 5
5. Postspiracular bristles present 6
Postspiracular bristles absent 8
6. Prespiracular bristles present (even if few) ....Genus *Psorophora* Robineau-Desvoidy 1827
Prespiracular bristles absent 7
7. Wing scales mostly narrow, or when broad, setae are present on upper side of base of R_1 (1st vein) ....Genus *Aedes* Meigen 1818
Wing scales broad; setae absent on uppper side of R_1 .... Genus *Mansonia* Blanchard 1901 (Subgenera *Mansonia, Mansonioides,* .... *Rhynchotaenia*)
8. Prespiracular bristles present; lower side of base of R_1 (1st vein) distinctly pilose ....Genus *Culiseta* Felt 1904
Prespiracular bristles absent; lower side of base of R_1 scaly or bare 9
9. Pulvilli present 10
Pulvilli absent 11
10. Second joint of antenna (first flagellar) very long in both sexes; antenna of male not plumose ....Genus *Deinocerites* Theobald 1901
Second joint of antenna normal; antenna of male nearly always plumose .... Genus *Culex* Linnaeus 1758
11. Fourth joint of front tarsus very short (both sexes); first segment of front tarsi longer than the last four together ....Genus *Orthopodomyia* Theobald 1904
Fourth joint of front tarsus not shortened in female; first segment of front tarsi not longer than last four together ....Genus *Mansonia* Blanchard 1901 (Subgenus *Coquillettidia*)

BIBLIOGRAPHY

1. Edwards, F. W., 1932. *Diptera, Family Culicidae.* Brussels: Genera Insectorum de P. Wytsman, 194me Fascicule. 256 pp. + 5 plates.

2. Matheson, Robert, 1944. *Handbook of the Mosquitoes of North America.* 2nd. ed. Ithaca: Comstock Publ. Co., Inc. viii + 314 pp.

3. Russell, Paul F.; Rozeboom, Lloyd E.; and Stone, Alan; 1943. *Keys*

to the Anopheline Mosquitoes of the World, with Notes on Their Identification, Distribution, Biology, and Relation to Malaria. Philadelphia: Am. Entomolog. Soc. 152 pp.

4. Freeborn, S. B., 1924. "The terminal abdominal structures of male mosquitoes," *Am. J. Hyg.*, 4:188–212.

5. Ross, Edward S., and Roberts, H. Radclyffe, 1943. *Mosquito Atlas: Part I. The nearctic Anopheles, important malaria vectors of the Americas, and Aedes aegypti, Culex quinquefasciatus.* iv + 44 pp. *Part II. Eighteen old world anophelines important to malaria.* iv + 44 pp. Philadelphia: Am. Entomolog. Soc.

6. Komp, W. H. W., 1942. "A technique for staining, dissecting, and mounting the male terminalia of mosquitoes," U. S. Public Health Service, *Pub. Health Rep.*, 57:1327–33.

7. Howard, L. O., 1900. *Notes on the Mosquitoes of the United States.* Washington, D. C.: Dept. Agric., in Div. Entomol. Bull., no. 25, p. 16.

8. Dyar, H. G., 1928. *The Mosquitoes of the Americas.* Carnegie Inst. Washington, Publ. no 387. 616 pp.

9. Christophers, S. R., and Puri, I. M., 1929. "Why do Anopheles larvae feed at the surface, and how?" *Tr. Far East. Assoc. Trop. Med.*, Seventh Congress held in India, 2:736–38.

10. Frost, Florence M.; Herms, W. B.; and Hoskins, W. M.; 1936. "The nutritional requirements of the larva of the mosquito, *Theobaldia incidens* (Thom)," *J. Exper. Zoöl.*, 73:461–79.

11. Gray, H. F., 1936. "Control of pest mosquitoes for comfort," *Civil Engineering*, 6:685–88.

12. Stage, H. H.: Gjullin, C. M.; and Yates, W. W.; 1937. "Flight range and longevity of floodwater mosquitoes in the lower Columbia River Valley," *J. Econ. Entomol.*, 30:940–45.

13. Eyles, Don E.; Sabrosky, Curtis W.; and Russell, John C.; 1945. "Long-range dispersal of *Anopheles quadrimaculatus*," U. S. Public Service, *Pub. Health Rep.* 60:1265–73.

14. Reeves, W. C., 1948. "A final summary of flight range studies on *Culex tarsalis* and notes on wild bird malaria in Kern County." *Proc. and Papers of 16th Ann Conf. Calif. Mosq. Control Assn.* (Berkeley), pp. 58–59.

15. Mayne, B., 1922. "How long does a mosquito retain malaria parasites?" U. S. Public Health Service, *Pub. Health Rep.* 37:1060–63.

16. Freeborn, S. B., 1932. "The seasonal life history of *Anopheles maculipennis* with reference to humidity requirements and hibernation.' " *Am. J. Hyg.* 16:215–23.

17. Nuttall, G. H. F., and Shipley A. E., 1903. "Studies in relation to malaria: Structure and biology of anophelines," *J. Hyg.*, 3:166–215.

18. Williams, F. X., 1931. *The insects and other invertebrates of Hawaiian sugar cane fields.* Exper. Sta. Hawaiian Sugar Planters' Assn. 400 pp. (p. 279).

19. Woke, P. A., 1937. "Comparative effects of the blood of man and of canary on egg-production of *Culex pipiens* Linn," *J. Parasitol.*, 23:311–13.

20. Smith, John B., 1902. *The salt marsh mosquito, Culex sollicitans Wlk.* New Jersey Agric. Exper. Sta., Special Bulletin T.

21. Freeborn, Stanley B., 1926. *The mosquitoes of California.* Univ. Calif., "Technical Bulletins." *Entomology,* 3:333–60.

22. Feng, Lan-Chou, 1938. "The tree hole species of mosquitoes of Peiping, China," *Chinese M. J.,* supp. 2, pp. 503–25.

23. Dyar, Harrison G., 1916. "New Aedes from the mountains of California," *Insec. Inscit. Mens.,* 4:80–90.

24. Jachowski, Leo A., Jr., and Schultz, Carlos, 1948. *Notes on the biology and control of mosquitoes at Umiat, Alaska.* Nav. Med. Research Inst., Nav. Med. Center, Bethesda, Md., Project NM 005-017, Rep. no. 1. 17 pp.

25. ———, and Schultz, Carlos, 1948. "Notes on the biology and control of mosquitoes at Umiat, Alaska," *Mosq. News,* 8:155–65.

26. Jenkins, Dale W., 1948. "Ecological observations on the mosquitoes of central Alaska," *Mosq. News* 8:140–47.

27. Hindle, Edward, 1914. *Flies in Relation to Disease: Blood-sucking flies.* London: Cambridge Univ. Press. xv. + 398 pp.

28. Woke, P. A., 1937. "Cold-blooded vertebrates as hosts for *Aedes aegypti* Linn.," *J. Parasitol.,* 23:310–11.

29. Hoffman, W. A., 1926. "Resting position of Haitian Anopheles," *Am. J. Trop. Med.,* 6:377–79.

30. Herms, W. B., and Freeborn, S. B., 1920. "Egg-laying habits of California anophelines," *J. Parasitol.,* 7:69–79.

31. Herms, W. B., and Frost, Florence M., 1932. "A comparative study of the eggs of Californian anophelines," *J. Parasitol.,* 18:240–44 (3 plates).

32. Christophers, S. R., and Barraud, P. J., 1931. "The eggs of Indian Anopheles, with description of the hitherto underscribed eggs of a number of species," *Records Malaria Survey India,* 2:161–92 (5 plates).

33. Hackett, L. W., 1934. "The present status of our knowledge of the subspecies of *Anopheles maculipennis,*" *Tr. Roy. Soc. Trop. Med. & Hyg.,* 28:109–28.

34. Hackett, L. W., and Lewis, D. G., 1935. "A new variety of *A. maculipennis* in Southern Europe," *Riv. di Malariol.,* 14:377–83.

35. Rozeboom, L. E., 1937. "The egg of *Anopheles pseudopunctipennis* in Panama," *J. Parasitol.,* 23:538–39.

36. Williams, L. L., Jr., 1937. "Mosquitoes and malaria," *J. Econ. Entomol.,* 30:20–26.

37. Russell, P. F., 1932. "The control of *Anopheles minimus* mosquito larvae in the Philippines by stranding and flushing," *Philippine J. Sc.* 47: 439–45.

38. Buxton, P. A., 1924. "Applied entomology of Palestine, being a report to the Palestine Government," *Bull. Entomolog. Research,* 14:289–340 (5 plates).

39. Herms, W. B., 1929. "Anopheline mosquito investigations in California," Fourth Internat. Congress of Entomology, Ithaca, August, 1928, 2:708–21.

40. Rozeboom, L. E., 1936. "The life cycle of laboratory-bred *Anopheles albimanus* Wiedemann," *Ann. Entomolog. Soc. Amer.,* 29:480–89.

41. Boyd, Mark F., 1929. "Studies on the bionomics of North American

anophelines. I. The number of annual broods of *A. quadrimaculatus*," *Am. J. Hyg.*, 7:264–75.

42. Greenwald, Margaret; Cochran, J. H.; and Scharff, Donald K.; 1948. "Large scale rearing of *Anopheles quadrimaculatus* Say at Orlando, Florida," *Mosq. News*, 8:50–56.

43. Carter, H. R.; Le Prince, J. A. A., and Griffitts, T. H. D.; 1916. *Impounded water*. Washington, D. C.: Govt. Print. Office, in Pub. Health Bull., no. 79. 34 pp.

44. Mitzmain, M. B., 1917. "Anopheline mosquitoes, their distribution and infection under field conditions," U. S. Public Health Service, *Pub. Health Rep.* 32:536–40.

45. Reeves, W. C., and Hammon, W. McD., 1943. "Feeding habits of the proven and possible mosquito vectors of western equine and St. Louis encephalitis in the Yakima Valley, Washington," *Am. J. Trop. Med.*, 24:131–34.

46. Aitken, Thomas H. G., 1945. "Studies on the anopheline complex of Western America," *Univ. Calif., Publ. in Entomol.* 7:273–364.

CHAPTER XII

MOSQUITOES AS VECTORS OF DISEASE

A. THE MALARIAS

Human Malaria. Malaria is a widely distributed disease, prevalent to a greater or less degree on every continent and on many islands of the seas. It is considered to be the most important disease of man. Whether or not malaria existed in North America prior to the discovery of the continent has not been definitely established; however, this disease was evidently recognized as a factor in colonization on the Massachusetts coast and the Georgia-Carolina coast as early as the middle of the 17th century.[1] In the United States, where malaria is on a rapid decline, there were an estimated 900,000 cases as late as 1935; approximately 3,900 deaths were reported for 1934 in 13 southern states.[2] The number of deaths in 1944 due to malaria was reported to have been 584, of which 540 occurred in the 14 southern states.[3]

The presence of endemic malaria is dependent upon a complex of environmental factors favorable to the development of large numbers of vector (anthropophilous) mosquitoes, as well as to the *Plasmodia* causing the disease. Temperature, particularly as it affects the development of the plasmodium in the mosquito, and temperature combined with humidity as it affects the life of the vector, are critical factors; a mean summer isotherm of 15° to 16° C in general limits its geographical distribution fairly well. The distribution of malaria is dependent upon the availability of water for mosquito breeding, not necessarily heavy rainfall; naturally arid regions may be seriously affected because of imperfections in irrigation if this is practiced. Although lowlands are more likely to be affected, this does not hold as a general rule, because if one or more important factors are lacking in a lowland region the area is nonmalarious. The disease may occur at high elevations (9,000 feet in Quito) under favorable circumstances. The writer found endemic malaria in California at an elevation of about 5,500 feet; it is present in Mexico at an elevation of near 7,500 feet.

Few diseases have so large a list of synonyms; among these are ague, chills and fever, jungle fever, paludism, marsh fever, remittent fever, intermittent fever, *Wechselfieber, Kaltesfieber,* ètc. The symptoms are

anophelines. I. The number of annual broods of *A. quadrimaculatus*," *Am. J. Hyg.*, 7:264–75.

42. Greenwald, Margaret; Cochran, J. H.; and Scharff, Donald K.; 1948. "Large scale rearing of *Anopheles quadrimaculatus* Say at Orlando, Florida," *Mosq. News,* 8:50–56.

43. Carter, H. R.; Le Prince, J. A. A., and Griffitts, T. H. D.; 1916. *Impounded water.* Washington, D. C.: Govt. Print. Office, in Pub. Health Bull., no. 79. 34 pp.

44. Mitzmain, M. B., 1917. "Anopheline mosquitoes, their distribution and infection under field conditions," U. S. Public Health Service, *Pub. Health Rep.* 32:536–40.

45. Reeves, W. C., and Hammon, W. McD., 1943. "Feeding habits of the proven and possible mosquito vectors of western equine and St. Louis encephalitis in the Yakima Valley, Washington," *Am. J. Trop. Med.,* 24:131–34.

46. Aitken, Thomas H. G., 1945. "Studies on the anopheline complex of Western America," *Univ. Calif., Publ. in Entomol.* 7:273–364.

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Human Malaria. Malaria is a widely distributed disease, prevalent to a greater or less degree on every continent and on many islands of the seas. It is considered to be the most important disease of man. Whether or not malaria existed in North America prior to the discovery of the continent has not been definitely established; however, this disease was evidently recognized as a factor in colonization on the Massachusetts coast and the Georgia-Carolina coast as early as the middle of the 17th century.[1] In the United States, where malaria is on a rapid decline, there were an estimated 900,000 cases as late as 1935; approximately 3,900 deaths were reported for 1934 in 13 southern states.[2] The number of deaths in 1944 due to malaria was reported to have been 584, of which 540 occurred in the 14 southern states.[3]

The presence of endemic malaria is dependent upon a complex of environmental factors favorable to the development of large numbers of vector (anthropophilous) mosquitoes, as well as to the *Plasmodia* causing the disease. Temperature, particularly as it affects the development of the plasmodium in the mosquito, and temperature combined with humidity as it affects the life of the vector, are critical factors; a mean summer isotherm of 15° to 16° C in general limits its geographical distribution fairly well. The distribution of malaria is dependent upon the availability of water for mosquito breeding, not necessarily heavy rainfall; naturally arid regions may be seriously affected because of imperfections in irrigation if this is practiced. Although lowlands are more likely to be affected, this does not hold as a general rule, because if one or more important factors are lacking in a lowland region the area is nonmalarious. The disease may occur at high elevations (9,000 feet in Quito) under favorable circumstances. The writer found endemic malaria in California at an elevation of about 5,500 feet; it is present in Mexico at an elevation of near 7,500 feet.

Few diseases have so large a list of synonyms; among these are ague, chills and fever, jungle fever, paludism, marsh fever, remittent fever, intermittent fever, *Wechselfieber, Kaltesfieber,* etc. The symptoms are commonly characterized by more or less regularly occurring febrile paroxysms. In most cases there are three fairly well-defined stages, viz.: the *cold stage* (the chill) in which the skin becomes pale and has the appearance of "gooseflesh," the patient's teeth may chatter, and he may shiver more or less violently; the next stage is the *hot stage,* or fever, the temperature rising during the chill, the skin is hot and flushed; the third stage is marked by the appearance of a general *perspiration,* the fever falls, and the temperature approaches normal. The entire paroxysm may last but a few hours. In many cases the stages are not well marked, neither do the paroxysms recur at the same intervals. The intervals depend largely on the type of infection. When the paroxysm occurs at intervals of 24 hours, as is often true in the early stages of infection or in multiple infections, it is *quotidian;* when the interval is 48 hours or every third day it is *tertian;* and when the interval is 72 hours or every fourth day, it is *quartan.*

The disease is caused by an infection with one or more of several species of blood-inhabiting protozoa (Class Sporozoa) belonging to the genus *Plasmodium.* Under the clincal term "malaria," there are really combined four or more diseases, which, while caused by closely related parasites and having a common means of transmission, nevertheless possess individual characteristics, namely: *vivax* malaria, tertian; *falciparum* malaria, estivoautumnal; *quartan* malaria, quartan fever; *ovale* malaria, ovale tertian.

The plasmodial parasites attack the red blood corpuscles, destroying them while undergoing asexual reproduction; this asexual reproduction or sporulation occurs at more or less regular intervals, i.e., 48 or 72 hours, depending upon the species of *Plasmodium.* The infection, according to Reed,[4] results in (1) changes in organs, such as enlargement of the spleen and liver, and heart involvements; in fatal cases of subtertian malaria capillaries in the brain and pia are found congested or blocked by schizonts and sporulating forms of plasmodia, with punctiform hemorrhages in the white matter of the cerebral cortex; (2) leucopenia with increase of mononuclears and varying degrees of anemia as the result of direct destruction of red cells by plasmodia and indirect degeneration of others; (3) malarial pigment (*melanin*) in macrophages in the splenic sinuses is characteristic, as are also the heavy pigment deposits in the cells in the splenic vein and liver; it is the same pigment as that produced in the red cells by the plasmodia and released with the rupture of infected red cells; (4) changes in physiology such as periodic febrile paroxysms; these are quite regular in benign tertian malaria, but because of irregular maturing of plasmodia the periodicity of the paroxysms is often concealed in falciparum (subtertian) malaria; focal symptomatology due to localization in subtertian [*Plasmodium falciparum* (Welch)] arising from the "sticky tendency" of parasitized red cells which causes agglutination and blockage; (5) malaria cachexia, a chronic condition following repeated malarial attacks. Persons concerned technically with malaria should consult Russell, West, and Manwell's *Practical Malariology*[5] and *Symposium on Human Malaria.*[6]*

Historical. Although the name "malaria" was not employed until the middle of the eighteenth century, the disease was known for many centuries, Hippocrates having divided periodic fevers into quotidian (daily), tertian (every third day) and quartan (every fourth day). The fable of Hercules and the Hydra is believed to refer to malaria. In 1753 Torti named the disease "malaria," believing it to be air-borne and emanating from the bad air (*mal aria*) rising from swamps and marshes. Credit for the discovery of the causal agent (1880) belongs to C. L. A. Laveran, a French army surgeon who was then stationed in Algeria. Although the mosquito transmission theory is said to have been held for many years by Italian and Tyrolese peasants and the natives of what was formerly German East Africa, the first well-formulated mosquito-malaria theory was advanced by Dr. A. F. A. King in 1883.[7] In 1885 and 1886 Golgi[8] discovered that the course of the fevers corresponded to the development of the parasites in the blood corpuscles and particularly to their periodic sporulation. He demonstrated this for both the quartan and terian parasites.

Manson expressed a strong belief in the malaria-mosquito theory as early as 1884, and it was his sustained guidance and encouragement that carried Ronald Ross[9] on to those brilliant discoveries in India in 1897–1898 that definitely incriminated mosquitoes as vectors of malaria, and twice won for him the Nobel prize. Although Ross made important discoveries in the field of human malaria and its anopheline vector, his chief discovery was the complete life cycle of the causal organism of bird malaria in a culicine mosquito; he established the bird-to-mosquito-to-bird cycle.

Credit is due MacCallum[10] for his discovery in 1897 that the flagellated bodies which Ross had found in the intestines of mosquitoes were actually male parasites and that these fertilize the female cells, thus giving the clue to the nature of the pigmented cells in the stomach wall of the mosquitoes. MacCallum actually observed the process of fertilization in blood taken from a patient suffering from aestivo-autumnal malaria. After describing the process of flagellation in the bird infection and entrance of the "flagellum" into the granular round form, MacCallum remarks (*loc. cit.*): "Have we not here, without much doubt, a sexual process in the organisms the result of which is the motile vermiculus. . . . We can thus consider the two forms of adult organism found in the fresh blood as male and female, the granular form containing more

* Also see Boyd, Mark F., 1950. *Malariology*. Philadelphia: W. B. Saunders Co. 2 vols. 1643 pp.

chromatophilic substance than the hyaline male form, which we may now say gives up its life in the production of four or more spermatozoa or flagella."

In an addendum MacCallum states that he had

> examined the blood of a woman suffering from an infection with the aestivo-autumnal type of organism, in which a great number of crescents were to be seen. These in a freshly made slide of blood, with very few exceptions, retained their crescentic shape for only a few minutes. They soon drew themselves up, thus straightened out the curve of the crescent while shortening themselves into the well-known ovoid form. After the lapse of 10 to 12 minutes most of them were quite round and extra-corpuscular, the "bib" lying beside them as a delicate circle or "shadow" of the red corpuscle.
>
> After 20 to 25 minutes certain ones of these spherical forms became flagellated; others, and especially those in which the pigment formed a definite ring and was not diffused throughout the organism, remained quiet and did not become flagellated. In a field where an example of each form could be watched, the flagella broke from the flagellated form and struggled among the corpuscles, finally approaching the quiet spherical form; one of them entered, agitating the pigment greatly, sometimes spinning the ring around. The rest were refused admission, but swarmed about, beating their heads against the wall of the organism. This occurred after 35 to 45 minutes.
>
> After the entrance of the flagellum the organism again became quiet and rather swollen, but although in the two instances in which this process was traced, the fertilized form was watched for a long time, no form analogous to the "vermiculus" was seen.

During this same period of discovery in the field of malaria Grassi[11] and associates proved that human malaria is transmitted by a particular genus of mosquito, namely, *Anopheles*.

In 1900 Sambon and Low, at the suggestion of Manson, built a mosquito-proof hut in the Roman Campagna, in which they lived during the most malarial months of that year without contracting malaria, having taken precautions against mosquito bites by promptly retiring within doors at sunset; otherwise they lived as did the natives. At this time these investigators sent infected *Anopheles* mosquitoes from the Roman Campagna to London, where Manson's son, Dr. P. Thurburn Manson, and Mr. George Warren permitted themselves to be bitten by these mosquitoes and "shortly afterwards both of these gentlemen, neither of whom had been abroad or otherwise exposed to malarial influences, developed characteristic malarial fever, and malarial parasites were found in abundance in their blood, both at that time and on the occurrence of the several relapses of malarial fever from which they subsequently suffered. The mosquito-malaria theory has now, therefore, passed from the region of conjecture to that of fact."[12]

Bass and Johns[13] in 1912 were the first to cultivate successfully the malaria parasite *in vitro*.

The plasmodia. The causal organisms of malaria belong to the genus *Plasmodium* of the family Plasmodiidae, suborder Haemosporidiidea, order Coccidiida, class Sporozoa, Phylum Protozoa. They are blood-inhabiting microparasites, passing the principal portion of their asexual cycle within the red blood cells of the host where they produce, with the aid of hemoglobin, a characteristic malarial pigment (melanin).

If parasites are present in the blood they should be visible, after proper staining, on careful thin-smear microscopic examination, as pigmented intracorpuscular bodies in the form of signet rings, ameboid forms, segmenting forms, or as crescents in estivo-autumnal fever of 10 or more days' duration. An excellent manual by Wilcox[14] is available for the microscopical diagnosis of malaria in man. Of interest in routine examination of malaria blood smears is the use of fluorescent dyes (0.1 per cent aqueous rivanol or berberine sulfate fluorochrome) and then irradiation by ultraviolet ray (Metcalf, 1945[15]).

Ross[16] states that the parasites "will not generally be numerous enough to cause illness unless there is at least one parasite to 100,000 haematids; that is, 50 parasites in 1 cmm of blood; or 150,000,000 in a man 64 kilograms (142 pounds) in weight. . . . Such calculations demonstrate the absurdity of supposing that there are no plasmodia present in a person because we fail in finding one after a few minutes' search. As a matter of fact, even if as many as 150,000,000 plasmodia are present in an average man, the chances are that ten to fifteen minutes' search will be required for each plasmodium found; while if we are careless or unfortunate, we may have to look much longer." The time elapsing from inoculation by the mosquito to appearance of infected erythrocytes (presumably one or more exoerythrocytic cycles) varies with the species of the plasmodium as well as with the strain and other factors. In *falciparum* malaria the incubation period for the Coker strain ranged from 6 to 25 days, while that for the Long strain ranged from 9 to 10 days.

a. *Plasmodium falciparum* (Welch 1897) (*Plasmodium praecox* Blanchard 1900) is the causal organism of *estivo-autumnal* fever (subtertian) of the tropics and subtropics, the most severe form of malaria, often resulting fatally. Although it is a *tertian* fever there is considerable irregularity in the occurrence and duration of the febrile stage owing to a corresponding irregularity in the sporulation of the parasites, schizogony usually requiring about 48 hours, though often less. The infected red corpuscles are usually normal in size, though some may be slightly shrunken, often crenated, and rather dark green (brassy). The intracorpuscular parasite in all its stages is small (not over two-thirds the size of a corpuscle) and fairly ovoid in outline; the pigment is darker than in other forms; clumps early in coarse granules, and "Maurer's dots" appear in the corpuscles in the later stages. The *signet ring* is thin and small and the chromatin dot is commonly double and out of line with the ring. There may be two and even four signet rings in one red corpuscle. The *segmented state*, rarely if ever seen in the peripheral blood, produces from 8 to 24 *merozoites*. Characteristic crescent-shaped or kidney-shaped bodies appear in the peripheral blood in about 10 days after infection; these are the sexual forms (*gametocytes*) and occur as crescents in this species of *Plasmodium* only. The *macrogametocyte* or female form, measuring from 10 to 15μ shows the chromatin granules well concentrated in the mid-region, while the *microgametocyte* or male form, measuring from 7 to 10μ has a more hyaline appearance. A remnant of the red blood corpuscle often remains slung from the opposite ends of the crescent and forms the so-called "bib."

b. *Plasmodium vivax* (Grassi and Feletti 1890) is the cause of tertian fever of temperate climates, which occurs also abundantly in the tropics and subtropics, with regularly recurrent paroxysms every 48 hours. The parsitized corpuscles are distinctly enlarged, quite pale, and usually contain fine pigment granules known as "Schüffner's dots." The signet ring is large and conspicuous and the dot is in line with the ring and rarely double. The fully grown *merocytes* or *schizonts* are very irregular and bizarre in form. The number of elements, *merozoites*, in the sporulating or segmented stage commonly seen in the peripheral blood is from 12 to 24 (usually about 16), and their arrangment is irregular. Sporulation occurs regularly every 48 hours. There are no "crescents" in this species; the gametocytes are round or oval in form, filling practically the entire red cell when full grown. The *macrogametocyte* has the chromatin arranged in a compact mass; the *microgametocyte* has the pigment well distributed and presents a more hyaline appearance.

c. *Plasmodium malariae* (Laveran 1881) is the cause of quartan fever, with recurrent paroxysms every 72 hours. This form of malaria is much less common but coincides in distribution with estivo-autumnal fever. The pigment is coarse and generally occurs in marginal streaks. The parasitized corpuscles are usually normal in size, and the parasite is small and more or less oval in shape though, when partly grown, it frequently extends across the equator of the corpuscle in the form of a band. The ring-forms have one vacuole and usually one dot. The gametocytes are rarely seen. The segmenting stage gives rise to the typical "daisy" form, each sporulated body radiating from the center. The number of bodies varies from 6 to 12, usually 8. Sporulation occurs every 72 hours. The gametocytes resemble those of *Plasmodium vivax*.

Rodhain[17] has shown that *P. malariae* can be transmitted intravenously from man to the chimpanzee and maintains its virulence and specific character for man; also the *malariae type* of plasmodium of the naturally infected chimpanzee is transmissible to man, producing a malaria fever of the quartan type.

d. *Plasmodium ovale* Stephens 1922 is the cause of a mild form of tertian fever in East Africa and probably South America. The name indicates the oval shape which is generally assumed by the parasite as well as the parasitized erythrocyte. The infected corpuscles may become somewhat enlarged. The pigment is dark and granular and "Schüffner's dots" are present in all stages. The merozoites range from 8 to 12 in number.

Life cycle of the plasmodia. The life history of malaria parasites consists of two cycles: first, the *asexual,* also known as the blood cycle, cycle of Golgi,[18] or schizogonic cycle; and, secondly, the *sexual,* also *known as the mosquito cycle, cycle of Ross, or sporogonic cycle.*[19]

The *asexual* cycle (Fig. 78), accomplished in the blood and other tissues of man, begins with the injection of spindle-shaped *sporozoites* (Fig. 78) into the circulation with the bite of an appropriate anopheline mosquito.

Though most diagrams illustrating the life cycle of the malaria parasite show the sporozoite entering the erythrocyte at once (including Fig. 78), actually there has been much speculation for many years as to what *did* happen in the life cycle of the *Plasmodium* between the inoculation of the sporozoite by the mosquito and the appearance of the trophozoite or signet ring in the erythrocyte (Huff[20]). That there are pre-erythrocytic stages in the developmental cycle of *Plasmodium elongatum* Huff and *P. gallinaceum* Brumpt and other bird malaria parasites has been proved by Huff and associates;[21] the first generation following infection by sporozoites being called "cryptozoites" and subsequent generations, "metacryptozoites." These stages "require a period of development in macrophages and related kinds of cells before they invade erythrocytes." Huff[22] raises the question, "Do such stages really exist in human or simian malarial infections?" He cites indirect evidence viz.: "(1) the existence of periods of inapparent parasitism followed by parasitemia in sporozoite-induced infections, and (2) the differences between sporozoite-induced and blood-induced infections with respect to their response to anti-malarial drugs. . . . The evidence is conclusive that the sporozoites and the 3 or 4 generations of development following them do not occur in the blood during this period"; i.e., subinoculations within about half an hour of blood from a heavily sporozoite (mosquito) inoculated malaria patient may occasionally result in an infection; however, following this brief period there is a period of seven or more days (depending apparently on the species of *Plasmodium*) when subinoculations will *not* result in infection.

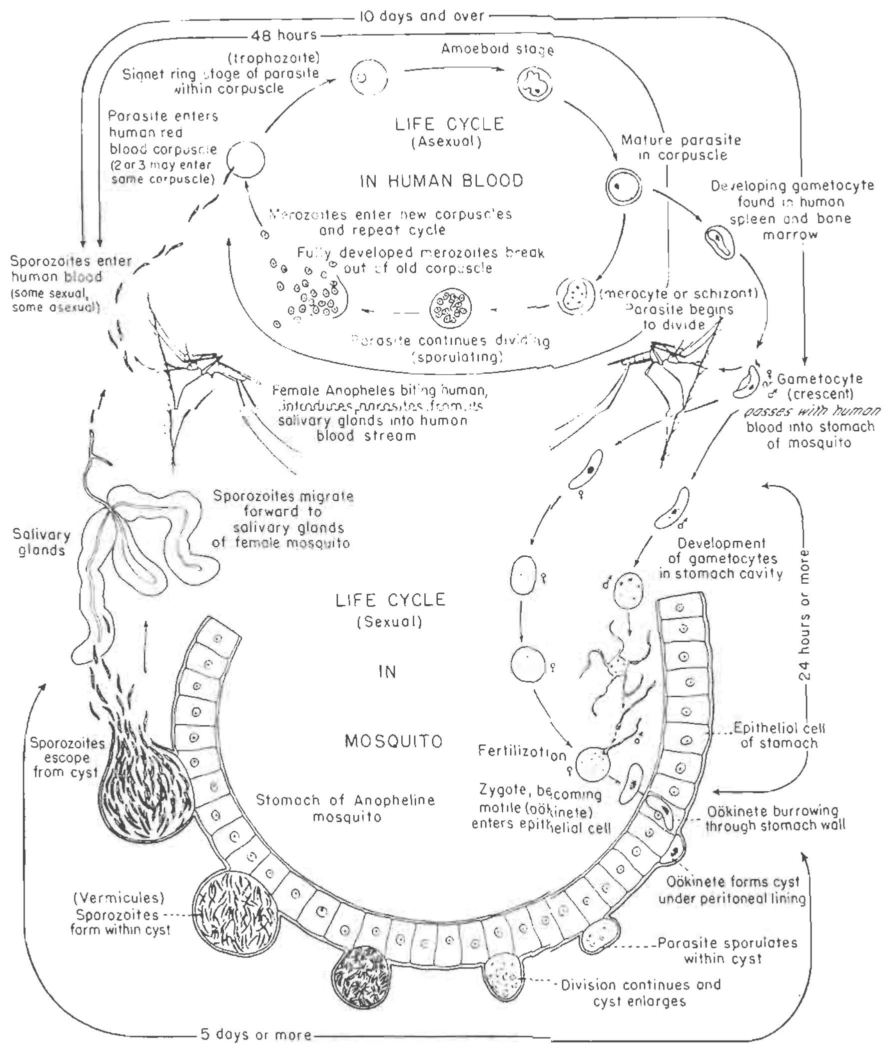


Fig. 78. The life cycle of the malarial parasite, *Plasmodium falciparum*, in man and the anopheles mosquito. (For exoerthrycytic cycles, see text.)

Following the presumable exoerythrocytic stages the cycle continues very much as is shown in the diagram (Fig. 78), i.e., the invasion of the erythrocytes and parasitemia, except that there may be repeated exoerythrocytic cycles in addition and concurrently, which helps to explain relapses as well as certain medicinal failures. The young trophozoite having parasitized an erythrocyte the "signet ring" stage is reached, growing rapidly until the erythrocyte is more or less filled, depending upon the species of parasite, and it is then known as a *merocyte.* The full-grown merocyte (also known as a *schizont*) now divides into a larger or smaller number of bodies (also depending upon the species) which are then liberated, and when free in the plasma are known as *merozoites.* The time required for this sporulation is from 24 to 72 hours according to the species. Merozoites, like sporozoites, presumably also have an exoerythrocytic cycle, after which the resulting trophozoites enter other red cells and again the cycle repeats itself until the infection is great enough to produce a paroxysm, i.e., in from 6 to 12 days, commonly about 10 days. The paroxysms may be due to the release of toxins, although there is no clear evidence concerning this.

The great majority of the merozoites are asexual, but some of them are potential males and females, which require a longer time, probably not less than 10 days, to develop to their full growth, and are then known as *gametocytes.* In *Plasmodium vivax* the sexual forms are not easily recognized; however, the following characters are useful: "(1) their larger size, (2) more abundant pigment, (3) there is usually only one fairly large chromatin mass, whereas in an asexual form (*schizont*) of nearly equal size the chromatin has already begun to divide into several portions (segmenting stage)" (Stephens and Christophers). In *P. falciparum* the sexual individuals are in the form of *crescents.* The female crescent (*macrogametocyte*) has the pigment collected at the center, while the male crescent (*microgametocyte*) has the pigment scattered throughout and is known as a hyaline crescent.

With development of the gametocytes completed all is ready for the next cycle (the sexual) which can only be accomplished within the body of anopheline mosquitoes. In the meantime the asexual cycles are repeated until senescence or some other biological process ends the life of the parasites or until quinine or other plasmodiacide destroys them. The gametocytes are not easily destroyed, persisting in the body for long periods, during which time the infected person is a carrier. A person eventually removed from reinfection becomes rid of malaria probably because of parasite senescence. Rejuvenation of the parasite brood and consequent relapses even after many years of latency may perhaps result from the exoerythrocytic cycle referred to earlier.

Sexual cycle, the cycle of Ross (Fig. 78), has been observed only in female anopheline mosquitoes; in the stomach of which flagellation of the male gametocyte takes place. After a peripheral arrangement of the chromatin (in clumps corresponding to the number of flagella) there are extruded from three to six long slender filaments (flagella), each of which separates from the parent body (exflagellation), forming the male

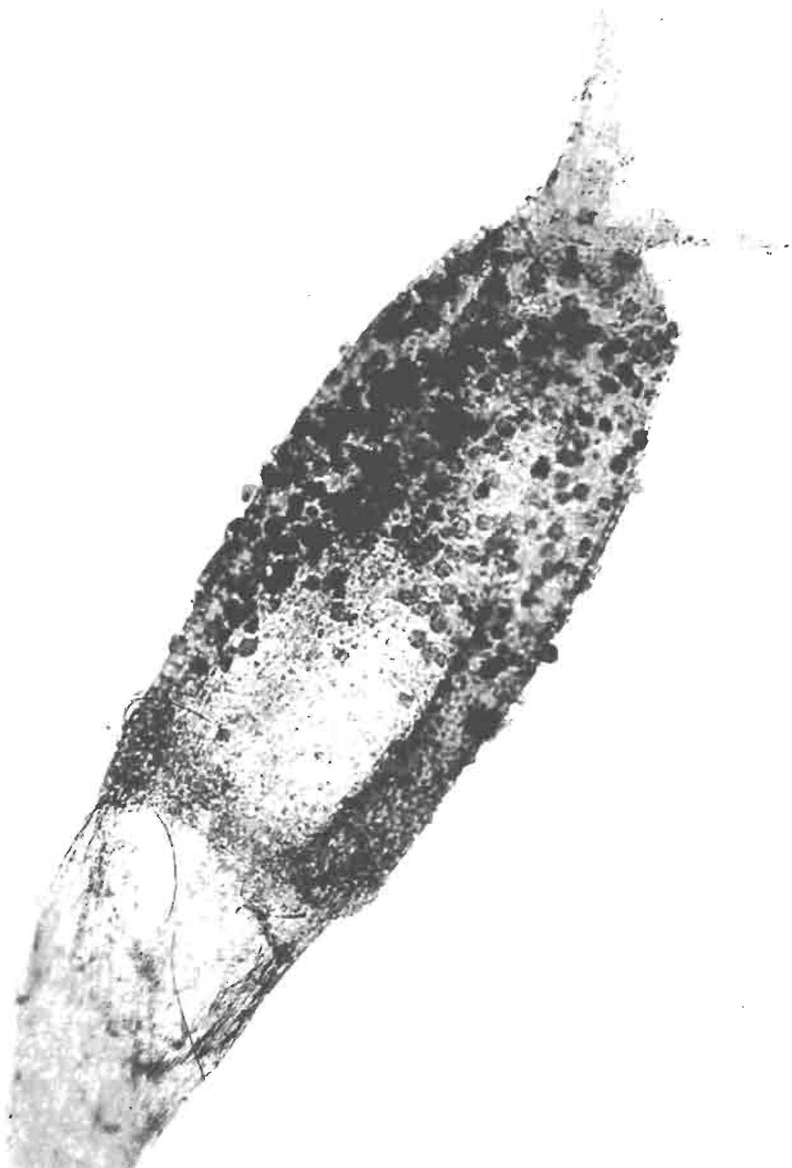


FIG. 79. Stomach of female anopheles mosquito with numerous plasmodial cysts. (Photograph by Mayne.)

gamete (*microgamete*) corresponding in function to the spermatozoön of higher animals. The female gametocyte, now known as the *macrogamete,* having been taken into the stomach of the mosquito with the microgametocytes in the act of sucking blood, also undergoes certain changes (maturation), becoming rounded or oval in form with the chromatin mass centrally located. In this condition and still in the stomach of the mosquito, the microgamete conjugates with the macrogamete, producing the *zygote,* which soon becomes motile and is then known as the *oökinete* or vermiculus, in which stage the epithelium of the stomach is penetrated and a position is shortly taken up just beneath the peritoneal membrane. Based on his studies of the plasmodia of birds Huff[23] points out that this penetration of the stomach wall

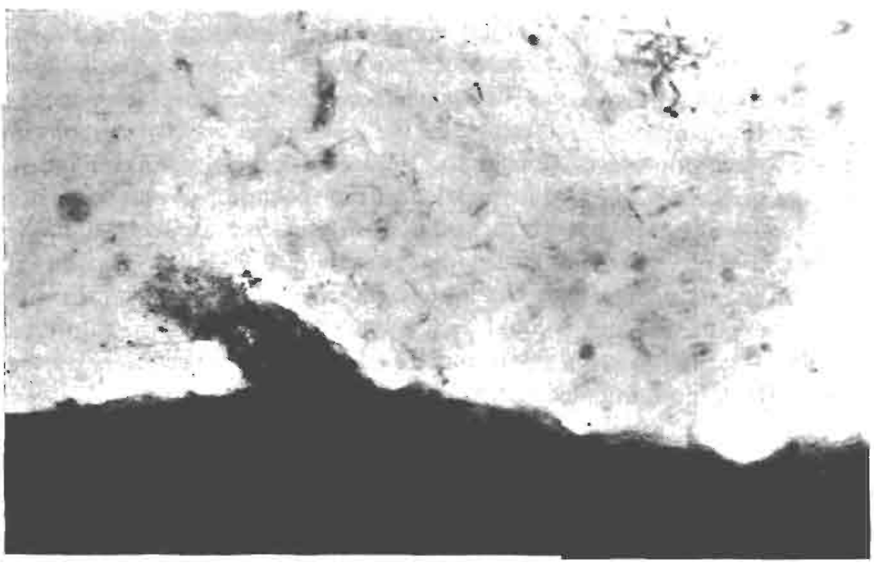


FIG. 80. A bursting plasmodial cyst on stomach of mosquito. Spindle-shaped sporozoites being liberated. (Greatly magnified.) (Photograph by Mayne.)

is not a boring process, for this zygote has lost its pointed ends long before the penetration begins. When the oökinetes are first found in the vicinity of the stomach wall, they are lying parallel to it in the serous mucoid layer adjacent to the cells of the stomach wall. As the parasite grows, it becomes relatively thicker and gradually force two of the stomach cells apart. It gradually becomes more spherical and forces the stomach cells apart nearer and nearer the outside of the stomach wall. The stomach cells now begin to come back to their original positions on the inner side. Finally the parasite, now an oöcyst, comes to lie under the outer envelope of the stomach.

In this position the parasite grows enormously, forming an *oöcyst* (Fig. 79) in which many nuclei appear in from four to five days. These tiny nucleated bodies give rise to hundreds of spindle-shaped organisms (*sporozoites*) which are shed into the body cavity of the mosquito in from 24 to 48 hours (Fig. 80). The majority of the sporozoites eventually collect in the salivary glands, remaining there until the mosquito bites again, when many of them may be injected with the saliva into the wound. The time required for the completion of the sexual cycle varies from 7 to 10 days under favorable conditions. Once infected the mosquito probably remains infected and infective for the rest of its life. In making malaria surveys one should be proficient in detecting plasmodial infections in anophelines.[24]

What makes a mosquito a good natural vector? Conclusive experimental evidence indicates that the *Plasmodia* of human malaria do not develop within the bodies of culicine mosquitoes even though gametocytes are ingested. Whether all anopheline species are infectable is, of course, not known; many malariologists are of the opinion that laboratory tests tend to prove that it is a matter of degree only, that no anopheline is completely refractory to the plasmodia of human malaria. There is not only a variation in the degree of mosquito species hospitality to plasmodia but also a variation in the species of plasmodia. Thus *A. maculipennis* Meig. and *A. quadrimaculatus* Say are known to be carriers of three species of plasmodia (Beyer, *et al.*,[25] Thayer,[26] King[27]) while A. *punctipennis* (Say) is a strong carrier of the tertian parasite and a very weak one (probably negligible) for the estivo-autumnal parasite (Darling[28]). Moore, Young, Hardman, and Stubbs[29] tested the infectivity of foreign malarias for four species of California anophelines, *A. freeborni* Aitken, *A. punctipennis* (*Say*), *A. occidentalis* D. and K., and *A. pseudopunctipennis franciscanus* McC. They all appeared to have about the same susceptibility; all developed sporozites. Successful transmission was effected with the first three species. The length of the sporogonous cycle in the four species at 75° F was about 10 days; the shortest cycle was 8 days in *A. freeborni.*

A good natural vector is an *Anopheles* species which is freely infectable by the several species of human plasmodia, offers a favorable environment for development to the sporozoite stage, breeds, successfully and abundantly, is a house invader, and takes human blood repeatedly, i.e., is anthropophilous. The maintenance of close and constant contact between an anthropophilous mosquito and its source of food supply is an important factor in endemicity. The concept of species sanitation is based on a knowledge of the factors referred to above. An anopheline may be common, even abundant, yet of little importance as a vector for one or more reasons.

Hindle[30] points out that, "the first instance of an *Anopheles* being shown not to transmit malaria was in the case of the common Indian species *Anopheles subpictus* (*rossi*) Grassi. This species is found quite commonly in very large numbers associated with every degree of prevalence of malaria, but it has not been shown to act as a transmitting agent (in India) under natural conditions, though it can be infected experimentally."

"**Anophelism without malaria.**" Hackett (*loc. cit.* p. 3) points out that because of the presence of anophelines in great numbers in areas without malaria, various theories have been advanced to explain this. "Anophelism without malaria" is an expression now commonly used in the study of malariology. Hackett refers to such theories as (1) robust mosquitoes produced under unusually favorable conditions are unsusceptible to plasmodial infection (Alessandrini); (2) brackish water breeds robust anophelines which easily become infected, live long, and are therefore most dangerous (Grassi); (3) coumarin, the active principle in a type of clover, *Melilotus altissima,* present in clover honey, either kills sporozoites in the mosquito glands or protects the mosquito against infection (D'Herelle); (4) adaptation of the maxillary tooth formula to feeding habits of the mosquito,[31] i.e., increase in the number of teeth might be an adaptation to tough skins of larger domestic animals (zoöphilism); when the average number of teeth (maxillary index) was between 14 and 15 this race fed constantly on domestic animals and did not feed on man, those feeding on human blood having an index of 14 or less (Roubaud); (5) gonotrophic dissociation, which refers to undeveloped ovaries in the female anopheline, following a blood meal, e.g., a strain of anophelines which passes the winter in warm stables and houses where there is plenty of available food, yet is not stimulated to oviposition (Swellengrebel). Normally the ovaries of anopheline females develop following a blood meal; also the females usually undergo true hibernation without a blood meal. Thus *A. maculipennis* variety *atroparvus* van Thiel resolves itself into two strains, one of which shows gonotrophic dissociation and is the explanation of so-called "malaria houses," i.e., when this strain "takes up winter quarters in a house, it lives upon the family; and if the mosquito should be infected or someone in the house should be a carrier, by spring most of the family will have contracted malaria and will have passed it on to the rest of the anophelines sheltering in the house." (6) Geographic subspecies distinguished by egg types, seven being recognized, indicating wide divergence in feeding and breeding habits; barred eggs of cattle feeders, and eggs of more uniform pattern of malaria vectors and brackish water breeders (Falleroni; and Hackett, Martini, and Missiroli, *loc. cit.* p. 3).

Number of persons infected by one mosquito. Whether an *Anopheles* once infected can infect more than one person without again feeding on infective blood is a matter of interest. Country-school privies as the writer has often observed, may be infested with *Anopheles* mosquitoes which have the opportunity to bite different persons in quick succession. In an instructive series of experiments conducted by Mayne (Mitzmain[32]) he reports that one mosquito proved to be the sole infecting agent in three cases. Mitzmain used *Anopheles punctipennis* (Say) with *Plasmodium vivax.* He also demonstrated in 11 experiments that short exposure to bites (interrupted feeding) was sufficient to cause successful transmission of the disease.

Effect of temperature on plasmodia in the mosquito. In spite of the fact that all conditions appear to be favorable i.e., presence of numerous anopheline mosquitoes together with ample human population with carriers of plasmodial gametocytes, malaria may be wholly absent in particular localities. An analysis of conditions will usually reveal the fact that the average temperature is low because the nights are cool although the days may be fairly warm, or because of prevailing cool fogs. It is generally agreed that malaria gametocytes cannot develop successfully within the body of the mosquito host when the average temperature is below about 60° F. It is nevertheless a matter of interest to know that King[33] observed the survival of the parasite of tertian malaria in the mosquito host (*Anopheles quadrimaculatus* Say) at a temperature of 30° F for a period of two days; at 31° F for four days; and at 46° F for 17 days; and the parasite of estivo-autumnal malaria survived a temperature of 35° F for 24 hours.

Knowles and Basu[34] (1943), working with the vector *Anopheles stephensi* Liston, found that the heaviest salivary gland infection of *Plasmodium vivax* was obtained at 80° F and 50 per cent relative humidity. Using the same species of *Anopheles* they found that at a temperature of 100° F and with all percentages of humidity between 50 and 100 no infection with any species of malaria parasite was obtained. The sporozoite stage of *Plasmodium vivax* was reached in the salivary glands of *A. stephensi* in 18 days at 60° F, 15 days at 70° F, 11 days at 80° F, and 9 days at 90° F. For *Plasmodium falciparum* also at 50 per cent relative humidity the time was 14 days at 70° F, 10 days at 80° F, and 9 days at 90° F. *Plasmodium malariae* has been reported to require 30 to 35 days at 67° F, with relative humidity not given.

Hibernating anophelines not carriers. Hibernation of the anopheline host presents the problem of the overwintering of the parasite. Mayne (Mitzmain[35]) again comes forward with an excellent discussion of the question, "Is mosquito or man the winter carrier of malaria organisms?" He reached the following conclusions:

> . . . hibernating *Anopheles,* collected in the region investigated (northwestern Mississippi), did not harbor parasites of malaria. This was determined after

an examination of 2,122 dissected anophelines, of which 1,211 specimens were examined before May 15, 1915. Among the remaining 911 specimens, serving as a malaria indicator for the spring season, 3 mosquitoes, between May 15 and May 26, were definitely shown to contain oöcysts, indistinguishable from those seen in mosquitoes experimentally infected with human malaria.

In the investigation of man as the responsible winter carrier, 1,184 persons, residing on the plantations selected, were examined for malaria parasites. Four hundred and ninety-two infections were identified microscopically; 317 cases were of subtertian type, 8 were mixed infections, and the remainder were of the simple tertian type with the exception of one quartan case.

In the consideration of these infections an important fact stands out: *nearly one-fourth (24.8 per cent) of the human carriers harbored gametocytes.*

It was proved that from a group of 103 persons, examined in March, 1915, 8 of the 15 gametocyte carriers identified were similarly infected during the preceding fall.

The incrimination of man as the sole winter carrier is emphasized by the fact that 3 malaria-infected *Anopheles quadrimaculatus* were found in the homes of these gametocyte carriers during May 15 to May 26, previous to which time 1,180 specimens of *Anopheles* from this source were found to be negative.

Anophelines overwintering in warm stables and homes, as explained in the previous chapter under races of *Anopheles maculipennis* Meig., may nevertheless play an important though highly circumscribed role in the transmission of malaria.

Vectors of human malaria. It has been abundantly proved that the causal organisms (plasmodia) of human malaria are transmitted from man to man only through the agency of mosquitoes belonging to the genus *Anopheles*. Russell, Rozeboom, and Stone[36] (1943) give identification keys to 240 species and 78 subspecies of anophelines of the world. Of this number Russell in *Practical Malariology* (1946, *loc. cit.*) named 54 species as the chief malaria vectors. These are distributed over the world, according to various authorities, as shown in the following tabulation:

TABLE I

IMPORTANT ANOPHELINE VECTORS OF MALARIA*

COUNTRY AND SPECIES	LOCALITY	BREEDING PLACES
UNITED STATES AND CANADA		
Anopheles quadrimaculatus Say	(Eastern, central, and southern United States; Gulf to Ontario)	Sunlit, impounded water, marshes, swamps, rice fields
Anopheles maculipennis freeborni Aitken	(Rocky Mountains, New Mexico, Pacific coast)	Sunlit seepage water, irrigation ditches, rice fields (in part)

* See also Herms and Gray.[37]

COUNTRY AND SPECIES	LOCALITY	BREEDING PLACES
MEXICO, CENTRAL AMERICA, WEST INDIES		
Anopheles albimanus Wied.	(Mexico, Central America, West Indies, Colombia to Venezuela)	Sunlit lagoons (brackish and fresh), swamps, pools
Anopheles p. pseudopunctipennis Theo.	(Southern United States to Argentina)	Sunlit streams with green algae, seepage, pools, etc.
Anopheles darlingi Root	(Central America, Venezuela to Argentina)	Sunlit fresh water, marshes, etc.
Anopheles aquasalis Curry	(Central America, West Indies, to Brazil)	Brackish marshes, irrigation seepages, etc.
Anopheles punctimacula D. and K.	(Mexico, Central America, West Indies, to Brazil)	Shaded pools, swamps, streams
Anopheles bellator D. and K.	(West Indies to Brazil)	Water at base of leaves of bromeliads
Anopheles cruzii D. and K.	(Brazil)	Water at base of leaves of bromeliads
Anopheles m. aztecus Hoffmann	(Mexico)	Sunlit ponds with green algae
SOUTH AMERICA		
Anopheles albimanus Wied.	(Colombia, Ecuador, Venezuela)	See above
Anopheles albitarsus L.-A.	(Argentina)	Rice fields, marshes, overflows, etc.
Anopheles p. pseudopunctipennis Theo.	(To Chile and Argentina)	See above
Anopheles darlingi Root	(Venezuela to Argentina)	See above
Anopheles gambiae Giles	(Imported into Brazil, now exterminated[38])	All sorts of collections of fresh water
EUROPE		
Anopheles l. labranchiae Falleroni	(Italy, Sardinia, Sicily, Corsica, Spain, N. W. coast of Africa)	Upland streams, rice fields, brackish coastal marshes
Anopheles labranchiae atroparvus van Thiel	(England, The Netherlands, Germany, Sweden, Spain, northeastern Italy)	Sunlit fresh or brackish marshes, swamps, etc.
Anopheles messeae Falleroni	(Norway, Russia, Siberia, Manchuria)	Fresh-water pools, ponds, marshes
Anopheles superpictus Grassi	(Spain, southern Europe, Greece, Asia Minor)	Pools in stream beds, irrigation canals, seepages, etc.
Anopheles sacharovi (= *elutus*) Favr.	(Balkans, Russia)	Sunlit coastal marshes, fresh or brackish water
NORTH AFRICA AND MIDDLE EAST		
Anopheles claviger Meig.	(Ukraine, Asia Minor, North Africa)	Rock pools, wells, cisterns, marshes
Anopheles l. labranchiae Fall.	(Northwest coast of Africa)	See above
Anopheles pharoensis Theo.	(North Africa, Palestine)	Rice fields, swamps
Anopheles sacharovi Favr.	(Balkans, central Russia)	See above

COUNTRY AND SPECIES	LOCALITY	BREEDING PLACES
Anopheles sergenti Theo.	(North Africa, Palestine, Turkey, Syria)	Rice fields, irrigation canals, borrow pits, seepage water
Anopheles superpictus Grassi	(Asia Minor)	Stream bed pools, irrigation canals, seepage water, hill district
CENTRAL AND SOUTH AFRICA		
Anopheles funestus Giles	(Tropical Africa)	Stream margins, swamps, ditches, seepage water
Anopheles gambiae Giles	(Tropical Africa, Egypt, Arabia)	Puddles, pools, sluggish streams, all sorts of collections of water
Anopheles moucheti Evans	(Uganda, Congo, Cameroons)	Stream margins, swamps
Anopheles pharaoensis Theo.	(Widely distributed in Africa, Palestine)	See above
Anopheles nili Theo.	(Widely distributed in central Africa)	Shade loving, stream margins
Anopheles pretoriensis Theo.	(Widely distributed in central and south Africa)	Sunlit rock pools, stream beds, hoof prints, ditches
PHILIPPINE ISLANDS		
Anopheles minimus flavirostris Lud.	(Many islands, Java, Celebes)	Foothill streams, ditches, wells
Anopheles mangyanus Banks	(Many islands)	Stream beds, irrigation ditches
JAPAN, NORTH CHINA, KOREA		
Anopheles hyrcanus sinensis Wied.	(Widely distributed)	Open clean water, rice fields, swamps, ponds, slow streams
Anopheles pattoni Christ.	(Northen China)	Beds of hill streams, rock pools
SOUTH AND CENTRAL CHINA, BURMA, TAIWAN		
Anopheles minimus Theo.	(Hilly regions southern China, Taiwan, Burma)	Sunlit, slow moving streams, rice fields, irrigation ditches
Anopheles hyrcanus sinensis Wied.	(Plain of central China, Burma)	See above
Anopheles jeyporicus	(Hong Kong area)	Rice fields in hilly country
Anopheles jeyporicus		
Anopheles culicifacies Giles	(Burma)	Stream breeder, irrigation ditches
Anopheles maculatus maculatus Theo.	(Burma, Southern China)	Stream and river bed breeder, rice fields, pools, lake margins
Anopheles philippinensis Lud.	(Burma, Indo-China)	Rice fields, borrow pits, swamps, ditches, tanks
INDIA AND CEYLON		
Anopheles culicifacies Giles	(India, Ceylon)	See above
Anopheles stephensi Liston	(India)	Well, cisterns, roof gutters, temporary water receptacles

COUNTRY AND SPECIES	LOCALITY	BREEDING PLACES
Anopheles maculatus maculatus Theo.	(India, Ceylon)	See above
Anopheles fluviatilis James	(India)	Foothill stream edges, springs, irrigation canals
Anopheles minimus Theo.	(Eastern and northern Ceylon)	See above
Anopheles philippinensis Lud.	(India)	See above
MALAYA (East Indies, Siam, Malaya		
Anopheles aconitus Dönitz	(East Indies, Malaya, Indo-China)	Rice fields, irrigation ditches, pools in creek beds, reservoirs
Anopheles culicifacies Giles	(Siam)	See above
Anopheles fluviatilis James	(Thailand)	See above
Anopheles hyrcanus nigerrimus Giles	(Malaya, East Indies)	Rice fields, impounded water, borrow pits, sluggish streams
Anopheles maculatus maculatus Theo.	(Thailand, Malaya, East Indies)	Stream and river bed breeder, seepage water, lake margins
Anopheles subpictus subpictus Grassi	(Malaya, East Indies)	Borrow pits, buffalo wallows, all sorts of temporary or permanent collections of water
Anopheles sundaicus (Rodenwaldt)	(Thailand, Malaya, East Indies)	Sea-water lagoons and swamps
Anopheles umbrosus (Theo.)	(Malaya, Cochin-China, East Indies)	Shaded jungle pools, mangrove swamps
Anopheles philippinensis Lud.	(Malaya, East Indies)	See above
AUSTRALIA-MELANESIA-POLYNESIA		
Anopheles farauti (= *A. moluccensis*) Lav.	(New Guinea, Solomons, New Hebrides)	Fresh or brackish water, all sorts of natural or artificial collections of water, clear or polluted
Anopheles punctulatus punctulatus Dön.	(New Guinea, Solomons)	Rain pools, stream margins, hoof prints, etc.
Anopheles bancrofti Giles	(New Guinea, northern Australia)	Shallow, slow moving streams

Malaria surveys. Although an inquiry into the facts about malaria in some particular area may be described as a survey, Christophers, Sinton, and Covell[39] point out that " . . . it is far from being merely routine or mechanical." These authors state that the circumstances affecting malaria are so varied "with its triple-linked chain of man, the parasite, and the mosquito, as well as all the various factors influencing this chain, that in the present state of our knowledge a malaria survey is almost always a true piece of research work. Such a survey is intended to guide policy and action. . . . " Elmendorf[40] defines a malaria survey as "a quantitative as well as a qualitative procedure directed toward securing information which will determine the status of malaria in a community. The survey should serve as a blueprint for guidance . . . and should provide an original base-line from which all subsequent measurements of the course of the disease are determined."

In general the survey staff, on a minimum basis, should consist of the following personnel: a *malariologist,* an *entomologist* trained as a culicidologist, a *sanitary engineer* with special training in hydraulics. The staff should include a laboratory technician trained in parasitology, a mapper, and a clerk. Headquarters and laboratory should be conveniently located and properly equipped. Having made the acquaintance, and having been assured the cooperation, of civil authorities, particularly the local health officer, the following data will be assembled, recorded, mapped, and card-indexed as required:

1. Demographical data: composition of the population by race, age, and sex; occupations, economic status, housing, etc.
2. Meteorological data: rainfall, temperature, humidity, winds, etc.
3. Topographical data: soil, vegetation, etc.
4. Agricultural data: crops, farming methods, irrigation, drainage, rice culture, marshes, etc.
5. Malaria statistics: morbidity and mortality; incidence (age, seasonal, etc.); distribution, occupation, and economic status of malarial population; spleen census, etc.
6. Parasitological and entomological data: parasite index based on blood smears; oöcyte and sporozoite index; numerical prevalence of all species of adult anophelines with habitat preference (houses, pig pens, cattle sheds, etc.); feeding habits of adults ascertained by use of precipitin test; larval incidence (use of dippers), showing kinds and distribution of breeding places; with a little practice collections of eggs can be made very readily. As previously stated, eggs are important in the identification of anopheline mosquitoes. (Aitken,[41] 1948).

Animal Malarias

Avian malaria. Blood protozoa were first reported from birds in 1885 by Danilewsky from the Russian Ukraine. Herman[42] lists 178 species and subspecies of North American birds in whose blood protozoön parasites have been found; 63 of these protozoon infections are listed as plasmodial. The species of avian plasmodia, of which there are many, can be divided roughly into two groups based on whether they produce an elongate or a round type of gametocyte. Among those belonging to the elongate type is *Plasmodium elongatum Huff,* and among those belonging to the round type are the following well-known species, *Plasmodium gallinaceum* Brumpt, *P. cathemerium* Hartman, and *P. relictum* (Grassi et Feletti). So far as is known avian plasmodia are transmitted in nature by culicine mosquitoes, among them *Culex pipiens* Linn., *C. quinquefasciatus* Say, and *C. tarsalis* Coq. (Reeves, *loc. cit.* p. 182). Numerous species of mosquitoes are susceptible to infection in the laboratory, including *Anopheles quadrimaculatus* Say. For laboratory purposes *Aedes aegypti* is an excellent vector of *Plasmodium gallinaceum* Brumpt, as is *Aedes atropalpus* (Coq.) recently (1946) reported by Trembly.

Other malaria-like infections of birds are caused by *Haemoproteus*, e.g., *Haemoproteus columbae* Celli and San Felice of pigeons and doves, transmitted by a louse fly, *Pseudolynchia canariensis* (Macq.); also quail malaria caused by *Haemoproteus lophortyx* O'Roke carried by *Lynchia hirusta* Ferris, the louse fly of quail. (See Chapter XVIII.) A malaria-like disease of ducks is caused by *Leucocytozoön anatis* Wickware and is carried by a simuliid fly, *Simulium venustum* Say. (See p. 153.)

Simian malaria. Among the several species of the genus *Plasmodium* peculiar to monkeys are *P. knowlesi* Sinton and Mulligan, *P. cynomolgi* Mayer, *P. kochi* (Laveran), *P. inui* Halberstadler and Prowazek, and *P. brasilianum* Gonder and Berenberg-Gossler. These plasmodia differ but slightly from the human species, and when *P. knowlesi* is introduced (as for paresis treatment) into the blood of man, a clinical *vivax* malaria response obtains (Milan and Kusch[43]). *Anopheles quadrimaculatus* Say is reported to be highly susceptible to *P. cynomolgi* (Coggeshall, 1941). Clark and Dunn,[44] in Panama, were able to infect certain species of Panamanian anophelines with this parasite.

Saurian and amphibian malaria. Plasmodia have long been known in lizards of the family Iguannidae; among these are *Plasmodium agamae* Wenyon, *P. diploglossi* Aragao and Neiva, *P. giganteum* Theiler., and *P. floridense* Thompson and Huff. Bullfrogs and toads have recently been shown to have plasmodial infections, viz.: *P. catesbianca* Fantham, Porter, and Richardson in the former and *P. bufonis* F., P., and R. in the latter.

Bats, squirrels, buffalo, antelopes, and many other animals are hosts for plasmodial infections. There are practically unlimited opportunities in this field of research.

B. YELLOW FEVER

Yellow fever derives its name from the jaundiced color of the skin, mucous membranes, and sclera, which usually develops about the third or fourth day of the illness. The onset is very sudden. It begins with a severe headache, backache, and fever, with frequent vomiting. The vomit becomes very dark after two or three days because it contains blood, whence the name "black vomit." In fatal cases death usually occurs between the fifth and eighth day of the illness; if the patient survives until the seventh day his chances for recovery are generally said to be good. One attack confers lifelong immunity. The death rate may reach as high as 50 per cent.

Carter[45] (1931, page 146) considers the epidemic in Yucatan, Mexico, in 1648 as the first certainly recognizable (from its description) epidemic of yellow fever. He also gives (page 190) 1649 as the date for the earliest introduction of yellow fever into Havana, and on page 192 he refers to its disappearance from Cuba after 1655 and its reintroduction from Vera Cruz in 1761. From that date until 1901 it was endemic in Havana with devastating results. The last major American urban epidemic took place in Rio de Janeiro in 1928 and 1929 with 435 recorded deaths. For nearly three centuries yellow fever took an huge annual toll of life. Outbreaks have occurred as far north as Quebec, Canada, and as far south as Tocopilla, Chile. New York City had epidemics of yellow fever many times, as did Philadelphia.

Walter Reed and yellow fever. Although Dr. Carlos Finlay (*loc. cit.* p. 3) of Havana had quite early (1881) advanced a mosquito-transmission theory, and had carried on what we now know to have been incriminating experiments with nonimmunes, his theory was discredited until renewed interest was given it by the work of the United States Army Yellow Fever Commission headed by Major Walter Reed in 1900. Reed[46] and his colleagues made a preliminary report in which they state: "Since we here, for the first time, record a case in which a typical attack of yellow fever has followed the bite of an infected mosquito, within the usual period of incubation of the disease, and in which other sources of infection can be excluded, we feel confident that the publication of these observations must excite renewed interest in the mosquito theory of the propagation of yellow fever, as first proposed by Finlay."

The United States Senate Document No. 822 (Jan. 27, 1911) is concerned with yellow fever and contains a compilation of various publications by the Commission as well as others. In this document, McCaw gives the following account of the work of the Commission:

> In June, 1900, Major Reed was sent to Cuba as president of a board to study the infectious diseases of the country, but more especially yellow fever. Associated with him were Acting Asst. Surgs. James Carroll, Jesse W. Lazear, and A. Agramonte.
>
> At this time the American authorities in Cuba had for a year and a half endeavored to diminish the disease and mortality of the Cuban towns, by general sanitary work, but while the health of the population showed distinct improvement and the mortality had greatly diminished, yellow fever apparently had been entirely unaffected by these measures. In fact, owing to the large number of nonimmune foreigners, the disease was more frequent than usual

in Habana and in Quemados near the camp of American troops, and many valuable lives of American officers and soldiers had been lost.

Reed was convinced from the first that general sanitary measures alone would not check the disease but that its transmission was probably due to an insect.

In June, July, and August, 1900, the commission gave their entire attention to the bacteriological study of the blood of yellow-fever patients and the post-mortem examinations of the organs of those dying with the disease. In 24 cases where the blood was repeatedly examined, as well as in 11 carefully studied autopsies, *Bacillus icteroides* was not discovered, nor was there any indication of the presence in the blood of a specific cause of the disease.

Application was made to General Leonard Wood, the military governor of Cuba, for permission to conduct experiments on nonimmune persons, and a liberal sum of money requested for the purpose of rewarding volunteers who would submit themselves to the experiment.

It was indeed fortunate that the military governor of Cuba was a man who by his breadth of mind and special scientific training could readily appreciate the arguments of Major Reed as to the value of the proposed work.

Money and full authority to proceed were promptly granted, and to the everlasting glory of the American soldier, volunteers from the Army offered themselves for experiment in plenty and with the utmost fearlessness.

Before the arrangements were entirely completed, Dr. Carroll, a member of the commission, allowed himself to be bitten by a mosquito that 12 days previously had filled itself with the blood of a yellow-fever patient. He suffered from a very severe attack, and his was the first experimental case. Dr. Lazear also experimented on himself at the same time, but was not infected. Some days later, while in the yellow-fever ward, he was bitten by a mosquito and noted the fact carefully. He acquired the disease in its most terrible form and died a martyr to science and a true hero.

No other fatality occurred among the brave men who, in the course of the experiments, willingly exposed themselves to the infection of the dreaded disease.

A camp was especially constructed for the experiments about 4 miles from Habana, christened Camp Lazear in honor of the dead comrade. The inmates of the camp were put into most rigid quarantine and ample time was allowed to eliminate any possibility of the disease being brought in from Habana.

The personnel consisted of three nurses and nine nonimmunes, all in the military service, and included two physicians.

From time to time Spanish immigrants, newly arrived, were brought in directly from the immigrant station; a person not known to be immune was not allowed to leave camp, or if he did was forbidden to return.

The most complete record was kept of the health of every man to be experimented upon, thus eliminating the possibility of any other disease than yellow fever complicating the case.

The mosquitoes used were specially bred from the eggs and kept in a building screened by wire netting. When an insect was wanted for an experiment it was taken into a yellow-fever hospital and allowed to fill itself with the blood of a patient; afterwards at varying intervals from the time of this meal of blood it was purposely applied to nonimmunes in camp.

In December, 5 cases of the disease were developed as the result of such applications; in January, 3, and in February, 2, making in all 10, exclusive of the cases of Drs. Carroll and Lazear. Immediately upon the appearance of the first recognized symptoms of the disease, in any one of these experimental cases, the patient was taken from Camp Lazear to a yellow-fever hospital, 1 mile distant. Every person in camp was rigidly protected from accidental mosquito bites, and not in a single instance did yellow fever develop in the camp, except at the will of the experimenters.

The experiments were conducted at a season when there was the least chance of naturally acquiring the disease, and the mosquitoes used were kept active by maintaining them at a summer temperature.

A completely mosquito-proof building was divided into two compartments by a wire-screen partition; infected insects were liberated on one side only. A brave nonimmune entered and remained long enough to allow himself to be bitten several times. He was attacked by yellow fever, while two susceptible men in the other compartment did not acquire the disease, sleeping there 13 nights. This demonstrates in the simplest and most certain manner that the infectiousness of the building was due only to the presence of the insects.

Every attempt was made to infect individuals by means of bedding, clothes, and other articles that had been used and soiled by patients suffering with virulent yellow fever.

Volunteers slept in the room with and handled the most filthy articles for 20 nights, but not a symptom of yellow fever was noted among them, nor was their health in the slightest degree affected. Nevertheless they were not immune to the disease, for some of them were afterwards purposely infected by mosquito bites. This experiment indicates at once the uselessness of destroying valuable property for fear of infection. Had the people of the United States known this one fact 100 years ago, an enormous amount of money would have been saved to householders.

Besides the experimental cases caused by mosquito bite, four nonimmunes were infected by injecting blood drawn directly from the veins of yellow-fever patients in the first two days of the disease, thus demonstrating the presence of an infectious agent in the blood at this early period of the attack.

Even the blood serum of a patient, passed through a bacteria-proof filter, was found to be capable of causing yellow fever in another person."

The conclusions reached by the Commission follow:

1. The mosquito (*C. fasciatus* = *Aëdes aegypti*) serves as the intermediate host for the parasite of yellow fever.

2. Yellow fever is transmitted to the nonimmune individual by means of the bite of the mosquito that has previously fed on the blood of those sick with this disease.

3. An interval of about 12 days or more after contamination appears to be necessary before the mosquito is capable of conveying the infection.

4. The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack.

5. Yellow fever can also be experimentally produced by the subcutaneous injection of blood taken from the general circulation during the first and second days of this disease.

6. An attack of yellow fever, produced by the bite of the mosquito, confers immunity against the subsequent injection of the blood of an individual suffering from the nonexperimental form of this disease.

7. The period of incubation in 13 cases of experimental yellow fever has varied from 41 hours to 5 days and 17 hours.

8. Yellow fever is not conveyed by fomites, and hence disinfection of articles of clothing, bedding, or merchandise, supposedly contaminated by contact with those sick with this disease, is unnecessary.

9. A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitoes capable of conveying the parasite of this disease.

10. The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes and the protection of the sick against the bites of these insects.

11. While the mode of propagation of yellow fever has now been definitely determined, the specific cause of this disease remains to be discovered.

The following is an excerpt from a letter which Walter Reed wrote to his wife in Baltimore late in the evening of December 31, 1900. The letter was written from the Barracks in Quemados, Cuba: "Only ten minutes of the old century remain. Here I have been sitting reading that most wonderful book, 'La Roche on Yellow Fever' written in 1853. Forty-seven years later it has been permitted to me and my assistants to lift the impenetrable veil that has surrounded the causation of this most dreadful pest of humanity and to put it on a rational and scientific basis. I thank God that this has been accomplished during the latter days of the old century. May its cure be wrought in the early days of the new! The prayer that has been mine for twenty years, that I might be permitted in some way at some time to do something to alleviate human suffering has been granted! A thousand Happy New Years! . . . Hark, there go the twenty-four buglers in concert, all sounding 'Taps' for the old year." (Herrick.[47])

Etiology. The search for the causal agent of yellow fever was carried on most assiduously for many years and various discoveries were announced from time to time. Sanarelli in 1897 declared the organism to be *Bacillus icteroides;* this was amply disproved by the United States Army Yellow Fever Commission in 1900. Seidelin in 1909 described *Paraplasma flavigenum* as the causal agent, and in 1919 Noguchi came to the conclusion that a spirochete, *Leptospira icteroides* Noguchi, was the cause of yellow fever. This turned out to be the cause of Weil's disease or infectious jaundice. Yellow fever is now classed among the virus diseases.

The virus is believed to be present in the circulation only during the first three days of the illness. *Aedes aegypti* (Linn.) reared from eggs in the laboratory and fed by Stokes, Bauer, and Hudson (*loc. cit.* p. 4) on infected monkeys on the first or second day of the fever, invariably became infective. They found that the mosquitoes were infective 16 days after feeding on an infected animal and remained so until death, one mosquito producing a fatal infection in two monkeys 85 and 92 days after the original infective meal.

Mechanism of transmission. The virus of yellow fever is not only present in the circulating blood during the first three or four days of the illness, but also at least six hours or possibly somewhat longer, before the onset of the fever. During this short period the vector mosquito, *Aedes aegypti* (Linn.), may at one feeding ingest several thousand infective doses of the virus. The mosquito then becomes a dangerous vector for the rest of its life, which may be over 200 days. The extrinsic incubation period normally under field temperatures takes about 12 days (Carter, *loc. cit.*) but may be considerably shortened by increased temperature, i.e., to 4 days at 98° F or extended to 18 days at 70° F (Davis[48]). After a susceptible person has been bitten and receives an infectious dose from an infected mosquito an incubation period of three or four days follows before the symptoms of the disease appear. Now again another source of infection is ready for other *aegypti* vectors restricted as before to the short period of three or four days. An epidemic may quickly develop in the presence of hordes of this rapidly multiplying species of domestic mosquito.

Summary of recent discoveries. Soper *et al.*[49] (1943) state that "when the medical historian of the future comes to write the chapter on yellow fever, he will be obliged to devote considerable space to the developments of the decade and a half from 1926 to 1940. This short period showed how impossible of fulfillment was the dream of final eradication of yellow fever, but it saw great advances in our understanding of the etiology, epidemiology, and prophylaxis of the disease, including the discovery of animal susceptibility; the rediscovery of the virus origin; the demonstration that mosquitoes other than *Aedes aegypti* can and do transmit the virus; the development of the protection test for determining immunity; the organization of viscerotomy for the diagnosis of unsuspected fatal cases; the proof that unrecognized yellow fever has been widespread in large silent endemic areas of South America and Africa; the demonstration that the disease exists in many countries of South America as one of jungle animals, independent of the distribution of *Aedes aegypti* and of man; and finally the modification of the yellow fever virus in such a way as to make mass vaccination practical. These outstanding developments have tended to overshadow the more prosaic improvements in the organization of measures against the aegypti mosquito in Brazil which have transformed expensive temporary aegypti reduction campaigns for the eradication of yellow fever into economical permanent services for the species eradication of *Aedes aegypti* itself from the infested areas."

Aedes vittatus (Bigot) has been reported as responsible for a recent (1940) epidemic of yellow fever of modified virulence in the Anglo-Egyptian Sudan. This species is said to breed abundantly in rock pools as well as tree holes. Filling such pools with sand is recommended as a partial means of control. (Horgan, Proc. Amer. Mosq. Control Assoc., Feb., 1949.)

Jungle yellow fever. Yellow fever is generally regarded as an urban or house disease transmitted solely by *Aedes aegypti* (Linn.), a domestic mosquito which breeds largely in artificial containers in and about human habitations. The epidemiological pattern of urban yellow fever is relatively simple; namely, "man to mosquito to man." Control seemed simple enough with meticulous inspection and careful application of proper control measures. The Rockefeller Foundation reported[50] that prior to 1929 the belief was expressed that yellow fever was not only fast disappearing as a human menace but that it had been practically eliminated. "In 1925 only three cases of yellow fever were reported from the entire Western Hemisphere; in the 11 months following April, 1927, no cases were reported, and it was assumed that the battle, which had cost the lives of research workers and millions of dollars, was practically won. Then almost without warning, the South American Jungle struck back (Soper and associates,[51] 1933), and in a few years' time the epidemiological strategy of the battle had to be completely altered." It is pointed out that vast areas of the hinterland of both South America and Africa are endemic centers of yellow fever. Burke[52] (1937) studied an epidemic involving 201 cases of yellow fever of the jungle type in the absence of *Aedes aegypti* (Linn.) on the Planalto of Matto Grosso, Brazil, during the seasons of 1934 and 1935. He reports that the identity of the disease was definitely established, the only difference being in the conditions under which infection occurs. "The paucity of human population in the infected district and the scattered distribution of cases in both time and space, together with the isolated circumstances attending many cases argue against man being the only vertebrate host involved. The sera from five *Cebus* monkeys captured for this study in known infected districts all gave positive protection test results, indicating immunity naturally acquired in the jungle. All available evidence points to infection occurring either in clearings next to uncleared jungle or in the jungle itself, especially during working hours." The Rockefeller Foundation (*loc. cit.*) points out that jungle yellow fever must be considered as a possible permanent source of virus for the reinfection of cities and towns where high densities of *Aedes aegypti* (Linn.) mosquitoes are tolerated. In jungle yellow fever the epidemiological pattern appears to be "monkey to mosquito to monkey." Man becomes infected when he goes into the forest and is bitten by infected mosquitoes and he brings the virus back with him when he returns to his community. There he may encounter *"aegypti"* mosquitoes and an epidemic of yellow fever may ensue. Students concerned with jungle yellow fever will need to consult "An epidemiological study of jungle yellow fever in an endemic area in Brazil," by Taylor, Laemmert, *et al.*[53]

Aedes leucocelaenus (D. and S.) and *Haemagogus capricornii* (Lutz), both forest-inhabiting mosquitoes, were incriminated by Shannon, Whitman, and Franca[54] during the 1938 outbreak of yellow fever in the State of Rio de Janeiro, Brazil. The presence of the virus in these mosquitoes as well as in certain Sabathine species taken in the Brazilian jungle was demonstrated. Subsequent investigations indicate that *Haemagogus capricornii* was probably *H. spegazzinii* (see below).

The 1947 report of the International Health Division of the Rockefeller Foundation states that *Haemagogus spegazzinii* Brèthes is by all odds the commonest and most widely distributed species of the genus *Haemagogus* in Brazil and is generally found in the endemic areas of jungle yellow fever in Colombia. As to reservoir jungle animals the above-mentioned report states that of the various kinds of animals tested only the howler monkey (*Cebus*) circulated virus in abundance.

Several mosquitoes have been incriminated as vectors of yellow fever in the tropical jungles of East Africa, among these *Aedes simpsoni* Theo. and *Aedes africanus* Theo., both widely distributed in endemic jungle areas. The latter species is apparently highly important because of its recently discovered presence both in the forest and in the plantations. There is thus presented the possibility of introducing yellow fever to human populations directly from endemic jungle foci through the agency of one species of mosquito.[55]

Other mosquito vectors. Prior to 1928 (see Chapter I) no experimental animal (other than man) was known to be susceptible to yellow fever. With the discovery that the Indian monkey, *Pithecus rhesus,* was susceptible as well as a score of other species of monkeys, and particularly with the work of Sawyer on white mice, experimentation with various species of mosquitoes grew apace. (See Soper *et al.,* 1933, *loc. cit.*)

There are evidently many species of jungle-inhabiting vertebrates which harbor the virus of yellow fever, more importantly perhaps several species of monkeys. Hedgehogs are highly susceptible animals and are of great value in laboratory investigations. Several species of mosquitoes other than those here referred to lend themselves well to laboratory transmission by the bite; among them is a common and widespread nearctic species *Aedes triseriatus* (Say)[56] in which the monkey, *Macacus rhesus,* was used as the host. It is of interest to note that the more important species of mosquitoes incriminated as probable or proved vectors of the virus of jungle yellow fever are tree-hole breeders thus sharing (perhaps roughly) the same ecological niche with their simian hosts.

Besides those species mentioned, many others (probably more than 30) have given positive results by the bite in laboratory tests; among these are *Aedes scapularis* (Rondani), *A. fluviatilis* (Lutz), *A. luteocephalus* Newst., *A. stokesi* Evans, *A. albopictus* (Skuse), *Culex thalassius* Theob., *Eretmapodites chrysogaster* Graham, *Mansonia africana* (Theob.), *Haemagogus equinus* Theobald, and *H. splendens* Williston.

C. DENGUE FEVER

Dengue fever, also known as breakbone fever or dandy fever, is a widespread disease of tropical and subtropical regions, but it also occurs in temperate climates where suitable vectors are present. In an epidemic which occurred in the State of Texas in 1922, originating in Galveston during the second week of June, spreading later to other parts of the state, beginning to abate late in September, and ending in late autumn, the number of cases of the disease was estimated at between 500,000 and 600,000.[57]

The disease is characterized by its sudden attack, severe rheumatic pains in the joints and limbs, intense headache, backache, and high fever; a remission of two to three days follows the first attack of three days; the second attack lasts usually but a day and is accompanied by a rapidly spreading rash. The "saddle back" type of fever, though quite common, is not constant. The entire course may be run in five to six days. Although the disease is of much importance because of its debilitating effects and prolonged convalescence, the death rate is very low. It is caused by a filtrable virus, as shown by Ashburn and Craig, 1907.[58] The virus is present in the peripheral circulation during the initial stages of the disease, i.e., the first three to five days, also probably for some hours previous to the onset; hence the mosquito vector must bite the patient during this short period of time in order to become infected.

Mosquito transmission. Transmission experiments conducted by

Chandler and Rice (*loc. cit.*) with *Aedes aegypti* (Linn.) were successful in four out of six cases, the mosquitoes having fed on patients in the second to fifth days of the disease.

Simmons, St. John, and Reynolds[55] (1931) found that all lots of *Aedes aegypti* (Linn.) that fed on blood from experimental cases of dengue during the first 48 hours of the disease became infected. The mosquito is able to transmit the infection after an incubation period of 11 days, though Chandler and Rice (*loc. cit.*) state that mosquitoes succeeded in transmitting the disease in from 24 to 96 hours. Infected mosquitoes remain infected as long as they live. The disease has been transmitted 174 days after infection, and *Aedes aegypti* (Linn.) has been kept alive for seven months. (Simmons *et al., loc. cit.*)

Graham[60] (1902) was the first to demonstrate that mosquitoes transmit dengue by the bite.

The incubation period in experimentally infected cases varies from three to eight days. Simmons *et al.* give the average incubation period at 5.66 days; average duration of the fever, 4.8 days. The virus is not transmitted from infected female *Aedes aegypti* (Linn.) through the egg to the offspring, neither does contamination of the skin by dengue virus from crushed *Aedes aegypti* (Linn.) result in infection. A brief immunity is conferred by an attack of dengue. (A description of *A. aegypti,* and of its breeding habits will be found in the preceding chapter.)

The investigations of Simmons, St. John, and Reynolds prove that *Aedes albopictus* (Skuse) is an important vector of dengue, also that *Culex quinquefasciatus* (= *fatigans*) Wied., hitherto so regarded is not a vector.

Aedes albopictus (Skuse) has the mesonotum ornamented with a central silvery stripe; white scales of the thoracic pleura are arranged in irregular patches. Its breeding habits under urban conditions are very similar to those of *A. aegypti,* i.e., the larvae live in fairly clear water in artificial containers; however, when away from human habitations the larvae occur in tree holes, bamboo stalks, plant axils, and stone cavities in the forests (up to 2,000 feet in Hawaii). It is particularly prevalent in depressions in monuments in Japanese cemeteries, as well as in flower containers. The species is found in Oriental regions, Madagascar, New Guinea, north Australia, and the Philippine and Hawaiian Islands.

Aedes scutellaris (Walker) resembles *A. albopictus* (Skuse) closely; the mesonotum has a central silvery stripe, white scales of thoracic pleura arranged in two wavy parallel lines. The distribution of the species overlaps that of *A. albopictus* in Micronesia and in Polynesia except Hawaii. It is reported to be a vector of dengue in New Guinea.

D. ARTHROPOD-BORNE VIRUS ENCEPHALITIDES

Equine encephalomyelitis is a disease of wide distribution; the causal agent is a virus with neurotropic properties, as shown by Meyer, Haring, and Howitt,[61] who describe symptoms as follows.

> Preceding the onset of symptoms which attract attention, the temperature may be found to vary from 103° F to 107° F. Not infrequently when the horse shows signs of drooping of the head, sleepiness, and circling motion or other psychic and motoric disturbances, the body temperature may be normal. The pulse and respiration are usually accelerated. Quite often the animal rests against the wall or corner and may show backward and sideways motions. Muscular twitchings are quite common. Many of the horses are down on the second or third day and may or may not get up when pressed to do so. Paresis of the lips and drooling are frequently noted. Mastication and swallowing may or may not be impaired, but grinding of the teeth is quite regularly observed. The conjunctiva is always infected and frequently icteric or grayish and studded with petechiae or ecchymoses. In the mild cases which were able to rise, recovery was as a rule uneventful but about half were so severe that they terminated fatally in 3 to 8 days or were destroyed for humane reasons.

Believing that bloodsucking insects might be instrumental in the transmission of the virus, the author conducted tests during the late summer of 1932 (Herms, Wheeler, and Herms 1934).[62] In these tests horseflies, *Tabanus punctifer* O. S., and horn flies, *Haematobia serrata* Desv., were used—all with negative results.

Early in 1933 Kelser[63] proved that the disease can be transmitted by *Aedes aegypti* (Linn.) not only from infected guinea pigs to normal guinea pigs but also to a horse which contracted the disease and died within five days after the onset of symptoms. Blood drawn from the horse at the height of the fever and injected into a guinea pig produced the disease, and mosquitoes fed on the horse during the period of high temperature and subsequently fed on a normal guinea pig likewise produced the disease. The largest percentage of deaths among the guinea pigs bitten by infected mosquitoes occurred on the sixth day following the infective mosquito bite. The mosquitoes were found to be capable of producing the disease as early as six days and remained infectious for at least 36 days. Kelser pointed out that it is possible that the mosquitoes, when once infected, may remain infectious the rest of their lives as is the case in yellow fever and dengue.

Following the work of Kelser the author conducted further tests, using *Anopheles maculipennis* Meigen (*A. freeborni* Aitken) and *Aedes dorsalis* (Meigen), a salt-marsh mosquito, bred from larvae taken from a salt marsh. Again all results were negative (Herms, Wheeler, and Herms, *loc. cit.*). However, later tests (unpublished) made in October, 1934, give evidence of six successful transmissions out of 29 (guinea pig to guinea pig) secured with *Aedes dorsalis* (Meigen) bred from fresh-water larvae. The elapsed time between initial feeding on an inoculated pig and feeding on a normal pig was six days in four of the positive cases and 12 days in the remaining two. From 4 to 50 mosquitoes were used in each test. Investigations made by Madsen, Knowlton, and Rowe[64] with *A. dorsalis* (Meigen) also indicate that this species may be a vector of the virus; although most tests failed, 2.5 per cent of the total trials (with guinea pigs) were positive, with incubation period varying between 9 and 19 days. Tests made by these authors with *Aedes nigromaculis* (Ludlow) gave 55.8 per cent positive transmissions, with incubation period varying between 4 and 10 days. Merrill, Lacaillade, and Ten Broeck[65] have demonstrated in repeated tests that *Aedes sollicitans* (Walker), a common Atlantic coast salt-marsh mosquito, will transmit both eastern and western strains of the virus from infected to normal guinea pigs. Transmission was obtained with the eastern virus 11 days after the initial feeding and at later periods. *Aedes cantator* (Coq.), another salt-marsh breeder, was shown to transmit the eastern virus, though less readily. These authors have demonstrated a 1,000- to 10,000-fold increase of the virus within the bodies of both *Aedes aegypti* (Linn.) (western strain virus) and *A. sollicitans* (Walker) (eastern strain virus). They report 63 days as being the longest period during which *Aedes aegypti* (Linn.) is capable of transmitting the western strain. Madsen and Knowlton[66] were successful in transmitting the western virus to guinea pigs by the bite of *Aedes nigromaculis* (Ludlow). Simmons, Reynolds, and Cornell[67] demonstrated that *Aedes albopictus* (Skuse) could transmit the western virus. Kelser[68] reported studies definitely proving the ability of *Aedes taeniorhynchus* (Wied.) to transmit the "western" type of equine encephalomyelitis from guinea pig to guinea pig.

In 1940 Kitselman and Grundmann[69] reported the isolation of equine encephalomyelitis virus from naturally infected conenose bugs, *Triatoma sanguisuga* Le Conte. Syverton and Berry[70] (1936) were able to transmit the western strain of the virus to a ground squirrel, *Citellus richardsoni* (Sabine) through the bite of ticks, *Dermacentor andersoni* Stiles. The animal died five days after the ticks, which had fed on an inoculated guinea pig, were placed on the animal. Tyzzer, Sellards, and Bennett[71] in 1938 reported the natural occurrence of the infection in the ring-necked pheasant. Man was reported susceptible to the infection in 1938 by Howitt.[72]

Reeves (William C.), who later joined Hammon (W. McD.) of the Hooper Foundation for Medical Research in the study of the arthropodan encephalitides, began his research work under the direction of the author in 1940 and completed his work for the doctorate in October, 1943, the title of his thesis being "*Culex tarsalis* and Other Mosquitoes as Vectors of the Virus Encephalitides of Western North America." In 1941 Hammon, Reeves, Brookman, and Izumi[73] reported the isolation of the viruses of both western and St. Louis encephalitis from *Culex tarsalis* mosquitoes in the Yakima Valley of the State of Washington. It should be recalled at this point that mosquitoes were strongly suspected as the vectors in the 1933 St. Louis outbreak, in which there were over 1,000 cases with about 200 deaths. In 1942 Hammon and Reeves[74] reported, "We can safely conclude that this species [*Culex tarsalis* Coq. referred to above] is a natural vector . . . in respect to St. Louis encephalitis virus . . . and in addition . . . that the chicken may serve as a satisfactory reservoir of the virus."

Hammon,[75] in a paper read at the 1948 meeting of the California Mosquito Control Conference, points out repeated failure to find virus in overwintering wild mosquitoes; a chronic or latent stage of the infection was not observed in chickens or other vertebrates, and thus no true reservoir was found. Then came announcement by Smith[76] and co-workers and by Sulkin[77] that these viruses had been found in chicken mites, and furthermore that the virus was transmitted through the mite eggs from generation to generation. Here, then, appears, to be a true reservoir for the infection. In 1947 Reeves, Hammon, Furman, McClure, and Brookman[78] announced the recovery of western equine encephalomyelitis virus from wild bird mites *Liponyssus sylviarum* (Canestrini and Fanzago). The wild birds were the yellow-headed blackbird, *Xanthocephalus xanthocephalus* (Bonaparte). This discovery is significant in that it brings a *wild* vertebrate reservoir animal (a bird) into the encephalitis picture. Much more work needs to be done with mites in relation to the virus in order to elucidate their role as vectors.

Recently Hammon[79] (1948) has pointed out that the hypothetical cycle now becomes a more complicated one. "Mites infected previously, congenitally or by feeding on an infected bird, may initiate infection in nestling birds or in newly hatched fowl in the spring. These birds in turn are fed on by mosquitoes. After the essential extrinsic incubation period, these mosquitoes may infect other birds. Several such cycles in mosquitoes and birds will build up the number of infected mosquitoes and infection may then appear in men or horses. Infection is usually seen first in horses, and later in men, undoubtedly because of the greater number of mosquitoes having access to the large, unprotected domestic animals, and because of the mosquitoes' feeding preference."

Japanese "B" virus. According to Hammon[80] the most severe epidemic of encephalitis ever described occurred in Tokyo in the summer of 1924, with over 6,000 cases resulting in 3,797 deaths. In the Japanese epidemic of 1948 there were about 8,000 cases, with a 30 per cent mortality. (LaCasse, Proc. Amer. Mosq. Control Assoc., Feb., 1949.) That this virus also occurs in horses was shown by Hodes, and Peck[81] in Okinawa by complement-fixing and neutralizing antibodies. Mitamura, J.,[82] and associates report a small epidemic of encephalitis in the prefecture of Okayama in 1938, which lasted about 40 days. The chief species of mosquitoes taken were *Culex pipiens* var. *pallens* Coq., *Culex tritaeniorhynchus* Giles, and *Anopheles hyrcanus* var. *sinensis* Wied. Females of both species of *Culex* taken between July 25 and August 24 infected mice on which they fed. It is also reported that 12 mice became infected out of 511 bitten by female *Culex pipiens pallens* that had developed in the laboratory from larvae and pupae taken in nature, hence had had no opportunity of acquiring the virus in the adult stages. Forty species of mosquitoes are reported for Japan. *Culex annulirostris* Skuse has recently been reported by Hurlbut and Thomas as a potential vector in the Caroline Islands.

Among other recently discovered mosquito-borne encephalitis viruses are the following: West Nile, Semliki Forest, Rift Valley, and Mengo strains.

E. FILARIASIS

Filariasis. Infestation with nematode worms belonging to the family Filariidae is known as filariasis. The larval worms of this family are commonly known as microfilariae and occur in the circulatory and lymphatic systems, connective tissue layers, and serous cavities of the vertebrate hosts.

Bancroft's filaria, Wuchereria bancrofti (Cobbold), is a widely distributed parasite of man, being indigenous to Polynesia and Melanesia, where advanced elephantiasis (see p. 247) is frequently encountered; the disease is found in many tropical and subtropical areas of the globe, such as central Africa, southeast coastal China, and the East Indies. In the United States only one endemic area has been known in the past, a small area including Charleston, South Carolina.[83] Puerto Rico is reported to be one of the best-known endemic areas of filariasis in the Western Hemisphere, although the disease is not regarded as a major health problem there.[84] The infection also exists in the northeast coastal portions of South America.

The sheathed microfilariae, first noted by Bancroft in 1876, measure about .3 mm in length and from 7.5μ to 10μ in diameter (Fig. 81). As observed by Manson in 1877–78, they occur in the peripheral blood, particularly at night. In the daytime most of the microfilariae are concentrated in the pulmonary vessels, the capillaries of the heart, and other visceral organs. This nocturnal periodicity[85] enables night-flying mosquitoes such as *Culex quinquefasciatus* Wied. to suck up the parasites while biting. Manson called attention to this adaptation and believed that mosquitoes served as "nurses" for the filariae, and that when the mosquitoes dropped into water and disintegrated the organisms were liberated and infection of human beings resulted from drinking infected water.

Life cycle. The fully grown, very slender, sexually mature worms, the females reaching a length of 80 to 100 mm and the males about 40 mm, are often found (post-mortem) in tangles in nodular dilations of the distal lymphatics, in lymphocytic varices, and in the glands them-

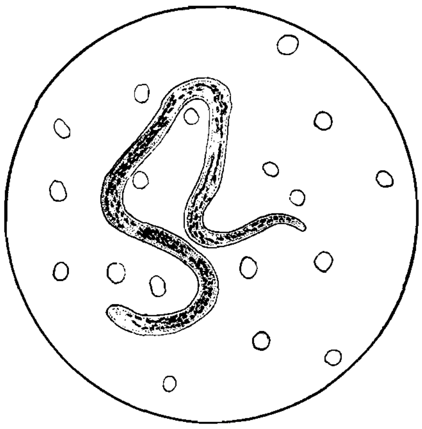


FIG. 81. *Wuchereria bancrofti,* in human blood. ×333.

selves or even in the thoracic duct. Here myriads of minute microfilariae are born, each in a sheath or sac. Although many remain in the lymph, others following a normal circulatory course will reach the peripheral circulation. Brown[86] reports an infection of 23,240 microfilariae per ml of blood in a Virgin Islander (except for a slight general glandular enlargement, there were no symptoms referable to this infection). In the so-called classic filariasis the microfilariae manifest a nocturnal periodicity (filariae sanguinis hominis nocturna), i.e., they are present in the peripheral blood of infected persons principally during the hours from 10 P.M. to 2 A.M. There is, however, a nonperiodic form of *bancrofti* in the South Pacific area. In either instance the microfilariae are available to appropriate species of mosquitoes, i.e., anthropophilous night feeders in the case of "filaria nocturna," and day biters in the case of "filaria diurna." Many species of mosquitoes belonging to four common genera, *Culex, Aedes, Mansonia,* and *Anopheles,* have been incriminated as vectors, some species being more readily infective than others.

The more numerous the individual mosquitoes in a large population of infected human beings the more dangerous is the situation. It must be remembered that this is an infection referred to earlier in which the pathogen does not multiply within the body of the vector; the mosquito must imbibe one microfilaria from the blood of an infected person for every filarial worm transmitted to a new victim. Having successfully reached the stomach of the mosquito, the tiny worm now loses its sac-like sheath and in a matter of hours forces its way through the stomach wall and works its way through the thoracic muscles, becoming somewhat "sausage-shaped" by the fourth or fifth day. There are two molts, and by the end of the twelfth to fourteenth day the digestive tract has developed, and at least some infective larvae, now about 1.5 mm in length, settle in the region of the head and labium of the mosquito (many larvae are lost in the abdomen) awaiting an opportunity to slip down the salivary channel when the mosquito bites. The number of larvae per mosquito successfully reaching this stage is evidently quite small (heavy infections may kill the mosquito); Hu and Yen[87] report an average of 3.9.

When the mosquito bites, the larvae quickly slip down the labium and escape from the labellum, coming to lie upon the warm skin of the person bitten. The larvae now penetrate the skin, enter the lymph stream, and are carried to the lymphatics, as mentioned in the beginning, where development to sexual maturity is accomplished in a matter of months; from 12 to 18 months is reported probable from the time of infection to the maturity and appearance of a new generation of microfilariae in the peripheral blood.

Asymptomatic filariasis. In endemic areas a considerable percentage of the population are infected with the parasite, as shown by blood smear examination: thus Murray[88] reported 19.1 per cent positive for microfilaria in a survey of 5,144 Samoans. Brown (*loc. cit.*) states that on physical examination the infected person, though unaware of the infection, may exhibit a general glandular enlargement especially of the inguinal lymph glands.

Inflammatory filariasis. The author has observed cases of so-called "mumu" in which there were recurrent attacks of inflammatory filariasis, many months after exposure to infection in the South Pacific area. Brown (*loc. cit.*) suggests that these inflammatory reactions "may be an allergic phenomenon due to a sensitivity to the products of the worms or to a superimposed bacterial infection, usually streptococci."

Obstructive filariasis (elephantiasis). The obstructive types of filariasis (grossly enlarged scrotum, vulvae, breasts, or legs) usually follow years after the original long exposure to mosquito bites in endemic areas and are the exception rather than the rule. Microfilariae are frequently absent from the blood, although Murray (*loc. cit.*) states that he found that, in American Samoa, persons afflicted with elephantiasis were much more likely to have microfilariae than those without this condition.

Brug's filaria, Wuchereria malayi Brug, the causal organism of "filariasis malayi" is very similar to that of "Bancroft's filariasis." It occurs in India, Ceylon, Thailand, the East Indies, the Philippines, and New Guinea. In some areas, such as portions of coastal China and some islands of the East Indies, the two Wuchereria infections overlap; the behavior of the worms in the mosquito vector, as well as in man, and the effects on the human host are very similar. *W. malayi*, which is essentially nocturnal in periodicity, differs from *W. bancrofti* only in minor details. The morphology of the sheathed microfilariae differs chiefly in the cephalic space, which is twice as long as broad, and the posterior extremity of the worm has a slight bulb at the tip with two minute terminal nuclei.

While there is little difference in the causal organisms, it is important to note that the principal vector mosquitoes of *W. malayi* are members of the *Mansonia* group in which the wrigglers attach themselves to submerged aquatic plants such as *Pistia* and *Eichhornia*, as described in a previous chapter. *Mansonia* (*Mansonoides*) *annulifera* Theobald is considered to be the principal vector, although several other related species as well as certain anophelines, e.g., *Anopheles hyrcanus sinensis* Wiedemann, are believed to be implicated.

Mosquito species involved. Throughout the filarial belt of the world (between 30° N and 32° S latitude) some 60 species of mosquitoes belonging to half a dozen genera have been implicated in greater or lesser degree as vectors of the infection. *Culex quinquefasciatus* Say, a widely distributed tropical and subtropical species, heads practically all lists of vectors; it is, however, significant that in the Samoan area experimental evidence disclosed that only 29 per cent of *C. quinquefasciatus* could be infected from a single blood meal, whereas 90 per cent of *Aedes pseudoscutellaris* (Theob.) became infected; also, it is reported that only one per cent of the former developed microfilariae to the infective stage in contrast to 18 per cent in the *Aedes* species. In several island groups of the South Pacific (among them Solomon, New Hebrides, Fiji, and Samoa) a primary vector is *Anopheles farauti* Lav. which often becomes tremendously infected and dies as a result; as many as 96 eight- to ten-day-old larvae have been observed (Reiber). Only 11 per cent of the total number of infected mosquitoes survived long enough to transmit larvae. Under natural conditions this percentage would no doubt be still lower.

In the China area *Anopheles hyrcanus sinensis* Wiedemann and *Culex pipiens pallens* Coquillett, are proven vectors, as they are also in Japan together with *Aedes togoi* (Theobald); in west Africa *Anopheles gambiae* Giles is listed as a vector (see above for filariasis malayi).

In testing the susceptibility of North American species of mosquitoes to *Wuchereria bancrofti* (Bora Bora strain) Eyles and Most[89] found that development to advanced or infective stage occurred in the following species: *Culex pipiens* Linn., *C. quinquefasciatus* Say, *C. erraticus* (Dyar and Knab), *C. salinarius* Coq., *Anopheles walkeri* Theob., *A. punctipennis* (Say), *Aedes triseriatus* (Say), *A. aegypti* Linn., *Aedes atropalpus* (Coq.), and *Mansonia perturbans* (Walker). In the following species development did not proceed beyond the first larval stage: *Anopheles quadrimaculatus* Say, *Anopheles freeborni* Aitken, *Aedes tormentor* Dyar and Knab, and *Psorophora ferox* Humboldt.

With only three specimens of *Culex tarsalis* Coq. tested, Scott, Richards, and Seaman[90] found that one of these developed infective stages of *W. bancrofti* in the thorax, head, and proboscis.

Heartworm of dogs. *Dirofilaria immitis* (Leidy), a cosmopolitan species, occurs in dogs (in over 30 per cent in some parts of the southern United States) and occasionally in cats, as well as in various wild carnivores. The adult worms, females measuring in length from 25 to 35 cm and the males 12 to 18 cm, invade the heart (right ventricle) and pulmonary artery of the host, where they often form tangled knots and may cause death as the result of embolism, asphyxia, and dilation of the heart. This parasite has become widely distributed in the United States.

Like *Wuchereria bancrofti* the microfilariae of *D. immitis* are found in the blood stream and manifest a similar periodicity, except that more of the latter appear to be present in the peripheral circulation during the daytime (Hinman). The larvae of *D. immitis* are without sheaths. *Culex pipiens* Linn., *Aedes aegypti* (Linn.), as well as a number of other species of mosquitoes, are known to be vectors. Development in the mosquito is similar to that of *Wuchereria* as described above. Sexual maturity of the worms in the body of the dog is said to require from 5 to 9 months. Short-haired dogs are believed to be more susceptible to heartworms than long-haired dogs, probably because they are less protected against mosquito bites. To prevent infection in endemic areas, dogs must be protected from mosquitoes, particularly at night.

Other filarial worms. Among other species belonging to the family Filariidae are *Mansonella ozzardi* (Manson), said to be nonpathogenic to man, transmitted by *Aedes aegypti* (Linn.); *Acanthoceilonema perstans* (Manson), transmitted by *Culicoides austeni* Carter, Ingram, and Macfie (see Chapter X); *Loa loa* (Cobbold), causing calabar swellings, transmitted by *Chrysops dimidiata* v. d. Wulp (see Chapter XIV); *Onchocerca volvulus* Leuckart, transmitted by *Simulium damnosum*

Theo., *Simulium ochraceum* Walker, and other species of black flies (see Chapter X).

F. RABBIT MYXOMATOSIS

Rabbit myxomatosis, also known as "big head" of rabbits, is caused by a virus closely related serologically and immunologically to the virus of rabbit fibroma (Shope).[91] Experimental observations by Philip[92] on the latter virus indicate that the infection can be transmitted mechanically in the laboratory by several species of conenose bugs (Family Reduviidae), e.g., *Triatoma protracta, Triatoma infestans,* and *Rhodnius prolixus.* It was presumed that the mechanical transfer of the infection in interrupted feeding tests was by the contaminated mouth parts of the bugs.

The virus of *rabbit myxoma* produces lesions in the skin, lymph glands, tunica vaginalis, testicle, spleen, and lungs. The disease runs a rapid course, and death ensues within a week or two. Rabbits are the only known susceptible hosts. Recently Aragão[93] (1943) has reported successful transmission of the virus by mosquitoes, *Aedes scapularis* (Rondani) and *A. aegypti* (Linn.). Here, as in the tests made by Philip with rabbit fibroma, the transmission of the virus was thought to be mechanical; i.e., only the proboscides of the mosquitoes were contaminated. The infected mosquitoes were able to transmit the virus two or three times up to 17 days after the infecting blood meal. Other mosquitoes as well as other bloodsucking arthropods have been incriminated in transmission.

G. FOWL-POX

Fowl-pox, an important virus disease of poultry, while spread in various ways, such as contact between diseased and healthy birds, may also be spread, according to Brody,[94] by *Aedes stimulans* (Walker) by intermittent feeding, harboring the virus in or on its body for at least two days following an infective meal, and by *Aedes aegypti* (Linn.), which can definitely transmit the virus more than once during its life. The latter species is able to transmit the disease within one hour after an infective meal and continues to be infective for at least about 40 days.

BIBLIOGRAPHY

1. Boyd, Mark F., 1941, "An historical sketch of the prevalence of malaria in North America," *Am. J. Trop. Med.*, 21:223–44.

2. Meleney, H. E., 1937. "The problem of malaria mortality in the United States," *Am. J. Trop. Med.*, 17:15–24.

3. Faust, Ernest C.; Hess, Archie D.; and Young, Martin D.; 1945. "Malaria mortality and morbidity in the United States for the year 1944." *J. Nat. Malaria Soc.*, 5:103–12.

4. Reed, A. C., 1937. "Ultimate prognosis of hookworm disease, malaria, and amebiasis," *Proc. 27th Annual Meeting Medical Section of American Life Convention,* pp. 176–206.

5. Russell, P. F.; West, L. S.; and Manwell, R. D.; 1946. *Practical Malariology.* Philadelphia: W. B. Saunders Co. 684 pp.

6. *Symposium on Human Malaria,* 1941. Washington, D. C.: Am. Assn. Advancement Sc., Publ. no 15.

7. King, A. F. A., 1883. "Insects and disease, mosquitoes and malaria," *Pop. Sc. Monthly,* 23:644–58.

8. Golgi, C., 1886. "Sulla infezione malarica." *Arch. per le Sc. Med.,* 10:110.

9. Ross, R., 1897. "Observations on a condition necessary to the transformation of the malaria crescent," *Brit. M. J.* 1:251; also, "On some peculiar pigmented cells found in two mosquitoes fed on malarial blood, *ibid.,* 2:1786–88.

10. MacCallum, W. C., 1898. "On the haematozoan infections of birds," *J. Exper. Med.,* 3:117–36.

11. Grassi, B.; Bignami, A.; and Bastianelli, G.; 1899. "Ciclo evolutivo delle semilune nell' *Anopheles claviger* ed altri studi sulla malaria dall' ottobre 1898 al maggio 1899." *Atti d. Soc. per studi d. malaria,* 1:14–27. (Cited by Ross 1910.)

12. Manson, Sir Patrick, 1909. *Tropical Diseases.* London: Cassel and Company, xx + 876 pp.

13. Bass, C. C., and Johns. F. M., 1912. "The cultivation of malarial plasmodia *in vitro,*" *J. Exper. Med.,* 16:567–79.

14. Wilcox, Aimée, 1942. *Manual for the Microscopical Diagnosis of Malaria in Man.* Washington, D. C.: U. S. Public Health Service, in Nat. Inst. Health Bull., no. 180. ix + 39 pp. (16 plates).

15. Metcalf, R. L., 1944. "The detection of the plasmodia of human malaria in blood films by fluorescence microscopy," *J. Nat. Malaria Soc.,* 4:223–29.

16. Ross, R., 1910. *The Prevention of Malaria.* New York: E. P. Dutton & Co. xx + 669 pp.

17. Rodhain, J., 1948. "Susceptibility of the chimpanzee to *P. malariae* of human origin," *Am. J. Trop. Med.,* 28:629–31.

18. Golgi, C., 1889. "Sul ciclo evolutivo dei parassiti malarici nella febbre terzana; diagnosi differenziale tra i parassiti endoglobulari malarici della," *Arch. per Sc. Med.,* 13:173–96.

19. Mayne, Bruce, 1938. *Graphic Reproduction of the Life Cycle of the Malaria Parasite in the Mosquito Host.* Washington, D. C.: U. S. Public Health Service, in Nat. Inst. Health Bull, no. 170. 11 pp. (26 plates).

20. Huff, Clay, G., 1947. "Life cycle of malaria parasites," *Annual Review Microbiol.,* 1:43–60.

21. "Symposium on Exoerythrocytic Forms of Malaria Parasites, Parts I–VI," *J. Parasitol.,* 34:261–320.

22. Huff, Clay G., 1948. "Exoerythrocytic stages of malarial parasites," *Am. J. Trop. Med.,* 28:527–31.

23. ———, 1934. "Comparative studies on susceptible and insusceptible *Culex pipiens* in relation to infections with *Plasmodium cathemerium* and *P. relictum*," *Am. J. Hyg.*, 19:123–47.

24. Hunter, George W., 3rd.; Weller, Thomas H.; and Jahnes, William G. Jr.; 1946. "An outline for teaching mosquito stomach and salivary gland dissection," *Am. J. Trop. Med.*, 26:221–28.

25. Beyer, G. E.; Potheir, O. L.; Courtet, M.; and Lemann, J.; 1901–02. "I. Bionomics–Experimental Investigation with *Bacillus sanarelli* and Experimental Investigation with Malaria," *New Orleans M. & S. J.*, 54:419–80.

26. Thayer, W. S., 1900. "On recent advances in our knowledge concerning the etiology of malarial fevers," *Philadelphia. M. J.*, 5:1046–48.

27. King, W. V., 1916. "Experiments on the development of malaria parasites in three American species of Anopheles," *J. Exper. Med.*, 33:703–16.

28. Darling, S. T., 1910. *Studies in Relation to Malaria. Isthmian Canal Commission Report.* Washington, D. C.: Gov. Print. Office. 38 pp.

29. Moore, Joseph A.; Young, Martin D.; Hardman, Newton H.; and Stubbs, Tradwick H.; 1945. "Studies on imported malarias: 2. Ability of California anophelines to transmit malarias of foreign origin and other considerations," *J. Nat. Malaria Soc.*, 4:307–29.

30. Hindle, Edward, 1914. *Flies in Relation to Disease–Bloodsucking flies.* London: Cambridge University Press. xv + 398 pp.

31. White, R. Senior, 1937. "The maxillary index of the Indian anophelines," *Records of the Malaria Survey of India,* 7:155–77. (Contains an excellent review of this subject with bibliography.)

32. Mitzmain, M. B., 1916. "An attempt to determine the number of persons one mosquito can infect with malaria," U. S. Public Health Service, *Pub. Health Rep.*, 31:2325–35.

33. King, W. V., 1917. "The effect of cold upon malaria parasites in the mosquito host," *J. Exper. Med.*, 25:495–98.

34. Knowles, R., and Basu, B. C., 1943. "Laboratory studies on the infectivity of *Anopheles stephensi*," *J. Malaria Inst. India*, 5:1–30.

35. Mitzmain, M. B., 1916. *Is mosquito or man the winter carrier of malaria organism?* Washington, D. C.: Govt. Print. Office, in Pub. Health Bull., no. 84. 32 pp.

36. Russell, Paul F.; Rozeboom, Lloyd E.; and Stone, Alan; 1943. *Keys to the Anopheline Mosquitoes of the World with Notes on their Identification, Distribution, Biology, and Relation to Malaria.* Philadelphia: Amer. Entomolog. Soc. 152 pp.

37. Herms, W. B., and Gray, H. F., 1944. *Mosquito Control: Practical methods for abatement of disease vectors.* 2nd ed. New York: The Commonwealth Fund. viii + 419 pp.

38. Soper, Fred L., and Wilson, D. Bruce, 1943. *Anopheles gambiae in Brazil 1930 to 1940.* New York: The Rockefeller Foundation, xviii + 262 pp.

39. Christophers, S. R.; Sinton, J. A.; Covell, G.; 1936. *How To Do a Malaria Survey.* 3rd ed. (revised by J. A. Sinton), in Health Bull. no. 14, Malaria Bureau. no. 6. Malaria Survey of India, 206 pp.

40. Elmendorff, John E., Jr., 1941. "Malaria survey: Methods and pro-

cedures," pp. 295–301 in *Symposium on Human Malaria.* Washington, D. C.: Am. Assn. Advancement Sc., Publ. no. 15.

41. Aitkin, Thomas H. G., 1948. "Recovery of anopheline eggs from natural habitats. An aid to rapid survey work," *Ann. Entomolog. Soc. Amer.,* 41:327–29.

42. Herman, Carlton M., 1944. "The blood protozoa of North American birds," *Bird Banding* (A Journal of Ornithological Investigations), 15:89–112.

43. Milam, D. F., and Kusch, Ernest, 1938. "Observations on *Plasmodium knowlesi* malaria in general paresis," *South. M. J.,* 31:947–49.

44. Clark, H. C., and Dunn, L. H., 1931. "Experimental efforts to transfer monkey malaria to man," *Am. J. Trop. Med.,* 11:1–10.

45. Carter, Henry Rose, 1931. *Yellow Fever–An Epidemiological and Historical Study of Its Place of Origin.* Baltimore: Williams and Wilkins Co. xii + 308 pp.

46. Reed, Walter; Carroll, James; Agramonte, A.; and Lazear, Jesse W.; 1900. "The etiology of yellow fever–a preliminary note," *Proc. Twenty-eighth Annual Meeting, Amer. Pub. Health Assn.,* Indianapolis, Oct. 22–26.

47. Herrick, G. W., 1939. "The last epidemic of yellow fever," *Scient. Monthly,* 49:401–8.

48. Davis, Nelson C., 1932. "Effect of various temperatures in modifying extrinsic incubation period of yellow fever virus in *Aedes aegypti,*" *Am. J. Hyg.,* 16:163–176.

49. Soper, Fred L.; Wilson, D. Bruce; Lima, Servulo; Antunes, Waldemar Sá; 1943. *The Organization of Permanent Nation-wide Anti-Aedes aegypti Measures in Brazil.* New York: The Rockefeller Foundation. xiii + 137 pp.

50. The Rockefeller Foundation, *A Review for 1936,* by Raymond B. Fosdick, President of the Foundation. 1937, pp. 13–17.

51. Soper, F. L.; Penna, H.; Cardosa, E.; Serafim, J., Jr.; Frobisher, M., Jr.; and Pinheiro, J.; 1933. "Yellow fever without *Aëdes aegypti:* Study of a rural epidemic in the Valle do Chanaan, Espírito Santo, Brazil, 1932," *Am. J. Hyg.,* 18:555–87.

52. Burke, A. W., 1937. "An epidemic of jungle yellow fever on the Planalto of Matto Grosso, Brazil," *Am. J. Trop. Med.,* 17:313–34.

53. Taylor, R. M., and Laemmert, H. W., *et al.,* 1946. "An epidemiological study of jungle yellow fever in an endemic area in Brazil," *Am. J. Trop. Med.,* 26 (Nov. Supp.): 1–69.

54. Shannon, R. C.; Whitman, L.; and Franca, M.; 1938. "Yellow fever virus in jungle mosquitoes," *Science,* 88:110–11.

55. Taylor, R. M., and Theiler, Max, 1948. "The epidemiology of yellow fever," *Proc. 4th Internat. Cong. Trop. Med. & Malaria,* Washington, D. C. (Abstracts.)

56. Bennett, Byron L.; Baker, Fred C.; Sellards, Andrew W.; 1938. "The behavior of the virus of yellow fever in the mosquito, *Aedes triseriatus,*" *Science,* 88:410–11.

57. Chandler, Asa C., and Rice, Lee, 1923. "Observations on the etiology of dengue fever," *Am. J. Trop. Med.,* 3:233–62.

58. Ashburn, P. M., and Craig, C. F., 1907. "Experimental investigations

regarding the etiology of dengue fever," *Philippine J. Sc.*, B., 2:93–147 (27 charts and one map).

59. Simmons, J. S.; St. John, Joe H.; and Reynolds, F. H. K.; 1931. *Experimental Studies of Dengue.* Philippine Bur. Sc., Monograph no. 29. 489 pp. (3 plates).

60. Graham, H., 1902. "The dengue: A study of its pathology and mode of propagation," *Med. Rec.*, 61:204–7.

61. Meyer, K. F.; Haring, C. M.; and Howitt, B.; 1931. "Newer knowledge of the neurotropic virus infections of the horse," *J. Am. Vet. M. A.*, 79, n.s. 32:370–89.

62. Herms, W. B.; Wheeler, C. M.; and Herms, H. P.; 1934. "Attempts to transmit equine encephalomyelitis by means of bloodsucking insects, especially mosquitoes," *J. Econ. Entomol.*, 27:987–98.

63. Kelser, R. A., 1933. "Mosquitoes as vectors of the virus of equine encephalomyelitis," *J. Am. Vet. M. A.*, 82, n.s. 35:767–71.

64. Madsen, D. E.; Knowlton, G. F.; and Rowe, J. A.; 1936. "Further studies on transmission of equine encephalomyelitis by mosquitoes," *J. Am. Vet. M. A.*, vol. 89, n.s. 42:187–96.

65. Merrill, M. H.; Lacaillade, C. Wm., Jr., and Ten Broeck, Carl; 1934. "Mosquito transmission of equine encephalomyelitis," *Science*, 80:251–52.

66. Madsen, D. E., and Knowlton, G. F., 1935. "Mosquito transmission of equine encephalomyelitis," *J. Am. Vet. M. A.* 86, n.s. 39:662–66.

67. Simmons, J. S.; Reynolds, F. H. K.; and Cornell, V. H., 1936. "Transmission of the virus of equine encephalomyelitis through *Aedes albopictus* Skuse," *Am. J. Trop. Med.*, 16:289–302.

68. Kelser, R. A., 1937. "Transmission of the virus of equine encephalomyelitis by *Aedes taeniorhynchus*," Science, 85:178.

69. Kitselman, C. H., and Grundmann, A. W., 1940. "Equine encephalomyelitis virus isolated from naturally infected *Triatoma sanguisuga* LeConte." Kansas Agric. Exper. Tech. *Bull.*, no. 50. pp. 1–15.

70. Syverton, J. T., and Berry, G. P., 1936. "Arthropod vector for equine encephalomyelitis, western strain," *Science*, 84:186–87.

71. Tyzzer, E. E.; Sellards, A. W.; and Bennett, B. L.; 1938. "The occurrence in nature of 'equine encephalomyelitis' in the ring-necked pheasant," *Science*, 88:505–6.

72. Howitt, B., 1938. "Recovery of the virus of equine encephalomyelitis from the brain of a child," *Science*, 88:455–56.

73. Hammon, W. McD.; Reeves, W. C.; Brookman, Bernard, and Izumi, E. M., 1941. "Isolation of the viruses of Western equine and St. Louis encephalitis from *Culex tarsalis* mosquitoes," *Science*, 94:328–30.

74. ———, and ———, 1942. "*Culex tarsalis* Coq., a proven vector of St. Louis encephalitis," *Proc. Soc. Exper. Biol. & Med.*, 51:142–43.

75. ———, 1948. "Advances in knowledge of the vectors and arthropod reservoirs of certain of the encephalitides," *Proc. and Papers of 16th Annual Conference California Mosquito Control Assn.*, (Berkeley), pp. 4–6.

76. Smith, M. B.; Blattner, R. J.; and Heys, F. M.; 1944. "The isolation of the St. Louis encephalitis virus from chicken mites *(Dermanyssus gallinae)* in nature," *Science*, 100:362–63.

77. Sulkin, S. Edward, 1945. "Recovery of equine encephalomyelitis virus (western type) from chicken mites," *Science,* 101:381–83.

78. Reeves, W. C.; Hammon, W. McD.; Furman, D. P.; McClure, H. E.; and Brookman, B.; 1947. Recovery of western equine encephalomyelitis virus from wild bird mites *(Liponyssus sylviarum)* in Kern County, California," *Science,* 105:411–12.

79. Hammon, W. McD., 1948. "The arthropod-borne virus encephalitides," *Am. J. Trop. Med.,* 28:515–25.

80. ———, 1945. "The encephalitides of virus origin, with special reference to those of North America," *Clinics,* 4:485–503 (with 57 references).

81. Hodes, Horace L.; Thomas, Lewis; and Peck, John L.; 1946. "Complement-fixing and neutralizing antibodies against Japanese B virus in the sera of Okinawan horses," *Science,* 103:357–59.

82. Mitamura, T.; Kitaoka, M.; Watanabe, S.; Hosoi, T.; Tenjiu, S.; Seki, O.; Nagahata, K.; Jo, K.; and Shimizu, M.; 1939. "Weitere Untersuchungen über die Uebertragung der japanischen epidemischen Enzephalitis durch Mücken." *Tr. Soc. Path. Japan,* 29:92–105.

83. Francis, Edward, 1919. *Filariasis in Southern United States.* U. S. Public Health Service, Hygienic Lab. Bull., no. 117. 34 pp., IX plates.

84. Tampi, M. K., 1931. "A study of filariasis in Porto Rico," *Porto Rico J. Pub. Health & Trop. Med.,* 6:435–41.

85. Hinman, E. Harold, 1936. "Attempted reversal of filarial periodicity in *Dirofilaria immitis,*" *Proc. Soc. Exper. Biol. & Med.,* 33:524–27.

86. Brown, H. W., 1945. "Current problems in filariasis," *Am. J. Pub. Health,* 35:607–13.

87. Hu, Stephen M. K., and Yen, Chia-Hsien, 1933. "Studies on the susceptibility of *Culex pipiens* Linn. var *pallens* Coq. to experimental infection with *Wuchereria bancrofti* Cobbold in Shanghai area," *Chinese M. J.,* 47:1359–66.

88. Murray, William D., 1948. "Filariasis studies in American Samoa," *Naval Med. Bull.,* 48:327–41.

89. Eyles, Don E., and Most, Harry, 1947. "Infectivity of Pacific Island *Wuchereria bancrofti* to mosquitoes of the United States," *Am. J. Trop. Med.,* 27:211–20.

90. Scott, O. K.; Richards, C. S.; and Seaman, E. A.; 1945. "Experimental infection of Southern California mosquitoes with *Wuchereria bancrofti,*" *J. Parasitol.,* 31:195–97.

91. Shope, R. E., 1932. "A filterable virus causing a tumor-like condition in rabbits and its relationship to *Virus myxomatosum,*" *J. Exper. Med.,* 56:803–22.

92. Philip, Cornelius B., 1942. "Mechanical transmission of rabbit fibroma (Shope) by certain haemophagous bugs," *J. Parasitol.* 28:395–98.

93. Aragão, H. B., 1943. "O virus do mixoma coelho do mato (Sylvilagus) sua transmissão pe los *Aedes scapularis e aegypti,*" *Mem. Inst. Oswaldo Cruz,* 38:93–99.

94. Brody, Arthur L., 1936. *The Transmission of Fowl-Pox.* Cornell Univ. Agric. Exp. Sta. Memoir no. 195. 37 pp.

CHAPTER XIII

MOSQUITO ABATEMENT

Historical. Ancient Greeks and Romans believed there was some etiologic connection between intermittent fevers and swamps, hence the use of *drainage methods* in Greece and especially Rome to overcome the "poisoning effects" of stagnant water. Lancisi, the greatest Italian physician of his time, published a treatise on swamp fevers in 1717, according to Russell, in which he suggests that since malaria disappeared after drainage, the poison from the marshes was possibly transmitted by mosquitoes which also came from the swamps.

The use of larvicides dates back to about 1793 (Russell, *et al., loc. cit.*, p. 215) when oil (probably whale oil) was used in Philadelphia on rain barrels to kill mosquito larvae.

L. O. Howard,[1] for many years (1894-1924) chief of the United States Bureau of Entomology, records "an experiment against mosquitoes" which he conducted in 1892. He "sprinkled four ounces of coal oil upon the surface of the pond" which "contained 60 square feet. . . . The pool which upon the evening of the 5th (July) had been teeming with animal life, contained no living insects during the following ten days. The kerosene, curiously enough, seemed to exercise no deterrent effect upon the adult female mosquitos. They still continued to attempt to deposit eggs and in this attempt were destroyed. This is in my opinion a most important point, and one which has hardly been anticipated." This experiment was made in the Catskill Mountains of New York at an elevation of about 2,500 feet.

Ronald Ross in his famous book, entitled *The Prevention of Malaria*,[2] states: "Since 1899 I had gradually become convinced that anti-mosquito campaigns had better be conducted, not only against Anophelines but against all kinds of mosquitoes at once." He refers to his advice given on the spot in 1902 to continue general mosquito reduction at Ismailia on the Suez Canal; within a year, he states, mosquitoes of both groups (Culicine and Anopheline) were almost entirely banished and the admissions for malaria reduced from about 2,000 a year to 214. Ross also refers to the brilliant campaign commenced in 1901 by Malcolm Watson in the Federated Malay States at Klang and Port Swettenham.

The remarkable work of Gorgas in controlling yellow fever in Havana in 1901 and that of Gorgas and Le Prince[3] in the control of yellow fever and malaria in the Panama Canal Zone attracted widespread attention to the subject of mosquito abatement. Ross (1902), in his book *Mosquito Brigades and How to Organize Them,*[4] gave much practical information regarding methods of mosquito control. In 1939 Herms and Gray published the first edition of their work on mosquito control (now in the second edition).[5]

L. O. Howard contributed a special article to Ross's book on malaria prevention. In this article, which was entitled "Anti-malaria work in the United States," Howard says " . . . beginning with 1900 and gradually increasing in amount year by year, a great deal of anti-mosquito work, more or less well planned, has been done in different parts of the United States; but, looking over the entire field it is astonishing to note how little of this work has been done with a direct sanitary object in view." He remarks that, "It is pleasing, in the face of so much of this sort of thing, to note a well-directed and rather large-scale bit of work with a direct anti-malarial bearing which was begun at an early date, and this is the work on Staten Island under Dr. A. H. Doty, the health officer of the port of New York." This work was evidently started in 1901.

Public interest in the United States was greatly stimulated by the appearance, in 1904, of J. B. Smith's report on the mosquitoes of New Jersey.[6] Salt-marsh mosquito abatement on the Pacific coast began in 1904 at San Rafael, Marin County, California, under the direction of C. W. Woodworth, and at Burlingame, California, in 1905.[7] In 1910[8] the first specific malaria-mosquito control work in California was begun by the author (Herms) at Penryn in Placer County. The campaign was locally supported. Somewhat prior to 1910 the author called attention to the danger from malaria due to anopheline breeding in seepage from poorly constructed irrigation ditches and canals in California. "The use of metal, cement, or tile irrigation ditches, which prevent lateral flow except where wanted, will help greatly in lessening the vast number of (malaria) mosquitoes now produced in or by poorly kept ditches." Farmers of northern California were admonished to "pay more attention to the improvement of their irrigation methods."

In 1913[9] Carter of the United States Public Health Service began to make malaria surveys in Virginia and North Carolina. In 1914 a Federal appropriation of $16,000 was provided for malaria investigations by the Public Health Service; 22 surveys and three malaria control demonstrations were made in seven states in that year. From 1914 to 1928 the United States Public Health Service conducted directly or cooperatively malaria-mosquito control work in 343 communities in 17 states and, including surveys and investigations, in 667 communities in 24 states.

Organization for abatement work. Before actual mosquito abatement work is undertaken, it must be well planned and organized (Herms and Gray, *loc. cit.*). There must be an adequate preliminary survey. The services of an experienced and practical mosquito-abatement expert should be secured to make this survey. The expenses for the preliminary survey may be defrayed from funds raised by public subscription or by any other means.

The preliminary report should include data and recommendations on the following matters:

1. The boundaries of the area which should be included within the project.
2. The population, area, and assessed valuation of the proposed district.
3. The location and areas of the principal breeding situations such as marshes, etc.
4. Tentative suggestions as to the best method of control for each principal breeding area.
5. The amount and types of domestic breeding, and measures for abatement.
6. The particular species of mosquitoes involved, with a brief discussion of their breeding habits.
7. The probable organization that will be required, including personnel and equipment.
8. Detailed preliminary estimates of cost, both for permanent work (including capital outlays) and for regular maintenance. With these should be submitted comparative costs for already operating districts of similar size and conditions.
9. The economic losses caused by mosquitoes in the proposed district, and the economic savings which should result from adequate mosquito abatement.
10. Where a mosquito-borne disease is involved, statistical material of epidemiological importance should be included (see malaria survey, p. 230, for pattern).

Personnel. The general supervision of organized community mosquito control work, particularly as to policies and finance, is usually in the hands of a board or commission appointed in various ways in different states. The proponents of the undertaking should make the proper representations to the appointing power or powers, so that only citizens of outstanding character and ability are chosen. It is desirable that the members of the board or commission serve without compensation, except that expenses incurred in the performance of duty should be defrayed. In making selections it is well to include an outstanding physician, a public-minded attorney, a respected and able civil or sanitary engineer, and a successful business man—all should have a deserved reputation for unselfish public service.

The most important duty of the board or commission is the selection of the executive officer, who for the larger districts should have at least the following qualifications: (1) agreeable personality and honesty, (2) successful experience in mosquito abatement work, (3) administrative ability, (4) training in medical entomology and sanitary engineering. For very small districts this officer may well be of the working-foreman type.

A responsible group of citizens whose duty it is to administer the expenditure of considerable sums of local tax money for mosquito control is not likely to appoint as manager or executive officer a man who is simply brimful of so-called personality on the assumption that he will make a good "public relations" man. A good personality is an important asset, but the tax-paying public is particularly concerned with the man's ability to "deliver the goods." Thorough familiarity with the goods, in this instance mosquito control, is essential to good public relations.[10] Personnel should be carefully selected so as not to freeze into the organization at the outset individuals who are unfit for this type of work.

In large districts it is necessary to subdivide the force into divisions which can be handled by a responsible *foreman* or *inspector* and a crew of laborers. The foreman should be a full-time employee, active, energetic, interested in his work, and able to supervise his men in all sorts of mosquito control operations. As he is in immediate contact with the public, he must have a good personality, and must be able to get along with people. He will require much "backbone," plus self-restraint and patience. He must be physically equal to sustained activity in the field, for the work is frequently arduous. The regular crews should be carefully selected and kept on a year-round basis, with temporary additions during the peak of the breeding season as needed.

Adequate office facilities must be provided, inclusive of clercial and telephone service, for public contact and business purposes. Detailed maps of the district must be available, a reporting system, and a bookkeeping system in conformance with legal requirements must be developed. The office should maintain a skillful and continuous program of publicity and public education.

Training of personnel and education of the public. In order to secure a high level of performance in mosquito control operations the administrative and operational staff of a mosquito abatement district must be properly trained in control techniques. Mosquito control involves highly technical skills and complicated procedures which unless correctly administered may injure the public health and in agricultural areas may prove injurious to agriculture. Also in order to secure full cooperation of the public there must be a program of public education. One or more members of the staff should be trained to carry this out; in fact, the nature of mosquito control is such that all members of the staff, and laborers as well, are in constant contact with the public while on duty, hence all have a measure of responsibility in this regard; even a minor "slip up" may cause a "break down" in public relations.

Among similar agencies in other states, the California State Department of Public Health through the Bureau of Vector Control maintains a "Mosquito Control Training Program" designed to provide the operational personnel of the mosquito control agencies of California with the fundamentals of mosquito control. Two training teams, each consisting of one entomological instructor, one operational instructor, and an assistant equipped with an outfitted station wagon, covered the state from San Diego to Redding in 1948 holding 13 field training programs serving 39 mosquito abatement districts, 7 local health departments, 4 United States Navy installations, and 5 other agencies during February, March, and April. In addition to lectures and laboratory instruction, there were field demonstrations in the use of insecticides and equipment. Attendance certificates were given, and much informational literature was distributed. Instructors used on these teams were required to have had field experience and top training, particularly since many of those receiving instruction were men of experience themselves.

Mosquito surveys. The simplest form of mosquito survey on which policy and action can be based has as its minimum objective the accurate *determination of all species* of mosquitoes present in a given locality, *estimating* their relative *abundance*, *mapping* their distribution, and *studying* their respective *breeding places*. The survey will delimit the boundaries of the area which should be included within the project; it should ascertain the population and assessed valuation of the area; it should specify in general the best method of control for the principal breeding areas and the probable organization that will be required (personnel and equipment); it should furnish estimates of cost both for permanent control work and for regular maintenance.

Surveys must be done intelligently (i.e., by qualified persons) and with adequate means; they are intended to guide policy and action, usually involving considerable sums of public money. Adequate maps are required to show topographical details. Surveys in greater detail should secure the following information concerning the area under consideration: (1) *meteorological* conditions (by the month), rainfall, humidity, temperature, winds, etc.; (2) *topography*, soil, vegetation, drainage, water courses, ponds, lakes, springs, swamps, salt marshes, ditches, etc.; (3) *agricultural crops*, methods of farming, major crops, irrigation practices, rice culture, etc.; (4) additional specific epidemiological information with regard to malaria and other mosquito-borne infections, if present, such as sporozoite rate and oöcyst rate in dissected anopheline mosquitoes; numerical prevalence of different species of adult anophelines in houses, privies, pig pens, barns, etc.; spleen rate and parasite index in blood; malaria morbidity, incidence, etc.; presence of arthropod-borne encephalitides calls for additional technical details (see Chapter XII).

Essentials of mosquito abatement. Bearing in mind the fact that no mosquito ever came into existence without water in which its larval stages were completed, and that a very small quantity of water may serve the purpose, the control of all collections of suitable water available to mosquitoes is a matter of basic importance. The study of water management is highly important. The objective of mosquito abatement operations is the elimination of mosquito production. The abatement method must fit the offending species of mosquito. Marsh drainage or the usual larvicidal treatments would be ineffective in the control of tree-hole species such as *Aedes varipalpus* (Coq.) where specialized treatment is required. Even under temperate-zone conditions the work of mosquito abatement is a continuous year-round program, with special alertness in the spring to curb the first brood of larvae, and intensive larviciding through the summer. At the end of the breeding season efforts should be intensified to reduce the last brood as far as possible so as to have fewer overwintering mosquitoes to start the following year's brood. An intensive adulticiding program is in order. Use the winter months for maintenance work on drainage systems; for construction of new drains and permanent structures; and for planning the following year's work. Permanent corrective work should be the aim.

Water management. Anyone concerned with mosquito control operations soon learns that much mosquito breeding is due to mismanagement of water intended for useful purposes. Irrigation systems so essential to crop production in arid regions too often do not provide proper drainage, or the canals and ditches may be so situated or so constructed as to permit seepage resulting in ideal breeding places for potent malaria vectors such as *Anopheles freeborni* Aitken. Improper flooding of rice fields or uncontrolled flooding, as well as improper irrigation practices, in the production of alfalfa and ladino clover may be productive of pest mosquitoes. Irrigated pastures may be a source of many mosquitoes. Water impoundment may result in the production of important malaria vectors such as *Anopheles quadrimaculatus* Say in the middle and southern United States. The significance of such impounded water, so far as malaria is concerned, depends upon the presence or absence of a vector *Anopheles* adaptable to such breeding conditions. Mosquito production is due in large measure to man's carelessness and indifference, so well illustrated by the ubiquitious borrow pits formed by the removal of earth for highway and railroad embankments and fills; and by the drainage ditch improperly constructed with respect to slope or outlet (see King[11]).

Irrigation. Where irrigation is properly practiced with due attention to the economical use of water and good agricultural practice, there need be little or no mosquito breeding and consequent malaria menace. However, when seepage results from breaks in ditches and particularly from side-hill canals, ideal swampy areas are produced and cattle and

FIG. 82. Breaks in the irrigation ditch are responsible for considerable inundation, producing favorable breeding places for mosquitoes. The rapidly running water in the ditch is unfavorable for mosquitoes.

horses leave water-filled hoof prints, an ideal breeding situation for *Anopheles freeborni* Aitken in the western United States. Because of the breeding habits of this important western malaria-bearing mosquito, poorly constructed and improperly operated or leaking irrigation ditches commonly account for malaria in the neighborhood (Fig. 82).

Wilson[12] points out that "malarial effects are not attributable directly to the results of irrigation where it is economically and properly practiced—where care is taken to irrigate only land which has an open soil and such slopes and natural drainage as to prevent waterlogging, no unhealthy effects will result. . . . It is desirable, in order to mitigate the possible evil effects of irrigation, to keep the canal as much as possible within soil so that its surface level may be low, and thus only raise the sub-surface water plane to the least height practicable; that earth wanted to complete embankments be never taken from excavations or borrow-pits except where such localities admit readily of drainage."

It may be pointed out that there is a distinct difference between agricultural drainage and mosquito abatement drainage as applied to irrigation districts. Agricultural drainage is concerned merely with the problem of lowering the ground water level to a point where crops can be raised successfully. Frequently considerable quantities of mosquito-breeding water remain, often in the drains themselves (Fig. 83). Such

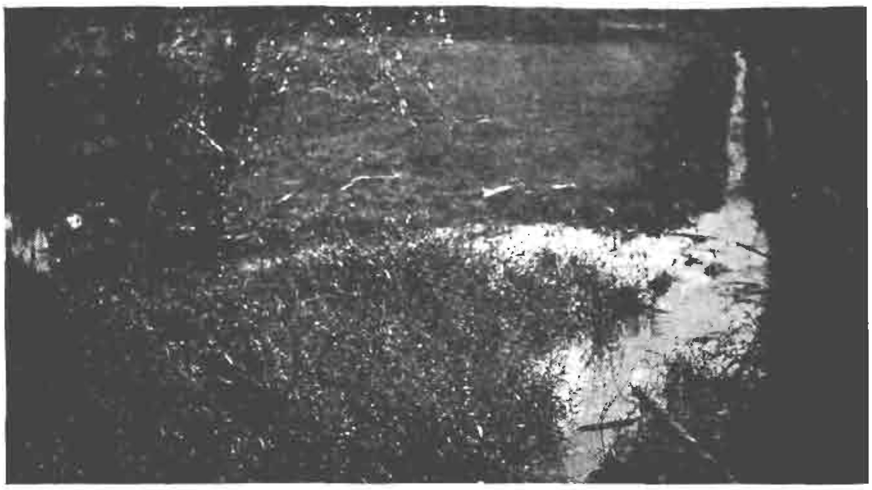


FIG. 83. Drainage water resulting from faulty irrigation, a source of numerous mosquitoes.

drainage consists usually of large deep main drains with comparatively few laterals. Mosquito abatement drainage, on the other hand, is a matter of more careful attention to detail, with great care to obtain uniform grades and smooth bottoms for the drains, so as to avoid mosquito breeding.

Impounded water. The storage of water in deep, clean, and steep-walled reservoirs seldom presents a mosquito problem; neither does storage of water in steep-sided reservoirs in rocky canyons. "The impoundage of flowing streams," Bishop, Hollis, *et. al.,*[13] (1947) point out, "profoundly alters the biology of the affected waters. One of the changes may be a vast increase in the potential for anopheline mosquito production unless appropriate control works and procedures are planned, designed, and operated. The choice is, therefore, between the probability of a disastrous 'man-made' increase in transmission of malaria or a successful resolution of the continuing conflict between adverse biological pressures and control practices." The magnitude of operations by the Tennessee Valley Authority begun in 1933, based on water management (control), is indicated in the fact that these involved 10,000 miles of reservoir shore line in a region of widely varying topography. The impounding of a lake in a region of flat topography within the Tennnessee Valley produces a wide variety of breeding places for *Anopheles quadrimaculatus* Say. There is here provided a huge food supply of microörganisms in the flotage and optimum protection against enemy fish and other aquatic predators. One of the important ways to control mosquito breeding in this situation is *water level* management. The effect of water level manipulations on anopheline larvae is both direct and indirect; direct in that as a result of drawing the water out of the marginal vegetation the larvae are exposed to an open and clean water surface wherein few can survive; and indirect in that the water is removed from the marginal plants, drift, and flotage which provide food and protection for the mosquito larvae. One of the important elements in water level management is periodic water level fluctuations. Complicated biological study and engineering features beyond the scope of this book are involved in accomplishing the desired results.

The author has seen mosquito breeding swamps in California made over into small recreational lakes by some dredging and impoundment of open water unsuitable for mosquitoes.

Controlled reflooding may be successfully employed in the control of salt-marsh *Aedes* mosquitoes. The eggs of salt-marsh *Aedes* do not all hatch on any one hatching; some eggs do not hatch until there have been a number of successive wettings. It is at least theoretically possible ultimately to hatch out all mosquito eggs in a given reclaimed marsh by purposefully flooding the marsh at the monthly high tide, holding the water on the marsh for several days until a brood of larvae appears, and then draining the marsh rapidly before the larvae develop into adult mosquitoes. The marsh is then allowed to remain unwatered until a subsequent monthly high tide when the flooding is repeated. As no adults have been permitted to emerge, no new eggs are deposited, and thus by successive wettings all eggs should be hatched eventually without additions of new eggs. Thus actually thousands of acres of salt marsh on Suisun Bay near San Francisco have been freed of *Aedes dorsalis* (Meig.), so that these marshes can now be flooded or dried out at the will of the owner without hatching hordes of mosquitoes. From 7 to 10 successive floodings and drainings were necessary to accomplish this.

Flushing. Several species of *Anopheles* mosquitoes, particularly *A. minimus flavirostris* (Lud.), the principal malaria vector of the Philippine Islands, breed in the running water of small upland streams, the larvae being able to hold fast against the current by means of small hooks; other anophelines breed in stream-bed pools. Flushing devices, either automatic or hand-operated, can be installed at intervals along the breeding length of such streams so as to release suddenly a relatively large quantity of water accumulated behind a dam. The mosquito larvae are swept downstream forcibly and are destroyed by stranding and crushing. (See Herms and Gray, *loc. cit.*)

Salinification. *Anopheles albimanus* Wied., the principal malaria vector of the West Indies, breeds prolifically in certain brackish coastal lagoons. Hurlbut[14] showed that where the salinity remains between 15 and 25 per cent sea-water breeding is abundant, while collections made concurrently where the salinity was 70 to 80 per cent were entirely negative. Breeding of this species can be eliminated by increasing the salinity to about 75 per cent sea water by making adequate sea connection, thus subjecting the marginal lagoons to tidal action.

Russell (*loc. cit.*) reports that Knipe and Hackett used tide gates in reverse position at Durazzo, in Albania, to transform a brackish swamp into a sea-water lagoon to inhibit the breeding of *Anopheles sacharovi* Favr.

Creeks and small streams. Except for flood water left behind during overflow from floods, great rivers themselves rarely afford opportunity for mosquito breeding. Flood water left behind when rivers recede may become a prolific breeding place for certain species of mosquitoes such as *Aedes vexans* (Meigen), and in some instances in appropriate regions a malaria hazard may be created in that a breeding place for *Anopheles quadrimaculatus* Say may result. In many parts of California, as creeks and smaller streams recede during the rainless summer, numerous sunny pools are left behind which soon become green with algae (e.g., *Spirogyra*) among which vast numbers of larvae of *Anopheles pseudopunctipennis franciscanus* McCracken occur; also quiet deeper pools occur along the banks shaded by wild grapes, willow, and other vegetation, in which *Anopheles punctipennis* (Say) find suitable breeding places. Pools along the banks can frequently be drained off or can at least be thoroughly larvicided.

Small streams or creeks that border communities or flow through the town frequently become clogged with rubbish and eventually become prolific mosquito breeders, and situations frequently result which are favorable for Anophelines. Communities should not permit rubbish to be thrown into stream beds. By neatly channelizing such stream beds and planting the banks with shrubbery, an eyesore and mosquito hazard may be rendered attractive and harmless.

Drainage. The removal or control of collections of water which produce mosquitoes presents a distinct problem in nearly every case. The type of drainage required in many instances will generally not require technical engineering skill. There are, however, drainage projects of considerable proportions for which engineering skill is essential; such skill is eminently necessary for large-scale salt-marsh drainage operations where land reclamation calls for dykes, drainage canals, pumps, tide gates, and the like. Unless the executive officer is himself an engineer, properly qualified engineering personnel must be employed to secure effective control.

In the case of a swamp caused by springs, a system of *deep* circumferential cut-off drains is recommended in order to intercept the seepage water and conduct it around the wet area. Where streams debouch from the hills onto a flat plain or valley, water from heavy rains, particularly in the spring, tends to spread out and leaves temporary pools which may produce mosquitoes. Usually these temporary pools can be more economically controlled by larviciding; but, in some cases at least, drains may be dug which lead back into the main stream at a lower elevation. These ditches will usually require considerable maintenance.

Mosquito breeding in rolling country is commonly due to artificial obstructions, particularly railroad or highway embankments with improperly placed culverts. Usually culverts are set too high so that pools or swampy areas are formed on the upper side of the embankment. Corrections are usually not speedily made if at all; hence heavy larviciding is a necessary remedy.

In some swampy situations because of small or negative gradients, drainage becomes very difficult or impossible. In such cases *sumpage ditches* or sumpage wells may be constructed, and the collected water may then be heavily larvicided. Surface water may also persist because of hardpan (impervious subsoil, etc.), in which case *vertical drainage* may be resorted to by digging sumpage wells, or by blasting.

Drain ditches. The purpose of laying out drain ditches is to secure effective and economical drainage. Although open drain ditches are most commonly used in mosquito control operations, subsurface drainage with the use of tile, or bundles of saplings laid in the ditches may at times be appropriate. Where the general slope of the ground is appreciable to the eye, this is usually a simple matter, i.e., following the low points to a place where the drainage water can be disposed of into some natural water course or other situation where there is sufficient fall to carry away the water. Laterals are then run from the main drain by the shortest distance to connect up with low spots or wet areas. The bottom of the main drain ditch must be kept deep enough so that the laterals can reach all the low spots in the area to be drained.

For most of the ordinary ditching for mosquito control transit and level are unnecessary. All that one needs are a few long stakes, 500 feet or more of stout chalk line or strong cotton cord, for line, and a hand level with a 10-foot board marked in feet and inches. If drains of considerable size and yardage are to be excavated, the usual surveying methods are employed, and the work should be done under contract with power machinery unless such equipment is owned or otherwise available to the district.

Hand labor, with pick and shovel, serves many purposes very well. As a rule the square-point long-handled shovel is to be preferred to the round-point shovel for ditching. Under some conditions long-handled spades may be satisfactory. Mattocks may be found useful in some soils.

In many cases, more or less dense vegetation has to be cut down and cleared before ditching can be performed with any speed or economy. Heavy grass or weeds may be cut with scythe, sickle, or machete. In open fields a horse-drawn hay mower may be economical. For brush, either axes, brush hooks, or machete may be used. The machete, however, is a dangerous tool in the hands of a man not accustomed to its use.

Dense grass and some forms of brush may be killed by applying stove distillates or Diesel oils, which are toxic to vegetation, or standard weed killers such as 2, 4 D. After killing and drying, the vegetation can be burned. Arsenical weed killers such as sodium arsenite, or an acid solution of arsenious chloride, may be used where there is no danger to cattle or other herbivorous animals. However, in any agricultural community the use of arsenical weed killers should be avoided in mosquito control operations.

Weed burners, capable of throwing a flame several feet, can be used for clearing dense vegetation, but the general experience seems to be that they are more expensive than other methods of clearing. If burning is to be done, due consideration must be given to the nesting season and nesting habits of wild life.

Ditching with dynamite. Swampy ground may often be economically ditched with dynamite. Special directions will have to be followed and trial shots will be necessary as a rule to determine the correct depth of holes, their distance apart, and the amount of dynamite per hole. The most satisfactory results are obtained by using 50 to 60 per cent straight dynamite, fired by self-propagating detonation.

Dynamite ditch construction is advantageous in that a ditch can be blown through land with stumps, boulders, etc., without first removing these obstructions; it is only necessary to place heavier loads at these points.

Maintenance of ditches. After the drainage ditch is constructed, it must be maintained in effective working order. Three main conditions making constant maintenance necessary are:

(a) Growth of vegetation,
(b) Caving or sloughing of banks,
(c) Artificial obstructions.

Under some conditions growth of vegetation in and adjacent to ditches is not a problem, but as a rule ditches will require clearing several times a year in order to keep them free from obstructing growths. In tropical or semitropical countries the problem of keeping drains free from vegetation is most difficult, and the source of considerable maintenance expense.

While weed killers may be helpful under some conditions, dependence must be placed in most cases on hand labor in cutting down and clearing out vegetation. The frequency of clearing will depend, of course, on local soil and climatic conditions, and it will be difficult as a rule to estimate in advance what the annual cost of maintenance of ditches will be.

Caving or sloughing of ditch banks is apt to occur in new ditches, for the first year or two. After that the banks usually become fairly stable, and but little further trouble is encountered, unless cattle are pastured along the ditches. In that case the animals may break down the ditch banks and cause some trouble.

Artificial obstructions are as a rule frequent only in the vicinity of public roads near a city or town. It is surprising how many people will haul refuse away from their homes out into the country and dump it, usually *in* a ditch close to a road and thus block the ditch completely or partially. The ditch thereupon becomes a mosquito breeder.

Frequent inspection of drainage ditches, say at least once every two or three weeks during the breeding season, should be carried on and all obstructions to flow, whether natural or artificial, removed promptly. During the winter all ditches should be gone over carefully and trimmed to grade, and the proper side slopes remade, so that all will be in first-class order at the beginning of each breeding season.

Salt-marsh drainage. Salt-marsh drainage requires special study and experience because of tidal action, soil conditions, differences in behavior of salt-marsh mosquitoes, and other factors. The rich reward in comfort and reclaimed land has given incentive to salt-marsh mosquito abatement. Strong public-spirited organizations have given marked support to this work on the Atlantic coast as well as on the Pacific, particularly in New Jersey and California.

These marshes include vast areas of tidal marshes affected by salt or brackish water along the shores of oceans and particularly the various bays, sounds, and estuaries. The effect of daily (diurnal) and spring tides resulting in fluctuations of water level is the principal feature distinguishing these from fresh-water marshes, although other characteristics, such as the salt-marsh vegetation, are of importance.

While salt marshes appear to be flat, there is a gradual slope between low tide level and the adjacent dry land. For practical purposes these marshes may be divided into two main areas, the area subjected to daily tidal action, where mosquito breeding seldom occurs, and the area between the elevation of mean high tidal water and the elevation of the extreme high tides. It is in the latter area that practically all the breeding of salt-marsh mosquitoes occurs.

Mosquito control operators concerned with projects involving salt marshes must acquaint themselves thoroughly with tidal phenomena. These vary in range and type in different parts of the world. The so-called "spring" tides, one or two of which occur each month, are the tides which fill pools along the upper portions of the marshes, and in these pools the principal salt-marsh mosquito breeding takes place.

The dates, times, and heights of the monthly highest tides are important in their bearing on the approximate time of emergence of a new crop of salt-marsh mosquitoes. It should be the invariable practice during the salt-marsh mosquito breeding season to inspect all known or suspected marsh breeding areas, beginning about two days after the highest tide and completing the inspection within six or seven days. The delay of two days is for the purpose of giving the larvae opportunity to develop to a sufficient size to be easily seen with the unaided eye. As under favorable conditions the time from egg hatching to emergence may be as short as eight days, it is obvious that if a flight is to be prevented, the inspection and necessary control measures must be completed before the time of emergence.

Because salt-marsh mosquito control involves the location of dikes and tide gates, the knowledge of the dates, times, and heights of both the lowest tides and the highest tides, particularly of "storm tides," the combined effect of "spring tides" and piling up of water on shore due to high winds and river flood waters as well, is important.

Marsh vegetation is often very dense and interferes with inspection and larviciding; hence burning is usually recommended. But it must be borne in mind that peat deposits commonly occur, and if the water level has been lowered as a result of drainage, which is usually practiced, dry peat may ignite and cause a peat fire. Peat fires can be extinguished only by flooding, which may be expensive. An additional hazard which may follow drainage is the shrinkage of the soil and the formation of "cracks." These cracks may be several feet in depth and may contain water in which mosquitoes will breed in abundance. Such cracks are difficult and expensive to larvicide though airplane application of DDT is helpful. An area of marsh that has cracked due to drainage operations should be plowed so as to break up the surface and fill in the cracks. An occasional disking after the initial plowing is recommended. For mosquito abatement purposes it is desirable to lower the water level only to the point where the water is drawn from breeding areas in dense vegetation but not to the extent that cracking of the soil occurs or a peat fire hazard is created. Drainage and burning must also be done with due regard for wild life conservation.

Types of salt-marsh drainage. There are two principal methods of marsh drainage, (1) open marsh drainage, and (2) reclamation. In open

FIG. 84. Salt-marsh drainage operations with drag line mounted on timber mats to prevent sinking in mud. The bucket is about to cut into a breeding pool which will be drained into ditch in foreground. (Photograph by H. F. Gray.)

marsh drainage the marsh is opened up by ditches to the free ebb and flow of tides so as to eliminate *standing* water suitable for mosquito breeding. In the reclamation type the area to be drained is surrounded on the low sides by a dyke which is pierced in one or more places by outlet structures, tide gates, which permit water behind the dyke to run out at low tides, but prevent the return flow at high tides. Suitable drainage ditches are dug to conduct water to the outlets (Fig. 84). The reclaimed marsh may be used for agricultural or industrial purposes.

Filling and pumping. In almost all mosquito abatement work low wet areas will be encountered which cannot be economically drained. Al-

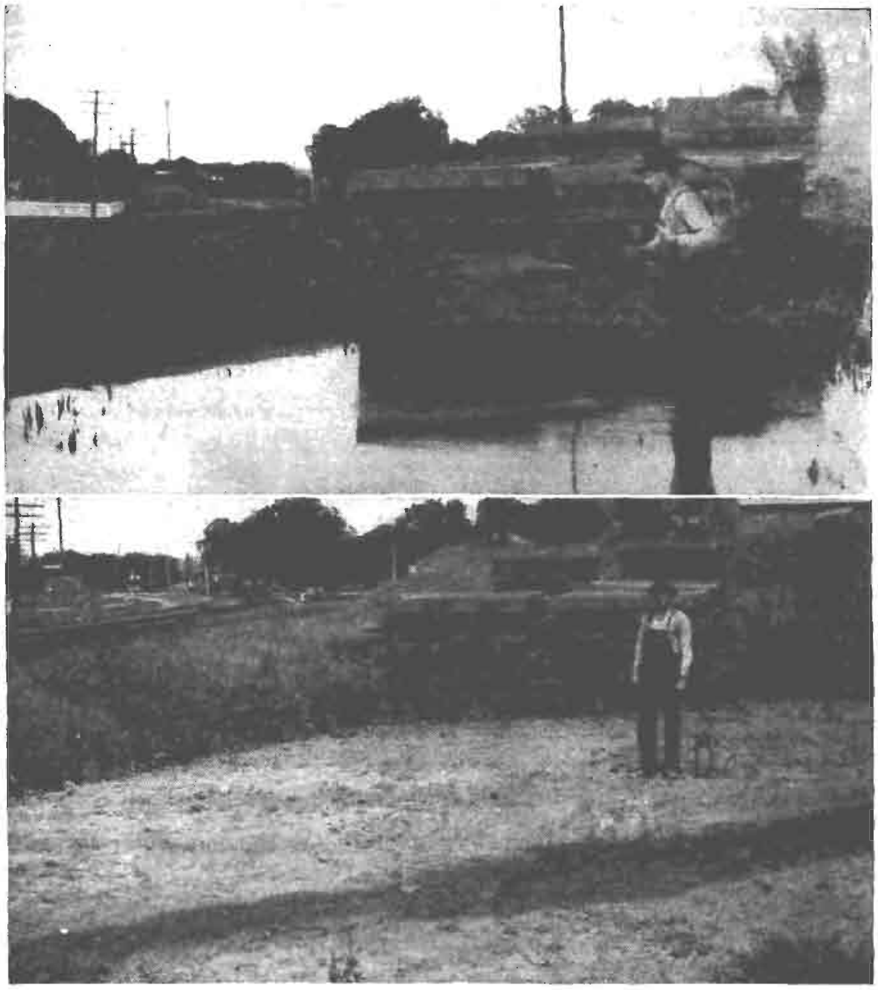


Fig. 85. Upper figure shows a pond adjacent to a railroad and caused by obstructing the natural drainage, a source of many mosquitoes every year. Larviciding, while serving the purpose of mosquito control, requires repeated expenditure of time, labor, and money. The lower figure shows the same spot after it had been permanently corrected by the railroad company.

though some such places may be ponded and the water stocked with top minnows, usually the most satisfactory method is filling (Fig. 85). Most smaller holes, such as borrow pits, can be filled in by hand shovel-ing; larger holes may be filled by means of power scrapers. If sanitarily handled, municipal garbage and refuse may be used in a "fill and cover" method. Such fills are covered with earth so as to prevent fly breeding and to some extent rat breeding.

Salt marshes may be filled by hydraulic dredges, which suck mud and sand from the bottom of an adjacent bay and pump the mud and water mixture through a pipe and discharge it on the marsh. Where harbor or channel improvements are being made by hydraulic dredging, very satisfactory arrangements may often be made to use the mud and sand to fill a near-by mosquito-breeding marsh.

FIG. 86. Rice field with roadside seepage ditch in which mosquitoes breed.

When the general land level is at or below the low-water level of an adjacent river or bay, pumping has to be resorted to. Portable pumping units are frequently of much value.

Mosquito breeding in rice fields. The control of mosquito breeding in rice fields is obviously a matter that must have the attention of those concerned with rice culture (agronomists) as well as the mosquito control experts. Much of the breeding can be controlled by proper management with respect to the habits of mosquitoes. Many species of mosquitoes are involved, some of which are malaria vectors. In the extensive rice-growing areas of Arkansas, 98 to 99 per cent of the population of noxious mosquitoes consist of four species, *Psorophora confinnis* (L.-A.) and *P. discolor* (Coq.) both flood-water breeders, constituting 83 to 94 per cent, and *Culex erraticus* D. and K. and *Anopheles quad-*

rimaculatus Say, a malaria vector constituting the rest (Horsfall[15]). In some newly developed areas the introduction of rice culture on a large scale is characterized by hasty, haphazard methods (quick returns at minimum cost), with utter disregard of sound agronomic practice. Poorly constructed irrigation systems and even poorer drainage methods result in a veritable bog adjacent to rice fields (Fig. 86). Too often as much as half of the mosquito production is due to poor practices and could be largely controlled by improved methods of water management. Growing rice in very porous soil requires much replenishment of water, and the continous freshening by replenishment may in certain areas invite abundant breeding of malaria vectors, e.g., *Anopheles freeborni* (Aitken) in California. Where rice growing is an old established practice great care is normally exercised in the matter of water management, particularly so in the vicinity of communities. People must learn to live in such an environment, and it is folly to endure mosquitoes which may be produced by rice fields.

The use of larvicides in rice fields is fraught with danger in that the rice plants may be damaged. Rao and Sweet[16] found that the use of a one per cent dilution of Paris green in road dust and wood ash produced no ill effects on the rice or in straw yield. There was no indication that dusting should be stopped during the period in which the paddy was in flower.

Several authors have recommended intermittent irrigation, e.g., Hill and Cambournac[17] in Portugal, and Russell, Knipe, and Rao[18] in South India. The latter recommend a cycle of five wet days and two dry days, particularly for the control of the vector, *Anopheles culicifacies* Giles.

In 1948 Cambournac and Fonseca,[19] reporting on anopheline and malaria control in the rice-growing regions of Portugal, point out that the control of anopheline larvae in rice fields by the use of DDT emulsions is both economical and practical. They recommend 5 liters of a one per cent emulsion per hectare, i.e., about 5.28 quarts to about 2.5 acres, poured in the irrigation ditches and thus automatically dispersed in the plots.

In California experimental work is in progress in the rice-growing area of the Sutter-Yuba Mosquito Abatement District in which the rice seed is treated with DDT (wettable powder which clings to the seed in the hopper) and is then seeded by airplane. In this same district studies were made during September, 1947, using DDT applied in airplane thermal exhaust aerosols for the control of *Anopheles* larvae. According to State Board of Health reports, better than 95 per cent mortality of *Anopheles freeborni* was obtained with as little as one pint of DDT solution per acre with a dosage of 0.025 pounds of DDT per acre. The cost of treating 18,000 acres of rice fields was 30 cents per acre;

this sum included cost of hiring the airplane pilot, ground crew wages, DDT, and solvents.

Anopheline control in Bromeliads. *Anopheles bellator* Dyar and Knab is regarded as the most important vector of malaria in Trinidad (West Indies), where it breeds in the rain water collected among the leaves of epiphytic Bromeliads, particularly the large so-called "tank plants," *Gravisia aquilega* and *Hohenbergia stellata,* and others. These plants are found in great profusion on immortelle trees which are planted to shade cacao; hence the problem of anopheline control is bound up with the cacao industry. Manual removal of the bromeliads is costly and has limited value. Gillette[20] reports that the spray killing of bromeliads by the mechanical application of copper sulfate is the method of choice. He states that one application of 0.25 per cent to 0.5 per cent solution may suffice for a 10-year period. The instance is cited of the treatment of about 1,500 acres in a district of 30,000 population has resulted in a spleen rate reduction from 28 per cent in 1945 to 5 per cent in 1947.

Sewer inlets and catch basins. In the newer types of street inlets little opportunity is afforded for water to collect and remain standing for mosquito breeding. Most of the old types of inlets and catch basins, especially those connecting with a combined sewer (for domestic sewage and storm water), are apt to produce mosquitoes, particularly *Culex pipiens* Linn. Larviciding is most economically done by means of a motorcycle sidecar (Fig. 87). One filling with 25 gallons of larvicide with air pressure to 50 pounds per square inch, will suffice for a day's work, i.e., from 200 to 300 catch basins.

Public utilities street vaults. In practically all urban areas the various public utilities, such as telephone, power and light, gas, electric railway, telegraph, and water, have numerous vaults in streets which are frequently the source of a severe local infestation of *Culex pipiens* Linn. during the breeding season. Heavy residual spraying with DDT will give good control.

Cesspools, privies, liquid manure pits. Where pit privies are built in wet areas, water collects and prolific mosquito breeding (*Culex quinquefasciatus* Say, *Culex pipiens* Linn., and *C. tarsalis* Coq.) may result. The use of DDT or cresylic acid larvicide is recommended. Where liquid wastes are disposed of in leaching cesspools, mosquito production may be very great if mosquitoes have access to them. Even small knot holes or vent pipes afford a ready means of entrance. If necessary repairs are made so the egress is cut off, the larvicide need not be applied; otherwise treatment at appropriate intervals of about 10 days must be practiced.

Liquid manure pits in connection with greenhouses and plant nurseries commonly produce prodigious numbers of *Culex pipiens* Linn.

and *Culex tarsalis* Coq. Larvicides must be used which are not dangerous to plants; DDT and several other newer insecticides may be employed.

Tree holes. In wooded areas and on estates tree-hole mosquitoes, such as *Aedes varipalpus* (Coq.), often cause great annoyance. Tree holes, particularly in oaks, collect water in which mosquito larvae thrive.

FIG. 87. Motorcycle sidecar equipped for larviciding. (Photograph by H.] Gray.)

Liberal application of DDT or cresylic acid larvicide is recommende(the larvicide should be applied not only to the water but also liberal] swabbed on the wood above the water line to repel egg-laying fema mosquitoes. The practice of tree surgery on estates has greatly reduce mosquito breeding in tree holes. Heavy applications of DDT aeros have been effective in canyons or narrow valleys.

Cemetery urns. Urns and other types of receptacles intended f(flowers on graves may stand partly filled with water for considerable lengths of time without changing and may furnish breeding places for many species of mosquitoes. Thus a mosquito survey of the cemeteries within the boundaries of the Alameda County (California) Mosquito Abatement District showed that 11 species were breeding in such receptacles, namely, *Culex pipiens* Linn. about 70 per cent, *Culiseta incidens* Thom. about 25 per cent, *Anopheles pseudopunctipennis franciscanus* McCracken and *A. punctipennis* (Say) about 5 per cent. *Culex tarsalis* Coq. and *C. stigmatosoma* Dyar larvae were only rarely found. Of the 18 cemeteries the largest contained 80,000 graves with some 27,000 containers for flowers. During the summer season at least half of these containers were breeding mosquitoes; in January, 20 per cent still contained larvae. Excellent control was effected by the use of DDT aerosol, 5 per cent emulsion in Diesel oil, applied at about three-week intervals. Each treatment (made before 7 a.m.) required only about one hour, whereas previously over 200 man-hours were required for hand treatment (Aarons,[21] 1948).

FIG. 88. Tin cans, tubs, and barrels in which water may stand, furnishing breeding places for mosquitoes.

Sundry nuisances. Water-holding receptacles of many kinds may prove suitable for mosquito breeding and must not be overlooked by the "mosquito man." However, it sometimes happens that an overemphasis of the tin can results in attracting the attention away from more important matters, such as dripping hydrants, stagnant ditches, etc. Indeed the water in tin cans unless in shady situations usually becomes too hot for mosquitoes during most of the summer. (Fig. 88.) Heaps of broken gourds commonly reek with mosquito larvae; tubs and barrels of water frequently produce many of these pests, even certain species of anophelines. Stagnant water in poorly constructed street gutters is often a serious menace. Dripping faucets (Fig. 89) may result in pools of water suitable for mosquito breeding.

Routine inspections for mosquito breeding. Inspections for the purpose of finding breeding places in organized mosquito abatement districts are made either upon the receipt of complaints or for routine purposes. The inspector should be guided by the nature of the complaint: (1) mosquitoes annoying at night or affecting sleep, and (2) annoying during the daytime or toward evening, while working in the garden or watering the lawn. In the first instance it is probably a domestic fresh water species, breeding on the premises or in the immediate vicinity. In the second instance it is probably a salt-marsh species, if salt marsh is near, or it may be a flood-water or tree-hole *Aedes*. In a previous chapter (p. 181) exceptions to this rule are discussed, particularly the spring dispersal flights of some species of *Anopheles*. Obviously the inspector must have a thorough knowledge of the species of mosquitoes and be well informed concerning breeding habits. When complaints are received, the inspector should visit the premises and if possible capture mosquitoes

FIG. 89. The dripping faucet with the resulting pool of water is often a constant menace in the entire absence of other mosquito-breeding sources.

for positive identification, so as to simplify inspection for larvae and to ensure effective abatement.

In searching for adult mosquitoes for species determination it is important to remember that few species are active during the day; hence one must search for them in dark, cool and moist places, under houses, in basements and cellars, behind pictures, in closets and dark corners. A flashlight is very useful in these operations. *Aedes dorsalis* (Meigen) and other day fliers often hide in shrubbery and may be found by shaking bushes and vines or kicking weeds. The mosquitoes may be collected in small cyanide bottles or by means of a sucking tube with an extension rubber tube.

Inspections, whether due to complaint or as a matter of routine, must be thoroughly and intelligently carried out; the breeding may be occurring in a rain barrel, a lily pond, or a concealed cesspool; it may be taking place in a concealed chamber fed by a natural spring; in an abandoned well, a broken or clogged sewer or drain; floor boards may have to be removed.

The work of routine or house-to-house inspection must be properly organized for efficient and economical coverage. The inspectors must be intelligent, well trained technically, and capable of meeting all sorts of people.

In inspecting large tracts of marsh to locate the precise producing areas, it is always advisable to mark off the marsh into definite sections, which can be examined one at a time, so that no portion is overlooked. Breeding areas, when located, are marked by setting up stakes in the center of the breeding area for the crew of larviciders which follow, or for future reinspection. Frequently both inspection and larviciding may be done concurrently by the same man.

Larvicides. All too commonly mosquito control is thought of as the practice of spraying oil (larvicide) on the surface of stagnant water. Larvicides have a useful place in mosquito abatement operations; however, all properly conducted operations should look upon larviciding as secondary to the methods set forth previously in this chapter. The correct use of larvicides should be observed at all times. The older larviciding methods such as oiling[22] and dusting with Paris green[23] have been replaced by methods using such materials as DDT, TDE, BHC, or new formulations.

Oil (petroleum) which is to be used on water to destroy the aquatic stages of mosquitoes must, of course, be lethal, at least in time, to all larvae and pupae; it must spread well on the surface of the water and must have lasting qualities. Mixtures suitable for larvicidal purposes should have a specific gravity of 31° to 39° Baumé (API) and a viscosity of 45 or lower Saybolt at 100° F; they must be economical, easily obtainable, not unpleasant to handle, and if possible nontoxic to animals.

In ordering petroleum oil for mosquito abatement purposes the following specifications should be designated: (1) it must be homogeneous; (2) it must not separate into fractions on prolonged standing; (3) it must be free from granular and fibrous material which might cause the clogging of spray nozzles; (4) other physical characteristics must lie within the following maxima and minima, specific gravity at 60° F not less than 0.830 nor more than 0.870; viscosity (Saybolt universal) at 100° F not less than 30 nor more than 45; boiling range from 330° F to 800° F.

Cresylic acid (boiling fraction 225°–250° C) must be emulsified with soap. The original "Panama" larvicide was made with a rosin soap as the emulsifying agent—200 pounds of powdered rosin with 30 pounds of caustic soda added to 150 gallons of crude carbolic acid, heated and stirred. Whaleoil soap or other cheap soaps may be used as emulsifiers. The preparation of this larvicide is best done by manufacturing chemists who have proper equipment. The larvicide is applied in the proportions of 1 part to 30,000 parts of water.

Pyrethrum extract is very toxic to mosquitoes in the larval as well as adult stages. Vannote and Ginsburg[24] prepared an emulsion of kerosene extract (extracted from 1 pound of pyrethrum flowers per gallon of petroleum solvent) emulsified with water and soap (66 per cent kerosene extract with 34 per cent water containing 3 per cent to 5 per cent of soap). This stock emulsion was diluted 1 to 10 with water for the field spray. Using in excess of 50 gallons of the dilution per acre Ginsburg obtained a complete kill of larvae on salt marsh. Formulations made of pyrethrins, which give a rapid knockdown, and insecticides such as DDT with long residual effect and heavy kill are desirable.

Paris green can be successfully used against the surface-feeding anopheline larvae. This material is suitable for airplane dusting on a large scale as well as for application with hand dusters. The Paris green is diluted with road dust, hydrated lime, finely powdered charcoal, finely powdered talc or other inert finely powdered material. It apparently acts as a stomach poison, the small particles of Paris green being ingested with larval food. The poison is usually mixed with the diluent in the ratio of 1 part of Paris green to 99 parts of diluent (by weight). For airplane dusting a higher percentage of Paris green is recommended, namely from 25 per cent to 33 per cent. The amount of Paris green per acre of marsh varies according to various authors from slightly less than one pound to two and even four pounds where vegetation is dense.

Concerning the dangers from the use of this poison Barber and Hayne[23] state:

> In our experiments we have used no precautions other than to stand to the windward of the dust cloud—the place where one would naturally stand in distributing dust—and we have experienced no harmful results whatever. However, even with the small quantities used in larvicide work, it is well to remember that one is working with a poison and that some precautions should be taken at least until the matter is further investigated. It is probably sufficient to keep to the windward of the dust clouds and to avoid inhaling the dust as far as possible. In case a great deal of exposure is necessary, one should use some precaution to keep any large amount of Paris green from entering the clothing or accumulating on the skin. The danger to domestic animals through drinking treated water seems very remote. . . . We have never observed any effect of the poison on culicine larvae or on any aquatic insect or animal, however delicate, other than the surface feeding anopheline larvae. In particular, we have not observed any indication of harm to top-feeding minnows or to any other natural enemy of larvae.

Dichloro-diphenyl-trichloroethane (DDT) has been rigidly tested by many mosquito control agencies in large-scale field operations in practically all parts of the world. Both as a larvicide and adulticide it has been proved a superior culicide. Its use is not "foolproof"; it is a poisonous chemical, and care must be exercised when using it in the presence of food and food containers in the home and in barns and stables. Furthermore, since DDT, a white crystalline substance, is practically insoluble in water, solvents are employed in the preparation of DDT sprays. The solvents are inflammable and explosive; hence when using such sprays indoors or in enclosed places without emulsifying in water reasonable caution must be exercised. Some of the solvents are toxic to man.

Emulsions. An emulsion concentrate may be made of 25 per cent DDT, 10 per cent Triton X-100, and 65 per cent xylene or other solvent. Concentrates are available commercially. Emulsions are made by adding the required amount of concentrate slowly to water with continuous stirring. To prepare a 5 per cent emulsion of DDT, for example, one volume of the concentrate is mixed with four volumes of water. When used in the field as a larvicide the emulsions should be applied at the rate of 0.1 to 0.2 pound of DDT per acre of water surface. Since DDT is not a strong pupacide, treatments must be made with this in mind or formulations must be employed which include pupacides.

DDT-oil solutions. Diesel oil, kerosene, and fuel oil will dissolve DDT in about 24 hours. To make a 5 per cent solution add about 2 pounds (2⅛ pounds for a true 5 per cent weight-per-volume solution) of technical DDT to each five gallons of oil. Used as a larvicide, control can be obtained with 5 quarts per acre of a 1 per cent DDT-oil solution. Oil without DDT for similar control would require from 15 to 25 gallons per acre. Using a 5 per cent DDT-oil solution as little as 1 to 2 quarts per acre will be effective when applied with pressure spray equipment.

The use of a DDT-oil solution is well illustrated in the control of *Anopheles farauti* Lav., vector of both malaria and filariasis in the New Hebrides Islands, as reported by Yust.[25] This vector bred largely during military operations in road ruts created by military vehicles. Native huts treated with 5 per cent DDT in kerosene at the rate of one gallon per 1,000 square feet were free of *A. farauti* adults for three months. Breeding sites were sprayed weekly with a 5 per cent solution of DDT in No. 2 Diesel oil at the rate of about two quarts per acre. Weekly spraying was necessary because of the formation of new water pools.

DDT dust larvicide is employed in the same way and for the same purpose as Paris green; however, used against the larvae of *Anopheles quadrimaculatus* Say it is said to be about 25 times as toxic. It is applied as a 10 per cent DDT dust in pyrophyllite, talc, or other inert dust at the rate of 0.1 to 0.2 pounds DDT per acre, i.e., one pound dust mixture. Where there is abundant vegetation the dosage must be increased. For the control of *Culex* and *Aedes* species a larger dosage is also required.

Dichloro-diphenyl-dichloroethane (TDE, also referred to as DDD) is an analog of DDT and in comparative tests made with fourth instar mosquito larvae by the author and many other investigators it is at least equal to DDT as a larvicide, except in lasting residual properties. The more rapid dissipation may have its advantages in that there is less accumulation in the soil or water after repeated treatments. TDE is also less toxic to fish and to warm-blooded animals as well.

Benzene hexachloride ($C_6H_6Cl_6$, also known as 666) and *chlordane* ($C_{10}H_6Cl_8$) both have larvicidal as well as pupacidal properties which deserve much more research, particularly with regard to formulations.

Larvicide drips. During the years when petroleum oil was the accepted larvicide, so-called "oil drips" were used to good advantage in giving continuous drop-by-drop treatment to ditches and small streams of moving water. This scheme is now followed with some modifications in a similar use of DDT. Oil drums or other containers properly equipped with a faucet permitting adjustment for automatic exit of small quantities of fluid are placed at the head gate or point where irrigation water enters the pasture or other large irrigated areas where there is mosquito breeding. Emulsible DDT is used and the drip is regulated according to the quantity of water to be treated. Larvicide drip dispensers must be constantly serviced to prevent clogging and mechanical difficulties.

Aerosols. Ginsburg[26] points out that the term "aerosol" is derived from the word *sol* used in chemistry to denote colloidal dispersions of solid particles in liquid, gaseous, or solid media; thus if the dispersion medium is water the system is called *hydrosol,* if air it is called *aerosol,* etc. The term *aerosol* has recently come into general use to specify certain insecticidal sprays, dusts, and fumes applied against mosquitoes and other insects. Aerosols include a variety of natural and artificial systems such as smoke, clouds, mists, fog, fumes, atomized oil in oil burners, smoke screens, etc. in which air constitutes the continuous *external* dispersion medium and the fine particles form the *internal* or dispersed phase.

The main object of aerosol spraying is to disperse insecticides, such as mosquito larvicides and adulticides, etc., in such a fine state of subdivision and in such a way that they remain suspended or floative in the air for long periods of time, thereby prolonging their effectiveness. The work of Ginsburg[27] in the development of pyrethrum aerosols for use out of doors is well known. DDT aerosols are now widely used both as larvicides and adulticides. Airplane thermal exhaust aerosols are commonly employed in rice-field and other large-scale mosquito control operations. Numerous types of equipment are available for producing aerosols, among them the engine exhaust of a "jeep," which is inexpensive and effective (Fig. 90).

Residual treatment. *Duration of toxicity of residual DDT.* Using a stock solution of 35 per cent technical grade DDT in xylene, with Triton X-100 as an emulsifying agent, Weathersbee, Arnold, and Hop-

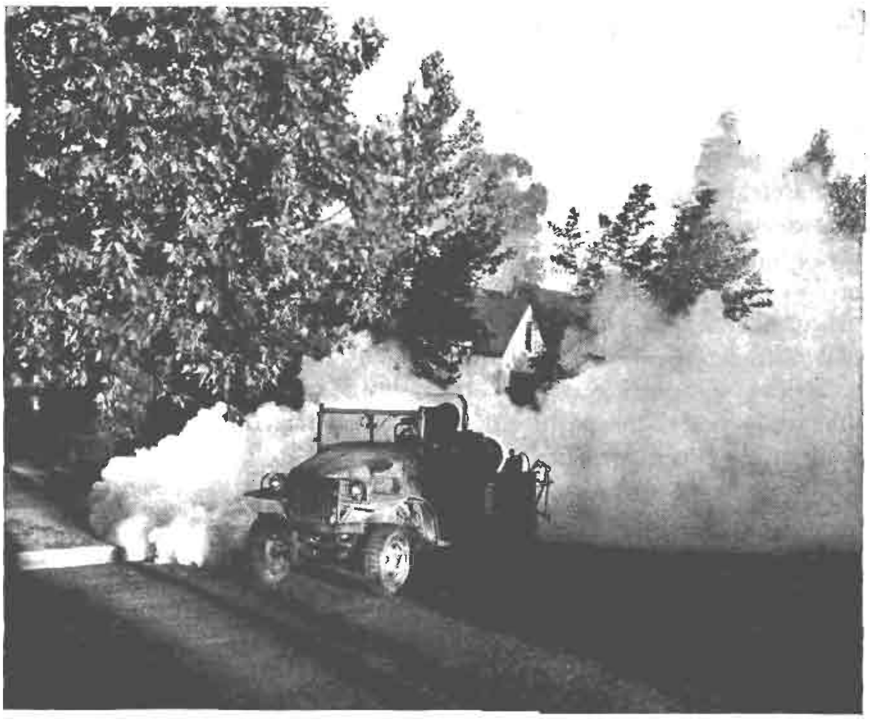


FIG. 90. Aerosol equipment in adulticiding operations. (Photograph by E. A. Smith.)

kins,[28] 1948, prepared water emulsions of 1, 2, 3, 4, and 5 per cent and applied each as a residual spray in five field tests on surfaces in stables, a farmhouse, and doghouses, where *Anopheles quadrimaculatus* was by far the predominant species of mosquito, as shown by preliminary observations. The one per cent concentration kept the sprayed surface free of mosquitoes for 27 days; the 2 per cent for 35 days; the 3 per cent for 62 days; the 4 per cent for 101 days; and the 5 per cent for 135 days. The duration, therefore, of toxicity to *A. quadrimaculatus* of DDT emulsion applied as a residual spray resulting in a residue varying from 40 to 200 mg per square foot is proportionate to the concentration used.

The authors concluded that concentrations of 4 per cent and 5 per cent (160 to 200 mg per square foot) DDT appear to be more economical and more efficient than lesser concentrations.

Indoor residual spraying to destroy adult mosquitoes is accomplished by treating all the wall surfaces and ceilings, undersurfaces of tables, beds, etc. with a 5 per cent DDT-oil emulsion spray using either a hand or air-pressure sprayer. The object is to deposit a residuum of about 200 mg of DDT per square foot. One thorough treatment may last in excess of three months. Rooms should be ventilated during spraying, and DDT should not be allowed to fall on food, dishes, or cooking utensils.

FIG. 91. Knapsack spray pump for mosquito control operations.

The residual effectiveness of DDT (190 mg of 5 per cent DDT emulsion per sq ft) applied indoors to adobe walls and straw-thatched roofs under subtropical conditions is shown in a report by Downs *et al.*,[29] who noted residual action 29 *months* after application.

Gray[30] points out that this residual effect of DDT and its analogs gives us not only a completely new technique in mosquito control, but an entirely new concept, particularly in the control of disease vectors.

Application of larvicides. Methods of applying larvicides will, of course, depend upon the materials used, i.e., whether liquids or dusts, and whether large areas are involved or only a few catch basins. For relatively small areas or for numerous small and widely separated areas, hand application is both economical and convenient (Fig. 91).

Hand spraying equipment such as the knapsack spray pump has had almost universal use. This type of equipment partly atomizes the larvicide, resulting in a better spread and a better coverage of the water surface and is at the same time more economical in the use of larvicide and in labor. Knapsack sprayers consist of closed metal cylinders of three to five gallons' capacity, fitted with a brass plunger pump which forces air into the container until a strong air pressure is built up. To the outlet pipe, which extends to the bottom of the container, is attached a piece of flexible hose and a spray nozzle. The spray nozzle is usually attached to the end of a one-quarter-inch metal pipe (Fig. 92). In

FIG. 92. Knapsack spray pump in use in mosquito control.

operation the tank is filled about three-quarters full with larvicide. A hand lever extending over one shoulder when the sprayer is strapped on the back operates the pump plunger. The container should be provided with a lip about one and one-half inches high extending around and above the tip of the can to prevent the larvicide from spilling down the back of the operator. One man with a knapsack sprayer can spray about five acres per day of eight hours under open field conditions. Power equipment has largely replaced the knapsack sprayer.

Paris green dusts may be broadcast by hand or by means of a mechanical blower, preferably the rotary type. In either case the dusting should be done with the wind so that the dust floats away from the workman, thus avoiding exposure to the arsenic which may cause a dermatitis. Where anopheline marshes of great area are to be treated, airplane dusting is to be preferred.

Oiled sawdust may be employed to good advantage. The practical use of this material first came to the notice of the writer (Herms, *loc. cit.*) while he was on military duty in 1918. A small detachment of troops was camped near an abandoned sawmill, and a huge hill of sawdust was available for filling numerous small anopheline breeding pools in the vicinity. It was observed that oil-soaked sawdust gave off a thin film of oil for many days. Thereafter liberal use of oil-soaked sawdust was made whenever it was available. For small-scale projects sawdust was moistened with fuel oil and broadcast by hand. A small winding creek much overgrown with shrubbery and weeds, with steep banks and many pools, can be successfully treated in this manner.

Power equipment to apply culicides. Enormous strides have been made in the development of power equipment designed to give efficient distribution of the newer insecticides in field practice. Many problems have had to be met. It has been pointed out that in order to capitalize on the increased toxicity and the heretofore unheard-of residual properties of these products, a much finer break-up of the insecticides is demanded as well as a more widespread and uniform distribution. There is new equipment for producing and distributing aerosols, sprays, and dusts. Much ingenuity has been employed in adapting various types of wartime vehicles such as "jeeps" and "weasels" to mosquito control operations.

Aircraft in mosquito control. The use of aircraft in the distribution of insecticides for mosquito control became almost world-wide during World War II. Dichloro-diphenyl-trichloroethane (DDT) in various formulations was the insecticide of choice in these operations. The American Mosquito Control Association has recently (1948) issued a valuable illustrated brochure[31] which presents practical information on when, where, and how aircraft may be used to distribute mosquito insecticides, chiefly DDT, and on appropriate methods for appraising results. All agencies using aircraft for mosquito control purposes should have this brochure available to the personnel concerned.

Historically the airplane in this connection was first used in Canada to survey areas in which mosquitoes were breeding (Hewitt,[32] 1919). The Alameda County (California) Mosquito Abatement District early in its formation (1930) used aerial photographs to map its salt-marsh mosquito breeding areas extending for some 25 miles along the shores of San Francisco Bay. These aerial photographic maps were made on a scale of 800 feet to the inch (Herms and Gray, *loc. cit.*). King and Bradley[33] (1925) and Williams and Cook[34] (1927) describe the earliest use of the airplane in dusting operations against malaria-bearing mosquitoes, the former near Lake City, Florida, and the latter in the vicinity of Quantico, Virginia. Although the airplane had been used to apply spray as early as 1930 (Ginsburg[35]), it was not until 1944–1945, when DDT became available, that spraying by airplane came into wide use (Fig. 93).

Much of the airplane work done in mosquito abatement districts is with military trainer biplanes which carry from 80 to 90 gallons of liquid insecticide or 650 to 750 pounds of dust. The helicopter is growing in favor because it can be used at any time of day, as it provides a downward thrust to the insecticide; the wing type plane is used principally in the early morning when the cooling air carries the insecticide down.

The dusting equipment consists of a hopper, a wind-driven agitator, feed control gate, and venturi spreader. Spray equipment consists of a

Fig. 93. Use of airplane in spraying operations. (Courtesy of Kern County, California, Mosquito Abatement District.)

tank, liquid distributing lines (gravity or pressure), and a device for discharging the liquid into the air. Various types of nozzles are used. Effective dispersal of sprays has been accomplished by introducing the liquid spray into the exhaust stream of the engine with resulting atomization. Insecticide formulations must be carefully selected on the basis of the intended purpose.

The use of aircraft makes it economically possible to treat extensive breeding areas and wild terrain; rice fields may be readily and rapidly treated, as may also extensive flooded areas caused by river overflow, etc. The airplane is particularly useful in large-scale "prehatching" treatments, such as the application of an appropriate insecticide, e.g. DDT-fuel oil spray, to breeding areas while they are covered with snow, which on melting in the spring automatically treats all pools harboring newly hatched boreal mosquito larvae. Preirrigation treatment of irrigated pastures may also be carried out by airplane, using water soluble DDT.

Airplanes are widely used in California in mosquito control operations. Reporting on this type of work at the Sixteenth Annual Conference of the California Mosquito Control Association (1948) Geib, superintendent of the Kern Mosquito Abatement District, stated that *spraying* 3 to 4 quarts of a 5 per cent aqueous emulsion of DDT at rates of .3 to .4 pounds of DDT per acre gave satisfactory control of *Aedes* larvae on pasture land and river bottom. Using Stearman PT-17 biplanes owned and operated by the district the average cost per acre during the 1947 season was 66 cents.

Repellents. There is little doubt that through the centuries man has tried to protect himself from mosquito bites by using herbs and smoke smudges, probably with little success. Oriental punk has long been widely employed; considerable quantities were used in the tropics by the armed forces of both sides during World War II. During World War I troops who were forced to operate in mosquito-infested regions were often advised to smear themselves with so-called "bamber oil" (among other ointments), a mixture of citronella oil, kerosene, and coconut oil, with a little carbolic acid added. Relief was probably not marked. Early during the World War II period steps were taken to develop repellents with some real effect. Granette[36] at Rutgers University (New Jersey) and Knipling and Dove[37] at the United States Bureau of Entomology and Plant Quarantine, Orlando, Florida, laboratories, in cooperation with the armed forces and other agencies (some commercial), developed the widely used repellents known as Rutgers 612, dimethyl phthalate, and indalone. Rutgers 612 is most effective against *Aedes* species, while dimethyl phthalate is most effective against *Anopheles quadrimaculatus.* Granette in a personal communication advised the author that effectiveness of repellents varies considerably with the various anopheline species. Indalone is an effective repellent against flies such as *Stomoxys.* All of these repellents are more or less effective for three to five hours and they remain effective for a much longer time (about a week) when sprayed liberally (50 to 100 cc) on clothing.

Screens to exclude mosquitoes. Where there is no control of mosquitoes or only incomplete control, particularly in the presence of malaria-bearing species, sleeping quarters, living quarters, porches, etc., should be adequately screened against intruding mosquitoes. The best size mesh for all purposes is No. 18, i.e., 18 strands to an inch. The screens must be suited to exposure and to the climatic conditions of the situation where they are used. They must be protected or reinforced against mechanical breakage at points of stress. Painting screens with a 5 per cent oil emulsion of DDT is a good practice.

Duck clubs. In most states the duck-shooting season does not begin until November first or later. By that time cold weather usually stops mosquito breeding. However, many duck clubs start flooding their duck ponds long before this, perhaps to have the ponds ready to attract the earlier migrants. These slowly filled shallow ponds may prove to be a mosquito menace. Some clubs keep their ponds well flooded throughout the year, and if the banks are steep and top minnows have access to all parts of the pond, there is no mosquito problem. The ponds which are most difficult to handle are those that are drained off in January or February after the duck season is over, and are allowed to remain dry during the summer, being again flooded in late summer or early autumn while the weather is still warm. Breeding is sure to occur. *Aedes* eggs, such as those of *Aedes dorsalis* (Meig.), from preceding years promptly hatch, and a plague of mosquitoes soon appears.

Neff (1935), of the United States Bureau of Biological Survey, states in the Proceedings of the Sixth Annual Meeting of Mosquito Abatement Officials in California, p. 16, "Impounding of water, or the maintenance of a constant water level by means of tide gates that permit an equalized flow of water through the salt marshes, seems to be the method of control which has aroused little or no adverse comment from biologists. This method also offers opportunity for experimental work on planting of better wild life food plants than may formerly have existed on the areas with fluctuating water levels."

Although expensive, pyrethrum should be used instead of oil where a larvicide becomes necessary. Neff (*loc. cit.*) states that pyrethrum larvicide has "no known deleterious effect upon wild life."

The following rules in dealing with mosquito control in connection with duck clubs are suggested:

1. Continuous all-year flooding of ponds is permissible and approved, provided the ponds are stocked with "mosquito fish" at all times.
2. Intermittent maintenance of ponds is permissible, provided, (a) the water is effectively removed early in the spring before breeding occurs, and (b) the water is not put in in the autumn until the weather is cool enough to prevent mosquito breeding.
3. Ponds must have sound, tight banks and bottoms to prevent wet areas due to seepage.
4. Ponds must have sufficient depth throughout to permit mosquito fish to penetrate freely all parts.
5. Ponds must not be overgrown with vegetation, especially along and near the margins, so that "mosquito fish" may have free access to all parts.
6. If the water is pumped, the supply and equipment should be adequate to fill the ponds with reasonable speed.
7. Duly authorized inspectors of mosquito abatement districts in which the

ponds occur should be permitted to inspect the area frequently to determine whether or not mosquito breeding is taking place.

8. If in spite of all precautions mosquito larvae do occur, a larvicide, preferably a pyrethrum emulsion, should be applied, but only where breeding is actually in progress.

Mosquito control and wild life conservation. Mosquito abatement operations if intelligently conducted need not be detrimental to wild life, though no doubt they have been so at times. In conducting control operations in suburban and rural areas an understanding of wild life ecology is urged, and a modification of measures to suit the situation is necessary. It is regrettable if wild life has been harmed; but there have also been unfounded complaints on the part of misinformed and intolerant wild life conservationists which have made it distinctly difficult for mosquito abatement officials to perform their proper function. It is important that the seemingly divergent viewpoints of conservationists and duck clubs and mosquito abatement officials be harmonized. No doubt each group will need to make reasonable concessions.

Cooperation between responsible mosquito abatement officials and the representatives of wild life interests is only made difficult when one side or the other sets itself upon a pedestal. Our properly trained experts in mosquito control are just as truly biologists as are wild life authorities, the only difference is usually in the fact that the former are trained in the field of invertebrate zoology and the latter in the field of vertebrate zoology. Both should be ecologists and have training in limnology. Fortunately, most of our authorities in the field of mosquito control have had training not only in the ecology of aquatic invertebrates but for obvious reasons also in vertebrate ecology. Entomologists trained in mosquito control and ecology, and wild life conservationists equally well trained in ecology ought to be able to see "eye to eye" as biologists. Working thus together as biologists on an equal footing, the aims and objectives of both sides will be advanced.

The almost universal use of the newer insecticides, DDT and others, in agricultural pest control operations as well as in mosquito control has brought to a focus the problem of the tolerance of each wildlife species, both aquatic and terrestrial, and the indirect effect as pertains to the food of the several species involved in treated areas. Cope[38] states that "in the case of DDT and wildlife (exclusive of fish), something over one pound per acre seems to be a safe level, while for fish a figure of 0.2 pound per acre would apply." For anopheline control 0.1 pound per acre of DDT is usually adequate and somewhat larger doses for culicine larviciding. For benzene hexachloride it is indicated that up to one pound per acre may be safe for wildlife, fish, and fish food organisms. Chlordane appears to be damaging to some fish at one pound per acre. TDE appears to be about one-fifth as toxic to warm water fishes as DDT.

Hess and Keener[39] (1947) made field tests with a 20 per cent solution by weight of technical grade DDT in a methylated napthalene (Velsicol NR-70) applied by airplane in the form of a thermal aerosol. The rate of application was approximately 0.1 pound of DDT per acre. They report the following results:

> The effect of routine treatments on an area basis was almost complete elimination of anopheline mosquitoes and surface Hemiptera and a significant reduction in culicine mosquitoes with no evidence of reduction of other forms. Observations in areas at the end of a full season of 16 applications at the rate of 0.1 lb. DDT per acre indicated that surface Hemiptera had been almost completely eliminated and mosquitoes were considerably reduced, but other forms were not significantly affected. Surface Hemiptera are not important as fish food and because of their predaceous habits may actually compete with fish for food. On the basis of these studies, it is therefore concluded that DDT applied in the manner indicated does not have any significant injurious effect upon the total population of fish food organisms—it is also concluded that the DDT treatment had no injurious effect upon the resident fish population.

Natural enemies of mosquitoes. A review and summary of the literature dealing with "Predators of the Culicidae" by Hinman[40] indicates that, excluding fish, the chances of finding satisfactory predators is not very encouraging. Among the natural enemies of mosquitoes few are so frequently referred to as dragonflies (also known as "mosquito hawks"), bats, and surface-feeding fish. Dragonflies, order Odonata, are predaceous in both the nymphal and adult stage. The aquatic nymphs are commonly found in quiet, shallow, permanent pools suitable also for mosquito breeding, and both may flourish in the same pool in spite of the fact that the dragonfly nymphs, usually relatively few in number, may feed on mosquito wrigglers. Since the nymphs feed in the mud and debris at the bottom, probably few wrigglers are captured. If the wrigglers are easily available, the nymphs will feed on mosquito larvae voraciously. Warren[41] reported a nymph of *Pantala* consuming 75 full-grown mosquito larvae by seven o'clock in the evening, which he had placed in a glass half-full of water in the morning. Adult dragonflies are exceedingly adept at capturing mosquitoes on the wing just before and at sunset. However, here again the number of dragonflies, which also feed on other insects, is no match for the mosquitoes. Dragonflies do not fly at night when night-flying mosquitoes are on the wing.

Bats are insectivorous and feed freely on mosquitoes; as many as 250, it is said, may be captured by one bat in a night, but with many other species of crepuscular and night-flying insects available, bats are not effective enough to be a large factor in control, even though one might tolerate them in large numbers near the home. Bat roosts, however, have been established to accommodate bats for the purpose of mosquito control.

Fish of various species have been advocated for many years. In *Nature* for December, 1891 (pages 223–224), there is this item: "An Englishman living on the Riviera, according to a correspondent, having been troubled by mosquitoes, discovered that they bred in the large tanks kept for the purpose of storing fresh water, which is rather a rare commodity at this Mediterranean resort. He put a pair of carp in each tank and succeeded in this way in extirpating the insect pest."

Howard (1910, *loc. cit.*) refers to the control of mosquitoes by goldfish in an ornamental aquatic garden near Boston:

> I took from the pond a small goldfish about three inches long and placed it in an aquarium where it could, if it would, feed upon mosquito larvae and still be under careful observation. . . . On the first day owing perhaps to being rather easily disturbed in its new quarters, this goldfish ate eleven larvae only in three hours, but the next twenty-three were devoured in one hour; and as the fish became more at home, the 'wigglers' disappeared in short order whenever they were dropped into the water. On one occasion twenty were eaten in one minute, and forty-eight within five minutes. This experiment was frequently repeated and to see if this partiality for insect food was characteristic of those goldfish only which were indigenous to this locality experimented with, some said to have been reared in carp ponds near Baltimore, Maryland, were secured. The result was the same. . . .

The most useful of all fishes for mosquito control is the top minnow or mosquito fish,[42] *Gambusia affinis* (Baird and Girard), a hardy, rapidly breeding, prolific surface-feeding fish which within its range normally inhabits shallow water suitable for mosquito propagation. It is viviparous and may produce as many as six to eight broods in a season with an average of 40 to a brood. The size of the fish ranges from 1¼ inches in length in the male to nearly 2 inches in the female. This fish is easily propagated and adapts itself to a variety of conditions with ease. It has been introduced into various parts of the world, even over great distances, for example, from Texas to the Hawaiian Islands and thence to the Philippines. Transportation of top minnows can be done satisfactorily in 10-gallon milk cans with tops punched with holes and water kept below the point where the top of the can begins to narrow. Although as many as 500 fish may be transported for an hour's trip with only moderate loss, not over 200 young fish per can should be shipped on longer trips, and special care must be exercised to remove dead fish at intervals and freshen the water.

For garden pools 10 square feet in diameter, 20 top minnows will be ample, and no artificial feeding will be necessary. The *Gambusia* will more or less regulate their own numbers according to the food supply available.

Top minnows will evidently not feed on mosquito larvae when these are motionless, hence are not markedly effective in the control of mosquitoes whose larvae are sluggish; e.g., the usually motionless larvae of *Anopheles quadrimaculatus* Say do not attract the attention of top minnows as readily as do the more active larvae of *A. m. freeborni* Aitken; hence the minnows are not so effective in control.

Other fish which have been found useful are *Heterandria formosa, Fundulus diaphanus,* and *Fundulus dispar* for fresh water, and *Cyprinodon variegatus, Fundulus heterocliteus, Fundulus similis, Fundulus majalis,* and *Lucania para* for salt or brackish water. To these may be added a number of species as listed by various authors, notably Hegh,[43] Radcliffe,[44] and Hamlyn-Harris.[45] The International Health Board of the Rockefeller Foundation has issued (1924) a comprehensive treatise entitled "The use of fish for mosquito control."

In Guayaquil, Ecuador, the problem of the yellow fever mosquito was solved, according to Connor,[46] by the use of fish. Connor states that the domestic water supply is delivered to the houses daily and is stored in tanks and other receptacles, there being at the time of his writing about 7,000 of the former and 30,000 of the latter, such as barrels, oil cans, earthenware bowls, etc. In these various containers yellow fever mosquitoes developed in countless numbers. Experimentation with several species of fish finally resulted in the selection of the "*chalaco*" (*Dormitator latifrons,* family Gobiidae). These fish, furnished to the Yellow-Fever Service by local fishermen, were placed in a specially prepared well, the conditions of which approximated those of the stream from which the fish were taken. After a few days the fish were removed to a second well, the water of which was the same as that used by the city. Connor writes further as follows:

> The fish are then taken from the wells and placed in tins or pails and delivered to the inspectors. Instructions have been given to each inspector that every fresh-water container in his district is to be supplied with one fish, regardless of the presence or absence of mosquito larvae in the container at that time. The public is encouraged, personally, by notices in the newspaper, and by the inspectors themselves, to exercise reasonable care in protecting the fish. The public of Guayaquil has responded in a whole-hearted manner to the requests of the Yellow-Fever Service, and many families have in their possession at this time the identical fish which was given them to mosquito-proof their water container nearly eighteen months ago.
>
> More than 30,000 water receptacles have in this way been purged of mosquito larvae in a relatively short time and at a minimum of expense. With

the continued use of fish it is believed that the yellow-fever mosquito can be reduced to such small numbers that, should a few cases of the disease be introduced into the community, it would not spread.

Transportation of exotic mosquitoes by airplane. The accidental importation of exotic mosquitoes may result in severe epidemics of disease with heavy loss of life and may necessitate expensive campaigns to root out the evil. Particularly noteworthy is the malaria epidemic in Brazil following the introduction of *Anopheles gambiae* Giles from Africa, referred to in an earlier chapter as described by Soper and Wilson (*loc. cit.* p. 228). Such dangers, particularly importation of the vector of yellow fever, have long been recognized as possible through air transportation. Various amendments have been made to the "International Sanitary Convention for Aerial Navigation of April 12, 1933," signed by the United States in April, 1934, according to Miller, Burgess, and Carpenter[47] (1947). Article 54, (a 1944 amendment) reads, "In view of the special risk of conveying insect vectors of malaria and other diseases by aircraft on international flight, all such aircraft leaving affected areas will be disinsected . . . further disinsectization of the aircraft on or before arrival may be required if there is reason to suspect the importance of insect vectors."

There are said to be about 35,000 airplanes entering the United States annually from foreign ports, 80 per cent of which are from the West Indies and Latin America. Scheduled flight times from all parts of the world are short enough to enable stowaway mosquitoes to arrive alive in the United States. Southern and California ports of entry, with more favorable year-round climatic conditions, are regarded as offering the most likely points of invasion. Although the enormous wartime movement of aircraft (Herms,[48] 1946) showed a tremendous potential of insect transport particularly at Honolulu, strict quarantine regulations including airplane disinsectization administered by the United States Navy proved very effective. Freon-pyrethrum–DDT aerosol "bombs"—continue to be used for disinsectization purposes. Automatic centrally controlled devices for releasing aerosols in aircraft are now being tested.

Recently Rosen, Reeves, and Aarons[49] reported the presence of *Aedes aegypti* (Linn.) on Wake Island. Reeves (1949) at the 8th Pacific Science Congress presented a paper on "Possible Recent Introductions of Mosquito Vectors of Human Disease in the Central Pacific."

BIBLIOGRAPHY

1. Howard, L. O., 1892. "An experiment against mosquitoes," *Insect Life,* 5:12–14.

2. Ross, Ronald, 1910. *The Prevention of Malaria.* New York: E. P. Dutton and Co. xx + 669 pp. (24 plates).

3. Le Prince, J. A., and Orenstein, A. J., 1916. *Mosquito Control in Panama: The eradication of malaria and yellow fever in Cuba and Panama.* New York: G. P. Putnam's Sons. xvii + 335 pp.

4. Ross, Ronald, 1902. *Mosquito Brigades and How to Organize Them.* New York: Longmans, Green, & Co. 100 pp.

5. Herms, W. B., and Gray, H. F., 1944. *Mosquito Control: Practical Methods for Abatement of Disease Vectors.* 2nd ed., New York: The Commonwealth Fund. viii + 419 pp.

6. Smith, John B., 1904. *Report of the New Jersey State Agricultural Experimental Station upon the Mosquitoes Occurring within the State, Their Habits, Life Histories, etc.* Trenton: MacCrellish & Quigley.

7. Quayle, H. J., 1906. *Mosquito Control.* Berkeley: Univ. Calif. in Agric. Exper. Sta. Bull., no. 178. 56 pp.

8. Herms, W. B., 1913. *Malaria–Cause and Control.* New York: The Macmillan Co. xi + 163 pp.

9. Le Prince, J. A., 1928. "Historical review of developments of control of disease-bearing mosquitoes," *Tr. Am. Soc. Civ. Eng.,* 92:1259.

10. Herms, W. B., 1948. "Public relations in mosquito control operations," *Proc. and Papers 16th Annual Conf. Calif. Mosq. Control Assn.* (Berkeley), pp. 51–54.

11. King, W. V., 1948. "Man-made malaria," *Proc. 4th Internat. Cong. Trop. Med. and Malaria* (Abstract), Washington, D. C.

12. Wilson, H. M., 1909. *Irrigation Engineering.* 6th ed., New York: John Wiley & Sons, Inc. 625 pp.

13. Bishop, E. L.; Hollis, M. D.; *et al.;* 1947. *Malaria Control on Impounded Water.* Washington, D. C., U. S. Public Health Service and Tennessee Valley Authority. xiii + 422 pp.

14. Hurlbut, Herbert S., 1943. "Observations on the use of sea water in the control of *Anopheles albimanus* Wied.," *J. Parasitol.,* 29:356–60.

15. Horsfall, William R., 1942. *Biology and Control of Mosquitoes in the Rice Areas.* Fayetteville: Univ. Arkansas, in Agric. Exper. Sta. Bull., no. 427. 46 pp.

16. Rao, B. A., and Sweet, W. C., 1937. "Paris green and paddy," *Records Malaria Survey India,* 7:185–89.

17. Hill, Rolla B., and Cambournac, F. J. C., 1941. "Intermittent irrigation in rice cultivation, and its effect on yield, water consumption and *Anopheles* production," *Am. J. Trop. Med.,* 21:123–44.

18. Russell, Paul F.; Knipe, Fred. W.; and Rao, H. Ramanatha; 1942. "On the intermittent irrigation of rice fields to control malaria in South India," *J. Mal. Inst. India,* 4:321–40.

19. Cambournac, F. J. C., and Fonseca da, A. E., 1948. "Experiments on the control of anopheline larvae and malaria in the rice-growing regions of Portugal," *Proc. 4th Internat. Cong. Trop. Med. & Malaria* (Abstracts), Washington, D. C.

20. Gillette, H. P. S., 1948. "The control of Bromeliad malaria in Trinidad, B. W. I.," *Proc. 4th Internat. Cong. Trop. Med. & Malaria* (Abstracts), Washington, D. C.

21. Aarons, Theodore, 1948. "Cemetery mosquito control by aerosol," *Proc. and Papers 16th Annual Conf. Calif. Mosq. Control Assn.*, Berkeley, pp. 84–85.

22. Le Prince, J. A., 1915. "Control of Malaria; oiling as an antimosquito measure," U. S. Public Health Service, *Pub. Health Rep.*, 30:599–608.

23. Barber, M. A., and Hayne, J. B., 1921. "Arsenic as a larvicide for anopheline larvae," U. S. Public Health Service, *Pub. Health Rep.*, 36:3027–34.

24. Vannote, R. L., and Ginsburg, J. M., 1931. "Practical application of pyrethrum mosquito larvicide," *Proc. 18th Annual Meeting, New Jersey Mosq. Extermination Assn.*, pp. 111–120.

25. Yust, Harold R., 1947. "DDT to control *Anopheles farauti* on Espiritu Santo, New Hebrides Islands," *J. Econ. Entomol.*, 40:762–68.

26. Ginsburg, Joseph M., 1943. "Aerosol sprays for killing and repelling mosquitoes," *Proc. 30th Annual Meeting, New Jersey Mosq. Extermination Assn.*, pp. 211–17.

27. ———, 1935. "Larvicides and a method of temporary protection from adult mosquitoes in limited areas," *Proc. 25th Annual Meeting New Jersey Mosq. Extermination Assn.*

28. Weathersbee, Albert A.; Arnold, F. T. Jr.; and Hopkins, Julian P.; 1948. "Observations on the duration of toxicity of DDT to *Anopheles quadrimaculatus* Say under field conditions," *J. Nat. Malaria Soc.*, 7:138–43.

29. Downs, W. G.; Iris, R. C.; and Gahan, J. B.; 1948. "Residual effectiveness of DDT in the third season after application," *Am. J. Trop. Med.*, 28:741–745.

30. Gray, H. F., 1948. "Some newer ideas in mosquito control," *Bol. Ofic. san. panam.*, 27:321–25.

31. American Mosquito Control Association, 1948. *The Use of Aircraft in the Control of Mosquitoes*, in Bull. no. 1., ed. P. F. Russell, G. H. Bradley, A. D. Hess, J. A. Mulrennan, and H. H. Stage. 46 pp. + 45 plates.

32. Hewitt, C. G., 1919. "The use of the aeroplane in entomological work," *Agric. Gaz. Canada*, 6:877.

33. King, W. V., and Bradley, G. H., 1925. "Airplane dusting controls malaria mosquitoes," *Aero Digest*, 7:652–53.

34. Williams, L. L., Jr., and Cook, S. S., 1927. "Paris green applied by airplane in the control of *Anopheles* production," U. S. Public Health Service, *Pub. Health Rep.*, 62:459–80.

35. Ginsburg, J. M., 1931. "Airplane application of larvicide on mosquito breeding places inaccessible from land," *New Jersey Agric. Exper. Sta.* Annual Rept, (1930–1931), 19:173–76.

36. Granett, Philip, 1943. "The significance of the development of mosquito repellents for the protection of military and civilian populations," *Proc. 30th Annual Meeting, New Jersey Mosq. Extermination. Assn.*, 203–10.

37. Knipling, E. F., and Dove, W. E., 1944. "Recent investigations of insecticides and repellents for the armed forces," *J. Econ. Entomol.*, 37:477–80.

38. Cope, Oliver B., 1948. "Toxicities and tolerances of new insecticides in relation to wildlife and fish," *Proc. and Papers 16th Annual. Conf. California Mosq. Control Assn.* (Berkeley), pp. 26–29.

39. Hess, A. D., and Keener, G. G. Jr., 1947. "Effect of airplane distributed DDT thermal aerosols on fish and fish food organisms," *J. Wildlife Management,* **11**:1–10.

40. Hinman, E. H., 1934. "Predators of the Culicidae (mosquitoes)," *J. Trop. Med. & Hyg.,* **37**:129–34, and 145–50.

41. Warren, Alfred, 1915. *A study of the Food Habits of the Hawaiian Dragonflies or Pinau with Reference to Their Economic Relation to Other Insects.* "College of Hawaii Publications," *Bull. No.* 3. 45 pp.

42. Seale, A., 1917. "The mosquito fish, *Gambusia affinis* (Baird and Girard), in the Philippine Islands," *Philippine J. Sc.,* **12**:177–89.

43. Hegh, E., 1918. *Comment nos planteurs et nos colons peuvent-ils se protéger contre les moustiques qui transmettent des maladies.* Report Minister of Colonies, Service of Agriculture of Belgium, no. 4. 200 pp.

44. Radcliffe, L., 1915. *Fishes Destructive to the Eggs and Larvae of Mosquitoes.* Washington, D. C.: Dept. of Commerce, in Bur. Fisheries, Circ. 17, pp. 1–19.

45. Hamlyn-Harris, Ronald, 1929. "The relative value of larval destructors and the part they play in mosquito control in Queensland," *Proc. Roy. Soc. Queensland,* vol. 40. 38 pp. + 8 plates.

46. Connor, Michael E., 1921. "Fish as mosquito destroyers." *Natural History,* **21**:279–81.

47. Miller, Albert; Burgess, Robert W.; and Carpenter, Stanley J.; 1947. "Potentialities of transportation of exotic anophelines by airplane," *J. Nat. Malaria Soc.,* **6**:227–43.

48. Herms, W. M., 1946. "Wartime aviation quarantine. Pests and their control," *J. Pest Control Industry,* **14**:24–25.

49. Rosen, L.; Reeves, W. C. and Aarons, T. 1948. "*Aedes aegypti* on Wake Island," *Proc. Hawaian Entomolog. Soc.* (for year 1947), **13**:255–56.

CHAPTER XIV

HORSEFLIES, DEER FLIES, AND SNIPE FLIES

A. HORSEFLIES

Family Tabanidae

Tabanidae. To this large cosmopolitan family of the Order Diptera, comprising about 2,500 species, belong the avidly bloodsucking flies commonly known as horseflies, gadflies, breeze flies, greenheads, deer flies, or mango flies. These flies are usually quite large and heavy-bodied (Fig. 94), measuring in length from 7 to 10 mm in the smaller species

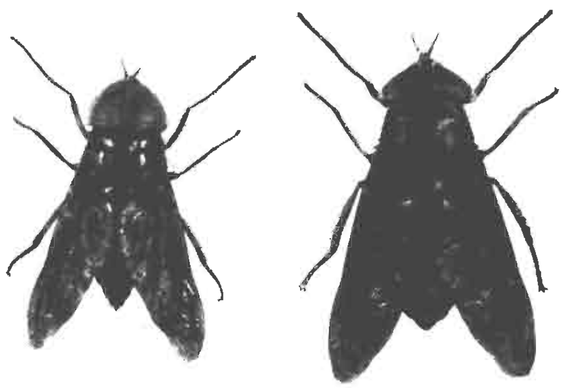


FIG. 94. The black horsefly, *Tabanus atratus*. (*Left*) male, (*right*) female. ×1.5. (Photograph by Hine.)

to from 20 to 30 mm in the larger species. They are strong fliers and notorious pests of horses, cattle, deer, and many other warm-blooded animals and are at times annoying to man, particularly the persistent members of the genus *Chrysops* (Fig. 95). The males feed on vegetable sap and some may suck the juices of soft-bodied insects, but they do not bite. The eyes are very large and widely separated (dichoptic) in the females, and contiguous (holoptic) in the male. The antennae are short (though fairly long in some genera such as *Chrysops*) and are porrect, consisting of three joints, the terminal segment or flagellum being elongate and annulated, without an arista. The wing venation (Fig. 12B) is simple and characteristic. The mouth parts of the females are blade-like and function as cutting instruments. The metamorphosis is complete (Fig. 96).

The family is divided into two subfamilies, the Pangoniinae, which includes all Tabanidae with apical spurs on the hind tibiae and in which ocelli are usually present, e.g., the species belonging to the following

FIG. 95. A deer fly (*Chrysops*) in the act of oviposition. Note also an egg mass farther down on the leaf. ×1. (Photograph by Hine.)

genera: *Silvius, Chrysops, Goniops, Apatolestes,* and *Pangonia;* and the subfamily Tabaninae, in which the apical spurs and ocelli are lacking, as in *Tabanus* (Fig. 97) and *Chrysozona.*

Since most of the species are aquatic or semiaquatic in breeding habit (some are able to develop in moist earth, leaf mold, and rotting logs), the eggs are deposited in layers on objects over water or moist situations favorable for the larvae, such as overhanging foliage, projecting rocks, sticks, and aquatic vegetation. The narrow cylindrical eggs (1.0 to 2.5 mm long) vary in numbers from 100 to 1,000, and are deposited commonly in layers covered with a waterproof secretion which also binds the eggs together tightly.

The larva (Fig. 98) has a slender, cylindrical, contractile body tapering at both ends and consisting of 11 segments (interpreted also as a head and 12 segments, the last very short). The head is small and retractile, with pointed mandibles capable of inflicting a sharp bite; at the posterior end is situated a tracheal siphon which telescopes into the anal segment. The pupa (Fig. 98), resembling naked Lepidoptera, is obtect, abruptly rounded anteriorly, tapering posteriorly, with wing and leg cases closely attached to the body; the abdominal segments are free and about equal in length, segments two to seven each bear a more or less complete ring of spines near the posterior third. The adult fly emerges from the pupal case through a split or slit along the dorsum of the thorax. The Tabanidae therefore belong to the Orthorrhapha, i.e.,

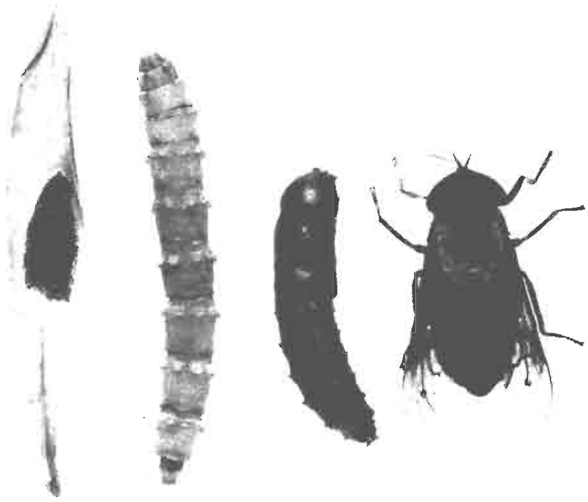


FIG. 96. *Tabanus punctifer.* Egg mass on willow leaf, larva, pupa, and adult female fly.

the flies escape from the pupal skin through a T-shaped opening. In contrast the adults of the Cyclorrhapha such as houseflies escape from the pupal case through a round opening made by pushing off the anterior end. In pushing off this end of the puparium the housefly and its allies use a frontal bladder-like structure known as a ptilinum, absent in the Orthorrhapha such as the Tabanidae.

Students concerned with the Tabanidae are referred to such monographic works as those of Osten Sacken,[1] Hine,[2] Enderlein,[3] Kröber,[4] and Stone.[5]

Breeding habits and life history. The eggs, numbering up to nearly a thousand, are deposited during the warmer months of the year in compact layers on objects such as the leaves of willow and emergent aquatic vegetation which grows from or overhangs swampy areas, ponds, etc.

(Fig. 95). The incubation period is greatly influenced by weather conditions, but during midsummer the usual range is from five to seven days. On hatching the larvae fall to the surface of the water, upon mud or moist earth, in clumps and quickly drop to the bottom or burrow individually into the wet or damp earth, where they begin feeding at once on organic matter. Many species are predators, sucking the juices of insect larvae, crustacea, snails, earthworms, and other soft-bodied animals; cannibalism has been observed in several species. The larvae of Tabanidae are commonly encountered buried in the mud along the

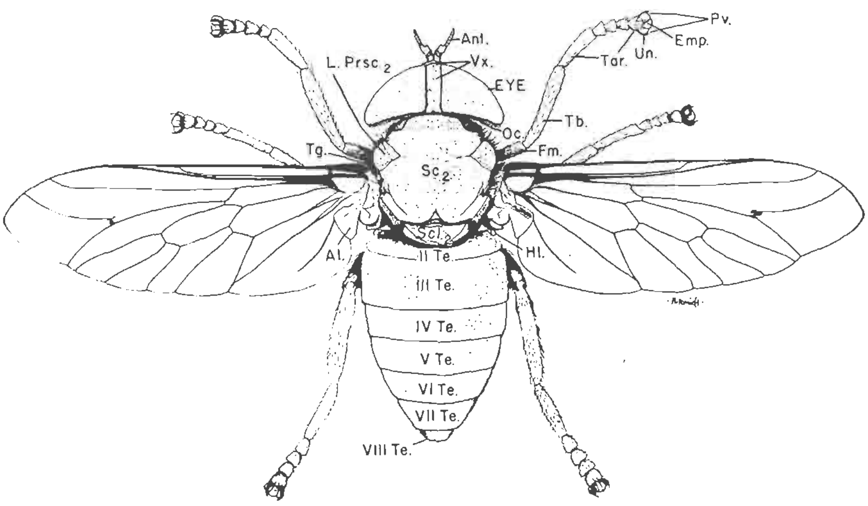


FIG. 97. Morphological details of a femal horsefly, *Tabanus punctifer*. Explanation of abbreviations: *Ant.*, antenna; *Al.*, alula; *Emp.*, empodium; *Fm.*, femur; *Hl.*, haltere; *L. Prsc.*, lobe of prescutum (notopleuron); *Oc.*, occiput; *Pv.*, pulvilli; *Sc.*, scutum; *Scl.*, scutellum; *Tar.*, tarsi; *Tb.*, tibia; *Te.*, tergite; *Tg.*, tegula; *Un.*, ungues; *Vx.*, vertex.

edges of marshy ponds, roadside ditches, and the overflow from rice fields; the writer has found numerous larvae of *Tabanus gilanus* Townsend in the mud at the edge of salt marshes. The larvae of certain species may be found in moist leaf mould and debris.

The larvae grow rapidly during the summer and autumn, and very slowly if at all during the winter in the single-brooded species, attaining full growth in the following early spring. There is some uncertainty as to the number of larval instars; it varies, according to some authors from four to nine, the first molt evidently taking place shortly after hatching. An excellent account of the early stages of Tabanidae may be found in the work of Marchand.[6]

When the full-grown larva prepares to pupate, it moves into drier earth, usually an inch or two below the surface, and in a day or two the pupal stage is reached. This stage requires from two to three weeks, varying with the species. Stone[7] reports that most of the *Chrysops* species emerge in less than two weeks, even in as short a time as five days. The flies emerge from the pupa at the surface, the wings soon unfold, and the insects take refuge among nearby foliage or rest on objects near at hand; in a short time they begin to feed, the females seeking blood and

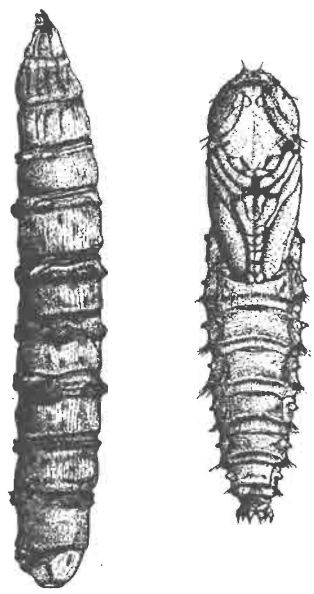


FIG. 98. Lateral view of larva (*left*) and ventral view of pupa (*right*) of *Tabanus gilanus*.

the males feeding on flower secretions and vegetable juices where one may find them by sweeping with a net.

Much important information based on rearing experience with many species may be obtained by consulting the various publications by Schwardt, among them *Horseflies in Arkansas*.[8] In an earlier paper (Research paper 219, Journal Series, University of Arkansas) Schwardt reports life-history records based on 202 individuals of *Tabanus lineola* Fabr., a common North American species of "greenhead," viz., average incubation period 4 days, average larval period 48.8 days, average pupal period 8.1 days, average preoviposition period 9 days, total developmental period averaging 96.9 days.

In the Sierra Nevada and other mountain ranges horseflies breed in great numbers at elevations of 8,000 to 9,000 feet in soggy ground caused by springs, and water from melting snow in the summer. Deer and other wild animals suffer much from the bites of these flies.

Bites. The horseflies have broad blade-like mouth parts (Fig. 24), which inflict a deep, painful wound, causing a considerable flow of blood; and owing to their intermittent biting habits there is danger of infection.

Webb and Wells,[9] working on *T. phaenops* O. S. in western Nevada, estimated that eight flies feeding to satiety would consume a cubic centimeter of blood. On this basis they calculated that 20 to 30 flies feeding for six hours would take an average of at least 100 cc of blood. This would amount to approximately a quart in ten days. Philip,[10] working in Minnesota, derived a larger estimate of blood loss. Basing his figures on a somewhat heavier infestation than that in Nevada, Philip placed the daily loss of blood for each animal at 300 cc, or nearly one-third of a quart. Neither of these estimates includes the blood which exudes from the bite after the fly leaves. Philip, however, calls attention to this additional loss. The horseflies most abundant in Arkansas are comparable in size to the species on which these estimates of blood loss were made, and the infestation is often heavier than 50 flies per animal.

(Schwardt, 1936, *loc. cit.*)

In describing an outbreak of gadflies in Kentucky, Garman[11] writes:

Beef cattle had lost an average of 100 pounds as a result of the constant annoyance from them. . . . On cattle I counted from ten to nineteen. On mules and horses in harness they were a constant annoyance and even hogs were not exempt. Seven of the flies were counted on the exposed side of one of these animals lying in a puddle.

The persecuted stock appeared to have given up fighting their enemies and allowed them to have their way. The switch of a cow's tail was observed to pass over the backs of clinging flies without causing them to move. . . . During the middle of the day animals suffered so much that they refrained from grazing at all, either standing close together about the barn or else lurking singly in thickets or standing in pools formed by small streams.

Relation to anthrax. Anthrax, also known as malignant pustule or carbuncle, wool sorter's disease, or charbon, is caused by *Bacillus anthracis*. Nearly all species of domesticated animals and man are susceptible; the herbivora and rodents are most liable to infection.

After the inoculation of the organism into the animal the incubation period is from three to six days. The bacilli are seen in the blood stream in advanced cases as chains of rod-shaped bodies. Entrance to the body is gained mainly in one of three ways: (1) through lesions or pricks, i.e., *inoculation*, producing local anthrax or malignant pustule; (2) by *inhalation* of the spores, producing pulmonary anthrax; and (3) by *ingestion* with food, producing intestinal anthrax.

The horseflies are decidedly intermittent in their biting habits and inflict a definite lancet-like prick from which blood exudes so that the proboscis becomes soiled. The flies will bite sick animals as well as healthy ones.

Horseflies relate directly to the first mode of infection (inoculation), and it is not altogether improbable that an epidemic of anthrax might thus be started and could be spread. Nuttall cites Bollinger (1874), who captured horseflies on a cow dead from anthrax and saw the bacilli in preparations made from the stomachs and intestines of the insects. Two rabbits inoculated therewith died of anthrax.

Mitzmain[12] (Mayne), in a series of experiments with *Tabanus striatus* Fabr., showed that direct mechanical inoculation of guinea pigs could be readily effected by the bite of the fly. He permitted the flies to feed on an inoculated guinea pig shortly before its death and transferred the flies soon thereafter (45 seconds to 30 minutes) to a healthy guinea pig. The death of these animals followed in from three to three and one-half days.

Instances are recorded in which apparently the simple bite of the infected fly was all that was needed to produce malignant pustule in

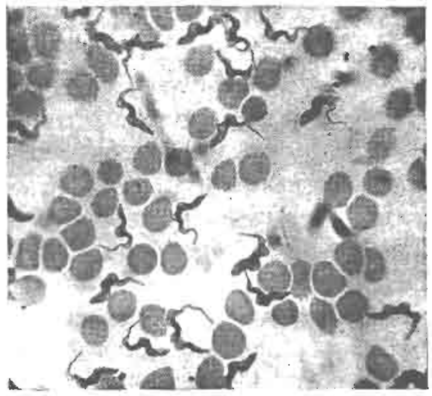


FIG. 99. *Trypanosoma evansi*, the causal organism of surra. (After Yutuc.)

humans. Several veterinarians have related instances to the writer in which this had occurred, notably one case in which a man was in the act of burying a cow dead of anthrax when he was bitten severely on the back of the neck by a horsefly and in due time developed a malignant pustule. Nuttall also cites a number of similar instances.

Surra is a highly fatal disease of equine animals in southeastern Asia and many of the islands of the Malay Archipelago. It was first described (1885) from India by Evans. The causal agent is *Trypanosoma evansi* Steel. Yutuc,[13] who reported on the first outbreak of surra in British North Borneo, states that it is highly fatal to dogs; carabaos are evidently not severely affected as a result of an infection and thus serve as reservoirs. In India camels also serve as reservoirs. In the Philippine Islands and elsewhere virulent outbreaks of surra occur among bovines.

The trypanosomes (Fig. 99) are found in the blood of infected animals, and especially in the lymph of swollen glands, from the beginning of the first symptoms. During the early stage of the disease, according to Yutuc, practically no clinical manifestations are visible save a variable appetite and an intermittent fever; there is progressive emaciation and edema of the abdomen and genitalia. Guinea pigs, white mice, and monkeys are highly susceptible laboratory animals.

Mitzmain[14] (Mayne) succeeded in transmitting the infection from animal to animal through the agency of a horsefly, *Tabanus striatus* Fabr.

In a series of experiments in which *Tabanus striatus* Fabr. was used, he first allowed the flies to bite an infected guinea pig or horse for not more than one minute, usually 45 seconds, and then transferred them to a healthy animal where they were allowed to complete the meal without interruption. An interruption of five seconds to three minutes was necessary to transfer the flies from animal to animal. The horses and mule employed in these experiments were kept in a screened stable for from six to eight months previously, and the monkeys, guinea pigs, and rabbits in fly-screened cages for about 90 days. In all cases the animals were examined frequently (blood examinations were made) and declared surra-free at the time the experiments began.

Flies bred from eggs were allowed to bite a guinea pig which had been inoculated with blood from a carabao which had been infected with surra for nearly a year previous to the test. Three flies were applied individually in tubes to the surra-infected guinea pig and allowed to feed from 45 seconds to one minute and 30 seconds, after which they were transferred to a monkey and allowed to feed until satisfied, i.e., from 5 to 21 minutes. The first rise in the monkey's temperature, 40.1° C, occurred on the eleventh day, accompanied by the presence of a few trypanosomes in the peripheral circulation; the trypanosomes increased in numbers until the death of the monkey on the twenty-fifth day.

Blood from the heart of this monkey was inoculated into a horse and two guinea pigs. The latter showed infection on the eighth and ninth days respectively, and the horse on the seventh day. Two flies were permitted to bite this horse, the insects being interrupted in their biting in from 40 to 45 seconds and then transferred to a healthy horse, where the feeding was completed. The latter animal showed numerous trypanosomes in its circulation on the ninth day. Thus positive results were secured in both a monkey and a horse.

Blood from this newly infected horse was inoculated into a mule, two monkeys, and two guinea pigs, all of which became infected in due time, both monkeys dying on the fourteenth and fifteenth days respectively.

A second series of experiments was carried on with captured flies, which were allowed to bite the surra-infected horse mentioned above and later a healthy horse, similar feeding methods being observed. This experiment also gave positive results, as did blood inoculations into monkeys and guinea pigs.

In order to eliminate the possibility of hereditary transmission of trypanosomes in the flies a further experiment was conducted with 74 flies, hatched from eggs of a fly which, previous to egg deposition, had fed on a surra-infected monkey; the 74 flies were allowed to bite a healthy monkey during a period of two weeks with negative results.

Mayne concludes that the " . . . contaminated labellum of the fly does not appear to be a factor in the conveyance of infection. The maximum length of time that *Trypanosoma evansi* (Steel) has been demonstrated microscopically in the gut of this species of fly after feeding on infected blood is thirty hours; the organisms were found in the fly's dejecta two and one-half hours after biting the infected animal; and suspensions of flies, when injected subcutaneously, were found infective for animals for a period of ten hours after the flies had fed on infected blood."

In a letter to the writer under date of November 18, 1913, Mayne states that " . . . infection is not transferred by *Tabanus striatus* Fabr. later than twenty minutes after the infective meal. The longest time I have succeeded in inducing flies to transmit was fifteen minutes and all results from twenty minutes to forty-eight hours were entirely negative. This despite the fact that trypanosomes survive in the intestinal tract of *T. striatus* Fabr. for a period of thirty hours." He believes this horsefly to be the principal carrier of surra and that the stable fly, *Stomoxys calcitrans* (Linn.), is ruled out, which is indeed indicated by the long and careful series of experiments conducted by that worker on both species of flies.

Basu[15] (1945) reports that the occurrence of surra in India is seasonal: the peak period for bovine surra is in August; for equine surra it is in October; and for surra of camels and others it is in September. The aggregate incidence reaches its peak in September. He also states that the seasonal curve of surra and human malaria in the Punjab coincide.

Tularemia. In 1919 a disease of man of hitherto unknown etiology occurring in the state of Utah (U.S.A.) was reported by Francis[16] as deer-fly fever or Pahvant Valley plague. It was later described by the same author[17] and given the name tularemia. It is a specific infectious disease traceable to *Pasteurella* (= *Bacterium*) *tularensis* (McCoy and Chapin). It was described by Francis as a disease of rural populations occurring during the summer months, coinciding with the prevalence of the newly discovered vector, *Chrysops discalis* Will., a deer fly (Tabanidae). Francis states, "Following the fly bite on some exposed surface of the body (neck, face, hands or legs) the onset is sudden, with pains and fever; the patient is prostrated and is confined to bed; the lymph glands which drain the bitten area become tender, inflamed and swollen, and commonly suppurate, requiring incision. The fever is of a septic type, lasting from three to six weeks, and convalescence is slow." The disease occurs not only in the United States but also in Canada, Alaska, northern Europe, Russia, Japan, and many other parts of the world. The pathology of tularemia is described in great detail by Lillie and Francis.[18] In the acute form of the disease a primary ulcer (eschar) develops at the site of the inoculation. Francis and his co-workers found that rabbits constitute an important reservoir for the infection and that it is transmitted from rabbit to rabbit by the *Chrysops* fly. The fly is undoubtedly merely a mechanical vector as indicated by the experiments of Francis and Mayne, *et al.* (*loc. cit.*). It has also been found that tularemia can be transmitted from rabbit to rabbit by means of the rabbit louse, *Haemodipsus ventricosus* (Denny). *Cimex lectularius* Linn., the common bedbug, was also found to be a successful vector in laboratory experiments with guinea pigs, as was the mouse louse, *Polyplax serratus* (Burm.), in the case of white mice. Mosquitoes and fleas have been shown to be able to transmit the infection. Several species of ticks are involved. *Dermacentor andersoni* Stiles, is an important vector, perhaps the most important one because the infection is transmitted transovarianly from generation to generation. Tularemia is now known to exist in nature in many species of animals; among these are meadow mice, ground squirrels, beavers, coyotes, sheep, and quail and other game birds. Infection may be acquired not only by insect bites but also by contact with infected insect feces, infected raw meat, and in rare instances through contaminated water. Over 2,000 cases of tularemia were reported in the United States in 1938, with a mortality rate of about 5 per cent.

Loiasis. The so-called mango fly, *Chrysops dimidiata* v. d. Wulp, has been shown by Leiper (*loc cit.* p. 6), to be a vector of *Loa loa* (Cobbold), the African eye worm of man in various endemic regions in Africa, particularly the Belgian Congo. *Chrysops silacea* Austen has been proved to be a carrier of the organism by Connall and Connall,[19] who completely elucidated the life cycle not only in this fly, but also in *C. dimidiata* v. d. Wulp. Microfilariae of *Loa loa* are found in the peripheral blood vessels during the daytime, showing a diurnal periodicity which gave rise to the term *Microfilaria diurna* Manson. The larvae measure about 300μ in length by 7.5μ in thickness, resembling

Wuchereria bancrofti (Cobbold) quite closely. In this stage they are ingested by the *Chrysops* flies and undergo development similar to that of *Wuchereria bancrofti* in the mosquito. Metamorphosis is completed in from 10 to 12 days, the larva increasing in length "tenfold." When the infected fly bites, the mature larvae issue from the proboscis, come to lie upon the skin of the host, and quickly disappear by burrowing.

The adult worms, females measuring from 50 to 70 mm in length and the males about half this length, inhabit the superficial subcutaneous connective tissue and are known to move about from place to place quite rapidly, giving rise to transient itching swellings known as Calabar swellings. The parasites have been observed in many parts of the body, such as the scrotum, penis, breast, eyelid, anterior chamber of the eye, tongue, finger, and back. The worms may be most readily excised when they travel across the bridge of the nose or conjunctiva.

El debab. El debab is a trypanosomiasis of Algerian horses and camels traceable to *Trypanosoma berberum* Edmond and Et. Sergent. This disease is evidently spread by horseflies, *Tabanus nemoralis* Meig. and *T. tomentosus* Macq. being considered the vectors.

Control. Comparatively little of a specific preventive nature has been done, except for the notable work of Porchinski, reported by Howard.[20] Porchinski observed that tabanids collect in great numbers in the neighborhood of damp places and lower themselves to the surface of pools to drink, actually touching the water with their bodies. It occurred to him that a covering of kerosene on the water would endanger the lives of the insects as they came in contact with the surface. Hence a quantity of kerosene was applied to a given pool, with most gratifying results. By the third day of the experiment, the "pool of death" was covered with "floating islands" of dead tabanids. Porchinski recommends that a favorite pool be selected, and that the oil be poured on so that a thick uniform layer of oil is formed covering the entire pool. Such "pools of death" apparently attract the tabanids from over a considerable adjacent area. The oil must of course be applied as early as possible during the season when the adult flies appear and begin to mate and deposit eggs.

The author[21] has called attention to the breeding of tabanid flies in rice fields, particularly in roadside pools, the result of rice-field drainage. Correction of drainage defects is an important procedure. Drainage of breeding areas is good practice where this can be done without interference with agriculture. In the California rice fields the author found characteristic egg masses of *Tabanus punctifer* O.S. attached to the stems and blades of rice plants. These eggs were commonly heavily parasitized by the hymenopterous egg parasite *Telenomus* (= *Phanurus*) *emersoni* (Girault). This parasite has been shown by Parman[22] to be a potent factor in the control of tabanids near Uvalde, Texas, where artificial dissemination of the parasite was practiced.

The species of Tabanidae. There are about 2,500 species of Tabanidae included in about 30 genera, with over 1,200 described species in the genus *Tabanus* alone. They are world-wide in distribution. Although the author has examined much of the literature concerning the Tabanidae, only a few species are briefly referred to in this work (see below). Students concerned with the Tabanidae of given localities should consult the work of specialists in that area, e.g., Fairchild's excellent articles on the Panamanian species.

1. *Tabanus atratus* Fabricius, the black horsefly (Fig. 94), measures from 16 to 28 mm in length. It is distributed over most of the United States east of the Rocky Mountains and into Mexico. The whole insect is uniformly black and the thorax and abdomen in well-preserved specimens are thinly covered with a whitish "dust" which is easily rubbed off if the specimens are not cared for properly.

2. *Tabanus stygius* Say is the black and white horsefly and is a widely distributed species east of the Rocky Mountains. Its length is 20 to 22 mm. The third segment of the antennae is red at the base, blackish at the apex; the first and second segments and palpi are dark; the legs are black, often the tibia are reddish at the base; the wings are yellowish brown with the posterior border approaching hyaline, a brown spot on the bifurcation of the third vein, and the transverse vein closing the discal cell margined with brown; the abdomen is a uniform black; in the female the thorax dorsally is plainly whitish pollinose with more intense longitudinal lines; the thorax of the male is dorsally a uniform grayish brown.

3. *Tabanus punctifer* O.S. (Fig. 96) is also a black and white horsefly resembling *T. stygius* Say except that the front tibiae are white on the basal third and the thorax is uniformly white in both sexes; there is usually a small dark spot near the tip of the wing. It is the largest and best known species of horsefly in western North America, particularly along the Pacific coast.

4. *Tabanus vicarius* Walker (= *Tabanus costalis* Wied.), the greenhead, is one of the most dreaded stock pests common throughout the southern United States.

Length 12–14 mm. Palpi yellowish, antennae brownish with the annulate portion darker; thorax including the scutellum uniformly grayish yellow pollinose; legs largely black, base of front tibiae and the middle and hind tibiae except at apex yellowish; wings hyaline with the costal cells yellowish, veins yellowish; abdomen above alternately striped with black and grayish yellow. In the female the frontal callosity black above, with a very much narrowed prolongation, the part of which adjacent to the callosity is sometimes obliterated, leaving the upper part as a separate spot. The male is much like the female and easily associated with it, but there is a tendency toward obliteration of the distinct markings of the abdomen, the black of the female is replaced by brownish and the stripes may blend so that the whole base of the abdomen is practically one color.

(Hine, *loc. cit.*)

5. *Tabanus lineola* Fabr., the lined horsefly, is also an important stock pest widely distributed in eastern, central, and southern North America.

Length 12–15 mm. Palpi white; antennae reddish, annulate portion of third segment darker; thorax brown and gray striped, the latter color not prominent; wings hyaline; legs reddish, apex of the front tibia plainly, apexes of middle and hind tibiae faintly, and all of the tarsi dark brown; abdomen above brown or black with three prominent, gray stripes. The males and females of this species are easily associated. In the latter sex there is sometimes a confusion of colors; the dark is replaced by reddish but the gray mid-dorsal stripe is always prominent in all well-preserved specimens.

(Hine)

6. *Tabanus sulcifrons* Macq. is known as the autumn horsefly.

Length 18–21 mm. Palpi brownish, antennae nearly black with the third segment brownish at base; legs dark, bases of tibiae darker; wings with a distinct brownish tinge, cross veins at the end of the discal cell and bifurcation of the third vein margined with brown. Female front with parallel sides, frontal callosity shining brown, not quite as wide at the front, nearly square and with a linear prolongation above. Segments of the abdomen above with prominent gray, hind margins which expand into large gray triangles in the middle; usually a black mark on the anterior part of each of the second and third segments at the apex of the gray triangle. In the males the division between the large and small facets of the eye prominent; head slightly more convex than in the female but nearly of the same size, coloration of the whole body the same as in the female.

(Hine)

7. *Tabanus striatus* Fabr. is said to be the most prevalent horsefly of the Philippine Islands, and is known to be an important carrier of surra. The following description is after Mayne (*loc. cit.*).

The male is very distinct from the female, being smaller and having a larger head and different color markings. Size: 14 to 15 millimeters. Wing expanse: 25 to 28 millimeters. The distinctly clavate palpi are shorter than in the female, only two-thirds as long as the labium; they are dirty white and fringed with moderately long black hairs. The abdominal color markings take the form of a T of pale cadmium yellow in a field of burnt sienna, bordered with pale clay yellow. The area of the large facets of the eye is colored Roman sepia surrounded by an elliptical band of ultra ash gray. The field of small facets has a mauve fringe bounding an area of iridescent mauve and Prussian green.

Female: The front is narrow, converges slightly anteriorly; the color is golden, marked with a black callosity of irregular form. Size: 15 to 17 millimeters. Wing expanse: 26.5 to 29 millimeters. The head is considerably smaller than that of the male; eyes iridescent mauve and Prussian green. The palpi are prominently conical, as long as, or slightly longer than, the labium; the color is the same as in the male, mottled with short black hairs. The abdomen is alternately striped with Cologne earth and pale clay yellow. The median stripe is pale clay yellow. In both sexes the thorax is indistinctly striped with pale clay yellow and pale brown, and the wings are transparent except the costal and subcostal cells, which are pale brown.

8. *Chrysops callida* O. S. is a widely distributed species, measuring from 7 to 9 mm, and is black in color with large pale yellow spots on the sides near the base of the abdomen.

9. *Chrysops celer* O. S. is black in color, the female with dense orange pile on the pleurae. It measures from 8 to 11 mm in length. It appears to have a more northerly distribution.

10. *Chrysops discalis* Williston is gray to yellow-gray in the female, with black spots on the abdomen; in the wing picture the hyaline discal cell and spot at the bifurcation of vein R_{4+5} are quite characteristic. In the male the color is predominately black with yellow-gray spots on the abdomen. Length 8 to 10.5 mm. It is reported from Utah, Nevada, California, Oregon, Washington, Nebraska, North Dakota, Wyoming, Montana, Colorado, also Manitoba and Saskatchewan.

11. *Chrysops dimidiata* v. d. Wulp is a southwest African species measuring 8.5 mm in length. The face is dusty brownish yellow, the thorax is piceous, and the abdomen is reddish ochraceous with a fulvous pilosity.

12. *Chrysozona americana* Osten Sacken occurs from central Alaska to New Mexico (type locality, Hudson Bay Territory) sharing generic characters:

Small, slender, greyish species; eye somewhat pilose; frons very broad and basal callus transverse; first antennal segment one and one-half to three times as long as wide and usually swollen; third with four divisions, the first division with no distinct angle above; wing brown or gray, with hyaline maculations . . . knob of haltere white or pale yellow.

(Stone)

*Key to the Tabanid Genera of Nearctic America**

(Arranged by T. H. G. Aitken after Brennan[23] and Stone.[5] Refer also to Surcouf.[24])

1. Hind tibiae with apical spurs; ocelli usually present............ Subfamily Pangoniinae (2)
   Hind tibiae without apical spurs; ocelli usually absent, if present rudimentary .... Subfamily Tabaninae (9)
2. Flagellum of antenna composed of eight annuli............ (3)
   Flagellum with five distinct annuli............ (7)
3. Second anal vein sinuous............ *Bequaertomyia* Brennan 1935
   Second anal vein not sinuous............ (4)
4. Eyes of female acutely angulate above; wings darkened anteriorly............ *Goniops* Aldrich 1892
   Eyes of female rounded (normal); wings of uniform color............ (5)
5. Palpi short, stubby, about equal in length to proboscis, which is conspicuously shorter than head............ *Apatolestes* Williston 1885
   Palpi slender, distinctly shorter than proboscis, which is often as long as or longer than head............ (6)
6. Cell R_5 petiolate............ *Esenbeckia* Rondani 1864
   Cell R_5 open............ *Stonemyia* Brennan 1935
7. (2) Pedicel of antenna about half as long as scape............ *Silvius* Meigen 1820
   Pedicel of antenna more than half as long as scape, often nearly as long .... (8)
8. Wings evenly infuscated; abdomen globose, much wider than thorax; antennae very slender and elongate; stump at bifurcation of vein R_{4+5}............ *Neochrysops* Walton 1918
   Wings irregularly infuscated, exhibiting a variety of patterns (entirely hyaline in *C. hyalina* Shannon); abdomen normal; antennae variable; bifurcation of vein R_{4+5} without stump (rarely appearing adventitiously)............ *Chrysops* Meigen 1803
9. (1) Scape of antenna considerably longer than thick; frons of female widened below, broader than high, with a velvety-black spot to each side at angle made by eye and subcallus;† flagellum of antenna with four annuli; wing gray, with small white maculations............ *Chrysozona* Meigen 1800
   Scape of antenna usually scarcely longer than thick; frons of female not broader than high, without velvety-black spots; flagellum of antenna usually with five annuli; wing pattern, if any, otherwise...... (10)

* Philip[25] has omitted the genus *Bequaertomyia* from his recent catalog of Nearctic Tabanidae, based on the elevation by Mackerras and Fuller (1942) of this primitive group of flies to family rank (Pelecorhynchidae) in which *Bequaertomyia* appears.

† That part of the frons below the level of the lower, inner angle of the eye and above the antennae is termed the *subcallus;* the frons proper (just dorsad) usually possesses two denuded calli, one at the lower margin, the *basal callus,* and the *median callus,* usually narrow and frequently broadly joined to the *basal callus.*

10. Eye bare; subcallus very swollen and shiny; genae denuded; dorsal angle of flagellum small and blunt; wing at least partially infuscated .... (11)
Without above combination of characters; if the subcallus is enlarged and denuded, the eye is densely pilose .... (12)
11. Scape of antenna swollen, at least below; apical half of vein R_4 turned abruptly forward; wing, at least anteriorly, infuscated with a cresent-shaped, hyaline apex; tibiae somewhat swollen .... *Bolbodimyia* Bigot 1892
Scape of antenna not noticeably swollen; vein R_4 not turned abruptly forward; apex of wing not hyaline; tibiae not swollen .... *Whitneyomyia* Bequaert 1933
12. (10) Flagellum of antenna with no dorsal angle; frons of female very narrow, the median callus a very slender line; no ocellar tubercle; wing with at least a subapical brown spot; eye bare .... *Diachlorus* Osten Sacken 1876
Not with this combination of characters .... (13)
13. Basal portion of antennal flagellum with a prominent, forward-projecting tooth reaching nearly to base of annulate portion; eye pilose .... *Dicladocera* Lutz 1909
Basal portion of antennal flagellum with or without a prominent dorsal angle, but if this is produced forward the eye is bare .... (14)
14. Basal callus in female lacking or very much reduced, separated from eye by a considerable space; neither palpus black nor abdomen with a narrow dorsal stripe .... (15)
Basal callus in female as wide, or nearly as wide, as frons, or, if narrowed, still considerably wider than median callus; either palpus black or abdomen with a narrow dorsal stripe .... (17)
15. Eye distinctly pilose; no distinct ocellar tubercle (eye of female usually with a single, diagonal, purple line which often shows even in dried specimens) .... *Atylotus* Osten Sacken 1876
Eye bare or very sparsely pilose; ocellar tubercle present or absent (frons of female about five times as high as width at base) .... (16)
16. Basal callus a swelling at base of a slender raised ridge; a distinct ocellar tubercle present in female; abdomen brownish, with white bands, the apex compressed .... *Leucotabanus* Lutz 1913
No basal or median calli or ocellar tubercle present; bright green or yellow species, the abdomen not distinctly compressed apically .... *Chlorotabanus* Lutz 1909
17. (14) Annulate portion of antennal flagellum hairy; no ocellar tubercle; second palpal segment short and stout, with erect hair; proboscis short .... *Anacimas* Enderlein 1923
Not agreeing entirely with above, the hair of antennal flagellum very inconspicuous if present .... (18)
18. Rather small, species with bare or sparsely pilose eye, scarcely any angle, and no dorsal excision on flagellum of antenna, and frequently a stump vein from vein R_4 .... *Stenotabanus* Lutz 1913

Eye bare or pilose, but if a stump vein from vein R_4 is present either the dorsal angle of the antennal flagellum is distinct or the eye is densely pilose, or both............................*Tabanus* Linné 1758

B. SNIPE FLIES

Family Rhagionidae (Leptidae)

Snipe flies belong to the dipterous family Rhagionidae formerly known as Leptidae. The family comprises nonbloodsuckers as well as bloodsucking species. Leonard[26] characterizes the family as follows:

> Flies of moderate to large size usually more or less elongate and nearly bare to moderately pilose, rarely rather densely hairy, never, however, with distinct bristles. Males usually holoptic; more rarely dichoptic. Empodium pulvilliform, there being three pads of about equal size between the tarsal claws. (*Hilarimorpha* has no visible empodia and no discal cell but is usually referred to this family.) Squamae small or vestigial. Antennae extremely variable: (a) segments of flagellum distinct, sometimes as many as thirty in number; (b) the segments not more than eight in number; more closely applied, without style or arista; (c) fewer in number with a differentiated segmented style or arista, altogether not more than eight; (d) the third segment simple with or without a dorsal or terminal arista. Veins of the wings distinct, not crowded anteriorly; third longitudinal cell furcate; basal cells large; five posterior cells usually present.

One of the members of the family is the "worm lion," *Vermileo;* the larvae excavate pits in sand as do "ant lions" (*Myrmeleon*). Among the bloodsucking genera are *Atherix*, e.g., *A. longipes* Bell, a severe biter in Mexico; and *Symphoromyia.*

The genus *Symphoromyia*[27] "includes leptid flies with five posterior cells, the anal cell open; third antennal joint simple, rather deep vertically, attached above its middle, usually kidney-shaped (sometimes concave in profile below the arista, then not quite kidney-shaped); arista subapical; tibial spurs none in front, two in the middle, one behind, but often quite weak in males." The females of several species are vicious biters, behaving somewhat as do the tabanid flies belonging to the genus *Chrysops.* They alight on the exposed parts of the body quite silently and singly and inflict a sudden painful bite usually before their presence is known. Among the severe biters are *Symphoromyia atripes* Bigot, a western species measuring 5.3 to 8 mm in length, black, with reddish legs; *S. pachyceras* Williston, particularly a Pacific coast species, measuring 6 to 9 mm in length, wholly black except narrowly on the knees, the inner proboscis, and stems of the halteres which are yellow; *S. kincaidi* Aldrich, pile of the thorax and head black, of abdomen largely yellow, front and middle knees narrowly red, a Pacific coast species. S. *hirta* Johns. is shown in Figure 100.

The mouth parts of Symphoromyia evidently vary considerably. The biting forms have a prominent stout retractile labial sheath which closely ensheaths the functional chitinized piercing structures.

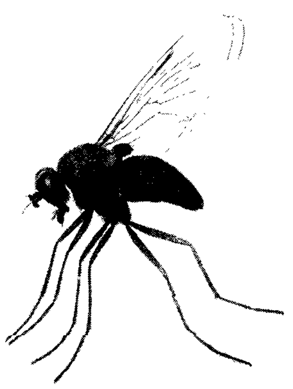


FIG. 100. A snipe fly, *Symphoromyia hirta*. (Adapted after Hearle.)

Practically nothing is known about the breeding habits and life history of the species of *Symphoromyia*. The rhagionids as a group are known to breed in moist soil, where there is decaying vegetation; the larvae are predaceous.

BIBLIOGRAPHY

1. Osten Sacken, C. R., 1875. *Prodrome of a Monograph of the Tabanidae of the United States.* Part I; Part II, 1876; Supplement, 1878, in Mem. Boston Soc. Nat. Hist., vol. 2, part iv, nos. 1, 4, 6.

2. Hine, J. S., 1904. "The Tabanidae of western United States and Canada," *Ohio Naturalist,* 5:217–48.

3. Enderlein, G., 1925. "Studien an blutsaugenden Insekten. I. Grundlagen eines neuen Systems der Tabaniden," *Mitt. a. d. Zool. Mus. Berlin,* 2:253–409 (5 text figures).

4. Kröber, O., 1927. "Die Chrysopsarten Afrikas," *Zoologische Jahrbücher. Abt. f. Syst., Ökol., u. Geogr. der Tiere.,* 53:175–268 (5 plates).

5. Stone, Alan, 1938. *The Horseflies of the Subfamily Tabaninae of the Nearctic Region.* Washington, D. C.: Dept. Agric., in Misc. Publ., no. 305. 171 pp.

6. Marchand, Werner, 1920. *The Early Stages of Tabanidae (Horseflies).* New York: Rockefeller Institute for Medical Research, in Monograph no. 13. 203 pp. + 15 plates.

7. Stone, Alan, 1930. "The bionomics of some Tabanidae (Diptera)," *Ann. Entomolog. Soc. Amer.,* 23:261–304.

8. Schwardt, H. A., 1936. *Horseflies of Arkansas.* Fayetteville: Univ. Arkansas, in Agric. Exper. Sta. Bull., no. 332. 66 pp.

9. Webb, J. L., and Wells, R. W., 1924. *Horseflies: Biologies and relation to western agriculture.* Washington, D. C.: Dept. Agric., in Bull., no. 1218. 36 pp.

10. Philip, C. B., 1928. "Methods of collecting and rearing the immature stages of Tabanidae," *J. Parasitol.*, 14:243–53.

11. Garman, H., 1910. *An Outbreak of Gadflies in Kentucky.* Kentucky Agric. Exper. Sta., in Bull., no. 151.

12. Mitzmain, M. B., 1914. "II. Summary of experiments in the transmission of anthrax by biting flies." Washington D. C.: Govt. Print. Office, in Public Health Bull., no. 94. pp. 41–48.

13. Yutuc, Lope M., 1938. "A report on the first outbreak of surra in British North Borneo and its control measure," *Philippine J. Animal Indust.*, 5:501–15 (4 plates).

14. Mitzmain, M. B., 1913. "The mechanical transmission of surra by *Tabanus striatus* Fabricius," *Philippine J. Sc.*, 8:223–29.

15. Basu, B. C., 1945. "Distribution and seasonal incidence of surra in India," *Indian J. Vet. Sc.*, 15:277–79.

16. Francis, Edward, 1919. "Deer fly fever or Pahvant Valley plague," U. S. Public Health Service, *Pub. Health Rep.* 34:2061–62.

17. ———; Mayne, Bruce; and Lake, G. C.; 1921. *Tularaemia Francis 1921: A New Disease of Man.* Washington, D. C.: U. S. Public Health Service, in Hygienic Laboratory Bull., no. 130. 87 pp.

18. Lillie, R. D., and Francis, Edward, 1936. *Pathologoy of Tularaemia.* Washington, D. C.: U. S. Public Health Service, in Nat. Inst. Health Bull., no. 167. 217 pp.

19. Connall, A., and Connall, S. L. M., 1922. "The development of *Loa loa* (Guyot) in *Chrysops silacea* (Austen) and in *Chrysops dimidiata* (van der Wulp)," *Tr. Roy. Soc. Trop. Med. & Hyg.*, 16:64–89.

20. Howard, L. O., 1899. *A Remedy for Gadflies. Porchinski's Recent Discovery in Russia, with Some American Observations.* Washington, D. C.: Dept. Agric., in Div. of Entomol. Bull., no. 20, n. s.

21. Herms, W. B., 1927. "Tabanids breeding in rice fields." *Pan-Pacific Entomologist,* 4:91–92.

22. Parman, D. C., 1928. *Experimental Dissemination of the Tabanid Egg Parasite, Phanurus emersoni Girault, and Biological Notes on the Species.* Washington, D. C.: Dept. Agric. in Circ. no. 18. 6 pp.

23. Brennan, J. M., 1935. "The Pangoniinae of Nearctic America. Diptera: Tabanidae," *Univ. Kansas Sc. Bull.*, 22:249–402.

24. Surcouf, J., 1921. *Diptera, family Tabanidae.* Brussels: *Genera Insectorum de P. Wytsman,* Fasc. 175.

25. Philip, Cornelius B., 1947. "A catalog of the bloodsucking fly family Tabanidae (horseflies and deerflies) of the Nearctic region north of Mexico," *Amer. Midland Naturalist,* 37:257–324.

26. Leonard, M. D., 1930. *A Revision of the Dipterous Family Rhagionidae (Leptidae) in the United States and Canada,* in Mem. Amer. Entomolog. Soc., No. 7. 181 pp. (3 plates).

27. Aldrich, J. M., 1915. "The dipterous genus *Symphoromyia* in North America," *Proc. U. S. Nat. Mus.,* 49:113–42.

CHAPTER XV

HOUSEFLIES

ORDER DIPTERA, SUPERFAMILY MUSCOIDEA

House-invading flies. Many species of robust flies belonging to various families of Diptera are commonly found indoors; however, relatively few of these species are of public health importance. Flies which habitually enter the house, coming in contact with household food or drink, and breeding normally in excrement and dead animal matter or feeding on these, are a potential menace to the public health. Flies of this character usually belong to the Muscidae, Anthomyidae, Sarcophagidae (flesh flies), and Calliphoridae (blowflies). Curran (1934, *loc. cit.* p. 143) has combined the Anthomyidae with the Muscidae and places the flesh flies (Sarcophagidae) and blowflies (Calliphoridae), in the family Metopiidae, which he designates as the flesh flies. The blowflies include the bluebottle and the greenbottle flies, which deposit their eggs on dead animals or garbage, also upon cold meat and other foods of man, which when ingested may cause intestinal disturbances. The flesh flies deposit living young (larviposit).

Family Muscidae. The family Muscidae, to which the true housefly and several other house-invading species belong, is characterized by Curran (1934, *loc. cit.*) as including "flies of medium to small size, usually dull colored, with the squamae large or of medium size, the hypopleural bristles absent; the second antennal segment grooved above. Arista plumose, pubescent, bare or pectinate, eyes approximate or widely separated in the males, the front rarely narrowed in both sexes; frontal bristles always present, intrafrontals frequently present; orbitals developed but rarely in the males. Abdomen composed of four segments in the male, five in the female. Male genitalia usually not prominent but sometimes conspicuous; fifth sternal lobes sometimes prominent."

The true housefly. Hewitt's[1] description (translation from Schiner) of *Musca domestica* Linn. (Fig. 101), the common housefly, is undoubtedly the best for our purpose:

Frons of male occupying a fourth part of the breadth of the head. Frontal stripe of female narrow in front, so broad behind that it entirely fills up the width of the frons. The dorsal region of the thorax dusty gray in color with four equally broad longitudinal stripes. Scutellum gray with black sides. The light regions of the abdomen yellowish, transparent, the darkest parts at least at the base of the ventral side yellow. The last segment and a dorsal line blackish brown. Seen from behind and against the light the whole abdomen shimmering yellow, and only on each side of the dorsal line on each segment a dull transverse band. The lower part of the face silky yellow, shot with blackish brown. Median stripe velvety black. Antennae brown. Palpi black. Legs blackish brown. Wings tinged with pale gray with yellowish base. The female has a broad velvety black, often reddishly shimmering, frontal stripe, which is not broader at the anterior end than the bases of the antennae, but becomes so very much broader above that the light dustiness of the sides is entirely obliterated, the abdomen gradually becoming darker. The shimmering areas of the separate segments generally brownish. All the other parts are the same as in the male. Mature insect 6–7 mm. in length, 13–15 mm. across the wings.

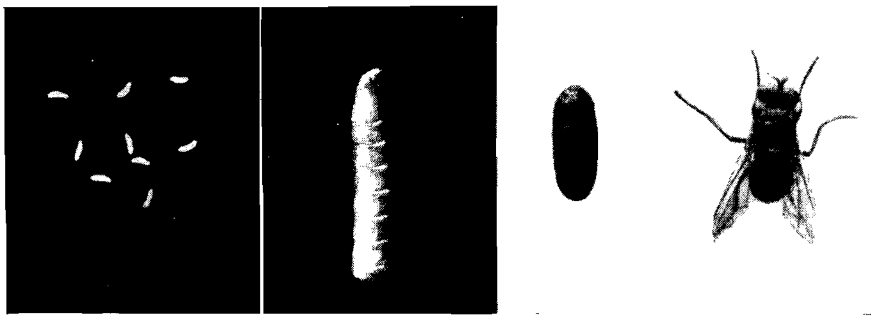


FIG. 101. Developmental stages of the common housefly, *Musca domestica:* egg, larva, pupa, and adult female.

Why called the housefly. Of a total of 23,087 flies collected by Howard[2] in dining rooms in different parts of the United States, 22,808 (98 per cent of the whole number) were *Musca domestica* Linn. Again, of a total of 294 flies collected by the writer, representing the entire fly population of one house, 202 (94.4 per cent) were *Musca domestica* Linn. Thus the term *common housefly* is not misapplied. It is furthermore a cosmopolitan species.

Distribution of sexes. In order to determine the distribution of the sexes of *Musca domestica* Linn. observations were made under two different conditions: first, six sweepings with an insect net were made over a pile of horse manure on which many flies had gathered (the results are shown in Table II); second, all but half a dozen flies were collected in one house, giving a fairly representative lot for indoors, even under screened conditions (Table III).

TABLE II

SHOWING RESULTS WITH REGARD TO SEX AND SPECIES IN SIX SWEEPINGS FROM A HORSE-MANURE PILE ON MAY 19, 1909

	First		*Second*		*Third*		*Fourth*		*Fifth*		*Sixth*		*Total*	
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
Housefly (*Musca domestica*)	7	153	4	81	3	64	9	77	4	210	5	112	32	697
Muscina sp.	2	6	0	7	0	5	2	5	3	10	1	4	8	37
Blowfly (*Calliphora* sp.)	2	2	0	1	1	0	0	0	0	0	1	0	4	3
Lucilia sp.	0	1	0	1	0	1	0	1	0	0	0	0	0	4
Other species	1	4	0	4	2	1	4	2	4	2	2	0	13	13
Totals	12	166	4	94	6	71	15	85	11	222	9	116	57	754

TABLE III

SHOWING NUMBER OF INDIVIDUALS COLLECTED IN A SCREENED DWELLING *JUNE* 1, 1909, *REPRESENTING THE ENTIRE FLY POPULATION OF THE HOUSE*

	♂	♀
Housefly (*Musca domestica*)	86	116
Muscina sp.	3	1
Homalomyia sp.	5	0
Calliphora sp.	1	2
Totals	95	119

These two tables give us some information as to the relative abundance of the housefly, and the distribution of the sexes. Table III shows clearly that of those flies which frequent both the manure pile and the home, the common housefly (*Musca domestica* Linn.) composes 90 per cent, and that of the total collected, over 95 per cent (95.4 per cent) were females. Thus, it is clear that it is the "instinct" to oviposit (to lay eggs) that mainly attracted these insects to the manure. In fact, fresher parts of the manure pile are often literally white with housefly eggs in countless numbers. Observations made in the near vicinity of the manure piles proved that certainly the same percentage (over 95 per cent) of the flies clinging to the walls of the stable, boxes, and so on were males.

That males and females are normally about equal in number is evidenced by the fact that of a total of 264 pupae collected indiscriminately and allowed to emerge in the laboratory, 129 were males and 135 were females.

Of the total number of houseflies (202) collected indoors (June, 1909), representing all but perhaps six of the total number in that particular house, 57 per cent were females, showing nearly equal distribution for the sexes. This would, it seems, indicate that males and females are equally attracted to the house by odors issuing therefrom.

Life history of the housefly. The housefly passes through a complex metamorphosis (Fig. 101) i.e., egg, larva (maggot), pupa, and adult or fully winged insect. Under warm summer temperatures the egg stage requires about 20 hours, the larval stage about 5 days, the pupa about 4 days, a total of about 10 days from egg to adult insect. This allows for the development of from 10 to 12 generations in one summer.

From 75 to 150 eggs are deposited singly, piling up in masses, and there are usually several such layings at intervals of three or four days. Female flies begin depositing eggs from 9 to 12 days after emerging from the pupa case. Dunn,[3] entomologist, Board of Health Laboratory, Ancon, Canal Zone, reporting on his observations, states that as many as 159 eggs may be deposited in one batch, that large batches are sometimes deposited at intervals of but 36 hours, and that one female may deposit as many as 21 batches, or a total of 2,387 eggs, in 31 days after emergence. He also states that oviposition may take place as early as 2¼ days after emergence, and that copulation may occur within 24 hours after emergence and one successful copulation seems to be sufficient to fertilize the female for her lifetime. Under our laboratory conditions houseflies reach sexual maturity in three or four days and begin depositing eggs on the ninth day after emergence from the puparium. Sunshine stimulates their breeding habits. Egg laying may continue throughout the lifetime of a fly, i.e., for more than two months.

Influence of temperature on life history. While conducting an extensive series of experiments in which many hundreds of houseflies were used in all stages, a record was made of the temperature at which the containers were kept. Ordinarily not more than one to three quarts of manure were used for the growing maggots, hence the temperature of the environment did not differ widely from that of the manure. The temperature of an average manure pile to which material is added daily varies from 18° C to 66° C. Young growing larvae are most numerous at temperatures varying from 45° to 55°. Below 45° half-grown and full-grown larvae occur; above 55° the temperature seems to become too great.

From the following table (Table IV) it will be seen that temperature influences very materially the time required for the development from egg to imago, but nevertheless with an average outdoor temperature of 18° C flies ordinarily require only from 12 to 14 days to pass through the same stages; this is, of course, due to the higher temperature of the manure pile, as indicated above. The shortest time required for complete metamorphosis of *Musca domestica* Linn. is seen to be 9⅓ days, and that at 30° C.

TABLE IV

SHOWING INFLUENCE OF TEMPERATURE ON THE LENGTH OF LIFE HISTORY OF MUSCA DOMESTICA

The insects were kept at the temperature indicated from egg to emergence of the imago. The average temperature is here given, the variation from the average was probably not more than ± 1°. Temperature of the air and not of the manure is here considered.

	16° C		18° C		20° C		25° C		30° C	
	Min.	*Max.*	*Min.*	*Max.*	*Min.*	*Max.*	*Min.*	*Max.*	*Min.*	*Max.*
Egg stage	36 hrs.	40 hrs.	27 hrs.	30 hrs.	20 hrs.	30 hrs.	12 hrs.	20 hrs.	8 hrs.	12 hrs
Larval stage	11 ds.	26 ds.	10 ds.	14 ds.	8 ds.	10 ds.	7 ds.	8 ds.	5 ds.	6 ds.
Pupal stage	18 ds.	21 ds.	12 ds.	15 ds.	10 ds.	11 ds.	7 ds.	9 ds.	4 ds.	5 ds.
Total time required from egg to imago	40½ ds.	48⅔ ds.	23⅛ ds.	30¼ ds.	18⅚ ds.	22¼ ds.	14½ ds.	17⅚ ds.	9⅓ ds.	11½ ds.
Average time required to develop from egg to imago	44.8 days		26.7 days		20.5 days		16.1 days		10.4 days	

Excrement the preferred breeding place. Excrement, especially of horses (Fig. 102), is the material upon which *Musca domestica* Linn. prefers to deposit its eggs, and this is the material on which the larvae feed. It seems quite safe to say that under rural conditions 95 per cent of the houseflies are bred in horse manure. The eggs of the housefly hatch in from 12 to 24 hours; the newly hatched larvae begin feeding at once and grow rapidly.

To gain an estimate of the number of larvae developing in an average horse-manure pile, samples were taken after four days' exposure to flies, with the following results: first sample (4 lb) contained 6,873 larvae; second sample (4 lb), 1,142; third sample (4 lb), 1,585; fourth sample (3 lb), 682; total 10,282 larvae in 15 pounds. All of the larvae were quite or nearly full grown. This gives an average of 685 larvae per pound. The weight of the entire pile was estimated at not less than 1,000 pounds, of which certainly two-thirds was infested. A little arithmetic gives us the astonishing estimate of 455,525 larvae (685 x 665), or in round numbers 450,000.[4]

The larval stage is the growing period of the fly, and the size of the adult will depend entirely upon the growth that the larva attains. An underfed larva will result in an undersized adult. The growing stage requires from four to six days, after which the maggots usually crawl away from their breeding place, many of them burrowing into the loose ground just beneath the manure pile, or under boards or stones, or into dry manure collected under platforms and the like. One and three-quarter pounds of dry manure, taken from beneath a platform, contained 2,561 pupae. The larvae spend three or four days in the prepupal or migratory stage before actually pupating. In a given set of individuals under similar conditions the various stages are remarkably similar in duration: when one pupates, the rest will certainly follow in short order, and when one emerges, the others quickly follow.

When the fly emerges from the pupa case the wings are folded in tight pads, and change in size is due only to expansion and addition in weight and not in growth. Stomach contents or development of eggs in the female add to weight. This is why no young houseflies are seen,

FIG. 102. A typical rural fly-breeding place—the manure pile. The principal menace is the fresh, warm manure added on top daily. The nearby vehicle is a remnant of the "horse and buggy days." The manure pile is far from obsolete.

i.e., young in the sense of being small. Little flies are not "baby" flies; they are either a different and smaller species or are undersized. One can influence the size of the adult fly by underfeeding it in the larval stage (see Herms,[5] 1907).

With one adult fly depositing from 120 to 150 eggs per lot, with at least six lots at intervals of from three to four days, Hodge[6] gives us the following astounding statement: "A pair of flies beginning operations in April may be progenitors, if all were to live, of 191,010,000,000,000,000,000 flies by August. Allowing one-eighth of a cubic inch to a fly, this number would cover the earth 47 feet deep."

Other breeding places. While horse manure is a favorite larval food and is commonly regarded as the chief factor in the production of houseflies under most rural and village conditions, situations may arise in which other materials are vastly more important, particularly in this day of the automobile and tractor. Cow manure if well mixed with bedding is frequently an important factor in the development of flies. Flies will also breed freely in hog manure, but the swarms of flies about the pig pens usually originate in the waste feed, slops, etc. Chicken manure is the most important factor in the breeding of flies in poultry districts, and the pest of blowflies in such areas is the result of dead birds buried in shallow pits or simply disposed of by throwing them into a gully or in a corner. The dead birds (large or small) should be burned or buried with crude oil or plenty of lime, or sprayed with creosote oil. Human excrement is a very dangerous substance, and if exposed to flies in open privies becomes a very prolific breeding place, which emphasizes the need of flyproof privies or the application of other means to prevent flies from breeding.

Great swarms of flies are often found around feed troughs; the animals (hogs and cattle) may be literally covered with them. An examination of the waste feed behind or beneath the troughs or in and about the mixing vats will almost invariably reveal numerous maggots. Storage receptacles for slops sometimes present a wriggling mass of maggots. The correction of such fly breeding manifestly depends upon greater care in handling the mash, wet or dry. Spraying fences, walls, and floors with DDT is also good practice.

It frequently happens that brewer's grain or spent hops, bran mash, and ensilage are only partly consumed by the animals and the waste is thrown out into the fields in heaps. Such heaps of waste are commonly a source of enormous numbers of flies (nearly all *Musca domestica*) about dairies where otherwise conditions may be very good and where no apparent reason for the swarms of flies exists. Such wastes should be scattered in a thin layer so as to hasten drying and thus prevent fly breeding.

Garbage heaps, particularly when fermentation and decomposition begin, are commonly sources of many flies of several kinds. Heaps of decaying onions and other vegetables, fruits, etc., as well as decaying straw and weeds, may become infested with maggots, often the larvae of the biting stable flies (*Stomoxys calcitrans*). Every household in every community should be provided with a garbage can equipped with a tight-fitting lid. All liquid matter should be drained from the kitchen refuse at the sink; only solids should be placed in the garbage can, and these should first be wrapped amply in paper. Household garbage should not be buried or allowed to accumulate in heaps in back yard or alley (Fig. 103). Rats as well as flies thrive in such an environment.

In the country, in the absence of sewers or septic tanks, the dishwater from the kitchen is frequently piped from the sink to a ditch in the back yard. On many occasions these ditches become clogged and vile smelling, and an examination will reveal numerous maggots developing in the muck—a source of flies which is commonly overlooked.

The writer has often seen septic tanks with open cracks and knot holes which permit flies to enter and lay eggs. Maggots have been found in countless numbers in the soft sludge mat covering the liquid in the defective tank. Crevices and knotholes are easily covered with tin, thus

FIG. 103. A poor excuse for a fly-tight garbage can. Care of garbage should be regulated by ordinance.

preventing ingress and egress for flies and eliminating breeding within the tanks.

Range of flight. Ordinarily under city conditions it may be safely said that where flies are abundant they have been bred in the immediate vicinity. The housefly can, however, use its wings effectively and may also be carried by the wind, though it usually seeks protection very quickly when there is a strong breeze. Where houses are situated close together flies have the opportunity to travel considerable distances by easy flights and they are often carried on meat delivery wagons and on animals.

In an illuminating experiment Copeman *et al.*[7] have shown that houseflies may invade a community at a distance of from 300 yards to 17,000 yards from their breeding place, such as a garbage dump.

In a city in Montana 387,877 marked flies were liberated from a release point, and a total of 1,056 of them were recaptured at 78 stations situated between 50 and 3,500 yards from the point of release.[8]

Longevity of flies. In order to determine the longevity of flies it is necessary to keep the same individual under observation from the time of emergence from the pupa to the time of death. The writer has done this by keeping each pupa in a separate vial, noting the time of emergence to the hour and spotting each fly lightly with Chinese white dorsally on the thorax. The spots can be arranged singly and in combination so that many different flies can be kept under observation at the same time. After marking, flies were liberated in bobbinet-covered cages (size of cages never more than 8 by 10 by 18 inches). Each cage was provided with sugar water and a receptacle of horse manure. A full set of experiments under sufficiently varying conditions indicate an average life of close to 30 days, with a maximum life of something over 60 days during the summer months. In hibernation flies may live over winter, i.e., from October to April, in the eastern and central United States. In California, flies emerge from their pupa cases throughout the winter, and their life history is then considerably longer than in summer.

Other house-invading flies. There are many species (more than 30) belonging to the genus *Musca,* most of which have habits similar to those of the cosmopolitan *M. domestica,* namely *M. vicina* Macq. the common housefly of the Orient, also common in Hawaii; *M. vetustissima* Walker, common in Australia; *M. nebulo* Wied., a tropical species; *M. sorbens* Wied., reported to be abundant on the central and south Pacific Islands.

Fannia (*Homalomyia*) *canicularis* (Linn.), known as the *lesser housefly,* is frequently seen hovering in mid-air or flying hither and thither in the middle of the room. Whereas the true housefly is encountered most abundantly in the kitchen or dining room, particularly on food, the "little housefly" will be seen as frequently in one room as another, and very seldom actually on the "spread" table. The writer commonly observes a half dozen or more of these little flies dancing weirdly in the center of the lecture room midway between the floor and the ceiling. Various observers have estimated that this species constitute from one to 25 per cent of the total population of flies in the average house.

In size the species varies from 5 to 6 mm. Its color is grayish, resembling that of the housefly very closely. Hewitt describes it as follows:

> Head iridescent black, silvery white, especially around the eyes. The antennae are blackish gray with non-setose arista. Palps black. The thorax is blackish gray with three indistinct black longitudinal stripes; the scutellum

is gray and bears long setae; the sides of the thorax are lighter. . . . The legs are black, and the middle femora bear comb-like setae below. The somewhat large squamae at the bases of the wings are white and the halteres are yellow. . . . The head of the female is gray with a wide frons, black frontal stripe and gray sides. The longitudinal stripes of the thorax are faint and the abdomen, which is more pyriform than that of the male, has a slightly golden attachment.

The eggs of this species are deposited on decaying vegetable matter and excrement, particularly of humans, horses, and cows. The larvae emerge in about 24 hours and may be recognized as compressed, spiny organisms about 6 mm long when full grown (Fig. 104). The pupal period lasts about seven days under favorable conditions.

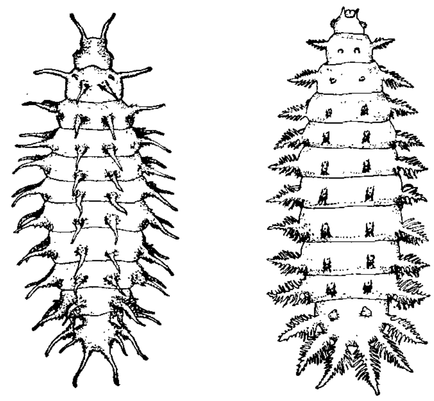


FIG. 104. (*A*) larva of *Fannia* (= *Homalomyia*) *canicularis,* (*B*) larva of *Fannia* (= *Homalomyia*) *scalaris.* ×6. (Redrawn and adapted after Hewitt.)

Fannia (= *Homalomyia*) *scalaris* (Fabr.), the latrine fly, is very similar to the foregoing. In size the two flies are about the same, if anything the latrine fly is somewhat the larger. The thorax and abdomen are bluish black; the antennae, palpi, and legs are black. The abdomen has a dark median stripe which, with segmentally arranged transverse bands, produces a series of dorsal triangular markings. The middle tibia is provided with a distinct tubercle.

The eggs of this fly are deposited on excrement of humans, horses, cows, etc., also on decaying vegetable matter. The egg stage lasts about 24 hours, the larval stage about six days and over, and the pupal stage about nine days.

While the larva of the "latrine fly" resembles the larva of the lesser housefly in general, it is readily distinguished because its single lateral protuberances are distinctly feathered (Fig. 104).

Other species of *Fannia* with similar breeding habits, referred to as "Old World" species but now widely distributed are *F. manicata* (Meig.) and *F. incisurata* (Zett.). The genus belongs to the family *Anthomyiidae.*

Musca autumnalis DeGeer (formerly called *Musca corvina* Fabr.) is described by Austen[9] whose valuable brochure on the "'housefly" should be read by all sanitary officers interested in housefly problems. He says:

. . . though agreeing approximately with the housefly in length, is a bulkier, more compactly built and thick-set insect . . . often decidedly larger. In the male the upper surface of the abdomen has a black base, from which there is a backward prolongation in the shape of a longitudinal, median stripe, both base and stripe being sharply defined, and presenting a well marked contrast to the cinnamon-buff of the remaining ground-color; in the female the upper surface of both thorax and abdomen is grey, with darker markings. In the case of both sexes, however, the surest criteria for distinguishing *Musca autumnalis* from *M. domestica* are those presented by the upper surface of the head. Whereas in the male *Musca autumnalis* the eyes are so close together as to be almost or actually in contact at one spot, in the male housefly the space between the eyes is always much broader, and, as already indicated, may be nearly equal to one-fourth of the total width of the head. As regards the opposite sex, in the female of *Musca autumnalis* the black longitudinal area (frontal stripe) in the center of the space between the eyes is approximately equal in width to the grey border on each side, separating it from the corresponding eye. In the female housefly, however, the frontal stripe is much broader, and its width greatly exceeds that of the border, yellowish-golden in front and below, blackish above, on each side of it. The resting position of the wings in *Musca autumnalis* is the same as in *M. domestica*. In the autumn, in country districts in the British Islands, *Musca autumnalis* frequently enters houses and public buildings, sometimes in large numbers, and subsequently hibernates in attics, roof-lofts, towers, in the folds of curtains in disused rooms, and in similar retreats.

Major Austen reports that it breeds in cattle droppings scattered in the field.

Muscina stabulans (Fallen) is larger and more robust than the true housefly, varying in length from 7 to nearly 10 mm. Its general appearance is dark gray. The head is whitish gray, the antennal arista bears setae on both the upper and lower sides. The thorax is gray with four longitudinal black lines: the abdomen is almost black in color, covered with gray in places, giving it a blotched appearance. The legs are slender and are reddish gold or cinnamon in color. The wings are folded like those of *Musca domestica* Linn.; the "fourth longitudinal vein is not elbowed and converges but slightly towards that of the vein before it" (Austen). The eggs of this species are laid upon decaying organic matter and excrement, inclusive of human feces and rotting cow dung, in which the larvae develop. The complete life cycle is said to require from five to six weeks.

Pollenia rudis (Fabr.), the cluster fly, may be distinguished from all other houseflies in that the wings when not in use close over each other at the tips scissors-like. According to Austen it measures normally about one-third inch in length; it is thus as a rule a much larger insect than the true housefly. It is more heavily built and slower in its movements. Austen continues the description:

The upper surface of the dark greyish-olive middle region of the body (thorax) is clothed with a thick coat of fine, silky, recumbent, yellowish or golden-yellow hair, easily visible to the naked eye, and, though readily rubbed off, still recognisable with the aid of a lens even in a much damaged specimen. The iron grey upper surface of the posterior division of the body (abdomen) is mottled with shimmering metallic patches of lighter grey. (In 1908) Dr. D. Keilin, working in Paris, made the extraordinary discovery that the maggot of the cluster-fly is an internal parasite of a small earthworm (*Allolobophora chlorotica,* Sav.) [which like the fly itself, is exceedingly common and widely distributed in Europe, North America, and elsewhere]. The popular name of the insect (namely cluster fly) is due to the habit of this fly of clustering together, sometimes in very large numbers like a swarm of bees, when hibernating in houses or other buildings.

Blowflies and flesh flies. The blowflies, comprising the bluebottles and greenbottles, included in the family Calliphoridae[10] as well as the flesh flies (family Sarcophagidae of most authors[11]), are placed in the family Metopiidae by Curran who characterizes this family as follows:

Flies of medium to moderately small size, the abdomen usually dark and tessellate or metallic green or blue. Front in both sexes broad, usually somewhat narrowed in the males, rarely very narrow; vibrissae present; antennae long or short, the arista plumose, pubescent or bare. Abdomen composed of four segments in the males, the fifth short in the females; abdominal bristles usually strong, at least on the apical segments. Hypopleura with a row of bristles; post-scutellum developed only in *Mesembrinella.* Apical cell usually open, rarely closed and petiolate, usually ending far before the apex of the wing. . . . The absence of the post-scutellum distinguishes the family from the Tachinidae, while the presence of hypopleural bristles separates it from the Muscidae.

The larvae of the blowflies and flesh flies usually feed on dead animals, garbage, excrement, etc. They are principally scavenger in habit.

Calliphora. Among the several species of bluebottle flies (metallic blue in color) two are quite common, namely, *Calliphora vomitoria* (Linn.), having black genae with golden-red hairs, and *C. vicina* R. -D. [= *C. erythrocephala* (Meig.)], with fulvous genae and black hairs (Fig. 105). The eggs of these species hatch in from 6 to 48 hours; the growing larvae feed on the flesh for from three to nine days, and after attaining full growth leave the food and bury themselves in loose earth or debris. This period (prepupal period) lasts from two to seven days, commonly four, after which pupation takes place. The pupal period varies considerably according to temperature, lasting from 10 to 17 days, commonly 11 days. Thus the life history of the blowfly requires from 16 to 35 days, usually 22 days. The life of the adult is about 35 days on an average.[12]

Phaenicia (= *Lucilia*) *sericata* (Meig.) is of a yellow-green or cupreous-green metallic color varying from metallic blue and green to copper. It may occur indoors but is typically a scavenger. The palpi are yellow. There are usually three (occasionally four) postacrostical bristles present on each side. The second abdominal segment is devoid of marginal macrochaetae present in *L. sylvarum* Meig., which also has black palpi. At a temperature of 80° ± 2° F, with beef lung or fish as food,

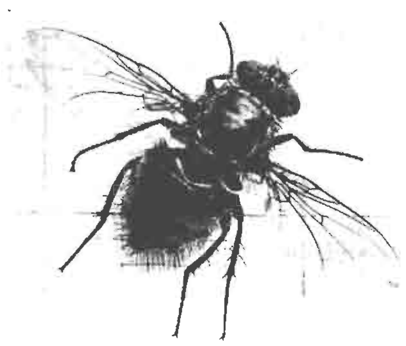


FIG. 105. A common blowfly, *Calliphora vomitoria.*

the entire life history of *Phaenicia sericata* (Meig.) from the deposition of the egg to emergence of the fly requires about 12 days: egg stage (the egg hatching the same day if deposited during early morning) about eight hours; larval stage (feeding period) about two and a half days; prepupal stage (migrating larvae) about three days; pupal period about six days. It is the most abundant species of the genus in North America, particularly in northern United States and southern Canada (Hall). *P. sericata* (Meig.) lends itself well to rearing in large numbers for experimental purposes. Rearing procedures are described by Dorman, Hale, and Hoskins.[13]

The size of the flies and the sex ratio[14] varies according to the amount of food available during the larval or feeding stage. The sex ratio of 2.8 to 3.1 males to 6.9 to 7.2 females for flies resulting from larvae which fed until they left the food voluntarily, i.e., from 72 to 78 hours, is reversed to 6.2 to 6.5 males to 3.5 to 3.8 females in flies in which the larvae were permitted to feed only 30 to 36 hours, i.e., were underfed.

Lucilia illustris (Meig.) is a holaractic species, widely distributed in North America; it is common in the midwestern portion of the United States where it ordinarily deposits its eggs on carcasses of animals in competition with *Phaenicia sericata* (Meig.). It appears to be an open woodland and meadow species. North American authors have heretofore not fully recognized the fact that they were pretty surely dealing with *L. illustris* and not with *Lucilia caesar* (Linn.) as they often stated. The two species resemble each other very closely. Hall (*loc. cit.*) states that *Lucilia caesar* (Linn.) does not occur in North America. The thorax of *L. illustris* is metallic blue-green with bronze and purple reflections; the legs are black; the normal number of lateral bristles in *Lucilia* is three.

Phormia regina (Meig.), known as the black blowfly, is a widely distributed holaractic species; it is found throughout the United States and as far south as Mexico City. It commonly deposits its eggs in the wool of sheep. It is said to be a cold-weather blowfly, occurring most abundantly during the early spring months and becomes less abundant as hot weather approaches. The thorax is black with a metallic bluish-green luster; there are darker black longitudinal stripes on the dorsum extending somewhat beyond the suture; the legs are shining black; the abdomen is olivaceous or bluish green to black, and shining; the length of the fly is 6 to 11 mm.

Sarcophaga haemorrhoidalis (Fall.), one of the numerous species of flesh flies, occurs throughout North America as well as Europe. It measures 10 to 14 mm in length; in color it is gray. The terminalia of the female are red. It reminds one of an overgrown housefly, but it is lighter gray, the eyes are brighter reddish brown in color, and it is larviparous. The larvae have a wide range of feeding habits, being, however, primarily scavengers. They feed on dead insects, carrion, mammalian excrement, etc.

The larvae may be deposited on the hand of a person holding the female fly. The life history in the presence of ample food and warm temperature requires from 14 to 18 days. The growth of the larvae is very rapid after extrusion when food such as carrion is available. The larval stage may be completed in about three days, followed by the prepupal or migratory stage lasting usually about three days. The pupal stage requires from 8 to 10 days. *Sarcophaga carnaria* (Linn.), measuring 10 to 16 mm in length, is another widely distributed and common species of flesh fly.

Germ carriers. The common housefly, *Musca domestica* Linn., is by accident of habit and structure an important and dangerous disease-transmitting insect. In habit the housefly is revoltingly filthy, feeding indiscriminately on excrement, vomit, and sputum, and is, on the other hand, equally attracted to the daintiest food of man. The housefly's proboscis is provided with a profusion of fine hairs which serve as collectors of germs and filth; the foot (Fig. 106) of the fly when examined under the microscope presents an astonishing complexity of structure. Each of the six feet is equally fitted with bristly structures and pads which secrete a sticky material, adding thus to their collecting ability. When the fly feeds it regurgitates droplets used in liquefying solid food, and extrudes droplets of excrement as well. Its structure, added to its natural vile habits, make the housefly an ideal transmitter of filth diseases.

The common housefly has long been known to contaminate food, but has, nevertheless, been regarded as a scavenger, and thus a beneficial

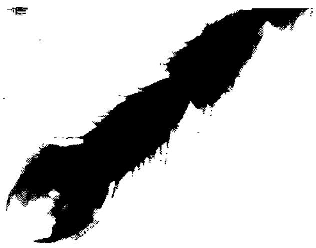


FIG. 106. Foot of the common housefly. (Much enlarged.)

insect; however, if there remains any doubt in the mind of the reader as to its harmfulness, after pondering what follows, let him take the time to make a few careful observations for himself.

In order to show that the housefly (*Musca domestica* Linn.) can carry "germs" of a known kind, a simple test can be made with a culture of *Staphylococcus aureus*. After the fly is allowed to walk about in the culture tube it is transferred to a sterile agar plate upon which it is permitted to walk for about three minutes. The plate is then incubated for 24 hours. Figure 107 shows the trail of the fly in one of our tests; every place that the foot touched is plainly marked by a vigorous bacterial growth.

Esten and Mason[15] in an article entitled "Sources of Bacteria in Milk" state:

> The numbers of bacteria on a single fly may range all the way from 550 to 6,600,000. Early in the fly season the numbers of bacteria on flies are comparatively very small, while later the numbers are comparatively very large. The place where flies live also determines largely the number that they carry. *The average for 414 flies was about one and one-fourth million bacteria on each.* It hardly seems possible for so small a bit of life to carry so large a number of organisms. The objectionable class coliaerogenes type was two and one-half times as abundant as the favorable acid type.

A significant study was made by Yao, Yuan, and Huie[16] in Peiping, China. This was based on a total of 384,193 flies, of which 98.4 per cent were *Musca domestica* Linn., 1.1 per cent *Fannia canicularis* (Linn.) and *F. scalaris* (Fabr.), 0.31 per cent *Lucilia caesar* (Linn.), 0.16 per cent *Calliphora vicina* R. D. and *C. vomitoria* (Linn.), and 0.03 per cent *Sarcophaga carnaria* (Linn.). They found an average of 3,683,000 bacteria per fly in the slum district, and an average of 1,941,000 for the cleanest district. They found eight to ten times as many bacteria inside the flies as on the outside.

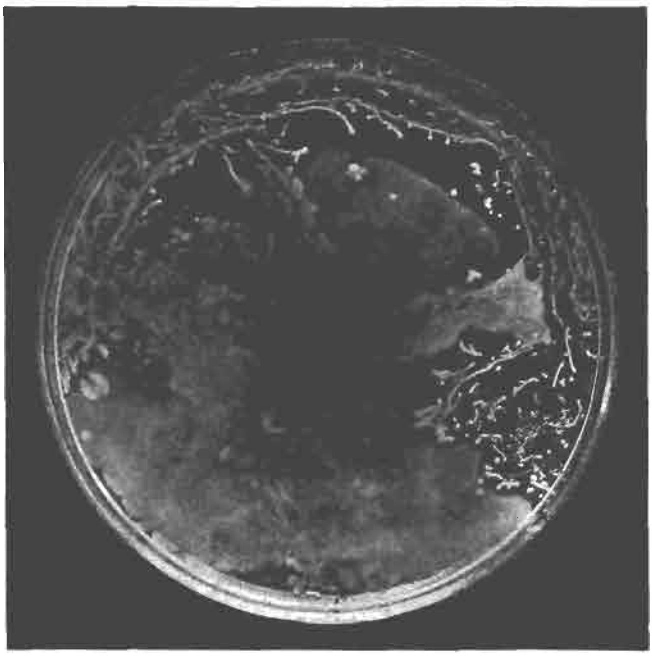


FIG. 107. Cultures of *Staphylococcus aureus* transferred by a housefly to a sterile agar plate upon which it was allowed to crawl for three minutes. Incubation period was 24 hours.

The fly usually acquires infection by walking over infectious materials, both its feet and wings becoming contaminated. The intestinal contents of flies also become charged with infection when feeding, and this is dejected in the fly "specks," and vomit droplets. It seems plausible that flies might become infected in the larval stage by developing in infectious fecal matter and that the newly emerged and unfed flies would be dangerous. Under experimental conditions Graham-Smith[17] has produced infected blowflies by feeding the larvae on meat infected with spores of *Bacillus anthracis*. He found that the blowflies remained heavily infected for at least two days after emerging and that the bacilli could be cultivated either from the legs and wings or intestinal contents of flies more than 15 or 19 days old.

The opportunity for flies to become infected is so great in all communities, even the most sanitary, that no fly should be trusted to alight on food prepared for human consumption. The following quotation is from Nuttall and Jepson.[18]

> It should be remembered that a fly may cause relatively gross infection of any food upon which it alights after having fed upon infective substances, be they typhoid, cholera or diarrhea stools. Not only is its exterior contaminated, but its intestine is charged with infective material in concentrated form which may be discharged undigested upon fresh food which it seeks. Consequently, the excrement voided by a single fly may contain a greater quantity of the infective agents than, for instance, a sample of infected water. In potential possibilities the droppings of one fly may, in certain circumstances, weigh in the balance as against buckets of water or of milk.

Leidy[19] in 1871 expressed an opinion that flies were probably a means of communicating disease to a greater degree than was generally suspected. From what he had observed in one of the large military hospitals, in which hospital gangrene had existed during the Civil War, he thought flies should be carefully excluded from wounds. It was, however, not until the Spanish-American War in 1898 that the real menace of the fly became evident as indicated by the following quotation from an article by Veeder in the Medical Record of September 17, 1898:

> To clinch the argument, and apparently to leave no loophole for escape, I have made cultures of bacteria from fly tracks and from the excrement of flies, and there seems to be not the slightest difficulty in so doing. Indeed the evidence of every sort is so clear that I have about reached the conclusion that the conveyance of infection in the manner indicated is the chief factor in decimating the army.

Gastrointestinal diseases. The housefly is primarily a food contaminator and vector of filth diseases because of its feeding and breeding habits, as already explained. Pathogenic organisms are collected on feet and mouth parts and ingested while feeding, then deposited mechanically while the fly is crawling on human food or deposited by regurgitation or with the fly's excrement.

Of the housefly's ability to transmit typhoid bacilli, Jordan[20] writes:

> Not only may bacilli stick to the legs and wings of these insects, but if swallowed they may survive the passage of the alimentary tract. Typhoid bacilli have been isolated from houseflies captured in houses in Chicago, in the neighborhood of badly kept privy vaults used by typhoid patients, and it has

been shown experimentally that living bacilli may remain in or upon the body of flies for as long as twenty-three days after infection.

Yao, Yuan, and Huie (*loc. cit.*) found *Shigella* (= *Bacillus*) *dysenteriae* (Shiga) in 15 of 50 batches of 100 flies tested. Alimentary tracts of flies also revealed cysts of *Endamoeba histolytica.* The specific death rate for gastrointestinal diseases roughly paralleled the number of flies captured.

Faichnie[21] (1929) in a study of the etiology of enteric fever came to the conclusion that comparatively little typhoid is carried on the feet of flies, but he found that both *E. typhosa* and *Salmonella paratyphi* (Kayser) (*B. paratyphosus* A) multiplied in the intestines of flies fed on infected excrement.

Cholera was among the first diseases in which the housefly was incriminated as a carrier. Tizzoni and Cattani in Bologna in 1886 isolated cholera vibrios from flies caught in cholera wards. Simmonds in 1892 captured flies in the post-mortem morgue in Hamburg and isolated cholera vibrios from these in large numbers. "Upon the surface of vegetables and fruits kept in a cool moist place, experiments have shown that the spirillum may retain its vitality for from four to seven days" (Jordan).

In their study of the epidemiology of cholera, Gill and Lal[22] found evidence to support the startling suggestion that possibly one phase of the life cycle of the cholera vibrio may be passed in the body of the housefly. The results of their work show that the vibrios disappeared from the body of the fly after about 24 hours but reappeared on or about the fifth day, at which time the fly was capable of infecting food by its feces.

Yaws (*Frambesia*) is caused by *Treponema pertenue* Castellani. The disease is widely distributed in the tropics. The spirochetes are found in the superficial ulcers on the hands, face, feet, and other parts of the body. The following statement by Nuttall and Jepson (1909, *loc. cit.*) presents evidence showing that *Musca domestica* Linn. is amply able to transmit this infection:

> Castellani (1907) tested the matter of the fly transmission of yaws by experimental methods. He allowed *M. domestica* to feed (1) upon yaws material (scraping from slightly ulcerated papules), and (2) upon semi-ulcerated papules on the skin of these yaws patients. In both cases he was able to discover the *Treponema (Spirochaeta) pertenuis* in microscopic preparations made from the flies' mouth parts and legs. Furthermore, he allowed *M. domestica* to feed on yaws material (1 and 2 as above) and afterwards transferred them to scarified areas upon the eyebrows of monkeys. Of 15 monkeys thus experimented upon, three developed yaws papules at the places which had been contaminated by the flies.

Ophthalmia. In commenting on ophthalmia as carried by flies, Howard (1911, *loc. cit.*) has the following to say:

Dr. Lucien Howe of Buffalo informed the writer [Howard] that in his opinion the ophthalmia of the Egyptians is also transferred by flies and presumably by the housefly, and referred the writer [Howard] to a paper which he read before the Seventh International Congress of Ophthalmology at Wiesbaden in 1888. He referred to the extraordinary prevalence of purulent ophthalmia among the natives up and down the river Nile and to the extraordinary abundance of the flies in that country. He spoke of their remarkable indifference to the visits of flies, not only children, but adults, allowing flies to settle in swarms about their eyes, sucking the secretions, and never making any attempt to drive them away. Doctor Howe called attention to the fact that the number of cases of the eye disease always increases when the flies are present in the greatest numbers and the eye trouble is most prevalent in the place where the flies are most numerous. In the desert, where flies are absent, eyes as a rule are unaffected. He made an examination of the flies captured upon diseased eyes and found on their feet bacteria which were similar to those found in the conjunctival secretions. Flies captured in Egypt swarming about the eyes of ophthalmia patients and sent to Washington, D. C., were identified as *Musca domestica.*

Poliomyelitis. Flies, biting and nonbiting, as well as mosquitoes have long been under suspicion as vectors of the virus of poliomyelitis. The virus of poliomyelitis has been isolated from human stools and sewage (Paul and Trask,[23] 1941). Various species of muscoid flies breed in excrement and sewage, as has been pointed out earlier in this chapter. The presence of the virus of poliomyelitis has been demonstrated in flies collected in the field during both urban and rural epidemics by various investigators. (Sabin and Ward,[24] 1941.) Sabin and Ward[25] (1942) state that there is no longer any doubt that flies can carry the virus. The virus was isolated from one of three batches of flies (in Atlanta), more than 95 per cent of which were *Musca domestica.* Isolation of the virus was made from 7 out of 12 batches of flies (collected in Cleveland, Ohio, where fresh meat was added to the banana and sugar); from 90 to 95 per cent of these flies were Calliphoridae (blowflies). Hall[10] who identified the flies points out that all of the positive samples of flies contained *Phaenicia* (= *Lucilia*) *sericata* (Meig.) and *Phormia regina* (Meig.). Just where and how these flies originally obtained the virus is an unsolved problem, as Hall remarks. Positive experimental evidence was secured by intraperitoneal inoculation into *Cynomolgus* monkeys of etherized fly extract; unetherized material was given both intranasally and by mouth. Precisely what role, if any, flies play in the *transmission* of poliomyelitis to human beings is not clear; that flies might serve as a link in the infection of animal reservoirs is of course not impossible.

Since it is known that muscoid flies may harbor the virus of poliomyelitis, every effort should be made to control flies by any means possible; be it by environmental sanitation or by the use of insecticides, which normally should be only supplemental to good sanitation. Fly control needs encouragement. However, in the face of evidence that poliomyelitis can be spread by human carriers and since evidence of transmission by flies is incomplete, fly campaigns recommended as a means of poliomyelitis control, particularly the community-wide spread of DDT or other insecticide by airplane, only creates false hope and false security. It must be borne in mind, too, that such treatment only affects the adult fly population; the residual effects are not likely to influence in any large measure the following broods of flies.

Tuberculosis. Investigations by Dr. Ch. André of the University of Lyons were reported at the Anti-Tuberculosis Congress at Washington, 1908, as follows:

> Flies are active agents in the dissemination of Koch's bacillus because they are constantly going back and forth between contagious sputa and feces, and foodstuffs, especially meat, fruit, milk, etc., which they pollute by contact with their feet, and especially with their excretions.
>
> The experimental researches of the author show the following:
>
> 1. Flies caught in the open air do not contain any acid-fast bacilli that could be mistaken for the bacillus of Koch.
> 2. Flies that have been fed on sputum evacuate considerable quantities of bacilli in their excretions. The bacilli appear six hours after ingestion of the sputum, and some may be found as long as five days later. These flies, therefore, have plenty of time to carry these bacilli to a great distance, and to contaminate food in houses apparently protected from contagion, because not inhabited by consumptives.
> 3. Food polluted by flies that have fed on sputa contains infective bacilli and produces tuberculosis in the guinea pig.
> 4. Flies readily absorb bacilli contained in dry dust.
> 5. Flies caught at random in a hospital ward produced tuberculosis in the guinea pig.

André's conclusions are: "The sputa and feces of tuberculosis subjects must be disinfected; flies should be destroyed as completely as possible; foodstuffs should be protected by means of covers made of wire gauze."

Eggs of parasitic worms. Extensive and careful work on the dispersal of eggs of parasitic worms by the housefly has been done by Nicoll.[26] The following is a summary of his investigations. Flies feed readily on excrement in which eggs from parasitic worms occur. Eggs may be conveyed by flies from excrement to food in two ways, namely on the external surface of the body and in the intestines. The latter is possible only when the diameter of the eggs is under .05 mm. Eggs with a diameter of up to .09 mm may be conveyed on the external surface; however, these adhering eggs are usually got rid of by the fly within a short time, while those harbored in the intestine may remain there for several days. It was found that material containing eggs of parasites, and in particular ripe segments of tapeworms, remain a source of infection through flies as long as two weeks.

The eggs of the following parasitic worms were shown experimentally to be capable of transmission by *Musca domestica* Linn.: *Taenia solium* Linn., *Taenia pisiformis* (Bloch), *Taenia hydatigena* Pallas, *Hymenolepis nana* (v. Siebold), *Dipylidium caninum* (Linn.), *Diphyllobothrium latum* (Linn.), *Enterobius vermicularis* (Linn.), *Trichocephalus* (= *Trichuris*) *trichiurus* (Linn.), both internally and externally, *Necator americanus* (Stiles), *Ancylostoma canium* (Ercolani), *Ascaris equorum* Goeze, *Toxascaris leonina* (v. Linstow), *Hymenolepis diminuta* (Rudolphi), externally only. No trematode parasites were experimented with, and the observations of Stiles that the larval fly can ingest ascarid eggs and pass them on to the adult fly was not confirmed.

Intestinal protozoa. Roubaud[27] found that the cysts of *Endamoeba coli* (Grassi), *Endamoeba histolytica* (Schaudinn), and *Giardia lamblia* Stiles passed through the intestine of the fly uninjured, and that free amebae (both *coli and histolytica*) when fed to flies were found dead in the fly's intestine in less than an hour. It is generally believed that the housefly plays an important role in the epidemiology of amebiasis through the spread of cysts of *Endamoeba histolytica.* Root[28] found motile *Chilomastix mesnili* (Wenyon) in a fly's feces seven minutes after it had fed on an infectious stool.

Murrina, a trypanosomiasis of horses, mules, and burros in Panama, is caused by *Trypanosoma hippicum* Darling. Darling[29] showed that the disease can be transmitted mechanically by houseflies by contact with fresh blood from an infected animal and thence to an open wound of another animal. The disease is, of course, transmitted in other ways, vampire bats playing an important role.

Bovine mastitis. Sanders (*loc. cit.*) reports investigations made at the Florida Agricultural Experiment Station which incriminated the houseflies (*Musca domestica*) as well as *Hippelates* flies as vectors of bovine mastitis. Houseflies (also *Hippelates*) were seen to alight at the natural openings of calves, yearlings, pregnant heifers, and lactating cows. They fed on lachrimal fluid, fatty body secretions, milk droplets accidentally spilled, and on secretion at the tip of the teats of animals in herds where mastitis prevailed. Exposure tests were made with flies feeding alternately on infected material and the teat orifice; also the teat orifice was exposed to flies taken directly from premises where mastitis prevailed. "Mastitis developed in each of the experimental animals by the exposure technique employed."

Cutaneous habronemiasis. An examination of certain persistent ulcerations, summer sores, on the lower portions of the bodies of horses (the sores have a tendency to disappear during colder weather) may reveal the presence of larval nematode worms belonging to the genus *Habrone-*

FIG. 108. Segment of intestine (inside out) of fowl infested with numerous tapeworms, *Choanotaenia infundibulum*.

ma measuring from 1 to 1.5 mm in length. The presence of these worms in sores on the eyes is termed *habronemic conjunctivitis*. Adults of *Habronema muscae* (Carter), measuring from 8 to 14 mm in length in the male and 13 to 22 mm in the female, occur in the stomach of the horse, where they lay their eggs, which pass out with the feces. The newly hatched larvae find their way into the bodies of fly larvae, which are evidently true intermediary hosts, and in these further development occurs. Flies resulting from these larvae may contain a number of the worms (from 1 to 20) in their bodies, often in the head. According to

Ransom[30] the infection of horses with *H. muscae* is apparently brought about by the swallowing of infected flies or infection by the worm larvae migrating from the insect as it feeds. The sores above-mentioned are evidently the result of larvae entering lesions while the animals are lying down in infested manure.

Fowl taeniasis. Domestic fowls are commonly infested with tapeworms and the extent to which this may occur is well illustrated in Figure 108. Several species of tapeworms inhabit the intestines of fowls. Although Grassi and Rovelli[31] as early as 1886 had made observations concerning the development of these parasites, it was not until 1916 that experimental evidence was published by Gutberlet,[32] proving the housefly (*Musca domestica* Linn.) to be an intermediate host.

The most important of the fowl tapeworms is *Choanotaenia infundibulum* (Bloch). It measures from 50 to 200 mm in length. The scolex is small and rounded, measuring about 0.4 mm in width. The rostellum is armed with a single row of 16 to 20 hooks. The cysts found in the housefly are oval in shape and measure about 200μ in length by 120μ in diameter. Gutberlet infected the adult flies by feeding them on liquids which were infested with tapeworm eggs, and it is assumed that fly larvae breeding in infested fowl droppings would become similarly infected. Reid and Ackert[33] in experiments with this tapeworm recovered proglottids in fowl feces at the end of the seventh week. These authors point out that buttermilk is very attractive to both flies and chickens and that chickens devour the flies eagerly.

Raillietina cesticillus (Molin), according to Gutberlet, also has the housefly as its host; and *Hymenolepis carioca* (Magal.) has *Stomoxys calcitrans* (Linn.), the stable fly as its host.

FLY CONTROL

Fly control. Effective fly control is based on a knowledge of the habits and life history of the particular offending species. First find the breeding places and then apply the appropriate remedy. The presence of flies always denotes defective sanitation, particularly in the disposal of manures, garbage, sewage, slops, food wastes, ensilage, brewer's grain, spent hops, wet mash, dead animals, etc. The prevention of fly breeding requires good housekeeping practices in a broad sense.

Rural fly control. Since the principal *rural* breeding places of houseflies are usually in and about barns and stables, particular attention must be paid to these as well as to barnyards and corrals, with special reference to the disposal of manures and the prevention of accumulations of manure in the stable. Concrete floors permit proper cleaning of stalls.

In constructing a concrete floor, provision must be made for carrying away the animals' urine and the water used in cleansing the floors and stalls; the stall floors should be given a one-inch drop from the manger to the manure gutter, which latter should be "6 inches deep and 14 inches wide. In order to facilitate the drainage of the liquids a 3-inch U-shaped channel is sometimes made in the bottom of the gutter next to the manure alley, but this is not necessary where a slope is given the gutter bottom. The gutter should be given a uniform fall of 3 inches to 100 feet, and the floor of the manure alley should have a slope towards the gutter of 1 inch to 10 feet. A small watertight liquid manure cistern may be provided outside the barn into which the gutter drains, but if a manure shed is used, the cistern should be in the shed. The gutter should be connected to the cistern by means of a drain pipe effectively trapped like the soil pipe in a house and so arranged that the trap may be easily cleaned."[34]

Concrete stall floors should be covered with wood to prevent animals from coming in direct contact with the concrete. The super-floor should be so made that it can be lifted while the concrete is being cleaned. If the crevices of the wood floor are not also frequently cleaned, fly larvae will develop in these also.

Manure wastage. Piling manure in a barnyard results in a loss of manurial value due to leaching and fermentation, estimated at from 25 to 50 per cent. The Cornell University Experiment Station has carried on investigations which show the loss of valuable plant food when manure is disposed of in the usual exposed manner for six months. When the manure was tested, it was found that the horse manure had lost 57 per cent and the cow manure 49 per cent in gross weight, and the loss in value based on plant food (nitrogen, phosphorus, and potassium) amounted to 65 per cent for the horse manure and 23 per cent for the cow manure. When manure wet with urine is thrown from the stable on to the heap it contains about 75 per cent water which holds most of the plant food. Exposed to leaching rains and weather the liquid sinks into the ground beneath or flows away. Thus not only does the barnyard manure pile result in flies but also in a loss to the farmer.

Manure disposal. Wherever manure is piled up and accessible to flies, these insects are afforded opportunity to breed. As stated before, it requires only about four days for the larvae to reach full growth, after which they begin to migrate into the drier portions of the heap and crawl out into nearby debris, beneath platforms, etc. It is therefore imperative, if fly breeding is to be prevented, that manure be properly handled at once.

Under ordinary rural conditions it would seem possible to remove the manure to the fields every two or three days. For this purpose a cart may be backed up against the stable doorway, and the manure may be thrown in, carted away to a field, and scattered. This saves much time in handling and is sound agricultural practice. Since moisture and warmth are both necessary for the production of fly larvae, the scattered manure cannot serve this purpose.

According to the Wisconsin Bulletin No. 221 "Manure is never so valuable as when perfectly fresh, for it is impossible under the best system of management to prevent all loss of its fertilizing ingredients. For this reason, whenever possible, the manure should be hauled directly to the field and spread. The system saves time and labor as it involves handling but once. The manure will be leached by the rain and snow, nevertheless the soluble portion will be carried into the soil, where it is needed. When spread in a thin layer, it will not heat, so there will be no loss from hot fermentation, and where manure simply dries out when spread on the ground, there is no loss of valuable constituents."

A manure spreader is a valuable part of farm equipment. Farmers and gardeners who wish to use "rotted" manure for fertilizing purposes should compost it; few flies breed in "rotted" manure. Fresh manure may also be placed in trenches and covered with lime and earth or it may be stored in fly-tight composting pits.

Composting pits. Composting pits are frequently maintained on country estates and truck gardens where quantities of rotted manure are used for fertilizing purposes. Such pits are usually made of concrete and covered with wood, all carefully constructed to exclude flies and mosquitoes, which latter may breed in the liquids collected in the sump. A properly constructed composting pit makes it possible to preserve the urine, which is very valuable in addition to the more solid excreta.

A cross section of a composting pit is shown in Figure 109. In this case a pump is indicated by means of which the urine and water are pumped out of the sump and returned to the manure from time to time. Water should be applied to the manure occasionally to prevent burning, which may destroy much of the value of the fertilizer.

The size of the pit depends also on the number of horses stabled. A pit such as is shown in the figure, with a length of 60 feet, ought to store the manure from 10 horses for a period of six months.

Close packing. The following description of close packing is from Austen (*loc. cit.*), who states that the essence of this method is the utilization of the natural heat of fermenting manure for the destruction of the eggs, maggots, and pupae (if present) of the housefly:

For *close packing* (a method introduced in 1915 by Lieut.—Colonel S. A. Monckton Copeman) an area of hard, level ground, at least three or four feet greater in extent each way than the ultimate size of the intended dump, must be selected or prepared to receive the manure. On this, each day's manure is utilised in forming or adding to a compact rectangular block, which may be of any desired dimensions horizontally, but for convenience of treat-

ment should not exceed five feet in height. Each load of manure on being added to the dump must be pressed down firmly with shovels, and if the weather be dry should be sprinkled slightly with water; finally the sides, which should be somewhat sloping, must be beaten and smoothed down with the shovel. . . . It was found by Colonel Copeman that, four inches beneath the surface of a heap of fresh stable manure treated in this way, the heat produced by fermentation may be as much as 169° F, though housefly maggots are speedily killed at temperatures only slightly above 114.8° F. It should

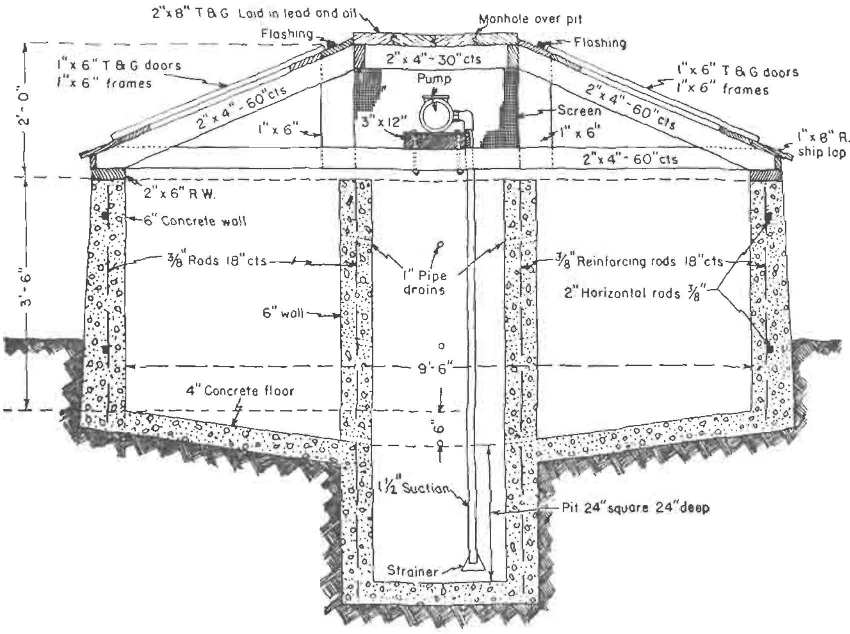


FIG. 109. Cross section of a concrete flyproof composting pit. (After Rosenthal in Advisory Pamphlet on Camp Sanitation and Housing, Commission of Immigration and Housing of California.)

be noted that the fertilizing value of *close packed* manure is greater than that of loosely stacked manure.

Although the success of close packing as a preventive of housefly breeding in horse-manure has been proved in England, the method has yet to be tested in warmer climates. In the event of failure from some unforeseen cause, the portion of the dump (both sides and top) in which maggots are seen should be covered over with a layer of sacking (old coal sacks, if without holes, answer well), soaked in heavy oil and secured by means of large stones; the sacking need only be allowed to remain for one week, after which if required it may be employed on another part of the dump.

Chemical treatment of manure. Although there is a strong popular demand for some chemical which, if applied to the manure, will result in fly prevention, the writer is not enthusiastic about this method of control for two reasons: *first*, this scheme may be used as a substitute for sanitation—cleaning up and keeping clean; and second, owing to the necessity for frequent application, there will certainly be neglect.

Ordinary applications of the insecticides are of little or no avail, because of the hardiness of fly larvae, and furthermore, the larvae cannot be reached easily, buried as they are in the bedding or manure.

Borax is recommended by the United States Department of Agriculture at the rate of 0.62 pound to 8 bushels of manure. The New Jersey Agricultural Experiment Station recommended the use of borax, stating:

> One ounce of borax to one cubic foot of manure will kill 90 per cent or more of the larvae and inhibit the eggs from hatching, provided it is applied in solution, and the larvae and eggs come in contact with it. It was figured out that the average output from one horse is one and one-half cubic feet of manure per day and that two and one-half gallons of water containing one and one-half ounces of borax should penetrate all parts of this amount of manure. In making treatments, it was noted that wet manure which had been soaked by rains, or when located in low wet areas, was difficult to treat, for it was next to impossible to make any additional water (by sprinkling) penetrate the wet manure. When manure is in this condition, particularly in low wet areas, and contains numerous larvae, it should be put on higher ground in order that it may dry out somewhat; or a thick layer of gravel or cinders placed under the manure in wet low areas will help considerably. Manure treated with borax is detrimental to some plants when the manure is used as a fertilizer, but, as far as known, it will not injure plants provided one does not use over 15 tons of treated manure (not over one pound of borax for every 16 cubic feet) to the acre.

As a practical matter 11 ounces of borax in 10 gallons of water is sufficient to treat the daily accumulation from 10 animals. Borax is an excellent chemical to use on dirt floors of stables, in privies, and on accumulations of decomposing matter which will not be used as fertilizer.

Dust has considerable value in fly control. The author found that very few flies bred in the manure from cavalry horses on duty during the summer in arid dusty regions when the manure was thoroughly mixed daily with road dust and disposed of in windrows a short distance from camp.

Use of DDT in rural fly control. Gunderson, extension entomologist, Iowa State College, who played an important role in the successful fly control campaign in Iowa ("Make Iowa Fly Free in 1947") sets up the following program of action:

1. Clean up fly breeding places, such as manure piles, hay and straw stack bottoms, weed piles, and other decaying plant and animal materials. Scatter on the fields every 3 days for greatest fertilizer value.

2. Treat outdoor toilets and manure piles with borax (11 ounces in 10 gallons of water on each day's accumulation for 10 animals). These places may be sprayed every 7 days with a 2½ per cent DDT spray to accomplish similar results.

3. Spray inside walls of an all farm out-buildings housing livestock or livestock products with 2½ per cent DDT (2 pounds 50 per cent DDT wettable powder in 5 gallons of water). Apply at the rate of one gallon per 1,000 square feet of surface. Dairymen should check with local milk inspector before applying this formula in the milk house. Some inspectors require DDT emulsion or oil spray in such places.

4. Spray cattle every two weeks or as often as necessary. Use one pound of 50 per cent DDT wettable spray powder in ten gallons of water for early sprays. Increase to two pounds of 50 per cent DDT wettable powder in five gallons of water when flies are found on legs and bellies of cows. Use 1 to 2 quarts of spray per animal.

Dairy barns. Michelbacher and Smith[35] (1946) report excellent results obtained with DDT in the control of flies in dairy barns. They recommend DDT water suspension sprays in preference to DDT-kerosene solution for several reasons: (1) Based on the actual amount of DDT used, water suspension sprays proved to be more effective, probably because kerosene carries part of the DDT into the sprayed surface where it is lost, while a water suspension spray deposits DDT on the surface. (2) Some fire hazard is involved where a kerosene spray is used, thus vitiating insurance contracts. (3) Water suspension is a much safer material to use since it is not harmful to man or animals if some of the spray is accidentally deposited on the skin. Water suspension sprays were used at the rate of 2 to 4 pounds of actual DDT to 100 gallons of water. The 4-pound dosage was preferred because of its longer lasting effect. It was applied, at pressures ranging from 100 to 400 pounds, to the interior surfaces of the barns, including alleyway, washroom, and milk-cooling room, also to ceiling and walls of the feed storage room provided the feedstuffs were protected from the spray. Frequently the calf barn and the bull pen were also treated. To do a thorough job 40 to 60 gallons of dilute spray were needed to treat a 24-cow barn. During the summer this treatment lasted for about six weeks; three to four spray applications a year are suggested. The following precautions must be taken before spraying begins: (1) All milk cans and utensils put under cover or removed; (2) milk coolers and sinks carefully covered with heavy paper or other material; (3) all feedstuffs removed or carefully covered to avoid wetting with spray; (4) feed troughs hosed clean just before and after the spray is applied in order to reduce spray residue which might collect in them. The authors point out that the butterfat production per cow increased from 394 to 429 pounds in dairies so treated; other factors may, of course, have played a part in bringing about this increase, but fly control was, no doubt, an important factor.

Feed wastes. The presence of numerous flies about the premises of a certified dairy, which upon inspection was found to be very clean and otherwise in excellent condition with no evident reason for the presence of the flies, was a matter of no small concern. The trouble was located in a field a few hundred feet from the dairy barn where numerous *heaps* of waste brewer's grain (used as cattle feed during late summer and autumn) had been hauled and dumped. Each heap was a veritable wriggling mass of housefly maggots. Liberal application of a larvicide (fuel oil in this case) to the heaps soon destroyed the source, and further waste feed was spread out in a thin layer to facilitate rapid drying and thus defeat fly breeding. The epidemic of flies was soon brought under control. An adulticidal spray such as DDT will quickly eradicate the infestation. Fly breeding about dairies is frequently traceable to accumulations of waste feed about the troughs, the waste becoming mixed with feces and urine. Thus bad conditions may arise in spite of the proper disposal of manure.

Community fly control. Among the earliest successful community-wide fly-control campaigns was one conducted by the author[4] in Berkeley, California, in 1909–1910 during the "horse-and-buggy" days when there was really a fly problem. The population of Berkeley was about 40,000 at that time. A letter from the then secretary of the Chamber of Commerce dated April 8, 1909, reads in part, " . . . it is my desire to enlist your aid for the relief of Berkeley. . . . This office is located between two livery stables that breed myriads of flies." The campaign was started at once with the full cooperation of all city officials and the Board of Health through the health officer. Much newspaper publicity was given to the work and the Board of Health distributed bulletins written by the author and entitled "Essentials of Housefly Control" and "Fight The Fly, Why, When and How." Twenty students from the class in medical entomology were officially appointed sanitary inspectors. A detailed house-to-house inspection was made to locate breeding places, and advice was given as to means of control. Police officers assisted when difficult situations arose. Although insecticides were used to some extent, the campaign was primarily based on sanitary procedures.

After a few years of community interest in housefly control there followed a lull in interest, perhaps due in large measure to conversion of livery stables into garages, a change over from the horse to the automobile. During the past three years there has been a widespread popular demand for fly-control programs, owing no doubt to the popular association of flies with the spread of poliomyelitis, and to the enthusiastic approval of the new miracle insect killer, DDT.

Rowe[36] of the United States Public Health Service, in a modern presentation (1948) points out that in the conduct of the most effective and economical community fly-control programs both preventive and insecticidal measures are essential. He states that too much emphasis cannot be placed upon preventive measures. These are incorporated in what are known as standard environmental sanitation practices. However, such practices must be specific and directed toward the abatement of all fly-breeding situations in so far as possible. In agricultural communities stables, corrals, and feed pens may be fly-breeding hot spots; the outdoor toilet (privy) may be a contributing factor; garbage and community garbage dumps may also contribute flies; these situations are all amenable to relatively simple sanitary and insecticidal procedures. However, according to Rowe, some of the most vexing fly problems in a community are created by the haphazard dumpage or the careless disposal of industrial end-products and wastes. Major fly-breeding foci are often created around stockyards, stock feeding pens in connection with beet-sugar refineries, abattoirs, hide and tallow plants, canneries, food and milk processing plants, et cetera. Vast numbers of flies—houseflies, blowflies, pomace flies, etc.—may be produced in the accumulated wastes and contaminated soil around the plants. Special study must be given to each situation and a practical remedy should be employed that is adaptable to each; the program should aim at elimination of the problem whenever possible rather than at insecticidal treatment. Fly problems are man-made.

DDT has been the insecticide of choice for community fly-control programs in the United States, primarily because of its residual value in the destruction of adult flies. The housefly lends itself particularly well to the lethal effect of residual DDT in that it spends much of its life resting or walking on surfaces which may be readily treated. The lethal dose of DDT for an average size housefly is a tenth of a microgram, according to Douglas.[37]

Three types of DDT sprays have been used, namely, emulsions, solutions, and wettable powder suspensions, with little difference in effectiveness. Because of the so-called "fire hazard" kerosene or other oil solutions are seldom used. For all-around uses, inside and outside, the emulsion type seems to be most suitable. The residual efficiency of emulsions in general is rather high as compared with solutions, and they leave little objectionable residue. There should be a minimum residue of 200 milligrams per square foot. Douglas (*loc. cit.*) believes 300 milligrams is better and in some situations even 400 to 500 milligrams is actually a desirable residue. He states that a 200-milligram residue has an effective residual life of 95 days, and 400 milligrams 110 days. One gallon of 5 per cent DDT spray to 1,000 square feet of surface gives the minimum of 200 milligrams of residue. Slick surfaces require an increase of the spray to 7.5 per cent. Application should be with a hand-compression sprayer or power sprayer giving a wetting spray in a uniform fan-shaped pattern. The principal resting places of flies should be covered, such as ceilings, around cracks and crevices, along edges of ceilings, doorways, and furniture. Electric and switch cords should be painted or sprayed. The United States Public Health Service warns: *Keep food and water containers covered after spraying the room. Flies fall into them as DDT takes effect.*

DDT-resistant houseflies. Evidence in support of observations made in many parts of the country that houseflies may be developing resistance to DDT has been accumulating steadily. Lindquist and Wilson[38] by selective breeding produced a strain of houseflies that was more resistant to DDT spray than were flies from their regular stock. About 10 per cent of the flies survived the initial "fine-mist spray" treatment, and these were used as the parent stock in establishing a new "special" colony. Each of 14 generations of flies was similarly exposed to treatment, and the survivors were allowed to propagate. The average mortality as the result of tests with the 14th generation was 69 per cent for the original strain of flies and 34 per cent for the selected flies.

That this resistance of the flies is not due to a deterioration of DDT under normal storage conditions has also been proved by tests. It is also unlikely that flies have changed their habits of alighting on surfaces; flies have been observed to alight on treated surfaces without toxic effect.

Several other factors may enter into the probable cause of increased fly populations in spite of DDT treatments, e.g., apparent failures came during extremely hot weather; DDT is said to be most effective in cool weather. Also careless use of DDT or inadequate treatment may be a factor. Certainly the wide use of DDT has resulted in a relaxation of sanitary practices, thus causing increased populations of flies.

No doubt satisfactory fly control will be achieved in many areas where proper DDT formulations are carefully applied and sanitation practices are rigidly adhered to. Much experimental work is being done with other insecticides, particularly now that fly control has been widely demonstrated. Among the promising insecticides is benzene hexachloride wettable powder, 12 per cent concentration at a dosage of 17 pounds in 100 gallons of spray (Marsh and Metcalf).

Garbage cans. Where garbage cans are used, and certainly every household should possess a garbage receptacle that is kept tightly closed against flies, it is strongly urged that all liquids be drained from the refuse before disposing of it and that the solids be wrapped in a newspaper before placing in the can. Keep the can clean. In this way fly breeding in garbage cans may be effectually prevented, and an act of mercy is done the scavenger and other handlers of garbage. It is a good practice to spray the outside of the garbage can and the rack on which it stands every week or two with a 5 per cent DDT oil solution.

Use of manure on lawns. Veritable swarms of flies may suddenly make their appearance on porches and windows and in the house after comparatively fresh manure has been spread on lawns as fertilizer. Such manure is commonly infested with full-grown maggots or pupae which, in a few days after the fertilizer has been applied, give rise to a pest of flies. It is, no doubt, wise to use old composted manure for this purpose or subject the manure to a thorough steaming or drenching with hot water at 195° F (nearly boiling) before applying it to the lawn.

Two objections are commonly raised against the steaming or drenching method of treatment: first, that the useful bacteria are destroyed, i.e., the manure may be rendered sterile; and second, that all other desirable constituents are leached out by the water. Not all of the useful bacteria, by any means, are destroyed by the hot water, and those remaining quickly multiply and soon render the manure as good as ever in this respect. In the second place, the leachings may be preserved quite easily by placing the manure to be treated in a shallow, tight box similar to those used by plasterers for mixing mortar, and adding a spigot in a hole with plug from which the leachings can be drawn and used as liquid manure.

This method is also useful to mushroom growers who must use rotted manure in which certain species of fly larvae, mites, etc., may occur in great numbers.

Railroad cars laden with manure are often sidetracked in or near communities and are responsible for swarms of flies. The writer has on several occasions recommended that the periphery of the entire carload be subjected to a treatment of live steam from the locomotive. This has been done with good results.

Lawn clippings. Grass clippings from lawns around public buildings are often dumped in heaps on the premises. Hot weather soon produces a vile-smelling, decomposing, warm mass of grass which is very attractive to flies, and within a few days numerous maggots, particularly those of the housefly, may be found infesting the mass. Thus, again, in spite of the absence of manure and garbage there may be a veritable plague of flies. The method of prevention is obvious: the clippings should be spread out thinly in order to dry out, and they may then be burned or otherwise disposed of.

Flies from septic tanks. Small invasions of flies may be traced to nearby septic tanks covered with knotty or imperfect wood superstructure which permit ingress of flies. Flies may be seen coming and going through these apertures, and examination of the top sludge will reveal countless maggots and pupae on the surface. Closing the knot holes or other apertures with tin or otherwise will soon end the trouble.

Sewage treatment plants. Flies of many species, notably the common housefly and blowflies, may be attracted to sewage treatment plants because of odors; and occasionally under improper management countless numbers of flies originate in sludge beds at the end of the treatment process. There may also be much fly breeding in wet sludge when this is applied to the soil as a fertilizer. The control of a breeding source of this kind may be accomplished by deep plowing and, of course, discontinuance of the use of wet sludge as a fertilizer.

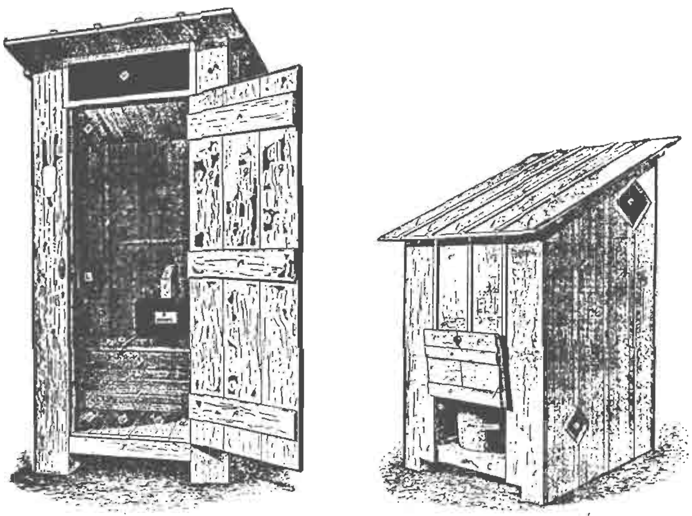


FIG. 110. A sanitary privy: front view to left, rear and side view to right. (After Stiles and Lumsden.)

Sewage treatment plants of modern construction do not as a rule breed flies. However, under all circumstances raw sewage must not be accessible to flies because of possible transport of pathogenic organisms of fecal origin. Sewage-works engineers suggest that fly breeding may be effectively prevented by quick drying of sludge. Chemical treatment of sludge is not generally favored, although spraying screenings with a solution made of one and a half pounds of pure carbolic acid and 40 pounds of caustic soda in 60 gallons of water has been recommended to stop fly breeding. The use of DDT as suggested in community fly control is recommended.

Privies. In the absence of modern plumbing particular attention should be paid to the location and construction of a box privy with receptacle or dug pit. No doubt many gastrointestinal disorders are traceable to insanitary privies. Two important precautions are involved:

first, selection of a location which will avert pollution of wells or other water supply; and second, choice of a type of construction which will prevent flies from gaining access to the excreta and will insure privacy.

A sanitary privy (Fig. 110) must meet the following requirements, according to Stiles and Lumsden:[39]

> (1) The excreta must not touch the ground; hence some kind of watertight receptacle (box, pail, tub, barrel, tank, or vault) for the excreta must be used under the seat. (2) Domesticated animals must not have access to the night soil; therefore the privy should have a trapdoor in the back to exclude them. (3) Flies and other insects must not have access to the excreta; therefore the entire privy must be made rigidly flyproof, or some substance must be used in the receptacle to protect the contents from insects.

Where the excreta are deposited in a pit or cesspool great care must be exercised in banking up around the outside of the building so as to prevent flies from gaining access to the pit. Borax applied to the excreta as suggested for manure is an added precaution to kill fly larvae. Paradichlorobenzene and orthodichlorobenzene are similarly useful. Also the liberal use of a 5 per cent DDT-oil emulsion spray is urged for application to all parts of the privy, inside and out, particularly the inside walls of the box seat. DDT does not kill the fly larvae.

If the privy is built on skids or can be otherwise easily moved, in addition to the treatment mentioned above, the accumulated excreta should be burned from time to time by adding straw and crude oil and setting them aflame. If sufficient water is available, the country home should be equipped to receive the sewage.

Flytraps. Flytraps, properly baited, are useful in making fly surveys for fly density data; however, other more valuable techniques to measure active fly populations have been devised, such as "bait cards" and "fly grills."[40]

BIBLIOGRAPHY

1. Hewitt, C. Gordon, 1910. *The Housefly*, Manchester, England: The University Press, xiii + 195 pp.
2. Howard, L. O., 1900. "A contribution to the study of the insect fauna of human excrement," *Proc. Wash. Acad. Sc.*, 2:541–604.
3. Dunn, L. H., 1923. "Observations on the oviposition of the housefly, *Musca domestica*, in Panama," *Bull. Entomolog. Research*, 13:301–5.
4. Herms, W. B., 1911. "The housefly in its relation to the public health." Berkeley: Univ. Calif., in Agric. Exper. Sta. Bull., no. 215. pp. 513–48 (16 figs.).
5. ——, 1907. "An ecological and experimental study of Sarcophagidae, *J. Exper. Zool.*, 4:45–83.
6. Hodge, C. F., 1911. In *Nature and Culture*, July, 1911.

7. Copeman, S. M., Howlett, F. M., and Merriman, G., 1911. "Flies as Carriers of Infection," in Reports to the Local Government Board on Public Health and Medical Subjects, London, n.s. no. 53, Report no. 4.

8. Parker, R. R., 1916. "Dispersion of *Musca domestica* Linn. under city conditions in Montana," *J. Econ. Entomol.*, 9:325–54.

9. Austen, E. E., 1926. "The housefly: Its life history, etc.," London: British Museum (Natural History) in Economic Series no. 1A 2nd Ed., pp. 5–52.

10. Hall, David G., 1948. *The Blowflies of North America.* Thomas Say Foundation of Entomolog. Soc. Amer. vol. 4, 477 pp.

11. Aldrich, J. M., 1916. *Sarcophaga and Allies in North America.* Thomas Say Foundation of Entomolog. Soc. Amer. 302 pp. (16 plates).

12. Herms, W. B., 1911. *The Photic Reactions of Sarcophagid Flies, etc.* Cambridge: Harvard Univ. in Contributions from Zool. Lab., Museum Comp. Zool., No. 217.

13. Dorman, S. C.; Hale, W. C.; and Hoskins, W. M.; 1938. "The laboratory rearing of flesh flies and the relations between temperature, diet, and egg production," *J. Econ. Entomol.*, **31**:44–51.

14. Herms, W. B., 1928. "The effect of different quantities of food during the larval period on the sex ratio and size of *Lucilia sericata* Meigen and *Theobaldia incidens* Thom.," *J. Econ. Entomol.*, **21**:720–29.

15. Esten, W. N., and Mason, C. J., 1908. "Sources of Bacteria in Milk," *Storrs Agric. Exper. Sta., Bull.*, no. 51, pp. 94–98.

16. Yao, H. Y.; Yuan, I. C.; and Huie, Dorothy; 1929. "The relation of flies, beverages, and well water to gastrointestinal diseases in Peiping," *Nat. Med. J. China*, **15**:410–18.

17. Graham-Smith, G. S., 1911. "Further observations on the ways in which artificially infected flies carry and distribute pathogenic and other bacteria," in Reports to the Local Government Board on Public Health and Medical Subjects, London (n.s., no. 53, further reports no. 4) pp. 31–48.

18. Nuttall, G. H. F., and Jepson, F. P., 1909. "The part played by *Musca domestica* and allied (non-biting) flies in the spread of infective diseases," in Reports to the Local Government Board on Public Health and Medical Subjects, London n.s., no. 16.

19. Leidy, Prof., 1871. "Flies as a means of communicating contagious diseases," *Proc. Acad. Nat. Sci. of Phila.*, p. 297.

20. Jordan, Edwin O., 1908. *A Textbook of General Bacteriology,* Philadelphia: W. B. Saunders Co. 557 pp.

21. Faichnie, N., 1929. "The etiology of enteric fever: Personal views and experiences," *J. M. A. S. Africa,* 3:669–75.

22. Gill, C. A., and Lal, R. B., 1931. "Epidemiology of cholera, with special reference to transmission: A preliminary report," *Indian J. Med. Research,* **18**:1255–97.

23. Paul, J. R., and Trask, J. D., 1941. "The virus of poliomyelitis in stools and sewage," *J. A. M. A.*, **16**:493–98.

24. Sabin, A. B., and Ward, R., 1941. "Flies as carriers of poliomyelitis virus in urban epidemics," *Science,* **94**:590–91.

25. ———, and ———, 1942. "Insects and epidemiology of poliomyelitis," *Science,* 95:300–301.

26. Nicoll, William, 1911. "On the part played by flies in the dispersal of the eggs of parasitic worms," in Reports to the Local Government Board on Public Health and Medical Subjects, London n.s. no. 53, further reports (No. 4) on flies as carriers of infection.

27. Roubaud, E., 1918. "Le rôle des mouches dans la dispersion des amibes dysentriques et autre protozoaires intestinaux," *Bull. Soc. path. exot.*, **11**:166–71.

28. Root, F. M., 1921. "Experiments on the carriage of intestinal protozoa of man by flies," *Am. J. Hyg.*, **1**:131–53.

29. Darling, S. T., 1912. "Murrina, a trypanosomal disease of horses in Panama, and the means used in controlling an outbreak," *Tr. 15th Internat. Cong. Hyg. & Demog.*, Washington, 5:619–31.

30. Ransom, B. H., 1913. *The Life History of Habronema muscae (Carter), a Parasite of the Horse Transmitted by the Housefly.* Washington, D. C.: Dept. Agric., in Bur. Animal Indust. Bull, no. 163. 36 pp.

31. Grassi, B., and Rovelli, G., 1889. "Embryologische Forschungen an Cestoden," *Centralbl. f. Bakt.*, **5**:370–77, and 401–10. Cited by Gutberlet.

32. Gutberlet, J. E., 1916. "Morphology of adult and larval cestodes of poultry," *Tr. Am. Micr.* Soc., 35:23–44 (2 plates).

33. Reid, W. M., and Ackert, J. E., 1937. "The cysticercoid of *Choanotaenia infundibulum* (Bloch) and the housefly as its host," *Tr. Am. Micr. Soc.*, **56**:99–104.

34. Dolve, R. M., 1912. *Barn Plans.* State College: North Dakota Agric. Coll., Govt. Agric. Exper. Sta. of North Dakota, in Bull. no. 97. 57 pp.

35. Michelbacher, A. E., and Smith, Gordon L., 1946. "Control of flies in dairy barns," Berkeley: Univ. Calif., in Agric. Exper. Sta. Circular, no. 365, pp. 94–96.

36. Rowe, John A., 1948. "Fundamentals of community fly control," *Proc. and Papers, 16th Ann. Conf. Calif. Mosq. Control Assn.* (Berkeley), pp. 11–14.

37. Douglas, James R., 1948. "Controllable noxious arthropods, other than mosquitoes, affecting man and animals," *Proc. and Papers 16th Ann. Conf. Calif. Mosq. Control Assn.* (Berkeley), pp. 14–16.

38. Lindquist, Arthur W., and Wilson, H. G., 1948. "Development of a strain of houseflies resistant to DDT," *Science*, **107**:276.

39. Stiles, C. W., and Lumsden, L. L., 1911. *The Sanitary Privy.* Washington, D. C.: Dept. Agric., in Farmers' Bull., no. 463. 32 pp.

40. Scudder, H. I., 1947. A new technique for sampling the density of housefly populations. U. S. Public Health Service, *Pub. Health Rep.* **62**:681–86.

CHAPTER XVI

BLOODSUCKING MUSCOID FLIES

Tsetse Flies, Stomoxys Flies, Horn Flies

ORDER DIPTERA–FAMILY MUSCIDAE

A. TSETSE FLIES

Introduction. The genus *Glossina,* a member of the family *Muscidae,* comprises the tsetse flies of Africa. The genus was established in 1830 by Wiedemann, and in the same year Robineau-Desvoidy described *Glossina palpalis.* Bequaert[1] states that the word "tsetse" was introduced into the English language by R. Gordon Cumming in 1850 in his "Five Years of a Hunter's Life in the Far Interior of South Africa," and David Livingstone in 1857 "focussed the attention of the scientific world upon the ravages of the fly."

That tsetse flies enjoyed a wide distribution in geological times is evidenced by the fact that several species of fossil *Glossina* flies from the Miocene shales of Colorado, U.S.A., have been described. Today the tsetse flies are restricted to continental Africa south of the Tropic of Cancer, an area known as "the fly belt." Buxton[2] states that the tsetses hold an area of about 4½ million square miles and are an immense obstacle to the development of tropical Africa.

Adult tsetse flies (Fig. 111), both male and female, depend on blood for their existence, and while they feed on a wide variety of animals, there are host preferences among the different species. Although man is freely attacked, he is not considered a favored host. In areas where large wild mammals have been decimated, the flies turn freely to man. *Glossina palpalis* (R.-D.) favors reptiles, particularly crocodiles and monitor lizards.

Students concerned with tsetse flies will consult the publications of Buxton,[2] Austen and Hegh[3] (1922), Newstead[4] (1924), Hegh[5] (1929) Swynnerton[6] (1936), as well as those other authors which cover particular segments of the subject, e.g., the work of Bequaert (1946)[7] which deals with the tsetse flies of Liberia.

General characteristics. The tsetses are medium-sized flies, ranging in size from that of a housefly to that of a blowfly. They are brownish in color; the body is wasp-like, and the wings when at rest are crossed scissors-like and extend well beyond the tip of the abdomen. The wing venation is characteristic in that the fourth longitudinal vein (M_{1+2}) bends suddenly upward before it meets the anterior transverse vein, which is very oblique (Fig. 111). The discal cell is remarkably "cleaver-like" in outline.

The palpi are nearly as long as the proboscis, which points bayonet-like in front of the head. The antennal arista (Fig. 112) bears a series of long bilaterally branched and regularly arranged hairs only on the upper surface. Grünberg[8] attached taxonomic value to the aristal hairs.

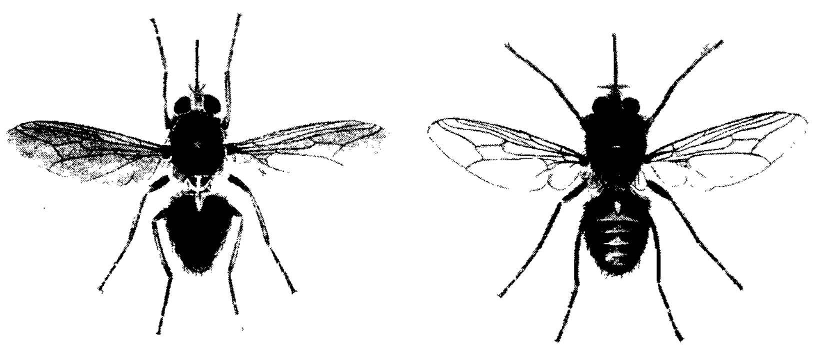
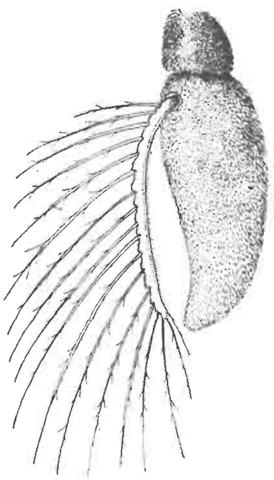


FIG. 111. (*Left*) *Glossina palpalis,* (*right*) *Glossina morsitans.* (After Newsstead.)

The mouth parts consist of the labium which ensheathes the two slender piercing stylets, the labrum-epipharynx, and the hypopharynx. A characteristic "onion-shaped" bulb is situated at the base of the haustellum (Fig. 113). Both sexes are avid bloodsuckers, feeding usually in broad daylight and outdoors. They are particularly attracted to moving objects.

Life history. The female tsetse fly gives birth to full-grown larvae which are extruded singly at intervals of about 10 to 12 days during the lifetime of the mother. During the intrauterine state (there are three larval stages) the larvae feed on fluid from special glands commonly referred to as "milk glands." The newly extruded larvae are creamy white to pale yellow and have a pair of intensely black, shining lobes at the posterior extremity. After each larviposition at least one full meal of blood is needed before the next larva begins to develop, eight to ten being produced during the life of the female.

The larvae are unable to crawl as do other muscoid larvae because of the reduced cephalopharyngeal armature. Lewis[9] points out that the larva moves and burrows by peristaltic movements and longitudinal contractions of the whole body. Coarse pebbly sand favors the larvae in burrowing, although a depth of only a few centimeters is reached. The behavior of the larva at this time, Lewis points out, is of great importance in determining its chances of survival. "If it is slow to penetrate the soil, it is exposed for a longer period to the possible attack of predators and parasites." Pupation takes place within an hour of larvi-



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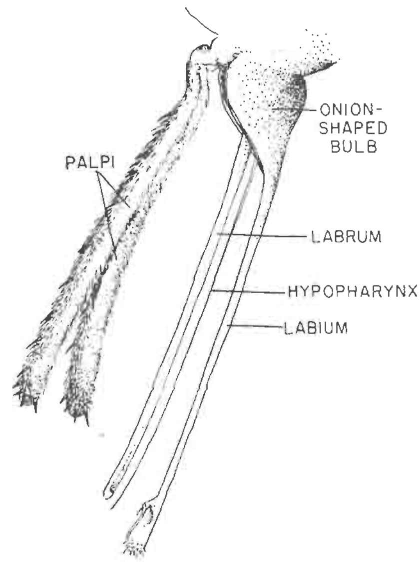


113

FIG. 112. Antenna of a *Glossina* fly, showing arista with branched hairs. (Much enlarged.)

FIG. 113. Mouth parts of a *Glossina* fly. ×17.

position. The pupa rapidly darkens to a blackish brown color with the posterior lobes and general form as shown in Figure 114. The pupal stage lasts from three to four weeks and longer, depending much on soil temperature and soil moisture. A great deal of work has been done on the ecology of the puparium (Nash[10]).

The fly emerges from the puparium by breaking loose the end of the pupal case by pressure from the ptilinum. The flies are said to have a striking dislike for excrementous matter, and the larvae are ordinarily deposited in the root tangles of the mangrove and shade of other vegetation where the soil is not too dry and is loose. The presence or absence of organic matter seems to be immaterial (Nash).

Trypanosomiasis. The tsetse flies are important vectors of trypanosomes. The term *trypanosomiasis* applies to all infections with flagellate protozoan parasites of the genus *Trypanosoma* and includes African sleeping sickness. The *Trypanosoma* belong to the class Mastigophora, order Protomonadida, family Trypanosomidae; they invade the blood, lymph, cerebrospinal fluid, and various organs of the body, such as the liver and spleen of many species of vertebrate animals, from fish to man. Many species of trypanosomes are regarded as nonpathogenic. Nearly all of the *Trypanosoma* require an intermediate insectan host.

The first trypanosome was discovered by Valentin in 1841 in the blood of the salmon. The name *Trypanosoma*[11] was given to these organisms by Gruby in 1842–43. Attention was not called to trypanosomes of mammals until the work of Lewis in 1878 on the parasites [*Trypanosoma lewisi* (Kent)] of the blood of the rat in India.° After that followed the

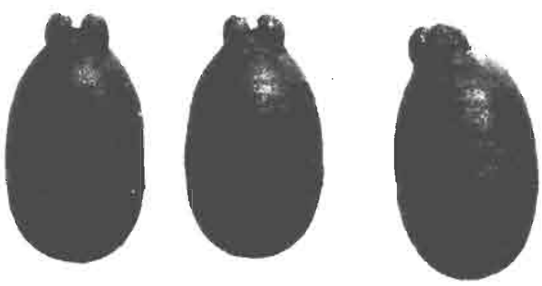


FIG. 114. Pupae of the *Glossina* fly. ×4.8.

discovery of other more important pathogenic trypanosomes, e.g., in 1880 Evans discovered the trypanosome, *Trypanosoma evansi* (Steel), of surra; in 1895 Bruce discovered *T. brucei* Plimm. and Bradf., the causal organism of nagana, the tsetse-fly disease of cattle and horses of Africa, and in 1897 he[12] showed that *Glossina morsitans* Westwood transmitted the infection. In 1901 Dutton[13,14] discovered trypanosomes in a blood smear prepared by Dr. R. M. Forde from a European patient in Gambia; he also found them in the blood of a native child. Following this discovery the organism was named *Trypanosoma gambiense* Dutton. In 1903 Bruce and Nabarro[15] proved *Glossina palpalis* (R.-D.) to be the carrier by feeding freshly caught flies ranging in number from 31 to 9 on a black-faced monkey daily, sometimes twice daily, beginning May 13. Trypanosomes were present in the monkey's blood on May 27. In 1903 Castellani[16] reported trypanosomes in the cerebrospinal fluid of negroes in Uganda suffering from sleeping sickness. The trypanosomes

° *Trypanosoma lewisi* is transmitted from rat to rat by the rat flea, *Nosopsyllus fasciatus* (Bosc), and the rat louse, *Polyplax spinulosus* (Burm.).

found by Castellani were supposed to be a different species from that of Dutton (*Trypanosoma gambiense* Dutton) and were called *T. ugandense* Castellani, 1903. Kruse later gave to this trypanosome the name *T. castellanii*. The important discoveries of Dutton and Castellani were quickly confirmed by David Bruce, who found these trypanosomes 38 times out of 38 in the cerbrospinal fluid obtained by lumbar puncture in natives of Uganda suffering from sleeping sickness, and 12 times out of 13 in the blood. According to the rules of priority, the last two species names mentioned must give way to *Trypanosoma gambiense* Dutton. A second species of trypanosome producing sleeping sickness in Rhodesia, Nyasaland, and adjoining territory was described in 1913 by Stephens and Fantham (*loc. cit.*, p. 5) as *T. rhodesiense*.

Sleeping sickness is widely distributed in Africa, extending along the west coast from Senegal (in part) to Angola (in part) and eastward to the valley of the upper Nile. It has been estimated that between 1896 and 1906 from 400,000 to 500,000 natives perished from this pestilence. Dutton and Todd found that in some villages from 30 per cent to 50 per cent of the population was infected. In many parts of Africa the number of cases remains high; on the other hand as Buxton (1948) points out there are a number of areas in East and West Africa where there are healthy people and cattle living permanently on ground from which tsetse flies have been exterminated.

Age does not affect the distribution of the malady, since children as young as 18 months to 2 years have been known to be infected. Sex does not influence the disease. Occupation and social position, however, do show a marked influence. The majority of the cases observed are among the agricultural and lower classes, where the degree of exposure to tsetse-fly bites is greatest. In areas where *Glossina palpalis* occurs, inland fishermen in particular may be exposed to the bites of this species.

The bite of an infected tsetse appears to produce more local reaction than that of the uninfected fly. The usual duration of the incubation period is ten days to three weeks, but clinical symptoms may be delayed for as long as two to five years. During the first phase of the disease, which may continue for many months, the trypanosomes are in the blood, the trypanosomiasis stage; this phase is characterized by an irregular fever, glandular enlargement, debility, and languor. In the second phase, the sleeping sickness stage, the trypanosomes are constantly found in the cerebrospinal fluid; a constant accompaniment is the enlargement of the posterior cervical lymph nodes, *Winterbottom's sign;* there is speech impairment and tremors of the tongue; there is nervousness, pronounced languor and drowsiness, which gives way to lethargy, and finally the victim falls into a comatose state, wasting rapidly, due largely to starvation, until death ensues. Although authors commonly refer to

Gambian sleeping sickness as caused by *Trypanosoma gambiense* and the Rhodesian sleeping sickness as caused by *Trypanosoma rhodesiense* there is as yet no uniformity of opinion in the matter; there may be but one causal species.

Transmission. The natives of French Guinea long attributed the transmission of sleeping sickness to flies; it had already been shown by Bruce that nagana, a disease of cattle and horses, was transmitted by tsetse flies, when Dutton and Todd gave attention to the biting flies of Gambia. These investigators found that of the flies which bite both man and animals, *Tabanus dorsovittata* Walker and *Glossina palpalis* (R.-D.), were the most important. The latter is very common in western Africa, where it abounds in the mangroves which line the inland rivers during the warmer months when these insects are very troublesome.

Experiments made by these workers, however, gave negative results. It was Bruce and his collaborators (*loc. cit.*) who subsequently went over the matter and showed that *Glossina palpalis* (R.-D.) is a vector. Animal experimentation showed that these flies can transmit the trypanosomes mechanically for a period of less than 48 hours; the organisms become more and more attenuated after the fly has bitten the infected individual, and it loses the power of infection in less than 48 hours. Thus the tsetse fly may be a mechanical vector for a few hours, during which time its soiled proboscis is involved, i.e., trypanosomes are introduced into the wound produced by the bite before the proboscis is completely cleaned. Interrupted feeding would thus be a factor. Mechanical transmission from man to man in nature is believed to be very uncommon, if it occurs at all.

Kleine[17] in 1909, working with *Trypanosoma brucei*, causal organism of nagana, was evidently the first to demonstrate its development in the tsetse fly, *Glossina palpalis*. Robertson[18] working with the causal organism of sleeping sickness, *Trypanosoma gambiense*, reported in 1913 that it is first established in the posterior part of the mid-gut of the insect, where multiplication occurs and trypanosomes of varying sizes are produced. From the tenth or twelfth day onward slender, long forms are to be found in increasing numbers. These finally move forward to the proventriculus and are the dominant type. The proventriculus becomes infected as a rule between the twelfth and twentieth days. The salivary glands become infected by the slender (proventricular) type which reaches the glands by way of the hypopharynx; arriving in the glands, they become attached to the wall and assume the crithidial condition. Multiplication takes place, and trypanosomes are formed which closely resemble the blood type. The development in the salivary glands takes from two to five days before the metacyclic forms are infective. The fly is never infective until the glands are invaded. The trypanosomes are never attached to the wall of the alimentary canal, and there is no intracellular multiplication in the gut cycle. The metacyclic forms become mature and multiply by longitudinal fission in the circulatory system of the mammalian host. The Sleeping Sickness Commission found that infectivity lasted at least 96 days. Only a small percentage of the tsetse flies become infective. The life of a female *G. palpalis* (R.-D.) in captivity has been observed to be about four and one-half months.

The problem of sleeping sickness is greatly complicated in that many species of game animals harbor the causal trypanosomes and may thus serve as natural reservoirs of the infection. Furthermore the affinities of *Trypanosoma rhodesiense* have not yet been definitely determined. There are some who contend that this is a varient of *T. gambiense;* others contend that it is a human strain of *T. brucei* and considerably less adapted biologically to man and his domestic animals than to the wild game. This might account for the greater virulence of this species in relation to man. *Glossina morsitans* and *G. swynnertoni* are vectors.

The principal vectors of African sleeping sickness, each in its respective zone, are *Glossina palpalis* (R.-D.), *G. morsitans* Westwood, and *G. swynnertoni* Austen.

Nagana. Trypanosoma brucei Plimm. and Bradf. is the causal organism of nagana, long known to be a fatal tsetse-fly disease of African horses, mules, and camels, less rapidly fatal to cattle, sheep, and dogs. Many other mammals are susceptible to the infection. Bruce (*loc. cit.*) found that many species of wild game animals harbor the trypanosome and thus form reservoirs. The disease is characterized by progressive emaciation, fever, edema of the abdomen and genitalia, and marked depression. The trypanosomes are found in the blood and especially the lymph-gland swellings from the beginning of the first symptoms.

Glossina morsitans West., *G. longipalpis* (Wied.), *G. pallidipes* Austen, *G. tachinoides* West., and *G. austeni* Newstead relate to its transmission in practically the same way that *Glossina palpalis* (R.-D.) and other *Glossina* flies relate to sleeping sickness of man, i.e., the flies are infective for a day or two after feeding on an infected animal, then become noninfective for a period of about three weeks, when they again become infective and remain so for the rest of life. The incubation period after inoculation into the body of the host is said to be about 10 days.

Glossina species. Newstead (*loc. cit.*) recognized twenty species, one subspecies, and five varieties belonging to the genus *Glossina.* These he divides into three groups, (1) the *fusca* group, which includes the 10 largest species, viz., *Glossina brevipalpis* Newstead, *G. fusca* (Walker), *G. fusca* var. *congolensis* Newst. and Evans, *G. fuscipleuris* Aust., *G. haningtoni* Newst. and Evans, *G. longipennis* Corti, *G. medicorum*

Aust., *G. nigrofusca* Newst., *G. schwetzi* Newst. and Evans, *G. severini* Newst., and *G. tabaniformis* West.; (2) the *palpalis* group, which includes *Glossina caliginea* Aust., *G. pallicera* Bigot, *G. palpalis* (Rob.-Desv.) and two varieties, var. *wellmani* Austen and var. *maculata* Newst., also one subspecies *G. palpalis* subspecies *fuscipes* Newst., and *G. tachinoides* West.; (3) the *morsitans* group, which comprises *Glossina longipalpis* (Wied.), *G. morsitans* West., *G. morsitans* var. *pallida* Shirc., *G. morsitans* var. *paradoxa* Shirc., *G. submorsitans* Newst., *G. pallidipes* Aust., *G. swynnertoni* Aust., and *G. austeni* Newst.

Glossina palpalis (R.-D.) (Fig. 111) is the most important vector of Gambian sleeping sickness. It covers an enormous area in Africa, but it occurs chiefly in the Congo and in west Africa. It is usually found on the shores of rivers and lakes, but it may occur quite far back from them; and as Swynnerton points out, it requires a combination of several types of country one of which must be relatively massive wooding or thicket of more or less evergreen type (see Bequaert[7]). It feeds mainly on large reptiles such as crocodiles and monitor lizards, but it can live on the blood of mammals as well. Although man is not regarded as one of its favored hosts, it will nevertheless feed freely on persons available in its riparian habitat.

Glossina morsitans Westwood (Fig. 111) is a most efficient vector of both Rhodesian sleeping sickness of man and nagana of animals. It has a wide distribution in Africa; it is of importance in the Sudan, northern and southern Rhodesia, the Belgian Congo, and many other localities. This species requires "savanna of sufficient shade value, and with sufficient logs, rocks, or tree rot holes to form a good rest-haunt and breeding-ground, and relatively open glades or plains in which to hunt for its prey." It is typically a "game fly," but attacks human beings readily, hence is a most dangerous tsetse.

Glossina swynnertoni Austen, like *G. morsitans* West., is a strong vector of both Rhodesian sleeping sickness and nagana. It is largely confined to the northern part of Tanganyika, according to Swynnerton, who describes it as "the fly of the driest and most open areas and apparently unable to inhabit the more mesophytic savannas. It breeds normally in thicket, though rock suits it as well. . . . It utilizes open spaces as feeding grounds. . . . It is primarily and essentially a 'game' fly." It attacks human beings with readiness, and like *Glossina morsitans* West. is a very dangerous tsetse.

Tsetse-fly control. Since the memorable discoveries of Bruce and others that tsetse flies are responsible for the transmission of nagana and sleeping sickness, few insects have been so minutely studied by the most capable investigators. The practical and extended control of breeding places offers serious difficulties, not the least of these being the fact that the larvae are retained within the body of the female until fully grown, hence are not directly dependent upon an external food supply. The monumental work of the late C. F. M. Swynnerton gives ample testimony to the tremendous ramifications of the tsetse-fly control problem. Among the many possible modes of attack there are the following: (a) direct attack, involving the use of flytraps; the direct effect of temperature and moisture on pupae; the use of natural enemies, etc.; (b) indirect attack by modification of cover, reducing or expelling game animals, thus depriving the flies of food supply; introduction of fly barriers by setting up clearings or thickets according to the species of fly to be dealt with; reclamation and appropriate agricultural practice. Because of the wide divergence in the ecological requirements of the several important species of tsetse flies, the utilization of appropriate control measures is a matter of long and tedious investigation.

Buxton (1948, *loc. cit.*) states that it is not difficult to exterminate *Glossina palpalis* from a linear fly belt along water; it is done by clearing or hand catching. In large areas of bush, such as those inhabited by *Glossina morsitans*, satisfactory results have been achieved by bush clearing, by attacking the restricted permanent habitats, and also by game destruction and human settlement. Buxton also suggests that the use of DDT "smoke" gives some promise.

B. STOMOXYS FLIES

Family Muscidae, Genus Stomoxys

General characteristics. Owing to similarity in color and size the stomoxys fly is often mistaken for the common housefly *Musca domestica* Linn. However, the former is more robust and has a broader abdomen. In color it is brownish gray with a greenish-yellow sheen; the outer of the four longitudinal thoracic stripes are broken, and the abdomen is more or less checkered. The wings when at rest are widely spread apart at the tips, and are distinctly iridescent; the apical cell is open. When resting the fly has its head thrown well up and the wings slope decidedly toward the surface upon which it has settled. The proboscis protrudes bayonet-like in front of the head. The antennal aristae, unlike those of the housefly, bear setae on the upper side only. *Stomoxys calcitrans* (Linn.) enjoys practically a world-wide distribution.

Habits. Although the stomoxys fly, *Stomoxys calcitrans* (Linn.), is commonly called the "stable fly," it occurs much less abundantly (often absent) about stables than does the housefly. It is also called the "biting housefly," since it may occur indoors, especially in the autumn and during rainy weather, and bites human beings viciously. It is often very annoying along the sandy vegetation-strewn shores of lakes. Recently the name "dog fly" has become attached to this species. The stomoxys fly is typically an out-of-door day-biting fly and is usually to be found in abundance during summer and autumn where large numbers of domesticated animals occur, horses and cattle affording an abundant food supply. This species does not breed in excrement as freely as does the housefly. Sunny fences, walls, light-colored canvas coverings, and

FIG. 115. Feed racks for dairy cattle afford an ideal breeding place for stomoxys flies in that the moist lower layers of material in the trough furnish abundant food for the larvae.

light objects in general, when in the proximity of animals, are abundantly frequented by stomoxys flies.

The stomoxys fly is a vicious "biter" which draws blood quickly and fills up to full capacity in from three to four minutes if undisturbed; but ordinarily even when undisturbed it changes position frequently or flies to another animal, where the meal is continued. This fly feeds readily on many species of warm-blooded animals, for example, rats, guinea pigs, rabbits, monkeys, cattle, horses, and man. Both sexes are bloodsucking The flight of the stomoxys fly is direct and swift. It may travel many miles.

Breeding habits and life history. Although the stomoxys fly can be successfully reared in the manures of horses, cattle, sheep, etc., it may be safely said that it does not breed commonly in excrement under field conditions unless this is well mixed with straw or hay. Very good breeding places are afforded by the leftover soggy hay, alfalfa, or grain in the bottoms of or underneath out-of-door feed racks (Fig. 115) in connection with dairies, feed lots, etc. This material becomes soggy and ferments, and here practically pure cultures of stomoxys larvae may be found. The material must be moist; dryness prevents larval development. Piles of moist fermenting weeds and lawn cuttings also furnish fairly good breeding material. Piles of decaying onions have been found by the writer to harbor myriads of larvae late in the autumn. Old straw piles that remain in the field through the year may produce an abundance of stable flies in the moist fermenting straw near the ground,

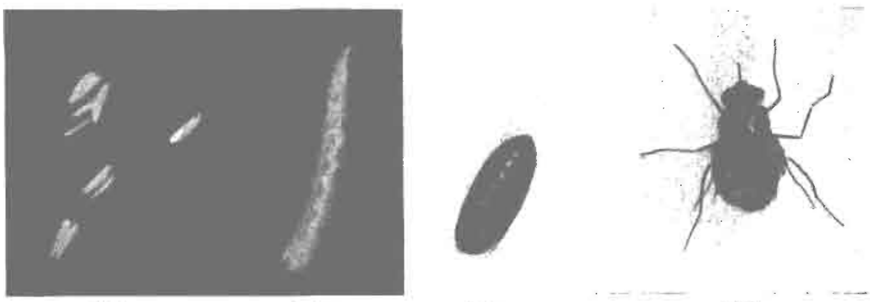


Fig. 116. Life cycle of the stable fly, *Stomoxys calcitrans*. ×2. (Photograph by H. F. Gray.)

particularly if cattle have access to it and moisten it with urine. Moist piles of fermenting peanut litter produce large numbers of these flies, as do beach deposits of grasses along the Florida coast. The author has also noted this along the southern shores of Lake Erie.

The larvae of stomoxys and of the housefly can readily be differentiated by the form, size, and position of the posterior spiracles; otherwise they resemble each other closely. The pair of posterior spiracles of the stomoxys larva are roughly triangular, widely separated, and situated near the periphery, while in the housefly larva they are elliptical, quite large, close together, and more central in position (Fig. 119).

The eggs (Fig. 116) of the stomoxys fly are about 1 mm long, curved on one side, and straight and grooved on the opposite side. In depositing her eggs the female fly often crawls far into the loose material, placing them usually in little pockets in small numbers, often in pairs. Egg depositions range in number from 23 to 102, usually between 25 and 50, and there are ordinarily four or five layings. Mayne (Mitzmain[19]) found in his observations made in the Philippine Islands that the maximum number of eggs produced by a single *Stomoxys* is 632 and possibly 820 and that there may be as many as 20 depositions during the lifetime of the female.

The incubation period varies from two to five days, commonly three days, at a temperature of 26° C. Higher temperatures result in a shorter incubation period. The newly hatched larvae bury themselves in their food at once, thus protecting themselves against light and dryness. At a temperature of from 21° to 26° C, the larvae reach full growth in from 14 to 26 days. Mayne found that the larval stage averaged 12 days at a room temperature of 30° to 31° C.

Before pupation the larvae usually crawl into the drier parts of the breeding material, where the chestnut-colored pupae are often found in enormous numbers. The pupae are from 6 to 7 mm long and may be recognized by the posterior spiracles as in the larva. The pupal period also varies, dependent largely on temperature. At a temperature of from 21° to 26° C, this period varies from 6 to 26 days, with the greatest frequency between 9 days and 13 days.

At an average temperature of 29° C, Mayne found the pupal period to average five days.

If not handicapped, the imago emerges with astonishing rapidity, crawls away, unfolds its wings, and is ready to fly away in less than half an hour. The fact that the proboscis is temporarily attached beneath the thorax gives the newly emerged insect a very peculiar appearance, and it may then be easily mistaken for a housefly.

Summarizing the life history of the stomoxys fly it may be said that at a temperature of 21° to 26° C, the *shortest* periods are: egg, 2 days, larva, 14 days, pupa, 6 days, total, 22 days; the *average*, egg, 3 days, larva, 15 days, pupa, 10 days, total, 28 days; and the *maximum*, egg, 5 days, larva, 26 days, pupa, 26 days, total, 57 days. The total time at 21° C, from the laying of the eggs to the emergence of the adults, was from 33 days to 36 days, as observed in five individual cases. Mayne reports the development of this fly in 12 days under optimum conditions.

Copulation takes place within a week, and egg deposition begins in about 18 days, after emergence from the pupa cases at a temperature of from 21° to 26° C. Higher temperatures undoubtedly decrease this time. In warmer parts of the world the stomoxys fly continues to breed throughout the year, slowing up the cycle during the colder months.

Longevity. With approximately 4,000 flies under continuous daily observation in glass quart jars, 50 flies to a set, the writer has found that the average length of life of the stomoxys fly under favorable laboratory conditions of feeding (i.e., daily feedings on monkeys or rabbits) is about 20 days. The maximum life under these conditions was found to be 69 days and several hours in a female.

Mayne has found the maximum for a female stomoxys fly to be 72 days and for the male 94 days.

The writer has observed that a set of flies which fed only on sugar water deposited no eggs, although many of them lived 20 days or longer, while control flies fed on blood did lay eggs. Hence it seems apparent that the flies must have blood in order to develop eggs.

As a cattle pest. Bishopp[20] regards this fly as one of the most important sources of annoyance to livestock. Injury is brought about in various ways, e.g., worry due to the attacks of myriads of flies, loss of blood, and loss of flesh.

Freeborn, Regan, and Folger[21] have shown that the reduction in milk production caused by the stable fly amounted to 9.26 per cent, which for a five months' period means a loss of 50 gallons of milk, amounting to $10 per cow per season. The total loss occasioned by the three dairy cattle pests—houseflies, stable flies, and horseflies—amounted to 14 per cent.

Poliomyelitis. The following is quoted from a report by Rosenau on poliomyelitis, presented at the 1912 meeting of the International Congress on Hygiene and Demography.[22]

When I first began to study the disease [poliomyelitis], I regarded it probably as one which is spread by direct contagion, by contact, either directly or indirectly, from person to person. The first circumstance which shook my faith that we were dealing with a contagious disease was the fact that we had eighteen negative results in attempting to prove the presence of the virus in the secretions from the nose and throat. I could not help asking at the time if it were not possible to find the virus, which is so potent, in the secretions of the nose and throat of persons who have the disease and those who are convalescing from the disease. These results were confirmed at the same time by Strauss, of New York, who had negative results in a large series and by Neustaedter's recent results and by other results, all of the examinations having proven negative excepting one recently reported by Kline, Patterson and his associates at this congress and in the literature recently.

A second circumstance which led me to believe we were not dealing with a contagious disease was the fact brought out by Dr. Richardson. Children in all stages of this disease were crowded into schools, institutions, tenement districts and other places where there was every chance for the spread of the disease, but it did not spread there, but it continued to spread in the rural, thinly scattered districts where one would not expect to find contagious disease. There was a resemblance to rabies. All those who have worked with this virus in laboratories were at once struck with the resemblance between poliomyelitis and rabies. The latter being a wound infection, there is some analogy between it and poliomyelitis, and poliomyelitis might be transmitted through some sort of wound. I was fortunate enough to have had experience with yellow fever, both in the investigation of it and the sanitary measures against it, before the mosquito period, and I was much struck with many analogies which came to me between that disease and certain features of poliomyelitis.

The work I bring to your attention consisted of taking a number of flies —*Stomoxys calcitrans*—caught in a net and bred for the purpose; you can catch several hundred of these flies in a stable in a very short time. We placed these flies in a large cage and exposed monkeys to their bites, the monkeys having been purposely infected with the virus of poliomyelitis. Care was taken to place the monkeys in the cages in all stages of the disease, before and after. In fact, a monkey would be exposed to the bites of the flies on the same day he was infected, so that the flies could drink the blood of the monkey during all stages of the period of incubation of the disease, for we do not yet know in what stage of the infection the virus appears in the blood at its maximum, or the best period for infecting these flies. Following this we exposed healthy monkeys to the bites of the same flies, and after several weeks' time these healthy monkeys came down with a disease which in all essential respects resembles anterior poliomyelitis. Out of twelve healthy monkeys so exposed, six of them now have symptoms of the disease, three of them in the virulent form. Of the other three monkeys, two are coming down, but one seems to have a milder infection than the other. This mild infection consists of trembling and weakness of the hand, and some weakness of the jaw which lasted about a week or so and then passed away. We cannot be sure whether that is true poliomyelitis or not until we are able to test the monkey subsequently. If it were poliomyelitis, that monkey would be "immune." In three of the six cases that came down with the disease, having been bitten by flies, there was some diarrhea. The disease in the monkey resembles more closely that which we see in children, rather than the disease we produce purposely experimentally by bringing the virus in direct association with the central nervous system. Of course, that may be only a coincidence, but it is interesting.

The work of Rosenau was repeated and confirmed during October, 1912, by Anderson and Frost[23,24] who summarize as follows: "Three monkeys exposed daily to the bite of several hundred stomoxys, which at the same time were allowed daily to bite two intracerebrally inoculated monkeys, developed quite typical symptoms of poliomyelitis eight, seven and nine days from the date of their first exposure."

In order to test the findings of the above-mentioned investigators and to secure further biological evidence if possible, the author in cooperation with W. A. Sawyer,[25] undertook a special investigation of the problem, beginning in October, 1912. Believing it unwise to use flies collected out of doors, these insects were reared for the purpose in an insectary. The importance of this precaution is made evident by the fact that flies captured out of doors in Berkeley were shown to transmit a pathogenic organism to a rabbit, infection undoubtedly having been acquired in nature. This infection, resulting in abscess, was successfully transmitted from rabbit to rabbit through the agency of the stomoxys fly.

Seven carefully planned experiments were conducted over a period of nearly a year; about 4,000 laboratory-reared flies, a large number of monkeys, rabbits, and other experimental animals were used; the results were entirely negative.

Control of stable flies. The more important breeding places of the stomoxys can be controlled by removing moist feed wastes from feeding troughs and from feed lots, stalls, stables, etc., and scattering the wastes to hasten drying. Moisture is necessary for the development of the larvae; therefore dry material is not suitable. Weeds, lawn cuttings, decaying onions, vegetation washed up on lake shores in the immediate vicinity of summer resorts, etc., should not be allowed to accumulate in piles long enough to ferment and decay.

Bishopp (*loc. cit.*) has shown that loosely piled straw stacks (oats and wheat) are important breeding places of the stomoxys fly, hence he recommends "that the straw for feeding and bedding purposes be baled and stored under cover. Where this is not practicable the stacks should be rounded up so as to make the top largely rain proof and the sides nearly vertical." DDT as recommended for housefly control can also be used successfully in the control of stable flies.

C. THE HORN FLY

Family Muscidae

Introduction. The so-called horn fly (also called Texas fly), which torments cattle and horses (the latter to a less extent) in many parts of North America is classified by some entomologists as *Haematobia stimulans* Meigen (= *irritans* R.-D.) and by others as *Lyperosia irritans* Linn. [= *Haematobia serrata* (R.-D.)]. In his description of the habits of these flies in Europe, Hammer[26] states that *Haematobia stimulans* extends farther north in Scandinavia and Russia than does *Lyperosia irritans*, the latter occurring only in the southern portions of this area. Hammer remarks that *L. irritans* has adapted itself to milder climates with higher temperatures. He also describes this species as attaching very closely to cattle, only leaving the animals to oviposit; it spends both day and night on the cows whether they are in the byre or in the field. *Haematobia stimulans* leaves the animals at night and hides away among vegetation. The horn fly with which we have dealt in California, where it is a severe cattle pest, sticks close to the cattle at all times; it also deposits large reddish-brown (or yellowish-brown) eggs, while Hammer states that *Haematobia stimulans* deposits white eggs. This insect is known as the horn fly because it rests on the horns of cattle much of the time, particularly at night, probably because they afford a safe resting place.

The horn fly was introduced into the United States from Europe, where it has been an important cattle pest for many years. (See Hammer, *loc. cit.*) According to the United States Bureau of Entomology and Plant Quarantine it was first reported in the fall of 1887 from Camden, N. J., appearing during the following year in Maryland and Virginia, probably having appeared in Philadelphia in 1886, and by 1892 was found over the entire continent from Canada to Texas and from Massachusetts to the Rocky Mountains. California cattlemen state that it made its appearance in this state in about 1893–1894. It appeared in Honolulu, Hawaii, in 1897.

Characteristics. The horn fly is about half the size of the common housefly, i.e., about 4 mm long. It has much the same color and in most

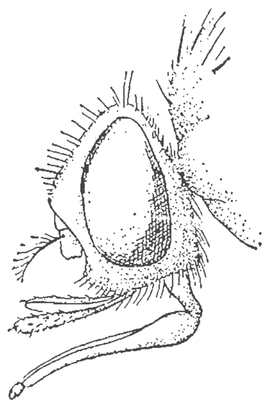


FIG. 117. Side view of head of the horn fly.

other respects resembles the stomoxys fly. The mouth parts (Fig. 117) are like those of stomoxys except that the labium of the horn fly is relatively heavier and the palpi, almost as long as the proboscis, are flattened and loosely ensheath this structure. The arista is plumose dorsally. The wing venation is as in stomoxys.

Horn flies appear early in the spring and become most abundant in late summer and autumn. When the horn fly is at rest on an animal or elsewhere its wings lie flat on its back and fold rather closely, but when it bites, the wings are spread and the insect stands almost perpendicularly, hidden between the hairs of the host. Apparently the habit of resting at the base of the horns is only done when flies are overabundant.

Life history. The horn fly deposits its eggs chiefly, if not exclusively, on freshly passed cow manure. The fly is seen to dart from the animal and deposits its eggs in groups of four to seven, or singly, on the surface of the dung. The eggs are relatively large (1.3 to 1.5 mm), larger than the eggs of stomoxys; they are reddish brown in color, hence not easily seen on the cow dung. Under laboratory conditions, at least, few eggs are deposited by the females, rarely over twenty. At a temperature of 24° to 26° C, the eggs hatch in 24 hours.

The larvae burrow beneath the surface of the droppings, reaching full growth in from three to five days, when they crawl underneath into drier parts and pupate. The pupal period requires from six to eight days. Hence the entire life history (Fig. 118) from the egg to the adult requires from 10 to 14 days at a temperature of from 24° to 26° C.

Damage done. The damage occasioned by the horn fly is chiefly through irritation and annoyance, which in dairy animals results in disturbed feeding and improper digestion causing loss of flesh and reduced milk production. Dr. James Fletcher estimated the loss in Ontario and

FIG. 118. Life cycle of the "horn fly," *Haematobia serrata.*

Quebec, Canada, at one-half of the product of meat and milk. Range animals literally run themselves thin in trying to get away from these pests.

The actual loss of blood must be considerable when literally thousands of these flies attack an animal. From 10 to 25 minutes are required for the fly to fully engorge itself; during this time it withdraws and reinserts its proboscis in the same puncture many times as in a pumping motion. Much undigested blood is discharged from the anus of the fly while in the act of feeding.

Control. The most effective method to prevent the multiplication of the horn fly is to scatter the droppings from cattle with a rake or other implements or simply by dragging a branch of a tree over the field. Hogs allowed to run with the cattle serve this purpose very well. The manure thus scattered dries out quickly, and the larvae that are present perish, owing to the fact that they require much moisture for development. The writer has seen this method applied successfully in various parts of California where the dry summer favors such means of handling the situation. On wide ranges this method is impracticable, but in connection with dairies it is entirely feasible. The use of DDT cattle sprays, as already described, gives good control on the animals.

Lyperosia exigua (de Meij.), commonly known as the "buffalo fly," is particularly important to the cattle and dairy industries of Australia.[27] Among the animals which it attacks are buffalo, cattle, horse, dog, and man. The fly oviposits in fresh dung from buffalo and cattle in particular.

Other species of bloodsucking muscoid flies. The genus *Philaematomyia* represented by the single species, *P. insignis* Austen, is of particular interest because of the form of the proboscis, which is intermediate between the biting and nonbiting muscid type. *P. insignis* Austen is a widely distributed African and Oriental species resembling *Musca domestica* Linn. in size and general appearance. According to Austen[28] the proximal portion of the proboscis is a strongly swollen, polished, chitinous bulb, while the distal portion is soft and fleshy and folded back under the distal end of the bulb when not in use; when in use its terminal section, consisting of a tubular extension, is protruded from between the labella; it is surrounded at the distal extremity with a circlet of stout chitinous teeth. When it is not being used the entire proboscis can be retracted within the buccal cavity. Austen states that the fly probably feeds by cutting through the epidermis with the teeth at the end of the tubular extension and then sucks up the blood.

The Ethiopian genus *Stygeromyia* (*S. maculosa* Austen, and *S. sanguinaria* Austen) is said by Austen (*loc. cit.*) to be in some respects intermediate between *Stomoxys* and *Lyperosia.* It resembles *Stomoxys* in general appearance and form of the body but is distinguished "by the relative stoutness of the short, chitinous, horizontal proboscis, and by the palpi being equal to the proboscis in length, large, expanded towards the tips, and curved upwards."

Stomoxys nigra Macquart, *S. omega* Newstead, and *S. inornata* Grünberg are all Ethiopian species and resemble *S. calcitrans* in feeding habits.

BIBLIOGRAPHY

1. Bequaert, J., 1930. "Tsetse flies—past and present (Diptera: Muscoidea)." *Entomological News,* 41:158–64, 202–3, and 227–33.

2. Buxton, P. A., 1948. "The problem of tsetse flies," *Proc. 4th Internat. Cong. Trop. Med. and Malaria,* Washington, D. C. (Abstracts).

3. Austen, E. E., and Hegh, E., 1922. *Tsetse-flies, Their Characteristics, Distribution, and Bionomics.* London: Imperial Bur. Entomol. 188 pp.

4. Newstead, R., 1924. *Guide to the Study of Tsetse Flies.* Liverpool: School Trop. Med. in Memoir, no. 1. n.s. 272 pp. (28 plates, 4 maps).

5. Hegh, Emile, 1929. *Les tsé-tsés,* vol. 1. Brussels: A. J. Engelterzi. xiv + 742 pp. (327 figs. + 15 colored plates).

6. Swynnerton, C. F. M., 1936. "The tsetse flies of East Africa." *Tr. Roy. Entomolog. Soc. London,* vol. 84. xxxiv + 579 pp. (7 maps, 22 plates).

7. Bequaert, Joseph C., 1946. "Tsetse-flies in Liberia: Distribution and ecology; possibilities of control," *Am. J. Trop. Med.* **26** (Suppl.):57–94.

8. Grünberg, Karl, 1904. *Die Blutsaugenden Dipteren.* Jena: Gustav-Fischer. xi + 188 pp. (127 figures).

9. Lewis, D. J., 1934. "The behavior of the larvae of tsetse flies before pupation," *Bull. Entomolog. Res.,* **25**:195–99 (1 plate).

10. Nash, T. A. M., 1939. "The ecology of the puparium of *Glossina* in northern Nigeria," *Bull. Entomolog. Res.,* **30**:259–84.

11. Laveran, Alphonse, et Mesnil, Felix, 1904. *Trypanosomes et Trypanosomiasis,* Paris. xi + 417 pp. Translated by D. N. Nabarro, 1907. Chicago Medical Book Co.

12. Bruce, D., 1897. *Further Report on the Tsetse-Fly Disease or Nagana in Zululand.* London: Harrison and Sons. 69 pp.

13. Dutton, J. E., 1902. "Trypanosoma in man," *Brit. M. J.* **1**:42.

14. ———, 1902. "Note on a Trypanosoma occurring in the blood of man," *Brit. M. J.,* **2**:881–84.

15. Bruce, D., and Nabarro, D., 1903. *Progress report on sleeping sickness in Uganda.* Rept. Sleeping Sickness Comm. Roy. Soc. London, no. 1. 88 pp. (10 plates).

16. Castellani, A., 1903. "Trypanosoma in sleeping sickness," *Brit. M. J.,* **1**:1218.

17. Kleine, F. K., 1909. "Positive Infectionsversuche mit *Trypanosoma brucei* durch *Glossina palpalis,*" *Deutsche med. Wochnschr.,* **35**:469–70.

18. Roberston, Muriel, 1913. "Notes on the life history of *Trypanosoma gambiense,* with a brief reference to the cycles of *Trypanosoma nanum* and *Trypanosoma pecorum* in *Glossina palpalis,*" *Phil. Tr. Roy. Soc. London* (Ser. B), **203**:161–84 (5 plates).

19. Mitzmain, M. B., 1913. "The bionomics of *Stomoxys calcitrans* (Linnaeus); a preliminary account," *Philippine J. Sc.,* **8** (Sec. B): 29–48.

20. Bishopp, F. C., 1939. *The Stable Fly: How to Prevent its Annoyance and its Losses to Livestock.* Washington, D. C.: Dept. of Agric., in Farmers' Bull., no. 1097. 18 pp. (revised).

21. Freeborn, S. B.; Regan, W. M.; Folger, A. H.; 1925. "The relation of flies and fly sprays to milk production," *J. Econ. Entomol.,* **18**:779–90.

22. Rosenau, M. J., 1912. In "Society Proceedings," *J. A. M. A.,* **59**:1314. Report on poliomyelitis presented at meeting of International Congress on Hygiene and Demography, 1912.

23. Anderson, John F., and Frost. Wade H., 1912. "Transmission of poliomyelitis by means of the stable fly *(Stomoxys calcitrans),*" U. S. Public Health Service, *Pub. Health Rep.,* **27**:332–35.

24. ———, 1913. Poliomyelitis: Further attempts to transmit the disease through the agency of the stable fly *(Stomoxys calcitrans).* U. S. Public Health Service, *Pub. Health Rep.,* **28**:833–37.

25. Sawyer, W. A., and Herms, W. B., 1913. "Attempts to transmit poliomyelitis by means of the stable fly *(Stomoxys calcitrans),*" *J. A. M. A.*, 41:461–66.

26. Hammer, Ole, 1941. "Biological and ecological investigations on flies associated with pasturing cattle and their excrement," *Videnskabelige meddelelser fra Dansk naturhistorisk forening*, vol. 105, 257 pp.

27. Kriggsman, B. J., and Windred, G. L., 1933. *Investigations on the buffalo fly, Lyperosia exigua de Meij.* Commonwealth of Australia: Council for Sc. and Ind. Res., Pamph. 43. 40 pp.

28. Austen, E. E., 1909. Illustrations of African bloodsucking flies other than mosquitoes and tsetse flies. London: Mus. Nat. Hist. 221 pp. + xiii plates.

CHAPTER XVII

MYIASIS

Myiasis is a term meaning an infestation of the organs and tissues of man or animals by fly maggots and the disturbances resulting therefrom. Such invasions may be benign in effect or may result in more or less violent disturbances, even in death. When the intestinal tract is invaded it is called *intestinal myiasis;* when the stomach is concerned it is *gastric myiasis;* invasion of the urinary tract is called *urinary myiasis;* invasion of the nasal passages, *nasal myiasis;* invasion of the ears, *auricular myiasis* or *otomyiasis;* of the eyes, *ophthalmomyiasis;* when wounds or ulcerations of the skin are infested by maggots the term *traumatic dermal myiasis* is applied; invasion of the skin is also known as *cutaneous myiasis,* etc. When maggot infestations are traceable to species which are normally scavengers, coprobionts, or saprobionts, the term *accidental myiasis* is usually employed; when the species of maggot is a necrobiont or facultative sarcobiont (involving fresh wounds) the term *semispecific* is used; and when the infestation is traceable to obligatory sarcobionts such as the warble flies and botflies the term *obligate myiasis* is used. All who are concerned with the subject of myiasis must become thoroughly familiar with *The Flies That Cause Myiasis in Man* by Maurice T. James.[1] *

ACCIDENTAL MYIASIS

Accidental myiasis may be caused by a considerable number of species of Diptera belonging to several families such as the *Calliphoridae* (blowflies, comprising the bluebottle and greenbottle flies), *Sarcophagidae* (flesh flies, scavenger flies), and *Muscidae* (houseflies, scavenger flies). The larvae of these flies normally feed on decomposing animal and vegetable matter, garbage, dead animals, and manures. Infestations in man are usually traceable to the ingestion of fly eggs or larvae with contaminated food or water.

Identification of fly maggots. Maggots, the larvae of muscoid Diptera, are footless, more or less cylindrical, tapering anteriorly, and truncated posteriorly; they are distinctly segmented, with ordinarily 11 or 12 visible

* Also see Hall, David G., 1948. *The Blowflies of North America.* Thomas Say Foundation. 477 pp. 5 color + 46 black and white plates.

segments (Fig. 119). Fully grown larvae differ greatly in length according to the species, ranging from 5 mm to 35 mm.

At the blunt or posterior end are found the spiracles (Fig. 119)

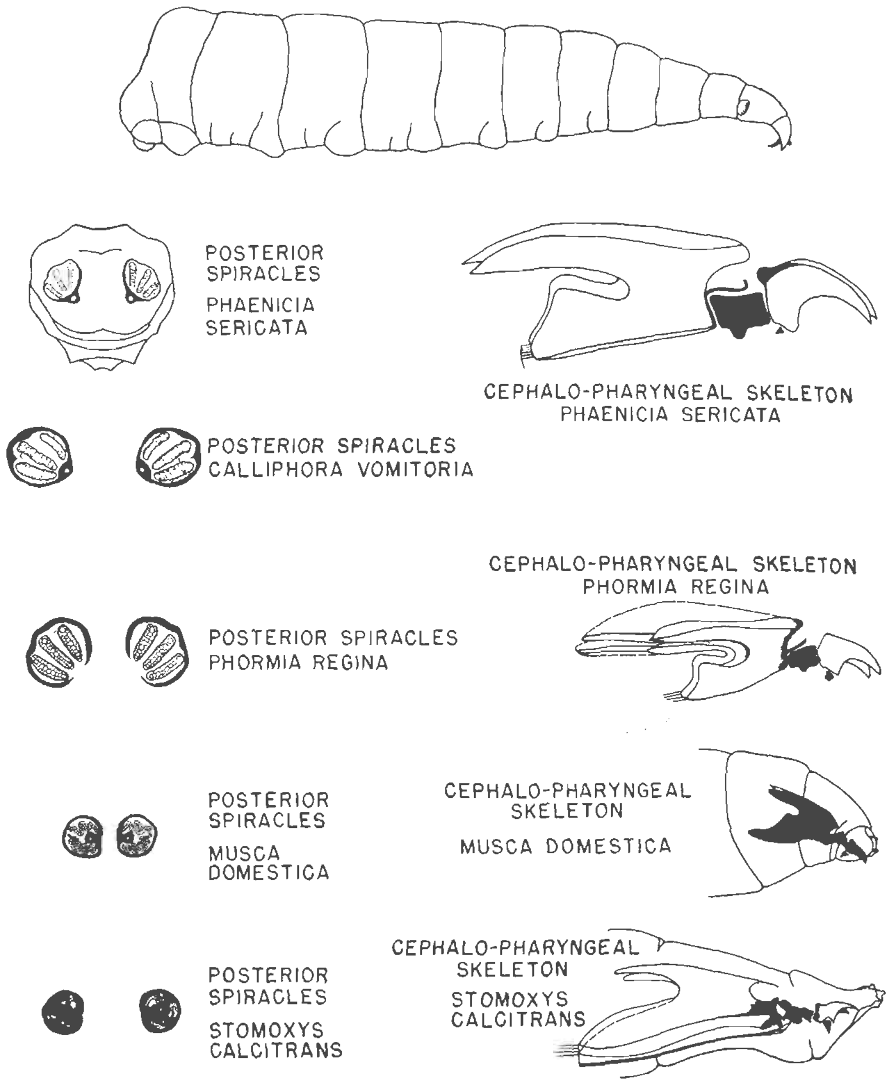


FIG. 119. Taxonomic details used in the classification of muscoid fly larvae. (Redrawn after various authors.)

which afford useful diagnostic characters. There are two *stigmal plates* more or less separated from each other, within which are situated *spiracles* one to three in number, either slit-like, sinuous, or more or less circular. A prominence known as a "button" is best seen in certain slit-like forms, as in *Phaenicia* (*Lucilia*) *sericata* (Meig.) (Fig. 119). The button may be absent or variously situated depending upon the species, hence has taxonomic value.

In using the posterior spiracles for purposes of classification the following characters are to be noted: (1) *diameter* of the stigmal plate, the space occupied by one stigmal plate on a line drawn through the center of both; (2) *length,* when slits are absent, the space occupied by a plate on a line drawn dorsoventrally through the center of the plate; or, when slits are present, the space occupied by a plate along a line drawn from the lower edge of the button (or space if button is absent) through the longest slit (middle slit) to the margin of the plate; (3) *width,* along a line drawn at the middle of the plate at right angles to the *length* line; (4) distance between the plates; (5) general form of the plates; (6) shape of spiracles; (7) presence or absence of *button;* (8) general structure of plate.

Intestinal myiasis. According to Braun and Seifert[2] at least 30 species of fly larvae had been reported (1926) from cases of intestinal myiasis. No doubt, other species have been added since. They are principally members of the families Muscidae, Calliphoridae, and Sarcophagidae, which commonly deposit their eggs or larvae on cold meat, cheese, and other foods of man and are thus ingested. It is also suggested that the flies may deposit their eggs in or near the anus, particularly in the use of old-fashioned open privies. The larvae on hatching are believed to make their way into the intestine.

Hoeppli and Watt (1933),[3] experimenting with the larvae of *Chrysomya megacephala* (Fabr.) and *Phaenicia* (*Lucilia*) *sericata* (Meig.), secured results that agree with those obtained by Desoil and Delhaye (1922)[4] with larvae of *Calliphora vomitoria* (Linn.) showing that fly larvae of certain kinds as well as the eggs are able to resist the influence of temperature, gastric juice, hydrochloric acid, and ferments in all the concentrations occurring in the human stomach, but that the larvae are very susceptible to mechanical injuries and to the obstruction of their stigmata. Ample food for the larvae is provided by the intestinal contents as well as the intestinal mucosa itself. On the basis of experiments with dogs and cats which were fed fly larvae with their food, with the result that no living larvae of any of the species were found either in the large intestine or in passed feces of these animals, Causey[5] (1938) remarks that " . . . it is time to revise our discussion of intestinal myiasis in accordance with known facts."

The clinical symptoms of intestinal myiasis depend on the number as well as the species of fly larvae and on their location in the intestine. No doubt many instances occur in which living fly larvae are passed in the stool without having caused any intestinal disturbance. In severe infestations there will be nausea, vertigo, and more or less violent pains in the abdomen; diarrhea with discharge of blood may occur as the result of injury of the intestinal mucosa by the larvae. Living and dead larvae are expelled with either the vomit or stool, or with both.

An obstinate case of intestinal myiasis was reported by Herms and Gilbert (1933).[6] The patient, Z. W., female, age 38 years, was first seen April 26, 1930. Her chief complaints were attacks of nausea, vomiting and diarrhea, nervousness, and joint aches. There were recurring attacks of nervousness, vomiting, and diarrhea, and apparently rather frequent hemorrhages from the bowels. The patient was considerably depressed at times, and treatment was difficult because of lack of cooperation except after she had had a bad spell. Because of the difficulty of obtaining stool specimens, especially during the acute attacks, and in view of the fact that it was felt that there must be other reasons for her condition, early in the spring of 1931 during an attack of nausea with vomiting and diarrhea the patient was kept for one entire day in the office under observation and stool and vomit specimens were obtained, both containing the first larvae which were studied. During these attacks it was difficult for her to obtain relief with fairly large doses of opiates. Following this observation she was given santonin by mouth and colonic irrigations containing thymol. Many larvae were recovered after this, all of which were dead. Following the attacks of diarrhea the patient had a number of severe hemorrhages. Tetrachlorethylene capsules were given by mouth, but they caused gastric distress. In the hospital a duodenal tube was passed and tetrachlorethylene was injected beyond the stomach. For a few weeks there was apparent improvement, but the attacks recurred, with the passage of larvae by vomiting and bowel discharge.

Three lots of fly larvae were studied in the laboratory, i.e.: March 31, May 12, and July 28, 1931. Adult flies belonging to three genera, *Calliphora, Phaenicia (Lucilia), and Sarcophaga,* were reared from these larvae.

The recurrence of violent symptoms with evacuation of larvae in vomit and stools would ordinarily point to repeated infections, but the fact remains that the patient as observed by her physician, the co-author, lived in a way that would seem to preclude repeated infestations, and the exposure of stools and vomit to flies is ruled out because of circumstances indicated in the case report. The authors have advanced a possible explanation based on the pedogenetic reproduction of fly larvae as suggested by observations made by Parker (1922),[7] viz.: "The increases led me to believe that *Calliphora erythrocephala* occasionally multiplies in an unusual way, and that this way is not polyembryony but pedogenesis." In the case described there were certainly broods of very young larvae at intervals, at which time also full grown larvae were present.

Cheese skippers as agents. The larvae of the cheese fly, *Piophila casei* (Linn.) of the family Piophilidae, frequently cause intestinal myiasis, as they are able to pass through the digestive tract without injury. Simmons[8] cites a number of instances indicating the frequency of their occurrence in the digestive tract of man. The adult flies measure from 2.5 to 4 mm in length; superficially they appear shining black, with reddish-brown eyes and wings held flat over the dorsum when at rest. The eggs are deposited on cured meats, old cheese, dried bones, smoked fish, and many similar materials. The eggs hatch in from 30 to 48 hours at a temperature of 65° F; the larval stage requires about 8 days, the pupal about 12 days. These stages are greatly influenced by temperatures. The larvae have the peculiar habit of curving the ends of the body together and then suddenly springing to a distance of from three to six inches.

Soldier fly. A case of intestinal myiasis caused by the larvae of a soldier fly, *Hermetia illucens* (Linn.) (Family Stratiomyidae), is reported by Meleney and Harwood.[9] This fly feeds mainly on flowers, and the eggs are deposited on decaying fruits, vegetables, and animal matter. The source of infection according to the authors was apparently raw fruit or vegetables. The symptoms were local irritation in the stomach and rectum, and spells of fainting. The patient was a boy of ten years. M. A. Stewart informed the author that he observed a patient in Houston, Texas, who continued to expel these larvae for several months.

Gastric myiasis. The frequency with which the "rat-tailed" larvae of the drone fly *Tubifera tenax* (Linn.), occur in liquid excrement must lead to extreme caution in accepting reports that these larvae have been evacuated with discharges from the bowels. The writer has on several occasions received specimens of "rat-tailed" larvae which were said to have been evacuated by the "double handful" and the patient was said to have "steadily improved" thereafter.

There are, however, several recorded cases which seem to be incontrovertible, notably the case reported by Hall and Muir,[10] who also brought together all information then available relative to *Tubifera* and myiasis. The case referred to was that of a boy aged five years "who had been ailing for about ten weeks and who had been under medical treatment for indigestion and obstinate constipation for about five weeks at that time. The child was emaciated and anemic. Very striking symptoms were the constant and pronounced twitching of the eyelids and other nervous movements. He gritted his teeth in his sleep at times, and made convulsive movements of the limbs. When awake he complained of pain in the limbs and headache. The emaciation seemed to be due to the fact that the boy had for some time vomited almost everything he ate. The breath was very bad, 'worse than rotten eggs,' according to his parents. On the basis of the nervous and digestive disturbance and the general debility, a diagnosis of worm infestation was made."

With this diagnosis in mind the mother of the boy gave him a dose of a proprietary worm remedy, resulting in the discharge of an object wriggling round vigorously in the feces and urine. The slop jar into which the stool was passed was in regular use and had been previously rinsed with tap water and allowed to dry during the day. The specimen was identified by the authors as one of the "rat-tailed larvae" measuring 3.2 cm. in length, including the long "tail." A second larva was said to have been discharged the following day. The case is believed by the authors to be probably a genuine case of "gastric myiasis."

After the passage of the larvae the child is said to have improved in health and become normal; the nervous symptoms and vomiting disappeared.

Three chances for infection were pointed out; namely, first, the eating of "overripe" or probably decaying peaches in which "rat-tailed" larvae might have occurred; secondly, the drinking of "ditch" water polluted with kitchen refuse, etc.; lastly, stable manure in a neighbor's yard where the child played.

The authors offer the following comment relative to the gastric disturbances:

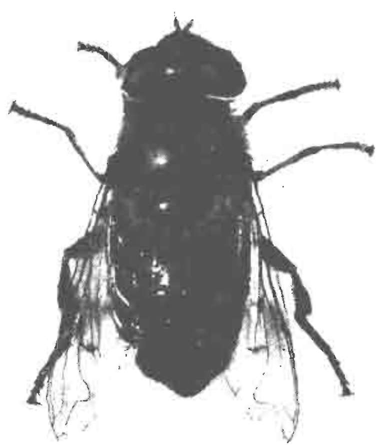
> A larva supplied with the stigmatic apparatus of *Eristalis* would apparently be fitted for life in a stomach with a small amount of food and plenty of the atmospheric air which is swallowed in eating and drinking and at other times. Such a condition would simulate the normal life conditions fairly closely. That the stomach would not fill to the point where it would drown the larva might be insured by the vomiting, perhaps automatically, the activity of the larva increasing as the stomach filled to where it threatened to cover the rising stigmatic tube, and so setting up an irritation leading to vomiting. The mother states that the child's stomach was extremely intolerant of milk and that drinking milk was promptly followed by vomiting. This suggests that milk, usually taken in long drinks and considerable quantities, quickly threatened the larva with drowning and set up such activity as promptly to cause vomiting.

Hall[11] has added the records of other cases, giving a total of 17 claiming the presence of syrphid larvae in the digestive tract of man, one claiming their presence in the nostrils of man, and two claiming their presence in the diseased vagina of cattle.

Tubifera (= *Eristalis*) *tenax* (Linn.), the drone fly (Fig. 120), is a large insect, somewhat larger than a honeybee and resembling the drone bee very closely; indeed it is commonly referred to as its mimic. The fly deposits its eggs on liquid manure or other filthy liquids in cans, slop jars, privies, septic tank effluent, etc. The larvae are known as "rat-tailed larvae" (Fig. 121); these also occur occasionally in heaps of horse manure.

The family Syrphidae includes a very large group of flies, varying greatly in size, many of which are brightly colored. They are nearly all flower loving, feeding on nectar mainly. Only one genus needs to be considered here, namely *Tubifera*, the larvae of which have a long anal breathing tube, i.e., "rat-tailed," and the adults of which are commonly called drone flies.

Urinary myiasis. As in intestinal myiasis the symptoms of urinary myiasis depend on the number and kind of larvae, and their localization. There may be obstruction and pain; pus, mucus, and blood in the urine;



120

121

FIG. 120. The drone fly, *Eristalis tenax*, whose larvae are commonly called "rat-tailed larvae." ×3.5.

FIG. 121. The "rat-tailed larva" of *Eristalis tenax*, drone fly. ×2.

and a frequent desire to urinate. Larvae are expelled with the urine. Chevril[12] reports that *Fannia canicularis* (Linn.) (see Chapter XV) is most frequently found in urinary myiasis, although *Fannia scalaris* (Fab.) and *Musca domestica* Linn.[13] have been encountered. Hoeppli and Watt (*loc. cit.*) believe that albumin and sugar in the urine may provide food, as may mucus and leucocytes; the lack of oxygen presents the chief difficulty, although very small amounts of oxygen are needed by the larvae.

Infection is probably usually accomplished at night in warm weather when persons may sleep without covering. The flies deposit their eggs around the urethral opening; these hatch in a few hours, and the larvae enter the urethra.

Traumatic dermal myiasis, the invasion of wounds or ulcers of the skin by fly larvae, is of common occurrence in warm, humid climates. A large number of species of flesh flies are responsible for this type of myiasis, particularly the screwworm flies, greenbottle flies, and related species.

The following description of a case caused by *Phormia regina* (Meig.) reported by Stewart[14] will serve to illustrate this form of myiasis:

> The dermatitic area was not large at first but it continued to spread after hospitalization. An extremely offensive odor was given off, but aside from the dermatitis and irritation of the sores, the patient appeared to be feeling well; appetite, digestion and egestion were good. At first treatment was applied only to the area around the ears, but on the night of the patient's admission to the hospital the discovery was made that the scalp was a mass of pus and a supersaturated sulphur wash was applied. The hair was parted to allow the wash to penetrate freely to the scalp and a towel was tied about the head, coming below the ears. The supersaturated sulphur wash was applied every two hours.
>
> After the second treatment was applied to the scalp the patient became very restless, working the fingers into the palms of her hands and alternately putting her hands to her ears. Soon she began to scream, acted frantic, and became nearly delirious. She was given a sedative without effect.
>
> On taking the towel from the patient's head the nurse observed fly larvae, which had been forced into activity by the treatment, crawling over the towel, hair and down the cheeks. The nurse estimates that she killed twenty-five or thirty larvae in the hour and a half she spent in removing them and still the hair and scalp remained full of them. Back of the ears the mass of living larvae was so great that they could almost have been spooned out. At this time the patient complained of a buzzing in the ears similar to that occurring when the ears are full of water, and said that she could not hear. The nurse then used toothpick swabs to remove the great quantity of larvae found in the pinnae of the ears; in so doing most of the larvae were killed, but some were kept alive and placed on raw beef in vials so that they might complete their larval growth and pupate.
>
> As soon as pupation occurred the puparia were removed to fresh vials and covered with fine dry soil until they emerged as adults, when they were identified as *Phormia regina* Meig.
>
> After the removal of all visible larvae had been completed the patient's hair was clipped, the supersaturated sulphur wash treatment was continued, and the scalp was bandaged. To the original area of dermatitis around the ears was applied a paste consisting of salicylic acid, 2 gm.; zinc oxide, 24 gm.; starch, 24 gm.; petrolatum, sufficient to make 100 gm.
>
> It is obvious that an adult female fly had been attracted to the suppurating scalp sores by the foul odor given off and had oviposited in one or more of these sores. The larvae were driven from the scalp to the pinnae of the ears by the application of the supersaturated sulphur wash.

Chrysomya megacephala (Fabr.) was responsible for much traumatic myiasis in the South Pacific Islands during World War II. Stewart[15] describes a new treatment for traumatic dermal myiasis:

> A new douche, composed of 15 per cent chloroform in light vegetable oil, has been employed in the treatment of seventeen cases of traumatic dermal myiasis. In every case all the maggots were removed with a single treatment, extending over a period of thirty minutes.
>
> The new douche has advantages over the commonly used chloroform-milk solution in that chloroform is entirely soluble in vegetable oil and only slightly soluble in milk; in that the chloroform-vegetable oil solution is very stable and can be kept indefinitely in closed containers, whereas the chloroform-milk mixture has to be made up fresh for each application, and in that the vegetable oil is very soothing to the raw tissue of the infested wound.

SEMI-OBLIGATE MYIASIS

Callitroga (= *Cochliomyia*) *americana* C. & P. (Fig. 122) is the name recently given to the New World screwworm fly by Cushing and Patton[16] to separate this dangerous form (the primary screwworm),

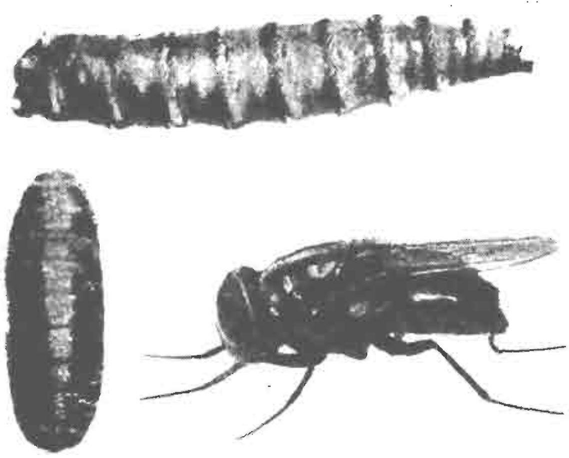


FIG. 122. *Callitroga* (= *Cochliomyia*) *americana*, the Texas screwworm fly, larva, and pupa. (After Bishopp.)

which feeds on the tissues of living animals, from *Callitroga* (= *Cochliomyia*) *macellaria* (Fabr.), the secondary screwworm fly (Fig. 123), which is more particularly a scavenger fly. *C. americana* is an obligate parasite and according to Laake, Cushing, and Parish initiates the great majority of cases of screwworm infestations in man and animals in the United States and probably in the entire Neotropical region. It is known to be the cause of nasopharyngeal myiasis in man. The following is a detailed description of a case which terminated fatally (from Osborn [1896] quoted by Richardson in the Peoria, Ill., Medical Monthly for February, 1883):

While traveling in Kansas in the latter part of last August, a citizen of this place had the misfortune to receive while asleep a deposit of eggs from this fly. He had been troubled for years with catarrh, hence the attraction to the flies. He returned home a few days after the accident and shortly after began complaining of a bad cold. Growing rapidly worse, I was called to attend

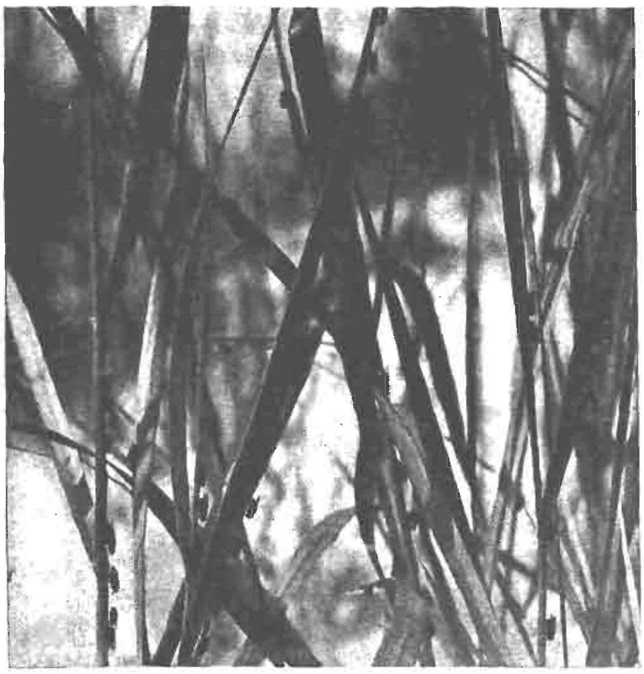


FIG. 123. *Callitroga* (= *Cochliomyia*) *macellaria* just emerged from the pupa cases. A dead animal near by furnished food for the larvae; pupation took place in the sand underneath the carcass. The newly emerged flies have crawled up on the grass and will soon be ready to fly away. Note characteristic resting attitude, with head down.

him. Monday, my first day, his appearance was that of a man laboring under a severe cold. Had slight congestion of the lungs, and moderate fever. His nose seemed greatly swollen and he complained of a smarting, uneasy feeling in it, and general misery through the head. Gave him treatment to relieve the congestion and fever. Tuesday, saw him again. His nose and face were still swollen, and in addition to the other symptoms he was becoming slightly delirious and complained a great deal of the intense misery and annoyance in his nose and head. A few hours after, I was sent for in haste with the word that something was in his nose. I found on examination a mass of the larvae of this fly (or 'screw worms' as they are commonly called in the South) completely blocking up one nostril. On touching them they would instantly retreat *en masse* up the nostril. Making a 20 per cent solution of chloroform in sweet milk I made a few injections up both nostrils, which immediately brought away a large number, so that in a few hours I had taken away some 125 of them. By Wednesday evening erysipelas had begun, implicating the nose and neighboring portions of the face. Another physician was called. By continual syringing with a strong antiseptic solution of salicylate of soda, bicarbonate of soda, and carbolic acid we hoped to drown out the remaining larvae. But they had by this time cut their way into so many recesses of the nose and were so firmly attached that we were unnable to accomplish much. Finally we resorted to the chloroform injections, which immediately brought away a considerable number. Friday I was able to open up two or three canals that they had cut, extracting several more that had literally packed themselves, one after another, in these fistulous channels. His speech becoming suddenly much worse, I examined the interior of his mouth and found that a clear-cut opening had been made entirely through the soft palate into his mouth and large enough to insert the end of a common lead pencil. Saturday the few remaining larvae began changing color and one by one dropped away. On Sunday for the first time hemorrhage from both nostrils took place, which continued at intervals for three days, but was not at any time severe. On this day the patient began to improve, the delirium and erysipelas having subsided, leaving but little or no annoyance in his head. In a few days he became able to go about home, and even to walk a distance of half a mile to visit a friend and return. But while there he began complaining of a pain in the neighborhood of his left ear, apparently where the eustachian tube connects with the middle ear. It proved to be an abscess. Being already so reduced by the first attack he was unable to withstand the second, and died after an illness of nearly three weeks, completely exhausted by his prolonged sufferings. Three days before his death the abscess discharged its contents by the left nostril. The quantity of pus formed was about 2½ ounces (78 grams).

In all about 250 larvae were taken away from him during the first attack, and, as the visible results, not only had they cut the hole through the soft palate, but had also eaten the cartilage of the septum of the nose so nearly through as to give him the appearance of having a broken nose. The case occupied, from the first invasion of the fly to its final result, nearly two months. He doubtless would have recovered but for the formation of the abscess, which from all the symptoms, was caused by one or more of the larvae having found their way up the left eustachian tube.

Callitroga (= *Cochliomyia*) *americana* C. & P. is strongly attracted to the wounds and sores of animals. Laake[17] estimates the loss occasioned by this fly in the southwestern United States at $5,000,000. He found the following predisposing causes of attack: among sheep and lambs, wounds caused by needle grass take first rank; among goats and kids, shear cuts take first rank; among cattle, injuries by the horns of other cattle; among calves, exposed tissue at birth; and among horses and mules, wire cuts.

Laake points out that the more common causes of screwworm attack are due to farm practices that can be corrected. He stresses particularly care in shearing, dehorning, removing and disposing of old barbed wire from dismantled fences, also the timing of dehorning, castrating, and branding so as to expose the wounds as little as possible to flies during the season of abundance.

An epidemic of human myiasis, 81 cases in 5 provinces of Chile occurring during the months of December to April, 1945–1946, is reported by Gajardo-Tobar and Honorato[18] who also describe the biology of the fly concerned, *Callitroga americana* (C. and P.) = [*Cochliomyia hominivorax* (Coq.)].

Cutaneous myiasis. The larvae of *Wohlfahrtia vigil* (Walk.), a member of the Sarcophagidae in eastern North America, frequently cause cutaneous myiasis. Walker[19] describes the cases of a five-month-old boy thus:

Most of the lesions were clustered together on the left side of the neck under the angle of the jaw, one being on the left cheek. They had been first noticed by the mother 24 hours earlier, and when seen by the writer they were already secondarily infected with pus organisms, the child being in a poor general condition and suffering from an intestinal disorder. They were similar to the lesions observed in the previous cases, each being a boil-like sore with an external opening, and from these openings five or six larvae had already been expressed. Only three additional larvae were obtained, these measuring 5 to 7 mm. in length. Each was placed on raw beef in a separate test-tube, plugged with cotton wool. In 24 hours they reached a length of 12 to 13 mm., and in another 24 hours they were full-grown, each measuring about 17 mm. in length.

On the third day after their removal from the child the larvae were placed with the meat in a jar of earth and immediately burrowed into the latter. Next day they were at the bottom of the jar and two of them had begun to contract. Three days later they were dug up and all had transformed into puparia.

On September 27, 18 days later, a male *Wohlfahrtia vigil* emerged. I waited for the others to appear until October 5, but neither having emerged by that time I opened one of the puparia on that day and the other a week later, and in both I found pupae which had evidently died some time before the proper time for emergence, as they were quite colorless. In this case, like the previous ones, the child recovered rapidly after the removal of the maggots.

Two new cases of cutaneous myiasis in Panama were reported recently (1948) by Calero,[20] viz.: Case I. Removed second-stage larvae of *Stephanostoma* (= *Sarcophaga*) *haemorrhoidalis* (Fall.) from fistulous tract in the region of the mentum. Case II. Removed 166 larvae of *Callitroga* (= *Cochliomyia*) *hominivorax* (Coq.) from right parietal region of the cranium; three points of perforation of the periosteum were found upon removing the larvae. The patient did not present either meningeal symptoms or encephalic irritation.

Life history of the screwworm fly. The adult fly of *Callitroga* (= *Cochliomyia*) *americana* (C. & P.) has a deep greenish-blue metallic color with yellow, orange, or reddish face, and three dark stripes on the dorsal surface of the thorax. It is difficult, unless one is experienced, to separate this species from *Callitroga* (= *Cochliomyia*) *macellaria* (Fabr.). Laake, Cushing, and Parish state that the females of *C. macellaria* (Fabr.) may usually be distinguished from *C. americana* C. & P. by the fact that the basicostal scale (a small sclerite at the base of the wing) of the former is of a yellowish color, whereas in the latter (*C. americana* C. & P.) it is black. Also *C. macellaria* (Fabr.) is covered on the mid-line of the underside of the abdomen by a dense white pruinosity absent in *C. americana* C. & P. The species can be easily and accurately determined by the use of the characters exhibited by the male terminalia.[21]

Individual females of *Callitroga* (= *Cochliomyia*) *americana* C. & P. according to Laake, Cushing, and Parish, may lay as many as 2,853 eggs, the eggs being deposited in characteristic batches of 10 to 393 eggs each, and the laying of as many as 300 eggs may be completed in from four to six minutes. The incubation period of the eggs on wounds in animals ranges from 11 to 21.5 hours, under natural conditions. The larval feeding period ranges from 3.5 to 4.5 days; the prepupal period from a few hours to about three days (7 hours to 76 hours); the pupal stage lasts about seven days. The prepupal and pupal stages are greatly influenced by temperature and moisture. The life history from egg to adult under optimum natural conditions requires about 11 days.

Screwworm control. The screwworm is a true parasite and lives only in the living flesh of warm-blooded animals; it is not found in snakes, lizards, or other cold-blooded animals, nor in carcasses, dead fish, decaying meats, or decaying vegetable matter. The maggots found in dead animals are not true screwworms (Bruce and Sheely[22]). In the light of this information much serious damage by screwworms may be prevented by good herd management, which involves such matters as controlled breeding and proper utilization of climate and natural growth of native grasses; having the calves dropped to come along with the grass; eliminating nonbreeders and shy breeders; separating the breeding herd from the steers; feeding bulls during the winter and having them in good condition in spring; castrating bull calves when they are young by the bloodless operation; nubbing the sharp points of horns; marking and branding during the winter season and using a repellent smear on these parts; using common sense in handling and driving cattle; eliminating the use of the whip and catch dogs; avoiding the jamming of cattle into pens and chutes; eliminating unnecessary wounds and bruises of all kinds. In addition, Bruce and Sheely point out that livestock should be examined frequently for wounds and injuries and that all cases of screw-worm should be treated promptly; in treating animals one should avoid making wounds bleed; animals should be castrated, marked, branded, and dehorned during the winter months; bloodless emasculaters should be used for castration; all wounds should be treated with a repellent smear; ears of cattle, sheep, and goats should be inspected for ticks; breeding should be controlled so calves will be dropped in the early spring.

SUNDRY MAGGOT INFESTATIONS

Tumbu fly and Congo floor maggot. Several tropical African species of calliphorine flies are commonly referred to in the literature on myiasis, among them *Cordylobia anthropophaga* (E. Blanchard), the "tumbu fly." Austen describes it as being a "thickset, compactly built fly, of an average length of about 9½ mm. . . . Head, body, and legs are straw

FIG. 124. Congo floor maggot, *Auchmeromyia luteola.* (After Blacklock, in Martini's *Zoönosen der Haut in wärmeren Ländern.*)

yellow." According to Blacklock and Thompson[23] the eggs are deposited in excrement-polluted sand and soil. The incubation period may be as short as 24 hours. If contact is made with the skin of man or other animals the larvae penetrate the unbroken skin, forming furuncular swellings, or where multiple and contiguous infection occurs, extensive "'sloughing and gangrenous" conditions result. In 8 to 10 days the full-grown larvae, measuring 13 to 15 mm in length, leave the host and pupate in a few days. Wild rats are looked upon as the main reservoir of the infection in nature.

In the same locality with the "tumbu fly" there may also occur *Auchmeromyia luteola* (Fabr.), the larva of which is a bloodsucker and is known as the "'Congo floor maggot" (Fig. 124). The fly is commonly found in and about human habitations. The eggs are deposited in small clusters in various situations, such as on sleeping mats spread on the ground in huts, in dusty crevices, in dry sand, situations where the larvae may readily find suitable food. According to Roubaud,[24] whose treatise on this insect should be read by all interested in this subject, the eggs hatch in 36 to 40 hours. The larvae are remarkably resistant to extreme dryness and lack of food. They are nocturnal in their feeding habits, sucking the blood of sleeping persons, producing a wound by means of powerful buccal hooklets. They feed for 15 to 20 minutes, detach and hide in the crevices of mats, etc., during the day, repeating the attack almost nightly if hosts are available. The larval period may be as short as two weeks or, in the absence of food, perhaps as long as three months when the larvae pupate in protected situations. The pupal stage lasts from 11 to 12 days.

Wool maggots. Blowflies, inclusive of the screwworm fly already discussed, were undoubtedly at one time solely scavengers feeding in the maggot stage on carrion and animal wastes, but with the introduction of herds of domesticated animals they have acquired the habit of attacking living animals. The term "blowfly strike" refers to cutaneous myiasis produced by the development of blowfly maggots on living sheep. Froggatt[25] writes:

> Blowflies have always been in Australia, and under natural conditions before such disturbing elements as cattle, horses, sheep, and rabbits were introduced, they were simply scavengers or flesh-flies that deposited their eggs in animal matter that happened to be festering in the sun. In the study of economic zoölogy the acquisition of new habits by an insect is no uncommon occurrence. In the case of the blowfly we know that first some one species learned the habit of blowing live wool from the practice (already acquired) of blowing the wool of sheep that had died from some other cause. The smell of the dead wool taught them that the damp or soiled wool on a live sheep was of a similar character, and once the maggots set up decomposition of the yolk and fibre the smell attracted other flies. Thus other species became sheep-maggot flies until all our common blowflies had learned the habit.
>
> To upset the balance of nature is always a dangerous undertaking, and there is not much doubt that it has been the destruction of the natural means of control of the fly and the provision of a much larger number of suitable breeding-grounds that has caused the remarkable increase in the number of flies. After great droughts landowners have had to fight millions of rabbits breeding all over the West, and poisoned water and poison-carts have been put to considerable use. Sometimes the dead rabbits were stacked up and left to rot; sometimes they filled up the water-holes; sometimes they were piled up feet high along the wire-netting fences. It can be easily understood that under such conditions the blow-flies must have increased a millionfold. Again, the poisoning of dingoes and rabbits has caused the death of many of our hawks and carrion crows, and I feel quite sure that one of these birds in tearing up a carcass will often kill far more maggots than a dozen of the smaller insectivorous birds that capture the flies, but often only after they have deposited their eggs on the sheep.
>
> The next, and perhaps the most important, factor in the development of the sheep-maggot pest is the work of sheep-breeders themselves. Forty years ago there were many thousands of Merino sheep of the bare-belly, bare-legged

type, which did not produce a third of the weight of wool of the modern, improved Merino. The ambition of every sheep-breeder has been to make every inch of the sheep's skin grow wool, and in the case of some classes of Merinos to produce a wrinkled skin, giving even more wool-bearing surface. A sheep clothed with such a mass of thick, close, fine wool, fitting closely over the rump and round the tail, is sure to get more or less stained and damp round the crutch, and to attract flies. This artificial increase in weight, quantity and fineness of wool is accompanied, too, by an increased secretion of yolk, which rising from the skin and spreading all through the wool fibre, forms an additional attraction for the flies, and supplies food for the maggots.

The following species of flesh flies are listed by Froggatt and other authors as attacking live wool: *Phormia regina* (Meig.), known as the black blowfly because of its blackish-blue color and regarded as the most important wool-maggot fly in the United States; *Phaenicia* (= *Lucilia*) *sericata* (Meig.), an important sheep maggot fly in the United States, Australia, and other parts of the world; *Chrysomya albiceps* (Wied.) [*Chrysomya* (*Pyconosma*) *rufifacies* (Macq.)], common in Australia and India; *Callitroga* (= *Cochliomyia*) *americana* (C. & P.), a menace to sheep raising, particularly in the southern United States, Mexico, and elsewhere in the range of this insect; *Chrysomya bezziana* (Villen.) [*Chrysomya flaviceps* (Wlk.) = *Chrysomya dux* (Esch.)], an important Australian and Oriental sheep maggot fly; *Wohlfahrtia magnifica* (Schin.), the principal sheep maggot fly of southern Russia, also causes human myiasis in Egypt.

Control of wool-maggot flies. The following measures are recommended for the prevention and treatment of "blowfly strike" by various workers, among them Babcock and Bennett[26] of the Texas Agricultural Experiment Station, and Belschner[27] of the New South Wales Department of Agriculture. (1) *Carcass burning:* dig a trench along the back of the carcass nearly as wide as the animal and as long, and 12 to 14 inches deep; fill trench with wood (one-quarter cord of wood is sufficient to burn cow or horse) or cow chips; start fire at windward end and entire carcass will be wholly consumed within 12 to 24 hours. (2) *Poisoning:* a freshly killed animal is very attractive to flies, and if treated with an arsenic solution will kill every fly that feeds on it, or if a carcass is already alive with maggots, a similar treatment will kill these also. For this purpose arsenite of soda at the rate of 3 pounds to 30 gallons of water is recommended. (3) *Crutching*, if properly done, prevents the attack of flies by reducing the opportunity for the wool of the crutch, rump, and flanks to become wet and soiled and thus attractive to flies. It consists particularly of shearing the wool away from the breech, over the tail and down the back of the hind legs. (4) *Jetting:* applying an arsenic solution (one pound of arsenic to 40 gallons of water) by means of a single jet under pressure of 125 pounds to the rump of the sheep; in this way the arsenic is forced through the wool to the skin, where it dries and protects the animal for a longer time than spraying. (5) *Parasites:* the use of parasites against the flies.

Foot maggots of animals. A lameness that varies in degree and is traceable to myiasis commonly occurs in Philippine cattle, carabaos, and goats and is caused by the larva of *Boöponus intonsus* Ald. Woodworth and Ashcraft[28] state that the eggs of this fly are attached to the hairs of the lower portions of the legs of the host animal, the incubation period varying from three to five days. The young larvae work their way down to the foot and enter the flesh, leaving their posterior ends exposed, and when full grown at the end of two to three weeks they leave the flesh and drop to the ground where they bury themselves and pupate, the pupal period requiring about 10 days.

The method of treatment suggested by these authors consists of cleaning the affected area with soap and water, removing as many maggots as possible and applying a chloroform pack, followed by heavy applications of *pix liquidae* every third day until the lesions heal. As a preventive means, daily inspection is recommended of all cattle, carabaos, and goats, especially during the dry season when the animals should have access to plenty of water and mud as wallows.

Toxic effect of ingested fly larvae. A disease known as "limberneck" in chickens is believed to be traceable at least in part to the ingestion of large number of fly larvae such as those of *Lucilia caesar* (Linn.), *Phaenicia sericata* (Meig.), and no doubt other species of flesh flies, or of meat infested with type C *Clostridium* (= *Bacillus*) *botulinum* (Van Ermengem).[29] It is believed that the organism multiplies in the unburied bodies of dead animals, as flesh is a favorable medium for growth, and that the flesh flies developing in the carcasses become infected, and in turn chickens eating the maggots (or the flesh) acquire the infection. This is another good reason why dead animals should be speedily and safely disposed of, preferably by incineration.

Bloodsucking maggots of birds. In a study of bloodsucking fly larvae in birds' nests Plath[30] found an average of 61 per cent of a total of 63 nests examined to be infested with an average of 47 maggots per nest. The species of birds were the Nuttall sparrow, California purple finch, greenback goldfinch, willow goldfinch, and California brown towhee. In a later paper the same author[31] adds several other species, namely, the rusty song sparrow, cliff swallow, Oregon towhee, yellow warbler, western robin, russet-backed thrush, and cedar waxwing. The species of flies responsible for the maggots were found to be *Protocalliphora azurea* (Fallén), *Phormia metallica* Town., both blowflies, and *Hylemyia nidicola* Ald., an anthomyid fly. Storer[32] reports taking 76 larvae and 24 pupae from the nest of the Bailey mountain chickadee. These were identified by Aldrich as *Protocalliphora splendida* Macq. variety near *hirudo* S. & D.

Plath concludes that from 5 to 10 per cent of the parasitized nestlings die from loss of blood, and some of them which do become full-fledged are so weakened by the loss of blood that they fall an easy prey to rapacious animals.

Guberlet and Hotson[33] (1940) reported larvae of *Protocalliphora hesperia* S. and D. infesting a young nestling English sparrow (*Passer domesticus*) at Seattle, Washington. These authors describe the larvae and reared adult flies which emerged in 14 days after the maggots had been removed from the bird and placed in soil to pupate.

OBLIGATE MYIASIS

Oestrid flies, now segregated into four families, were heretofore placed in one family, the Oestridae. The larvae of all oestrid flies are obligate parasites in the digestive tract or other parts of the body of many mammals. *Bots* are the larvae of members of the family Gasterophilidae, medium-sized, 9–18 mm long, stout, pollinose flies; the eyes are large; the antennae are depressed in facial grooves, the arista is bare; mouth parts are rudimentary; the thorax is pilose; the wings are large, the apical cross vein is absent, the squamae are small. *Warbles* are the larvae of the family Hypodermatidae, large robust, hairy, bee-like flies; with arista bare, palpi small or absent; mouth parts rudimentary; wings large, apical cell open. The term "warbles" is also applied to the larvae of the family Cuterebridae, large hairy flies often black and white in color. As in other oestrids the mouth parts of these flies are nonfunctional although there is a large oral orifice; the palpi are very small; the arista is naked or plumose; wings and squamae are large. *Head bots* refer to the larvae of the Oestridae (in a restricted sense), bee-like flies known as "nose flies"; with eyes naked and small, antennae depressed in sunken facial furrows, mouth parts vestigial, wings and squamae large; the females are larviparous.

The horse botflies belong to the family **Gasterophilidae** with only one genus, *Gasterophilus*,[34] and four North American species, (1) *Gasterophilus intestinalis* (DeGeer), with cloudy patches near the center and apex of the wings and possessing a prominent spur on the third trochanter; (2) *G. inermis* (Brauer), which also has wings with cloudy patches, but the trochanter is without a spur; (3) *G. haemorrhoidalis* (Linn.), without cloudy patches on the wings, and with the anterior basal cell markedly shorter than the discoidal cell and the tip of the abdomen reddish; and (4) *G. nasalis* (Linn.), also with hyaline wings and anterior basal cell equal or nearly equal in length to the discoidal cell. The flies of this genus are somewhat smaller than honeybees, the mouth parts are rudimentary, the antennae are very small and sunken in pits, the arista is bare, and according to Curran the apical cross-vein is absent; the vein closing the discal cell is also absent and the fourth and fifth veins evanescent apically; the squamae are small; the ovipositor of

FIG. 125. Eggs of the horse botfly, attached to a hair of the host. ×20.

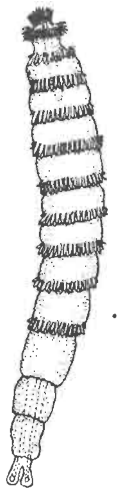
the female is large and protruberant. They are strong fliers. The larvae live in the stomach and intestines of horses.

Gasterophilus intestinalis (DeGeer) [*Gasterophilus equi* (Clark)] is the common horse botfly or nit fly, a widely distributed, nearly cosmopolitan, species commonly seen in the United States during midsummer

A B C

FIG. 126. Horse botflies. (*A*) *Gasterophilus intestinalis,* (*B*) *G. haemorrhoidalis,* and (*C*) *G. nasalis.* (Adapted after Hearle.)

to early autumn, June to September. The light yellow eggs (Fig. 125) are firmly attached to the hairs of the forelegs, belly, flanks, shoulders, and other parts of the body of the horse, but chiefly on the inside of the knees where they are accessible to the tongue, teeth and lips. The female fly (Fig. 126A) hovers from two to three feet away from the animal, darting swiftly and repeatedly at the horse, each time attaching an egg to a hair. Wells and Knipling[34] report one fly placing 905 eggs in 2¾ hours. Friction and moisture from the tongue of the horse seem necessary for the hatching of the eggs; the incubation period is from 7 to 14 days, but may be greatly prolonged by cool weather so that viable eggs may be found unhatched on the hair of the horse until late autumn, long after the flies have disappeared. Eggs kept in dry cartons may remain viable at room temperature for at least three months and hatch when moistened with saliva. The larvae on hatching (Fig. 127) are provided with an



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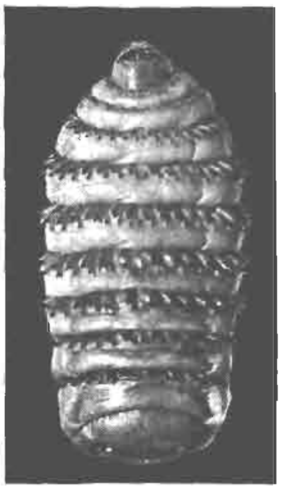

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FIG. 127. Newly emerged larva of the horse botfly. ×60.
FIG. 128. Larva of the horsebot, *Gasterophilus intestinalis*. ×4.

armature which enables them to excavate galleries in the subepithelial layer of the mucous membrane. Wehr,[35] who has studied the behavior of the larvae, states, "Many very small, thread-like subepithelial burrows, ramifying in every direction, were visible on the anterior half of the tongue, while those on the posterior half of the tongue were larger in size. Larvae were visible at the terminations of many of these galleries." Wehr found that newly hatched larvae when placed on the tongue of a freshly killed rabbit almost immediately began burrowing and within one minute nearly all became entirely embedded in the mucous membrane. From the mouth in the normal host the larvae apparently pass rapidly to their preferred site in the alimentary canal, the left sac or esophageal portion of the stomach, where third (even second) instar and the final instar larvae remain fixed with little or no change in position until the following spring and early summer, when they detach themselves and pass out of the intestine with the droppings. They are then from 1.5 to 2 cm in length (Fig. 128). Pupation takes place shortly thereafter in loose earth or in dry droppings. The pupal stage varies considerably, depending upon moisture and temperature, but the usual time is from three to five weeks when the winged botfly emerges. Copulation soon takes place and egg laying begins in early summer. The life history requires about one year.

Gasterophilus haemorrhoidalis (Linn.) (Fig. 126B) is a North American and European species. It is commonly known as the "nose fly," because the female fly forceably "strikes" the animal in the region of the nose, where it attaches its black eggs to the fine hairs of the lips or may even thrust the screw-like stalks, with which the eggs are provided, directly into the tender skin. Because of the orange-red terminal segments, this fly is also known as the "redtailed bot."

The fully grown larvae have the habit of moving from the stomach during the early spring and attaching close to the anus before finally dropping to the ground.

Gasterophilus nasalis (Linn.) [*G. veterinus* (Clark)] (Fig. 126C) is the chin fly or throat botfly, also a widely distributed species said to be especially abundant in the Rocky Mountain region. This fly is very annoying to horses, since its eggs are attached to hairs under the jaws, and when the fly darts at the throat, it causes the animals to throw their heads up as though struck under the chin. Egg deposition takes place during late spring and early summer. Unlike *G. intestinalis* (DeGeer) moisture is not required for the liberation of the larvae. The larvae hatch in from four to five days. The newly hatched larvae travel along the jaw and enter the mouth between the lips. There seems to be no tendency to burrow through the skin of the throat. From the mouth of the horse the larvae travel to their preferred site in the alimentary tract, the pyloric portion of the stomach and the anterior portion of the duodenum, where they are found in groups and remain for 10 to 11 months, i.e., until they are mature. Pupation takes place in a few hours after the larvae are voided with the manure during the early summer. The pupal stage requires about three weeks.

Gasterophilus inermis (Brauer) is a European species recently reported from North America (Illinois) by Knipling.[36] The eggs are deposited on the hairs of the cheeks of the host and, according to Knipling, when hatched the larvae penetrate the epidermis and work their way under it until the mouth is reached, thence after molting in the epithelial layer of the cheek they migrate to the rectum, where they remain until fully mature. The larvae drop to the ground and pupate as do other bots. The pupal period is 21 days in the case of Knipling's material. The adult is small, and "densely covered with silvery to yellowish hair, contrasting with the more or less orange-colored hair in *G. haemorrhoidalis, G. nasalis* and *G. intestinalis*."

Gasterophilus pecorum (Fabr.) is a European and African species, which does not occur in the United States. It is said to deposit its eggs on the food of the host animal and on nearby objects. The larvae burrow into the mucous membrane of the mouth, migrating soon to the stomach and rectum.

Pathogenesis. While a moderate infestation of bots will give no outward indications, a heavy infestation will be indicated by digestive dis-

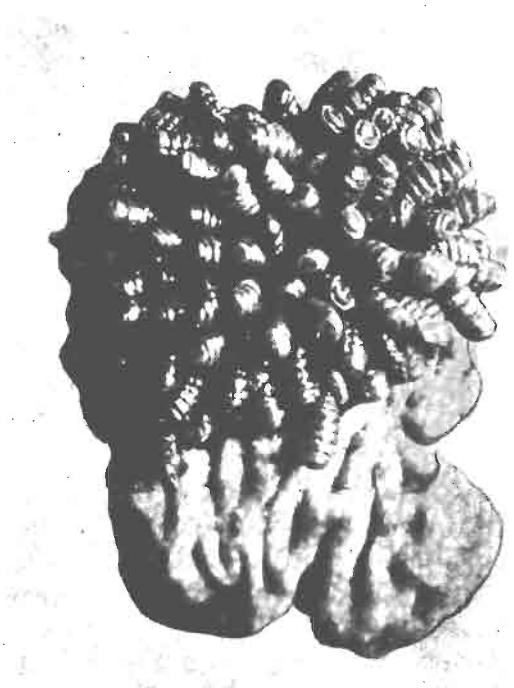


FIG. 129. Horse bots, *Gasterophilus intestinalis*, attached to mucous lining of the stomach of a horse. ×.75. (Photograph by Wherry.)

orders (which may of course be traceable to other causes as well). The discovery of bots in the manure is sufficient evidence. A light infestation is probably of no consequence; there are indeed some individuals who erroneously maintain that a horse must have at least a few bots in order to be well.

The injury which bots produce is: (1) abstraction of nutriment, both from the stomach and its contents; (2) obstruction to the food passing from the stomach to the intestine, particularly when the larvae are in or near the pylorus; (3) irritation and injury to the mucous membrane of the stomach (Fig. 129) due to the penetration of the oral hooklets; (4) irritation of the intestine, rectum, and anus in passage.

Treatment. Although carbon disulphide had been in use for many years in Europe as a remedy for horse bots, no wide use was made of the chemical for this purpose in the United States until after the experimental work of Hall[37] in 1917. The treatment should be administered only by veterinarians. After preparation of the animal by fasting it for 18 hours (water may be allowed), the chemical is administered in gelatine capsules at the rate of 1.5 fluid drams for each 250 pounds of weight. The bots begin to appear in the animal's droppings in five or six hours. Purgatives should not be used in this treatment.

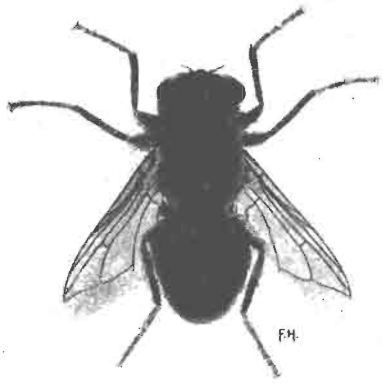


FIG. 130. The common ox warble fly or heel fly, *Hypoderma lineata.* (Adapted after Hearle.)

Cattle grubs (ox warbles) are the larvae of flies belonging to the family **Hypodermatidae,** genus *Hypoderma,* the heel flies. Although the normal host is cattle, horses and humans are occasionally parasitized. Persons dealing with cattle are familiar with the tumorous swellings on the backs of cattle during the late winter and early spring, and most stockmen have squeezed out the large grubs which inhabit these tumors. There are two well-known species: *Hypoderma lineata* (de Villers), widely distributed in the United States, as well as in Europe and Asia; and *Hypoderma bovis* (DeGeer), less widely distributed and more northerly in its occurrence[38] in the United States; it is found particularly in the New England states, but it does occur in California.

Hypoderma bovis (DeGeer) is the larger of the two species, measuring about 15 mm in length, while *H. lineata* (de Villers) measures about 13 mm. The former has the thorax covered with dense yellow hairs in front and black ones behind, with the terminal yellow hairs on the abdomen, while the latter, *H. lineata* (de Villers) (Fig. 130), has a fairly uniform hairy covering of mixed brownish black and white with four prominent smooth and polished lines on the thorax, the hairs of the terminal segment of the abdomen being reddish orange. The full-grown larvae are easily distinguished by examination of the spiny armature: *H. bovis* (DeGeer) has the last two segments entirely devoid of spines, while *H. lineata* (de Villers) has only the last one smooth. It may also be said that the full-grown larva of the former measures from 27 to 28 mm in length and that of the latter about 25 mm (Fig. 131).

Life history and habits. The eggs of both species are laid on the hairs of cattle, *H. lineata* (de Villers) attaching as many as a dozen in a row to a single hair, while *H. bovis* (DeGeer) is said to attach but a single egg to a hair. As many as 800 eggs, it is stated, may be laid by a female of either species (Warburton[39]). The eggs are evidently deposited by preference on the legs from the hock to the knee of the standing animal,

FIG. 131. Larva or grub of the ox warble fly, *Hypoderma lineata*. ×1.3.

but in recumbent animals the eggs may be attached to the hairs of other parts of the body close to the ground. Although no pain is inflicted at the time of oviposition, cattle become terror-stricken when the fly is discovered and gallop madly for water or shade in which to stand to escape the enemy. This is termed "gadding" and often spreads to the whole herd.

The eggs of both species hatch within a week and the tiny armored larvae crawl down the hairs of the host and bore either directly into the skin or into the hair follicles.[40] Knipling's[41] studies indicate that there are only three stages, although others have suggested there might be four or five.

Bishopp, Laake, and Wells[42] state that the eggs are ordinarily deposited only on sunny days, although *H. bovis* (DeGeer) may continue to oviposit during cloudy periods. A stiff breeze apparently deters the flies, although egg deposition was observed at temperatures as low as 40° F. The eggs hatch in from three to four days, and the larvae penetrate the skin causing considerable irritation. The larvae then work upward between the muscles and in a few months thereafter are found in the abdominal and chest cavities of the host. The above-mentioned authors state further, "During the following seven or eight months they constantly burrow about over the surface of the paunch, intestines, spleen, and other organs. Grubs are especially numerous between the muscular and mucous layers of the oesophagus or gullet. The grubs in these situations are slender and their length ranges from about one-tenth to about two-thirds of an inch. In the fall, winter, and spring the grubs migrate through the muscular tissues of the back and in a short time reach the under surface of the skin. During this last journey some of them enter the spinal canal and may burrow along the spinal cord for considerable distances. Soon after the skin is reached the grub cuts a minute hole through to the surface. At this time it is still slender and white and about two-thirds of an inch long, and is smooth except for small spines at each end. From one to five days later the grub molts for the third time." Upon emerging "from this molt the skin is closely set with spines. The body of the host now begins to isolate the invading parasite by forming a pocket or cyst around it. The growth of the grub from this time on is rather rapid, and a fourth molt occurs about 25 days after the third. In this last stage of its development the color gradually darkens, first becoming yellow, then brown, and finally almost black. During this entire development beneath the skin a breathing hole is kept open to the surface, and the grub lies with its two breathing pores, which are located on the posterior end, applied rather closely to the opening in the skin. As growth proceeds the hole in the skin is gradually enlarged." In late spring and early summer "at the end of the period of development in the back, which requires from 35 to 89 days, growth is complete, and the repulsive, spiny grub works its way out and falls to the ground." There the larvae crawl away into the loose earth or debris, becoming rapidly dark brown to black in pupation, and in from four to five weeks emerge as warble flies. The complete life cycle requires about a year.

The warbles begin to appear in the backs of cattle in some parts of California about October first, and Warburton reports mid-February as the time when the indications of newly forming dorsal tumors are most numerous in England.

In a summary of his work in Anatolia (Turkey) Kurtpinar[43] (1947) reports that of a total of 7,025 cattle examined 67.3 per cent were infested with *Hypoderma bovis* (DeGeer). The larvae appeared in the backs of cattle in January and were found there until the last of June; adults were found from early May until the end of June. Kurtpinar also reports that 75 per cent of 1,200 goats examined were infested by a different species, namely, *Hypoderma aegagri* Brauer.

Injury done. The injury done by the warbles is first that of *irritation* caused by their migrations in the body of the animal and later in their emergence from beneath the skin; secondly, the escape of the larva from the tumor leaves an open, running wound which persists for a long time and is attractive to screwworm flies and other tormenting insects. The direct pathogenesis is of minor importance, however, in the face of the economic loss produced by this insect.

Economic losses. The economic losses produced are: (1) Reduction in the milk secretion of cattle, which is estimated at from 10 to 20 per

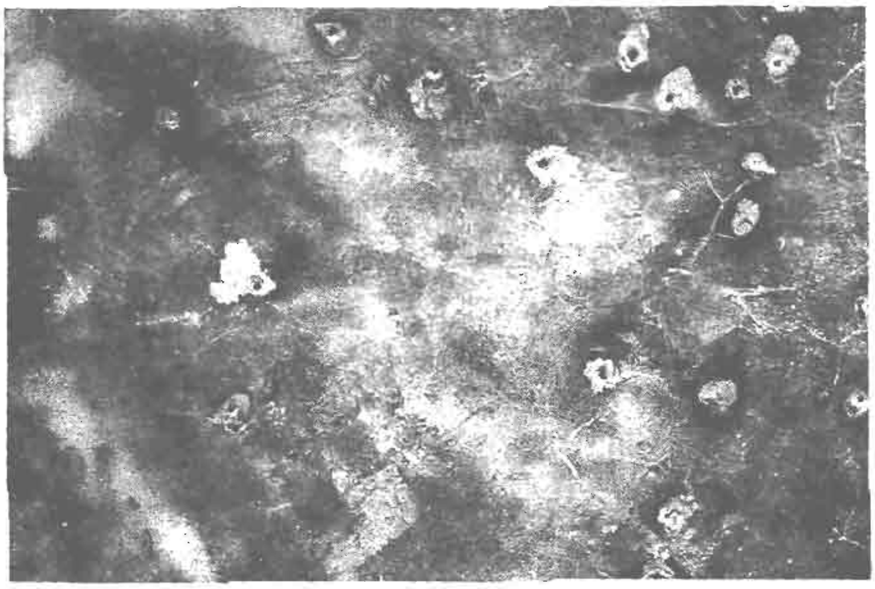


FIG. 132. A piece of sole leather 21 × 31.5 cm, showing work of ox warble. ×.3.

cent of the normal yield. (2) Loss of flesh due to the wild endeavor of the animals to escape from the flies and the irritating larvae (which is pointed out by Holstein: "A cow quietly grazing will suddenly spring forward, throw up her tail, and make for the nearest water at a headlong gait. Seemingly deprived at the moment of every instinct except the desire to escape, she will rush over a high bluff on the way, often being killed by the fall. This, with miring in water holes and the fact that cattle are prevented from feeding, causes the loss"). (3) Depreciation of the value of the carcass as flesh, which becomes greenish yellow and jelly-like in appearance at the points where the grubs are located, and is not fit for consumption. (4) Injury produced to the hide which becomes "grubby," full of holes, where the grubs have emerged (Fig. 132).

The following is quoted from *Tanners' Work* for October, 1913:

The case is recorded by Boas of Denmark of a cow which remained in poor condition and gave 33 pounds of milk per day. Forty-six grubs were extracted from this animal and eight days later she was giving 44 pounds of milk per day, continued to do so most of the summer and was in good flesh and condition in the fall. In this case the loss of milk due to the grub infestation was 25 per cent. The loss in flesh on account of grubs has been variously estimated at from $1.00 to $5.00 or more per head. If we assume that 25 per cent of all of the cattle in the United States are more or less infested with grubs, a quite conservative estimate, 50 per cent probably being nearer the actual percentage, the loss in flesh on account of grubs amounts to from $15,000,000 to $75,000,000 a year, the total number of cattle in the United States being calculated as approximately 60,000,000. If we also assume that infested milch cows lose 10 per cent in milk production and that 25 per cent of the 20,000,000 milch cows in the United States are affected, there should be added to the account a loss of not less than $30,000,000 per year.

As to the loss in hides it is stated by European tanners that a grubby hide is, on the average, less in value by one-third than a perfect hide, but for this country I have no definite information other than that grubby hides in the green state are commonly valued at one cent a pound less than perfect hides. On this basis the depreciation in value of a hide of average weight of 65 pounds, if grub-infested, would be 65 cents and the depreciation in the value of the estimated 15,000,000 grubby cattle of the United States so far as their hides are concerned thus amounts to $9,750,000. It is, however, quite probable that the actual loss in the value of hides when made into leather is much greater than this.

Without including the loss on account of the direct damage to beef carcasses from the presence of grubs, we may, on the basis of the foregoing, estimate the total loss from grubs in the United States in round numbers at from $55,000,000 to $120,000,000 per year.

Treatment. Although hand extraction of the warbles is possible in the case of small herds, the most effective method is the application of derris dust or derris washes in various formulations. Where wet applications are undesirable, dry powder may be used, as follows: equal parts of wettable sulfur (325 mesh) and finely ground (200 mesh or finer) cube or derris powder containing 5 per cent rotenone. One pound of the mixture applied with a dusting can or jar is sufficient to treat from 12 to 18 cattle. In preparing the mixture for application the cube or derris and wettable sulfur must be thoroughly mixed (Laake[44]).

For large-scale treatment of cattle, the use of rotenone suspensions applied by means of power sprayers is recommended by Wells.[45] A spray is made of cube or derris powder (5 per cent rotenone), 5 pounds; wettable sulfur, 10 pounds; and water, 100 gallons.* Application is made

* The most efficient control is obtained with 7½ pounds of 5 per cent rotenone to 100 gallons of water, plus a small amount of a wetting agent. (Douglas, J. R., and Furman, D. P., 1949. *J. Ec. Ent.*, 42:884–87.)

with a power sprayer, about 350 pounds pressure, directed downward to the backs of cattle at a distance of about 15 inches. The spray is applied as the animals pass through a chute. It is estimated that with one nozzle a herd of 1,000 cattle could be treated in one day.

Hearle[46] reports that derris as a wash has proved effective in large-scale experiments in several countries, including Canada. The formula recommended is standardized derris powder, one pound; soft soap one-quarter pound; water one gallon. The soft soap is boiled in a quart of water, and when cooled a little is poured into the derris powder in a bucket and mixed into a paste. Cold water and the remainder of the soap solution are then added slowly while stirring, to make up one gallon, and the mixture is ready for use. Standardized derris warble-fly powders ready for use are sold commercially. Before application the derris wash must be agitated frequently to ensure a good mixture. Although the keeping qualities are good if the liquid is placed in a well-stoppered container, it is advisable to prepare only an amount sufficient for immediate application. Where infestation is heavy, the wash should be liberally applied to the backs of the animals with a soft cloth or a worn stable brush, care being taken to cover completely the area affected by the grubs. In many cases, however, it is more economical to pour a little derris wash from a bottle on to each cyst, and to rub it in with the fingers. In the case of animals that are not stall-tied, a crush or dehorning chute is an aid to handling and treating them.

The date for the first application of the derris wash, varying in different parts of Canada, is in early spring when the swellings in the backs of the animals caused by the grubs first become conspicuous. In the interior of British Columbia this treatment is given in mid-February; in the prairie provinces and eastern Canada, about the third week in March. The second and third applications are made after intervals of 28 days, and the fourth after a further interval of about 35 days. A fifth dressing 35 days after the fourth is necessary in milder regions such as the interior of British Columbia, where the first application is made in mid-February. The intervals between the third and fourth, and fourth and fifth dressings are longer than between the preceding ones, being timed to accord with the larval development of *Hypoderma bovis* (DeGeer).

With regard to the treatment of beef herds for *H. lineata* (de Villers) during winter and early spring, the main objection of many ranches is that working cattle through a chute endangers the calf crop. In our experience these fears are baseless. Ice is a more serious menace, but the danger to stock from slipping may largely be overcome by sanding the yards. The April treatment can be combined with dehorning, if this is practiced.

Kurtpinar (*loc. cit.*) describes a treatment consisting of derris elliptica (Cooper's extract) and 12 per cent hydrogen peroxide plus one per cent methylene blue which killed the second-stage warbles in one to two minutes and the third instar in two to five hours; 2,664 cattle having a total of 17,232 larvae were treated. The average larval kill was 98.2 per cent.

The Caribou warble fly, *Oedemagena tarandi* (Linn.), is widely distributed over the range of its host both in northern Europe and northern North America. Hearle (*loc. cit.*) states that the fly is yellowish orange in color and has a bee-like appearance. The life history resembles that of the warble fly of cattle.

Head maggots, grub in the head, or **sheep bots** are the larvae of *Oestrus ovis* Linn., a very widely distributed species belonging to the family *Oestridae.* The fly (Fig. 133) is about 12 to 14 mm in length,

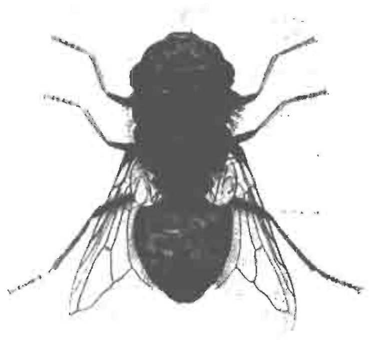


FIG. 133. The sheep botfly or head maggot fly, *Oestrus ovis.* (Adapted after Hearle.)

smaller than a honeybee, which it resembles; it is yellow to brownish gray in color, and hairy. The abdomen is variegated with brown and straw yellow; the feet are brown. It is further described by Osborn as follows:

> The under side of the head is puffed out and white. The antennae are extremely small and spring from two lobes which are sunk into a cavity at the anterior and under part of the head. The eyes are purplish brown, and three small eyelets are distinctly visible on the top of the head. It has no mouth and cannot, therefore, take any nourishment. The wings are transparent and extend beyond the body, and the winglets (calypteres) which are quite large and white, cover entirely the poisers. It is quite lazy and, except when attempting to deposit its eggs, the wings are seldom used.

Life history. The "head maggot fly" deposits living young during early summer to autumn in the nostrils of sheep and goats and may also attack human beings. One female fly may deposit as many as 60 larvae in an hour. The larvae at once begin to move up the nasal passages, working their way into the nasal and frontal sinuses often as far as the base of the horns in rams and attach themselves to the mucous membranes. Here numbers of these whitish grubs may be found wedged in closely in various conditions of development. The posterior ends of the larvae present conspicuous spiracles. The grubs reach full growth with a length of from 25 to 30 mm by the following spring, a larval period of from 8 to 10 months. At the end of this time they work their way out of the nostrils (they are usually sneezed out), fall to the ground, bury themselves in the earth, and pupate in a few hours. The pupal period lasts from three to six weeks and over, 19 to 34 days according to Fallis[47] who found that reared flies lived on an average of 16 days, one surviving as long as 28 days. Fallis also found that the complete development of the parasitic stage in spring lambs in Texas and New Mexico required from 2½ to 3½ months.

Symptoms. In the presence of the fly the sheep (or goats) are very much excited, shake the head, rush with their noses between their fellows, push their noses into the dust, snort, and otherwise indicate that they are trying to escape something that persists in entering the nostrils. Once infected there is a purulent discharge from the nostrils, vigorous shaking of the head, and perhaps the occasional discharge of a maggot, loss of appetite, grating of the teeth; and, when the animal walks, the fore feet are lifted in a pawing movement. The great majority of the cases do not result fatally, but death often comes in a week or less after the appearance of aggravated symptoms.

Grub-in-the-head is distinguished from "gid," caused by a larval tapeworm, *Multiceps multiceps* (Leske) [= *Coenurus cerebralis* (Batsch)], in that the former is always associated with purulent discharges from the nostrils, absent in the latter, that the symptoms of the former appear during the summer, and that the latter occurs ordinarily in lambs and yearlings only. There is no undue sneezing or rubbing of the nose in gid. Because of the mucus exuding from the nostrils of the sheep, the infection is commonly known as "snotty nose."

Treatment. Materials such as snuff, pepper, etc., may be introduced into the nostrils or sprinkled among the flock, to induce violent sneezing, which causes the expulsion of many of the larger grubs. Law recommends the injection of benzine, lifting the sheep's nose somewhat and pouring a teaspoonful of the remedy into each nostril. The lower nostril into which the benzine is poured is held shut for thirty seconds; the other side is then turned and the treatment repeated. The application is repeated daily or more often until the maggots are all expelled.

Prevention. "Salt logs" are used in sheep pastures by some sheep raisers. These logs are made by boring 2-inch holes at intervals of about 6 inches along the length on top. Salt is placed in the holes, which are kept about half full, and in turn the edges of the holes are repeatedly smeared with pine tar, or other repellent material. In endeavoring to reach the salt the sheep involuntarily smears its nose with the substance, which protects it to some extent against the head maggot fly.

Head maggot of horses. An important species of head maggot attacking horses in Russia and parts of Europe and in Egypt is *Rhinoestrus purpureus* (Brauer). Its habits are said to be similar to those of *Oestrus ovis* Linn. Like other species of related genera it may attack man either in the nose or eye.

Head maggot of deer. Deer, reindeer, elk, and other related wild animals are commonly infested with head maggots (Fig. 134); among these are the European species *Cephenemyia stimulator* Clark in the roe deer, *C. rufibarbis* Meig. in the red deer, *C. ulrichii* Brauer in elk, and *C. trompe* Linn. in reindeer, also in Alaskan reindeer. Among the

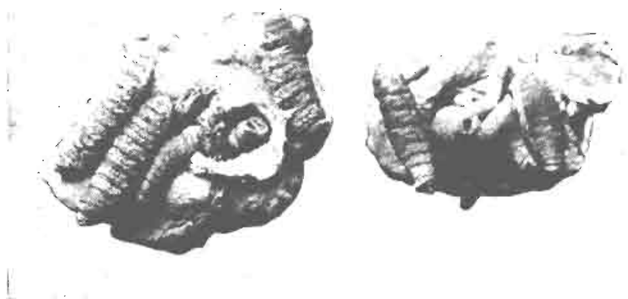


FIG. 134. Head maggots attached to tissue in nasal sinuses of the deer. ×.6.

known species in the United States besides *C. trompe* are *C. phobiger* Clark (a robust black species with yellow pile on thorax; legs black) from the white-tailed deer (*Odocoileus virginianus*), eastern United States; *C. pratti* Hunter from black-tailed deer (*Odocoileus hemionus*) in the western United States, often at high elevations; *C. jellisoni* Town. from the Pacific coast black-tailed deer (*Odocoileus hemionus columbianus*).

Dermal creeping myiasis of man is commonly caused by the wandering larvae of flies belonging to the family **Cuterebridae**, particularly *Dermatobia hominis* (Linn.); and also *Hypoderma lineata* (de Villers) and *Hypoderma bovis* (DeG.), both known as ox warble flies, belonging to the family *Oestridae.*

Dermatobia hominis (Linn.) (Family **Cuterebridae,**) is commonly found in Central and South America and Mexico. The larva is known in its early stage as *ver macaque* and in its later stages as *torcel* or *berne.* The fly measures from 12 to 16 mm in length and is entirely brown in color. This fly parasitizes a large number of species of mammals and even birds. It has been found in cattle, pigs, dogs, mules, monkeys, man, and various wild animals. In man the larva has been reported from various regions of the body, mainly head, arm, back, abdomen, scrotum, buttocks, thigh, and axilla.

Although it is not certain that this *Dermatobia* does not deposit its eggs directly on or in the human skin, it is now known that several species of mosquitoes, bloodsucking flies, and ticks, particularly the mosquito *Psorophora (Janthinosoma) lutzii* (Theo.) and possibly other insects act as carriers of the eggs. The female *Dermatobia* is said to oviposit on the undersides of the bodies of the mosquitoes so that when the latter suck blood, it is possible for the eggs to come in contact with the warm-blooded host where either contact or warmth stimulates the larvae; rapid emergence results and entrance to the skin of the host is effected. The larval period in the body of the host is said to require about two and a half months when, like the *Hypoderma* species, the larvae leave the tumorous swellings they have produced, drop to the earth, and pass through a pupal period requiring from three to six weeks.

Dunn[48] (1930) has described the life history of the human botfly most accurately as the result of an infestation which he suffered in the Panama Canal Zone. In his case the fly *Limnophora*, not a bloodsucker, was the vehicle for the eggs. Two larvae were observed to enter the skin of his arm, requiring 42 minutes for the first and one hour and 35 minutes for the second. Dunn experienced "absolutely no sensation caused by the entrance of the (first) larva until after the first 30 minutes. Then, as the posterior end was being drawn inside, a sharp pricking, which lasted for about two minutes, was experienced." He states that there was at first a sharp itching at night, and by the end of two weeks the lesions had the appearance of small boils and by the end of three weeks these were excruciatingly painful. At the end of 46 days and 15 hours and 50 days and 15½ hours respectively the larvae emerged from the skin, causing "absolutely no pain or sensation." The pupal periods were from 22 to 24 days.

The foregoing account of an infestation of human bots supports the opinion of other authors that the larvae remain in a relatively fixed spot in the subcutaneous tissues. The larvae of the ox botfly on the other hand have the tendency to migrate in the subcutaneous tissues often for considerable distances.

The author[49] (1925) described the migrations of the larvae of *Hypoderma bovis* (DeG.) (Family Hypodermatidae) as follows:

Mr. C. is a ranch superintendent, spending much of his time on horseback. On a Sunday toward the end of July, 1924, he had ridden to a point known as Mission Ridge near San Jose, when feeling tired, he dismounted and lay down on the ground in the open and slept. He distinctly remembers that his shirt had rolled up above his belt, exposing his skin, but felt no irritation at the time. Whether this exposure was taken advantage of by the fly can only be a matter of conjecture. Several days later, exact time not remembered, soreness was experienced and a slight swelling in the region of the right groin appeared. In about a week the swelling had increased to the width of a hand with no discoloration. The swelling then crept downward toward the left side affecting the scrotum, thence downward along the left leg to the knee and calf, thence back up the left leg following about the same course to the left groin, thence across to the right groin and back again to the left and upward along the left side of the body, slightly anterior to the shoulder, thence downward to the upper right arm to near the elbow, when the arm could not be raised without great pain, thence the swelling travelled upward again to the neighborhood of the shoulder blade where a "hive-like" local swelling was formed without any itching sensation. Mr. C. stated that at this point he was "bothered" all night, and while rubbing his arm and manipulating his shoulder muscles a larva of some insect "popped" out. This emergence took place about the end of October (1924). The larva was placed in a vial for shipment but was lost in transit.

Relative to the second larva which was delivered to the writer in person on the day following its emergence, Mr. C. states that since October when the first larva emerged, no further swellings were observed, but soreness in the region of the thigh and lower abdomen, similar to severe strain, persisted. However, on January 28 (1925) he experienced a severe "soreness" in the region of his right thigh which gave much distress, particularly when walking. By that night a swelling had developed and the following day the muscular soreness continued to spread; by January 31 a hernia-like swelling had developed which enlarged upward and outward to the region of the belt-line, the lower hernia-like swelling gradually disappearing. Sunday night, February 1, a hive-like swelling as observed in the case of the first larva began to form, enlarging to an area of about four by eight inches. Tuesday evening, February 3, lymph exuded from a small opening near the middle of the swollen area. About a tablespoonful of lymph stained with blood was pressed out and in the process of manipulation a larva similar to the first "popped" out. This specimen was delivered to the writer February 4 in good condition and identified as a third-stage larva of *Hypoderma bovis* DeG. The larva was milky white in color, about 12 mm. in length by 2 mm. in width at the middle, tapering bluntly at both ends. Very little swelling and practically no discoloration were visible on examination, although the point of emergence was clearly seen.

Gasterophilidae as human parasites. The larvae of the horse botflies burrow freely, as already explained, and may cause a form of creeping *cutaneous myiasis* in human beings. *Gasterophilus intestinalis* (DeGeer) is the species usually involved.[50] The course made by these creeping larvae is quite tortuous and plainly visible. The infection causes severe itching. The larvae, which measure from 1 to 2 mm in length, can be easily extracted surgically.

Ophthalmomyiasis of man is more particularly traceable to the larvae of head-maggot flies of sheep, deer, and related animals. Cases traceable to *Oestrus ovis* Linn. [*Cephalomyia ovis* (Linn.)] of sheep and *Rhinoestrus purpureus* (Brauer) are frequently reported in European horses. Three first-stage larvae of *Oestrus ovis* Linn. measuring about 1 mm in length that had been removed from the eye of a patient in Honolulu by Dr. R. Faus were studied by the author[51] (Fig. 135). The attending physician reported that the three larvae were buried in the sclera and were extremely adherent to the conjunctiva, causing acute conjunctivitis, larchrymation, ulceration, and neurosis.

The larvae of *Oestrus ovis* Linn. at times invade the nasal cavities of man as is normally the case with sheep. Severe frontal headaches result.

Rodent bots. The larvae of the family Cuterebridae are commonly parasitic upon rodents, wild and domestic rabbits, and mice, which may

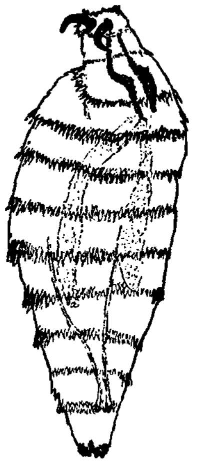


FIG. 135. Larva of *Oestrus ovis* from eye of human.

be severely infested with dermal tumors in which the large grubs lie. The adult flies are robust and bumblebee-like, having the scutellum elongate, the arista plumose or pectinate, the oral opening large, and the palpi small. There are four genera in this family, one of which includes the human botfly, *Dermatobia hominis* (Linn.) previously described; the other three, *Cuterebra, Pseudogametes,* and *Rogenhofera,* are parasitic on rodents and rabbits, and also on some other animals, including cats and dogs. *Cuterebra horripilam* Clark and *C. buccata* Fabr. usually infest rabbits; *C. peromysci* Dalmat infests white-footed mice (see Dalmat[52] for life cycle); *C. tenebrosa* Coq.; *C. beameri* Hall parasitizes pack rats; *C. emasculator* Fitch is the emasculating scrotum-inhabiting bot of squirrels.

Surgical maggots. Although now largely discontinued in favor of other treatments, the use of sterile maggots, maggot therapy, in the disinfection of osteomyelitis and other infected wounds was introduced into professional medical practice by Baer shortly after the end of World War I. Baer[53] had noticed that when men wounded in battle had been lying out on the ground for some time before being carried into dressing stations, their wounds were infested with maggots. He noticed particularly that these men whose wounds were crawling with maggots did not develop infections, as did the men whose wounds had received early treatment. It was discovered that the maggots were eating the dead tissue in which the bacterial infection throve; the maggots actually served as a "viable antiseptic." Baer's work attracted a great deal of attention and much experimentation followed, resulting in numerous publications by many investigators.

In 1932 Livingston and Prince[54] reported that filtered, uncontaminated products derived from the bodies of larvae in culture, when brought into contact with pyogenic organisms in petri dishes, destroyed the cultures.

The fly larvae used in earlier osteomyelitis treatment apparently belonged indiscriminately to the following species, namely, *Phaenicia sericata* (Meig.), *L. caesar* (Linn.), and *Phormia regina* (Meigen). It was assumed that all these species fed only on dead tissues. Stewart[55] has shown that even *Phaenicia sericata* (Meig.) larvae, which have been most commonly used in practice, will establish themselves in and feed upon normal healthy tissue, although they prefer necrotic tissue. He warns that they, and probably the larvae of *Phormia regina* (Meigen), *Lucilia caesar* (Linn.) and *Wohlfahrtia nuba* (Wiedemann), are potentially dangerous to normal tissue and must be utilized with care by an experienced person.

Stewart[56] also came to the conclusion that not only do the scavenging activities of the maggots play an important role in the successful results obtained, but the calcium carbonate, which was found to be constantly exuded by the larvae, is also of importance because of its property of alkalinizing the wound and of markedly increasing phagocytosis. Robinson in his later investigations discovered that allantoin and urea are present in maggot excretions, and that both have good effect in the treatment of osteomyelitis; however, because of its low cost and high solubility urea is now generally used, thus largely disposing of the use of maggots.

BIBLIOGRAPHY

1. James, Maurice T., 1947. *The Flies That Cause Myiasis in Man.* Washington, D. C.: Dept. Agric., in Misc. Publ., no 631. 175 pp.

2. Braun, M., und Seifert, O., 1926. *Die tierischen Parasiten des Menschen.* Leipzig: Curf Kabitzsch. x + 608 pp.

3. Hoeppli, R., and Watt, John Y. C., 1933. "Experiments on resistance

of dipterous larvae in connection with the problem of intestinal and urinary myiasis," *Chinese M.J.*, 47:1298–1306.

4. Desoil, P., et Delhaye, R., 1922. "Contribution à la pathogénie des myiases intestinales par l'étude de la résistance des oeufs et larves de Calliphorées aux agents physiques et chimiques intervenant dans le tube digestif." *Compt. rend. Soc. de biol.*, 87:1098–1100.

5. Causey, O. R., 1938. "Experimental intestinal myiasis," *Am. J. Hyg.*, 28:481–86.

6. Herms, W. B., and Gilbert, Q. O., 1933. "An obstinate case of intestinal myiasis," *Ann. Int. Med.*, 6:941–45.

7. Parker, G. H., 1922. "Possible pedogenesis in the blowfly, *Calliphora erythrocephala* Meigen," *Psyche*, 29:127–31.

8. Simmons, Perez, 1927. *The Cheese Skipper as a Pest in Cured Meats.* Washington, D. C.: Dept. Agric., in Dept. Bull., no. 1453. 55 pp.

9. Meleney, H. E., and Harwood, P. D., 1935. "Human intestinal myiasis due to the larvae of the soldier fly, *Hermetia illucens* Linné (Diptera, Stratiomyidae)," *Am. J. Trop. Med.*, 15:45–49.

10. Hall, M. C., and Muir, J. T., 1913. "A critical study of a case of myiasis due to *Eristalis*." *Arch. Int. Med.*, 2:193–203.

11. Hall, M. C., 1918. "A note regarding myiasis, especially that due to syrphid larvae," *Arch. Int. Med.*, 21:309–12.

12. Chevril, R., 1909. "Sur la myiase des voies urinaires," *Arch. de Parasitol.*, 12:369–450.

13. Mumford, E. P., 1926. "Three new cases of myiasis in man in the north of England," *Parasitology*, 18:375–83.

14. Stewart, M. A., 1929. "A case of dermal myiasis caused by *Phormia regina* Meig.," *J.A.M.A.*, 92:798–99.

15. ———, 1934. "A new treatment of traumatic dermal myiasis," *J.A.M.A.*, 103:402.

16. Cushing, E. C., and Patton, W. S., 1933. "Studies on the higher Diptera of medical and veterinary importance. *Cochliomyia americana* sp. nov., the screwworm fly of the New World," *Ann. Trop. Med.*, 27:539–51.

17. Laake, E. W., 1936. "Economic studies of screwworm flies, *Cochliomyia species* (Diptera, Calliphorinae), with special reference to the prevention of myiasis of domestic animals," *Iowa State Coll. J. Sc.*, 10:345–59.

18. Gajardo-Tobar, R., and Honorato, Armando, 1947. "Anotaciones acerca de una epidemia de miasis humana," Hospital de Viña del Mar, Chile, 3:5–14.

19. Walker, E. M., 1922. "Some cases of cutaneous myiasis, with notes on the larvae of *Wohlfahrtia vigil* (Walker)," *J. Parasitol.*, 9:1–5 (3 plates).

20. Calero, Carlos, 1948. "Cutaneous myiasis in Panama," *J. Parastiol.*, 34:343–44.

21. Cushing, E. C., and Hall, D. G., 1937. "Some morphological differences between the screwworm fly, *Cochliomyia americana* C. and P., and other closely allied or similar species in North America (Diptera: Calliphoridae)," *Proc. Entomolog. Soc. Wash.*, 39:195–98 (2 plates).

22. Bruce, W. G., and Sheely, W. J., 1944. *Screwworms in Florida.* Gainesville: Univ. Florida in Agric. Extension Serv. Bull., no. 123, 28 pp.

23. Blacklock, B., and Thompson, M. G., 1923. "A study of the tumbufly, *Cordylobia anthropophaga* Grünberg, in Sierra Leone," *Ann. Trop. Med.*, 17:443–501 + 4 plates.

24. Roubaud, E., 1913. "Recherches sur les Auchmeromyies," *Bull. scient. France et Belgique*, 47:105–202.

25. Frogatt, W. W., 1922. *Sheep-Maggot Flies*. Dept. Agric., New South Wales, Farmers' Bull., no. 144. 32 pp.

26. Babcock, O. G., and Bennett, D. H., 1921. *The Screwworm and the Wool Maggot*. Texas Agric. Exper. Sta., in Circ. no. 27. 15 pp.

27. Belschner, H. G., 1937. *A Review of the Sheep Blowfly Problem in New South Wales, (Studies on the Sheep Blowfly Problem)*. Sydney: Dept. of Agric., New South Wales: pp. 1–60.

28. Woodworth, H. E., and Ashcraft, J. B., 1923. "The foot maggot, *Boöponus intonsus* Aldrich, a new myiasis-producing fly," *Philippine J. Sc.*, 22:143–56 (8 plates).

29. Bengston, Ida A., 1923. "A toxin-producing anaerobe isolated principally from fly larvae," U. S. Public Health Service, *Pub. Health Rep.*, 38:340–44.

30. Plath, O. E., 1919. "A muscid larva of the San Francisco Bay region which sucks the blood of nestling birds," *Univ. Calif. Pub. in Zool.*, 19:191–200.

31. ———, 1919. "The prevalence of *Phormia azurea* Fallén (larva parasitic on nestling birds) in the Puget Sound Region and data on two undescribed flies of similar habit," *Ann. Entomolog. Soc. Amer.*, 12:373–81.

32. Storer, T. I., 1929. "*Protocalliphora* in the nest of a mountain chickadee," *The Condor*, 31:227.

33. Guberlet, John E., and Hotson, H. H., 1940. "A fly maggot attacking young birds, with observations of its life history," *The Murrelet*, 21:65–68.

34. Wells, K. W., and Knipling, E. F., 1938. "A report of some recent studies on species of *Gastrophilus* occurring in horses in the United States," *Iowa State Coll. J. Sc.* 12:181–203.

35. Wehr, Everett E., 1933. "The life history of *Gastrophilus* larvae of the horse and lesions produced by the larvae," *Cornell Veterinarian*, 23:254–71.

36. Knipling, E. F., 1935. "*Gastrophilus inermis* Brauer, a species of horse bot not previously recorded from North America," *Entomological News*, 46:105–7.

37. Hall, M. C., 1917. "Notes in regard to bots, *Gastrophilus spp.*," *J. Am. Vet. M. A.*, 51 n.s., 5:177–84.

38. Bishopp, F. C.; Laake, E. W.; Brundrett, H. M.; and Wells, R. W.; 1926. *The Cattle Grubs or Ox Warbles: Their Biologies and Suggestions for Control*. Washington, D. C.: Dept. Agric., in Dept. Bull., no. 1369. 119 pp.

39. Warburton, Cecil, 1922. "The warble flies of cattle," *Parasitology*, 14:322–41.

40. Hadwen, S., 1915. "Warble flies—a further contribution on the biology of *Hypoderma lineatum* and *Hypoderma bovis*," *Parasitology*, 7:331–38 (2 plates).

41. Knipling, E. F., 1935. "The larval stages of *Hypoderma lineatum* de Villers and *Hypoderma bovis* DeGeer," *J. Parasitol.*, 21:70–82.

42. Bishopp, F. C.; Laake, E. W.; and Wells, R. W.; 1929. *Cattle Grubs or Heel Flies* with Suggestions for Their Control. Washington, D. C.: Dept. Agric., in Farmers' Bull., no. 1596. 22 pp.

43. Kurtpinar, Hasip, 1947. *Anadolu ehlî hayvanlarinda görülen* Hypoderma *nevileri, iktisadî önemi ve müscadelesine dair en uygun tedbirler üzerine araştirmalar. (Species of Hypoderma found in domesticated animals of Anatolia; their economic importance and most suitable control measures.)* Çalişmalar: 153, T. C. Tarim Bakanligi Ankara Yüsek Ziraat Enstitüsü. 60 pp.

44. Laake, E. W., 1942. "Dry application of cube or derris combination with wettable sulfur for the control of cattle grubs," *J. Econ. Entomol.*, 35:112–13.

45. Wells, R. W., 1942. "The use of power sprayers in the control of cattle grubs," *J. Econ. Entomol.*, 35:112–13.

46. Hearle, Eric, 1938. *Insects and Allied Parasites Injurious to Livestock and Poultry in Canada.* Dominion of Canada, Dept. Agric., in Publ., no. 604. 108 pp.

47. Fallis, A. Murray, 1940. "Studies on *Oestrus ovis L.*," *Canad. J. Research*, 18:442–46.

48. Dunn, L. H., 1930. "Rearing the larvae of *Dermatobia hominis* Linn., in man," *Psyche*, 37:327–42 (1 plate).

✓49. Herms, W. B., 1925. "A case of human myiasis caused by the ox-warble, *Hypoderma bovis* DeG.," *J. Parasitol.*, 11:149–50.

✓50. Bedford, G. V.; Williams, D. H.; and Newton, M. V. B.; 1933. "Creeping eruption, with special reference to cutaneous myiasis and report of a case," *Canad. M. A. J.*, 28:377–82.

51. Herms, W. B., 1925. "Ophthalmomyiasis in man due to *Cephalomyia (Oestrus) ovis* Linn.," *J. Parasitol.*, 12:54–56.

52. Dalmat, Herbert T., 1943. "A contribution to the knowledge of the rodent warble flies (Cuterebridae)," *J. Parasitol.*, 29:311–18.

53. Baer, W. S., 1931. "The treatment of chronic osteomyelitis with the maggot (larva of the blowfly)," *J. Bone & Joint Surg.*, 13:438–75.

54. Livingston, S. K., and Prince, L. H., 1932. "The treatment of chronic osteomyelitis; with special reference to the use of the maggot active principle," *J.A.M.A.*, 98:1143.

55. Stewart, M. A., 1934. "The rôle of *Lucilia sericata* Meig. larvae in osteomyelitis wounds," *Ann. Trop. Med.*, 28:445–60.

56. ———, 1934. "A new treatment of osteomyelitis: Preliminary report," *Surg. Gynec. & Obst.*, 58:155–65.

CHAPTER XVIII

LOUSE FLIES

FAMILY HIPPOBOSCIDAE

Order Diptera (Series Pupipara)

Characteristics of Hippoboscidae. Three families of flies constitute the Pupipara: (1) Hippoboscidae (louse flies), (2) Nycteribiidae (bat tick flies), and (3) Streblidae (bat flies). The bloodsucking parasitic flies belonging to the family Hippoboscidae are readily recognized as Diptera when winged. The larvae are retained within the body of the female, being nourished by special glands within the mother until time for pupation is reached; they are then extruded and pupation quickly follows, whence the term, "pupipara." The adult flies are described as follows by Williston: "Head flattened, usually attached to an emargination of the thorax; face short, palpi forming a sheath for the proboscis, not projecting in front of the head; antennae inserted in pits or depressions near the border of the mouth, apparently one-jointed, with or without a terminal bristle or long hairs. Eyes round or oval, ocelli present or absent. Thorax flattened, leathery in appearance; scutellum broad and short. Halteres small or rudimentary. Abdomen sac-like, leathery in appearance, the sutures indistinct. Legs short and strong, broadly separated by the sternum; tarsi short; claws strong and often denticulated." The members of this family are all parasitic in the adult stage upon birds or mammals. There are about 400 species widely distributed throughout the world. They range in size from 2.5 to 10 mm.

The sheep "tick" or ked, *Melophagus ovinus* (Linn.), is a wingless bloodsucking species, reddish brown in color, about 5 to 7 mm in length. It is a world-wide parasite of sheep and goats. The head is short and sunken into the thorax, the body sac-like, leathery, and spiny (Fig. 136).

Life history. The eggs are retained and hatch within the body of the female ked, where the larvae develop in about seven days and are extruded fully grown ready to pupate. The extruded larva pupates during the course of a few hours, becoming chestnut brown in color; the secretion with which it is covered hardens and serves to glue the pupa firmly to the wool of the host. The pupae are commonly found on infested animals in the region of the shoulders, thighs, and belly. Pupae may be found on sheep at all times of the year, though the time required for development in the winter is longer than in the summer. Swingle,[1] who has observed this insect very carefully, states that pupae require from 19 to 23 days to hatch in the summer, whereas 19 to 36 days are required during the winter on sheep kept in the barn and probably 40 to 45 days on sheep out of doors. The time required for the females to reach sexual maturity is from 14 to 30 days and over, when they begin extruding young at the rate of one about every seven to eight days. Swingle (*loc. cit.*) considers about four months as the average life of the insect, during which time from 10 to 12 pupae are deposited.

The entire life of the ked is spent on its host; when off the sheep the insects die in from two to eight days, the majority in about four days.

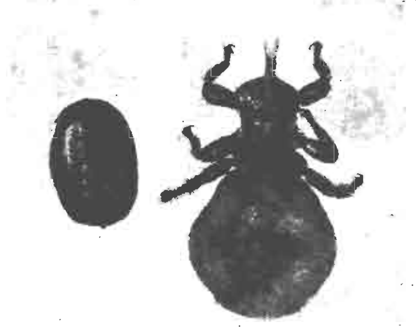


FIG. 136. The sheep "tick" or louse fly, *Melophagus ovinus*. (*Left*) pupa, (*right*) adult. ×4.5.

Damage done. The presence of a few louse flies on the body of a sheep does not materially affect it. Ordinarily the presence of the insect is indicated by the fact that the animal rubs itself vigorously, bites the wool, and scratches. Badly infested animals show emaciation and general unthriftiness. The injury to lambs is especially marked.

Control. Since the principal time for migration from the sheep to the lambs is at shearing when the insects are removed from the hosts with the wool, it is wise to take particular pains at this time to store the wool at some distance from the lambs. Inasmuch as the "ticks" die within a week when away from the host and cannot well crawl any great distance, this suggestion is well worth considering. Swingle states that "sheep free from 'ticks' can be kept for months beside a heavily infested one with a tight partition only three feet high between them without becoming infested. . . . A bunch of females placed in the wool of a sheep will be found in practically the same place for two days. Males, however, are most inclined to migrate." A flock of sheep once freed from "tick" can therefore be kept clean unless infested animals are introduced.

The writer has reasons to doubt the efficiency of "lime-sulphur" sheep-dip for the sheep "tick." Tobacco dips when used in 0.07 per cent solution will eradicate sheep "tick" according to Imes,[2] if two dippings are given with an interval of 24 to 28 days between them.

Louse flies of deer. *Lipoptena depressa* (Say) and *Lipoptena subulata* Coq. (= *L. ferrisi* Bequaert) are common parasites of deer in North America. These species are smaller than *Melophagus ovinus* (Linn.), but otherwise resemble it; they are wingless when established on the host but have well-developed filmy wings on emergence from the pupal stage (Fig. 137). The parasites have been found in chains, three or four individuals attached to one another, the first fly drawing blood from the host, the second with its proboscis thrust into the abdomen (dorsally) of the first, the third drawing on the second, and so on to the last in-

FIG. 137. Louse fly of the deer, *Lipoptena depressa,* showing wingless and winged forms. ×5.

dividual. *Lipoptena cervi* (Linn.), known as the "deer ked," is reported to be a common species on European deer, and according to Bequaert[3] has become naturalized in a few localities of the northeastern United States on the Virginia white-tailed deer. *Lipoptena mazamae* Rondani occurs on deer in South and Central America and in the southeastern United States.

Concerning *Lipoptena depressa* (Say), Hare[4] states that it is a common parasite of deer (genus *Odocoileus*) in western North America. The pupating larvae are smooth and clean, *dropping* from among the hairs of the host to the ground, where the entire pupal period is passed. At emergence the imago possesses fully developed though fragile wings and flies among the trees in the woodland haunts of the host. Upon reaching a deer these winged forms (volants) immediately crawl between the hairs and begin sucking blood. Here they remain as permanent parasites for the rest of their lives, soon loosing the wings by a simple process of wear. Under natural conditions the volants of *L. depressa* may be expected to live and remain active without a host for only one to eight days. Bloodsucking begins the first day after contact with the host, and sexual maturity is reached after about 12 days of feeding. After copulation the first larva is deposited by the female on the sixteenth to nineteenth day and thereafter singly at three-day intervals, up to 35 mature larvae. Adults of both sexes live up to four months on the host.

By actual count (Herman[5]) 1,350 flies have been reported from one host. Hare states that his observations indicate that the seasonal peak in California may extend into late fall in some localities; he found these flies abundantly on the wing on October 5 (1948) in Monterey County.

THE GENUS HIPPOBOSCA

Nine species of the genus *Hippobosca* are recognized as valid by Bequaert.[6] The wings are always well developed in the genus and are functional throughout adult life. With the exception of the ostrich louse fly, *Hippobosca struthionis* O. E. Janson, the species of this genus are ectoparasites of mammals. Except for *H. struthionis* O. E. Janson, an abundant parasite of the ostrich in South Africa, host specificity is not pronounced.

Hippobosca equina Linn. is a common species in England and is known as the "forest-fly." It is usually found on horses, mules, and donkeys, sometimes on cattle and other animals. *H. rufipes* v. Olfers is also primarily a parasite of equines and occurs in South Africa.

Hippobosca longipennis Fabr. (= *H. capensis* v. Olfers) is a louse fly reported by Bequaert to be commonly found on domestic dogs, especially on the pariah dog of India. It is also common in many parts of the Mediterranean region.

Hippobosca variegata von Mühlfeld (= *H. maculata* Leach) occurs on domestic cattle and equines and is widespread in distribution. *H. fulva* Austen off the hartebeest is known only from its type locality, northeastern Rhodesia. *H. hirsuta* Austen is reported to be a parasite of the water bucks and allied antelopes of Africa.

H. camelina Leach is a parasite of the camel and dromedary. *H. martinaglia* Bedford, found on antelopes in South Africa, is recognized as valid by Bequaert.

LOUSE FLIES OF BIRDS

The pigeon fly, *Pseudolynchia canariensis* (Macq.) [= *Lynchia maura* (Bigot) = *Olfersia maura* Bigot = *Lynchia lividicolor* Bigot], is an important parasite of domestic pigeons throughout the tropics and warmer regions of the world. It is found throughout the southern United States and California. The dark brown flies have long wings, 6.5 to 7.5 mm, and are able to fly swiftly from the host but usually alight near by. They move about quickly among the feathers of the host and bite and suck blood from parts that are not well feathered.

The mature larvae, at first pale yellow and later jet-black in color, are deposited on the body of the bird while it is quiet, but they soon roll off and collect in the nests. Bishopp[7] gives the duration of the pupal stage at from 29 to 31 days when the mean daily temperature is about

73° F. Thus the thorough and regular cleaning of the nests at intervals not to exceed 25 days is probably the most important single step in control. The pupae are very resistant, hence ordinary insecticides are of little use. Bishopp states that "one of the most effective and easily applied treatments for squabs is *fresh* pyrethrum powder, one to three pinches (depending upon the size of the squab) scattered among the feathers."

In addition to its evil effects as a bloodsucking parasite, the pigeon fly is the vector of pigeon malaria caused by *Haemoproteus columbae* Celli and San Felice.[8]

Bequaert[9] reports that *Microlynchia pusilla* (Speiser) is probably the most widely spread hippoboscid fly of doves. It is a strictly New World species; *Stilbometopa podopostyla* Speiser, also a New World species, has been taken from doves. *Ornithoctona erythrocephala* (Leach) has been reported from both wild doves and the domestic pigeon, as well as from birds of prey and others.

Pseudolynchia brunnea (Latreille) is regarded as a distinct species by Bequaert and is a parasite of birds of the nighthawk family. It is very dark brown in color, often nearly black.

Lynchia hirsuta Ferris is a common and abundant parasite of the Californian valley quail, *Lophortyx californica californica* Shaw, and has been shown by O'Roke[10] to be a vector of quail malaria caused by *Haemoproteus lophortyx* O'Roke. *Lynchia fusca* (Macquart) is a parasite of the owl, *Bubo virginianus pacificus* Cassin, in California and has been experimentally shown to be a vector of quail malaria by Herms and Kadner.[11] The flies feed readily on quail and deposit their mature larvae freely on these birds. The incubation period of the infection in the fly was found to be from 9 to 13 days and in the quail about 25 days.

Stilbometopa impressa (Bigot) is also a parasite of the Californian valley quail.[12] *Lynchia americana* (Leach) is characteristically a parasite of owls in North America.[13]

Bat flies are pupiparous bloodsucking parasites belonging to the family **Streblidae**; they are all parasitic on bats in tropical and subtropical climates. The members of the family may be separated from the **Hippoboscidae** by the large leaf-like palpi which project in front of the head and do not form a sheath for the proboscis. They differ from the **Nycteribiidae** (the spider-like bat flies) in that they do not have the head resting in a groove on the dorsum of the thorax. Little is known about the life history of these insects. The species of the family Streblidae have been reviewed by Kessel.[14]

Spider-like bat flies belong to the family **Nycteribiidae**. They are very small (2 to 3 mm long) wingless spider-like parasites of bats. Except for a very few species described from North and South America, they are primarily parasites of Old World bats. Ferris[15] has reviewed the New World species.

BIBLIOGRAPHY

1. Swingle, Leroy D., 1913. *The Life History of the Sheep Tick, Melophagus ovinus.* Laramie: Univ. of Wyoming, Agr. Exper. Sta. Bull., no. 99.

2. Imes, Marion, 1932. *The Sheep Tick and Its Eradication by Dipping.* Washington, D. C.: Dept. Agric., in Farmers' Bull., no. 798. 22 pp.

3. Bequaert, J., 1937. "Notes on Hippoboscidae, 5, The American species of Lipoptena," *Bull. Brooklyn Entomolog. Soc.*, 32:91–101.

4. Hare, John Edward, 1945. "Flying stage of the deer louse-fly, *Lipoptena depressa* (Say), in California," *Pan-Pacific Entomologist,* 21:48–57.

5. Herman, Carlton M., 1945. "Hippoboscid flies as parasites of game animals in California," *California Fish and Game,* 31:16–25.

6. Bequaert, J., 1939. "Notes on Hippoboscidae, 13. A second revision of Hippoboscinae," *Psyche,* 46:70–90.

7. Bishopp, F. C., 1929. "The pigeon fly, an important pest of pigeons in the United States," *J. Econ. Entomol.,* 22:974–80.

8. Huff, C. G., 1932. "Studies on Haemoproteus of mourning doves," *Am. J. Hyg.*, 16:618–23.

9. Bequaert, J., 1939. "Hippoboscid flies from North American doves," *Science,* 89:267–68.

10. O'Roke, E. C., 1930. "The morphology, transmission, and life history of *Haemoproteus lophortyx* O'Roke, a blood parasite of the California valley quail," *Univ. Calif. Publ. in Zool.*, 36:1–50.

11. Herms, W. B., and Kadner, C. G., 1937. "The louse fly, *Lynchia fusca,* parasite of the owl, *Bubo virginianus pacificus,* a new vector of malaria of the California valley quail," *J. Parasitol.,* 23:296–97.

12. Herman, Carlton M., 1944. "Notes on the pupal development of *Stilbometopa impressa,*" *J. Parasitol.*, 30:112–18.

13. Ferris, G. F., 1927. "Some American Hippoboscidae (Diptera: Pupipara)," *Canad. Entomologist,* 59:246–51.

14. Kessel, Q. C., 1925. "A synopsis of the Streblidae of the world," *J. N. Y. Entomolog. Soc.*, 33:11–34 (4 plates).

15. Ferris, G. F., 1924. The New World Nycteribiidae (Diptera: Pupipara). *Entomological News,* 35:191–99.

CHAPTER XIX

FLEAS

ORDER SIPHONAPTERA

Structural characteristics. "No part of the external anatomy of an adult flea could possibly be mistaken for that of any other insect. The head, the mouth parts, the thorax, the legs, the abdomen, the external genitalia, all present features that are not elsewhere duplicated among the hexapods." Snodgrass[1] (1946) continues to marvel, as well he may, "there are numerous peculiarities that strain the imagination for a plausible explanation, and the complexity of the male intromittent apparatus is almost beyond belief." Yet internally, except for the array of spines in the proventriculus, the flea is a fairly generalized insect.

Fleas constitute the order Siphonaptera (Siphunculata of some authors); they are laterally compressed, highly sclerotized, totally wingless, minute bloodsucking ectoparasites of warm-blooded vertebrate animals. The commoner species vary from 1.5 to 4 mm in length. The males are as a rule smaller, often considerably smaller, than the females; both sexes are bloodsuckers. The posterior pair of legs is strikingly adapted for leaping; some species, such as the chigoe fleas, are able to burrow partly into the skin of the host and are practically sessile.

The head is a highly specialized cranial capsule; it is set closely against the notum of the pronotum with a very short neck which precludes much movement of the head, but does allow some. On the sides of the head are depressions (grooves) that contain the tiny knobbed antennae (the knobs are segmented), and in front of these are the simple eyes when these are present. The position of a conspicuous bristle (ocular bristle) in certain position in front of the eye, may be useful in classifying fleas; also useful in classification is the presence in some species of a conspicuous comb of bold spines, *ctinidium,* located just above the mouth parts—the oral or *genal ctenidium* (Fig. 138).

The mouth parts of the adult flea include a minute *labrum,* a long, slender, unpaired *epipharyngeal stylet,* a pair of maxillae with paired *maxillary stylets,* a small *hypopharynx,* and a simple *labium.* Mandibles are lacking in the adult flea, though present in the larva in the form of toothed jaws.

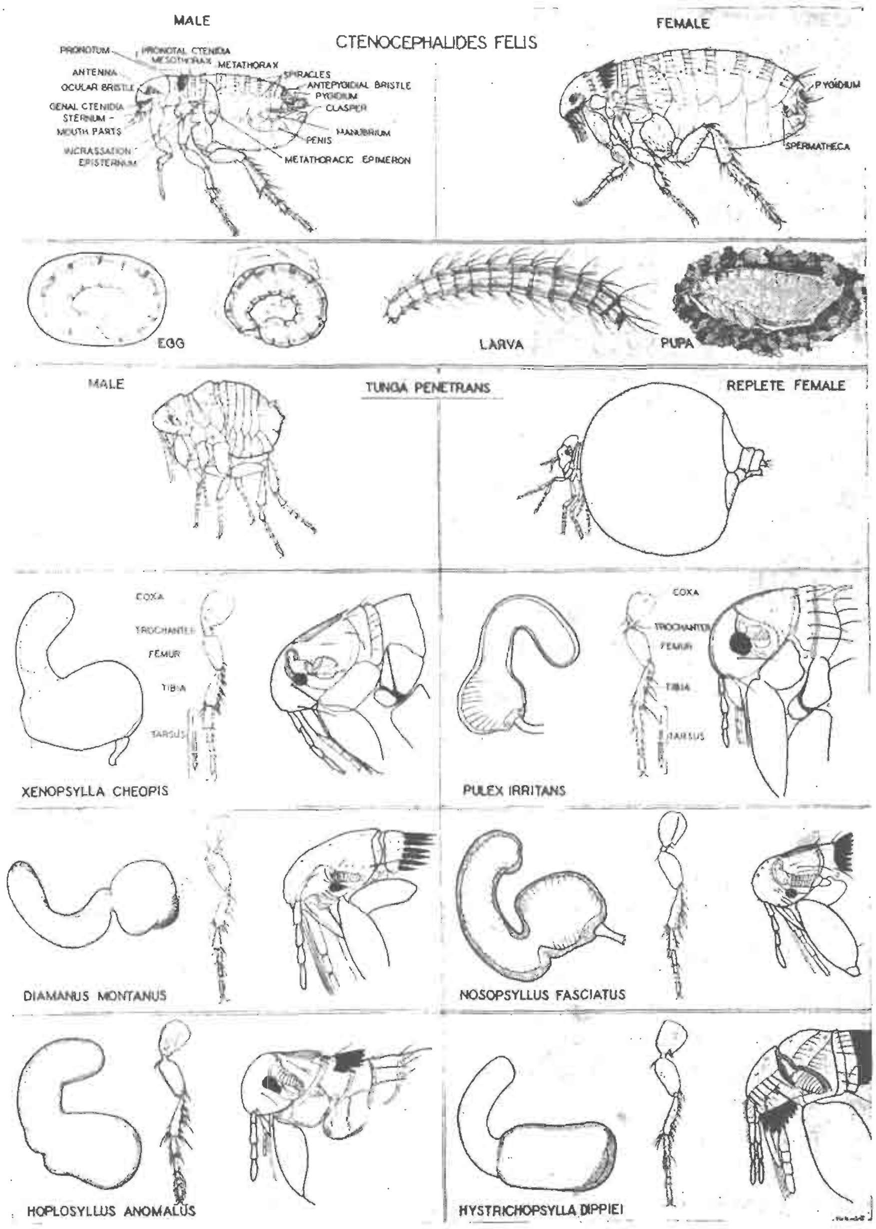


FIG. 138. Showing the structural details used in the classification of Siphonaptera, and also the life history.

The thorax of the flea is compact, consisting of the pro-, meso-, and metathorax. The *pronotum* lies immediately behind the head, and at its posterior margin in many species there is a ctenidium of spine-like processes known as the *pronotal ctenidium,* which is useful in rough classification. The *mesothorax* is the middle segment of the thorax; the *mesonotum,* the middle segment, is a simple arched plate. The *metathorax* is highly developed and is specially fitted to sustain the jumping mechanism. The several segments of the thorax and abdomen as well are made up of plates (sclerites); the dorsal plates are known as *tergites,* the ventral plates are the *sternites,* and the lateral plates are *pleurites.*

The abdomen consists of ten (actually eleven according to Kessel[2]) segments, which like the thoracic segments are made up of plates (scle-

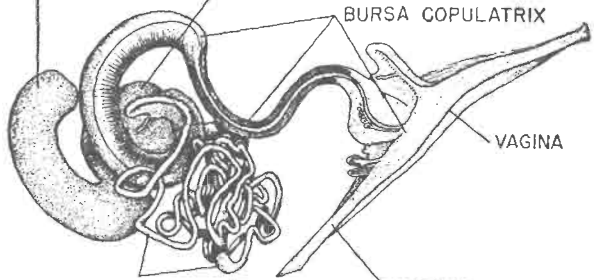


Fig. 139. Copulatory organs of the female *Nosopsyllus fasciatus.* (After Fox.)

rites), except that the pleurites are concealed. There are numerous backward-pointing spines. On the apical edge of the seventh tergite are the *antepygidial bristles;* the ninth tergite consists of a peculiar pincushion-like structure known as the *pygidium,* probably a sensory organ.

The male terminalia are particularly important in classification. Among the parts to be observed are the *claspers,* movable and nonmovable portions, and the *manubrium* (Fig. 138). In cleared specimens the spring-like *penis* may be seen lying in the region of the fifth and sixth segments which in copulation projects out from between the upper and lower claspers. The females possess a sacculated spermatheca (Fig. 139), situated in the region of the eighth or ninth segment and easily visible in cleared specimens. Some species have two spermathecae. This organ is characteristic for many species and is, therefore, an important taxonomic character (Fig. 138).

The legs consist of five joints: the *coxa,* the joint nearest the body; the *trochanter,* a very small segment; the *femur;* the *tibia* (strongly spined); and the five-jointed *tarsus* terminating in a pair of *ungues* or claws which may be considered as a sixth segment.

Digestive tract. As soon as blood begins to flow from the wound made by the protraction and retraction of the maxillary laciniae, it is drawn up into the *pharynx* by the action of both the cibarial and the pharyngeal pumps. By means of powerful muscles the blood is aspirated from the wound and on relaxation it is carried to the long narrow *esophagus* which begins in the region of the brain and passes through the

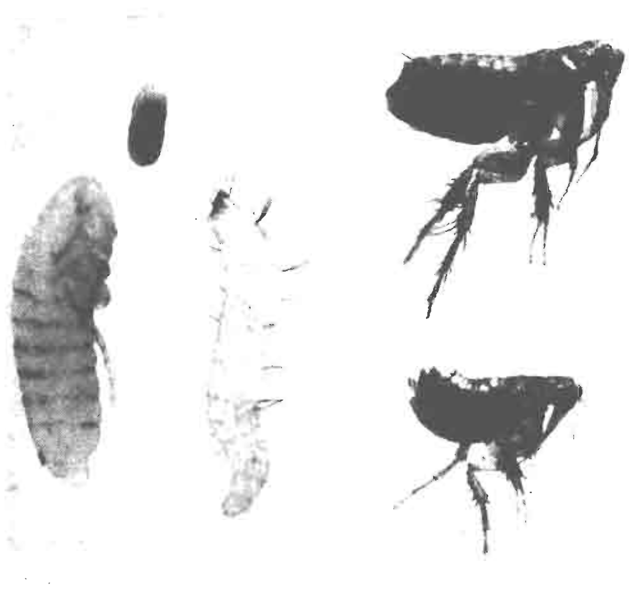


FIG. 140. Life cycle of a flea: (*upper left*) egg, (*center*) larva, (*lower left*) pupa, (*upper right*) female, (*lower right*) male.

circumesophageal ring. The esophagus opens into the stomach through the bulbous *proventriculus*, which is provided internally with radially arranged (seven rows) hair-like chitin-covered processes (Fig. 141) which, when the encircling bands of muscles contract, cause them to meet and form a valve, thus preventing regurgitation from the stomach. The *stomach* is a capacious distensible organ nearly as long as the abdomen, emptying into the short *intestine* which in turn empties into the wide *rectum* with its six *rectal glands*. Where the stomach joins the intestine, four filamentous *Malpighian tubules* arise.

Life history. The eggs of a flea (Fig. 140) are comparatively large (5 mm long), glistening white, and rounded at both ends. Relatively few, from 3 to 18, are deposited at one laying; however, during the entire lifetime of a female the number may be quite considerable. Bacot (1914)[3] records a total of 448 eggs over a period of 196 days deposited by a single female flea, *Pulex irritans* Linn. Most species deposit dry eggs which do not become attached to the hairs of the host even though oviposition takes place on the host. Fleas seldom oviposit among the hairs of the host, preferring the nests of the hosts where flea excrement occurs. Captured fleas will readily oviposit in glass vials or other receptacles in which they are trapped. If deposited on a dog or cat the eggs fall off readily when the animal stretches and shakes itself; thus myriads of eggs may be found on the sleeping-mat of a flea-infested animal. Temperatures of 65° to 80° F when combined with a fairly high humidity, 70 per cent and over, appear to favor egg laying. The incubation period varies from 2 to 12 days.

High mean temperature from 35° C to 37° C inhibits development, which may account for the fact that the eggs do not hatch well on the host. At a temperature of from 17° C to 23° C, Mayne (Mitzmain[4]) found that the egg stage lasted from seven to nine days; at from 11° C to 15° it lasted about 14 days. Others have found that this stage may be completed in from two to four days in *Ctenocephalides felis* (Bouché). The best growth and survival of this species was obtained at 65 to 90 per cent relative humidity (Bruce[5]).

The embyro is provided with a sharp spine (egg burster or "can opener") on the head by means of which the eggshell is cut into shreds by a tumbling motion of its inhabitant, which is thus liberated. (Kessel, *loc. cit.*) The larvae (Figs. 138 and 140) are very active, slender, 13-segmented, yellowish-white maggots, with segmentally arranged bristles. The mouth parts are of the biting type, and the newly hatched larvae of some species, e.g., *Nosopsyllus fasciatus* (Bosc), may subsist wholly on the feces of the adult fleas. Very little food seems to be necessary for their development, though excrementous matter, e.g., feces from rabbits, rats, squirrels, and other rodents, and also dry blood may be used as food. Excessive moisture is certainly detrimental to the life of the larvae, although a high percentage of moisture in the air is needed. The larvae are frequently found in houses in the crevices of the floor under the carpet or matting, and also in stables, coops, kennels, nests of rodents, pig pens, etc. When conditions are favorable, the time required for the larval period may be but 9 to 15 days; if they are unfavorable, it may extend over 200 days. At the end of the active feeding period when full growth has been achieved, the larva enters a quiescent stage, spins a cocoon, and pupates. The cocoon is whitish in appearance and so loosely spun that one may see the pupa within it.

The pupal period (Figs. 138 and 140) is influenced by temperature and varies greatly, from as short a time as seven days to nearly a year. The life cycle (egg to adult) accordingly may vary from as short a time as 18 days to many months. Under laboratory conditions at a temperature of 24° C Kerr[6] (1946) reports the life cycle of *Ctenocephalides felis* (Bouché) to be 20 to 24 days (larval stage 11 to 12 days). His cultures were from fleas fed on a cat.

Mayne (Mitzmain, 1910[7]) observed one individual of the squirrel flea, *Diamanus montanus* (Baker) (*Ceratophyllus acutus* Baker), from the moment the egg was laid to the emergence of the adult flea, securing the following data: incubation period of the egg, 8 days; first instar larva, 6 days; second instar larva, 10 days; third instar larva, 12 days; cocoon (pupal stage), 31 days; total, 67 days.

Longevity of fleas. Bacot (*loc. cit.*) states that with nearly saturated air at 45° to 50° F fleas can live for many days unfed. He reports that *Pulex irritans* Linn. survived for 125 days, *Nosopsyllus fasciatus* (Bosc) for 95 days, *Xenopsylla cheopis* (Roth.) for 38 days, *Ctenocephalides canis* (Curt.) for 58 days, and *Ceratophyllus gallinae* Schrank for 127 days. If fed on their natural host, *P. irritans* Linn. may live upward of 513 days, *N. fasciatus* (Bosc) for 106 days, and *X. cheopis* (Roth.), fed on man, 100 days. *Ct. canis* (Curt.) and *C. gallinae* Schrank have lived for periods of 234 and 345 days respectively when fed on man. Thus Bacot indicates that the maximum possible length of life of the various species mentioned is 966 days for *Pulex irritans* Linn., 738 days for *Ctenocephalides canis* (Curt.), 680 days for *Nosopsyllus fasciatus* (Bosc), 481 days for *Ceratophyllus gallinae* Schrank, and 376 days for *Xenopsylla cheopis* (Roth.). In a moist medium such as sprouting wheat grains and sawdust Mayne (Mitzmain, 1910, *loc. cit.*) has kept squirrel fleas alive for 38 days in one case and 65 days in another, the former a male, and the latter a female. Male rat fleas fed on human blood alone averaged 2½ days (maximum 17) of life, and the females 34⅘ days (maximum 160).

Hosts and occurrence of species. As will be seen later in this chapter, the rodent fleas are important from the public health standpoint, and ready transfer of fleas from host to host of different species adds much to the danger of disease transmission.

While it is apparent that ordinarily a certain species of flea predominates on a given species of host, e.g., *Ctenocephalides canis* (Curt.) on the dog, and particularly the cat, *Nosopsyllus fasciatus* (Bosc) on the rat in Europe and the United States, *Xenopsylla cheopis* (Roth.) on the rat in Asia, *Ctenopsyllus segnis* (Schön.) on the mouse, and *Pulex irritans* Linn. on the human, etc., host specificity in fleas is not strongly marked in many species.

In an unpublished report to the writer on the species of fleas found on rats in San Francisco, Rucker states that a great preponderance of the rat fleas recovered in San Francisco were *Nosopsyllus fasciatus* (Bosc) as based on 10,972 specimens as follows:

	PER CENT
Nosopsyllus fasciatus (Bosc)	68.07
Xenopsylla cheopis (Roth.)	21.36
Pulex irritans Linn.	5.57
Ctenopsyllus segnis (Schön.)	4.48
Ctenocephalides canis (Curt.)	0.52

The following tables (Tables V to XI) adapted after McCoy[8] throw much light on the interchange of hosts and the predominance of species:

TABLE V

FROM BROWN RATS [*Rattus r. norvegicus* (ERXLEBEN)]

No. of rats combed	*N. fasciatus*		*X. cheopis*		*P. irritans*		*C. segnis*		*Ct. canis*	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
606	570	1,252	790	1,146	225	425	44	137	13	15

TABLE VI

FROM BLACK RATS [*Rattus rattus rattus* (LINN.)]

No. of rats combed	*N. fasciatus*		*X. cheopis*		*P. irritans*		*C. segnis*		*Ct. canis*	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
11	7	32	6	5	0	0	4	17	0	2

TABLE VII

FROM MICE (*Mus musculus* LINN.)

From an unknown number of *Mus musculus*

	N. fasciatus		*X. cheopis*		*P. irritans*		*C. segnis*		*Ct. canis*	
	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
	1	5	2	0	0	0	3	10	0	0

TABLE VIII

FROM CALIFORNIA GROUND SQUIRRELS [*Citellus beecheyi beecheyi* (RICHARDSON)]

No. of squirrels combed	*Diamanus montanus*		*Hoplopsyllus anomalus*	
	Male	Female	Male	Female
132	2,065	2,306	86	140

TABLE IX

FROM THE DOG (*Canis familiaris* LINN.)

No. combed	*Ct. canis*		*P. irritans*		*Ct. felis*		*D. montanus*	
	Male	Female	Male	Female	Male	Female	Male	Female
4	10	44	8	17	0	1	1	0

TABLE X

FROM THE CAT (*Felis domestica* LINN.)

No. combed	*Ctenocephalides felis*	
	Male	Female
2	5	15

TABLE XI

FROM MAN (*Homo sapiens* LINN.)

No. of individuals	*P. irritans*		*Ct. felis*		*Ct. canis*		*D. montanus*	
	Male	Female	Male	Female	Male	Female	Male	Female
29	117	220	1	0	1	0	1	2

Plague. The first recorded pandemic of plague according to Wu *et al.*[9] was that of Justinian in the sixth century, starting in Egypt in 542 A.D. and spreading to Constantinople. It lasted 50 to 60 years, and its victims are estimated at 100,000,000. The second plague pandemic, the "Black Death," took place in the middle of the fourteenth century in Europe and claimed 25,000,000 victims or about one-fourth of the population. In Great Britain from half to two-thirds of the people perished. The great plague epidemic of London, 1664–1666, is said to have killed 70,000 persons out of a total population of 450,000. Plague disappeared from England in about 1680, having been almost continously present for nearly 140 years, with five epidemics.

Gradually this infection receded from Europe and the Near East, and as Wu *et al.* (*loc. cit.*) points out "the existence of epidemic foci, comparable to stagnant pools left behind by the lowering tide, was recognized. . . we now know a whole series of endemic plague foci, usually with epizoötics among the wild rodents situated near or even contiguous with Central Asia . . . the whole of this vast territory with its hosts of wild rodents might be compared with a heap of embers where plague smoulders continuously and from which sparks of infection may dart out now and then in various directions." The present pandemic is believed to have originated in a wild hibernating rodent, the tarabagan (*Arctomys sibirica* Schreber) in the interior of China and began as an epidemic in Hongkong in 1894 and was transported along world trade routes to many parts of the globe. The rat, as transported in commerce, constitutes the chief means of spreading the diseases, the infection being carried from rat to rat by means of rat fleas. For this reason plague may appear in a city far removed from the original focus of infection.

The first recorded appearance of plague on the North American continent, according to Hampton[10] occurred in San Francisco, California, on March 6, 1900, when the body of a Chinese who had died of plague was discovered in the Chinese quarter. Rat infection was not demonstrated until 1902. Human cases of plague continued to appear in San Francisco, and 121 cases with 113 deaths were reported up to February, 1904, when the last case in the first outbreak was recorded. In May, 1907, plague was again discovered in San Francisco, and the last of this series with 159 cases and 77 deaths occurred in June, 1908.

The next outbreak of plague in the United States, according to Hampton, occurred in New Orleans in 1914 with 30 cases and 10 deaths reported from June 21 to September 8. The Los Angeles outbreak of pneumonic plague—November 1, 1924, to January 5, 1925—resulted in 41 cases with 34 deaths. The outbreaks traceable to plague-infected rats and their fleas were quickly stamped out. However, as Hampton points out, large areas in our Pacific coast states and adjacent territory harbor plague-infected wild rodents such as ground squirrels and their ectoparasites (fleas) presenting a problem fraught with potential danger.

The answer to the question, "Is the disease in man and rodents identical?" was not forthcoming until 1894 with the work of Yersin and Kitasato in Hongkong. The former found the organism in the corpses of dead rats and according to Wu *et al.* gave the first detailed and accurate description of *Pasteurella pestis,* Yersin[11] calling it Bacille de la peste. To Kitasato, Wu states, we owe the earliest account of the organism, as he found the plague bacilli in the "finger blood of a patient with axillary bubo."

The disease in man. Wu *et al.* (*loc. cit.*) give the period of incubation from 2 to 10 days; the onset usually occurs within a period of 3 days. Fox in *Insects and Disease of Man,*[12] page 294 (P. Blakiston's Son & Co., by permission), describes the disease as follows:

It develops suddenly with a rapid rise of temperature, reaching 103° or 104° F. in two or three days, after which it is more or less irregular. There is headache, the eyes are injected and the facies are characteristic of extreme illness. Prostration is profound and comes on early. Delirium also appears early. The characteristic lesion of the disease, the bubo, usually is sufficiently pronounced by the second day to be readily detected. The most common site for the bubo is the femoral or inguino-femoral region, then the axillary region, cervical, iliac and popliteal. Over the enlarged glands oedema appears and pressure elicits great tenderness. The individual lymph nodes cannot be palpated. This swelling forms the primary bubo. Secondary buboes may appear in other parts of the body. In these, the glands are not matted together as in the primary bubo. Four forms of skin eruption may be described—a petechial eruption, ecchymoses, a subcuticular mottling, and the so-called plague pustule . . . a bulbous-like formation containing thin, turbid material teeming with plague bacilli. It is believed to indicate the original point of inoculation, the flea bite. Extending from this to the nearest lymphatic glands faint red lines indicating lymphangitis may be observed. A secondary pneumonia due to the deposit of plague bacilli in the pulmonary tissues may occur. In about a week if the patient survives, the bubo breaks down leaving an ulcer which heals slowly.

Plague is an acute infectious disease caused by *Pasteurella* (= *Bacterium*) *pestis* (Lehman and Neumann), (Atlas u. Grund. d. Bakt., 1896, p. 194). It is essentially a disease of rodents, usually transmitted by rodent fleas, but it may under certain conditions cause serious epidemics among human beings. The term *bubonic plague* is applied when inflammation of lymph glands results from the infection and buboes are formed; these are the first foci and may remain so localized and cause little discomfort. The buboes vary from 2 cm to 10 cm in diameter and are usually located in the groin (femoral glands) and axilla (axillary glands). When invasion of the blood stream occurs, a secondary *pneumonic plague* results. This form of plague may be transmitted from man to man as droplet infection and is not dependent upon either rodents or fleas. Pneumonic plague is almost invariably fatal.

A third type is known as *septicemic plague,* a fulminating type, due to invasion of the blood stream, which runs a very rapid course; death results before local signs are evident. Wu *et al.* (*loc. cit.*) state that "this fulminating type of plague is met with in pneumonic as well as in bubonic epidemics. Fulminating instances in pneumonic outbreaks are often peculiar to the final stage and are presumably instrumental in bringing about a spontaneous decline of the epidemic as droplet infection is absent, the patients succumbing before cough develops."

Fleas as vectors. Ogata[13] in 1897 came to the conclusion on epidemiological grounds that fleas were the agents of transmission, pointing out that fleas leave the rat as it becomes cold after death and so may transmit the virus direct to man. He pointed out that the flea can ingest plague bacilli while feeding, having produced plague in mice by injecting an emulsion of crushed fleas taken from plague rats.

Simond[14] in 1898 was the first to succeed in transmitting plague from a sick rat to a healthy rat through the agency of infected fleas. Simond's work was discredited for several years, but was successfully repeated by Verjbitski[15] in 1903.

Liston[16] in 1904, working in Bombay, came to the following conclusions: (1) There was one flea infesting rats in India far more commonly than did any other, viz., *Xenopsylla cheopis* (Roth.); (2) that these fleas when feeding on a plague rat harbored the plague bacilli in their bodies and that these multiplied therein; (3) that where fatal plague occurred, many of these infected fleas were at large, and (4) that after a local epizoötic of rat plague, man was also found to harbor these rat fleas and might become infected as had the guinea pigs used in the experiment.

The following is a very brief summary of experiments conducted by the Indian Plague Commission before and after its organization in 1905.

In the first instance healthy rats were confined in close proximity to rats which, inoculated with plague, were beginning to succumb to that disease and were artificially infested with rat fleas [*X. cheopis* (Roth.)]. The separate confinement of the rats in each case was so arranged that both contact with and access to all excreta were excluded, although it was provided that the fleas could pass from the inoculated to the healthy rats; this transfer actually did take place, and in many cases these fleas contained virulent plague bacilli; and when healthy nonimmune rats were thus infected they died of plague to the extent of 79 per cent; this extent of infection fell to 38 per cent, when partly immune rats of local origin were employed.

That the plague had originated in the healthy rats through the intermediary of rat fleas was further demonstrated when these were actually transferred from artificially plague-infected to healthy English rats, and the disease developed in 61 per cent of the latter.

Further, on constructing a series of miniature houses so as to reproduce the conditions pertaining to ordinary domiciles, it was found that whenever these were so constructed as to admit rats to their roofs, but not to their interiors, guinea pigs confined therein became successively infested with rat fleas and infected by plague, but that in those houses to which rats could not gain access plague was originated in guinea pigs living therein, either by transferring rat fleas to them, derived from plague-infected guinea pigs, or by an accidental admission of rat fleas from other sources. Also, when so confined, guinea pigs had under these conditions died of plague; healthy flea-free guinea pigs, subsequently introduced, became infected, and the infection remained in the place in proportion as the test animals were accessible to, and were found to be infested with, fleas: in other words, that "if the fleas be present, the rate of progress is in direct proportion to the number of fleas present." Further, that when healthy guinea pigs were confined in one of the houses, to the interior of whose roof fleas could not gain access, they became flea-infested and infected when running on the ground, but to a less extent when the cage was placed two inches therefrom, and not at all when it was suspended two feet above it. The fact that infection took place where pigs were located two inches above the ground indicates that contact with infected soil is not necessary for plague to originate, and that "an epizoötic of plague might start without direct contact of healthy with infected animals."

To demonstrate that this communication of plague from guinea pig to guinea pig was through the intermediary of fleas, rat fleas were taken from a morbid guinea pig and allowed to feed through muslin on healthy animals. The results were positive.

The state of affairs that existed in actual domiciles in which plague occurred or had existed was next inquired into, advantage being taken of the fact that plague-susceptible guinea pigs would serve as hosts as well as for the collection of fleas.

Guinea pigs free from fleas were introduced into rooms in which persons had died of plague, or from which plague-infected rats had been taken. They were allowed to be at large in these rooms for periods of from 18 to 24 hours. These guinea pigs not only collected the fleas on their bodies, most of which were rat fleas, but 29 per cent of them contracted plague and died of plague within a few days after being restored to ordinary confinement. As before, many of the fleas which they yielded harbored plague bacilli in their stomachs and were capable of infecting additional animals.

Further, after first washing the floors and walls of the rooms with mercuric chloride and so adequately disinfecting them for plague, but not for fleas, and then introducing guinea pigs, these became plague-infected when rat fleas were present.

That the infection was actually due to fleas was also shown by the positive results when fleas collected from rats occurring in plague-infected houses were transferred to healthy rats or guinea pigs in the laboratory. These in due course became infected and died of plague.

Similarly fleas taken from the clean guinea pigs allowed to run in plague-infected houses, and transferred to fresh animals, communicated plague to them in 8 out of 40 tests.

In the next place plague-free white rats, guinea pigs, and monkeys were placed in enclosures, which precluded contact as well as soil infection in plague-infected rooms, pairs of one animal or another being used in each of the 42 experiments of this class conducted, one individual being confined to a flea-proof receptable and the other to an adjacent one accessible to these insects (one animal being thus a control). In the latter case plague resulted in four instances, or 10 per cent gave postive results.

As a variation of the same experiments the enclosures for individual animals, while protected from soil or contact infection, were surrounded as a screen to fleas by two and one-half inches of "tanglefoot" or were unprovided with this protection, the "tanglefoot" being replaced by sand. (Twenty-nine experiments were conducted.) In the latter case the animals became infested with fleas, one having as many as 20; seven became fatally infected with plague. In the former, individual fleas were found on only three of the rats and no animals became plague-infected.

Examining the fleas entrapped, 247 in number, it was found that 147 were human fleas, 84 were rat fleas, and 16 cat fleas. Moreover, a large proportion of each kind was examined. No plague bacilli were found in the cat fleas, one only in 85 of the human fleas was infected, and no less than 23 out of 77 of the rat fleas harbored plague organisms.

It was also shown that, when rats in the course of an epizoötic died of plague, the pathological features manifested in their bodies corresponded to those exhibited by artificially rat-flea-infested animals, and hence it was inferred that in nature and under experimental conditions the animals had alike succumbed to a single agency. This similarity especially related to the site in which buboes arose, as in both instances, where the place of inoculation could be observed, it was the same.

Further observations. Blue[17] reports a number of observations made in San Francisco during 1906: Two small boys found the body of a dead rat in an unused cellar; the rat was buried with unusual funeral honors and in forty-eight hours both were taken ill with bubonic plague. Again, a laborer picked up a dead rat with the naked hand and threw it into the bay. He was taken ill with plague three days later. The case of a physician's family is also cited in which foul odors pervaded their second story apartment over a grocery store. On removing the wainscoting around the plumbing to ascertain the cause of the odor, two rat cadavers were found in the hollow wall. In two or three days thereafter the two members of the family who used the room sickened, one dying on the fifth day of cervical bubonic plague. Blue believes that the removal of the wainscoting set free infected rat fleas.

The following instance is reported in the United States Public Health Reports (November 7, 1913, page 2356): A fatal case of plague occurred in Manila (P. I.) in the person of an American, editor of the Manila Daily Bulletin. A plague rat had been found on September 6 in the block adjacent to the one in which the newspaper offices were located. The editor was admitted to the hospital September 19 and died at the Plague Hospital three days later. A mummified rat was found in the desk of the late editor, together with live fleas, *Xenopsylla cheopis* (Roth.). Both the fleas and rat revealed bipolar staining organisms, and inoculations into healthy laboratory rats produced typical cases of plague terminating fatally.*

That the mummified rat must have been dead at least two weeks and that the live fleas contained plague bacilli suggests "strong proof that plague might be introduced into a country without either the importation of human or rat cases of plague and that fleas might be alone concerned."

Role of the flea in plague transmission. The Indian Plague Commission showed that the average capacity of a flea's stomach [*Xenopsylla cheopis* (Roth)] was .5 cubic millimeter, and that it might receive as many as 5,000 *Pasteurella pestis* while imbibing blood from a plague rat. The Commission found that the bacillus multiplies in the stomach of the flea and that the percentage of fleas with bacilli in the stomach varied with the season of the year. In the epidemic season the percentage was greatest for the first four days, and on one occasion the stomach was found filled with the organisms on the twentieth day. In the nonepidemic season no plague bacilli were found in the stomach after the seventh day. They also found that in the epidemic season fleas might remain infective up to 15 days, while in the nonepidemic season but seven days, and in the latter case the percentage of infection in animals was much less than in the epidemic season. They showed that while one flea was occasionally able to carry the infection, this was not usual. It was found that both males and females were capable of transmitting the disease. As to the manner of dissemination the Commission found bacilli in the stomach and rectum only, never in the salivary glands or body cavity, and but rarely in the esophagus, and then only when the flea was killed immediately after feeding. After digestion, the blood in the stomach passes into the rectum and is ejected as a dark red or tarry droplet, containing virulent plague bacilli, which if rubbed into recent flea-bites resulted in infection of the animal. The actual inoculation therefore, it was believed, was accomplished indirectly by the flea-bitten person's scratching or rubbing the site of the bite after the infected flea had discharged fecal material upon the skin. That there is a great deal of difference in the consistency and other characteristics of the fecal deposits of the various species of fleas is easily observable, e.g., the human flea, *Pulex irritans* Linn., defecates freely while feeding but is not a ready vector of bubonic plague, while the rat flea, *Xenopsylla cheopis* (Roth.) seldom defecates while feeding and is a potent vector. Bacot and Martin[18] (1914) demonstrated a mechanism of infection which is based on observations made by Swellengrebel[19] (1913) in Java that *Xenopsylla cheopis* (Roth.) seldom defecates when feeding and showed that infection resulted when the flea's only contact with the experimental animal was by means of the proboscis, i.e., the infection is introduced with the bite directly. That this mode of infection was in the manner of regurgitation due to a temporary obstruction at the entrance to the stomach was discovered by Bacot and Martin (*loc. cit.*), who state that on

examining the contents of the stomach of a flea a day or two after it has fed upon infected blood, clusters of minute brown specks darker in colour and firmer in consistency than the rest of the contents are visible. . . . Later the stomach and proventriculus show jelly-like masses of a brown colour. These masses are possessed of considerable cohesion and are with difficulty teased out so as to make a film suitable for microscopical examination. The plague-culture grows in the proventriculus as well as in the stomach. Owing to its gelatinous consistency, it not infrequently leads to incompetence and even complete blocking of the proventricular valve. . . . Although with the proventriculus obstructed in this manner fresh blood cannot find its way into the stomach, this does not prevent the insect sucking, as the pump which aspirates blood up the sucking tube and propels it into the stomach is situated in the pharynx. On the contrary the flea suffers from thirst and is persistent in its efforts to satisfy this appetite, but only succeeds in distending the oesophagus. The blood in the distended oesophagus may flow out again on cessation of the sucking act, and we have seen drops of blood escape from the mouth parts of "blocked" fleas when the insect withdrew its proboscis. . . . Given the opportunity, the insects suck blood again and again and if the pharyngeal pump ceases for a moment, some of the blood will by the elastic recoil of the oesophageal wall be driven back into the wound and carry with it plague bacilli.

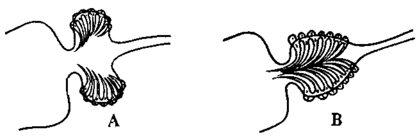
Bacot and Martin found that infected fleas lived as long as 50 days at from 10° C to 15° C and 23 days at 27° C and died infected. Working with two species of rat fleas, *Xenopsylla cheopis* (Roth.) and *Nosopsyllus* (= *Ceratophyllus*) *fasciatus* (Bosc), fed on septicemic blood, Bacot and Martin conclude that they "can transmit plague during the act of sucking and that certain individuals suffering from a temporary obstruction at the entrance to the stomach were responsible for most of the infections obtained, and probably for all." In the course of time the plague culture forming the proventricular plug undergoes autolysis and the normal passage of blood is re-established.

Figure 141A shows the position of the spine-like epithelial cells in the proventriculus when at rest, the opening into the stomach being free for the passage of blood. Figure 141B shows the opening into the stomach closed against the outward passage of blood on contraction of the muscular bands. Bacot[20] points out that it is the lodgment and growth of the bacilli among the spines that is the initial stage of the blockage.

In describing the mechanism of plague transmission by fleas, Eskey and Haas[21] (1940) show numerous microphotographs of blood-distended stomachs of fleas after feeding. The elapsed interval between an infective blood meal and an infective bite (transmission) for *Xenopsylla cheopis* (rat flea) averaged about 21 days (shortest interval 5 days, longest 31 days); for *Diamanus montanus* (ground-squirrel flea) the average was 53 days; the longest interval in the series was 130 days. The average length of life of fleas after being plague infected was 17 days (maximum 44 days) for *X. cheopis*, and 47 days (maximum of 85 days) for *D. montanus*.

Eskey[22] has shown that virulent plague organisms are more constantly present in the feces of some species of fleas than in others. He

FIG. 141. Spine-like epithelial cells in the proventriculus of *Xenopsylla cheopis:* (*A*) at rest, (*B*) showing opening into stomach closed.



reports that plague followed every inoculation of feces deposited by infected *Diamanus montanus* (Baker), while less than one-third of the fecal inoculations of *Nosopsyllus fasciatus* (Bosc) gave positive reactions. He also reports that the feces of *Xenopsylla cheopis* (Roth.) gave positive reactions, but these fleas did not survive long enough to determine whether or not the results would be constant for any length of time. Eskey points out that there seems to be danger of infection from virulent plague organisms present in the feces of all plague-infected fleas.

Still another possible mode of transmission, which applies, however, only to transfer from rodent to rodent, has been suggested by various workers, namely, that of crushing infected fleas with the teeth, with infection through the mucosa of the buccal cavity resulting in lymph-node involvement in the region of the neck.

Plague in field rodents. In California plague was demonstrated in ground squirrels, *Citellus beecheyi beecheyi* (Richardson), under natural conditions in 1908 by McCoy. According to that author at the time of his writing a report on this finding (1910),[23] about a dozen persons had contracted the disease under circumstances that pointed conclusively to squirrels as the cause. The two species of fleas commonly infesting the ground squirrel in California are *Diamanus montanus* (Baker), (= *Ceratophyllus acutus* Baker) and *Hoplopsyllus anomalus* Baker, of which the former is far more numerous. McCoy proved the first-named species a carrier as follows: He inoculated a ground squirrel subcutaneously with a broth culture of *P. pestis* derived from a human case of plague. This squirrel died on the fifth day, but three days before its death, 100 fleas, *D. montanus* (Baker), were put in the cage with it. The dead animal was removed from the cage while warm, and 27 live fleas taken from its body. Smears made of the crushed bodies of two of these fleas showed an abundance of pest-like bacilli in each. The remaining 25 fleas were put into a clean cage with a healthy squirrel. This animal died of subacute plague 10 days later, the buboes being in the region of the median, posterior inguinal, and pelvic glands. A pure culture of *P. pestis* was obtained from the liver. McCoy states that the experiment is conclusive in showing that *D. montanus* (Baker) may convey plague from a sick to a healthy squirrel. The squirrels used in the experiment were kept in quarantine for at least a month prior to their being used, which was necessary to exclude any naturally infected ones. McCoy found the bacilli in squirrel-flea feces four days after removal of the fleas from the host.

Sylvatic (selvatic) plague. The designation selvatic plague was proposed by Ricardo Jorge (1928) (see p. 4) for the plague of field rodents. Fleas play an important role in transmission from rodent to rodent and consequently in the endemicity of the disease. It is now known that under certain ecological conditions in vacated squirrel burrows fleas may continue to harbor virulent *P. pestis* for many months thus providing a virtual insectan reservoir for the infection under sylvatic conditions. Fleas have been known to survive though starved for more than six months (196 days).

Aside from the matter of flea transmission it is important to bear in mind that the great epidemic of plague in Manchuria resulting in 60,000 deaths in 1910–1911 was of the pneumonic type and sprang from the wild tarabagan, *Arctomys sibirica* Schreber (Siberian marmot), which was hunted for its valuable reddish brown fur by numerous Chinese hunters unfamiliar with its dangers. The mountainous portions of Central Asia, i.e., portions of Siberia, Mongolia, Tibet, and Manchuria, are regarded as the original home of plague, and the tarabagan as well as its flea parasites play an important role as reservoirs of the infection in this area. These large rodents are about half a meter in length with a bushy tail about 15 cm long. It is pointed out that the low body temperature of the tarabagan during hibernation enables the animal to survive and thus to carry over the infection from one season to the next, and the flea, *Oropsylla silantiewi* (Wagner), as well as perhaps other bloodsucking ectoparasites, transmit the infection from animal to animal.

Comparable endemic foci of sylvatic plague occur in South Africa, where the gerbilles (Muridae, Berbillinae) belong to three genera, particularly *Tatera*, e.g., *Tatera lobengulae* De Wint.; the multimammate mouse, *Mastomys coucha* (A. Smith) (Muridae, Murinae), and their flea parasites play the leading role.[24] In the Russian steppes the susliks, *Spermophilus refuscens* and other species (Sciuridae), and their flea parasites, *Citellophilus tesquorum* (Wagner) and *Neopsylla setosa* Wagner, play a similar role. In North America, as already explained, ground squirrels (*Citellus* spp.) (Sciuridae) and their fleas, e.g., *Diamanus montanus* (Baker) (= *Ceratophyllus acutus* Baker), may be important reservoirs of sylvatic plague. In South America, the cavy, *Cavia asperea* Pallas, and its fleas, e.g., *Rhopalopsyllus cavicola* (Wey-

FIG. 142. A California ground squirrel, *Citellus beechyi*. (Photograph by R. Maynard, California State Department of Public Health.)

enb.), play a similar role. Burroughs[25] gives further consideration to the matter of animals (other than rats) naturally infected with plague.

Sylvatic plague remains localized or at best spreads slowly, and in each endemic region a particular native animal or group of animals (rodents) maintains the infection, and when other small house-invading rodents such as mice and rats come in contact with such a focus, the infection may be carried to human habitations, and human cases may result; or likewise if humans invade the territory of sylvatic plague, infection may occur. Under such circumstances man may contract the infection by direct traumatic contact with infected rodents, or possibly by flea bites, which is less likely. Meyer[26] points out that by 1915 at least 24 cases with 15 deaths were directly attributed to exposure to ground squirrels in six California counties. Meyer[27] (1947) lists 38 species of wild rodents and rabbits which have been found plague-infected in 14 states of the Western United States. Three groups of rodents constitute the primary natural reservoirs of plague in that part of the country: (1) ground squirrels (*Citellus*), Pacific coast and northern intermountain area (Fig. 142); (2) wood rats (*Neotoma*), southwest desert areas; (3) prairie dogs (*Cynomys*), plateau regions of Arizona and New Mexico.

Although there is some evidence to support the hypothesis setting forth the probable presence of plague among wild rodents in North America, particularly ground squirrels, *Citellus*, long before the demonstration of plague in ground squirrels in California by McCoy (*loc. cit.*) in 1908, and therefore long before the discovery of plague in rats in San Francisco in 1902; nevertheless Meyer states that the available evidence is not adequate to warrant a decision. There appears to be some paleontological evidence as well connecting the genus *Citellus* of North America with ancestors on the Asiatic high plateau which is also the home of plague.

Meyer in "The Ecology of Plague" (1942, *loc. cit.*) points out that many of the unknown factors in sylvatic plague are intimately related to the influence of the climatic conditions on the life and longevity of the insects involved (fleas and lice). Too little attention has been given to the host-parasite relationships of this as well as other diseases involving arthropod vectors. The factors influencing the low transmissibility of sylvatic plague are as yet little understood. In the meantime Meyer (1947, *loc. cit.*) urges the maintenance of so-called "rodent free" belts around towns and "research and more research into the ecology of sylvatic plague."

Wild rodent fleas. Dunn and Parker[28] investigated the flea population of a variety of species of wild animals in the Bitter Root Valley of Montana. *Oropsylla* (= *Ceratophyllus*) *idahoensis* (Baker) was found infesting a large percentage of the 94 ground squirrels [*Citellus columbianus* (Ord)] examined, the average per animal being 3.86. While *O. idahoensis* (Baker) was by far the most common species of flea on this species of ground squirrel, six other species were taken in order of abundance, viz.: *Opisocrostis tuberculatus* (Baker), *Neopsylla inopina* Roth., *Monopsyllus eumolpi* (Roth.), *Cediopsylla inequalis* (Baker), and *Monopsyllus vison* (Baker) (one specimen). It is of interest to know that these authors took *Oropsylla idahoensis* (Baker) from the following species of wild animals: cottontail rabbits, *Sylvilagus n. nuttallii* (Bachman); snowshoe rabbits, *Lepus bairdi* Hayden; pine squirrels, *Sciurus hudsonicus richardsoni* Bachman; woodchucks, *Marmoto f. flaviventer* (Aud. and Bach.); and the bushy-tailed woodrat, *Neotoma c. cinerea* (Ord). The woodchuck showed a heavy infestation (average

15.47 per animal) of *Thrassis acamantis* (Roth.). Observations made on the marmot in California also show a heavy flea infestation, averaging 26.57 fleas per animal according to unpublished data by Stewart.

Pearse[29] in his study of fleas on rodent hosts in Nigeria concludes that "the ecological factors which are associated with a high degree of infestation are dry climate or habitat, the occupation of a more or less permanent home by the host, and large size of the host. Factors associated with low degree of infestation are wet climate or habitat, lack of permanent abode of host, small size of host, and wandering or arboreal habits of host."

While California ground squirrels, e.g., *Citellus beecheyi beecheyi* (Richardson), have several species of fleas infesting them, among these *Diamanus montanus* (Baker), *Hoplopsyllus anomalus* Baker, and *Malaraeus telchinum* (Roth.), there is usually a preponderance of the first-named species. Wood rats (*Neotoma*) commonly have several species, among them *Ctenopsyllus segnis* (Schön.), *Orchopeas sexdentatus sexdentatus* (Baker), *Anomiopsyllus nudatus* Baker, and *Malaraeus telchinum* (Roth.).

Infected and infective fleas. In relation to the spread of sylvatic plague Meyer[30] calls attention to certain paradoxical observations: (1) that despite active reservoirs with hundreds of infected rodents very few cases of human plague were diagnosed on the North American continent; (2) that plague-infected fleas are taken from animals which had anatomically been declared noninfected; (3) squirrel hunters and plague-survey crews are commonly covered by fleas and are bitten by squirrel fleas yet are not infected.

Meyer points out that in sylvatic plague man becomes infected with bubonic plague by immediate contact with the sick or dead rodents, flea transmission being infrequent. The danger represented by individual fleas appears therefore more limited than was originally believed. Wild rodent fleas serve as "preservers" of plague infections in suitable rodent burrows for many months and under such natural conditions, while infected, harbor bacilli which are of low virulence or avirulent. These "preserver" fleas are believed to be "nonblocked."

Meyer continues,

> Rodents with latent infections will hibernate only to develop acute plague early in spring (March and April). Since the flea population is as a rule simultaneously very high, a great reservoir of infected vectors is thus created. The cadavers of the dead rodents are rapidly and effectively removed by the larvae of the *Lucilia* flies, while the fleas persist in the nests. With the migration of the young squirrels and chipmunks into the empty abandoned burrows and nests, highly susceptible hosts are thus brought in contact with infected and infective fleas. They may bring the vectors to the surface and some may thus

contribute to the intensity and the expansion of the virus. These events are probably accompanied by a variable degree of subclinical immunization favored by factors of age and reduced metabolism due to the approaching hibernation. Again latent infections and infected fleas in the burrows furnish the chain which connects the epidemic of one year with that of the next. Thus sylvatic plague smoulders for years and is everlasting. Suppressive measures against sylvatic plague in order to be effective must by necessity be directed against the hosts, the rodents, and the vectors, the various species of fleas. In selecting the procedures to reduce the rodent populations chemicals, preferably gases which are also insecticidal, must be chosen. [See methyl bromide fumigation, p. 458.]

Laboratory tests indicate that many species of Siphonaptera (more than 30) can be infected experimentally, but relatively little has been done on *vector efficiency* except for the very significant work of Wheeler and Douglas[31] (1941). These investigators point out that the vector efficiency of a species of arthropod incriminated in the transmission of an infectious agent must necessarily take into consideration the following factors: (1) the *infection potential,* (2) the *vector potential,* and (3) the *transmission potential.* The first is based upon the percentage of a given species in which the infection becomes established; the second, upon the percentage of infected individuals which become infective, and the third, is the mean number of transmissions effected by a group of infective individuals. Thus these workers demonstrated that *Diamanus montanus* (Baker) has an infection potential of .85, a vector potential of .52, and a transmission potential of 2.58 with a *vector efficiency of 1.14,* compared with an infection potential of .98, a vector potential of .29, a transmission potential of 1.44, and a *vector efficiency of* .39 for *Xenopsylla cheopis* (Roth.).

Reporting on the results of his vector efficiency studies in relation to sylvatic plague, Burroughs (*loc. cit.,* 1947) states that experimental evidence was obtained that different strains of a species of flea taken in different geographical areas may differ markedly in their biological vector capacity. This observation was based primarily on experiments with *Diamanus montanus,* which proved much less efficient than *Xenopsylla cheopis* in striking contrast to the results obtained by Wheeler and Douglas (*loc. cit.*), whose *D. montanus* came from a widely separated area.

Eskey and Haas (*loc. cit.,* 1940) conducted transmission experiments with *individual* fleas collected from wild rodents in areas known to be sylvatic plague foci. Many of the infective fleas (proved on guinea pigs) transmitted plague to more than one experimental animal (guinea pigs). Both male and female fleas transmitted the infection, although males proved to be much less efficient on the whole; however, one *Xenopsylla* *cheopis* female infected 10 guinea pigs, and one male ground-squirrel flea, *Opisocrostis labis* (Jordan and Rothschild) infected 11.

Murine (endemic) typhus fever. For many years a mild form of typhus has existed in seaport communities in warmer climates in many parts of the world. This infection was long believed to be of murine origin. Mild typhus was first reported in the United States (New York) in 1898 and was referred to as Brill's disease by some authors and by others as classical typhus (see Zinsser). It was later reported in Atlanta, Georgia; Charlotte, North Carolina; and Galveston, Texas. In 1931 Mooser, Castañeda, and Zinsser[32] reported rats as the carriers of typhus fever in Mexico. The causal organism received the name *Rickettsia mooseri* Monteiro in 1931. In 1932 Dyer *et al.*[33] reported transmission of endemic typhus by rubbing crushed infected fleas or infected flea feces into wounds. The mechanism of transmission, including infection by means of dry flea feces, is essentially the same as in the classical louse-borne typhus previously described. Several species of rat fleas have been incriminated, including *Xenopsylla cheopis* and *Nosopsyllus fasciatus*, as well as (experimentally) the sticktight flea, *Echidnophaga gallinacea* (Westw.) (Alicata,[34] 1942). Dyer and associates report that the incubation period in one experimental animal, a guinea pig, after inoculation of a flea emulsion was 10 days; also the virus remained viable in the flea for at least 52 days, showing the importance of the flea as a reservoir under natural conditions.

Zinsser[35] (1937) held to the belief that both types of typhus may be either endemic or epidemic. He states: "Although the murine disease reaches man first from infected rats by flea vectors, this virus can also, like the European, pass from man to man by the louse . . . capable of epidemic dissemination of the murine as well as of the classical typhus. . . . Brill's disease is an imported classical typhus, endemically established in cities with large immigrant populations."

Recently (1948) Castañeda,[36] reporting on flea-borne and louse-borne typhus in Mexico, states that the geographic distribution of both types of typhus in Mexico follows the climate of each zone. Pure murine typhus is found in warm regions and is flea-borne. Classical typhus occurs in cold and mild zones, where louse-borne infection is frequent. In these zones *murine typhus* also occurs and *may become louse-borne* and cause important epidemics of high mortality. In Mexico City he reports that from 1927 to 1931 it was demonstrated that murine typhus was the prevalent type, while in 1938 most of the isolated strains from typhus patients were classic. He continues, " . . . during the last seven years murine strains have been found only on rare occasions; complement fixation tests have corroborated the prevalence of classic typhus in the cold and temperate zones where typhus is endemic." Castañeda states that rat campaigns are advisable in zones where there is danger of louse-borne murine typhus.

Beck and van Allen[37] (1947) state that two epidemiologic periods have occurred in California. The first period was probably of epidemic type and louse-borne, 1916 to 1917, consisting of an isolated epidemic of 32 cases and 4 deaths with source of infection in Mexico. The second period of the murine type and flea-borne was from 1919 to 1945 inclusive, representing 362 cases and 17 deaths. Cases during the latter part of the second period occurred during every month of the year with a slight increase during late summer and autumn. *Rattus norvegicus, R. rattus, R. alexandrinus,* and *Mus musculus* were found naturally infected. Ground squirrels, *Citellus beechyi,* were experimentally infected with a murine strain of typhus.

THE COMMONER SPECIES OF FLEAS

Systematic. There are said to be more than 900 described species of Siphonaptera; for North America and the West Indies, Ewing and Fox[38] (1943) list 209 species and 63 subspecies and varieties in 61 genera and 14 subgenera. The order is divided into six families as follows: (1) Hectopsyllidae, (2) Pulicidae, (3) Dolichopsyllidae, (4) Ischnopsyllidae, (5) Hystrichopsyllidae, (6) Macropsyllidae. An extensive index to the literature of Siphonaptera of North America has been published by Jellison and Good.[39*]

Key to the Families of Siphonaptera

(Modified by Stewart after Ewing)

1. The three thoracic tergites together longer than the first abdominal tergite 2
   The three thoracic tergites together shorter than the first abdominal tergite .... Hectopsyllidae
2. No vertical suture from dorsal margin of head to bases of antennae; frontal region almost evenly rounded along margin 3
   A vertical suture passing upward from the bases of the antennae to the dorsal margin of head; margin of frontal region usually most strongly curved at vertex 4
3. Abdominal tergites with but a single row of setae .... Pulicidae
   Abdominal tergites with at least two rows of setae .... Dolichopsyllidae
4. Head without a pair of dark anteroventral flaps on each side 5
   Head with a pair of dark anteroventral flaps on each side .... Ischnopsyllidae
5. Occipital region without dorsal incrassation; frontal region entire .... Hystrichopsyllidae
   Occipital region with dorsal incrassation; frontal region divided, the anterior part bearing a border of spines .... Macropsyllidae

* Also see Holland, G. P., 1949. *The Siphonaptera of Canada.* Ottawa: Dominion of Canada Dept. Agric. Pub. 817, Tech. Bull. 70. 44 maps + 42 plates.

Family Pulicidae. *Pulex irritans* Linn. (Fig. 143) is commonly known as the human flea. It is cosmopolitan in distribution and occurs on many domesticated animals, particularly swine. This species has neither oral nor pronotal ctenidia. The metacoxae have a row or patch of short spinelets on the inner side; the mesosternite has an internal rod-like incrassation extending dorsoanteriorly. The maxillary lacinae extend about halfway down on the fore coxae, which distinguishes this species from *Pulex dugesii* Baker (mandibles extending at least three-fourths the length of the fore coxae), also known as a human flea but restricted to Mexico and the border states of the United States. *Pulex irritans* Linn. transmits

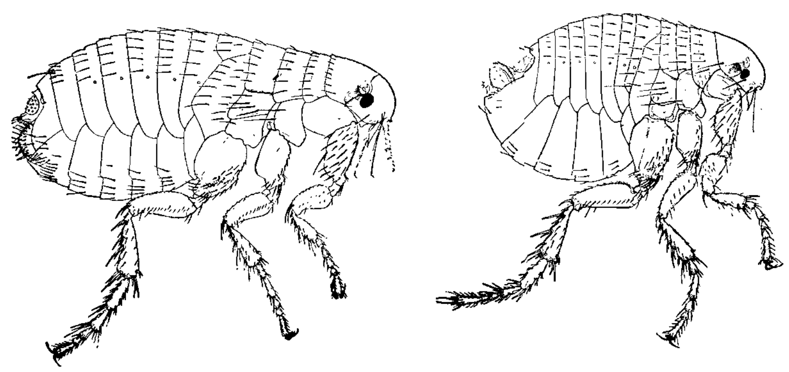


FIG. 143. *Pulex irritans,* the human flea; male (*right*), female (*left*). ×17.

plague under laboratory conditions and may be the chief vector of two unusual types of plague, e.g., *viruola pestosa* (a vesicular form) and *angina pestosa* (a tonsillar form) found in Ecuador.

Ctenocephalides canis (Curtis) and *Ctenocephalides felis* (Bouché) are the dog flea and cat flea respectively. Both species attack cats and dogs as well as man. Both have the oral ctenidia consisting of eight spines and the pronotal comb of 16 spines. They may be separated as follows:

In the female the head is fully twice as long as high (seen from side); two or three bristles on metathoracic episternum; bristles on metathoracic epimeron, first row, four to eight, second row, five to seven; seven to ten bristles on inner side of hind femur .... *felis*

In the female the head is less than twice as long as high (seen from the side); three or four bristles on metathoracic episternum; bristles on metathoracic epimeron, first row, seven to eleven, second row, seven to nine; ten to thirteen bristles on inner side of hind femur .... *canis*

Xenopsylla cheopis (Rothschild) (Fig. 144) is the Oriental or Asiatic rat flea. It habitually inhabits buildings and bites man freely. It resembles *Pulex irritans* Linn. in that both the oral and pronotal ctenidia are absent. The ocular bristle is in front of and just above the middle of the eye; there are two bristles on the gena; oral bristles placed low down just above the base of the maxillae; each abdominal tergite has but one row of bristles; the hind femur has a row of about eight bristles. The mandibles reach nearly to the end of the anterior coxae. Incrassation of mesosternite consists of one rod extending anterodorsally and one rod extending upward nearly perpendicularly (Fig. 138).

Mellanby[40] (1933) has performed experiments proving that *X. cheopis* (Roth.) can complete its life history between 18° C and 35° C in moist air. Between 18° C and 29° C air with a relative humidity of 40 per cent is unfavorable, while with 60 per cent pupation takes place successfully. Pupation at 18° C required eight days, at 22° C it required

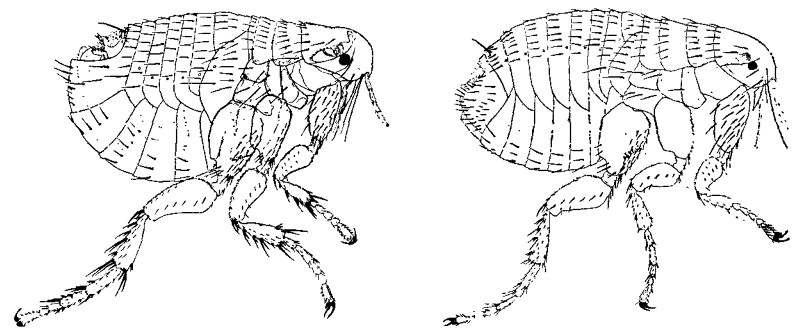


FIG. 144. *Xenopsylla cheopis,* the Oriental rat flea; male (*left*), female (*right*). ×17.

six days, and at 29° C to 35° C it required four days. The developmental zero for pupation is about 15° C.

Xenopsylla brasiliensis (Baker) is an African species, the predominant rat flea in Uganda, Kenya, and Nigeria. It has spread to South America and certain areas in India. It is regarded as a more important vector of plague than *X. cheopis* (Roth.) in Kenya and Uganda, since it is "the flea of the hut" while the latter infests rats of stone and brick buildings.

Xenopsylla astia Roth. has a restricted distribution

being found mostly along the low-lying coast of Ceylon, the east coast of India, and along the opposite coast of Bengal . . . while *X. astia* may be the responsible vector [of plague] in certain circumscribed and isolated outbreaks, the available evidence . . . points to its inferior position in the epidemiological picture. . . . Moreover *astia* outbreaks, if and when they do occur, are not known to carry over from one season to another.

(Wu, *et al., loc. cit.*)

Xenopsylla hawaiiensis Jordan is a common flea of the Hawaiian rat, *Rattus hawaiiensis*. According to Eskey reported by Jordan[41] this species of flea has a very peculiar distribution.

It has not been found in Honolulu or vicinity, while it is quite common on rats caught about nine miles away on the opposite side of the island. It is essentially a flea of field rats and rarely found on rats caught in buildings.

Family Dolichopsyllidae. *Nosopsyllus* (= *Ceratophyllus*) *fasciatus* (Bosc) is the European rat flea (Fig. 145). It is widespread over Europe and America, being less common in other parts of the world. It has been recorded on rats, house mice, pocket gophers, skunk, man, and many other host animals. It has but one ctenidium, the pronotal, which has

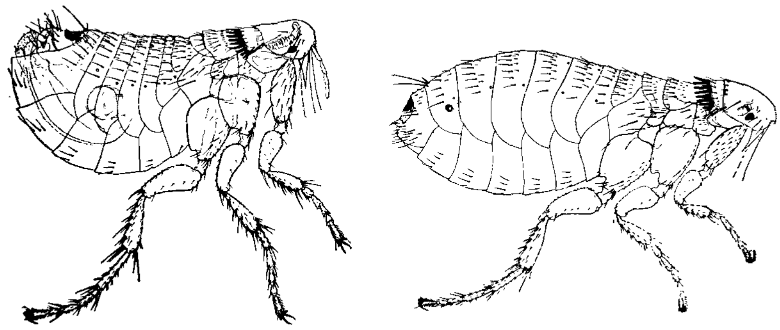


FIG. 145. *Nosopsyllus fasciatus,* the rat flea; male (*left*), female (*right*). ×17.

18 or 20 spines. There are three bristles in front of the eye; and in the female two bristles, and in the male four in front of these. There are three or four hairs on the inner surface of the hind femur. *N. fasciatus* (Bosc) is regarded as a negligible factor in the causation of natural outbreaks of plague.

The genus *Nosopsyllus* may be distinguished from the genus *Diamanus* by the fact that in *Diamanus* there are long, thin bristles on the inside of the mid and hind coxae from the base to the apex, while in *Nosopsyllus* such bristles occur at most in the apical half.

Diamanus montanus (Baker) (*Ceratophyllus acutus* Baker) (Fig. 146) is a common species of squirrel flea described from California. This species may be recognized by a spine at the tip of the second joint of the hind tarsus longer than the third joint and reaching over on to the fourth joint; the abdominal tergites have each two rows of bristles; the male claspers are very large and long, and sickle-shaped.

Ceratophyllus niger Fox was originally described from specimens taken from man and from *Rattus r. norvegicus* in California.

Ceratophyllus gallinae Schrank is commonly known as the European hen flea although it has a wide range of hosts. See previous pages.

Family Hectopsyllidae. *Tunga penetrans* (Linn.) [*Dermatophilus* (= *Sarcopsylla*) *penetrans* (Linn.)] is commonly known as the chigoe flea.

Echidnophaga gallinacea (Westw.) is commonly known as the stick-tight flea of poultry and other animals.

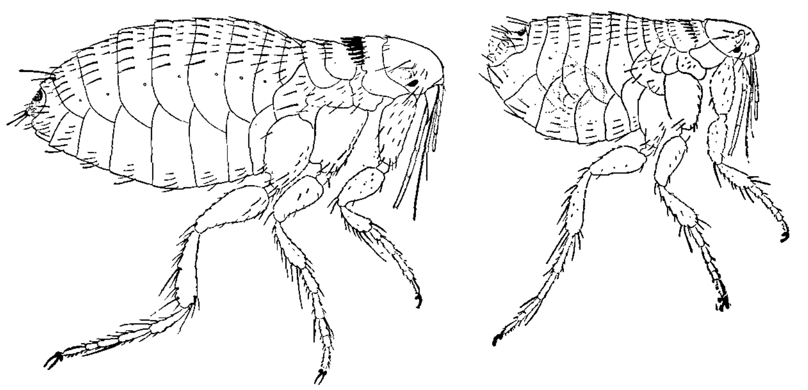


FIG. 146. *Diamanus montanus,* the squirrel flea; male (*right*), female (*left*). ×17.

Family Hystrichopsyllidae. *Ctenopsyllus segnis* (Schön.) (*Leptopsylla musculi* Duges) is the cosmopolitan mouse flea (Fig. 147). It is commonly found on rats. It bites man reluctantly and is regarded as a weak vector of plague; its role in human outbreaks is considered negligible.

Rat fleas on ships and at seaports. The United States Public Health Service has conducted a number of rat-flea and rat surveys at various seaports. Williams[42] reports that on a two-year survey of ships at the Port of New York 1,913 ships produced 18,265 rats, an average of 9.6 rats per ship. The ship rat is almost exclusively the black rat, *Rattus r. rattus* (Linn.) and the roof rat, *Rattus rattus alexandrinus* (Geoffroy-St. Hilaire and Audouin), constituting 99.65 per cent of all rats. Because

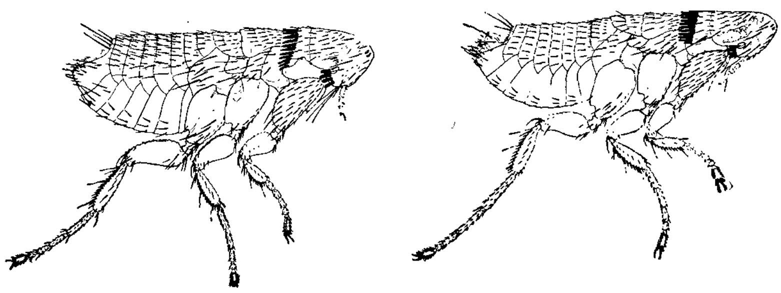


FIG. 147. *Ctenopsyllus segnis,* a mouse flea; male (*right*), female (*left*). ×17.

of the climbing habits of these rats, they are more likely to get into cargo and aboard ships than the Norway rat. The report indicates that the majority of ships carry few rats, and only about 50 per cent of arriving ships constitute about 90 per cent of the potential plague menace.

The dead rats collected after ship fumigations (hydrocyanic acid) were examined for fleas. A total of 7,886 fleas were taken from 18,265 rats, an average of 0.43 per rat, which was about 30 per cent of the expectation of fleas from live rats. Of the total number of fleas, 6,992 (88.68 per cent) were *Xenopsylla cheopis* (Roth.) and 786 (9.97 per cent) *Nosopsyllus* (= *Ceratophyllus*) *fasciatus* (Bosc). The remaining number of rodent fleas were *Ctenopsyllus segnis* (Schön.), 63, *Xenopsylla brasiliensis* (Baker), 4. *Pulex irritans* Linn. appears as a single specimen and the cat and dog flea numbered but 7.

The rat-flea survey at Norfolk, Va. (Hasseltine, 1929),[43] resulted in the capture of 1,561 rats, of which 883 harbored fleas; 4,898 fleas were taken. Of these fleas 81.6 per cent were *Xenopsylla cheopis* (Roth.), and 17.7 per cent were *Nosopsyllus* (= *Ceratophyllus*) *fasciatus* (Bosc). The Norfolk survey was based on caged, trapped rats taken under favorable conditions for harborage and propagation. Consequently, the number of fleas per rat (the living rats were chloroformed) was much higher than the average per ship rat on fumigated vessels, i.e., 5.5 against 0.43; also *Rattus r. norvegicus* (Erxleben) constituted all but four of the total number of rats taken at Norfolk.

At the port of New Orleans, Fox and Sullivan[44] report that from 3,839 live rats 17,559 fleas were taken, of which 6,566 were *Xenopsylla cheopis* (Roth.), 10,269 were *Ctenopsyllus segnis* (Schön.), (*Leptopsylla musculi* Duges), and 724 were *Nosopsyllus* (= *Ceratophyllus*) *fasciatus* (Bosc) —an average of 4.83 per rat. These authors state that it is quite significant that at the port of New Orleans, where plague has actually existed, *Xenopsylla cheopis* (Roth.) is the predominant rat flea present during every month of the year.

Inspection of vessels for rat infestation has two principal objects according to Williams:[45] first to determine the presence or absence of rats so as to determine whether or not a vessel shall be fumigated or otherwise treated to kill fleas and rats; and second, to determine the location of rats when present so as to apply fumigation effectively and to maintain proper rat-proofing.

The chigoe flea. *Tunga* (= *Sarcopsylla*) *penetrans* (Linn.) (Fig. 148) belongs to the family Hectopsyllidae and is known as "chigoe," "jigger," "chigger," "chique," "nigua" or "sand flea." The head of this flea is definitely angular and is usually larger proportionately than the heads of other fleas; there are no ctenidia on the head or pronotum; the mouth parts are conspicuous; the palpi are four-segmented. *Tunga penetrans*

(Linn.) is a tiny burrowing flea found in the tropical and subtropical regions of North and South America, also in the West Indies and Africa. Its introduction into Africa is said to have occurred as late as 1872. The chigoe is reddish brown and measures about 1 mm in length except that the impregnated female may become as large as a small pea. The adult fleas are intermittent feeders but adhere closely to the host. The female when impregnated proceeds to burrow into the skin of the host, frequently between the toes and into the soles of the feet, causing nodular swellings which ulcerate. The eggs are deposited either in the ulcer or drop to the ground when discharged from the body of the female, the tip of whose abdomen protrudes slightly from the nodule. The larvae

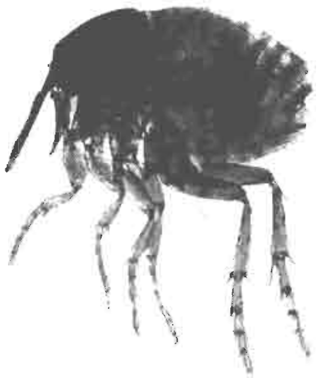


FIG. 148. The chigoe flea, ***Tunga penetrans***.

which emerge in a few days from the eggs are typical flea larvae. Those hatching in the ulcer usually drop to the ground to develop under conditions similar to those having hatched on the ground. Faust and Maxwell (1930)[46] report a case in which the eggs had hatched in or on the body around the sites of the burrows of the gravid females, and the larvae had thrived and grown there. The larval period under favorable conditions probably requires not more than 10 to 14 days and the cocoon or pupal period about a like number of days.

Pathogenesis. The chigoes commonly attack the bare feet, these being nearest the ground, infesting the skin between the toes and soles; but no part of the body is exempt from attack. The burrowing female flea causes extreme irritation; the area surrounding it becomes charged with pus, producing a distinct elevation. The ulcerations due to the presence of numerous chigoes become confluent. Wellman in a personal communication to the author attributes the commonly observed autoamputation of toes of natives in Angola to the work of the chigoe. Tetanus and gangrene frequently result.

Where the chigoe flea occurs, walking in bare feet should be avoided. Parts of the body attacked by the fleas should receive immediate attention. Repeated bathing of the infested part with Lysol gives good results. The insect can be removed quite easily by means of a sterile needle or very fine-pointed knife blade. The wounds caused by this treatment must be carefully dressed and allowed to heal.

The sticktight flea. *Echidnophaga gallinacea* (Westw.), also known as the "sticktight," is a serious poultry pest in many parts of subtropical America. It commonly attacks poultry of all kinds, also cats, dogs, rabbits, horses, and man. It differs from *Tunga penetrans* (Linn.) in having the angle of the head acutely produced, while in *T. penetrans* (Linn.) the head is obtuse instead of rounded and the eyes and antennae are in the posterior half of the head. It is from 1 to 1½ mm in length.

Before copulation both sexes are active, hopping about much as do other species of fleas. Shortly after feeding, the females attach themselves firmly to the skin of the host and begin to burrow. At this time the sexes are in copulation. The burrowing females deposit their eggs in the ulcers which have been produced by the infestation. The usual incubation period according to Parman[47] is from six to eight days at a temperature average of 76° F. If the eggs are deposited in the ulcer, the larvae crawl out and drop to the ground, where they grow rapidly under favorable conditions, feeding on nitrogenous matter, dry droppings, etc. The full grown larva, which is not unlike other flea larvae, is about 4 mm in length, reaching this stage in about two weeks. The larva then spins a cocoon, pupates, and in about two weeks (9 to 19 days) emerges as a fully developed flea. The life history requires from 30 to 60 days. Eggs are also deposited in the dust or dry droppings of poultry or in old nests, etc.

The fleas are most likely to attack the skin around the eyes, the wattles and comb, and the anus or other bare spots. The ulceration and wart-like elevations around the eyes often become so aggravated that blindness occurs, the host is unable to find its food, and death results. Since this flea also lives on dogs, cats, rats, quail, blackbirds, and sparrows, suitable precautions should be taken to exclude these from chicken pens.

The western hen flea, *Ceratophyllus niger* Fox, is considerably larger than the sticktight and does not attach except when feeding and then only for a brief period. It readily attacks man and cats and dogs. It breeds primarily in fowl droppings. The European hen flea, *Ceratophyllus gallinae* Schrank, which has habits similar to *C. niger* Fox, is also at times a serious pest of poultry (Stewart, 1927).[48]

Fleas in the household. Very few species of fleas are annoying to household pets. Among these are particularly the dog and cat fleas. *Cteno-*

cephalides canis (Curt.) and *Ctenocephalides felis* (Bouché) (Fig. 149), and the so-called human flea, also known as the sand flea, *Pulex irritans* Linn. While the common name might imply that there is a specific host relationship, this is not the case, since interchange of host species is quite usual. Cat and dog fleas readily attack humans, and the human flea is often remarkably abundant on swine.

Fleas in the house generally indicate that cats or dogs or both are present or have been present fairly recently. Fleas may be carried on clothing into the house from pig pens or from flea-infested public meeting places. The exclusion of cats and dogs or their proper management is necessary to prevent flea infestations. Cats and dogs (unless properly

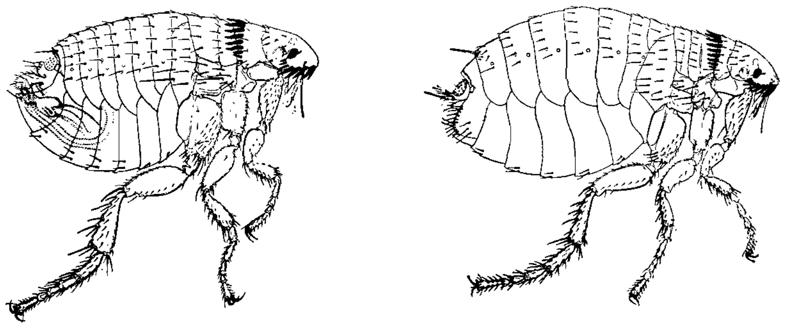


FIG. 149. *Ctenocephalides felis*, the cat flea; male (*left*), female (*right*). ×17.

bedded), as well as rats, must be excluded from the basement and from beneath the house.

Ordinarily fleas lay their eggs on the infested animal, but because the eggs are dry, they drop off when the host shakes itself. For this reason mats should be provided upon which the animals may sleep at night, and these should be shaken off every day or two over fire or into kerosene (see flea control). The eggs are minute glistening white objects. The incubation period varies considerably, but they usually hatch in from five to six days, sometimes less, and the worm-like sparsely haired larvae emerge. The larvae feed on particles of dry blood, fecal matter, and various organic substances collected in corners and crevices. The larvae are quite active and in two to four weeks reach a length of about one-fourth inch and then spin a crude cocoon in which they pupate. The flea emerges from the cocoon in about a week; thus three to four weeks are required for the entire life history of a common house flea under favorable conditions. Undisturbed mats, rags, and carpets favor the development of fleas. All carpets or matting tacked down and covering the floor should be dispensed with and smaller rugs substituted. Houses that have been vacant for several weeks may be badly infested with fleas because these insects are able to live without food for several weeks.

Fleas as intermediate hosts of cestodes. Although Melnikoff in 1867 showed that the biting louse of the dog, *Trichodectes canis* DeGeer, serves as an intermediate host of the double-pored dog tapeworm, *Dipylidium caninum* (Linn.); it has since been shown by other workers that fleas play a more important role in the transmission of this tapeworm, particularly the cat and the dog flea, *Ctenocephalides felis* (Bouché) and *Ct. canis* (Curt.). Although *Dipylidium caninum* (Linn.) is a tapeworm of dogs, cats, and certain wild carnivores, it also occurs in man, particularly in young children. The tapeworm measures from 20 to 70 cm in length; the mature proglottids are shaped like pumpkin seeds, and each has a double set of reproductive organs with a genital pore on each side. The scolex has a rostellum which is armed with three to seven circlets of spines and has four deeply cupped oral suckers. The embryonated eggs of the tapeworm are discharged in the fecal material of the host and are ingested by the larval flea and develop into cysticercoids in the body cavity of the insect. Thus the mature flea is infected and when ingested by a cat or dog or human, the cysticercoids are liberated and develop into tapeworms in the animal's digestive tract.

A common tapeworm of rats and mice, rarely of man, *Hymenolepis diminuta* (Rudolphi), has numerous intermediate arthropod hosts, among them *Nosopsyllus fasciatus* (Bosc) and *Xenopsylla cheopis* (Roth.).

Flea control. With a substantial background of knowledge concerning the parasitic habits, the life history, and particularly the ecology of fleas, control of these pestiferous, disease-bearing insects can be accomplished. Management of host animals and the practice of good sanitation in the household and in public places are fundamental principles which must not be neglected granting the effectiveness of the newly discovered insecticides, the proper use of which must be understood as well as their limitations.

Dichloro-diphenyl-trichloroethane (DDT) may be applied either as a 10 per cent dust (particularly when used in murine typhus fever control operations) or as a 5 per cent DDT-kerosene solution for application as a spray to floors and beneath rugs in the house and sleeping places of cats and dogs. One gallon of 5 per cent DDT-kerosene solution sprayed lightly over areas of 1,000 to 2,000 square feet will completely eradicate fleas. DDT is not a repellent, and it kills slowly. It retains its killing effect against fleas for about two months.

When DDT is used as a dust it is mixed with pyrophyllite, Fuller's earth, calcium dust, or walnut shell flour and is applied with a hand duster or with a hand shaker. That DDT dust kills fully grown flea larvae [*Ctenocephalides felis* (Bouché)] as well as adults was shown by Kerr (*loc. cit.*), who used among other rates a 5 per cent DDT-pyrophyllite dust. All larvae died within 24 hours after treatment. Adult fleas remained normal in behavior for about 10 minutes after exposure to toxic doses, then followed a period of increasing activity, with the fleas jumping vigorously in 15 to 20 minutes; within an hour they were incapable of movement except a slow waving of the legs.

Wherever pets sleep or are accustomed to lie down DDT dust (10 per cent recommended) is applied where fleas will come in contact with it. The dust should be forced into cracks and crevices and in all places where fleas may breed. Excess powder if objectionable after a few days can be wiped up with a cloth or removed with a vacuum cleaner. Flea-infested basements as well as yards and other out-door areas should be similarly dusted.

Dogs may be safely dusted with DDT; often only a line of 10 per cent dust along the middle of the back is sufficient to kill all the fleas and to prevent reinfestation for several weeks. Since DDT agitates fleas before killing, this activity causes the dog to scratch and bite vigorously until the insects cease crawling about.

Although DDT dust is not absorbed through the skin of mammals, cats should not be treated with DDT because of their cleaning habits; however, treating the cat's bed is usually sufficient. Flea powders containing rotenone and pyrethrum are safe to use on cats.

In the use of DDT dust (10 per cent DDT) in murine typhus control, the United States Public Health Service points out that this is aimed at the flea link in the typhus chain. Proper dusting of rat runs and harborages with DDT combats fleas both on and off their rodent hosts.

With regard to the use of DDT for the control of murine typhus fever the United States Public Health Service points out that rats in passing over the dusted places, pick up a considerable amount of dust on their feet, bellies, and tails, and fleas on the rats come in contact with the DDT dust and, if exposed to it for a sufficient length of time, are killed. Thorough residual dusting (not too heavy) of rat runways, both inside of buildings and in outside areaways, is essential not only to ensure killing fleas on the rats but also to kill the fleas (off the animals) in or near rat runs, nests, or harborages. Treatment of premises and buildings should be repeated at intervals of two or three months throughout the flea breeding season. Success depends upon a good knowledge of the habits of rats and careful inspection.

Flea repellents. Tests made by Lindquist, Madden, and Yates[49] show that insect repellents used during World War II, dimethyl phthalate, Rutgers 612, and indalone, are effective flea repellents giving an average repellent period of 139–260 minutes, the first giving the longest. Tests made with a mixture of one per cent pyrethrins and 2 per cent IN-930 in mineral oil gave even a longer repellency.

Rodent control. Officials responsible for the prevention and control of plague, murine typhus, and other infections traceable to rats and other rodents must recognize the fact that prevention and eradication of such diseases depends on intelligent rodent control, a function that should be placed on a much higher plane than is usually implied in the duties of the so-called "rat catcher." The position of rodent control officer calls for a high degree of intelligence, much technical training, and a substantial understanding of the whole problem; this person deserves the same respect and credit that is accorded any other individual who functions in the field of public health. The rodent menace must be met with intelligence; rats in particular must not be tolerated; they constitute not only a sinister menace to health, sufficient in itself to condemn them, but if further incentive to their extermination is necessary it is reported on good authority that a rat eats $2.00 worth of food each year and destroys ten times this amount by fouling, gnawing, and otherwise damaging grain, stored products, and other articles; 200,000,000 bushels of grain are reported to be destroyed or contaminated by rats annually in the United States; thus rats cause an aggregate annual loss of over $2,500,000,000 in the United States alone.

Rats. Together with the house mouse, *Mus musculus* Linn., the cosmopolitan species of rats belong to the order Rodentia (gnawing mammals), family Muridae. *Rattus rattus norvegicus* (Erxleben), the brown rat or sewer rat, better known as the Norway rat, is the largest of the common house rats. Full-grown adults measure about 16 inches in total length (tail 7½ inches), and weigh 11 to 12 ounces (exceptionally 24 ounces). The nose is blunt; the ears are of moderate size and slightly hairy; the tail is scaly, nearly naked, and blunt-ended. The body fur is coarse and generally brown, with scattered black hairs; the under parts are pale grey to yellowish white (Fig. 150). *Rattus rattus rattus* (Linn.) is the black rat, which is almost solidly blue-black in color; it is smaller and more slender than the Norway rat. The nose is sharply slender, and the ears are noticeably large, thin-membraned, and naked; the tail is slender and longer than the body and head. *Rattus rattus alexandrinus* (Geoffroy-Saint Hilaire and Andouin) is called the Alexandrine or roof rat, and also the tree rat. In color it is grayish brown above and white or yellowish white below (hence sometimes called yellow-, gray-, or white-bellied tree or roof rat). It is usually classified as a subspecies of the black rat, as it has the same general characteristics in form and habits. Both can climb readily and may nest in trees, palms, and attics of houses. *Rattus rattus frugivorus* Rafinesque is called the white-bellied frugivorus rat and is said to be an outdoor rat that does not live close to man. It is usually regarded simply as a color phase of *R. r. alexandrinus*. *Rattus hawaiiensis* Stone is now a common species of rat on Maui and other Pacific Islands. It is called the Polynesian rat and is a small species averaging about 60 grams in weight. It lives in grass and brush areas away from human habitations. Doty[50] states that it is very destructive to sugar cane. In color it is "cinnamon brown above, cinnamon buff on the sides, and light buff or buffy white below, strongly mixed with black hairs on the back and sides. The feet are nearly white above."

Rat biology. Since successful control is based on a full knowledge of the biology, particularly feeding habits and nesting habits, of the several species of rodents involved in a given problem, much attention must be given to this subject. Sufficient literature is available to serve as a guide,

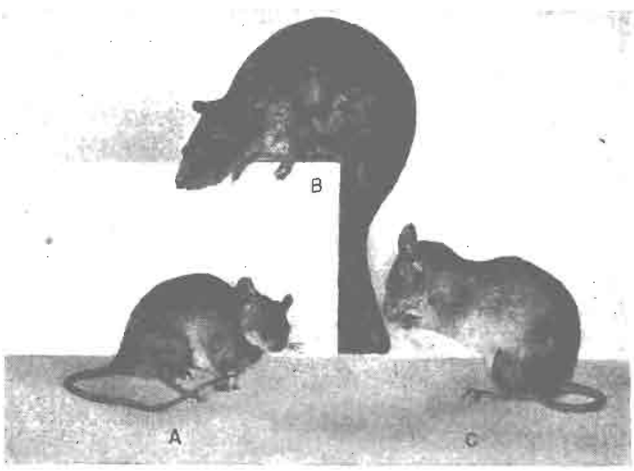


FIG. 150. House rats. (*A*) *Rattus r. rattus,* the black rat; (*B*) *Rattus n. norvegicus,* the brown rat; and (*C*) *Rattus r. alexandrinus,* the roof rat. ×.13.

but there is no substitute for field observation and experience. Each species of rat must be studied carefully inasmuch as their habits may differ considerably; a certain procedure which results successfully in one case may fail utterly in another.

Most species of rats are active at night; however, *Rattus hawaiiensis* feeds commonly during daylight hours; also unlike other species which are omnivorous, the Polynesian rat lives on grass and weed seeds along with some vegetative parts of plants such as sugar cane (Doty, *loc. cit.*). Likewise this latter species is said to be quite docile, while the cosmopolitan species listed above are as a rule vicious. Norway rats are cannibalistic and will devour young rats in nests, eat dead rats, and attack one another under stress. A knowledge of food habits and feeding habits is particularly important in the use of poisoned baits and prebaiting.

The Norway rat stays mainly at the ground level and seldom goes above the first floor of a building. It burrows underground a foot or two and makes tunnels several feet in length with several hidden exits or "bolt holes." Crude nests are made in burrows under buildings, beneath piles of lumber, in garbage dumps, along banks of ditches and streams. It favors pigpens, slaughterhouses, barns, chicken yards, and sewers. Roof rats, Alexandrine and black rats, climb readily and often live in attics and upper stories of buildings; they commonly nest in trees, especially tops of palms and in dense hedges or vines growing on fences. Like the Norway rat, the roof rat can swim readily. Alexandrine and black rats are far more common on ships than the Norway rat.

According to Storer[51] female Norway rats average 8 to 9 embryos (extremes 2 and 17) at a pregnancy and may breed four or more times during a year. Pregnancy lasts 21 to 22 days. The young are able to shift for themselves when about 3 weeks old. A female may first breed at the age of 4 to 5 months. Although it is not known how long wild rats live (some authorities believe about three years), the population is probably largely replaced by new individuals each year. One pair of rats may produce a population of more than 1,500 by the end of a year. Early spring and summer are the periods of greatest production, though young rats are found during any month of the year.

There are nine signs made by rats which are useful in detecting infestations (*Rats and their Control;* Rat and Mosquito Control Commission, Chamber of Commerce of Honolulu, 1943): (1) droppings, (2) runways, (3) tracks over dust or soft surfaces, (4) burrows, (5) gnawings, (6) live rats, (7) dead rats, (8) nests, (9) rat odor. Storer (*loc. cit.*) lists in addition sound, i.e., squeals, scampering in an attic; smears along beams, etc., made by contact of the body of the rat; and urine stains. With adequate information as here suggested, a fairly accurate rat census can be made.

Methods of rat control. One should aim at permanent control as the only satisfactory solution of the rat problem. There should be good sanitation the year round. Good housekeeping is good insurance against rats; cleanliness is the rat's worst enemy. The United States Fish and Wildlife Service (Circular 13, 1948) points out that food and shelter are the two most important factors in a rat's existence. Any program of rat control to be successful must incorporate these four major phases: (1) elimination of rat harborages, (2) elimination of food supply for rats, (3) rat proofing of buildings, and (4) destruction of rats.

Rat proofing and rat stoppage. Silver, Crouch, and Betts[52] state that the rat problem will have been largely solved when rat proofing becomes the regular practice. Storer (*loc. cit.*) points out that rat proofing (exclusion) is not enough; such construction must be frequently inspected and kept in good repair. Rat proofing can be done at the time of construction at slight extra cost, but when applied to old buildings it is usually an expensive procedure. However, exterior rat proofing, i.e., *rat stoppage* can be done inexpensively (Holsendorf and Clark[53]). Loss occasioned by rats on farms in the United States is reported to amount to $63,000,000 annually, much of which could be averted by ratproofing barns, corncribs, granaries, poultry houses, and pig pens (*building out* and *starving out* rats) as described by Silver, Crouch and Betts (*loc. cit.*).

Cutting off the rat's food supply (starving out the rat) refers not only to ratproofing so as to make food inaccessible; it also refers to the sanitary disposal of refuse from the household, garbage, slaughter-house refuse, feed refuse in connection with piggeries, poultry houses, barns, feeding lots, etc. Cleanliness, neatness, good housekeeping in general, and efficient farm management will aid materially in the control of rats and other rodents.

Destruction of rats. In the use of rat poisons a system of prebaiting and baiting is employed. Since the rat poison is usually mixed with rat food, one should have some knowledge of the food habits of these rodents. Rolled oats and other cereals, corn meal, bread crumbs, diced bread, raw meat (horse meat is acceptable to rats), and raw fish are the commonest baits used. Prebaiting is a practice based on the fact that rats are suspicious and avoid new objects, even a new food. If a clean prebait is offered they will overcome their fear in a few nights (two or more) and will accept the new food. Small piles of clean unpoisoned baits are placed at selected sites or in special containers for four or five nights replenishing the supply whenever the bait is eaten. On the fifth or sixth day the unpoisoned prebait is replaced with poisoned bait. If the poisoning is successful the above-described routine may not need to be repeated for several months; otherwise a follow-up campaign may be undertaken in a couple of weeks.

Among the commoner less dangerous rat poisons are the following: *red squill, antu, zinc phosphide, and barium carbonate*; but *1080* and *thallium sulphate* should only be used by trained professional operators. (1) *Red squill* is obtained from an onion-like plant (*Urginea maritima*) native to the Mediterranean area. The bulbs are sliced, dried, and ground to a fine reddish powder. Different batches of this powder vary in toxicity; hence squill of lower toxicity is fortified until its toxic strength reaches the point where rats are killed by 500 to 600 milligrams per kilogram of live rat (such tests are known as bioassays). For use one part by weight of red squill powder is well mixed with 9 parts of bait such as raw hamburger steak (horse meat is acceptable to rats) mixed with rolled oats or bread crumbs wet with water. A convenient way of handling the bait is to place a small quantity about the size of a marble in a paper square and twist in the form of a "torpedo." A little glycerin mixed with the bait prevents hardening and sodium sulphite may be used as a preservative. Red squill is the safest rat poison for general use since experiments show that cats, dogs, chickens, pigeons, and hogs usually will not eat it, probably because of its disagreeable taste, or will quickly vomit any which may have been eaten. *Red squill is a poison, hence should be treated accordingly and be placed out of reach of children and pets.*

(2) *Antu* is an abbreviation for the chemical alpha-napthyl-thiourea, a light, gray powder, quite insoluble in water, highly stable and non-irritating to the human skin. It is highly toxic to the Norway rat in concentrations of 1 to 1½ per cent, but much less toxic to Alexandrine and black rats. This deficiency can be corrected by increasing the percentage of "antu" in baits to 5 per cent. It kills rats by causing an accumulation of body fluids within the chest cavity. Sublethal dosage quickly builds up a tolerance. Antu is particularly toxic to dogs and young chicks; it can also poison pigs. Monkeys tolerate large quantities, hence it may not be dangerously toxic to human beings; however, *antu should be guarded like any other poison to prevent children, pets, domestic animals, or foodstuffs from coming in contact with it. Keep antu out of the mouth; do not eat or smoke when handling it.* As a rat poison it is used in baits in concentrations referred to above, or it can be used as a dust on runways or pumped into burrows in a mixture of 20 per cent antu, 2 per cent DDT, and 78 per cent pyrophyllite. This formula tends to control rat fleas as well as the rats.

(3) *Zinc phosphide* is a heavy dark-gray powder with a faint odor of phosphorus. It is used in baits at a percentage of from 2½ to 5 per cent.

(4) *Barium carbonate* is a fine white powder. The proportions used are: bait, 4 parts by weight, and barium carbonate, 1 part by weight. It is slow acting and although regarded as mild it may still be fatal to chickens, dogs, and cats; hence great care must be exercised in its use. *It must be inaccessible to children and to pets and other animals. It is a poison.*

(5) *Compound 1080 (sodium fluoroacetate)* is a violent poison (Ward and Spencer[54]). Water poisoned with ½ ounce of 1080 per gallon will kill monkeys on the basis of about 3 ounces of the solution to a 150-pound animal. It is highly fatal to man. Cats and dogs are almost sure to be fatally poisoned if rats, mice, or other rodents killed with 1080 are eaten. Compound 1080 should be used only by carefully instructed, reliable personnel competently supervised. It should not be used in dwellings.

(6) *Thallium sulfate* is a heavy-metal poison obtained from smelters. It is used by governmental agencies for the control of both rats and field

rodents. For thallium, as for 1080, there is no certain antidote. It is a slow acting poison in contrast to 1080 which acts very quickly.

Toxic gases such as *calcium cyanide, sulfur dioxide, methyl bromide,* and *carbon monoxide* (from automobile exhaust) are commonly forced into rodent burrows by means of special pumps to kill rats and other burrowing rodents.

Use of traps. In trapping rats the proper placement of the trap is far more important than the selection of a bait. Bacon strips, a piece of fresh fish, or bacon-scented oatmeal is better than cheese as a bait according to the "Fish and Wildlife Service." Such baits should be tied firmly to the trigger of the trap.

Dead rats resulting from poisoning, gassing, trapping, or any other cause should be treated copiously with kerosene, boiling water, or an insecticide before disposal, to destroy any fleas still present on the body. Incineration or deep burial of dead rats is good practice. *Deodorants* such as powdered activated charcoal may be used where rats have died within walls. Storer (*loc. cit.*) refers to a commercial product, Hyamine 1622, as useful to reduce such odors.

Field rodents. Among the more important field rodents are the ground squirrels belonging to the genus *Citellus,* family Sciuridae. They are important reservoirs of sylvatic plague and tularemia and may cause losses to cereal crops amounting to from 10 to 15 per cent or more. They materially reduce the forage in pastures and may damage ditch banks and levees by burrowing.

Storer[55] (1947) states that there are seven varieties or subspecies of ground squirrels in California, each occupying a separate area. The most widespread is the dark-colored Douglas ground squirrel, *Citellus beechyi douglasii* (Richardson), with much black between the shoulders; it occurs northward from San Francisco Bay throughout the region west and north of the Sacramento and Feather rivers. The brownish Beechey ground squirrel, *Citellus beechyi beechyi* (Richardson) (Fig. 142) occupies coastal California from the Golden Gate and Carquinez Strait south nearly to San Diego. The gray-toned Fisher ground squirrel, *Citellus beechyi fisheri* (Merriam), inhabits the greater part of central California from the Feather and Sacramento rivers south to the southern end of the San Joaquin Valley. The Oregon ground squirrel, *Citellus oregonus* (Merriam), a short-tailed brownish-gray species of the northeastern part of California, is essentially an inhabitant of grasslands. The Belding ground squirrel, *Citellus beldingi* (Merriam) is known as the picket-pin; it is also bob-tailed and is found in the high Sierra at elevations of 6,500 feet to 12,000 feet.

Every ground squirrel has two thin, internal cheek pouches, opening inside the lips, one on each side of the mouth, which are used to carry food. Storer points out that this characteristic is important in control because ground squirrels may be killed by absorbing poison on baits through the lining of the pouches.

Habits. Ground squirrels find their food on the surface of flat country, hillsides, or embankments. They are active by day during the warmer season of the year and on warm days in winter. All species dig burrows for safety, for shelter during very hot or rainy weather, for hibernation, for storage of food, and for the rearing of young. Burrows are about 4 inches in diameter, within 2½ to 4 feet below the surface, and may reach a length of 30 feet; occasionally they are much longer. Entrances to the burrows are always open. These tunnels, often branched, are made in hillsides, in ditch banks, in railroad embankments, and in flat lands. Storer describes a complicated system of tunnels with a total length of 471 feet and 33 opening.

During the rainy months ground squirrels feed chiefly on green herbage and seeds on the ground; when seed crops ripen these animals collect unhulled seeds of both wild and cultivated plants in their cheek pouches and hide these in caches in the ground and in various crevices for later use.

Estivation and hibernation. In the lowlands ground squirrels are reported to estivate as early as mid-May, whereas in the high mountains they do not begin hibernation until as late as early August. Emergence occurs in late winter or early spring. Storer (*loc. cit.*) points out that estivation explains why old breeding adults suddenly appear in spring, after all squirrels active in a field during autumn had been killed, and there seemed no chance for migration from surrounding fields. It is extremely important to carry on intensive control in the spring and early summer when all squirrels are active. During hot summer weather and in the autumn and early winter, some of the squirrels may be underground and out of reach. There is some doubt whether dormant animals are killed by gassing their burrows with carbon disulfide.

Breeding. Ground squirrels probably produce only one litter a year, of which about 5 or 6 are said to survive long enough to appear above ground. The breeding season is during the months of December to April. The young are seen in greatest numbers during May and June, when they scatter to new territory or move to unoccupied old burrows. Control operations, therefore, should be especially intensive in late winter and early spring.

Ground-squirrel control. Storer (*loc. cit.*) states that there are six general means of controlling ground squirrels, viz.: (1) poison bait, (2) poisonous gases, (3) trapping, (4) shooting, (5) exclusion, and (6) encouragement of natural enemies.

Strychnine-coated barley. In the use of poison for squirrels several important factors must be considered; namely, it must not be distasteful to the rodents and must enter the circulation readily; the poison must be applied to food readily eaten by the squirrels and at a time when the usual green food is at its minimum. It has been found that strychnia sulphate (the pure alkaloid should not be used) is most effective, but the bitter taste must in some manner be concealed. The use of whole barley, i.e., threshed but still retaining the husk, is recommended. In this form the barley is not eaten by birds and is most acceptable to the rodents; it is also cheaper in this form. Wheat is very acceptable to the rodents but when poisoned is very destructive to birds, particularly quail, doves, and other grain eaters.

Piper has devised a formula whereby the bitter taste of the strychnine is delayed about two minutes, thus enabling the squirrel to fill its cheek pouches before the bitter taste is noted. The formula is as follows:

Formula for strychnine-coated barley. Following is a government formula for preparing poisoned barley for California ground squirrels.

Barley (clean whole grain)	16 quarts
Strychnine (powdered alkaloid)	1 ounce
Bicarbonate of soda (baking soda)	1 ounce
Thin starch paste	¾ pint
Heavy corn sirup	¼ pint
Glycerin	1 tablespoonful
Saccharin	1/10 ounce

Mix thoroughly one ounce of powdered strychnine (alkaloid), and one ounce of common baking soda. Sift this into three-fourths pint of thin, hot starch paste and stir to a smooth, creamy mass. (The starch paste is made by dissolving one heaping tablespoonful of dry gloss starch in a little cold water, which is then added to three-fourths pint of boiling water; boil and stir constantly until a clear, thin paste is formed.) Add one-fourth pint of heavy corn sirup and one tablespoonful of glycerin and stir thoroughly. Add one-tenth ounce of saccharin and stir thoroughly. Pour this mixture over 16 quarts of clean barley and mix well so that each grain is coated.

Caution.—All containers of poison and all utensils used in the preparation of poisons should be kept *plainly labeled* as *POISON* and *out of reach* of children, irresponsible persons, and livestock.

Grain poisoned with strychnine placed in proper containers will retain its poisonous character and remain effective for an indefinite period, but heavy dews and rain may remove the poison and destroy the effectiveness of the bait. Therefore, this method is applicable during the dry season only. Simpson[56] reported that 30 kernels in the cheek pouches of squirrels rapidly prove effective, whereas 60 or 90 or more in the stomach may produce only a few convulsions and recovery ensues. He says, "This fact should be remembered in placing poison, for by scattering the grain a few kernels here and there near the burrow the squirrel is induced to store the grain temporarily in the cheek before a sufficient quantity is obtained for a meal. . . . " It should be scattered where the squirrel is accustomed to find food, and will probably be found most efficient if placed early in the morning, between the hours of 3 A.M. and 7 A.M.

Other poisons such as zinc phosphide may be used with such baits as oat groats, whole oats, and whole barley; mineral oil, petroleum jelly or lecithin hold the poison to the bait.

1080. Field rodents such as ground squirrels and prairie dogs are very susceptible to 1080 poisoning. Effective control has been obtained by the use of concentrations of 1 to 2 ounces mixed with 100 pounds of grain; however, the control of field rodents is a complex procedure and should only be undertaken by experienced persons authorized by law to do so. The careless use of 1080 will endanger domestic livestock and beneficial wild life found in the same habitat. The secondary hazard to dogs, cats, pigs, and carnivorous wild life following the use of 1080 in field rodent control is considerable.

Gassing with carbon bisulfide. Carbon bisulfide is an effective fumigant for ground squirrels and is commonly employed. Carbon bisulfide is obtained commercially in the form of a liquid, which is readily vaporized or is converted chemically into other gases. While it is a useful material against ground squirrels, there are some objectionable features; namely, it is very inflammable, must be kept in tightly closed containers, and, under certain conditions may explode; furthermore, during the dry season if "exploded" in the burrow there is danger of igniting dry grass or other inflammable material in the vicinity. If handled with as much care as gasoline, for example, the danger is not great. The advantages in its use are these: it is readily converted into a poisonous gas, diffuses quickly, destroys life rapidly, and can be used most readily during the rainy season, when green food is abundant, and the use of poisoned grain is not practicable.

Carbon bisulfide is applied in two ways: with a special pump to force the fluid or gas into the burrow or by soaking waste balls in the fluid and then placing them in the burrow. The balls of waste may be ignited, but this is hazardous. In either case it is suggested that from one to three days prior to the application of the poison all squirrel burrows in the area to be treated should be stopped with earth. The holes found opened indicate the burrow in which there are squirrels.

The method of applying carbon bisulfide by the ignition method is as follows: to handle a large area to the best advantage two men should work together.

One man is provided with a supply of "waste," "sacking," or other absorbent material, divided into a number of small balls about half the size of the fist. The bisulfide is carried in an ordinary one-gallon oil can, and refilled from time to time from a supply kept in a cool place out of the sun. He is supplied with matches. (Matches are dangerous, hence other methods of exploding the gas should be used.) His "pardner" carries a mattock or long-handled shovel. On arrival at an opened squirrel burrow, a ball of "waste" is saturated with two ounces of bisulfide, dropped deeply in the burrow and then a match applied. After a moment's time the man with the shovel stops with earth this burrow and all other burrows near from which the gas escapes. On subsequent inspection of the field all opened burrows will indicate holes lacking affective treatment.

Exploding the bisulfide causes considerable gas to escape, but

the ignition produces a violent chemical reaction and as a result sufficient oxygen from the air combines with carbon and sulphur elements to produce a volume of gas three times that which the original bisulfide would produce on evaporation. The gases produced, carbon dioxide and other sulphur dioxide, in the proportion of one to two, seem just as effective as bisulfide of carbon, and the method is superior in that the explosion produced drives these gases deeply into the burrow.

Two ounces of 60 cc of the bisulfide produce about 12 gallons of gas. To use the gas unexploded, simply omit igniting it.

A much cheaper and more efficient method of destruction with carbon bisulfide was early devised by Long,[57] namely, a pump with a device measuring the quantity of liquid and serviceable at all seasons of the year. This pump loaded with nine pints of refined bisulfide weighed 25 pounds. Only one-half ounce (15 cc) is required for each hole, against two ounces by the ignition method, and it is claimed that the men using the pump have been able to treat from 200 to 250 holes with each gallon of the bisulfide, against 50 to 60 holes per gallon with the waste ball method above described.

The use of Long's pump is described as follows: "Insert the hose in the squirrel hole at least one foot; then run one-half ounce of bisulfide from the reservoir into the measuring cup; then turn cock with handle down to allow liquid to run into vaporizing chamber, meanwhile covering the hole with dirt with the aid of a mattock. Then pump thirty strokes (in cold weather use one ounce with forty strokes). This equals 12 cubic feet or 1.5 per cent bisulfide gas. Withdraw the hose, close hole opening by stamping in the dirt with the heel and proceed to the next hole." Twenty minutes' to a half hour's treatment with air containing 2 per cent of carbon bisulfide is certain to be fatal. Improved carbon bisulfide pumps are on the market.

Gassing with methyl bromide. With the discovery that rodent fleas may serve not only as vectors of sylvatic plague but also as pseudo reservoirs, the necessity of flea control as well as rodent control is indicated in directing sylvatic plague suppressive measures. Carbon bisulfide is very uncertain and inefficient in its lethal action on adult fleas. Berry[58] found that methyl bromide, CH_3Br, 10 cc of liquid per burrow opening, may be used effectively against ground squirrels in wet or dry soils and at various temperatures. Methyl bromide is colorless, practically odorless, and noninflammable, but *it is a dangerous fumigant and should be used only by persons well informed concerning its hazards.*

Stewart and Mackie[59] (1938) have shown that ground-squirrel fleas of all stages are susceptible to this fumigant but that the adults are more easily killed than those in immature stages. These authors also found that the dosage used as a rodenticide, approximately 10 cc per burrow opening, is sufficient to kill the fleas in all stages of development. They point out that this fumigant has a peculiar characteristic in that "it does not kill immediately but manifests its toxicity in delayed kill without, however, exciting either the rodent or the flea before stupefying them."

Trapping and shooting. Where ground squirrels are digging into ditch banks, and in other cases where they must be disposed of promptly at any cost, special means must be adopted. Trapping and shooting are valuable under such circumstances. Both of these methods can be used at any season.

Natural enemies. Among the natural enemies of ground squirrels listed by Storer are coyotes, badgers, weasels, wildcats, red-tailed hawks, golden eagles, and gopher snakes. Badgers, weasels, and snakes capture the ground squirrels in their burrows. Wildcats and coyotes lie in wait near the burrows until the squirrels venture forth in search of food. "Dixon examined 186 stomachs of wildcats from 40 different localities in California; 26 contained ground squirrels and these with other rodents were found to constitute more than half of the food. Hawks and eagles swoop down on the squirrels from the air. The importance of preserving as many as possible of these native enemies of the ground squirrels is evident." When poison is used in the control of ground squirrels, reasonable precaution should be exercised to prevent killing their natural enemies.

Recently several investigators have called attention to the possible relationship of flesh-eating birds to the epidemiology of sylvatic plague. Jellison[60] states, "The abundance and variety of rodent fleas found in the nest of a burrowing owl (*Speotyto cunicularia*) suggest that this species may prove of particular interest. Casts from predatory birds fed plague-infected guinea pig tissue were consistently infectious." Wheeler, Douglas and Evans[61] report collecting sticktight fleas, *Echidnophaga* *gallinacea,* from the same species of owl in Kern County, California, during a plague epizoötic. Seventy individuals of this flea taken from one owl were mass-inoculated into a test guinea pig which proved positive for plague. This is probably the first record, according to these authors, of a bird host as a carrier of plague-infected parasites, and the first demonstration of natural plague infection of this species of flea.

BIBLIOGRAPHY

1. Snodgrass, R. E., 1946. *The Skeletal Anatomy of Fleas (Siphonaptera).* "Smithsonian Misc. Collect.," vol. 104, no. 18, Washington, D. C.: Smithsonian Inst. (Publ. no. 3815). 89 pp. (21 plates).

2. Kessel, Edward L., 1939. *The Embryology of Fleas.* "Smithsonian Misc. Collect." vol. 98, no. 3, Washington, D. C.: Smithsonian Inst. (Publ. no. 3527). 69 pp. (12 plates).

3. Bacot, A., 1914. "A study of the bionomics of the common rat fleas and other species associated with human habitations, with special reference to the influence of temperature and humidity at various periods of the life history of the insect," *J. Hyg.*, 13, (Plague Suppl. 2):447–654 (8 plates).

4. Mitzmain, M. B., 1910. *General Observations on the Bionomics of the Rodent and Human Fleas.* Washington, D. C.: Govt. Print. Office, in Pub. Health Bull., *no.* 38. 34 pp.

5. Bruce, W. N., 1948. "Studies on the biological requirements of the cat flea." *Ann. Entomolog. Soc. Amer.*, 41:346–52.

6. Kerr, R. W., 1946. "Control of fleas: Laboratory experiments with DDT and certain other insecticides," *J. Council Sci. & Indust. Research*, 19:233–40.

7. Mitzmain, M. B., 1910. "Some new facts on the bionomics of the California rodent fleas," *Ann. Entomolog. Soc. Amer.*, 3:61–82.

8. McCoy, George W., 1909. "Siphonaptera observed in the Plague Campaign in California," U. S. Public Health Service, *Pub. Health Rep.*, 24:1013–20.

9. Wu, Lien-Teh; Chun, J. W. H.; Pollitzer, R.; and Wu, C. Y.; 1936. *Plague, a Manual for Medical and Public Health Workers.* Shanghai: Weishengshu National Quar. Service. xxxiii + 547 pp.

10. Hampton, Brock C., 1940. "Plague in the United States," U. S. Public Health Service, *Pub. Health Rep.*, 55:1143–58.

11. Yersin, 1894. "La peste bubonique a Hong-Kong." *Ann. Inst. Pasteur,* 8:862–67.

12. Fox, Carroll, 1925. *Insects and Disease of Man.* Philadelphia: P. Blakiston's Son & Co. xii + 349 pp.

13. Ogata, M., 1897. "Ueber die Pestepidemie in Formosa," *Centralbl. f. Bakt.*, 21:769–77.

14. Simond, P. L., 1898. "La propagation de la peste," *Ann. Inst. Pasteur,* 12:625–87.

15. Verjbitski, D. T., 1908. "The part played by insects in the epidemiology of plague," *J. Hyg.*, 8:162.

16. Liston, W. G., 1905. "Plague rats and fleas," *J. Bombay Nat. Hist. Soc.*, 16:253–73.

17. Blue, Surgeon Rupert, 1910. *Rodents in Relation to the Transmission of Bubonic Plague. The Rat and Its Relation to the Public Health.* Washington, D. C.: U. S. Public Health and Marine Hospital Service. 254 pp.

18. Bacot, A. W., and Martin, C. J., 1914. "Observations on the mechanism of the transmission of plague by fleas," *J. Hyg.*, 13 (Plague Suppl. 3): 423–39.

19. Swellengrebel, N. H., and Otten, L., 1914. "Experimentelle Beiträge zur Kenntnis der Uebertragung der Pest durch Flöhe und Läuse," *Centralbl. f. Bakt.* 74:592–603.

20. Bacot, A. W., 1915. "Further notes on the mechanism of the transmission of plague by fleas," *J. Hyg.*, 14 (Plague Suppl. 4): 774–76.

21. Eskey, C. R., and Haas, V. H., 1940. *Plague in the Western Part of the United States.* Washington, D. C.: Govt. Print. Office, in Pub. Health Bull., no. 254. 83 pp.

22. Eskey, C. R., 1938. "Recent developments in our knowledge of plague transmission," U. S. Pub. Health Service, *Pub. Health Rep.*, 53:49–57.

23. McCoy, George W., 1910. "Bubonic plague in ground squirrels," *N. Y. Med. J.*, 92:652–65.

24. Roberts, Austin, 1935. "Mammals concerned in the bubonic plague and rabies problems in South Africa," *South African J. M. Sc.*, 22:414–60.

25. Burroughs, Albert Lawrence, 1947. "Sylvatic plague studies—the vector efficiency of nine species of fleas compared with *Xenopsylla cheopis*," *J. Hyg.*, 45:371–96.

26. Meyer, K. F., 1942. "The ecology of plague," *Medicine*, 21:143–74.

27. ———, 1947. "The prevention of plague in the light of newer knowledge," *Ann. New York Acad. Sc.*, 48 (art. 6): 429–67.

28. Dunn, L. H., and Parker, R. R., 1923. "Fleas found on wild animals in the Bitterroot Valley, Montana," U. S. Public Health Service, *Pub. Health Rep.*, 38:2763–75.

29. Pearse, A. S., 1928. "Fleas found on rodents and insectivores in Nigeria," *Bull. Entomolog. Research*, 19:167–68.

30. Meyer, K. F., 1938. "The rôle of the infected and the infective flea in the spread of sylvatic plague," *Am. J. Pub. Health*, 28:1153–64.

31. Wheeler, C. M., and Douglas, J. R., 1941. "Transmission studies of sylvatic plague," *Proc. Soc. Exp. Biol. & Med.*, 47:65–66.

32. Mooser, H.; Castañeda, M. R.; and Zinsser, H.; 1931. "Rats as carriers of Mexican typhus fever," *J.A.M.A.*, 97:231–32.

33. Dyer, R. E.; Ceder, E. T.; Workman, W. G.; Rumreich, A.; and Badger, L. F.; 1932. "Transmission of endemic typhus by rubbing either crushed infected fleas or infected flea feces into wounds," U. S. Public Health Service, *Pub. Health Rep.*, 47:131–33.

34. Alicata, Joseph E., 1942. "Experimental transmission of endemic typhus fever by the sticktight flea, *Echidnophaga gallinacea*," *J. Washington Acad. Sc.*, 32:57–60.

35. Zinsser, Hans, 1937. "The Rickettsia diseases, varieties, epidemiology, and geographical distribution," *Am. J. Hyg.*, 25:430–63.

36. Castañeda, M. Ruiz, 1948. "Flea-borne and louse-borne typhus in

Mexico." *Proc. 4th Internat. Cong. Trop. Med. & Malaria,* Washington, D. C., 1:408–13.

37. Beck, M. Dorothy, and van Allen, Alwine, 1948. "Typhus fever in California, 1916–1945, inclusive, an epidemiologic and field laboratory study," *Am. J. Hyg.*, 45:335–54.

38. Ewing, H. E., and Fox, Irving, 1943. *The Fleas of North America; Classification, Identification, and Geographic Distribution of these Injurious and Disease-spreading Insects.* Washington, D. C.: Dept. of Agric., in Misc. Publ., No. 500. 142 pp. (91 references, 13 figs.).

39. Jellison, Wm. L., and Good, Newell E., 1942. *Index to the Literature of Siphonaptera of North America.* Washington, D. C.: U. S. Public Health Service, in Nat. Inst. Health Bull., no. 178. 193 pp.

40. Mellanby, Kenneth, 1933. "The influence of temperature and humidity on the pupation of *Xenopsylla cheopis,*" *Bull. Entomolog. Research,* 24:197–202.

41. Jordan, Karl, 1932. "A new *Xenopsylla* from Hawaii," *Novitates Zoölogicae,* 38:264–66.

42. Williams, C. L., 1929. "A rat and rat-flea survey of ships at the port of New York," U. S. Public Health Service, *Pub. Health Rep.*, 44:443–76.

43. Hasseltine, H. E., 1929. "Rat-flea survey of the port of Norfolk, Va.," U. S. Public Health Service, *Pub. Health Rep.*, 44:579–89.

44. Fox, Carroll, and Sullivan, E. C., 1925. "A comparative study of rat-flea data for several seaports of the United States," U. S. Public Health Service, *Pub. Health Rep.*, 40:1909–34.

45. Williams, C. L., 1932. "Rat infestation inspection of vessels," U. S. Public Health Service, *Pub. Health Rep.*, 47:765–800.

46. Faust, E. C., and Maxwell, J. A., 1930. "The finding of the larvae of the chigoe, *Tunga penetrans,* in scrapings from human skin," *Arch. Dermat. & Syph.*, 22:94–97.

47. Parman, D. C., 1923. "Biological notes on the hen flea, *Echidnophaga gallinacea,*" *J. Agric. Research,* 23:1007–9.

48. Stewart, M. A., 1927. "A means of control of the European hen flea, *Ceratophyllus gallinae* Schrank," *J. Econ. Entomol.*, 20:132–34.

49. Lindquist, A. W.; Madden, A. H.; and Watts, C. N.; 1944. "The use of repellents against fleas," *J. Econ. Entomol.*, 37:485–86.

50. Doty, R. E., 1945. "Rat control on Hawaiian sugar cane plantations," *Hawaiian Planters' Record,* vol. 49, no. 2, 239 pp.

51. Storer, Tracy I., 1948. *Control of Rats and Mice,* Berkeley: Univ. Calif., in Agric. Exten. Serv. Circ., no. 142. 37 pp.

52. Silver, James; Crouch, W. E.; and Betts, M. C.; 1942. *Rat-proofing Buildings and Premises.* U. S. Fish and Wild Life Service Washington, D. C.: Conservation Bull., no. 19. 26 pp.

53. Holsendorf, B. E., and Clark, P. W., 1937. "The rat and rat-proof construction of buildings," U. S. Public Health Service, *Pub. Health Rep.*, Suppl. no. 131. 68 pp.

54. Ward, J. C., and Spencer, D. A., 1947. "Notes on the pharmacology of sodium fluoroacetate–Compound 1080," *J. Am. Pharm. A.*, 36:59–62.

55. Storer, Tracy I., 1947. *Control of Field Rodents in California,* in Calif. Agric. Ext. Circ., no. 138. 51 pp.

56. Simpson, F., 1911. "Ground squirrel eradication," Calif. State *Bd. of Health Bull.,* 6:507–12.

57. Long, John D., 1912. "A squirrel destructor," U. S. Pub. Health Service, *Pub. Health Rep.,* 27:1594–96 (4 plates).

58. Berry, C. E., 1938. "Methyl bromide as a rodenticide," Bull. *Dept. Agric.,* State of Calif., 27:172–80.

59. Stewart, M. A., and Mackie, D. B., 1938. "The control of sylvatic plague vectors," *Am. J. Hyg.,* 28:469–80.

60. Jellison, William L., 1939. "Sylvatic plague: Studies of predatory and scavenger birds in relation to its epidemiology," U. S. Public Health Service, *Pub. Health Rep.,* 54:792–98.

61. Wheeler, C. M.; Douglas, J. R.; and Evans, F. C.; 1941. "The rôle of the burrowing owl and the sticktight flea in the spread of plague," *Science,* 94:560–61.

CHAPTER XX

TICKS AND TICK-BORNE DISEASES

CLASS ARACHNIDA, ORDER ACARINA, SUPERFAMILY IXODOIDEA

Introduction. Probably all species of vertebrate animals higher than fishes are subject to attack by ticks, but particularly mammals, whose warm blood is highly attractive to these parasites. The food of ticks consists entirely of blood and lymph, and both males and females are bloodsuckers, as are all immature stages. Hunters have long observed tremendous infestations on the bodies of wild animals. Stockmen suffer enormous losses due to ticks on cattle, horses, and other stock; poultry is often severely parasitized. Hunter and Hooker,[1] United States Bureau of Entomology, reported that as many as two hundred pounds of blood may be withdrawn from a large host animal by ticks in a single season. Woodward and Turner,[2] using the common cattle tick, *Boöphilus annulatus* (Say), found that tick-infested cows under experimental conditions gave only 65.8 per cent as much milk as tick-free cows. Furthermore, the tick-free cows gained 6.1 per cent in body weight during the time of the experiment, while the tick-infested animals gained 3.6 per cent. Death due to exsanguination by ticks is believed to be possible. Jellison and Kohls[3] found that an adult female *Dermacentor andersoni* Stiles withdrew from 1.7 to 2 gm of blood in the act of engorgement, and they concluded that "tick-host anemia is not only an experimental disease but occurs with some frequency in nature, and may be the immediate cause of death in animals." During the months of October to March, 1935–36, one riding academy in Alameda County, California, lost 83 horses, whose deaths were reported to be due to loss of blood (exsanguination) through huge infestations of *Dermacentor albipictus* (Packard). Autopsies, blood examinations, and inoculation of blood into other horses produced no symptoms not attributable to simple secondary anemia.

There are many disorders and diseases of man and animals traceable to ticks, among these are (1) *dermatosis,* inflammation, itching, swelling, and ulcerations at the site of the bite, also skin ulceration and lesions resulting from improper or partial removal of tick mouth parts; (2) *envenomization,* inoculation of toxic salivary fluids at the site of the bite often resulting in severe systemic disturbances; (3) *exsanguination,* a serious matter when an animal is badly infested with ticks, resulting in a secondary anemia and possibly death; (4) *tick paralysis,* an acute ascending, flaccid type of paralysis, often fatal, caused directly by the bite of certain species of ticks, affecting children and young animals such as lambs, calves, colts, pigs, and dogs; (5) *otoacariasis,* invasion of the auditory canal by ticks; (6) *predisposition* to myiasis and secondary bacterial infections through tick-bite injuries. *Infections* transmitted by ticks may be classified in part as follows: *babesiosis, piroplasmosis, rickettsiosis,* and *spirochetosis;* they may also assist in the spread of *tularemia* and certain viral and helminthic infections.

High vector potential of ticks. Some of the factors which account for the potency of ticks in the spread of diseases of man and animals are the following: they are first of all *persistent bloodsuckers*—they *attach firmly* while feeding and cannot be dislodged easily; the later stages (nymphs and adults) are *highly chitinized,* hence very resistant to environmental stresses; they are relatively *free from natural enemies;* most species have a *wide host range,* thus ensuring a relatively certain source of blood; very few species of ticks are dependent upon a single host species; usually many species are available. Cooley reported 28 species of hosts for the adult of *Dermacentor andersoni* Stiles, and 32 for the immature stages. Other factors adding to the vector potential of ticks are: *longevity*—the life span of some species may be over five years; the *long duration of the life cycle* in many species, due to longevity of the several developmental stages, is an important factor in that, for example, the tick vector of relapsing fever may be a dangerous carrier of the infection for several years having received the infection through the egg from the previous generation; the *reproductive potential* of ticks is quite great—some species may deposit as many as 18,000 eggs; ticks apparently have the power to *regenerate lost parts* such as amputated legs, and the ability to repair mutilated mouth parts.

Man not a natural host for ticks. Ticks are in general parasites of animals in nature; they parasitize man and his domesticated mammals fortuitously. However, man has set the stage for enormous infestations of his herds of cattle, horses, and other animals. Certain tick-borne infections in wild animals have persisted for many centuries, and a host-parasite relationship has developed resulting in a benign (nonpathogenic) condition of infection rendering the wild animal a true reservoir. Thus rabbits (several species) harbor the rickettsiae of Rocky Mountain spotted fever, which infection is transmitted from rabbit to rabbit by a relatively host-specific tick. There is thus maintained in nature a continuous reservoir of infection; man is not an essential factor. Other species of ticks, more particularly *Dermacentor andersoni* Stiles, which bite other animals as well as man, pick up the infection from an infected rabbit, and if man intervenes (a fortuitous host) he will suffer a rickettsial infection often of great severity, probably because he is an *inappropriate* host. Spirochetal infections are described later in this chapter. No doubt, other infections not yet described will be revealed.

Diseases limited to animals, i.e. not transmissible to man may also possess similar tick-host relationships. Thus the causal agent of bovine anaplasmosis is harbored by wild deer without causing apparent symptoms, but when it is transmitted to domestic cattle by ticks a severe disease results.

Historical. Although ticks were referred to as "disgusting parasitic animals" by Aristotle in the fourth century B. C., the orderly classification of these parasites dates from the publications of Linnaeus in 1746 (Fauna Suecica). Linnaeus placed them among the Acari in the genus *Acarus* specifically, according to Nuttall (Monograph of the Ixodoidea, Cambridge University Press, May 1911). Pliny is said to have used the word *ricinus* to signify a "tick" because the fully engorged female resembles the seed of the castor-oil plant (*Ricinus*). The scientific nomenclature of ticks began with Latreille in 1804 when he set up eleven genera, among them *Ixodes;* therefore, the species of Linné became as we have it today, namely, *Ixodes ricinus* (Linnaeus). It was not until 1844 that C. L. Koch separated the ticks from the Acari, which included both ticks and mites. In 1896 Neumann placed the ticks in the order Acarina and divided them into two subfamilies, namely, (1) Argasinae and (2) Ixodinae. Finally, in 1901, Salmon and Stiles raised the two subfamilies of Neumann to family rank and placed them in the superfamily **Ixodoidea** established by Banks in 1894. Thus, systematic knowledge of the ticks grew slowly, and knowledge of their biology was exceedingly scant. Nevertheless, when Nuttall and associates published their bibliography on ticks in 1911 there were already 2,004 titles.

Theobald Smith (see p. 2) was the first (1893) experimentally to incriminate ticks as vectors of disease. He had previously (1889) discovered that Texas cattle fever was caused, like malaria, by a blood-corpuscle-invading protozoön, *Babesia bigemina.* Earlier discoveries pertaining to malaria were, no doubt, helpful. Smith and his associates, of course, also knew well the habits of the bloodsucking ticks, *Boöphilus annulatus* (Say) which abundantly parasitized the diseased as well as healthy cattle. They knew that this species is host-specific, hence needs but one host for its complete development. The astonishing fact was revealed by Theobald Smith that the etiological agent of Texas cattle fever was carried from the infected mother tick to her offspring (generation to generation) through the eggs. This discovery gave the answer to the puzzling question as to how this tick could infect other cattle when it was well known that once it had dropped off its host (the female deposits her eggs on the ground and then dies) it never again resumes its parasitic position on another host animal. Transovarian (generation to generation) transmission explained this readily—the newly hatched ticks were already infected, hence infectious, when feeding began.

In 1903 Marchoux and Salimbeni proved fowl spirochetosis, caused by *Borrelia anserina* (= *Spirochaeta gallinarum* Blanchard), to be tick-borne. They proved the common fowl tick, *Argas persicus* (Oken), to be a vector. This was apparently the first demonstration that a tick can transmit spirochetosis. In the meantime, the puzzling so-called "tick-bite" fever of man in the African Congo was nearing a solution. This fever was long associated with the bite of certain ticks by the natives. David Livingstone, the explorer, in 1857, was the first to report upon the evil effects following the bite of a tick. Murray in 1877 named this tick, *Ornithodoros moubata*. It was not until 1904 that Ross and Milne, Dutton and Todd in 1905 (reporting from Uganda and the Congo), and simultaneously Robert Koch reporting from (German) East Africa proved that this tick, *Ornithodoros moubata* (Murray), was a vector of the causal organism, a spirochete; the "tick-bite fever" of the natives, therefore, proved to be a tick-borne relapsing fever. Generation-to-generation transmission through the egg was also demonstrated. These were epoch-making discoveries and quickened the interest of workers in the fields of parasitology and epidemiology everywhere. In the brief period of a dozen years both families of ticks, the **Ixodidae**, hard ticks, and the **Argasidae**, soft ticks, had been incriminated as disease vectors involving both man and his domesticated animals.

Characteristics of ticks. With the few wingless insectan forms well in mind, such as the sac-like Pupipara, particularly the sheep ked, ticks are easily distinguished from insects, in that the body is not definitely divided; i.e., there is a strong fusion of the thorax and abdomen producing a sac-like leathery appearance. A discrete head is lacking, but the mouth parts, together with the *basis capituli* in many species (**Ixodidae**) form a structure known as the capitulum. Like other Arachnida the mature ticks bear four pairs of legs; the larvae are hexapod.

All ticks bear a pair of spiracles, situated lateroventrally on the abdomen, one on each side near the third and fourth coxae. A pair of simple eyes may be present. Many species of ticks are eyeless.

Ticks vary considerably in size according to species but rarely exceed 15 mm in length in the fully engorged females. The females are capable of great distention, and when fully engorged are seed-like in form.

Tick mouth parts and feeding habits. The capitulum (head) bears the mouth parts and accessory external structures. (Fig. 30, Chapter VI.) The basal portion is known as the *basis capituli,* from which projects a pair of protrusible *chelicerae.* The distal portions (*digits*) of the chelicerae are divergent and are provided with recurved teeth. Projecting forward and situated ventrally and medianly on the *basis capituli* is the *hypostome* bearing many recurved teeth. Laterally are located the *palpi* (one pair), consisting of four articles, of which two or more may be used; commonly three are visible.

When sucking blood both the hypostome and the chelicerae are inserted into the skin of the host. The impression that the mouth parts are formed like a corkscrew and may be removed by "unscrewing" is, of course, erroneous. By "unscrewing" one is more likely to leave the mouth parts in the flesh. Because of the recurved teeth the tick is enabled to hold so fast to the host that it is difficult to remove it without tearing the capitulum from the body of the tick. The tick itself, however, withdraws its mouth parts quickly and apparently with little effort by slipping the hood-like portions of the capitulum over the relaxed mouth parts and, with a quick jerk, drops off and escapes.

One does not usually feel the tick when it is biting. As Cooley[4] has so well said, "a person is always completely surprised when he finds a tick attached." The best way to remove a tick, and this should be done without delay, is to take hold of it with the fingers and pull it off slowly with a firm straight pull without jerking.

The length of time that a tick remains attached in the act of uninterrupted feeding depends on the species and the stage of development. The seed ticks commonly feed for a number of days; the nymphs and adults differ greatly in this respect, thus the common poultry tick, *Argas persicus* (Oken) feeds nightly and intermittently, while the nymphs and adults of the southern cattle tick, *Boöphilus annulatus* (Say) feed from six to eight days before becoming fully engorged. Other species of ticks, notably the California relapsing fever tick, *Ornithodoros hermsi* Wheeler, is able to engorge fully in from 15 to 20 minutes. Both male and female ticks suck blood; however, in the case of the **Ixodidae**, only the females become greatly distended when engorged, while both sexes become distended in the **Argasidae.**

Life history. Under natural conditions a few species of ticks show a marked host specificity, e.g., *Boöphilus annulatus* (Say), the southern cattle tick, and *D. parumapertus* Neum. on jackrabbits. However, most species have a fairly wide range of hosts, e.g., *Dermacentor occidentalis* Neum. The life histories of ticks vary considerably for the several species hence it is quite impossible to generalize, except that it may be said that all species of ticks pass through four stages (*egg, seed tick, nymph,* and *adult*) in from six weeks to two years, e.g., the Rocky Mountain spotted fever tick, *Dermacentor andersoni* Stiles, requires normally about two years to complete its life history. The fully engorged female ticks usually deposit their eggs on the ground, the number varying from 100 in some species to 18,000 in others. The newly hatched larvae, known as seed ticks, are hexapod (six-legged) and remain in this condition until the first molt is completed. The nymph emerges from the first molt with its fourth pair of legs present, and remains in this stage until the second molt, from which the adult tick emerges. As many as seven molts may occur in certain Argasine ticks. Copulation takes place after the last molt, when the females engorge and then deposit eggs. In the majority of species the ticks drop off the host animal to molt, but in several species, notably the Texas cattle fever tick *Boöphilus annulatus* (Say), the molting takes place on the host. There may be two or possibly three generations of ticks in one year under very favorable climatic conditions in species which molt on the host.

The seed ticks emerging from the eggs on the ground commonly climb up grasses and other low vegetation in order to come within easy reach of grazing or passing animals. The nymphs and adults employ the same method. Wild lilac (*Ceanothus*) is a favorable shrub for the purpose, in fact in some localities in California it is known as a "tick bush."

The larvae having reached the body of a host, there follows a sequence of feeding and molting until maturity is reached. When this sequence is completed on one animal, as for example, in the case of the cattle fever tick, *Boöphilus annulatus* (Say), the species is said to be a one-host tick. When the sequence is completed on two host animals, as with the African red tick, *Rhipicephalus evertsi* Neum., the species is said to be a two-host tick. The larvae of this two-host species hatch on the ground like other ticks, then proceed to attach themselves to the ears (often inside) and flanks of cattle, where they become fully engorged and molt while on the host. The nymphs then engorge and drop off to molt, after which the adult tick emerges and must now find a second host upon which it engorges itself and then drops to the ground, where the females lay eggs.

When the tick species requires three different hosts to complete its cycle it is called a three-host tick, as for example, *Dermacentor andersoni* Stiles, the Rocky Mountain spotted fever tick. In this species the larvae select smaller animals such as ground squirrels upon which engorgement is achieved after which the larvae drop to the ground to molt and reach the nymphal stage. In this stage the second host is usually a larger animal such as a rabbit or coyote. After engorgement on this host the nymph drops to the ground and with its last molt it reaches the adult stage and once more finds a host animal (the third host) upon which it feeds and thereafter drops to the ground, where the females lay their eggs.

In such species of ticks as *Ornithodoros hermsi* Wheeler, the vector of relapsing fever in California, several individual host animals are required; such species are known as many-host ticks. There are usually five molts in this species, each of which is completed off the host, hence at least five host animals are needed to complete the cycle.

Longevity. The longevity and hardiness of ticks is something truly remarkable, a matter not to be overlooked in applying control measures. Furthermore, chemicals which kill most insects in a few minutes act very slowly on these arachnids.

Unfed larval ticks of the species, *Argas persicus* (Oken), remain alive quite readily for more than a month; nymphs survive a longer time and the adults even longer than the nymphs. Nuttall and Warburton[5] cite cases in which nymphs of this species survived two months, and adults (unfed) "a little over two years." Graybill[6] reports considerable variation in the longevity of the Texas fever tick, depending on the season of the year; unfed larvae survived from 7 to 85 days (average 38.6) for July, and 30 to 234 days (average 167.4) for October. Nuttall and Warburton (*loc. cit.*) mention cases in which the unfed larvae of *Ixodes ricinus* (Linn.) survived 19 months, unfed nymphs 18 months, and unfed adults 15 to 27 months. Unfed adult *Dermacentor andersoni* Stiles survived 413 days. The author had a female *Ornithodoros megnini* Dugès remain alive without food in a pill box for two years and seven months. A specimen of *O. coriaceus* Koch remained alive similarly over six years with an average of two blood meals annually.

Classification. The superfamily **Ixodoidea,** which includes well over 450 species of ticks, is divided into two families, namely: (1) **Ixodidae,** also known as the hard-bodied ticks, comprises scutate ticks with a terminal capitulum; sexual dimorphism is marked; the males have a scutum which almost covers the dorsum and are incapable of great distention; in the females the scutum is a small shield immediately behind the capitulum; the females are capable of enormous distention. (2) **Argasidae,** also known as the soft-bodied ticks, includes ticks without a scutum (nonscutate); sexual dimorphism is not marked; the capitulum is ventral and the palpi are leg-like; the eyes when present are lateral and situated on the supracoxal folds; the spiracles are very small.

The two families may be readily separated by means of the following table (Table XII), and Figure 151.

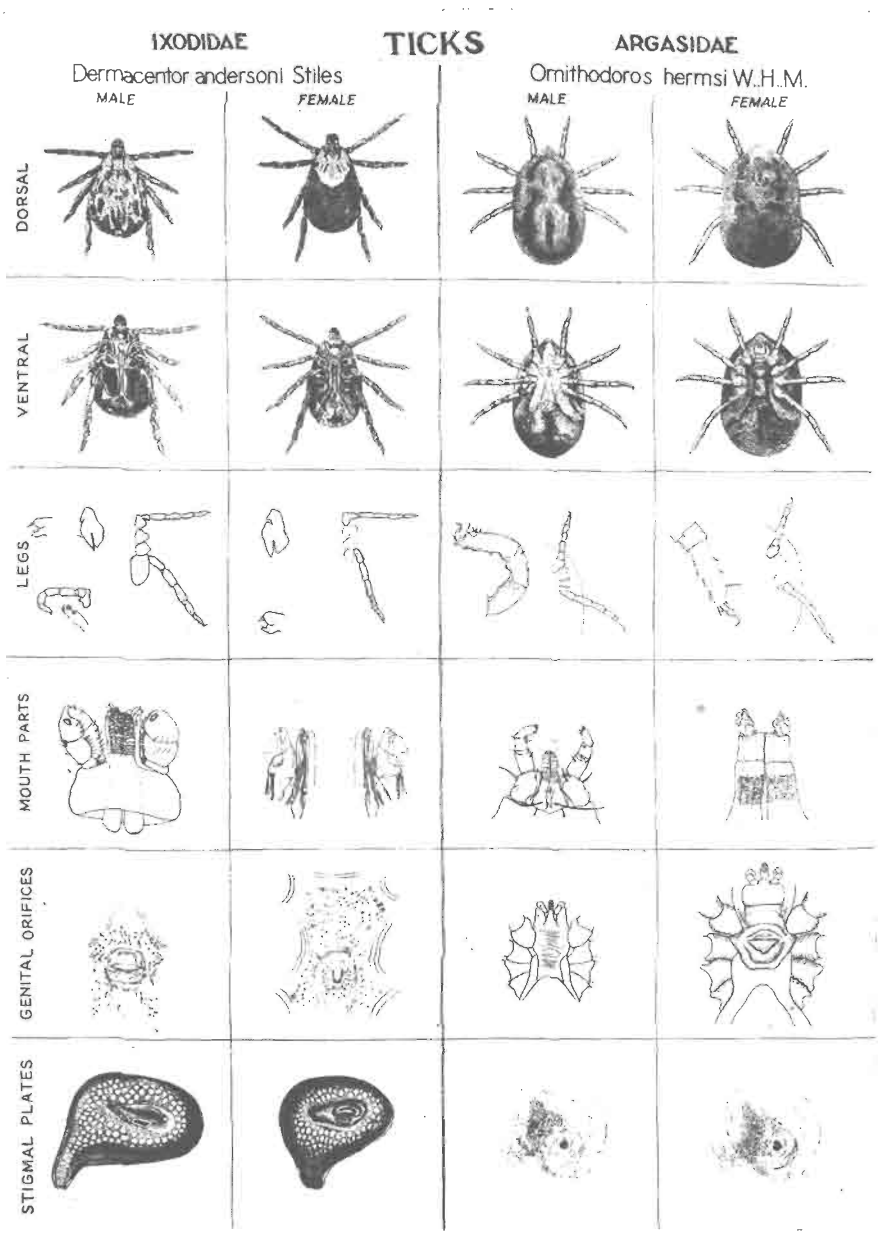


FIG. 151. Structural details of the two families of ticks, Ixodidae and Argasidae.

TABLE XII

DIFFERENCES BY WHICH THE TWO FAMILIES OF THE IXODOIDEA MAY BE SEPARATED (ADAPTED AFTER NUTTALL)

	Argasidae	Ixodidae
Sexual dimorphism	Slight	Marked
Capitulum	Ventral	Anterior
Base	No porose areas	Porose areas in—
Palpi	Leg-like, with subequal articles	Relatively rigid, of very varied form
Body		
Scutum	Absent	Present
Festoons	Absent	Generally present
Eyes (when present)	Lateral on supracoxal folds	Dorsal on the sides of the scutum
Legs		
Coxae	Unarmed	Generally armed with spurs
Tarsi	Without ventral spurs	Generally armed with 1 or 2 ventral spurs
Pulvilli	Absent or rudimentary	Always present

FAMILY IXODIDAE (HARD-BODIED TICKS)

The family Ixodidae. Nuttall and Warburton (1911, *loc. cit.*) include nine genera* in the family Ixodidae; namely, *Ixodes, Haemaphysalis, Dermacentor, Rhipicentor, Rhipicephalus, Margaropus, Boöphilus, Hyaloma,* and *Amblyomma,* divided into five groups as follows (see also *Fig. 152 showing typical capituli*):

Ixodidae
- Prostriata
  - 1 Ixodes
- Metastriata
  - Brevirostrata
    - Group 1
      - 2 Haemaphysalis
    - Group 2
      - 3 Dermacentor
      - 4 Rhipicentor
      - 5 Rhipicephalus
      - 6 Margaropus
      - 7 Boöphilus
  - Longirostrata
    - Group 1
      - 8 Hyalomma
    - Group 2
      - 9 Amblyomma

Genus Ixodes. This genus, which comprises about 60 species, is clearly separated from all other genera of the family **Ixodidae** by the

* Colley, 1938, added a new genus, *Otocentor,* which becomes a monotypic genus with *Dermacentor nitens* Neumann the genotype. The horse and related animals are evidently the favored host.

anal groove surrounding the anus in front (Prostriata) and the absence of festoons. The remaining genera fall naturally into two divisions (see diagram), the one characterized by a comparatively short capitulum, and the other by a comparatively long one. Eyes are absent; spiracles are round or oval; palpi and *basis capituli* are of variable form; coxae either unarmed, trenchant, spurred, or bifid; tarsi without spurs; sexual dimor-

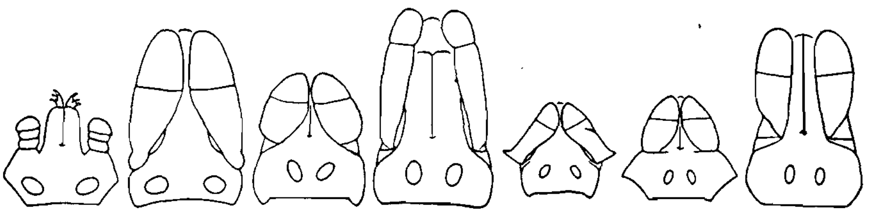


FIG. 152. Characteristic capituli of the several genera of ticks belonging to the family *Ixodidae:* (*A*) *Boöphilus,* (*B*) *Ixodes,* (*C*) *Dermacentor,* (*D*) *Amblyomma,* (*E*) *Haemaphysalis,* (*F*) *Rhipicephalus,* and (G) *Hyalomma.* (Adapted after Cooley.)

phism is pronounced, especially with regard to the capitulum; in the male the venter is covered by nonsalient plates; one pregenital, one median, one anal, two adanal, and two epimeral plates; e.g., *Ixodes ricinus* (Linn.), commonly known as the European castor bean tick. It is very widely distributed and feeds on a wide variety of hosts. The species widely referred to as *Ixodes californicus* Banks (Fig. 153) is now

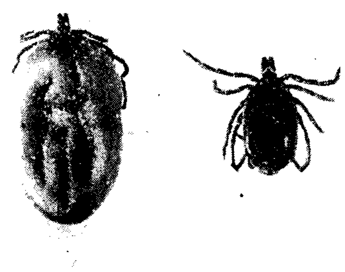


FIG. 153. A common deer and cattle tick of the Pacific coast, *Ixodes pacificus;* (*left*) female, (*right*) male. ×3.5.

described as *Ixodes pacificus* Cooley and Kohls[8] (1943). This is a common deer tick in California but flourishes on cattle as well. It bites human beings freely and often causes severe disturbances. Among the ticks referred to as bird ticks is *Ixodes howelli* Cooley and Kohls (1938) taken from the Sierra Nevada rosy finch, *Leucosticte tephrocotis dawsoni* Grinnell, at an elevation of 12,000 feet.

Genus Dermacentor. Usually they are ornate ticks with eyes; festoons 11 in number; palpi short, broad or moderate; *basis capituli* rectangular dorsally. In some species coxae I to IV of the male increase progressively in size; in all species coxa IV is much the largest; the male shows no ventral plates or shields. Coxa I is bifid in both sexes. The spiracles are suboval or comma-shaped. The genus includes about 20 species. Students concerned with species of the genus *Dermacentor* must consult the publications of R. A. Cooley (e.g., 1938, *loc. cit.*).

Dermacentor variabilis (Say) is the principal, if not the only, vector of Rocky Mountain spotted fever in the central and eastern portions of the United States. It is also an important vector of tularemia and bovine anaplasmosis. It may cause canine paralysis and is a common pest of dogs, which are the preferred host of adult ticks of this species. It also freely attacks horses and many other animals, including man. The immature stages feed almost exclusively on small rodents, very largely on meadow mice, [*Microtus pennsylvanicus pennsylvanicus* (Ord) in the Atlantic states]. It is a widely distributed North American species. It is commonly referred to as the "American dog tick" (Smith, Cole, and Gouck[9]).

The fully engorged females drop from the host and in four to ten days lay eggs (4,000 to 6,500) on the ground. The incubation period during the summer months is about 35 days; however, temperature influences this stage greatly. The larvae remain in clumps on the ground or on low-growing vegetation while awaiting a host, usually a field mouse. The period of larval engorgement varies from 3 to 12 days, after which the larvae drop to the ground and molt in about a week, though often greatly prolonged. The nymphs, having reached a host, again usually a field mouse, engorge in from 3 to 11 days, and once more drop to the ground and molt in from three weeks to several months. The unfed adults may live for more than two years; however, having reached the adult stage the ticks usually attack dogs and other large host animals including man. Engorgement of the females requires from 5 to 13 days. Mating takes place on the host. Like the unengorged adults the immature stages have a remarkable longevity in the absence of suitable hosts, which may prolong the life history two or more years, although under favorable conditions the life cycle from egg to adult may not require more than three months.

Dermacentor andersoni Stiles, the Rocky Mountain wood tick (Fig. 154) is a widely distributed and very common species throughout the Rocky Mountain region of the United States including Idaho, Wyoming, Montana, parts of Utah, Colorado, Nevada, Oregon, Washington, California, and British Columbia. "It is most abundant in regions or localities where the predominating vegetation is low, brushy, and more or less open, i.e., in areas where there is good protection for the small mammalian hosts of the larvae and nymphs and sufficient forage to attract the large hosts (either wild or domestic) of the adult ticks. It is relatively scarce in heavily timbered areas or country of a strictly grassland, prairie type" (Parker *et al.*[10])

This species may be recognized by comparison with Fig. 154. The adult ticks feed mostly on large animals, such as horses, cattle, and sheep, and also on deer, bear, and coyote; the larvae prefer to feed on rodents such as ground squirrels, pine squirrels, woodchucks, and chipmunks; the nymphs feed on the same host species as the larvae; all three stages may feed on animals of intermediate size such as jack rabbits, marmots, and badgers. In the wilder parts of its range where there are no domesticated animals, the adult ticks feed on deer, elk, bear, mountain goat, and coyote.

The life cycle of the Rocky Mountain wood tick is fully described by Cooley,[11] and from his description the following summary is largely

FIG. 154. The Rocky Mountain spotted fever tick, *Dermacentor andersoni*; (*left*) male, (*right*) unengorged female. ×3.5.

taken. Copulation takes place on the host, and when fully fed, the greatly distended female drops to the ground. The preoviposition period is about a week, after which egg-laying begins, continuing over a period of about three weeks. If undisturbed, the eggs pile up ahead of the female in one large mass, averaging some 6,400 eggs. The incubation period of the eggs requires about 35 days, when the young hexapod seed ticks emerge and find suitable rodent hosts, feed for three to five days, drop off, and molt in from 6 to 21 days, emerging as nymphs with four pairs of legs. These nymphs, the progeny of overwintered adults, go into hibernation to come up for feeding the next spring when they seek larger-sized hosts as already explained, to which they attach for feeding over a period of from four to nine days. When fully engorged, the nymphs drop to the ground and in 12 to 16 days or more, molt for the second time, emerging as adults. Some of these adults may find hosts in the same summer in which they have emerged as adults, but by the time they have emerged, Cooley explains, the season has generally become hot and dry, making it necessary for them to seek protection under waste and vegetation. The "normal cycle" is therefore two years. The larvae are found feeding through the summer months, and while the adults disappear by about July 1, the nymphs continue in diminishing numbers until late summer. Since man is usually bitten only by the adult ticks, danger from this source exists only from early spring to about July 1.

Like other species of ticks, *Dermacentor andersoni* Stiles is remarkably resistant to starvation. Hunter and Bishopp[12] report that all unfed seed ticks hatching from a mass of eggs usually die within one month after the first eggs hatch if food is not available. However, in one instance a period of 117 days elapsed between the beginning of hatching of the eggs and the death of the last seed tick. (A later record noted by these investigators exceeded 317 days.) Unfed nymphs have been found to survive one year and 11 days, and adults collected on vegetation during the spring months may survive for 413 days without food. Students concerned with the anatomy of this important species of tick will need to consult the work of Douglas[13] (1943).

Dermacentor albipictus (Packard) is commonly known as the winter tick, elk tick, or horse tick. It is a widely distributed North American species. It is a one-host tick and does not occur on the host during the summer months, it is distinctly a winter tick. The eggs are laid during the spring months and hatch in from three to six weeks. The seed ticks then bunch tightly together, remaining in a torpid condition until the first cold weather in autumn, when they become very active and seek host animals. Molting takes place on the original host animal. The females reach maturity with the final molt and engorgement usually in about six weeks after the seed ticks have become attached. Although the females drop off the host after final engorgement as do other ticks, egg laying is delayed until spring, often after an interval of several months.

Hearle (1938, *loc. cit.*, p. 399) states that heavy infestations of horses, cattle, moose, elk, and deer may result in death from "tick poverty" due to the drain on the vitality of the infested hosts. In this respect, Hearle states, it is the most important species occurring in Canada. This tick, it is reported, caused a loss of at least 20 per cent of the moose population in Nova Scotia. A disease of moose (*Alces americana americana* Jardine) is described by Thomas and Cahn[14] as occurring in northeastern Minnesota and the adjacent region. It is reported that guinea pigs and rabbits infested with the tick, *Dermacentor albipictus* (Packard), from diseased moose have reproduced in detail the symptoms of weakness, anemia, paralysis, excessive blind activity, and death as exhibited by infected moose. The causal bacterium is described as *Klebsiella paralytica* "because of the paralysis it causes" (Cahn, Wallace, and Thomas[15]).

Dermacentor occidentalis Marx has a narrow Pacific coast distribution, being particularly abundant in southwestern Oregon, rare northward and southerly. Adults of this species have been taken from cow, horse, mule, ass, deer, rabbit, sheep, dog, man, and the immature stages from many species of smaller animals such as ground squirrel, rabbit, skunk, and field mouse. *Dermacentor andersoni* and *D. occidentalis,* though distinct species, are very closely related and have been hybridized.

Key to the adults of the genus Demacentor
in the United States (after Cooley)

1. Spurs on coxa I widely divergent 2
   Spurs on coxal I with proximal edges parallel or only a little divergent 3
2. Scuta with deep large punctations .... *parumapertus* Neum.
   Scuta with the larger punctations shallow and moderate in size (known from peccary in southern Texas) .... *halli* McIntosh
3. Spiracular plate oval, without dorsal prolongation and with goblets few and large .... *albipictus* (Packard)
   Spiracular plates oval, with dorsal prolongations, and with goblets many or of moderate numbers 4
4. Cornua* long .... *occidentalis* Marx
   Cornua short or of moderate length 5
5. Spiracular plate with goblets very numerous and small .... *variabilis* (Say)
   Spiracular plate with goblets moderate in size and number 6
6. The larger punctations of the scuta very large and deep .... *andersoni* Stiles
   The larger punctations on the scuta moderate in size and depth (known from Rocky Mountain sheep in southern Arizona) .... *hunteri* Bishopp

Genus Haemaphysalis. The members of this genus, numbering about 45 species, are usually small and but slightly chitinized, and the sexes are very similar. They are inornate and eyeless, but have festoons; the second segment of the usually short conical palpi projects laterally beyond the *basis capituli,* forming an acute angle. The spiracles of the males are ovoid or comma-shaped; in the female rounder or ovoid. The genus is commonly found on mammals but may severely parasitize ground-nesting birds.

Haemaphysalis leporis-palustris (Packard) is a widely distributed North and Central American species. Although it is commonly known as the rabbit tick, it has been taken on a number of species of birds and occasionally on domestic animals such as the horse, cat, and dog. It

* Cornua: caudad projection extending from the lateral posterior dorsal angles of the basis capituli.

rarely bites man but is important in the spread of Rocky Mountain spotted fever and tularemia among wild animal reservoir hosts.

Haemaphysalis leachi (Audouin), the dog tick of Africa, is common in Asia, Australia, and Africa. It is usually found on wild and domestic carnivores, frequently on small rodents and rarely on cattle. This tick is a vector of malignant jaundice in dogs.

Genus Rhipicentor. This small and relatively unimportant genus of ticks is inornate, with eyes and festoons present; the palpi are short, with *basis capituli* hexagonal dorsally and having very prominent lateral angles. Coxa I is bifid in both sexes; coxa IV is much the largest; there are no ventral plates or shields; spiracles subtriangular in the female or comma-shaped in the male. The genus is represented by *Rhipicentor bicornis* Nuttall and Warburton.

Genus Rhipicephalus. This genus comprises about 30 species. They are usually inornate and possess eyes and festoons; the palpi are short and the *basis capituli* is usually hexagonal dorsally. Coxa I is bifid. The male possesses a pair of adanal shields and usually a pair of accessory glands; some males when replete show a caudal protrusion. The spiracles are bluntly or elongatedly comma-shaped.

Rhipicephalus sanguineus (Latr.) is the cosmopolitan brown dog tick; its principal host is undoubtedly the dog, although it is known to attack numerous other animals. It is a widely distributed tick occurring primarily in tropical and subtropical climates and is widely known as a vector of malignant jaundice of dogs. The adult ticks are most often found in the ears and between the toes of dogs, and the larvae and nymphs in the long hair at the back of the neck. McIntosh[16] states that the eggs are deposited in cracks and crevices of the kennel or other quarters frequented by the dog. The ticks have a strong tendency to crawl upward, hence are often found hidden in cracks in the roofs of kennels or in the ceilings of porches. It may become a very great nuisance. The eggs hatch in from 20 to 30 days and over, depending upon temperature. The life cycle corresponds to that of other species of Ixodine three-host ticks.

Genus Margaropus. This genus has an obsolete anal groove, and no ornamentations or festoons; the palpi are short and the capitulum is highly chitinized and similar in shape to that of *Boöphilus.* The coxae are conical and unarmed except for a small spine posteriorly on coxa I. The male has a median plate prolonged into two long spines projecting beyond the anus on both sides; when replete it shows a caudal protrusion. It may be separated from *Boöphilus,* which it closely resembles, by the presence of greatly thickened posterior legs and by the prolonged median plate.

Margaropus winthemi Korsch, the Argentine tick, is a native of South

America introduced into South Africa; it is frequently found on horses and occasionally on cattle. When engorged the females may easily be mistaken for *B. decoloratus* (Koch) but may be distinguished by the dark bands at the joints of the legs. This is a one-host tick, usually present in larger numbers during the winter.

Genus Hyalomma. Ornamentations are absent or present and may be at times confined to the legs; eyes are present, and there may or may not be festoons. The palpi are long and the *basis capituli* is subtriangular dorsally. The female approaches *Amblyomma*. The male has a pair of adanal shields, with or without accessory adanal shields and two posterior abdominal protrusions capped by chitinized points. Coxa I is bifid. Spiracles are comma-shaped. The genus includes less than half a dozen species.

Hyalomma aegyptium (Linn.). The "bont-leg" tick, with its two varieties, is fairly widespread over much of southern Europe, Asia, and

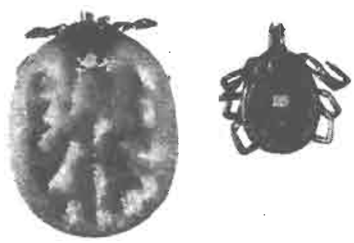


FIG. 155. The lone star tick, *Amblyomma americanum*. ×3.5.

Africa. The adults are parasitic on cattle, horses, sheep, goats, wild mammals, and occasionally birds, while the larvae and nymphs are found on rodents, hares, and birds, frequently infesting domestic poultry. It is usually a two-hosted tick, the larvae and nymphs feeding on the same host, though the larvae may drop off and seek a new host.

This tick is very hardy and exists under extremes of heat and cold. The adults attach by preference around the anus and genitalia and may produce severe lesions. Lameness is frequently encountered in sheep as a result of tick bite.

Genus Amblyomma. These are generally ornate ticks, with eyes and festoons; the palpi are long, article 2 especially so; the *basis capituli* is of variable form. The male is without adanal shields, but small ventral plaques are occasionally present close to the festoons. Spiracles are subtriangular or comma-shaped. There are about 90 species in this genus. *Amblyomma americanum* (Linn.) is the "lone star" tick (Fig. 155) of the southern United States, particularly Oklahoma, Texas, and Louisiana. Its range appears to extend considerably northward, and southerly into

Mexico, Central America, and South America (Brazil). This species has a wide variety of hosts, including wild and domestic animals, birds, and man.[17] It takes high rank as a pest and vector of disease, e.g., Rocky Mountain spotted fever, Bullis fever, and tularemia. It is a three-host tick.

Amblyomma cajennense (Fabr.) is reported as occurring in the southern United States, Central America, the West Indies, and South America. It commonly attacks man, horses, cattle, sheep, dogs, pigs, and many other animals. This species appears to be abundant in a few counties in Texas (Cooley and Kohls, *loc. cit.*).

Genus Boöphilus. Members of this genus have neither festoons nor ornamentations, but eyes are present. The palpi and hypostome are short, compressed, and dorsally and laterally ridged. The *basis capituli* is hexagonal dorsally and slightly chitinized. Coxa I is bifid; the anal groove is obsolete in the female and faintly indicated in the male. Unfed

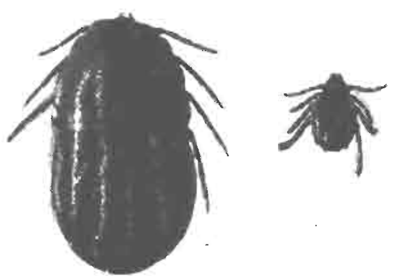


FIG. 156. The Texas fever tick, *Boöphilus annulatus;* (*left*) female, (*right*) male. ×3.5.

adults are small; the scutum of the female is quite small and the spiracles are circular or oval in both sexes. There are less than half a dozen species.

The Texas cattle fever tick, *Boöphilus annulatus* (Say) (*Margaropus annulatus* Say) (Fig. 156) is normally restricted to North America south of the 37° latitude into parts of Mexico. It is typically a cattle tick, although it occurs at times in smaller numbers on deer, sheep, horses, mules, and other animals. The bison is evidently also a suitable host. *Boöphilus annulatus microplus* (Can.) (= *australis* Fuller) is referred to as the tropical cattle tick (Tate[18]).

Fully engorged females range in length from 10 to 12 mm, while the male ranges from 3 to 4 mm. The body of the female is about equally rounded both posteriorly and anteriorly, with a slight median incurving. The anterior pair of legs is set well out on the shoulders away from the capitulum (in *Dermacentor* close to the capitulum). The palpi are short, and the capitulum is inconspicuous. The relatively small (about 1 mm long) scutum is solid chestnut brown in color.

Economic importance. Tremendous strides have been made in the last few years in the control of the Texas fever tick. It is of interest to note that as late as 1906 it was estimated that the annual losses to southern United States occasioned by the "cattle tick" directly and indirectly amounted to $130,500,000.[19] This sum included (1) death, from Texas fever, of pure-bred cattle imported from the North for breeding purposes; (2) death, from Texas fever, when cattle reared in isolated tick-free areas were unintentionally or accidentally placed with ticky cattle, or on tick-infested areas; (3) death of native cattle from excessive parasitism and fever, occasioned by the ticks; (4) universal loss of weight by all tick-infested cattle, and their failure to gain flesh at a rate great enough to make beef production profitable; (5) the lower price which "Southern" cattle brought upon the market, regardless of how perfect their condition; (6) sterility induced in high-grade cattle by tick infestation; (7) the expense of maintaining the Federal quarantine for the protection of the North against invasion by the tick, and the added expense of maintaining quarantine pens for southern cattle shipped North for slaughter; (8) the discouraging effect on the breeding of pure-bred cattle in the South by reason of southern breeders not being allowed to exhibit in northern show rings; (9) by no means least, the potential loss in fertility of southern farm lands due to a one-crop system which, with the tick eradicated, would quickly give way to a diversified agriculture thus tending to conserve and increase the fertility of soils; (10) shrinkage in milk production of tick-infested cattle.

From 1906, when cooperative tick-eradication work was undertaken between the United States Bureau of Animal Industry and state authorities, to December, 1918, a total of 458,529 square miles of territory was released from quarantine against Texas cattle fever, which speaks well for the methods employed.

Life history of Texas fever tick. *Boöphilus annulatus* (Say), is a one-host tick. The life history may be divided into two phases: (1) the *parasitic* phase during which the tick is attached to the host and which terminates when the mature tick drops to the ground after fertilization; (2) the *nonparasitic* phase when the tick is on the ground. After the mature female tick drops to the ground, there is a preoviposition period of three or four days to perhaps a month. Oviposition usually begins about 72 hours after the tick drops and continues usually for eight or nine days but may be greatly prolonged if the temperature is adverse.

The maximum number of eggs deposited by a female tick according to Graybill (*loc. cit.*) was 5,105, the minimum 357, with an average ranging from 1,811 to 4,089. The incubation period, also dependent on temperature, ranged from 19 days in summer to 180 days in the early autumn, with the average of 43.6 days for April, 26.3 days for May, 24.5 days for June, 20.5 days for July, 21.2 days for August, and 35.9 days for September. The hatching period depends on the time when the eggs are laid, the eggs first deposited ordinarily hatching first. The seed ticks (Fig. 157) on hatching are very active; they climb onto blades of grass or other objects which they ascend to the top, remaining clustered there until a suitable host animal brushes against them to which they can attach themselves. The time during which the seed ticks remain alive, i.e., longevity of the newly hatched ticks, again varies considerably, depending on temperature; the longevity for April ticks was found to be 65.1 days, May ticks 62.3 days, June ticks 65.1 days, July ticks 38.6 days, August ticks 84.9 days, October ticks 167.4 days. The total average ranged from 86.9 days for June to 279.6 for October.

The three stages considered in the parasitic period of the ticks are larval (seed tick), nymphal, and adult. As Graybill has well said, "The

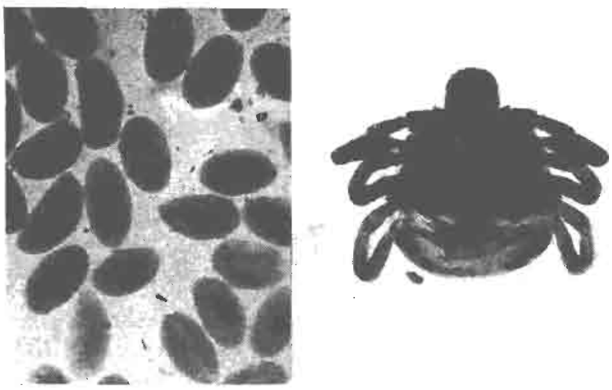


FIG. 157. (*Left*) eggs and (*right*) larva of the Texas fever tick, *Boöphilus annulatus*. ×50.

duration of each of these stages and the duration of a single infestation upon cattle during different portions of the year are of great practical importance. Upon the duration of an infestation depends the time animals must be kept on the tick-free fields in order to become free from ticks." After the seed tick has attached itself to the host, the larval period ranges from 5 to 10 days, the nymphal period from 5 to 20 days, and the life of the adult female from 5 to 35 days, with a total period of infestation, including the time for molting twice, which is accomplished on the host, from 20 to 65 days. The entire life cycle may be completed in about 40 days under most favorable conditions, usually nearer 60 days under natural conditions.

DISEASES CARRIED BY IXODINE TICKS

Texas cattle fever. Red water, splenic fever, bloody murrain, Mexican fever, tick fever, etc., are names given to a widely distributed disease of cattle, being endemic in southern Europe, Central and South America,

TABLE XIII

COMPARISON OF THE LIFE CYCLE OF A WOOD TICK AND THE TEXAS CATTLE FEVER TICK

Wood tick (*Dermacentor* sp.)	Texas cattle fever tick (*Boöphilus annulatus*)
I. Adult tick becomes engorged on host animal and drops to ground	Adult tick becomes engorged on host animal and drops to ground
II. Adult tick begins egg laying (3,000 ± eggs) after 3–5 days	Adult tick begins egg laying (3,000 ± eggs) after 3–5 days
III. Seed ticks hatch from eggs in about 30 days	Seed ticks hatch from eggs in about 30 days
IV. Seed ticks bunch on grass and await coming of host animal, from one day to several weeks	Seed ticks bunch on grass and await coming of host animal from one day to several weeks
V. After feeding 7–12 days seed ticks drop to ground and molt	After feeding 7–12 days seed ticks molt *on* host animal
VI. Nymphs crawl up on grass and await coming of second host animal from one day to several weeks	
VII. Ticks get on second host animal and feed 5–10 days, then *drop to ground* and molt second time	Nymphs feed 5–10 days, then molt second time *on* host animal and the newly emerged mature ticks mate
VIII. Mature unengorged ticks crawl up on grass and await coming of third host animal from one day to several weeks	
IX. Adult ticks mate and feed 5–8 days to engorgement, then drop to the ground and lay eggs	Adult ticks feed 4–14 days to engorgement, then drop to the ground and lay eggs

parts of Africa, Mexico, the Philippines, and the southern United States where it has been known for more than a century, having been introduced into this country probably from Europe. The causal organism is *Babesia begemina* (Smith and Kilbourne).

The name Texas fever became attached to the disease in the United States because of the large herds of cattle which were driven northward from Texas and gave a certain disease in some mysterious manner to northern cattle that crossed the trail of the southern cattle. The first account of the disease was given by James Mease in 1814 before the Philadelphia Society for Promoting Agriculture. In 1879 Salmon began an investigation of the disease; and in 1889 Theobald Smith made his epoch-making discovery of the intracorpuscular protozoan parasite inhabiting the blood of the diseased cattle. Immediately thereupon followed the experiments of Smith and Kilbourne, on suggestion of Salmon, which proved the disease to be tick-borne, a suspicion held as early as 1869 according to Smith and Kilbourne. Until that time (1889) infection was variously attributed to saliva, urine, or feces.

The disease may assume either an acute or a chronic form, the acute form occurring during the summer months and the chronic during the autumn and early winter. The symptoms of the acute form are as follows: The temperature often rises to 106° to 108° F within 24 to 48 hours. The sick animal leaves the herd, stands with arched back, head lowered, and ears drooping, the muzzle dry, appetite lost, and rumination stopped. There is constipation during the first stage of the disease, which may give way to diarrhea later. The manure is frequently stained with bile and may be tinged with bloody mucus; the urine is often very dark red or coffee-colored. The blood becomes thin and watery, so that when making an incision into the tip of the ear and allowing the blood to flow over the hand it does not stick to the hand as does the blood from a healthy animal.

Vast numbers of red blood corpuscles are destroyed by the parasites, which accounts in a measure for the reddish color of the urine through the elimination of hemoglobin by the kidneys; and it is believed that the excessive work that the liver has to perform in attempting to transform the excess of destroyed corpuscles into bile, causes this organ to become deranged in function, and eventually a complete stagnation may result with fatal termination. Mortality ranges from 50 to 75 per cent.

The chronic form of the disease according to Mohler[20] shows all the symptoms of the acute type, but in a milder degree. The temperature usually remains about 103° and never exceeds 105° F. There is loss of appetite, stoppage of rumination, constipation, and albumin in the urine. An anemic condition of the blood, as indicated by the pale and bloodless mucous membranes, is also present, but hemoglobin is not usually excreted by the urine; hence the red-water symptom is absent. There is also excessive loss of flesh, and before the end of the attack the affected animal is greatly emaciated. Although death rarely occurs, the value of the animal is much reduced.

Experimental evidence. In 1888 an "investigation into the nature, causation and prevention" of the disease was undertaken by the United States Department of Agriculture, Bureau of Animal Industry, under the direction of D. E. Salmon. The work was done by Theobald Smith and F. L. Kilbourne[21] and marks a most important epoch in our knowledge of protozoön diseases and in the history of preventive medicine.

During a period of about four years of almost continuous investigation, the problem was exhaustively studied in both the field and the laboratory. The field experiments were carried out along three different lines:

(1) Ticks were carefully picked from Southern animals so that none could mature and infect the ground. The object of this group of experiments was to find out if the disease could be conveyed from Southern to Northern stock on the same inclosure without the intervention of ticks. (2) Fields were infected by matured ticks and susceptible cattle placed on them to determine whether Texas fever could be produced without the presence of Southern cattle. (3) Susceptible Northern cattle were infected by placing on them young ticks hatched artifically, i.e., in closed dishes in the laboratory (Smith and Kilbourne, 1893, *loc. cit.*).

Healthy native cattle (Washington, D. C.) were exposed to sick native cattle free from ticks for months without contracting the disease, proving that the excretions had nothing to do with the transmission of the disease. In the absence of ticks, sick animals are harmless. Again several thousand, mostly full-grown ticks, collected from cattle in North Carolina, were scattered over the ground in a field on September 13. Four native cattle were placed in the field September 14; of these animals three contracted Texas fever. This experiment was repeated with five experimental animals, and a newborn calf, all of which contracted the fever. A yearling heifer was placed in a box stall and a number of young ticks, hatched artificially in glass dishes, were placed on the animal at intervals. The heifer contracted Texas fever. A repetition of this experiment on various occasions always gave similar results. It was definitely concluded that "Texas fever in nature is transmitted from cattle which come from the permanently infected territory to cattle outside this territory by the cattle tick, *Boöphilus bovis,* and that the infection is carried by the progeny of the ticks which matured on infected cattle, and is inoculated by them directly into the blood of susceptible cattle." The authors above cited state that the contents of the bodies of ticks in various stages of growth were examined microscopically with considerable care, but that the abundant particles resulting from the breaking up of the ingested blood corpuscles obscured the search so that nothing definite was discovered. "The very minute size of the microörganism renders its identification well-nigh impossible, and any attempt will be fraught with great difficulties."

Other tick carriers of the protozoön are *Boöphilus microplus* (Canestrini) and *B. decoloratus* (Koch) within their range—the former Australia, Philippine Islands, South America, and the latter Africa.

The causal agent. Babesia bigemina [= *Piroplasma bigeminum* (Smith and Kilbourne)], the causal protozoön parasite of Texas cattle fever, was discovered by Theobald Smith in 1889 and was named *Pyrosoma bigeminum.*

Rees[22] reports that the piroplasms described and illustrated by Smith and Kilbourne (1893) included two species: (1) *Babesia bigemina* and (2) *B. argentina* Lignières, which is of minute size and possesses other characteristics as well. He was able to cultivate the latter in vitro but could not cultivate the former.

The life cycle of *Babesia begemina* (Smith and Kilbourne) in the tick was fully elucidated by Dennis (1932).[23] He summarizes his results:

> When blood which is infected with *B. bigemina* is taken into the gut of the tick, many of the intracorpuscular parasites are soon freed. Certain of these normal-appearing parasites become transformed into gametes through growth and slight structural modification. The gametes are motile vermicule-like bodies which show no differentiation between the sexes. The gametes become associated in pairs, the individuals of which eventually fuse to form the zygote. The zygote becomes a motile oökinete which passes through the thin wall of the gut and penetrates the contiguous reproductive organs. The ova of the tick are invaded by the oökinetes which round up and grow to form sporonts. The sporont secretes a cyst within which it divides to form naked sporoblasts. The sporoblasts form multinucleate sporokinetes which migrate and are carried by cell proliferation throughout the tissues of the developing tick; some of the sporokinetes come to occupy the *anlagen* of the salivary glands. The sporokinete undergoes fragmentation to form the minute infectious sporozoites" (Fig. 158).

Rocky Mountain spotted fever has been known in the Bitter Root Valley of Montana (U.S.A.) since 1872.[24] It is also known as "tick fever," "black fever," "blue disease," and "black measles." The most characteristic and constant symptom is the rash which appears about the second to the fifth day on the wrists, ankles, and less commonly on the back, later spreading to all parts of the body. Parker[25] states that the rash is sometimes preceded by a mottled appearance of the skin. The symptoms most often complained of at the outset are frontal and occipital headache, intense aching in the lumbar region, and marked malaise. The incubation period is from two to five days in the more severe infections and from 3 to 14 days in the milder ones. The fever rises rapidly in the more virulent infections to 104° and 106° F. In fatal infections death usually occurs between the ninth and fifteenth day. Both mild and virulent strains of spotted fever occur in the same locality. Because errors in diagnosis are easily made, laboratory tests are advised. One of the common tests consists of the intraperitoneal injection of blood (1 cc whole blood) into male guinea pigs. In positive tests the guinea pigs show scrotal swelling, reddening, and sloughing of the skin.

The causal agent was discovered by Wolbach[26] in 1919; he named

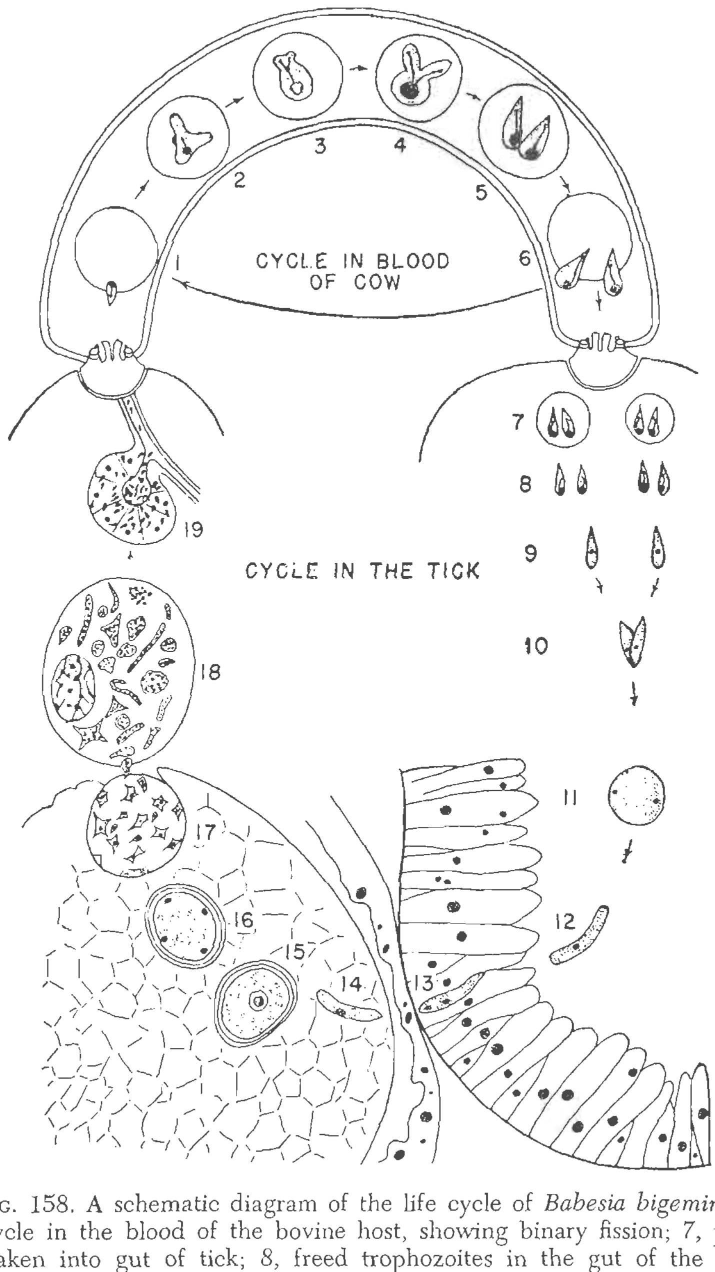


FIG. 158. A schematic diagram of the life cycle of *Babesia bigemina*. *1–6*, the cycle in the blood of the bovine host, showing binary fission; *7*, parasite just taken into gut of tick; *8*, freed trophozoites in the gut of the tick; 9, vermicule-like isogametes; *10*, beginning of syngamy, association of the gametes in pairs; *11*, completion of syngamy; *12*, motile zygote, or oökinete; *13–14*, oökinete passing through wall of the gut of the tick, through the oviduct, and entering the ovum; *15*, sporont formed by the rounding-up and growth of the oökinete; *16–17*, formation of sporoblasts; *18*, sporokinetes in one of the large cells which are destined to form part of a salivary acinus; *19*, sporozoites in the salivary gland (a single acinus shown) of the larva of the tick, whence they are transferred into the blood of a new host. (After Dennis.)

it *Dermacentroxenus rickettsi* in honor of Dr. Howard T. Ricketts, who made great contributions to our knowledge of both Rocky Mountain spotted fever and typhus fever and lost his life in the conduct of investigations in Mexico. The several rickettsioses transmitted by lice, fleas, mites, and ticks are caused by infectious agents known as rickettsiae; hence the causal agent of Rocky Mountain spotted fever is generally referred to as *Rickettsia rickettsi* (Wolbach).

Rocky Mountain spotted fever is endemic in the United States, in some parts of Canada (western), in Mexico, and in some parts of South America where it is known as São Paulo fever and by other names (see below). The greatest number of cases occur in populations engaged in outdoor occupations, principally agriculture. Both sexes and all ages are subject to the disease. In the western part of the United States most of the cases occur in men, while in the eastern part of the United States more women and children contract the disease. Parker suggests that this is probably due to the fact that the eastern vector is a tick, *Dermacentor variabilis* (Say), which infests the dog, a household animal. The Rocky Mountain wood tick, *Dermacentor andersoni* Stiles, is found far less frequently on dogs.

Tick transmission of spotted fever. In 1902, after a preliminary investigation, Wilson and Chowning[27] advanced for the first time the theory that a tick ("wood tick") acts as the natural vector of the disease. According to Ricketts (in the 48th Biennial Report of the Montana State Board of Health, p. 106) as recorded by Hunter and Bishopp[28] "the first experiments which resulted in the proof of the transmission of spotted fever by a tick were conducted by Drs. McCalla and Brenton of Boise, Idaho, in 1905. In these experiments a tick which was found attached to a spotted fever patient was removed and allowed to bite a healthy person. In 8 days this person developed a typical case of spotted fever. The experiment was continued by allowing the same tick to bite a second person. In this case again a typical case of spotted fever resulted."

The famous experiments of Dr. H. T. Ricketts[29] began in April, 1906. The more important published work of this lamented investigator has been brought together in a memorial volume[30] from which the following summary is made of his reports on spotted fever. First of all it was shown that the disease could be transmitted to guinea pigs by direct inoculation and that the duration of the fever and cutaneous phenomena resembled very closely the conditions as observed in humans. Hence, knowing the susceptibility of this species, it was used for further experimentation.

On June 19, 1906, a small female tick was placed at the base of the ear of a guinea pig inoculated intraperitoneally June 11 and 3 cc of defibrinated blood of a spotted fever patient. The tick fed for two days on this animal and was then removed and kept for two days in a pill box and on June 23 placed at the base of the ear of a healthy guinea pig, the former animal dying on the same day with characteristic symptoms. On June 28 the second guinea pig showed decided rise in temperature, which continued high until July 5 and became normal on July 7. Proper controls were conducted and two guinea pigs which were in the same cage with the tick-bitten guinea pig for two weeks did not become infected, indicating that mere association did not result in contracting the disease. It will be noticed that Ricketts called the wood tick which he used *Dermacentor occidentalis* Neum. Evidently the species was actually *Dermacentor andersoni* Stiles.

In addition to many other successful experiments during the following year Ricketts found that the disease can be transmitted by the male[31] as well as by the female tick and that "one attack of the disease establishes a rather high degree of immunity to subsequent inoculation." Furthermore a collection of ticks taken in the field transmitted the disease to a guinea pig in the laboratory, indicating the fact that infective ticks occur in nature in small numbers.

It was also ascertained that "the disease may be acquired and transmitted . . . by the tick during any of the active stages . . . and that the larvae of an infected female are in some instances infective. . . . The disease probably is transferred through the salivary secretion of the tick, since the salivary glands of the infected adult contain the virus." The transmission is believed to be "biological rather than purely mechanical."

Experiments conducted by Moore (Ricketts, 1911, *loc. cit.*, pp. 428–436) show that the "minimum duration of feeding necessary for a tick to infect a guinea pig was one hour and forty-five minutes. The average time necessary seems to be about ten hours, while twenty hours were almost constantly infective." Maver (see Ricketts, 1911, *loc. cit.*, pp. 440–444) in a series of experiments with other species of ticks found that spotted fever can be transmitted from infected to normal guinea pigs by nymphal *Dermacentor variabilis* (Say) infected as larvae, by adult *Dermacentor parumapertus marginatus* Banks and nymphs of *Amblyomma americanum* (Linn.). Ricketts showed transmission by adult *Dermacentor albipictus* (Packard) infected as nymphs.

The infection in nature. Parker points out that field observations made in eastern Montana in 1916 and 1917 suggested that under the "epizoötilogic" conditions concerned, some agent other than *Dermacentor andersoni* Stiles was likely involved in the natural maintenance of the virus. In 1923 Parker[32] established the fact that the rabbit tick, *Haemaphysalis leporis-palustris* (Packard), is capable of transmitting the infection from rabbit to rabbit and also that infected rabbit ticks occur in nature. A third important fact was established, namely that the infection is transmitted by infected female ticks to the egg as in the case of *Dermacentor andersoni* Stiles. While the rabbit tick only rarely bites man, it is important indirectly in that it is a potent vector under natural conditions and is furthermore the only known vector which occurs in all parts of the United States. The infection carried by this tick is reported by Parker to be extremely mild. Rabbits of all species studied are hosts of both wood ticks and rabbit ticks. Rabbits are natural reservoirs of the infection in western North America, field mice in other parts. Dogs are believed to be important reservoirs of the infection in the closely related disease, boutonneuse fever of Europe.

The American dog tick, *Dermacentor variabilis* (Say), was proved to be a carrier of the eastern type of Rocky Mountain spotted fever in 1931 by Dyer, Badger, and Rumreich,[33] who used larvae bred from eggs. The larvae were fed on a guinea pig infected with eastern spotted fever and after engorgement were allowed to molt, and the nymphs were fed to engorgement on a noninfected guinea pig and were then ground up and injected into fresh guinea pigs, thus establishing a strain of virus in guinea pigs. The results of these investigators confirmed the early work of Ricketts and Maver (1911). They also proved that transmission is transovarian. Parker,[34] 1937, reports successful stage-to-stage and generation-to-generation transmission with *Dermacentor occidentalis* Neum., the Pacific coast tick, and *Rhipicephalus sanguineus* (Latreille), the brown dog tick. In the latter species virus continuity was shown from larval ticks of one generation through six successive stages to adults of the next. In *Amblyomma americanum* (Linn.), Maver (1911) had already reported larva-to-adult continuity, and transmission from female to larvae was accomplished by Parker. With *Amblyomma cajennense* (Fabr.), the Cayenne tick, transmission from larvae to adults has been shown, and for *Dermacentor parumapterus marginatus* Banks, a rabbit tick, transmission from nymphs to adults was shown by Maver (1911), and continuity from larvae to nymphs as well as survival of the virus in adults was shown by Parker (1937). Parker states that these data are considered sufficient to indicate that each of these six additional species is a possible natural carrier of spotted fever virus and that four of these, *D. occidentalis* Neum., *R. sanguineus* (Latr.), *A. americanum* (Linn.), and *A. cajennense* (Fabr.), are possible present or future agents of transmission to man. Actually *Amblyomma americanum* Linn., the lone star tick, is now known to be the vector of spotted fever in Oklahoma and Texas (Parker, Kohls, and Steinhaus[35]); and *Amblyomma cajennense* (Fabr.) is the vector of the São Paulo fever (spotted fever) of Brazil and of Tobia petechical fever (spotted fever) of Columbia.

Mechanism of infection. The infection is picked up with the bite from the reservoir animal by a tick in any stage of its life history and is passed on from stage to stage; e.g., infected blood is ingested by the larva, the infection is passed on to the nymph (which may infect), and thence to the adult which in turn may infect, and at least some infected female ticks will pass the virus on through their eggs to the larvae of the next generation.

When normal adult ticks are first fed on an infected host, just long enough to ensure ingestion of the virus, and are then transferred directly to a normal host (the tick-feeding thus being interrupted but essentially continuous) Parker's[37] tests showed a period of from 9 to 12 days between the ingestion and the transmission of the virus. Except under experimental conditions infection is not transmitted by the same stage of the tick that acquires it, but by the next and subsequent stages. The infection is transmitted by the *bite* of the tick. The great majority of persons with cases of Rocky Mountain spotted fever give definite history of "tick bite from 2 to 10 or 12 days before onset." The percentage of ticks that contain infectious virus is reported to be very small: it may be less than one per cent and is rarely as high as five.

Parker[37] states that a minority of perhaps 5 per cent of infected persons deny the possibility of having been infected by the tick bite. "It has long been known that blood-virus of experimental animals would not pass the unabraded skin, nor would it infect if ingested. The possibility that tick virus would act differently was not considered until a short time ago. In an exceptionally clear-cut series of tests it was shown that tick virus would infect if merely dropped among the hairs on the unabraded skin of guinea pigs; also if dropped into the eye. In another series of tests it was demonstrated, contrary to previously accepted ideas, that tick excrement is frequently infectious, but tests to determine whether it will cause infection by contamination of the unabraded skin have proved negative."

The virus survives the winter in infected nymphal or adult ticks. At the end of winter the virus in these ticks is present according to Parker (*loc. cit.*, 1933) as a nonsymptom-producing and frequently immunizing virus, and does not produce symptomatically recognizable infections until its level of virulence is raised, either by heat or by the ingestion of blood. This phenomenon of *reactivation* has direct bearing on the fact that the bites of hibernated ticks in nature very early in the spring do not result in frank infection. It also explains why tick bites grow more dangerous as the season advances. Parker states, "In the early spring when the virus in the recently emerged tick is at its lowest ebb of infectiousness, and while the days and the nights are still cool, it takes so long for the virus to become reactivated after a tick has become attached that it is usually detected and removed before the virus has become virulent." It further gives force to the advice so seriously given by all authorities on Rocky Mountain spotted fever to detach ticks at once. It may be a matter of some hours and not infrequently a day or more after the tick attaches itself before reactivation occurs. The shortest recorded interval is one and three-quarters hours (Ricketts). If a tick which has become attached is removed within a few hours, the danger of infection is materially minimized. The danger from infection in the Atlantic coast states is later in the summer.

Control and prevention. The control of Rocky Mountain spotted fever over a wide area by the control of the tick vectors has been discouraging. According to Parker (*loc. cit.*, 1933) definite local good has been accomplished by dipping, grazing control, and the control of rodents, but lack of permanence of results and lack of knowledge as to the relative importance of the various factors of the local complex have all militated against substantial gains.

In 1926 Cooley obtained parasitized nymphal ticks from a tick parasite colony established by Larrousse on Naushon Island. The parasite, introduced originally from France, is named *Ixodiphagus caucurtei* du Buysson, a chalcidoid parasite of the subfamily Encyrtinae (Hymenoptera), which is said to attack only ticks. Cooley[38] introduced these parasites into Montana, where they were reared in quantity under laboratory conditions, and a field release of 381,190 parasites was made during the summer of 1928. The parasite has become established but it is believed unlikely that it will prove useful as an agency of tick control.

The avoidance of tick bites is urged by keeping out of known tick-infested areas during spring and early summer; or by wearing suitable clothing, such as high boots, leggings, puttees or socks outside the trouser legs. The advantage of early removal of ticks that may have escaped attention and have become attached has already been pointed out.

Parker[39] reported in 1935 on the results of the Public Health Service vaccine for the prevention of Rocky Mountain spotted fever and in 1935 (*loc. cit.*) he comments as follows:

> The vaccine has definite protective value. The degree and the duration of protection vary with the person vaccinated and with the degree of virulence of the infecting strain of spotted fever virus.
>
> The average person vaccinated in the spring retains marked immunity during at least the remainder of that year. The immunity is usually sufficient to afford full protection against the relatively mild strains of spotted fever but is apparently progressively less effective as the virulence of infecting strains is increased.

Colorado tick fever. Parker, in 1937, reported a febrile diseases of frequent occurrence in many parts of the Rocky Mountain region following the bite of *Dermacentor andersoni* Stiles. The disease is now known as "Colorado tick fever." There is a close similarity between this disease and dengue (see Chapter XII) both clinically and in the particle size of the respective viruses; however, the two diseases do not give cross immunity hence "appear to be distinct disease entities." (Florio, Hammon *et al.*)[40] Parker describes Colorado tick fever as a "disease of a remittent type and is commonly characterized by the occurrence of two febrile periods, each of two to four days' duration, with a remission period of one to several days between. The onset is sudden and the climax is often reached within the first 24 hours. *There is no rash.* Symptoms other than fever are malaise, chilly sensations, severe headache, conjunctivitis, photophobia, and generalized muscular and joint pains with particularly severe aching in the lumbar region. The malaise is usually intense. Constipation is the rule. The temperature often reaches 104° to 105° F or over, but may not exceed 101° to 102° F. The pulse rate is frequently 120 to 130. In most instances, though not always, the symptoms are more severe during the first febrile period. . . . It is non-fatal."

Tick transmission of tularemia. Parker[41] states that tularemia infection in ticks was supected in numerous instances during the seasons of 1922 and 1923 on account of the gross lesions at death in guinea pigs into which such ticks had been injected. Confirmation was made by cultivation of *Pasteurella* (= *Bacterium*) *tularensis* from guinea pigs in which the tick strain had been propagated. *Dermacentor andersoni* Stiles collected from nature proved infective; also experimentally, infection acquired by immature ticks was passed on to subsequent stages of the same generation. Later Parker and Spencer[42] (1926) demonstrated congenital transmission. This is believed to be the first record of "hereditary transmission" of a known bacterial infection. Probably several species of ticks are able to transmit the infection; *Dermacentor occidentalis* Neum. and *Dermacentor variabilis* (Say) have been incriminated. *Haemaphysalis leporis-palustris* (Packard), the so-called rabbit tick, is largely responsible for the maintenance of the infection in nature. *Pasteurella tularensis* has been recovered both from infected sage hens in Montana and from the tick, *Haemaphysalis cinnabarina* Koch, taken from the same birds. Davis and Kohls[43] (1937) discovered evidence indicating that *Ixodes pacificus* Cooley and Kohls may also be a carrier of tularemia to human beings.

Tick paralysis. A paralysis of sheep and calves attributable to ticks has been known in Australia according to Henning,[58] since 1843. Paralysis reported as "acute ascending paralysis" associated with tick bite was described in 1912 by Temple[44] in Oregon. The case reported was that of a child in which there was a complete paralysis of the motor and sensory nerves extending to the knees, causing inability to stand in the morning after retirement in apparent good health. On the third day the paralysis had involved the nerves of the throat and the child was unable to swallow or speak. Upon removal of two fully engorged ticks from the occipital region recovery was rapid and complete within a week. The ticks were not positively identified, though they were presumably *Dermacentor andersoni* Stiles. In 1913 Hadwen[45] reported cases of paralysis in sheep following the bite of *Dermacentor andersoni* Stiles (*D. venustus* Banks). He also cites excerpts from letters (*Canad. M. A. J.*, 1912) from physicians in British Columbia indicating frequent occurrence of paralysis in children following tick bite. The ticks were commonly removed from the nape of the neck.

In 1913 Hadwen and Nuttall[46] report having produced paralysis experimentally in the dog by means of *Dermacentor andersoni* Stiles (*D. venustus* Banks). The paralysis was the same as in sheep. In the experiment reported, paralysis was caused by a single tick in eight days. The authors state: "On the hypothesis that the symptoms are due to *toxins* given off by the tick, 'the period of incubation' might be explained on the supposition that it is only when the tick commences to engorge or feed rapidly, some days after it has become attached, that it gives off the hypothetical toxin in its saliva in sufficient quantity to produce pathogenic effects."

Tick paralysis is widely known in the western United States and Canada adjacent to the Rocky Mountains and coincides with the distribution of *Dermacentor andersoni* Stiles. Hearle, 1938 (*loc. cit.* p. 399), states:

> As an index of the wide distribution of this trouble in British Columbia, incomplete returns from medical practitioners indicated that nearly 150 human cases had been noted in the province. . . cases in sheep have been particularly numerous, and many deaths have resulted. Cattle are usually less susceptible, but trouble from tick paralysis has been noted from time to time, and in the spring of 1930 a serious outbreak in steers was investigated; over 100 paralysis cases, sixty of them fatal, being noted in one herd. We know of only one equine case. In sheep districts where this trouble is prevalent, flock masters are obliged to examine their animals frequently for the purpose of removing the offending ticks from sheep showing symptoms of weakness or staggers. . . . When animals become paralyzed all ticks showing engorgement should immediately be removed; in searching for them special attention should be paid to the back of the head, the region of the spine and along the neck, since this is where they mainly become attached.

Ixodes holocyclus Neumann causes paralysis in calves, sheep, and dogs in Australia. According to Ross[47] this condition is caused only by the mature engorging female tick two days before it is ready to drop from the host. He used the dog in his experimental work. Several mild cases of paralysis in children have been reported to the author in California, evidently as the result of attachment by *Ixodes pacificus* C. and K. *Dermacentor variabilis* (Say) may also be incriminated.

Bullis fever, also known as "lone star fever" referring to the vector, the lone star tick, is a virus infection, resulting in an unusually low white blood cell count and a severe postorbital and occipital headache and lymphadenitis. The onset is usually abrupt, with an initial chill or chilliness; fever soon follows; and there is pronounced lassitude and prostration, with nausea. The fever lasts from 4 to 14 days. Convalescence is protracted. (Woodland, McDowell, and Richards,[48] 1943.) This disease was first observed at Camp Bullis, Texas, in 1942. Anigstein and Bader,[49] as well as the above-mentioned group, express the belief, based on both epidemiological and experimental evidence, that the vector of this infection is *Amblyomma americanum* (Linn.), known as the "lone star tick"; it is believed to be at least one of the carriers of Bullis fever in nature.

Q fever was first reported from Australia (Queensland) in 1937,[50] where most of the infected persons were abattoir workers; the causal organism is *Coxiella* (= *Rickettsia*) *burneti* (Derrick) (1939). The disease is characterized by an acute onset with chills, prostration, and a continuous fever, which lasts from a few days to two to three weeks; death seldom results. In 1937 the finding of a rickettsial infection was reported in the tissues of animals in western Montana (U.S.A.); the organism was later named *Rickettsia diaporica* by Cox[51] and the disease was called "Nine Mile Fever," "Nine Mile Creek" being the place in Montana where *Dermacentor andersoni* ticks were collected and proved naturally infected with *Rickettsia diaporica*,[52] now shown to be the same as *Coxiella burneti*. The first known human infection in the United States, a laboratory infection, was reported by Dyer (1938), who pointed out its relationship to Australian Q fever (see Steinhaus,[53] 1946, pp. 295–302). In 1943, Parker and Davis[54] reported the *Rickettsia* (*Coxiella*) of American Q fever in the lone star tick, *Amblyomma americanum* Linn. In Australia *Haemaphysalis humerosa* Warburton and Nuttall, which does not bite man, is considered an important vector of the infection among bandicoots (*Isodon* spp.), serving as an important factor in maintaining endemicity of the disease. Rats are also regarded as important reservoirs. *Ixodes holocyclus* Neum., which bites man and marsupials, may be a factor in areas where the disease exists in marsupials, in that the infection may be deposited on the skin with tick feces or may be otherwise transmitted. Infection may result through the unabraded skin when it is contaminated with tick feces. Actual transmission by the bite of ticks has not been satisfactorily demonstrated. Transovarial passage in ticks has been observed. Several species of ticks other than those mentioned can at least be experimentally infected.

Philip[55] (1948) reports that feces from infected *Dermacentor andersoni* are very rich in rickettsiae, which remain viable in storage as long as 586 days. Infection from tick feces emulsion penetrated the unbroken skin as well as the abraded skin of test animals; also powdered feces dusted in the nostrils, eyes, and mouth produced infection. Philip also recovered the infection from houseflies, *Musca domestica*, caught at large in an animal room in which animals infected with Q fever were kept. Attempts to transfer the infection through these flies to normal animals failed.

In their Q fever studies in California, Huebner and Jellison and associates[56] recovered *Coxiella burneti* from raw milk, and they conclude that raw milk may serve as a source of infection to man by some mode as yet undetermined. Available evidence did not indicate that the drinking of raw milk was the cause of the majority of cases thus far studied.

Lymphocytic choriomeningitis is an infectious disease caused by a virus which is discharged in nasal secretions and in the urine of naturally infected mice. The manner in which naturally infected mice contract the disease is unknown. Experimental infection of all stages of *Dermacentor andersoni* with the virus was demonstrated by Shaughnessy and Milzer[57] (1939) by feeding the ticks on infected guinea pigs. They report stage-to-stage transmission of the virus from larvae to nymphs and nymphs to adults, as well as generation-to-generation transmission from adults to eggs. Typical symptoms were produced in all test animals, and typical pathology was shown by those that had died; surviving animals were immune to inoculation of the virus. Feces from feeding adult ticks applied to the lightly scarified skin of guinea pigs caused typical symptoms.

East Coast fever is a highly fatal disease of cattle along the East Coast of equatorial Africa. The mortality, it is said, may run over 90 per cent. The disease is caused by the protozoön, *Theileria parva* (Theiler), and is therefore a form of piroplasmosis (theileriosis) though, unlike red water, it is not readily transmitted by means of blood inoculations, nor is it accompanied by jaundice or hemoglobinuria. A very characteristic symptom is swelling of the superficial lymphatic glands.

The incubation period varies from 9 to 19 days and is usually from 10 to 15 days. The disease is transmitted by several species of ticks as reported by Lounsbury as early as 1906. He proved the adult brown tick, *Rhipicephalus appendiculatus* Neum., to be a vector, and later he showed that the disease may also be transmitted by the Cape brown tick, *Rhipicephalus capensis* Koch, and the red tick, *Rhipicephalus evertsi* Neumann.

Henning[58] states that unlike red water, East Coast fever is not transmitted from the adult female tick to the larva through the egg, but only by an adult tick which became infected during its nymphal stage, or by a nymph that became infected during its larval stage. While *Rhipicephalus appendiculatus* Neum., *R. capensis* Koch, and *R. simus* Koch are three-host ticks, *R. evertsi* Neum. is a two-host tick, hence as the tick remains on the same host during both its larval and nymphal stages transmission of the infection is possibly only during the adult stage. The larvae of the two-host tick, *R. evertsi,* hatch from eggs on the ground as do other ticks, then proceed to attach themselves to the ears (often inside) and the flanks of cattle. The larvae become fully engorged and molt while on the host. The nymphs then engorge and drop off the host to molt and give rise to the adult. The adult then attaches itself to a second host, engorges, and drops to the ground where the females lay eggs. Lounsbury and Theiler both found that ticks of the three-host species which have sucked infected blood during their larval stage can transmit the infection only during their nymphal stage; i.e., whether the nymph feeds on either a susceptible or a nonsusceptible host, infectivity is lost. A single tick can transmit the infection only once, and that during the stage that follows the one in which it had the infectious meal.

Equine piroplasmosis. At least two types of piroplasmosis are found in horses, mules, and donkeys, namely true equine piroplasmosis, traceable to *Babesia caballi* (Nuttall), occurring in Africa, Russia, Transcaucasia, and probably Siberia, and secondly a similar though distinct disease traceable to *Nuttallia equi* (Laveran) occurring in Transcaucasia, Italy, Africa, India, and South America (Brazil). *Babesia caballi* is transmitted by *Dermacentor reticulatus* (Fabr.) in Russia; and *Nuttallia equi* is transmitted by *Rhipicephalus evertsi* Neum. in South Africa.

Canine babesiosis (piroplasmosis), also known as "malignant jaundice" of dogs, is prevalent in southern Europe, Asia, South Africa, and more recently in the United States (Florida). The causal organism is *Babesia canis* (Piana and Galli-Valerio); the carrier is *Rhipicephalus sanguineus* (Latr.) in India, southern Europe, and the United States; *Haemaphysalis leachi* (Audouin) is a South African vector, and *Dermacentor reticulatus* (Fabr.) and *Ixodes ricinus* (Linn.) transmit the infection in southern Europe. Brumpt and Larrousse[59] have shown that *Dermacentor andersoni* Stiles can carry the disease. The infection is hereditary in the tick, but transmission to the dog is effected by the bite of the adults, but not by the larvae and nymphs, according to Brumpt.[60] The incubation period varies from 10 to 20 days. Sanders[61] (1937) reported that *R. sanguineus* (Latr.) is by far the most common species encountered in kennels and on animals affected with canine babesiosis in Florida.

Sanders (*loc. cit.*) states that the acute form of this infection is not difficult to diagnose; "the high temperature, increased pulse and respiration, progressive anemia, jaundice, the history of tick infestation and the demonstration of the causal organism are usually observed."

Heartwater, a dreaded disease of South African sheep, goats, and cattle is caused by *Rickettsia ruminatium* (Cowdry). It is carried by the "bont" tick, *Amblyomma hebraeum* Koch.

Henning[62] states that the bont tick may subsist on any warm-blooded animal; it has been found during all its stages on man, all species of domestic animals, and several species of antelopes. Bovines are its favorite host. This tick may lay as many as 18,000 eggs. The tick becomes a vector of the infection only after it has sucked blood from a diseased animal; i.e., hereditary infection does not occur, hence the newly hatched larvae are not infectious. *Amblyomma variegatum* (Fabr.) is able to transmit the infection in the adult stage.

Bovine anaplasmosis. Anaplasmosis is an important and practically world-wide infection of cattle caused by minute punctiform blood parasites described by Theiler in 1910 as *Anaplasma marginale* with the organism at or near the periphery of the red cell and *Anaplasma marginale* variety *centrale*, a somewhat smaller body, located approximately in the center of the infected corpuscle.

Anaplasmosis is described by Stiles[63] as an acute, subacute, or chronic, febrile, infectious, protozoön disease, characterized by loss of flesh, labored breathing, suspension of milk flow, anemia, jaundice, and marked degenerative changes in the red blood corpuscles owing to the activity of the microscopic parasites. The average mortality ranges from 30 to 50 per cent in the animals affected. Mechanical transmission of the infection by several species of Tabanid flies has been reported, and Stiles records a total of seventeen species of ticks which have been incriminated by various investigators in transmission, among them *Boöphilus annulatus* (Say), *B. decoloratus* (Koch), *B. microplus* (Canestrini), *Rhipicephalus simus* Koch, *R. bursa* Canestrini et Fanzago, *Ixodes ricinus* (Linn.), *Hyalomma lusitanicum* (Koch), *Rhipicephalus sanguineus* (Latreille), *Dermacentor variabilis* (Say), *D. andersoni* Stiles, and *D. occidentalis* Neum.

In 1936 Herms and Howell[64] reported five cases of tick transmission, two, nonhereditary, with *Dermacentor albipictus* (Packard) and *D. andersoni* Stiles, and three in which the infection passed through the eggs of the western dog tick, *Dermacentor occidentalis* Neum, the offspring being infective in both the larval and nymphal stages. In the congenital transmissions from the time the infective larval ticks were applied to the host animal to the time that marginal bodies first appeared in the blood the elapsed time was 37 days, 32 days, and 123 days respectively. In the two nonhereditary transmissions the elapsed time was 28 and 29 days. Howell, Stiles, and Moe[65] have demonstrated transovarian transmission by *Dermacentor andersoni* Stiles.

That deer, namely the southern black-tailed deer, *Odocoileus hemionus columbianus* (Richardson) and the Rocky Mountain mule deer, *Odocoileus hemionus hemionus* (Rafinesque), may serve as reservoirs for anaplasmosis was proved by Boynton and Woods in 1933.[66]

COMBATING TICKS (IXODINE)

Ticks on live stock. *Rotenone-sulphur* dips are commonly used for the control of ticks on the bodies of livestock. The dip is prepared as follows: 10 pounds 5 per cent rotenone, 10 pounds wettable sulphur (325 mesh or finer), 1 pint wetting agent, 100 gallons water. This dip is nontoxic to the animals and according to Stewart and Furman (mimeo. series, Univ. of California, Agric. Exp. Sta.) generally gives effective control. This formula is also effective against lice, cattle grubs, and sheep keds.

Arsenical dips are likely to be more effective but are also more toxic. These dips are made as follows: 24 pounds sodium bicarbonate (sal soda), 8 pounds arsenic trioxide, 1 gallon pine tar, 500 gallons water. This dip is particularly useful in the control of the Texas cattle fever tick, *Boöphilus annulatus*.[67] From the time the arsenic is purchased to the final disposal of the old dip, great care must be exercised in storing, handling, and using it because of its very toxic nature. The animals should always be watered a short time before they are dipped so that they will not be thirsty and drink the dip. After the animals emerge from the dipping vat, they should be kept on a draining floor until the dip ceases to run from their bodies; then they should be herded into a place free of vegetation until they are entirely dry. If they are allowed to drain in places where pools of dip collect from which they may drink or if they are turned at once to pasture where the dip may run from their bodies on grass or other vegetation, serious effects may result. Also crowding animals before they are dry should be avoided, and they should not be driven far within a week after dipping, especially in hot weather.

After the animals are watered, they are driven from the pen one by one into the chute and thence into the dip. The plunge will wet the animal all over, but a second plunge of the head by using a forked stick is necessary as the animal proceeds through the dip. After emerging, the animals should be driven to a tick-free pasture (see above precautions); the dipping process should be repeated in from 7 to 10 days.

Dipping vats containing arsenical dip must be protected when not in use; also when vats are emptied for cleaning the dip must not be allowed to flow on land or vegetation to which cattle or other animals have access; seepage into wells or springs must also be guarded against.

Other dips. Benzene hexachloride has been reported as effective against several species of ticks on live stock. A 0.5 per cent suspension of BHC (of 10–13 per cent gamma isomer) is suggested. Lactating dairy cattle must not be given a benzene hexachoride dip since the milk may have an extraneous odor for a few days following dipping. Likewise cattle that are to be slaughtered within 60 days should not be dipped in this solution. However, no harm to cattle results from its use.

DDT has been reported as effective in controlling the winter tick, *Dermacentor albipictus,* when used as an emulsion of 0.8 per cent DDT. Information concerning this dip is incomplete.

To free dogs of ticks a thorough application of derris powder or wash is recommended by Bishopp and Smith.[68] Derris when used as a powder should have a rotenone content of at least 3 per cent and should be applied lightly next to the skin at intervals of two or three days. A dip or wash can be made by dissolving an ounce of neutral soap in a gallon of tepid water and mixing two ounces of derris power of which the rotenone content is 3 per cent. The dip should be applied at intervals of five or six days. The dog may be put in a tub or a brush may be used. The dip is allowed to dry on or if necessary surplus liquid may be removed with a towel. Do not permit the dip or powder to get into the eyes of the animal.

The application of the powder is simpler and kills the ticks with which it comes in contact, but a more thorough covering is secured when a wash or dip is applied. The wash also has a longer repellent action. Kennels in which the dogs sleep should be thoroughly sprayed with a coal tar creosote solution, but this should not be allowed to come in contact with the dogs. Since the dog is the principal host upon which the adult *Dermacentor variabilis* (Say), the vector of eastern Rocky Mountain spotted fever, feeds, it is important in these areas to keep dogs tick-free. It is also important that humans exercise every precaution to avoid getting ticks on the body in order to prevent infection.

Control of ticks on vegetation. As has already been explained, when Ixodine (hard) ticks reach maturity on the host they drop to the ground where the eggs are deposited and the seed ticks (larvae) emerge, climbing up low shrubbery, weeds, and grass ready to cling to any suitable passing animal. Enormous numbers of larvae may thus be encountered, particularly along cattle and deer paths in more or less wooded areas and on the well-marked paths of meadow mice and other rodents in less wooded situations. As has also been explained, in the commoner species of hard ticks the nymphs likewise drop to the ground to molt. In some areas large numbers of nymphs and young adult ticks (before engorgement) climb up the branches and twigs of low shrubbery, where they are in an excellent position to attach themselves to larger animals, including man, as these brush against the vegetation; wild lilac (*Ceanothus*) where abundant and thus infested in commonly called "tick bush." Manifestly much good could be accomplished if an economical insecticidal treatment were available both for use on the ground and on vegetation along such pathways.

Smith and Gouck,[69] 1945, report marked success with both DDT sprays and dusts against *Amblyomma americanum* and *Dermacentor variabilis*. A spray was used containing one avoirdupois ounce of DDT (technical grade), 5 fluid ounces of soluble pine oil, and water to make 15 gallons; the entire surface of the ground and all vegetation up to 3 or 4 feet above ground were covered. No injury to plants was apparent in any of the tests. At the rate of one pound of DDT per acre highly satisfactory control was maintained for a period of about two months, proving pronounced residual effect on ticks. Dust containing 5 per cent DDT in pyrophyllite was effective at 3 pounds per acre.

FAMILY ARGASIDAE (SOFT-BODIED TICKS)

The family Argasidae includes the so-called soft-bodied or nonscutate ticks, in which sexual dimorphism is slight. The integument of all stages except larvae is leathery, wrinkled, granulated, mammillated, or with tubercles. The capitulum is either subterminal or distant from the anterior margin; in larvae the subterminal or terminal capitulum lies in a more or less marked depression, the camerostome. The articulations of the palpi of all stages are free; the porose areas are absent in both sexes. There are about 60 species. Cooley and Kohls[70] (1944) provide us with an excellent account of this family. They recognize four genera, namely, *Argas, Ornithodoros, Otobius* and *Antricola.*

GENUS ORNITHODOROS

The genus Ornithodoros. In this genus the capitulum is either subterminal or distant from the anterior margin; the hypostome is well developed. In the integument discs and mammillae commingle in a variety of patterns; hood, camerostome, cheeks, and eyes are present or absent; dorsal humps and subapical dorsal protuberances on legs are progressively more prominent in the successive nymphal stages. The body is more or less flattened but strongly convex dorsally when distended; the integumental pattern is continuous over the sides from dorsal to ventral surfaces. (Cooley and Kohls, *loc. cit.*). The genus includes about 50 species, some 20 of which occur in North America, Central America, and Cuba.

Ornithodoros moubata (Murray) occurs only in Africa where it is a widely distributed vector of relapsing fever, hence is known as the African relapsing fever tick (Fig. 159). It occurs in native huts, hiding in dust and thatch. It feeds chiefly at night and engorges rapidly. It is an eyeless species with a specific arrangement of the "humps" on the protarsus of the first pair of legs, being "subequal and tooth-like." The adults measure from 8 to 11 mm in length and about 7 mm in breadth. The color varies from dusty brown to greenish brown in living specimens. Eggs are deposited in batches of from 35 to 340 at intervals after blood meals during the lifetime of the female. The maximum number of eggs laid by one female was 1,217 according to Jobling.[71] The incubation

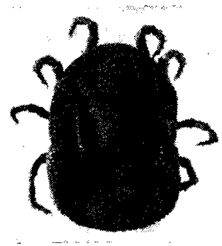


FIG. 159. African relapsing fever tick, *Ornithodorus moubata*. ×3.

period lasts from 7 to 11 days and over, depending on temperature. Davis[72] calls attention to the fact that the larvae are completely quiescent, with legs closely applied to the body, and within a few hours molt in the split egg shell to the first nymphal stage. The form that many authors refer to as the newly hatched larva is actually the first nymphal stage, which attaches itself to its first host animal and remains there for about a week feeding on lymph, then disengages itself and molts for the second time. There then follow several other feeding periods and molts, six to nine in all before sexual maturity is reached. Young females may molt even after sexual maturity. This species will feed on a wide range of host animals besides man including pigs, dogs, goats, and sheep; also on rabbits, mice, rats, monkeys, and fowls in the laboratory. It appears to be essentially a parasite of man, however, and is a man-to-tick-to-man vector of relapsing fever.

Since the discovery that African relapsing fever is transmitted by *Ornithodoros moubata* (Murray), many other species of *Ornithodoros* have been found naturally infected with spirochetes infective to man; indeed some believe that any species of this genus is capable of transmitting all strains of relapsing fever normally transmitted by ticks belonging to this genus. With this the author cannot fully agree, since he and his students have been unable to transmit the California strain by means of *Ornithodoros coriaceus* Koch; indeed tick-borne spirochetes seem to possess a tick fastness or specificity to a high degree.

Ornithodoros talaje (Guérin-Méneville) is a South and Central American (south to Argentina) and Mexican species, occurring also in Florida, Texas, Arizona, Nevada, and Kansas, and has been taken in several California counties.[73] It feeds on wild rodents, also on swine, cattle, horses, man and other mammals. It inflicts a very painful bite. It is the vector of relapsing fever in Panama, Venezuela, and Colombia. Bates, Dunn, and St. John[74] proved this tick a vector of relapsing fever in Panama by human experimentation in 1921; in one instance the bites of a naturally infected tick resulted in infection. This species has been reported from New York by Matheson,[75] who remarks, "How this tick reached there (Ransomville, N. Y.) can only be surmised. It probably maintained itself in the heated house and fed on the occupants or more probably on mice or rats or the domestic cat and dog, though none of these is listed as its normal host."

Ornithodoros venezuelensis Brumpt is a Central and South American species. It transmits relapsing fever in Colombia, Venezuela, and Panama. Dunn[76] collected 4,880 ticks of this species in 68 homes in twenty villages, towns, and cities in various parts of Colombia. Ticks infested with relapsing fever spirochetes were present in nearly 28 per cent of the homes in which collections were made. The altitude mentioned for one of the localities, a barracks at Muzo, was 2,700 feet. Like *O. moubata* this species appears to be essentially a parasite of man, although it is known to feed on other animals.

Ornithodoros erraticus Lucas occurs in Spain and northern Africa. It is an important vector of relapsing fever in northern Africa. *O. papillipes* Birula [*O. tholozani* (Lab. et Mégnin)] is a vector in Central Asia.[77] *O. normandi* Larrousse is a very small Tunisian species, reported to be a vector of relapsing fever.

Ornithodoros parkeri is described by Cooley[78] from ground squirrels (*Citellus* sp.), jack rabbits (*Lepus*), and the cottontail in Wyoming and Washington.

Tick-borne relapsing fever. Although we are told that the natives in many parts of Africa dreaded tick bites for many generations, David Livingstone, the explorer, was the first to report (1857) upon the evil effects following the bite of a tick which was later named *Ornithodoros moubata* (Murray, 1877). It was not, however, until 1904 that Ross and Milne,[80] and in 1905 Dutton and Todd,[81] reporting from Uganda and the Congo, and almost simultaneously Robert Koch reporting from German East Africa, gave us the knowledge that these evil effects were due to relapsing fever, a spirochetosis, and that the tick so dreaded by the natives, was actually the vector.

The symptoms of the disease are described by Nuttall and Warburton (*loc. cit.*, 1908), as follows: "Headache (especially at the back of the head), vomiting, abdominal pains and purging, with severe fever, a pulse of 90 to 120, dry hot skin, congested eyes and shortness of breath. After a period of fever lasting about two days, there is a fall of temperature, but a fresh attack soon follows. These relapses occur more frequently than in European (louse-borne) relapsing fever, being usually 5 or 6 in number, but there may be more. The attacks leave the patient in a weak condition for a long time after recovery, which usually follows, but death occurs in about 6 per cent of the cases."

How infection is transmitted. Dutton and Todd (*loc. cit.*) proved that the infection is not only introduced with the bite of the tick but is also transmitted to the offspring of the female tick through the egg. The newly hatched ticks were proved to be infective. Once infected the tick remains so, and the infection may be transmitted from generation to generation without renewal for at least three generations. The infection is transmitted by the bite of either the male or the female tick during all of its active stages. The coxal fluids are believed to play an important role since they contain spirochetes which are washed into the tick feces rendering these infectious as well. The attack of fever takes place in the human in from 5 to 10 days after the tick has bitten. Feng and Chung[82] report that "shortly after the spirochaetes are ingested by the ticks they penetrate the stomach wall and reach the body cavity as evidenced by finding spirochaetes in the legs six hours after the infective feed. . . . The spirochaetes gradually disappear from the stomach and reach the body cavity with the result that from the twelfth day on no more spirochaetes could be found in the stomach contents. . . . From the body cavity the spirochaetes invade the salivary glands, the coxal glands. . . . The constant presence of numerous spirochaetes in the acini of the salivary glands and the finding on several occasions of spirochaetes actually inside the small salivary ducts suggests that besides the coxal fluid the bite alone may also be infective. . . . [The spirochetes] multiply by transverse division after they have penetrated the stomach wall and reached the body cavity and other organs of the tick. . . . Mice inoculated . . . with feces remained sterile."

Tick-borne relapsing fever in the United States. The earliest known focus of endemic (tick-borne) relapsing fever in the United States in believed to have been in Colorado, where cases were reported by Meador[83] in 1915; this focus was in the mountains near Denver. In 1921 Briggs[84] reported two cases of relapsing fever in which the infection had been acquired at Polaris, Nevada County, California, at an elevation of about 5,750 feet. It would seem that Briggs suspected lice since he remarks, "Many tramps, put off trains at Truckee, find a day or so of employment here, only to move on. It is quite evident, therefore, that there are great opportunities for the dissemination of vermin by a nomadic population." According to a letter to Briggs from Mark F. Boyd, ticks collected some years previously in the vicinity of Lake Tahoe (vicinity of Briggs's cases) in northern California were sent by Boyd to Nathan Banks, United States National Museum and were identified as *Ornithodoros talaje* (Guérin Méneville), a species known to be a vector of relapsing fever in Panama. This was evidently an incorrect identification since diligent search for that species in this neighborhood proved futile. However, in searching for a vector during the summers of 1931–1933, an *Ornithodoros* tick, new to science, was discovered by Wheeler (1935) and was given the name *Ornithodoros hermsi* Wheeler.[85] A re-examination of one of the ticks sent by Boyd to Banks proved it to be a specimen of the new species described by Wheeler.

It is of interest to note that prospectors and others who worked in the Sierra Nevada at altitudes of 5,000 feet and over frequently reported suffering from a malaria-like disease which they called "squirrel fever." In the light of the discoveries described here and particularly the experience of C. M. Wheeler noted below, it is obvious that this infection was relapsing fever, infection having been due to contact with the blood of spirochete-infected squirrels or due to tick bites not then suspected.

Porter, Beck, and Stevens[86] state that, since the cases reported by Briggs, no further cases of relapsing fever came to the attention of the California State Department of Public Health until September, 1930, when Dr. George Stevens reported a case of a school teacher who had lived at Big Bear Lake (San Bernardino County) during July and August. In 1930 Major V. H. Cornell reported a case in which infection had been contracted at Lake Tahoe (Eldorado County) during July. In June, 1932, C. M. Wheeler, an assistant on field duty, contracted relapsing fever at Packer Lake (Sierra County) by accidentally smearing his fingers with blood from a Sierra chickaree squirrel (*Sciurus douglasii*) which with four others he had shot a few minutes previously.[87] The blood from the squirrels proved positive for spirochetes (*Spirochaeta recurrentis* Lebert). Seven days after the accident Wheeler came down with relapsing fever.

On August 12, 1931, three specimens of mature, undescribed *Ornithodoros* ticks were taken in a cottage near Brockway on Lake Tahoe (Eldorado County) at an elevation of approximately 6,000 feet, where cases of relapsing fever had occurred about a month previously. In August, 1934, more ticks of the same undescribed species were collected at Big Bear Lake at an elevation of about 5,700 feet in a cabin where there had been cases of relapsing fever. Other specimens were taken in various localities of the three counties already named (Eldorado, Placer, and San Bernardino) at elevations of from 5,000 to 8,000 feet. This new species of tick was named *Ornithodoros hermsi* by Wheeler (*loc. cit.*)

in 1935, and at the same time Wheeler, Herms, and Meyer[88] reported transmissions to a monkey and white mice by the bite of this tick.

The first demonstration of the vector capacity of this new species was made in October, 1934, on a monkey bitten by ticks collected in an endemic area. (Wheeler, Herms, and Meyer.) The monkey showed the first characteristic symptoms 16 days after being bitten by the ticks. Proof that this species is capable of transmitting the infection to human beings was secured by Wheeler[89] in May, 1937, when an experimental infection resulted in a series of tests made on human subjects. From 1921 to 1944, inclusive, 283 cases of relapsing fever were reported in California, with epidemiological evidence pointing to their origin in the Sierra Nevada at elevations of 5,000 to 10,000 feet. The same hitherto undescribed tick was collected in significant numbers in the vicinity of Big Bear Lake in southern California (San Bernardino County) at an elevation of 6,700 feet during 1933 and 1934. This tick, *Ornithodoros hermsi,* has since been taken in all endemic areas as high as 10,000 feet elevation.

It should be added here that a more northerly focus of human relapsing fever was reported in 1940 at the foot of Mount Lassen, Manzanita Lake (California), elevation about 5,850 feet. Clinical human cases were recognized, and ticks *Ornithodoros hermsi* taken in chipmunks' nests in the infected cabin proved positive when allowed to bite white mice. Ticks of this species have now been taken as far north as Kamloops, British Columbia.

Weller and Graham[90] reported infected ticks in central Texas in 1930; they found a cave in the Colorado River Valley of central Texas which was "literally alive with ticks, a handful of sand yielding thirty or forty of different sizes." The cave, it was reported, is "frequented by goats and sheep, also probably wild animals such as bats, foxes, skunks and rabbits." Some of the ticks were applied to three rabbits, allowed to feed for 15 minutes, and then crushed and rubbed into the abrasions. Spirochetes were later observed in the blood of the rabbits.

Ornithodoros hermsi Wheeler (Fig. 161), the vector of relapsing fever in California, transmits the infection by the *bite* of both male and female ticks and in all stages of development. The proportion of infective larvae in hereditary transmission appears not to exceed one per cent. Wheeler[91] reports that from 35 per cent to 48 per cent of noninfective ticks when allowed to feed as larvae on infected laboratory white mice were able to acquire the spirochetes and transmit them to normal animals in some one or all of the subsequent developmental stages. One female tick has caused four infections in white mice during a period of about four months. Larvae from a presumably noninfective female tick produced infections, and conversely the larvae of an infective female may not be infective although occasionally an infection may result from bites of later stages. The spirochetes are usually present in the blood of white mice about five days after the animals have been bitten by an infective tick. The time elapsing before infected ticks transmitted the infection depended, of course, on the time when the ticks were ready to feed again with molts intervening; this elapsed period was 15 to 20 days at the shortest.

The life history of *Ornithodoros hermsi* Wheeler (Fig. 160) as described by Herms and Wheeler[79] is as follows: The very tiny light amber-colored eggs are deposited at intervals in batches of 12 to 140 from May to October and range well over 200 per female. A specific example fol-

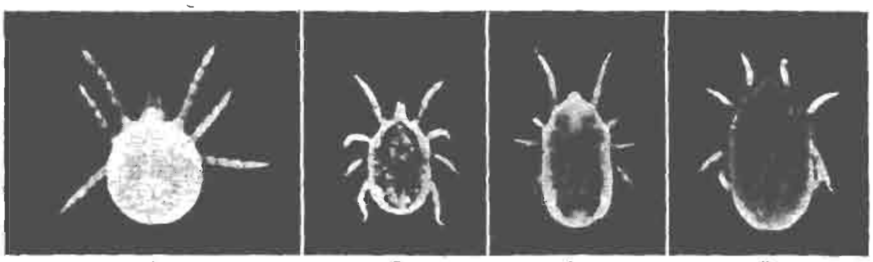


FIG. 160. Immature stages of *Ornithodorus hermsi*, (*A*) larva, (*B*) first nymphal stage, (*C*) second nymphal stage, and (*D*) third nymphal stage.

lows: Female tick, no. 5, taken as a last-stage nymph August 17, 1934, deposited a total of 232 eggs in four batches, 98 (April 8), 73 (May 19), 49 (May 26), and 12 (June 12) with but one feeding (i.e., after first batch was laid) between egg-layings, and died October 21, 1935. Under natural conditions the eggs are deposited in the hiding places of the ticks: in summer cabins (Fig. 162) the eggs are laid in such corners and crevices as afford protection to the ticks.

The incubation period at a temperature of 75° F and 90 per cent humidity ranges from 15 to 21 days. The number of eggs and the percentage of larvae hatching seems to grow less in the later egg layings, decreasing from as high as 95 per cent for the first batches to less than 50 per cent in the last. The first molt is usually accomplished within the egg; however, the larva (seed tick) remains hexapod until after the second molt. After about three days the larvae is ready to feed, remaining attached to the host for only about 12 to 15 minutes as is the case in later stages, although this attachment in the latter may be for from one-half to one hour in many cases. The larvae when fully engorged increase as much as three times in size and acquire a bright red color due to the imbibed blood. In this condition these tiny ticks have been referred to as a "strawberry seed insect" by persons living in relapsing fever areas. Molting takes place in about 15 days after feeding. With this molt the fourth pair of legs appears, and this stage is termed the first nymphal instar. Ticks in the first nymphal stage may feed within a few hours after molting, and again a period of 11 to 15 days elapses before the third molt and the appearance of the second nymphal instar. Then follows the third feeding and again an elapsed period, in this case about

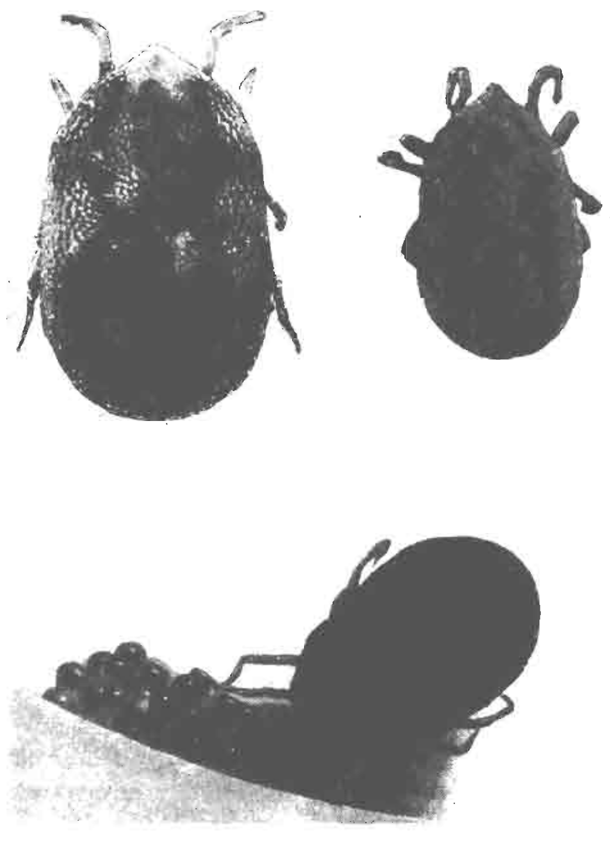


FIG. 161. *Ornithodoros hermsi.* (*Top left*) mature female, (*top right*) mature male, (*bottom*) female despositing eggs.

10 to 32 days, before the fourth molt and the appearance of the third nymphal instar, or even the adult may appear with this molt. Usually a fifth feeding and a fifth molt are necessary before sexual maturity is reached. Egg laying may begin in about 30 days after the last molt, fecundation taking place in a few days after maturity is reached. The cycle from egg to egg under our laboratory conditions required about

four months—e.g., from April 29, when eggs were laid, to August 24, when eggs were laid by a female from the April 29 hatch of eggs.

The life cycle may be greatly prolonged in the absence of food because of the ability of these ticks to withstand starvation; thus larvae may live as long as 95 days without food; unfed first-molt nymphs may live as long as 154 days; unfed second-stage nymphs may live as long as 79 days; third stage, as long as 109 days, and adults well over 7 months. Adult ticks have been kept alive in pill boxes with occasional feedings for a period of over four years.

The mature female tick (Fig. 161) measures from 5 to 6 mm in length by 3 to 4 mm in width. The male resembles the female closely in general appearance but is slightly smaller. This species is described as ovoid, conically pointed anteriorly, broadly rounded posteriorly. The anterior dorsal portion of the hood is visible from above. Unengorged specimens are of a light sandy color with the black of the intestinal diverticulae visible through the integument of the dorsal surface; freshly engorged specimens are of a dull, deep garnet shade with a grayish sheen over the body. The anterior conical point is whitish. The color changes to a grayish blue a few days after feeding. Legs and hood are pale yellow. In newly molted forms the body and legs are lighter but gradually assume the light sandy appearance and the leg darken correspondingly.

Tick vectors in various parts of the world. The known vectors of relapsing fever in the United States[92] are *Ornithodoros turicata* (Duges) in Texas and Kansas, and *O. hermsi* Wheeler in California, Colorado, and Idaho. *O. talaje* (Guérin-Méneville) has been collected in Arizona and California, and also *O. parkeri* Cooley,[93] the only known species in a large area from which a number of cases of relapsing fever have been reported; however, the latter species has not been adequately tested on humans.

In Mexico the vector is *Ornithodoros turicata* (Duges). In Central America *O. talaje* (Guérin-Méneville) and *O. venezuelensis* Brumpt are vectors; in South America, *O. venezuelensis* and *O. talaje;* in Africa, *O. moubata* (Murray), *O. savignyi* (Audouin), and *O. erraticus* Lucas; in Asia, *O. asperus* Warburton, *O. tartakovskyi* Olenev, and *O. tholozani* (Laboulbène and Mégnin) (= *O. papillipes* Birula; in Europe, *O. verricosus* Olenev Sassuchin and Fenik and *O. erraticus* Lucas (= *O. moracanus* Velu).

Spirochetes of endemic relapsing fever. The causal organism of tick-borne Central African relapsing fever was named *Spirochaeta duttoni* by Novy and Knapp in 1906. The South and Central American strain was called *Spirochaeta venezuelense* by Brumpt in 1921, and the Texas, U.S.A., strain was called *Spirochaeta turicatae* by Brumpt in 1933.

Other strains, such as *S. kochi* Novy, 1907, *S. hispanica* Sadi de Buen, 1926, *S. persica* Dschunkowsky, 1912, and *S. neotropicalis* Bates and St. John, Central and South America, have been recognized.

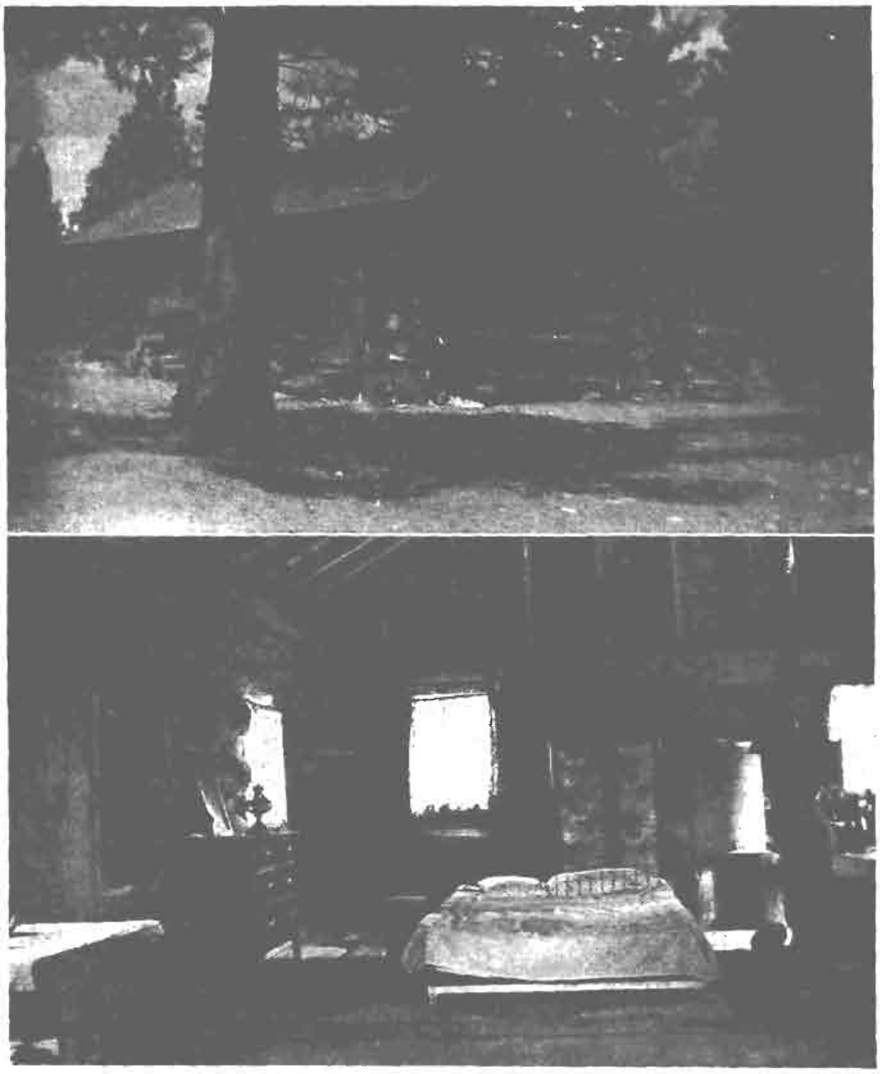


FIG. 162. (*Top*) a Sierrian summer cabin in which relapsing fever was contracted. (*Bottom*) interior view of the cottage.

That these are probably all strains or local varieties of one widely distributed species, *Borrelia* (*Spirochaeta*) *recurrentis* Lebert, is believed to be the case by various authors. Students should consult Publication

No. 18 (1942), American Association for the Advancement of Science, entitled *"A Symposium on Relapsing Fever in the Americas."*

Porter, Beck, and Stevens (*loc. cit.*, 1932) report finding spirochetes in blood smears from a chipmunk shot near Polaris, the location of the first endemic case reported by Briggs (1921), and also from a squirrel killed at Packer Lake, another endemic focus. Beck (1937) described three of the California foci (Big Bear Lake, Lake Tahoe, and Packer Lake) and gives an account of the rodent population in each. The results of her laboratory inoculations of white mice indicate that of a total of 183 inoculated, 13 were found to be infected with spirochetes resembling *Borrelia* (= *Spirochaeta*) *recurrentis.* Herms and Wheeler (*loc. cit.*, 1936) proved this spirochete to be the causal organism of relapsing fever in man. Only chipmunks and Tamarack squirrels were discovered harboring this spirochete in these areas. Three varieties of chipmunks are

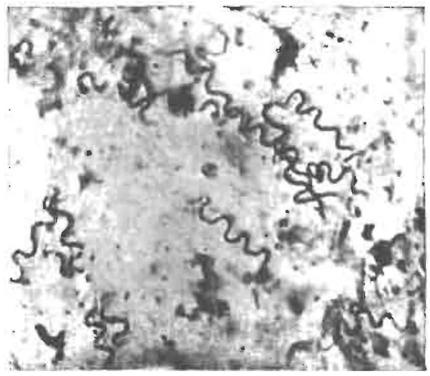


FIG. 163. Spirochetes of relapsing fever in a blood smear.

listed as hosts, mainly *Eutamias quadrimaculatus;* the other principal host of the spirochete is listed as the Tamarack squirrel or Sierra chickaree, *Sciurus douglassi albolimbatus.* Beck reports 13 rodent strains and 6 human strains, those of the chipmunk being most virulent.

The spirochetes of relapsing fever in man and rodents in California (Fig. 163) have been carefully studied by Miss M. Dorothy Beck.[94] She reports 13 strains of spirochetes resembling *Borrelia* (= *Treponema*) *recurrentis* from rodents in the field. Only chipmunks (*Eutamias* sp.) and Tamarack squirrels or Sierra Nevada chickarees (*Sciurus douglasii albolimbatus* Allen) were found to harbor spirochetes in the districts surveyed. She reports long periods of latency in mice, up to 114 days. She also found that the spirochetes of both human and animal origin show remarkable resistance to freezing and remain viable in defibrinated sheep blood for at lease 195 days. She concludes that the rodent and human strains are identical in morphology and that laboratory animals show a similar susceptibility. "These strains are undoubtedly the same since the rodent strains are directly transmissible to man."

The spirochetes show a high degree of tick-host specificity. Based on experimental evidence, Davis[95] proposes the name *Borrelia* (*Spirochaeta*) *hermsi* for the infection transmitted by *Ornithodoros hermsi* and *Borrelia parkeri* for the infection transmitted by *O. parkeri*, a species which resembles *O. hermsi* very closely and an experimental vector of spirochetosis. Although *O. parkeri* bites man freely and may be a vector of relapsing fever to man, though experimental evidence to that effect is lacking, its elevational range is below the endemic areas of relapsing fever, and its wild host relationships seem to differ. Nevertheless one human case is attributed to *O. parkeri* by Davis, Wynns, and Beck[96] (1941) at an elevation of about 250 feet, far below the usual elevation of known endemic areas.

Eradication of the infection from summer cabins (Fig. 162) known to harbor the infection because of the origin of cases in the cabins is rendered somewhat difficult because the manner of their construction permits chipmunks and smaller rodents to gain entrance and establish nests inside or between walls. By driving these animals out and keeping them out by securely closing all crevices, knotholes, etc., to prevent their return is good practice; however, it must be remembered that this deprives the ticks of their natural food, and they will turn avidly to the human occupants for blood. These ticks are also extremely "hardy" and may go without food for many months and continue to harbor the causal organisms (spirochetes) of relapsing fever so that a long vacancy does not ensure freedom from ticks; they do not starve easily.

Some of the measures useful in the control of ticks and of chipmunks and other small animals infesting summer cottages are the following: (1) When a new cottage is being built, it should be rodent-proofed—this is a simple and inexpensive procedure known to all carpenters in such areas; (2) all crevices, knotholes, and other points of ingress should be closed; all doors should be tight fitting, and the chimney should be covered with wire netting to keep out bats and rodents; (3) yards, woodpiles, etc., should be cleaned up and kept in order to prevent chipmunks and rodents from concealing themselves and building their nests; (4) rodent reproduction should not be encouraged by feeding and making pets of these animals; the rodent population should be kept at a low level; (5) old tree snags in the vicinity which may harbor nesting squirrels and chipmunks should be destroyed; nests in such snags have been found to harbor numerous relapsing fever ticks in all stages of development; (6) firewood covered with loose bark beneath which ticks may be concealed should not be brought into the house or woodshed; (7) although only the younger stages of the ticks are certainly affected, it is of value to apply a 10 per cent DDT dust to all surfaces inside the cabin making sure that there is a visible layer of the dust throughout;

treatments early in the summer and again in the autumn are recommended; (8) a 5 per cent solution of residual DDT spray should be applied thoroughly to beds, mattresses, rugs, under rugs, on walls and floors, under tables and chairs, in closets, porches, cracks in the walls and along baseboards, etc. (food and dishes should be protected from the DDT powder and spray); (9) in an area-wide program of relapsing fever control, the elimination of breeding niches for chipmunks (and other rodents) by complete destruction by fire (or other means) of "snags," partly dead or dead trees is strongly urged—this is an important aspect of forest sanitation in recreation areas.

Genus Otobius. In adults the integument is granulated; nymphs have spines. The sexes are similar. The capitulum is distant from the anterior margin in adults and near the margin in nymphs. The hood and eyes are absent; the hypostome is vestigial in adults but well developed in nymphs. Cooley and Kohls recognize two species *O. megnini* (Dugès)

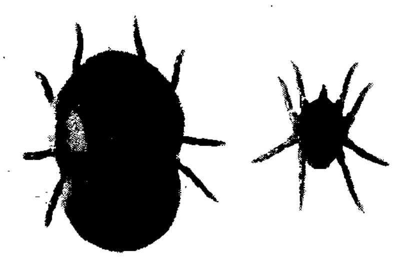


FIG. 164. Spinose ear tick, *Otobius megnini.* ×3.5.

and *O. lagophilus* Cooley and Kohls,[97] the latter a parasite on the face of rabbits in western United States and Canada.

The spinose ear tick, *Otobius megnini* (Dugès), is a widely distributed species, being found in warmer parts of the United States, South America, South Africa, India, and no doubt other parts of the world. It receives its name from the fact that the nymph is covered with spines (Fig. 164), and in all stages the tick invades the ears of cattle, horses, mules, sheep, cats, dogs, other domesticated animals, and man, as well as deer, coyotes, rabbits, and other wild animals. Rather large dark eggs are deposited by this species on the ground; under laboratory conditions at a temperature of about 21° C the incubation period is from 18 to 23 days. In the field newly emerged larvae crawl up weeds and other vegetation like the larvae of other ticks, coming in contact with suitable host animals such as cattle and gradually working their way to the shoulders, neck, and head, and thence to the deeper inner folds of the outer ear of the host, where they assume a peculiar sac-like form. After molting in the ear the nymphs attach themselves and remain attached for long periods of time; this, the second nymphal stage, is the stage in which this species is most easily distinguished from other ticks. Individual ticks may remain in the ear as long as 121 days, as observed in our tests. On detaching they crawl out of the ear, drop to the ground and molt again (there are three molts), after which maturity is reached. Copulation takes place within a day or two after the final molt, and oviposition occurs in from 14 to 42 days, with a maximum oviposition period of 155 days in the individuals observed, during which time 562 eggs were laid.

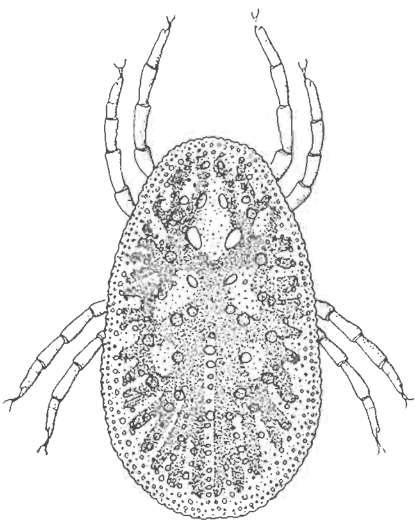


FIG. 165. The poultry tick, *Argas persicus*, dorsal view.

The longevity of unfed larvae at room temperature ranged from 19 to 63 days, with an average of 44 days.[98]

Damage done. The writer has received many complaints from various cattle-grazing districts in California relative to the "ear tick." Ears are occasionally sent in thoroughly infested with these pests in all stages. It is commonly asserted that this tick is responsible for much deafness in domesticated animals, and it is also believed to be responsible for illness and even death, particularly in calves.

Treatment. Owing to the position occupied by the ticks on the host, only local treatment is of any avail. A mixture of two parts of pine tar to one part of cottonseed oil, injected with a warm metal syringe about ½ ounce in each ear (cattle and horses), is recommended by Imes.[99] The ear should be manipulated during the injection. This mixture kills the ticks and affords protection against the invasion of others for about thirty days. Another mixture for similar treatment is as follows: benzene hexachloride (15 per cent gamma isomer) 5 per cent, xylol 10 per cent, pure pine oil, q.s. ad. 100 per cent.

Genus Argas. The members of this genus are distinctly flattened, with the margins quite thin even when the tick is fully engorged. The integument is leathery, minutely wrinkled in folds, often intermingled with small, rounded "buttons," each with a pit on top and often bearing a hair in the pit. Eyes are absent; sexes are similar. There are only two species in North America.

Argas persicus (Oken) (*Argas miniatus* Koch, *Argas americanus* Packard), a cosmopolitan *fowl tick,* is one of the most important poultry parasites in existence (Fig. 165). In addition to "fowl tick," this pest is commonly called "adobe tick," "tampan," or "blue bug." In color it varies from a light reddish brown to a dark brown, depending on the stage of engorgement. In size the obovate, flattened adults average about 8.5 mm long by 5.5 mm wide in the female, and 6.5 mm long by 4.5 mm wide in the male. When unengorged their thickness is about .75 mm and when fully engorged may be nearly 3 mm at the thickest part. The marginal border is always distinct even when the tick is engorged. The sexes are not easily distinguishable; the males are smaller, and though they may be as large as smaller female individuals, they taper slightly more anteriorly, i.e., are more obovate. The genital orifice of the male is "half-moon–shaped," while in the female it is "slit-like" and situated farther forward, i.e., immediately behind the capitulum as in other argasines. The capitulum has four long hairs, two hypostomal, and one near the articulation of each palp, all directed forward. The palpi are about twice as long as the hypostome, second article longest, the others equal in length. The hypostome, apically rounded, has six or seven fine denticles on each half distally, followed by stout teeth 2/2, the numbers increasing to 3/3, 4/4, 5/5, basally, the teeth decreasing in size, neither attaining the external border nor extending beyond half the length of the hypostome (Nuttall).

Life history and habits. The nymphs and adults of *Argas persicus* (Oken) are strikingly active at night, migrating long distances to find their host, and hiding in an inactive condition during the day. The writer has observed this pest in vast numbers hiding beneath the loose bark of the eucalyptus tree in California. Occasionally specimens are sent in with the inquiry, "Are they parasites of the tree or do they attack roosting chickens? The chickens seem to do very poorly, yet we find nothing on them." At night if one observes somewhat closely, one may see hordes of these ticks climbing up the sides of the chicken coop to the roosts and upon the fowls, filling up leisurely with blood and before daybreak departing for their hiding places. The females deposit their large reddish-brown eggs in the crevices occupied during the day. The eggs are laid in masses of from 25 to 100 and over, and there are usually several layings, each preceded by a meal of blood, with a total of seven hundred eggs per female. Egg deposition takes place in almost any sort of receptacle in which the ticks may be kept for observation. Hatching takes place in from 10 days to 3 or 4 weeks. The larvae (Fig. 166) are hexapod and very active, attacking a host apparently as readily by day as by night. Once attached the larvae feed for about five days, occasionally longer, remaining firmly attached during this time. When fully engorged they appear like little reddish globules, causing severe irritation. At the end of this feeding period the larvae detach themselves, having become rather flattened in the meantime, and then crawl away from the host, hiding in some convenient crevice near by. The larvae molt in about a week, when the fourth pair of legs appear and they are now in

FIG. 166. Larva of the poultry tick, *Argas persicus*. ×30.

the first nymphal stage, looking like miniature adults. Nocturnal feeding now takes place, and in 10 or 12 days another molt occurs and the second nymphal stage is reached. Again the tick attaches itself, being now able to engorge itself in about an hour; again, after the expiration of something over a week, a third molt takes place (there may even be a fourth molt) and then the adult stage is reached. The adults are able to engorge themselves in from 20 to 45 minutes. Under favorable conditions the adult stage is reached in about 30 days. Absence of hosts to feed upon may greatly prolong the life history.

Since eggs are deposited mainly during July in California, the adult stage may or may not be reached before the rainy season begins, and the overwintering stage may be in the second nymphal condition or as adults, appearing in pestiferous numbers early during the following summer. Hence there is ordinarily one generation of ticks per year under normal conditions. In the absence of a host this species can live more than two years without food.

The species will bite man. Instances are recorded in which transient laborers occupying long-vacated but renovated poultry houses have been badly bitten by the poultry tick. It might perhaps under certain circumstances become involved in the transmission of human spirochetosis.

Damage done. Each tick when engorging requires considerable blood to become replete, hence, when myriads of these parasites attack fowls great quantities of blood must be extracted. The writer has known of chickens being picked up under the roost in the morning with no apparent cause for death, and believes this to have been due directly to the work of ticks. Weakened and unthrifty condition of a flock may be traceable solely to ticks. Poultry suffering from ticks have dull, ragged plumage, suffer from diarrhea, are weak, and lay poorly. Turkeys are most likely to suffer.

Avian spirochetosis. A very dangerous disease, known as "fowl spirochetosis," is traceable to *Borrelia* (*Spirochaeta*) *gallinarum* Blanchard (*Spirochaeta marchouxi* Nuttall), occurring in India, Australia, Brazil, Egypt, and Persia, and is no doubt very widely distributed. The disease attacks chickens, geese, turkeys, guinea fowls, and other birds. The symptoms are described as follows:

> The disease begins with diarrhea, followed by loss of appetite, the birds appearing somnolent; the feathers being ruffled and the comb pale. The birds cease to perch, lie down with the head resting upon the ground and death takes place during a convulsive attack. At times the disease runs a slower course, the legs become paralyzed, then the wings, and the bird grows thin and dies in eight to fifteen days. Recovery may take place, but it is rare after paralytic symptoms have appeared. At autopsy, during the acute period of the disease, the spleen appears much enlarged and the liver swollen with more or less fatty degeneration; at times the liver is dotted with focal necroses. In chronic cases both these organs may appear atrophied. The blood is fluid and dark. Spirochaetes are plentiful in the blood until shortly before death, and they disappear as recovery sets in (Nuttall).

Argas persicus (Oken) was proved to be a vector of this spirochete infection by Marchoux and Salimbeni, Balfour, Nuttall, and others. These investigators have found that when the tick sucks blood from an infected fowl the spirochetes multiply within the body of the tick when it is kept at from 30° to 35° C, and it is capable of transmitting the disease; but when it is kept at from 15° to 20° C it fails to do so. However, if the tick is later kept at the higher temperature it becomes infective. The spirochetes are transmitted by the bite and by fecal contamination; the tick is said to be infective for six months or more. The infection is carried over from one generation of ticks to the next through the egg. The incubation period in the fowl is from four to nine days. Recovery from the disease is followed by immunity.

Seddon[100] reported several mild cases of avian spirochetosis in Australia (N.S.W.) in 1926. He states that the mildness of the attack may be attributed to the fact that the cases had occurred during the winter when the cold weather would inhibit the activity of the ticks. The disease is now commonly found in the southern parts of New South Wales and in several other parts of Australia.

Combating the fowl tick. Henhouse roosts should be painted thoroughly with kerosene or a solution of creosote and put in position with the ends in cups of crude oil or creosote or embedded in oil-soaked waste, or suspended by wires from the ceiling. Roost poles must be free from bark. All old nests and rubbish should be burned, and the interior, especially crevices, sprayed liberally with kerosene. Boiling water or steam may be used instead of kerosene emulsion. A repetition of the procedure once every five or six weeks during the tick season is recommended. The use of considerable crude oil or creosote oil in and about the houses is very desirable. Fowls should not be permitted to roost in trees, because of the hiding places afforded the ticks beneath the bark, particularly when this is loose.

If the henhouses can be made tight, fumigation with sulphur is useful; about five pounds per 1,000 cubic feet of space should be employed.

For the treatment of fowls infested with larval ticks, an ointment of kerosene, lard, and sulphur is advised.

Argas reflexus (Fabr.), commonly known as the "pigeon tick," differs from *A. persicus* (Oken) in that the body narrows rather suddenly toward the anterior end and that the thin margin is flexed upward. The capitulum has "two long post-hypostomal hairs ventrally, directed forwards. Palps with articles sub-equal, the third the shortest, denticulated hairs dorsally. . . . Hypostome apically notched, some small denticles at the tip, followed by 2/2 stout teeth merging into 3/3 to 6/6 progressively smaller teeth" (Nuttall).

Other species of Argas are the following: *A. brumpti* Neumann, the largest known species of the genus, measuring 15–20 mm in length by 10 mm in width. It is known to attack man in Africa. *A. vespertilionis* (Latreille) occasionally attacks man in Africa. *A. mianensis* Brumpt occurs in human habitations in Iran (Mianeh) where it is believed to be a vector of a spirochete infection in man known as Mianeh fever.

Genus Antricola. The dorsal walls are flattened and marginated; below the flattened dorsum the body is convex and deep; the integument is semitranslucent and the surface smooth, shining, and with tubercles; the mouth parts are adapted for quick feeding and not for clinging to the host.

Antricola coprophilus (McIntosh) feeds on bats in Arizona and Mexico.

Antricola marginatus (Banks) is found in bat caves in Cuba and other parts of the West Indies.

BIBLIOGRAPHY

1. Hunter, W. D., and Hooker, W. A., 1907. *Information Concerning the North American Fever Tick*. Washington, D. C.: Dept. Agric., in Bur. Entomol. Bull., no. 72. 87 pp.

2. Woodward, T. E., and Turner, W. F., 1915. *The Effect of the Cattle Tick upon the Milk Production of Dairy Cows*. Washington, D. C.: Dept. Agric., in Bur. Animal Indust., Bull., no. 147. 22 pp.

3. Jellison, W. L., and Kohls, G. M., 1938. "Tick-host anemia: a secondary anemia induced by *Dermacentor andersoni* Stiles," *J. Parasitol.*, 24:143–54.

4. Cooley, R. A., 1932. *The Rocky Mountain Wood Tick*. Bozeman: Montana State Coll., in Agric. Exper. Sta., Bull., no. 268. 58 pp.

5. Nuttall, G. H. F., and Warburton, Cecil, 1908. *Ticks, a Monograph of the Ixodoidea. Part I, Argasidae.* x + 104 + 35 pp. *Part II, Ixodidae.* xix + 105 + 348 pp. London: Cambridge Univ. Press.

6. Graybill, H. W., 1911. *Studies on the Biology of the Texas Fever Tick*. Washington, D. C., Dept. Agric., in Bur. Animal Indust., Bull., no. 130. 42 pp.

7. Cooley, R. A., 1938. *The genera Dermacentor and Otocentor (Ixodidae) in the United States with Studies in Variation*. Washington, D. C.: U. S. Public Health Service, in Nat. Inst. Health Bull., no. 171. v + 89 pp. (30 plates).

8. ———, and Kohls, Glenn, M., 1943. "*Ixodes californicus* Banks, 1904, *Ixodes pacificus* n. sp., and *Ixodes conepati* n. sp." *Pan Pacific Entomologist*, 19:139–47 (*see also* errata same vol.).

9. Smith, Carroll N.; Cole, Moses M.; and Gouck, Harry K.; 1946. *Biology and Control of the American Dog Tick*. Washington, D. C.: Dept. Agric., in "Tech. Bull.," no. 905. 74 pp.

10. Parker, R. R.; Philip, C. B.; Davis, G. E.; and Cooley, R. A.; 1937 "Ticks of the United States in relation to disease in man," *J. Econ. Entomol.*, 30:51–69.

11. Cooley, R. A., 1932. *The Rocky Mountain Wood Tick*. Bozeman: Montana State Coll., in Agric. Exper. Sta., Bull., no. 268. 58 pp.

12. Hunter, W. D., and Bishopp, F. C., 1911. *The Rocky Mountain Spotted Fever Tick*. Washington, D. C.: Dept. Agric., in Bur. Entomol. Bull., no. 105. 47 pp.

13. Douglas, J. R., 1943. "The internal anatomy of *Dermacentor andersoni* Stiles," *Univ. Calif. Publ. in Entomol.*, 7:207–72, plates 8–26, 7 text figures.

14. Thomas, L. J., and Cahn, A. R., 1932. "A new disease of moose. I.," *J. Parasitol.*, 18:219–31.

15. Cahn, A. R.; Wallace, G. O.; and Thomas, L. J.; 1932. "A new disease of moose. III," *Science*, 76:385–86.

16. McIntosh, Allen, 1931. "The brown dog tick," *North Am. Vet.*, 12:37–41.

17. Cooley, R. A., and Kohls, Glen M., 1944. "The Genus *Amblyomma* (Ixodidae) in the United States," *J. Parasitol.*, 30:77–111.

18. Tate, H. Douglas, 1941. "The biology of the tropical cattle tick and other species of ticks in Puerto Rico, with notes on the effects on ticks of arsenical dips," Univ. of Puerto Rico, *J. Agric.*, 25:1–24.

19. State Crop Pest Commission of Louisiana, 1906. *The Cattle Tick.* Circ. no. 10.

20. Mohler, John R., 1930. *Tick Fever.* Washington, D. C.: Dept. Agric., in "Farmers' Bull.," no. 1625.

21. Smith, T., and Kilbourne, F. L., 1893. *Investigations into the Nature, Causation, and Prevention of Texas or Southern Cattle Fever.* Washington, D. C.: Dept. of Agric., in Bur. Animal Indust., Bull., no. 1. 301 pp.

22. Rees, C. W., 1934. "Characteristics of the Piroplasms, *Babesia argentina* and *B. bigemina* in the United States," *J. Agric. Research,* 48:427–38.

23. Dennis, E. W., 1932. "The life-cycle of *Babesia begimina* (Smith and Kilbourne) of Texas cattle-fever in the tick, *Margaropus annulatus* (Say)," *Univ. Calif. Publ. in Zool.,* 36:263–98 (6 plates).

24. Stiles, Ch. Wardell, 1905. *A Zoölogical Investigation into the Cause, Transmission and Source of Rocky Mountain "Spotted Fever."* Washington, D. C.: Public Health and Marine Hospital Service of the United States, in Hygienic Bull., no. 20. 121 pp.

25. Parker, R. R., 1938. "Rocky Mountain spotted fever," *J.A.M.A.,* 110:1185–88 and 1273–78.

26. Wolbach, S. B., 1919. "Studies on Rocky Mountain spotted fever." *J. Med. Research,* 41:1–197.

27. Wilson, Louis B., and Chowning, William M., 1902. "The so-called 'spotted fever' of the Rocky Mountains. A preliminary report to the Montana State Board of Health," *J.A.M.A.,* 39:131–36.

28. Hunter, W. D., and Bishopp, F. C., 1911. *The Rocky Mountain Spotted Fever Tick.* Washington, D. C.: Dept. Agric., in Bur. Entomol., Bull., no. 105. 47 pp.

29. Ricketts, H. T., 1906. "The transmission of Rocky Mountain spotted fever by the bite of the wood tick *(Dermacentor occidentalis),*" *J.A.M.A.,* 47: 358.

30. ———, 1911. *Contributions to Medical Science.* Chicago: University of Chicago Press. 497 pp. (See pp. 278–450.)

31. ———., 1906. "Observations on the virus and means of transmission of Rocky Mountain spotted fever," *J. Inf. Dis.,* 4:141–53.

32. Parker, R. R., 1923. "Transmission of Rocky Mountain spotted fever by the rabbit tick, *Haemaphysalis leporis-palustris* Packard," *Am. J. Trop. Med.,* 3:39–45.

33. Dyer, R. E.; Badger, L. F.; and Rumreich, A.; 1931. "Rocky Mountain spotted fever (eastern type) transmission by the American dog tick *(Dermacentor variabilis),*" U. S. Public Health Service, *Pub. Health Rep.,* 46:1403–13.

34. Parker, R. R., 1937. "Recent studies of tick-borne diseases made at the United States Public Health Service Laboratory at Hamilton, Montana," *Proc. Fifth Pacific Science Cong.,* pp. 3367–74.

35. ———; Kohls, Glen M.; and Steinhaus, Edward A.; 1943. "Rocky Mountain Spotted Fever; spontaneous infection in the tick, *Amblyomma americanum*," U. S. Public Health Service, *Pub. Health Rep.*, 58:721–29.

36. ———, 1933. "Certain phases of the problem of Rocky Mountain spotted fever," *Arch. Path.*, 15:398–429.

37. ———, 1928. "Rocky Mountain spotted fever," Montana State Board of Entomol., *Seventh Biennial Rept.*, 1927–28, pp. 39–62.

38. Cooley, R. A., 1928. "Preliminary report on the tick parasite, *Ixodiphagus caucertei* du Buysson," Montana State Board of Entomol., *Seventh Biennial Rept.*, 1927–28, pp. 17–31.

39. Parker, R. R., 1935. "Rocky Mountain spotted fever: results of ten years' prophylactic vaccination," *J. Infect. Dis.*, 57:78–93.

40. Florio, Lloyd; Hammon, W. Mc.D.; Laurent, A.; and Stewart, M.; 1946. "Colorado tick fever and dengue," *J. Exper. Med.*, 83:295–300.

41. Parker, R. R., 1924. "Tularaemia. XI. Tularaemia infection in ticks of the species *Dermacentor andersoni* Stiles in the Bitter Root Valley, Montana," U. S. Public Health Service, *Pub. Health Rep.*, 39:1057–73.

42. ———, and Spencer, R. R., 1926. "Hereditary transmission of tularaemia infection by the wood tick, *Dermacentor andersoni* Stiles," U. S. Public Health Service, *Pub. Health Rep.*, 41:1403–07.

43. Davis, G. E., and Kohls, G. M., 1937. "*Ixodes ricinus californicus* (Banks) a possible vector of *Bacterium tularense*," U. S. Public Health Service, *Pub. Health Rep.*, 52:281–82.

44. Temple, J. U., 1912. "Acute ascending paralysis, or tick paralysis," *Med. Sentinel*, 20:507–14.

45. Hadwen, S., 1913. "On 'tick paralysis' in sheep and man following bites of *Dermacentor venustus*," *Parasitology*, 6:283–97, 2 plates.

46. ———, and Nuttall, G. H. F., 1913. "Experimental 'tick paralysis' in the dog," *Parasitology*, 6:298–301.

47. Ross, I. C., 1926. "An experimental study of tick paralysis in Australia," *Parasitology*, 18:410–29.

48. Woodland, John C.; McDowell, Mordecai M.; and Richards, John T.; 1943. "Bullis fever (Lone Star fever-tick fever)," *J.A.M.A.*, 122:1156–60.

49. Anigstein, Ludwick, and Bader, Madero N., 1943. "Investigations on rickettsial diseases in Texas. Part 4. Experimental study of Bullis fever," *Texas Rep. Biol. & Med.*, 1:389–409.

50. Derrick, E. H., 1937. " 'Q' fever. A new fever entity. Clinical features and laboratory investigation," *M. J. Australia*, 24:281–99.

51. Cox, H. R., 1939. "A filter-passing infectious agent isolated from tick. III. Description of organism and cultivation experiments," U. S. Public Health Service, *Pub. Health Rep.*, 53:2270–76.

52. Parker, R. R., and Davis, Gordon E., 1938. "A filter-passing infectious agent isolated from ticks. II. Transmission by *Dermacenter andersoni*," U. S. Public Health Service, *Pub. Health Rep.*, 53:2267–70.

53. Steinhaus, Edward A., 1946. *Insect Microbiology*. Ithaca: Comstock Publ. Co., Inc. x + 763 pp.

54. Parker, R. R., and Davis, Gordon E., 1943. "American Q fever: the occurrence of *Rickettsia diaporica* in *Amblyomma americanum* in eastern Texas," U. S. Public Health Service, *Pub. Health Rep.*, 54:1510–11.

55. Philip, Cornelius B., 1948. "Observations on experimental Q fever," *J. Parasitol.*, 34:457–64.

56. Huebner, R. J.; Jellison, W. L.; Beck, M. D.; Parker, R. R.; and Shepard, C. C.; 1948. "Q fever studies in southern California," U. S. Public Health Service, *Pub. Health Rep.*, 63:214–22.

57. Shaughnessy, H. J., and Milzer, Albert, 1939. "Experimental infection of *Dermacentor andersoni* Stiles with the virus of lymphocytic choriomeningitis," *Am. J. Pub. Health*, 29:1103–8.

58. Henning, M. W., 1932. *Animal diseases of South Africa.* "South African Agriculture Series," 11:298–329.

59. Brumpt, E., et Larrousse, F., 1922. "Transmission de la Piroplasmose canine française par le *Dermacentor venustus*," *Bull. Soc. path. exot.*, 15:540–45.

60. ———, 1919. "Transmission de la piroplasmose canine tunisienne par le *Rhipicephalus sanguineus*," *Bull. Soc. path. exot.*, 12:757–64.

61. Sanders, D. A., 1937. "Observations on canine babesiasis (piroplasmosis)," *J. Am. Vet. M. A.*, 90, n.s. 43:27–40.

62. Henning, M. W., 1932. *Animal Diseases of South Africa.* "South *African Agricultural Series,*" 2:545.

63. Stiles, Geo. W., 1939. *Anaplasmosis in Cattle.* Washington, D. C.: Dept. Agric., in Circ., no. 154 (revised). 10 pp.

64. Herms, W. B., and Howell, D. E., 1936. "The western dog tick, *Dermacentor occidentalis* Neum., a vector of bovine anaplasmosis in California," *J. Parasitol.*, 22:283–88.

65. Howell, D. E.; Stiles, Geo. W.; and Moe, Lewis H.; 1941. "The hereditary transmission of anaplasmosis by *Dermacentor andersoni* Stiles," *Am. J. Vet. Research*, 11:165–66.

66. Boynton, W. H., and Woods, Gladys M., 1933. "Deer as carriers of anaplasmosis," *Science*, 78:559–60.

67. Ellenberger, W. P., and Chapin, R. M., 1940. *Cattle-fever Ticks and Methods of Eradication.* Washington, D. C.: Dept. Agric., in Farmers' Bull., no. 1057. 27 pp.

68. Bishopp, F. C., and Smith, C. N., 1938. *The American Dog Tick, Eastern Carrier of Rocky Mountain Spotted Fever.* Washington, D. C.: Dept. Agric. in Circ., no. 478. 26 pp.

69. Smith, Carroll N., and Gouck, Harry K., 1945. "DDT to control ticks on vegetation," *J. Econ. Entomol.*, 38:553–55.

70. Cooley, R. A., and Kohls, Glen M., 1944. *The Argasidae of North America, Central America, and Cuba.* Notre Dame: The University Press, in American Midland Naturalist. 152 pp.

71. Jobling, B., 1925. "A contribution to the biology of *Ornithodoros moubata* Murray," *Bull. Entomolog. Research*, 15:271–79 (1 plate).

72. Davis, Gordon E., 1947. "A note on the larval stage of the argasid tick, *Ornithodoros moubata* (Murray) 1877," *J. Parasitol.*, 33:495–96.

73. Aitken, T. H. G., 1939. "*Ornithodoros talaje* on the California mainland," *Pan-Pacific Entomologist,* 15:12–13.

74. Bates, L. B.; Dunn, L. H.; and St. John, J. H.; 1921. "Relapsing fever in Panama," *Am. J. Trop. Med.,* 1:183–210.

75. Matheson, R., 1931. "Note on the *Ornithodoros talaje* (Guer.-Men.)," *Parasitology,* 23:270.

76. Dunn, L. H., 1937. "Studies on the South American tick, *Ornithodoros venezuelensis* Brumpt in Colombia," *J. Parasitol.,* 13:249–59.

77. Hindle, E., 1935. "Relapsing fever: some recent advances," *Trop. Dis. Bull.,* 32:309–27.

78. Cooley, R. A., 1936. "*Ornithodoros parkeri,* a new species on rodents," U. S. Public Health Service, *Pub. Health Rep.,* 51:431–33.

79. Herms, W. B., and Wheeler, C. M., 1936. "*Ornithodoros hermsi* Wheeler as a vector of relapsing fever in California," *J. Parasitol.,* 22:276–82.

80. Ross, P. H., and Milne, A. D., 1904. "Tick fever," *Brit. M. J.* 2:1453–54.

81. Dutton, J. E., and Todd, J. L., 1905. *The Nature of Human Tick Fever in the Eastern Part of the Congo Free State, with Notes on the Distribution and Bionomics of the Tick.* Liverpool: School Trop. Med. Memoir, no. 17. 18 pp.

82. Feng, Lan-Chou, and Chung, Huei-Lan, 1936. "Studies on the development of *Spirochaeta duttoni in Ornithodoros moubata,*" *Chinese M. J.,* 50:1185–90.

83. Meador, Charles N., 1915. "Five cases of relapsing fever originating in Colorado, with positive blood findings in two," *Colorado Med.,* 12:365–68.

84. Briggs, LeR. H., 1922. "Relapsing fever in California," *J.A.M.A.,* 79:941–44.

85. Wheeler, C. M., 1935. "A new species of tick which is a vector of relapsing fever in California," *Am. J. Trop. Med.,* 15:435–38.

86. Porter, G. S.: Beck, M. Dorothy; and Stevens, I. M.; 1932. "Relapsing fever in California," *Am. J. Pub. Health,* 22:1136–40.

87. Herms, W. B., and Wheeler, C. M., 1935. "Tick transmissions of California relapsing fever," *J. Econ. Entomol.,* 28:846–55.

88. Wheeler, C. M.; Herms, W. B.; and Meyer, K. F.; 1935. "A new tick vector of relapsing fever in California," *Proc. Soc. Exper. Biol. & Med.,* 32:1290–92.

89. ———, 1938. "Relapsing fever in California—attempts to transmit spirochaetes of relapsing fever to human subjects by means of the bite of the vector, *Ornithodoros hermsi* Wheeler," *Am. J. Trop. Med.,* 18:641–59.

90. Weller, B., and Graham, G. M., 1930. "Relapsing fever in central Texas," *J.A.M.A.,* 95:1834–35.

91. Wheeler, C. M., 1938. Progress of spirochaete infection in the developmental stages of the host tick, *Ornithodoros hermsi* Wheeler. *Am. J. Trop. Med.,* 18:413–19.

92. Davis, Gordon E., 1940. "Ticks and relapsing fever in the United States," U. S. Public Health Service, *Pub. Health Rep.,* 55:2347–51.

93. ———, 1941. "*Ornithodoros parkeri* Cooley: observations on the biology of this tick," *J. Parasitol.*, 27:425–33.

94. Beck, M. Dorothy, 1937. "California field and laboratory studies on relapsing fever," *J. Infect. Dis.*, 60:64–80.

95. Davis, Gordon E., 1942. "Species unity or plurality of the relapsing fever spirochaetes," *A Symposium on Relapsing Fever in the Americas.* Washington, D. C.: Amer. Assoc. Advancement Sc., Publ. no. 18, pp. 41–47.

96. ———; Wynns, Harlin L.; and Beck, M. Dorothy; 1941. "Relapsing fever: *Ornithodoros parkeri* a vector in California," U. S. Public Health Service, *Pub. Health Rep.*, 50:2426–28.

97. Cooley, R. A., and Kohls, Glen M., 1940. "Two new species of Argasidae (Acarina: Ixodoidea)," U. S. Public Health Service, *Pub. Health Rep.*, 55:925–33.

98. Herms, W. B., 1917. "Contribution to the life history and habits of *Ornithodoros megnini*," *J. Econ. Entomol.*, 10:407–11.

99. Imes, Marion, 1918. *The Spinose Ear Tick.* Washington, D. C.: Dept. Agric., Bur. Animal Indust., in *Farmers' Bull.*, no. 980. 8 pp.

100. Seddon, H. R., 1926. "A note on spirochaetosis in fowls," New South Wales: Dept. of Agric., in Science Bull., no. 26, *Veterinary Res. Rept. no.* 2. pp. 17–19.

CHAPTER XXI

MITES

CLASS ARACHNIDA, ORDER ACARINA

Characteristics. In the mites, as in the ticks, the unsegmented abdomen is broadly joined to the cephalothorax with little or no evidence of separation. All but a few species are minute, i.e., just about visible to the naked eye. Mites, like other arachnids, have four pairs of legs as adults but only three pairs (exceptionally, fewer) in the larval stage. The mouth parts are quite varied but follow the general pattern of the ticks. The chelicerae in the parasitic species are piercing structures (see Snodgrass). One or more pairs of simple eyes are usually present. The respiratory system is in most species similar to that of the ticks, i.e., tracheal, while others absorb oxygen through the general body surface, which in these is quite soft. Nearly all species deposit eggs; however, there are a few which are ovoviviparous, among them, *Pediculoides ventricosus* (Newport). From the egg there emerges the hexapod larva, which usually soon molts and then presents its fourth pair of legs. The life history of many species requires less than four weeks; in some it is as short as eight days.

An infestation of mites is termed *acariasis*. Those species which burrow into the skin, producing channels in which their eggs are deposited are said to cause *sarcoptic acariasis*, e.g., *Sarcoptes scabiei* var. *hominis* (Hering) of human scabies; while those species which deposit their eggs at the base of the hairs of the host or on the skin, producing scabs are the cause of *psoroptic acariasis*, e.g., *Psoroptes communis* var. *ovis* (Hering) of sheep scab. Other forms of acariasis are described in this chapter.

Although Banks[1] recognized 29 families of mites in 1915, and Vitzthum[2] in 1943 recognized 80, actually only a few of these affect man and his domestic animals. Students concerned with the body of mites will need to consult the monumental work of Oudemans[3] (1937), also the excellent publications of Ewing, particularly his *Manual of External Parasites.*[4] Radford[5] has published a very helpful list of "Genera and species of parasitic mites," which gives the authority, year of validity, and, where possible, the original host.

MANGE, OR ITCH MITES–SARCOPTIC ACARIASIS

Family Sarcoptidae

Characteristics. All members of the family **Sarcoptidae,** commonly known as the itch mites, mange mites, or scab mites, are very small (just about visible to the naked eye), whitish, and somewhat hemispherical in form. Banks describes this family as follows:

> The body is entire, and the surface transversely striated and provided with a few bristles, often short, stout and sharp pointed. The legs are short and stout, arranged in two groups. The anterior legs are usually larger than the others. The tarsi commonly terminate in a stout claw. There is generally a long pedicellate sucker, sometimes with a jointed pedicel. The claw or sucker may be absent and in its place a long bristle. The legs often show a chitinous framework of rings, both transverse and oblique. On the front of the body is a prominent beak. The palpi are small, three-jointed, and appressed to the sides of the beak beneath. . . . There are frequently sexual differences. Some males have the third pair of legs very large and long, while the fourth pair is very small. Sometimes there are plate-like lobes at the tip of the male abdomen, and the tarsi may terminate differently in the two sexes.

The family **Sarcoptidae** includes a number of important genera, among them *Sarcoptes, Psoroptes, Notoedres, Chorioptes, Otodectes* and *Cnemidocoptes,* each producing a particular type of acariasis.

Sarcoptic mites. The mange or itch mites belong to the genus *Sarcoptes;* they have very short legs, the posterior pair not extending beyond the margin of the nearly circular body; suckers are present on the first and second pair of legs. The sarcoptic mites burrow in the skin, where they produce definite burrows in which the females deposit their eggs.

The species of *Sarcoptes* inhabiting the skin of mammals are ordinarily regarded as varieties of *Sarcoptes scabiei* (Linn.) (Fig. 167). They differ very slightly, and many of them may interchange hosts, e.g., *Sarcoptes scabiei* var. *suis* Gerlach, is parasitic on swine and may be temporarily parasitic on man; *Sarcoptes scabiei* var. *equi* Gerlach of the horse is also temporarily parasitic on man. The species appropriate to a given host, however, ordinarily exist only for a limited time on a different host species.

Human scabies or itch. The itch mite attacking man is known as *Sarcoptes scabiei* var. *hominis* (Hering). The infection is known as "scabies," "seven-year itch," or "Norwegian itch." It is universal in distribution. The female mite measures 330μ to 450μ in length and 250μ to 350μ in breadth; the male is slightly more than half as large. The mite attacks by preference the thin skin between the fingers, the bend of the knee and elbow, the penis, the breasts, and the shoulder blades, although any part of the body is subject to attack. An almost intolerable itching results due to toxic secretions and excretions. The sinuous burrows which the mite makes in the epidermis may reach 3 cm in length (Warburton[6]), and tiny vesicles and papules are formed on the surface. Scratching causes weeping and bleeding, which favor spread of the mites. It is also a means of secondary infection. Transmission is due to intimate personal contact such as hand shaking or sleeping with an infested person; clothing, towels, bedding, etc., are much less involved. Infection is usually effected by the adult fertilized female.[7] By tracing a mite burrow to its end with a hand lens, the adult female mite can usually be removed with a needle or scalpel and after treatment with

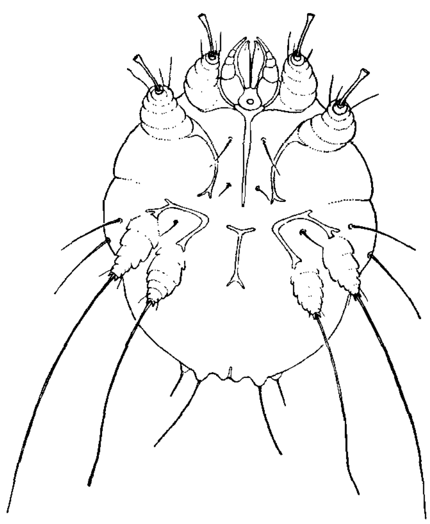


FIG. 167. *Sarcoptes scabiei.*

KOH on a glass slide can be readily seen under a microscope. Longstanding chronic cases show few parasites.

Life history of itch mite. The female mites deposit their rather large oval eggs ($150\mu \times 100\mu$) at intervals in the tortuous tunnels which they have made in the epidermis. From 10 to 25 eggs are deposited by each individual during a period of about two weeks. The female, having deposited her complement of eggs, dies at the end of her burrow. The hexapod larvae hatch in three or four days. In this stage the area of infection is most rapidly increased. Maturity is reached in 10 to 12 days, during which time there are three molts.

Treatment for human itch. Inasmuch as the mites are protected in their tunnels in the epidermis, the skin must be thoroughly softened by scrubbing vigorously with green soap and hot water before a remedy is applied. Sulphur ointments (10 to 15 per cent precipitated sulphur) give very good results if overnight applications are repeated at intervals of three or four days. Benzyl benzoate ointment is also highly recommended. Underclothing coming in contact with the parts affected should be boiled, steam sterilized, or baked.

Swine mange. Mange of swine is caused by *Sarcoptes scabiei* var. *suis* Gerlach (Fig. 168). Mange attacks swine commonly about the top of the neck, shoulders, ears, and withers, and along the back to the root of the tail. A microscopical examination of deeper tissue from beneath scabs will usually reveal the mites. Suckling pigs and young shoats suffer most. The affected animals scratch and rub vigorously, which may, however, be due to lice, but if the skin is cracked and thickly encrusted with heavy scabs, and the hair stands erect, an examination for scab mites

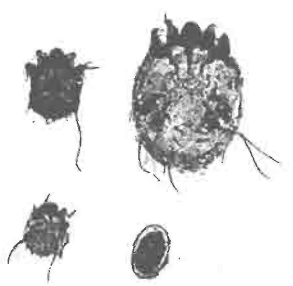


FIG. 168. Life history and general characteristics of a typical sarcoptic (mange or itch) mite: (*lower right*) egg, (*lower left*) larva, (*upper left*) male, (*upper right*) female. *Sarcoptes scabiei* var. *suis*, the itch or mange mite of swine. ×57.

should be made. The life history and habits of the swine mange mite correspond in every respect with those of the itch mite of humans.

Treatment for swine mange. In the treatment of swine mange it is necessary to apply external remedies, in addition to sanitary precautions to prevent spread and the reinfection of treated animals. Remedies are best applied in the form of solutions such as "lime and sulfur" dip because all parts of the animal's body are easily reached in the dipping process. Hand dressing, scrubbing, and application of ointments may be practiced where dipping is not practical; however, all parts of the animal should be thoroughly treated. A suitable dip may be made as follows: 8 pounds of fresh lime and 24 pounds of flowers of sulphur to 100 gallons of water, slaking the lime with sufficient water to form a thick paste, sifting in the sulphur, and mixing with a hoe. This mixture is placed in a kettle with 25 to 30 gallons of water and boiled for one hour at least, two hours being better. The entire mass is used for swine but must not be used for sheep. The dip is used warm at a temperature of from 100° to 110° F.[8]

Lime and sulphur dips may be purchased ready made and are commonly used at the rate of one part of the solution to 15 parts of water; however, care should be exercised to use the dip as directed, owing to variation in constituents. Coal-tar dips are also used extensively and are effective if given properly.

Dipping vats may be made of wood or concrete and are usually set in the ground at a slight elevation to ensure drainage away from the vat. A convenient size for a vat is "ten feet long on top, eight feet long on the bottom, one foot wide on the bottom and two feet wide at the top. The end where the hogs enter should be perpendicular and the other end inclined, with cleats, so that the hogs can emerge after passing through. The entrance should be by a slide. For pigs and small shoats that can be easily handled, a barrel serves the purpose well; the pigs can be caught, plunged in the dip and held there the required time. Some successful swine raisers build cement bathing places or wallows for swine and keep these filled with a watery solution of some dip or disinfecting solution." The time to treat young pigs, and this is important, is at weaning time. Dipping twice as for older animals is necessary, and if placed in uninfected quarters they ought to remain clean.

Mangy swine should be hand dressed with a stiff brush before dipping in order to loosen up scabs and then kept in the dip long enough to permit the solution to soak through the scabs, certainly not less than two minutes. All the animals must be dipped a second time in eight or ten days, in order to destroy the mites which have hatched from the eggs which are not destroyed.

Mayo[8] recommends a disinfecting whitewash to be applied to pens, etc., "Fresh lime, 25 pounds, flowers of sulphur, 15 pounds; mix the sulphur with a little water, to a paste, add 30 gallons of water and cook for an hour, then add water sufficient to make 50 gallons and apply with a spray pump, using a 'Bordeaux' nozzle."

Equine mange. Sarcoptic acariasis in horses, mules, and asses is caused by *Sarcoptes scabiei* var. *equi* Gerlach. This species is also transmissible to man and is said to be the chief cause of a transitory itch of cavalrymen and others handling horses extensively.

The most reliable diagnostic character is the discovery of the mite, as already explained. The usual symptoms are first of all a strong tendency to rub some circumscribed part such as the head, root of the mane or tail, withers, or back, due to an incessant itching. If a person scratches the affected parts, the animal moves its lips as though it were nibbling. The skin of these parts also shows an eruption of "fine conical papillae." The hair stands erect and bristly, much having dropped out, but totally bare spots where there are no isolated hairs apparently do not occur in mange but do in ringworm. The affected parts are at first scurfy, then become covered with yellowish scabs, which later exude matter due to the rubbing and inflammation, and finally there are formed scabs and crusts, often with deep crevices. During the first 14 days the progress of the disease is usually slow, but by the sixth week the ravages of the disease become extensive and there is rapid progress.

The life history and habits of *Sarcoptes scabiei* var. *equi* Gerlach correspond in every respect with those of the species already described.

Treatment for equine mange. Before applying a remedy for mange the animal should be clipped if possible so as to disclose all points of attack which might otherwise be hidden by hair. The clipped hair must not be blown away by the wind but should be burned. The parts affected are next thoroughly lathered and left for a short while to soften, after which warm water is applied and the scabs rubbed off as far as possible with wisps of hay or straw which should also be burned. The affected parts are now ready for a parasiticide, which should be applied by hand.

Various remedies may be obtained for mange, all of which have more or less value, but the writer has found that those containing sulphur are the most effective. Cameron,[9] Veterinary Director General, Health of Animals Division, Department of Agriculture, Dominion of Canada, prescribes for hand treatment of horses (and cattle as well, but not sheep) a mixture of sulphur, 2 pounds, oil of tar, 8 ounces, and raw linseed oil, 1 gallon. These ingredients are gradually heated together but not boiled. This dressing should be very thoroughly applied over the whole surface of the body and extremities of the animal, including the ears and tail, at a temperature not less than 110° F and not more than 120° F. Two dressings are necessary. Forty-eight hours before the second application (twelfth to fourteenth day inclusive) the first dressing should be washed off with hot water and castile soap. Ten days after the second application the animals should again be washed to remove the dressing. In severe cases it may be necessary to repeat the treatment many times.

For dipping animals Cameron (*loc. cit.*) uses a mixture of flowers of sulphur, 24 pounds; fresh unslaked lime, 10 pounds; and water, 100 gallons. The lime should be carefully slaked and made into a paste, and the sulphur added and thoroughly incorporated with the paste; the whole should be added to sufficient water, preferably boiling water, and thoroughly boiled for at least two hours and while boiling must be frequently stirred. The mixture should then be a rich chocolate-brown color. It should be allowed to stand for a few hours or overnight, then the liquid should be drained off without disturbing the sediment and sufficient water should be added to make 100 gallons. The dipping vat (cage or swimming) is filled with the solution in the proportions mentioned and heated to not less than 110° F or more than 115° F, maintaining this temperature while the animals are held in the vat for at least two minutes, during which period all crusts and scabs *must* be loosened with a stiff brush. A second dipping must be undertaken between the twelfth and fourteenth days after the first dip in order to destroy the new generation of mites (eggs are not killed by the dip) before they mature.

Stalls in which mangy animals have been kept must be disinfected with live steam or boiling water or be treated with an acaricide such as lime and sulfur. Brushes, scrapers, "rubbers," etc., should be boiled; harnesses should be rubbed thoroughly with a strong disinfectant.

Bovine mange. Sarcoptic acariasis, or mange, in cattle caused by *Sarcoptes scabiei* var. *bovis* Robin is not so common as the psoroptic form (scabies) but is far more difficult to cure. It usually attacks the parts of the body where the hair is short, namely the brisket and around the base of the tail. Treatment is the same as for horses described above.

Canine mange. The common mange of dogs is caused by *Sarcoptes scabiei* var. *canis* Gerlach, closely resembling the swine parasite. Mange in the dog appears first on the muzzle, around the eyes, on ears and breast, and later spreads to the back, abdomen, and elsewhere. Symptoms are much as in swine and horses.

Infected long-haired dogs should be clipped before treating. The following treatment is recommended:

> The whole skin may be covered with a solution of equal parts of green potash soap and alcohol and just enough carbolic acid to give it the odor. This is washed off next day and the surface is covered with the following: Napthalin, ½ oz.; Vaseline,* 2½ oz.; oil of lavender, 8 drops. This may be applied daily until a cure is established. Another very effective dressing and equally safe is sulphur, 1 oz.; carbonate of potash, ½ oz.; lard, 4 oz. For house dogs balsam of Peru or styrax, ½ oz. to Vaseline,* 2 oz., or alcohol, 1 pint, makes a most agreeable, if somewhat expensive dressing, which though slow is effective. (Law[10])

Notoedric mange. Mange of cats is caused by *Notoedres minor* var. *cati* (Hering) (*Sarcoptes minor* var. *felis* Gerlach), smaller and more circular than *Sarcoptes* but otherwise quite similar. Notoedric mange of cats begins at the tips of the ears and gradually spreads over the face and head. After clipping the hair in the region of the affected parts, apply ointments only, such as sulphur two parts, potassium carbonate one part, and lard eight parts. *N. minor* var. *cuniculi* Gerlach causes a severe mange of rabbits, beginning at the muzzle and spreading over the whole body.

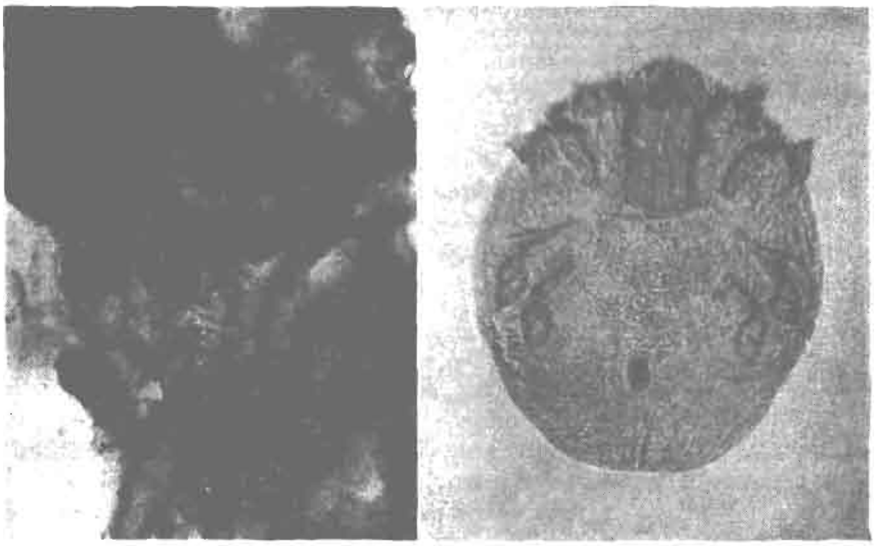
N. muris (Meigen) occurs on rats, and recently, 1934, Skidmore[11] describes an acariasis of white rats caused by a mite for which he proposes the name *Myobia ratti* (Family Cheyletidae).

Other mange mites. *Sarcoptes scabiei* var. *ovis* Megnin of sheep occurs on the face primarily and causes "black muzzle." Repeated dipping for several weeks in lime-sulphur is recommended. *S. scabiei* var.

* "Vaseline" Petroleum Jelly.

auckeniae Railliet occurs on llamas, and *S. scabiei* var. *caprae* Fürstenberg on goats.

Scaly leg mite on poultry. The legs of domestic fowls (chickens, turkeys, pheasants, etc.) are frequently attacked by a microscopic burrowing mite, *Cnemidocoptes mutans* (Robin and Lanquentin) (Fig. 169), which causes a lifting of the scales and a swollen condition of the shank with deformity and encrustation. The mites burrow and live in the skin, depositing their eggs in channels as do the mange mites. Scaly leg (Fig. 170) is easily transmitted from fowl to fowl; hence the infested birds should be segregated and treated.



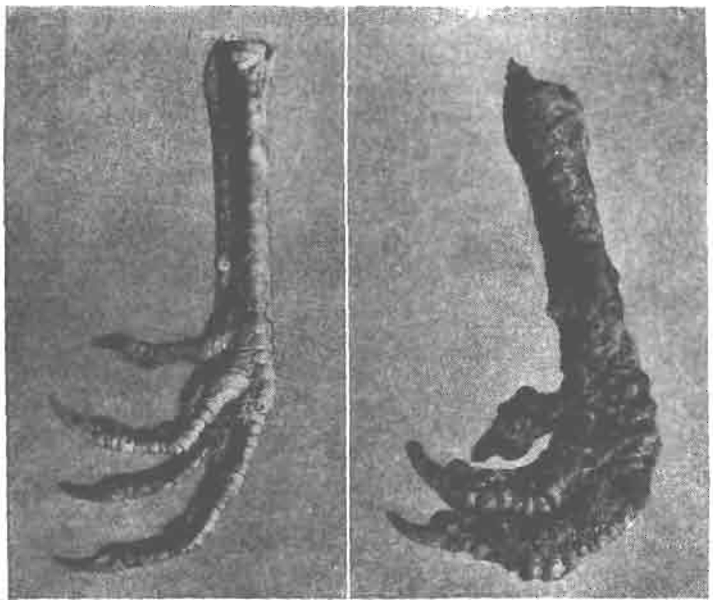
A B

FIG. 169. (*A*) photograph of a portion of scale from a fowl affected with "scaly leg," showing mites (*Cnemidocoptes mutans*) *in situ;* (*B*) enlargement of an individual mite. ×170.

In treating scaly leg the legs should be soaked and manipulated with the hands in soap and warm water in order to soften the scabs. Then when dry, "apply a coating of balsam of Peru or an ointment, containing 2 per cent of carbolic acid," or "a mixture of one part of oil of caraway with five parts of Vaseline*" (United States Department of Agriculture, Farmers' Bulletin 530). A simple treatment consists in dipping the legs of the fowl in a mixture of equal parts of kerosene and raw linseed oil; the oil must not touch the feathers on the legs; otherwise the skin may suffer. This dipping process is best done while the birds are roosting, lifting each bird from the roost, dipping, and then replacing it. The process should be repeated in about a week.

* "Vaseline" Petroleum Jelly.

Depluming mite. *Cnemidocoptes laevis* var. *gallinae* (Railliet), known as the depluming mite, is closely related to the "scaly leg mite," but attacks the skin of the fowl near the base of the feathers. The mites themselves do not cause the bird to lose its plumage, but the intense itching caused by the mites impels the host to pluck its feathers in an attempt to reduce the itching.



A B

FIG. 170. (*A*) normal leg and claw of a fowl; and (*B*) one affected with sarcoptic mites, *Cnemidocoptes mutans*, causing scaly leg.

No satisfactory treatment is available, but some benefit may be obtained by repeatedly rubbing the skin with a sulphur ointment made by mixing three parts of flowers of sulphur with one part of lard.

FOOT AND TAIL MANGE–CHORIOPTIC ACARIASIS

Chorioptic or symbiotic scabies. Mites belonging to the genus *Chorioptes* produce a mange which is restricted to certain parts of the body such as the feet, tail, and neck. *Chorioptes symbiotes* Verheyen [= *C. equi* (Gerlach)] attacks the lower parts of the legs of horses, particularly those with long hairs on the fetlocks. The infection is known as "aphis foot" in Australia.[12] A mixture of one part carbolic acid to 15 to 20 parts of linseed oil, or equal parts of kerosene and linseed oil, is recommended as treatment. Several applications are needed to effect a cure.

Tail mange in cattle caused by *Chorioptes bovis* (Gerlach) is localized on the tail or legs and is uncommon. Foot mange in sheep is caused by *Chorioptes ovis* (Railliet).

SCAB MITES—PSOROPTIC ACARIASIS

Characteristics of psoroptic mites. The psoroptic or scab mites belong to the family **Sarcoptidae** as do the itch and mange mites, hence partake of the family characteristics. However, in the psoroptic mites the legs are long and slender, all four pairs extending beyond the margin of the body, which is elongate. The "pedicel of the suckers is jointed" and the "mandibles styliform, serrate near tip" and suited for piercing. The

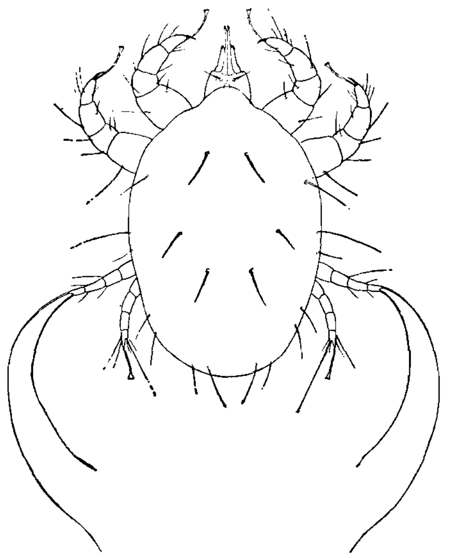


FIG. 171. *Psoroptes communis.*

psoroptic mites do not burrow, as do the sarcoptic mites but live at the base of the hairs of the host, piercing the skin and introducing a toxic saliva which causes inflammation. An exudate follows which partially hardens, forming a scab. As the mites multiply bites increase as well as itching; more serum oozes out to form a crust of loose humid matter. The parasitized area increases, and the skin becomes hardened and thickened. This condition is known as *scabies* or *scab.* The eggs are deposited among the piled-up scabs. Owing to the loose condition of the scabs and the hardihood of the mites, this form of acariasis becomes quickly and easily distributed from animal to animal by contact and by rubbing against fences, trees, and the like.

The commonest scab mites belong to the genus *Psoroptes* (Fig. 171) of which *Psoroptes communis* var. *ovis* (Hering) of sheep is best known. Other varieties of this species infest cattle and horses.

Ovine scabies (sheep scab). *Psoroptes communis* var. *ovis* (Hering) is the causal organism of scabies in sheep. This is by far the most important species of scab mites. However, with the widespread use of dips and rigid quarantine regulations, scabies in sheep is gradually being controlled.

The sheep scab mite is easily visible to the naked eye. The adult female measures about "one-fortieth" of an inch in length by "one-sixtieth" of an inch in breadth, and the male "one-fiftieth" by "one-eightieth" of an inch. As in all psoroptic species the mites are found on the surface

Fig. 172. Showing life history and general characteristics of a typical psoroptic or scab mite. (*Lower left*) egg, (*lower right*) larva, (*upper left*) female, (*upper right*) male. *Psoroptes communis*. ×85.

of the body among the scabs at the base of the hairs. The parts of the body most thickly covered with wool are chiefly affected. Scabies[13] is indicated by a "tagging" of the wool; the coat becomes rough, ragged, and matted at the points affected. Tags of wool are torn away by the sheep or are left attached to rubbing posts and other objects against which the animal rubs. The sheep scratches vigorously and shows signs of intense itching.

Life history of the scab mite. The female scab mite deposits an average of about 15 (maximum 30) eggs, one at a time, and the period of oviposition often lasts several days, after which the female evidently dies. The eggs (Fig. 172) are either attached to the wool near the skin or deposited directly upon the latter. The hexapod larvae hatch in from two to three days when next to the skin but take longer (up to six or eight days) when on wool not close to the body; the first molt takes place in three or four days, when the fourth pair of legs appears; second and third molts take place within the next four or five days. The males are said to molt but twice, and the female is fertilized after the second molt with a third molt before egg deposition. The length of time elapsing from egg to egg averages about nine days. The infection is spread by contact with infected sheep.

Treatment for sheep scab. Several kinds of dips with variations are used for sheep scab, among them lime and sulphur, nicotine and sulphur, nicotine cresol, coal-tar products, sodium silicofluoride, and derris. If proprietary dips are used, extreme care must be exercised in following the directions. The dip should have the approval of the United States Department of Agriculture. All dips must be repeated in ten to twelve days in order to destroy the mites newly hatched from eggs, which are not killed by the dip. The dips commonly used and permitted by the United States Bureau of Animal Industry for official dippings of sheep for scabies are lime-and-sulphur and nicotine solutions. See Imes[13] for directions for dipping sheep.

Psoroptic mange of horses is caused by *Psoroptes communis* var. *equi* Gerlach. It generally makes its appearance at or near the root of the mane or tail. It is regarded as a serious disease. Treatment is the same as for sarcoptic mange.

Bovine scabies (Psoroptic mange). Scabies in cattle is caused by *Psoroptes communis* var. *bovis* (Gerlach) and is a comparatively common infection. The disease usually appears at the root of the tail, thighs, neck, and withers and spreads rapidly to other parts of the body. Heavy losses may result from this disease.

Treatment for scabies in cattle is the same as for horses, namely lime and sulphur as previously described.

AURICULAR MITES–OTOACARIASIS

Auricular mites. A comparatively common affection of cats, dogs, and foxes, known as *otoacariasis* or parasitic *otitis*, is traceable to *Otodectes cynotis* (Hering), which resembles *Psoroptes* very closely. These mites belong to the family **Sarcoptidae.** They literally swarm in the ears of the host causing much discomfort, tenderness of the ears, auricular catarrh, loss of appetite, wasting, torticollis, "fits," etc.

Cleansing the ears first with soapsuds and warm water and then applying a sulphur ointment or a 10 per cent solution of tincture of iodine in glycerin, or a one per cent solution of carbolic acid in linseed oil is recommended. Banks recommends injecting olive oil containing one-tenth part of naphthol. The hutches or kennels must be thoroughly disinfected with a strong lime-and-sulphur solution or other effective solution to prevent further contagion.

FOLLICLE MITES—FOLLICULAR MANGE

Family Demodicidae

Characteristics of follicle mites. The **Demodicidae** comprise the follicle mites, very minute (.3 to .4 mm) mites with an elongated (vermiform) transversely striated abdomen and four pairs of "stubby" three-jointed legs. They parasitize the skin of mammals. All stages of the life history are often present together in a follicle.

The follicle mite of man (*Demodex folliculorum* Simon) (Fig. 173) inhabits the hair follicles and sebaceous glands "causing inflammation of

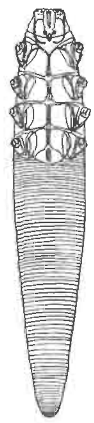


FIG. 173. A follicle mite. *Demodex folliculorum.* ×110.

the gland (comedones); their agglomeration in the meibomian glands (in man) sets up inflammation of the margins of the eyelids" (Braun). While the follicle mites may, under certain conditions, produce acne-like conditions, it is hardly probable that many cases of "blackhead" if any, may be traceable to these mites. They are nevertheless very common—said to occur in 50 per cent of mankind in all parts of the world.

In most animals the follicle mites are found in the region of the muzzle, and the infection is known as *follicular mange,* characterized by a raw appearance in advanced cases. Among those listed by Hirst[14] are the following cosmopolitan species: *Demodex canis* Leydig, which parasitizes dogs; *D. cati* Megnin, parasite of cats; *D. equi* Railliet, horses; *Demodex bovis* Stiles, which causes swellings about the size of a pea in the hides of cattle (shoulder and neck), affecting their value; *Demodex phylloides* Csokor, which infests the skin of swine, producing white tubercles.

Owing to the fact that the follicle mites occur so deeply in the skin, treatment is very difficult. Penetrating materials are necessary, for example, benzine, one part, and olive oil, four parts, or applications of tincture of iodine. Frequent applications must be made until a cure has been effected, which is doubtful. The following treatment for follicular mange in dogs is said to be of value—wash the dog with soap and water, then cleanse thoroughly and empty the pustules, after which soak the parts affected (or dip the animal) for five minutes in 5 per cent Formalin or 2 per cent formaldehyde, following this with an application of sulphur ointment. Repeat the treatment every three or four days until four or five treatments are given. The use of Ichthyol prepared with lard or lanolin in the proportions of one to seven is suggested by various authors. The chances for the cure of follicular mange are slight, and valuable dogs should be placed under the care of a skilled veterinarian where facilities are available for the production and administration of autovaccines.

LIPONYSSID MITES

Family Macronyssidae

Family Macronyssidae. The important group of Liponyssid mites was heretofore included in the family **Dermanyssidae** Kolenati, 1859 until 1931, when Vitzthum (*loc. cit.*) erected the family **Liponissidae.** In 1936 Oudemans proposed **Macronyssidae** as the family name for this group. Da Fonseca[15] calls attention to the fact that the type species of *Liponyssus* (*Liponyssus setosus* Kolenati), 1858, is lost; hence the generic characters cannot be ascertained. It is, therefore, an unscientific procedure to accept *Liponyssus* with unknown characters as a type for a family, and *Macronyssus* Kolenati is proposed as a substitute. "Macronyssidae," therefore, becomes the new family name. Fonseca summarizes the diagnosis of the new family as follows: "Mandibles with fixed and moveable arms devoid of any armature in the female. Palps with a bifid seta at the fifth segment. Dorsal shield of adults entire or divided in females. Sternal plate with two or three pairs of setae. Anal plate more or less piriform, never enlarged or prolonged. Holoventral shield of males entire or divided. Haemotophagus; oviparous or ovoviparous; sometimes facultative parthenogenetic; permanent ectoparasites on mammals, birds and reptilia."

All representatives of this group (Liponyssids) were heretofore included in the genus *Liponyssus* Kolenati, 1858; now, however, Fonseca (*loc. cit.*) lists 17 genera of which 5 are new. *Liponyssus* is retained as a monotypic genus *Liponyssus setosus* (Kolenati); the familiar tropical Liponyssid rat mite and several other species are placed in a new genus

Bdellonyssus Fonseca 1941. *Liponyssus bacoti* (Hirst), therefore, becomes *Bdellonyssus bacoti* (Hirst) (Fig. 174).

The genus *Bdellonyssus* Fonseca includes a homogeneous group of mostly tropical species in which the "dorsal shield is undivided; sternal plate of female with three pairs of setae and two pairs of slit-like pores; genital shield of female tapering to a more or less sharp point, not scale-like and with only a pair of setae; tibia I more than 1½ times as long as broad in the female; coxae of females and of nearly all males devoid of spines other than the dorsal one of coxa II; first palpal segment with a spur; holoventral shield of male undivided and undilated at the ventral surface" (Fonseca).

Tropical rat mites. According to Fonseca (*loc. cit.*) the triad of species *Bdellonyssus* (= *Liponyssus*) *bacoti* (Hirst), *B. brasiliensis*

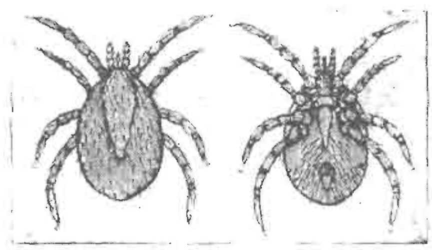


FIG. 174. The tropical rat mite, *B. bacoti;* (*left*) dorsal view, (*right*) ventral view. (After Dove and Shelmire.)

(Fonseca), and *B. nagayoi* (Yamada) forms so homogeneous a group that a correct identification will always be difficult. All three species have been taken on the Norway rat as well as other animals, and all have been reported to be annoying to man.

The tropical rat mite was first recorded from rats (*Rattus r. norvegicus*) in Egypt by Hirst[16] and described as *Leiognathus bacoti.* This mite is now reported from many parts of the world as irritating to man; Bishopp[17] reported it from the southern United States in 1928, and it is now known to be widely distributed. Bishopp states: "The bite is distinctly painful at the time the mouth parts are inserted. A sharp itching pain is usually experienced. Usually there is more or less irritation and itching at the site of the bite for several hours along with the development of a small haemorrhagic area."

Dove and Shelmire[18] (1932) report having experimentally transmitted the Texas strain of endemic typhus through bites of the tropical rat mite, *B. bacoti* (Hirst) from guinea pig to guinea pig. Various other workers have been unable to confirm the work of Dove and Shelmire.

Williams and Brown[19] (1945) have demonstrated that *B. bacoti* acts as an intermediate host of a filariid worm, *Litosomoides carinii* (Trav.), a parasite of the cotton rat, *Sigmodon hispidus.* Bertram, Unsworth, and Gordon[20] were also successful. Recently Philip and Hughes[21] (1948) have shown by preliminary tests that *B. bacoti* can transmit the causal agent of rickettsial pox, *Rickettsia akari,* from mouse to mouse by the bite. Transovarial passage to at least some of the mite offspring was also demonstrated.

Life cycle. Bertram, Unsworth, and Gordon (1946, *loc. cit.*) summarize the life cycle of *B. bacoti* (Hirst) as consisting of adult male and female, the egg, the nonfeeding larva, the bloodsucking protonymph, and the nonfeeding deutonymph. Two engorgements, one by the protonymph and one by the adult, are required to complete the life cycle in from 10 to 12 days at a temperature of 25° C. These authors state that unfertilized females reproduce parthenogenetically. Adult females were kept alive for 33 days on six engorgements, eggs up to minimum of 12 being laid between meals.

The nymphs and adults are very active and readily leave the nests and harborages of the hosts and travel freely for some distance to attack persons in restaurants, warehouses, offices, the home, and other situations where rats abound or have recently been.

Control of tropical rat mites is essentially one of rat control. In the absence of food the mites perish in about two weeks; therefore, if no other measures are taken, the pest will abate itself in about that time. However, with the elimination of the appropriate murine hosts they may become more annoying because of their enforced search for a blood meal. Along with rat control the author has recommended that tables, desks, chairs, cabinets, and woodwork be rubbed with a cloth moistened with kerosene. Bishopp recommends a mixture of anthracene oil, one part, and kerosene, two parts, for floors. Dusting rat harbors, floors, and other surfaces on which these mites crawl with 10 per cent DDT also gives good results.

The tropical fowl mite, *Bdellonyssus* (= *Liponyssus*) *bursa* (Berlese, 1888) is a widely distributed poultry pest reported from South America (Argentina, Brazil), India, Africa, China, and more recently (1917, Wood) from the United States. It appears to be a widespread parasite of the English sparrow, *Passer domesticus*, as well as of the chicken, *Gallus domesticus*. Although man is frequently bitten by this mite, no symptoms are reported other than a slight local irritation. This mite is a very tiny, fast-moving species measuring barely 1 mm in length. Wood[22] reports that the mites tend to accumulate on a few feathers about the vent of the fowl but when abundant may be quite generally distributed over the body. The fluffy down of the hen feathers lining the nest is a favorite location. This species feeds both by day and by night; the adults remain on the host and feed intermittently. The eggs of this mite are laid either on the fluff feathers of the host or in the nest. The incubation period according to Wood is three days; the minute larva does not feed but molts in about 17 hours and is ready to feed in this, the first nymphal, stage. The life cycle is said to require from 8 to 12 days.

The northern fowl mite, *Leiognathus** (= *Liponyssus*) *sylviarum* (Canestrini et Fanzago, 1877), resembles the tropical fowl mite very closely; in fact, Hirst, according to Cameron,[23] while pointing out the slight differences suggests that the tropical mite may be but a variety of the northern species. However, Fonseca places it in a different genus, *Leiognathus* characterized as follows: "Dorsal shield undivided; sternal shield with two pairs of setae; genital plate not scale-like, tapering to a point posteriorly and provided with only a pair of setae; coxae devoid of spines; holoventral shield of the male undivided." While reports of damage, often considerable, to chickens (*Gallus domesticus* Linn.), are from northerly regions, the first record came from Pisa, Italy, and severe infestations are reported from California and Florida. Furthermore, the host distribution as shown by Cameron (*loc. cit.*) indicates that it is a widespread parasite of wild birds, sparrows, starlings, and blackbirds being frequently mentioned.

On the fowl, these minute, very active mites prefer the region of the vent and accumulate on a few feathers like the tropical fowl mite and similarly spread to all parts of the body when the infestation is heavy. They also spread to the nests, roosts, and crevices of walls, but evidently deposit their eggs only on the birds. Several authors believe this species aestivates during the summer months; however, mites do occur on wild birds and in their nests during the summer months.

The egg stage lasts about 30 hours; the larva, which is quite sluggish, does not feed; the average duration of this stage is 9 hours. Information concerning the life cycle of this species of mite is very fragmentary as it is also for *B. bursa*. The cycle from egg to adult is believed to require from 8 to 10 days.

Control. The following recommendations for the control of these mites are made by Cameron (1938, *loc. cit.*). About 20 minutes before the birds go to roost during frosty weather (dropping boards, nest boxes, and roosts having been previously scraped clean) smear the roosts and roost supports and the front edge of each nest box with nicotine sulphate, applying about 2 oz for every 50 birds and treat every pen on the same evening, making sure that every bird goes to roost. Single birds may be hand-treated with a few drops of nicotine sulphate smeared around the vent, under thighs and wings, and on the neck. Make a practice of treating all birds as soon as they enter winter quarters in the fall, since they may have picked up infestations from wild birds. Prevent wild birds from nesting near poultry houses. The use of extremely fine

* This generic name is preoccupied and will, no doubt, have to be changed.

sulphur applied to the birds around the vent, under the wings, and on other feathered parts is also recommended. This treatment must be repeated in 10 days.

DERMANYSSID MITES

Family Dermanyssidae

The family Dermanyssidae is characterized by Ewing[24] as follows: "Mouthparts adapted for piercing, the chelicerae being either devoid of one or both chelae or of all true teeth; but, however, recurved 'holdfast' hooklike structures are sometimes present. Integument somewhat leathery and distensible, but covered to a large degree by one or more dorsal shields and one or more ventral plates. Each tracheal trunk opening through a peritreme situated on the side of the body. Sexual dimorphism evident and at times marked. Parasitic in habits and on vertebrates." In the genus *Dermanyssus* the female chelicerae are long and needle-like, being composed of two closely adhering elements; in the male the chelicerae are not needle-like but are variously formed, both arms being always present and easily recognized. The dorsal shield is large and undivided in both sexes; the legs are rather slender, especially the front pair. The rim of anus is greatly thickened behind, and the anus itself usually situated in the posterior half of anal plate. (Ewing.)

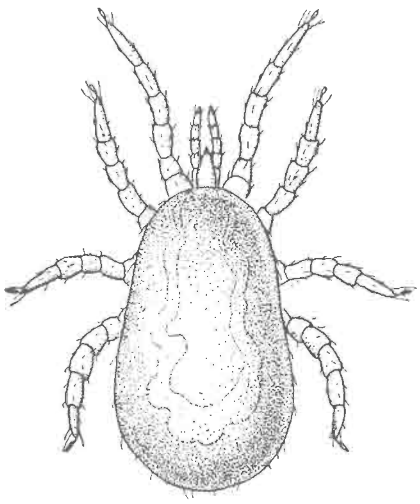


FIG. 175. The poultry mite. *Dermanyssus gallinae.*

The common red chicken mite, Dermanyssus gallinae (DeGeer), also known as the roost mite (Fig. 175), is the most important member of the family Dermanyssidae. It appears to be a pest of chickens throughout the world. Other poultry such as turkeys as well as pigeons are infested. English sparrows and some other wild birds may serve as hosts.

While man is commonly annoyed by crawling mites, instances of actually having been bitten appear to be quite rare. The size of the mites varies from .6 to .8 mm in length; the males average slightly less in length. They are somewhat pear-shaped and are light grey in color when unengorged and a dark red when engorged. Ewing[25] describes the female of *Dermanyssus gallinae* (DeGeer) as follows:

> Rather large species, with a long body and long legs; no shoulders present. *Dorsal plate.*—Covering considerably less than half of the dorsal surface of the body, broadest above the second pair of coxae, with lateral margins converging posteriorly behind widest part until truncate end is reached. Each posterolateral corner of dorsal plate is occupied by an oval light spot. *Ventral cephalothoracic characters.*—The posterior pair of sternal pores is represented by two minute circular openings on the posterior margin of the sternal plate near the posterolateral corners. *Anal plate.*—Slightly longer than broad, anterior margin slightly out-curved. Anal opening more than twice as long as broad, with lateral margins parallel. Anal covers very long, occupying more than half of the anal opening; behind them in the anal opening a minute, transverse, sclerotized bar. Paired anal setae situated at a level slightly in front of middle of anal opening and at about one-third the distance from rim of anal opening to lateral margins of anal plate; unpaired anal seta situated at a point about one-third the distance from posterior rim of anal opening to posterior margin of anal plate.

Habits and life history. During the daytime the mites remain hidden in crevices of the henhouse and roosts, under boards and debris. In these hiding places the mites deposit their eggs. At night the pests swarm out from their hiding places and attack the birds upon the roosts. Their attack is not restricted, however, altogether to the nighttime, for swarms of mites may be found on setting hens and laying hens during the day if these are nesting in dark places.

Bishopp and Wood,[26] and Wisseman and Sulkin[27] have made important contributions to a knowledge of the life history of this mite. Adult female mites usually begin ovipositing in from 12 to 24 hours after feeding and lay as many as 7 eggs, which hatch in from 48 to 72 hours at summer temperatures. The hexapod larvae are sluggish and do not feed. The first-stage nymph (eight-legged and active) appears in from 24 to 48 hours and, after a blood meal, molts and gives rise to the second nymphal stage. After feeding and a second molt the adult stage is reached. Occasional instances of a third nymphal stage are reported. The entire life cycle may be completed in as short a period as 7 days.

Damage done and control. The poultry mite is a serious pest in many parts of the world. The damage which this mite causes is very considerable and may be summarized as follows: Egg production is greatly reduced or entirely prevented; setting hens are often caused to leave their nests or perish; newly hatched chicks perish in large numbers in the presence of these mites; chickens lose flesh, are unthrifty, and are unprofitable for marketing; loss of blood and reduced vitality cause birds to be easily susceptible to disease. Inasmuch as the mites are seldom found on the bodies of the birds during the day, except in the first feeding period when they sometimes remain attached for a night and a day, or in dark nest boxes, control measures are directed most advantageously when they are in their hiding places. A thorough clean-up of the premises to which the birds have access, together with the elimination of every useless article therein, such as boxes, coops, boards, etc., is the first step. Old nesting material should be burned and if the infestation is severe, roosts and nests should be dismantled to be replaced by construction that will facilitate future clean-ups. Methods must now be directed against the cracks and crevices of the floors, walls, and even the roof of the poultry house. In heavy infestations the mites sometimes migrate to the outside of the house when the inside is sprayed. They should be looked for in cracks on the outside and if present the outside should also be treated. The most efficient method of treating poultry quarters is by the use of liquid insecticides applied by "knapsack" spray pump. A coarse spray is most effective and should be applied to each area from several different angles to ensure penetration into all hiding places. Many of the existing sprays are efficient. Any of the dips used on domesticated animals made up in a slightly stronger solution than directed for the dipping of animals will give fairly satisfactory results. The most satisfactory of all applications, however, are the wood preservatives that have coal-tar product anthracene oil as a base. These have the advantage of being effective over a long period of time and soak into the wood rapidly without leaving a greasy residue to soil the feet, feathers and eggs of the birds. They should be diluted about one-half with kerosene to facilitate spraying. One thorough treatment with this type of spray is generally sufficient. *Whitewash* containing 3 to 5 per cent of crude carbolic acid kills large numbers of mites but is not nearly so effective as anthracene oil. Nicotine sulphate used on the roosts, as for lice, is effective in killing young mites feeding on the birds and in protecting roosting birds. Used as a spray at the rate of three tablespoonfuls per gallon of water to which is added one-half teaspoonful of baking soda it gives very satisfactory results if applied carefully and in drenching quantities.

Prevention. The chicken mite is introduced into clean flocks in many cases in contaminated shipping coops. In some cases the introduction may be accomplished by a few young mites that are engorging for the first time on the introduced fowls. To overcome this possibility, newly acquired birds should be kept in special coops for two or three days before being placed in clean houses. These coops should then be destroyed or disinfected with boiling water or with one of the sprays suggested above. Shipping coops from other poultry plants should not be left in or near clean houses, nor should second-hand equipment be introduced unless properly disinfected. Mites will live for from three to five months without food, a fact which should be considered when vacant buildings are to be occupied by clean flocks. In cases where the control of the mite is impossible owing to the character of the quarters or lack of them, some relief is afforded by wrapping the ends of the roosts and other points of contact with rags soaked in crude oil to prevent the mites from gaining access to the fowls after they have gone to roost. To make this procedure effective crowding should be discouraged, and the back roosts should not be near enough to the wall to permit the movement of the mites to the plumage of the birds.

Other Dermanyssid mites. *Dermanyssus americanus* Ewing was described from English sparrows in Washington, D. C. The palpi of the female extend beyond the tips of the anterior femora; chelicerae are needle-shaped; the peritreme very short, not extending beyond the third coxa; the anus is situated almost entirely behind a middle transverse line; the paired setae are slightly behind the level of the anterior margin of the anus; the legs are very short and stout, the last pair falling far short of the tip of the abdomen. The length of the mite is 0.70 mm, the width, 0.39 mm.

Allodermanyssus sanguineus (Hirst), a mouse mite, the vector of rickettsialpox of man, is described by Ewing (*loc. cit.*) as follows: "Palpi slender, reaching to the tips of anterior femora; chilicerae showing plainly the needle-like elements representing both arms. Dorsal shield divided; anterior shield broadest at the shoulders, lateral margins behind the shoulders concave; posterior shield circular, minute. Sternal plate squarish, lying entirely between the second coxae, with three pairs of subequal marginal setae. Anal plate egg-shaped in outline, anterior margin broadly rounded; anus situated centrally, rim very thin in front and on the sides, but enormously thickened behind; paired setae situated at the level of the center of the anus; median seta situated about two thirds its length behind anus; caudal area reaching about halfway to the base of median seta. Legs very long and slender. Length, 0.91 mm; width, 0.46 mm."

Rickettsialpox of man was first observed in New York City during the summer of 1946, the causal organism, *Rickettsia akari,* being isolated by Huebner, Jellison, and Pomerantz.[28] The epidemiology of the disease is described by Greenberg, Pellitteri, and Jellison;[29] the infection is transmitted by the rodent (mouse) mite, *Allodermanyssus sanguineus* (Hirst), and the house mouse, *Mus musculus,* serves as a reservoir.

Pulmonary acariasis of monkeys is traceable to mites of the genus

Pneumonyssus which live in the lungs of the host, e.g., *Pneumonyssus simicola* Banks. *Halarachne zalophi* Oudemans occurs in the nasal passages of the California sea lion; and *Sternostomum rhinolethrum* Trouessart is said to produce catarrhal inflammation of the respiratory tract of fowls. Lung mites commonly infest the respiratory system of snakes. (Turk.[30])

The role of bird mites in the epidemiology of arthropod-borne encephalitides. In 1942 Hammon *et al.*[31] demonstrated that a large percentage of wild birds as well as domestic fowl possess neutralizing antibodies for the western equine and St. Louis encephalitis. This discovery gave impetus to an investigation already under way concerning the part that avian bloodsucking ectoparasites may have in the infection chain of these viruses. (See Chapter XII, Mosquitoes as Vectors of Disease.)

In 1944, Smith, Blattner and Heys,[32] reported the isolation of the St. Louis encephalitis virus from chicken mites, *Dermanyssus gallinae* (DeGeer), in nature during a nonepidemic period. Evidence was presented that this mite is capable of transferring the virus of St. Louis encephalitis congenitally to its offspring and "that a colony of mites once infected probably remains infected indefinitely." Sulkin[33] (1945) recovered the virus of the western type of equine encephalitis in the same species of mite, also in nature, but at the time of an outbreak of the disease among horses in Texas Howitt and associates[34] (1948) reported the isolation of the virus of the eastern type of the disease from *D. gallinae*. It had previously been shown (pp. 242 to 244) (as early as 1942) that mosquitoes belonging to several species are capable of acquiring, and transmitting to animals, the virus of both the St. Louis and the western equine encephalitides. However, these viruses appear not to be transmitted transovarianly in the mosquito, nor have they been recovered in hibernating mosquitoes; hence poultry and/or wild birds mites serving as reservoirs would constitute a strong link in the infection chain of the arthropod-borne encephalitides. In 1948 Smith and associates[35] reported having transmitted the virus of St. Louis encephalitis to hamsters by mosquitoes, *Culex pipiens*, at periods varying from 4 to 27 days after these insects had fed on chickens having viremia as a result of the bite of infected mites. Thus the concept is supported that two bloodsucking vectors may be involved, one the chicken *mite* which does not bite man and in which the virus is maintained in nature by transovarian passage, and the other, a *mosquito*, which transmits the infection from birds to other vertebrates, including man. Two other species of bird mites are involved, perhaps even more plausibly (Hammon[36] obtained negative results with *D. gallinae*), namely, *Leiognathus* (= *Liponyssus*) *sylviarum* (C. and F.) from which Reeves and associates[37] isolated the virus of western equine encephalomyelitis in California (mites were from nest of English sparrow), and *Bdellonyssus* (= *Liponyssus*) *bursa* (Berlese) from which Sulkin and Izumi[38] isolated the western equine virus in Texas.

While apparently complicating the infection chain, the discoveries implicating mites do furnish the factor of maintenance of the virus in nature. Thus mites infected previously either congenitally or by feeding on an infected bird may initiate infection, as Hammon (*loc. cit.*, 1948, p. 244) points out, in nestling birds or in newly hatched fowl in the spring. The young birds are then fed on by mosquitoes, and after the essential extrinsic incubation period, these mosquitoes may infect other birds. Hammon continues to reason that several such cycles in mosquitoes and birds will build up the number of infected mosquitoes, and infection may then appear in men or horses. Infection is usually seen first in horses and later in man, no doubt, because of the greater number of mosquitoes having access to these large unprotected animals and because of the zoöphilism of these insects.

CHIGGER MITES

Family Trombidiidae (Subfamily Trombiculinae)

Family Trombidiidae. The family of mites (**Trombidiidae**) to which the chiggers (larval Trombidids) belong is characterized by Banks (*loc. cit.*) as follows (adult characters):

> the body being divided into two portions, the anterior (cephalothorax) bearing the two anterior pairs of legs, the palpi, mouth parts, and eyes; the posterior (abdomen) is much larger and bears the two posterior pairs of legs. The mandibles are chelate; at least there is a distinct jaw or curved spine-like process. They are always red in color, some, however, being much darker than others. The body is covered with bristles or feathered hairs according to the species. The palpi are five-jointed, quite prominent, often swollen in the middle, the penultimate joint ending in one or two claws, the last joint (often clavate) appearing as an appendage or "thumb" to the preceding joint. The legs are seven-jointed; the tarsi terminate in two small claws. The legs are clothed in the same manner as the body. There are two eyes upon each side of the cephalothorax, quite frequently borne on the distinct pedicel.

The chiggers belong to the subfamily Trombiculinae, in which "each tarsus is 3-clawed, outer claws nearly always equal, forming a pair, middle claw usually longer than others; pseudostigmatic organs, when simple, long and more or less flagelliform" (Ewing[39]). There are about 50 recognized genera and well over 100 species.

Brennan[40] has made an effort to clarify the terms used in describing chiggers; the arrangement of the setae is considered important, but the dorsal formula is not always constant and cannot be relied upon entirely in the diagnosis of a species. Recently Wharton[41] (1948) has shown that the chaetotoxy of the legs is an important aid to the taxonomic study of larval trombiculids. He points out that four types of modified setae are found on the legs and palps as follows:

1. Blunt striated sensory setae, one on the dorsal side of each tarsus of the first two pairs of legs and one at the base of the palpal tarsus on the ventral surface.
2. Pointed striated sensory setae, on the genua and tibiae of all legs, the tarsus of leg I, and the pretarsus of legs I and II. There may be one on tarsus III and the palpal tarsus, but this is unusual.
3. Microsensory setae, may be present on the genua, tibiae, and tarsi of legs I and II.
4. Whip-like setae, one or more, may be present on telo-femur, genu, tibia, and tarsus of leg III.

The literature pertaining to the mite family Trombidiidae has grown enormously during the past few years as shown by the bibliography published by Williams[42] in 1944 consisting of 375 references.

Among the Trombiculid mites causing a severe dermatosis are the European species, *Trombicula autumnalis* (Shaw), known as the harvest mite. *Eutrombicula* differs from *Trombicula* in having bifid rather than trifid claws in the larva. There are at least three species of chiggers known to attack man in the Americas (Jenkins[43]); namely, *Eutrombicula alfreddugèsi* (Oudemans), *E. batatas* (Linn.), and *E. masoni* (Ewing).

E. alfreddugèsi = *Trombicula thalzahuatl* (Murray); early mention of *Leptus irritans* Lucas also probably refers to this species of larva; it is the common chigger of the United States, ranging from New England to Nebraska, south to Florida and Texas, and west to California; it also occurs in Mexico, Central America, and South America, where it is known as *bête rouge.* "It occurs abundantly in second growth cut-over areas, especially blackberry patches, forest edges, and river valleys where many species of birds, mammals and reptiles are often found" (Fig. 176).

E. masoni, according to Jenkins (*loc. cit.*), is distributed along the Atlantic coast from Florida to Massachusetts and along the Gulf coast to Texas, with isolated "islands" in Michigan and Minnesota. It is the most abundant species in Florida and parts of Georgia and is often present in large numbers when *E. alfreddugèsi* is rare or absent.

E. batatas is a tropical species and has been collected in the warmer southeastern part of the United States, and recently (Doetschman and Furman, 1948, in MS) it was reported from California. It is said to attack man rarely in the United States but is known to infest man freely in Panama and other tropical areas.

The bright red adult mites are found on the surface of moist ground or to a depth of 2 or 3 inches in drier soil, being often seen in the bases and root stocks of clumps of grass, in sphagnum moss, and under logs. In central Florida, Jenkins (*loc. cit.*) found adults of *E. alfreddugèsi* and *E. masoni* in largest numbers in winter and early spring; they became less common in summer and fall. Their food is said to be insect eggs.

According to Michener,[44] the spherical pale-dull orange-colored eggs of *Eutrombicula batatas* are laid singly on the surface of moist soil. The

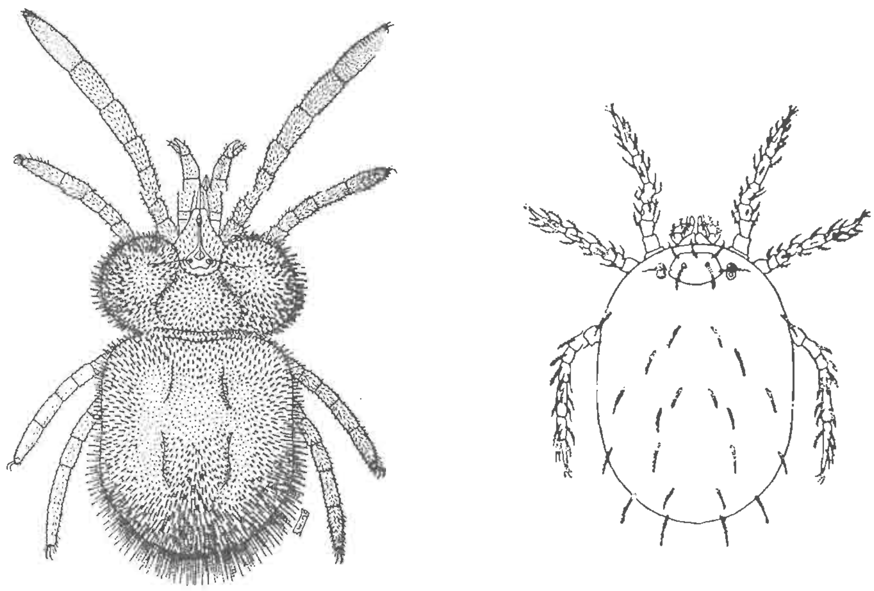


FIG. 176. Chigger (mite) or harvest mite. Adult (*left*), larva (*right*) of *Eutrombicula alfreddugesi.* (From Ewing's *Manual of External Parasites.* Courtesy of Charles C. Thomas, Publishers.)

incubation period ranges from 4 to 5 days, when the egg shell breaks into two portions, anterior and posterior, which become separated; but the nearly fully developed larva remains inside this "deutovum" for six or seven days more. Having completed the deutoval stage the fully formed red hexapod larva emerges and crawls rapidly about in search of a host. The larvae can live for about two weeks (probably longer) without feeding. The opportunity to find a suitable host should be good since it is known that close to a hundred species of vertebrates, mammals, birds, amphibians, and reptiles may serve as hosts. Ewing (1944, *loc. cit.*) reports rearing *E. alfreddugèsi* from box turtles, *Terrapene carolina* (Linn.), and from young toads, *Bufo* species, as well as from snakes.[45]

Keay[46] reports numerous hosts for *T. autumnalis,* among these the dog, horse, shrew, fowl, and sparrow. Engorgement of the chiggers (see next section explaining feeding habits) takes from 2 to 10 days, after which they drop to the ground and remain quiescent (protonymph) within the dead larval skin for 5 to 7 days, emerging with eight legs (the nymph). The nymphal stage lasts from 21 to 52 days, including the preadult stage, when another molt liberates the adult. The life cycle of *E. batatas,* according to Michener's (*loc. cit.*) summary requires from 39 to 85 days. The adult may live up to 45 days.

Chigger dermatitis. In many parts of the world, particularly in the warmer portions and during the late summer months in temperate climates, persons who have walked among tall weeds and grass, brambles, and low scrub often suffer an intolerable itching, which begins 3 to 6 hours after exposure and is followed by a severe dermatitis consisting of pustules and wheals. When, after a trip through tall weeds or grass or a patch of berry brambles, itching begins around the ankles, the knees, and the waist, a careful examination will pretty surely reveal at least one (there will be others) bright red mite barely visible to the naked eye either traveling fast or about to attach itself to the skin. A microscopic examination will show that it has three pairs of legs and no doubt is the larva of a trombiculid mite—a chigger.

Chiggers do not burrow into the skin as is commonly believed. Ewing[47] observed and studied daily 26 chiggers on his own skin; 21 were attached to the smooth surface of the skin, while five were attached at the bases of hairs, each having the capitulum thrust into the mouth of the hair follicle; not a single one had penetrated a pore or hair follicle. The chigger does not suck blood but injects a digestive fluid (a proteolytic enzyme) when firmly attached. This fluid dissolves the tissues so that the resulting liquid may be utilized by the chigger. The skin of the host becomes hardened, and there is formed a tube (stylostome) in which the chigger lies and continues to imbibe the nourishing liquid until it is replete; then it retreats and drops to the ground. According to Michener and Michener[48] it is presumably the action of the digestive fluid which causes the "bite" to itch after a few hours. Williams[49] points out that histological preparations of chigger bites on rabbit ears show that the epidermis is completely penetrated. A tube lined with stratum germinativum cells is formed which extends to the derma and subcutis. This tube appears to represent a reaction of the host to the secretion of the chigger, and its inner layer of cells are necrotic and give evidence of digestion.

To free infested areas of chiggers, lawns and gardens may be treated with very fine dusting sulphur applied with a hand duster at the rate of one pound per 1,000 sq ft, or 50 pounds per acre. Apply at intervals of two weeks during the chigger season, May to August. The ground should be carefully treated; the sulphur dust will not injure vegetation, but bright metal objects are easily tarnished. Dusting with DDT as recommended for tick control is also suggested by some authors. The use of benzene hexachloride (12 per cent gamma isomer) applied as a dust or spray at the rate of 4 pounds per acre has recently been reported as a successful treatment by the United States Bureau of Entomology and Plant Quarantine. Keeping down weeds and mowing the grass short so as to expose chiggers to the sun and dry the soil are good preventive measures.

Having been exposed to chiggers one should immediately take a bath lathering the body with a sulphur soap and allow the lather to remain about ten minutes before rinsing off.

Chigger repellents such as dimethyl phthalate and benzyl benzoate are available for the impregnation of clothing, particularly about the ankles and at the waistline. Relief from the "bites" may be had by applying various remedies such as a 5 per cent solution of benzocaine in alcohol.

Tsutsugamushi disease, also known as "Japanese flood fever," "Japanese river fever," "kedani fever," and more recently "scrub typhus" and "Mossman fever" (Australia), is an ancient disease (Blake *et al.,*[50] 1945) first described from Japan. It was long said that the infection is transmitted to man by the larval trombiculid (chigger) mite, *Trombicula akamushi* (Brumpt, 1910) from rodent reservoirs (voles, *Microtus montebelli* Sado). It was not, however, until 1916 that positive experimental evidence to that effect was secured by Miyajima and Okumura[51] in tests with monkeys. The specific rickettsial nature of the infection was determined in 1930 by Nagayo *et al.,*[52] who described *Rickettsia orientalis.* However, as early as 1920, Hayashi[53] reporting on the etiology of tsutsugamushi disease gave the name *Theileria tsutsugamushi* to what he then believed to be the causal agent. It is contended by Steinhaus[54] and others that, since it cannot be proved that Hayashi did not see the causal agent of this disease, the specific name which he gave should not be ignored. Under the rules of priority the specific name of the causal organism would then be *Rickettsia tsutsugamushi* (Hayashi), which name is accepted by Philip[55] in his account of tsutsugamushi disease in World War II.

The disease has an incubation period of 6 to 12 days and over. During the first 5 to 7 days it is characterized as follows (Blake, *et. al.,* 1945, *loc. cit.*): "headache (postorbital), apathy and general malaise, fever (chills), relative bradycardia, anorexia, conjunctival congestion, lymphadenitis, often regional, and an eschar." The *eschar* is the primary lesion which originates at the point of "chigger" attack (ankle, shin, groin, waistline, or axilla) in the great majority of cases. It is a painless papule at first, usually unnoted by the patient. It slowly enlarges to a diameter of 8 to 12 mm, the center becoming very dark and necrotic; a shallow ulcer may result eventually, leaving a scar. Between the fifth and eighth day in nearly all cases a characteristic dull red macular or maculopapular rash appears on the trunk and may spread to the extremities involving the palms, soles, face, and scalp. This may persist for about two weeks, often fading in a few days. Enlargement of the spleen, nervous disturbances, delirium, and prostration are common symptoms; in many cases there is deafness. The majority recover by lysis in three to five weeks. In extremely severe cases "symptoms and signs of more severe pneumonitis and encephalitis are constant. Evidences of peripheral circulatory collapse are common and signs of myocarditis may appear. . . . Thromboses and cerebral or gastrointestinal hermorrhage may take place." Mortality ranges up to 40 per cent; death results from cardiac failure, usually in about three weeks. The rickettsiae may readily be recovered from the blood of patients during the acute stage of the disease by the intraperitoneal inoculation of 0.2 to 0.3 ml of blood in white mice. (Blake *et al., loc. cit.*)

Although the greatest number of cases occur at low elevations (sandy bottom land overgrown with grasses and scrub), infections occur at elevations of 2,000 to 3,000 feet and, in Formosa, as high as 6,500 feet. The "chigger" mites have been reported as high as 8,000 feet in India. The infection is endemic in many parts of southeastern Asia and adjacent islands in the Indian Ocean and southwest Pacific (Ceylon, Japan, Philippines, Netherlands Indies, Taiwan), and the coastal area of North Queensland, Australia.

The importance of scrub typhus (tsutsugamushi disease) as a "medical casualty producer in some areas during the Asiatic-Pacific operation, 1941–1945," according to Philip (*loc. cit.*), "was second only to malaria and was more dreaded by the men." Interference with actual combat operations was greatest, Philip states, on the Assam-Burma front where "eighteen per cent of a single battalion got scrub typhus in two months and in that time 5 per cent of the total strength had died of it."

"American Task Force operations in the Schouten Islands resulted in a thousand cases in the first two months on Owi and Biak, reaching a total of 1,469 casualties in 6 months time, while at Sansapor beachhead the curve for weekly admissions on a thousand-per-year basis shot up to over 900 at the end of the second week, a rate higher for an individual episode than any yearly rate for all causes in the entire American Army in all theatres. . . . These two disasters alone provided a potential estimated loss of over 150,000 man days to the American Sixth Army."

The vector. There are a number of species of chiggers in endemic areas which attack man and cause "scrub itch" but are not vectors of tsutsugamushi disease. In spite of much research during the late war years, *Trombicula akamushi* (Brumpt) and *Trombicula deliensis* Walch are the only proved vectors, as Philip (1948) points out. Furthermore, Philip states that he has specimens of supposed *T. akamushi* from Japan that are indistinguishable morphologically from *T. deliensis;* hence there is good reason to suspect that the latter may really be but a variety of *T. akamushi* (Philip and Kohls, 1948[56]).

The adult mites measure from 1 to 2 mm in length, are generally reddish in color, often pale; they are ground-inhabiting and nonparasitic and are said to feed on insect eggs and minute arthropods; many authors refer to them as scavengers. The winter is spent in this stage. They are said to deposit their eggs in late spring or early summer in loose top soil under leaves, etc., in damp places. The hexapod larvae (0.32 to 0.43 mm in length) hatch in about 3 weeks under favorable temperature conditions (about 20° C) and wait the coming of a suitable vertebrate host—mammal or bird (man is an accidental host). Having successfully encountered a host the chigger attaches itself and begins to feed. Numerous hosts are available, but continuous propagation of the infection is dependent upon susceptible so-called reservoir animals (many of the host animals are suitable for the maintenance of the vector species including birds and reptiles), the first of which was demonstrated to be a vole, *Microtus montebelli* (see above). Wartime studies as reported by Philip and Kohls (1948, *loc. cit.*) indicate that rats play an important widespread role in the disease cycle in nature. "Six kinds of rats (among them *Rattus concolor browni* and *R. flavipectus yunanensis*) indigenous to New Guinea, Burma, or India, plus a species each of field mouse and tree shrew (*Tupaia belangeri versurae*) have been added to two murid species already demonstrated to carry natural infection in Japan and Malaya, and to others under strong indictment in Formosa, Sumatra, and Australia." Infection has been demonstrated in the circulation of English sparrows, pigeons, and turkeys (Philip).

The larval mite (akamushi) is said to remain on the host, commonly in the ears, feeding for three or four days, then drops to the ground where in five or six days it molts and the nymphal stage (eight-legged) is reached. This stage like the adult stage, which it is said to reach with another molt in about ten weeks, is nonparasitic (Miyajima and Okumura, *loc. cit.*). Experimental evidence is complete showing that larval mites (chiggers, i.e., akamushi) hatched from eggs layed by such female mites may transmit the infection to experimental animals. The proportion of infected larvae is said to be perhaps less than one per cent. That there is a chigger mite in the United States which is a member of the "akamushi" group is a matter of some interest. Wharton[57] states that the larva of *Trombicula myotis* Ewing is more closely related to *T. akamushi* than to any other species, and could easily be confused with it.

Control. During the early months of warfare in the southwest Pacific area little or nothing was known about the ecology of scrub typhus, information needed to plan successful measures for the control and prevention of the disease. Thus because of the high incidence of infection among troops bivouacked or maneuvering in the abundant tall kunai grass warning against such practices were issued; however, severe epidemics occurred under the "coconut canopy" along the beaches. There were outbreaks under various ecological conditions, causing considerable confusion. There appear to be several factors, however, that do provide some basis for control: the mites are extremely sensitive to lowered humidity. Therefore, clearing of grass and scrub (clean camp sites) is a practical procedure, with the use of flame thrower and bulldozer suggested; secondly, rat harborage and a plentiful supply of food for native rats provide an abundance of hosts for the larval mites, hence rat control is an important chigger-control procedure.

Protection of the person against chiggers by repellents (dimethyl phthalate) applied to clothing was achieved during the latter part of World War II. Dipping uniforms in a dibutyl phthalate-benzyl benzoate emulsion gives good protection.

LOUSE-LIKE MITES

Family Tarsonemidae

Characteristics of Tarsonemidae. This is a small family of soft-bodied mites having in the female a characteristic "prominent clavate organ of uncertain use" between the first and second pairs of legs. The third and fourth pairs of legs are separated from the first and second pairs by a long interspace. Sexual dimorphism is marked in the several species. The piercing, sucking mouth parts are provided with slender needle-like stylets. Many of the species are predaceous or parasitic on insects, while others suck the juices of plants.

Pediculoides ventricosus (Newport) (Fig. 177) is a widely distributed predaceous mite which attacks the larvae of a number of species of insects such as the Angoumois grain moth [*Sitotroga cerealella* (Oliv.)], the wheat joint-worm [*Harmolita grandis* (Riley)], the peach twig borer (*Anarsia lineatella* Zell.), the cotton-boll weevil (*Anthonomus grandis* Boh.), the bean and pea weevils [*Mylabris quadrimaculatus* (Fabr.) and *M. obtectus* (Say)], etc. It is, therefore, normally a beneficial mite, but unfortunately it also attacks man, producing a very disagreeable dermatitis commonly called "straw itch."

While the male mite is very tiny, just about visible to the naked eye, the female when pregnant becomes enormously swollen, measuring nearly a millimeter in length, the abdomen presenting a globular appearance, the cephalothorax and appendages barely visible.

Within the enlarged abdomen of the female may be found rather large eggs which hatch internally, and the young mites develop to maturity within the body of the mother before being extruded. The number of young produced by the female is said to range from a few to nearly 300. The female mites are often fertilized within the body of the mother.

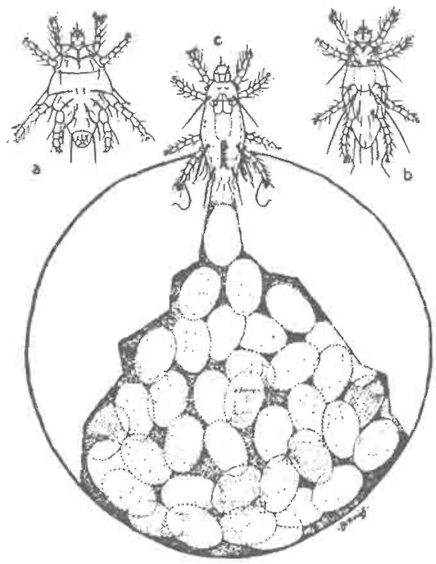


FIG. 177. *Pediculoides ventricosus.* (*a*) Male; (*b*) female; (*c*) gravid female, showing developing eggs. (Redrawn after various authors.)

A number of epidemics of dermatitis have been traced to these mites, infection having been brought about by sleeping on straw mattresses or while laboring in grain fields at harvest time. The infection has been confounded with hives, scabies, and even chickenpox; the neck, chest, abdomen, back, arms, and legs—in fact, the whole body—may be involved, and the itching is very intense. The eruption, which appears in 12 to 16 hours after the attack, is commonly accompanied with some fever.

According to Goldberger and Schamberg[58] the itching subsides under continuous exposure in from three to seven weeks. They recommend treating the infection with an ointment of beta naphthol, sulphur, benzoate, and lard.

To destroy mites in the straw of mattresses or in other situations, fumigation with sulphur, or steaming, or dry heat is recommended.

As to prevention, Webster[59] suggests burning the grain stubble during the fall, winter, or spring, and also states that threshing direct from the shock resulted in the control of the grain moth and consequently of the parasitic mites.

FLOUR AND MEAL MITES, GROCER'S ITCH

Family Tyroglyphidae

Characteristics of Tyroglyphidae. This family includes a small group of very tiny mites, ordinarily about 0.5 mm or less in length. Several of the species attack grain, flour, meal, dried meat, hams, dried fruits, insect collections, etc. Their development is so rapid that literally millions of them may appear in a stored product in a few days.

The metamorphosis of this group involves a peculiar stage known as the *hypopus,* appearing after the larval and nymphal stages, very unlike either of these and very different from the adult. In this stage the mites are said to attach themselves, nonparasitically, to flies and other insects, which disseminate them.

Persons handling stored products of various kinds, cereals, flour, meal, etc., may be attacked temporarily by the mites and experience a severe dermatitis, known as "grocer's itch," "copra itch," etc.

Tyroglyphus (= *Acarus*) *siro* (Linn.) is the grain mite or cheese mite, found in grain, stored products, and cheese; this mite causes a rash known as "vanillism" in vanilla-pod handlers; *T.* (= *Aleurobius*) *farinae* (DeG.) is known as the flour mite and is common in flour mills and granaries; *T. americanus* Banks is known as the cereal mite and is widespread and abundant on cereal products, seeds, stored prunes and other fruits; *Tyroglyphus longior* Gerv. attacks grains, cereals, dry seeds, etc., and causes "copra itch" among workers handling copra. *T. farinae* (DeG.) and *T. longior* Gerv. have been reported from the urinary tract and *T. longior* Gerv. from the intestinal tract (intestinal acariasis[60]).

RED SPIDERS

Family Tetranychidae

Characteristics of Tetranychidae. To this family belong the "web-spinning mites," most commonly infesting vegetation and destructive to fruit trees and other plants. The term "red spiders" is ordinarily applied to the group. *Tetranychus bimaculatus* Harvey, the two-spotted mite, attacks many species of plants as does the common red spider, *T. telarius* (Linn.).

Persons employed in picking hops and harvesting almonds, etc., often complain of itching produced by the red spiders, but this soon disappears.

QUILL MITES

Family Cheyletidae

Syringophilus bipectinatus Heller, the quill mite of poultry, lives in the shafts of the primary wing feathers. Rebrassier and Martin[61] report that this mite caused a peculiar molt; the loss of feathers extending over half of the body in most cases and in many instances over the whole body. The birds were reported to be apparently in good physical condition. S. *columbae* Hirst is a quill mite of pigeons.

AIR-SAC MITES

Family Cytoleichidae

Cytoleichus nudus (Vizioli) is known as the air-sac mite of poultry because of its habitat in the air passages. *Laminosioptes cysticola* (Vizioli) occurs in the subcutaneous tissue but is considered of no economic importance.

THE PENTASTOMIDS

Tongue Worms

The Class Pentastomida, also known as the Linguatulida, comprehends the so-called tongue worms, parasites of numerous species of vertebrate animals, from fishes to man. Because of certain resemblance to the phytoptid mites the tongue worms have been classed with the Acarina (Sambon,[62] 1922). Heymons[63] points out that the difference between the pentastomids and annelids is far less than the difference between them and the arthropods.

These endoparasites inhabit primarily the lungs and air passages of their hosts. The body consists of a head or cephalothorax and a not distinctly separated abdomen. The head possesses five ventral openings: the median opening is the mouth, and the remaining two pairs lead to hollows from which chitinous hooks protrude. They are legless, vermiform, and pseudo-annulated. Development is indirect, in that an intermediate host is required.

The class Pentastomida is divided into two orders (1) Cephalobaenida and (2) Porocephalida. The order Porocephalida consists of two families (1) Porocephalidae, which includes among others the genus *Porocephalus* and the genus *Armillifer;* (2) Linguatulidae, which includes the genus *Linguatula,* to which the tongue worm *Linguatula serrata* Frölich belongs.

Pentastomiasis of man is commonly encountered, the cause being either linguatulids (linguatulosis) or porocephalids (porocephaliasis).

Linguatulosis infection is primarily due to ingestion of the eggs of *Linguatula serrata* Frölich (= *L. rhinaria* Moniez), the adult of which normally inhabits the nasal and frontal sinuses of the dog and wolf, occasionally also those of the fox. The adult female reaches a length of 80 to 120 mm and a breadth of 8 to 10 mm (Fig. 178). The males are hardly half this size. The eggs are discharged from the nostrils and mouth of the infected animal and are ingested by the intermediate host with food and water. Intermediate hosts under natural conditions are as a rule wild rabbits in whose body cavities the larvae travel freely, invading

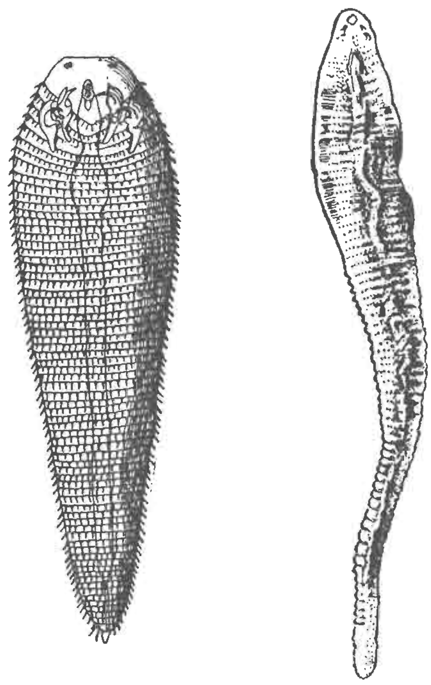


FIG. 178. *Linguatula serrata.* Larva (*left*), adult (*right*). (Redrawn after Brumpt.)

the liver and lungs; if infested rabbits are eaten by a dog, wolf, or fox the larvae develop into the mature form in these animals. Faust[64] has reported upon the presence of the immature form (encysted nymph) in the liver and lungs of a laboratory rabbit in Peking. He states that the material from the Peking rabbit averaged 4 mm in length by 0.8 mm in breadth.

Hobmaier and Hobmaier[65] report natural infections of brown rats (*Rattus norvegicus*) and artificial infections of ground squirrels (*Citellus beechyi*), both of which may contribute as intermediate hosts to the cosmopolitan distribution of *Linguatula rhinaria.*

Another linguatulid of man reported by Faust is *Armillifer moniliformis* (Diesing) from the liver of a Tibetan. This species as an adult described by Hill[66] is parasitic in the respiratory tract of pythons and has for nymphal host, among many others, such animals as mongooses, monkeys, ground squirrels and rats. *Leiperia gracilis* (Diesing) and a very closely related species, *Porocephalus crocodili* (Wheeler), occur in the crocodile.

BIBLIOGRAPHY

1. Banks, Nathan, 1915. *The Acarina or Mites.* Washington, D. C.: Dept. Agric., in Bur. of Entomol. Rep., no. 108. 153 pp.

2. Vitzthum, H. Graf, 1943. *Acarina, 7 Lieferung. Dr. H. G. Bronns' Klassen u. Ordnungen des Tierreichs.*, 5:913–1011. Akademische Verlagsgesellschaft, Becker u. Erler Kom. Ges.

3. Oudemans, A. C., 1937. *Kritisch Historisch Overzicht der Acarologie.* Tijdschrift voor Entomologie intzegeven door de Nederlandsche Vereeniging, Jan. 1926, (850 v. c. tot 1758), vol. G. (1805–1850), Leiden: E. J. Brill. 3379 pp.

4. Ewing, H. E., 1929. *A Manual of External Parasites.* Baltimore: C. C. Thomas. 225 pp. (96 figures).

5. Radford, C. D., 1943. "Genera and species of parasitic mites (Acarina)," *Parasitology,* 35:58–81.

6. Warburton, Cecil, 1920. "Sarcoptic scabies in man and animals," *Parasitology,* 12:265–300 (1 plate).

7. Mellanby, Kenneth, 1948. "Mites in relation to scabies," *Proc. Fourth Internat. Cong. Malaria and Trop. Dis.,* Washington, D. C. (Abstracts).

8. Mayo, N. S., 1910. *Some Diseases of Swine.* Blacksburg: Virginia Polytechnic Inst., in Agric. Exper. Sta. Bull., no. 189. 19 pp.

9. Cameron, A. E., 1942. *Mange in Horses, Cattle and Sheep.* Dominion of Canada: Dept. of Agric., in Health of Animals Div., Publ., no. 734 (Farmers' Bull. no. 109). 14 pp.

10. Law, James, 1909. *Textbook of Veterinary Medicine.* Ithaca, Vol. 5, 621 pp.

11. Skidmore, L. V., 1934. "Acariasis of the white rat *(Rattus norvegicus* form *albinus),*" *Canad. Entomologist.,* 46:110–15.

12. Froggatt, W. W., 1911. "'Aphis foot' of horses in the Tamworth District," *Agric. Gazette of New South Wales,* Sept. 2, pp. 789–91.

13. Imes, Marion, 1935. *Sheep Scab.* Washington, D. C.: Dept. of Agric., in Farmers' Bull., no. 713. 29 pp.

14. Hirst, Stanley, 1919. *Studies on Acari. No. 1: The Genus Demodex, Owen.* London: British Museum (Natural History). iii + 44 pp. (13 plates).

15. Fonseca, Flavio da, 1948. "A monograph of the genera and species of Macronyssidae Oudemans, 1936 (synom.: Liponissidae Vitzthum, 1931) (Acari)," *Proc. Zool. Soc. London,* 118 (part 2): 249–334.

16. Hirst, S., 1913. "On three new species of gamasid mites found on rats," *Bull. Entomolog. Research,* 4:119–24.

17. Bishopp, F. C., 1923. *The Rat Mite Attacking Man.* Washington, D. C.: Dept. Agric., in Department Circular, no. 294. 4 pp.

18. Dove, W. E., and Shelmire, B., 1932. "Some observations on tropical rat mites and endemic typhus," *J. Parasitol.*, **18**:159–68, 3 plates.

19. Williams, R. W., and Brown, H. W., 1945. "The development of *Litomosoides carinii,* filariid parasite on the cotton rat in the tropical rat mite," *Science,* **102**:482–83.

20. Bertram, D. S.; Unsworth, K.; and Gordon, R. M.; 1946. "The biology and maintenance of *Liponyssus bacoti* Hirst, 1913, and an investigation into its rôle as a vector of *Litomosoides carinii* to cotton rats and white rats, together with some observations on the infection in the white rats," *Ann. Trop. Med.*, **40**:228–52.

21. Philip, Cornelius B., and Hughes, Lyndahl E., 1948. "The tropical rat mite, *Liponyssus bacoti,* as an experimental vector of rickettsial pox," *Am. J. Trop. Med.*, **28**:697–705.

22. Wood, H. P., 1920. *Tropical Fowl Mite in the United States.* Washington, D. C.: Dept. of Agric., in Cir. no. 79. 8 pp.

23. Cameron, Donald, 1938. "The northern fowl mite, *Liponyssus sylviarum* C. & F., 1877," *Canadian J. Research,* Sec. D, **16**:230–54.

24. Ewing, H. E., 1922. "The dermanyssid mites of North America," *Proc. U. S. Nat. Museum,* **62** (no. 2459, art. 13): 1–26.

25. ———, 1936. "A short synopsis of the North American species of the mite genus *Dermanyssus,*" *Proc. Entomolog. Soc. Washington,* **38**:47–54.

26. Bishopp, F. C., and Wood, H. P., 1939. "Mites and lice on poultry," Washington, D. C.: Dept. of Agric., in Farmers' Bull., no. 801. pp. 1–26 (revised).

27. Wisseman, Charles L., Jr., and Sulkin, S. Edward, 1947. "Observations on the laboratory care, life cycle and host of the chicken mite, *Dermanyssus gallinae,*" *Am.J.Trop. Med.*, **27**:463–68.

28. Huebner, Robert J.; Jellison, William L.; and Pomerantz, Charles; 1946. "Rickettsialpox–a newly recognized Rickettsial disease. IV. Isolation of a Rickettsia, apparently identical with the causative agent of Rickettsialpox from *Allodermanyssus sanguineus,* a rodent mite." U. S. Public Health Service, *Pub. Health Rep.*, **61**:1677–82.

29. Greenberg, Morris; Pellitteri, Ottavio J.; and Jellison, William L.; 1947. "Rickettsialpox, newly recognized rickettsial disease. III Epidemiology." *Am. J. Pub. Health,* **37**:860–68.

30. Turk, F. A., 1947. "Studies of Acari. IV. A review of the lung mites of snakes," *Parasitology,* **38**:17–26.

31. Hammon, W. McD.; Lundy, H. W.; Gray, J. A.; Evans, F. C.; Bang, F.; and Izumi, E. A.; 1942. "A large-scale serum neutralization survey of certain vertebrates as part of an epidemiological study of encephalitis of western equine and St. Louis types," *J. Immunol.*, **44**:75–86.

32. Smith, Margaret G.; Blattner, Russell J.; and Heys, Florence, M.; 1944. "The isolation of the St. Louis encephalitis virus from chicken mites *(Dermanyssus gallinae)* in nature," *Science,* **100**:362–63.

33. Sulkin, S. Edward, 1945. "Recovery of equine encephalomyelitis virus (western type) from chicken mites," *Science,* **101**:381–83.

34. Howitt, B. F.; Dodge, H. R.; Bishop, L. K.; and Gorrie, R. H.; 1948.

"Virus of eastern equine encephalomyelitis isolated from chicken mites *(Dermanyssus gallinae)* and chicken lice *(Eomenacanthus stramineus)*," *Proc. Soc. Exper. Biol. & Med.*, 68:622–25.

35. Smith, Margaret G.; Blattner, Russell, J.; Heys, Florence M.; and Miller, Albert; 1948. "Experiments on the rôle of the chicken mite, *Dermanyssus gallinae*, and the mosquito in the epidemiology of St. Louis encephalitis." *J. Exper. Med.*, 87:119–38.

36. Hammon, W. McD., 1947. "The arthropod-borne virus encephalitides," "The Charles Franklin Craig Lectures," 1947, *Am. J. Trop. Med.*, 28:515–25.

37. Reeves, W. C.; Hammon, W. McD.; Furman, D. P.; McClure, H. E.; and Brookman, B.; 1947. "Recovery of western equine encephalomyelitis virus from wild birds *(Liponyssus sylviarum)* in Kern County, California," *Science*, 105:411–12.

38. Sulkin, E. S., and Izumi, E. M., 1947. "Isolation of western equine encephalomyelitis virus from tropical fowl mites, *Liponyssus bursa* (Berlese)," *Proc. Soc. Exper. Biol. and Med.*, 66:249–50.

39. Ewing H. E., 1944. "The trombiculid mites (chigger mites) and their relation to disease," *J.Parasitol.*, 30:339–65.

40. Brennan, James M., 1947. "New species of chiggers (Acarina, Trombiculidae) from bats of the Nearctic region," *J. Parasitol.*, 33:245–52.

41. Wharton, G. W., 1948. "Four new Peruvian chiggers (Acarina, Trombiculidae)," *Psyche*, 55:87–100.

42. Williams, Roger W., 1944. "A bibliography pertaining to the mite family Trombidiidae," *Am. Midland Naturalist*, 2:699–712.

43. Jenkins, Dale W., 1948. "Trombiculid mites affecting man. I. Bionomics with reference to epidemiology in the United States," *Am. J. Hyg.*, 48:22–35.

44. Michener, Charles D., 1946. "Observations on the habits and life history of a chigger mite, *Entrombicula batatas* (Acarina: Trombiculinae)," *Ann. Entomolog. Soc. Amer.*, 39:101–18.

45. Miller, A. E., 1925. "The native host of the chigger," *Science*, 60:345–46.

46. Keay, Gladys, 1937. "The ecology of the harvest mite *(Trombicula autumnalis)* in the British Isles," *J.Animal Ecology*, 6:23–35.

47. Ewing, H. E., 1921. *Studies on the Biology and Control of Chiggers.* Washington, D. C.: Dept. Agric., in Bull. no. 986. 19 pp.

48. Michener, Mary H., and Michener, Charles D., 1947. "Chiggers!" *Natural History*, 56:231–35.

49. Williams, Roger W., 1946. "A contribution to our knowledge of the bionomics of the common North American chigger, *Entrombicula alfreddugèsi* (Oudemans), with a description of a rapid collecting method," *Am. J. Trop. Med.*, 26:243–50.

50. Blake, Francis G.; Maxcy, Kenneth F.; Sadusk, Joseph, F., Jr.; Kohls, Glen M.; and Bell, E. John; 1945. "Studies on Tsutsugamushi disease (scrub typhus, mite-borne typhus) in New Guinea and adjacent islands: Epidemiology, clinical observations and etiology in the Dobadura area," *Am. J. Hyg.*, 41:243–373.

51. Miyajima, M., and Okumura, T., 1917. "On the life cycle of the

'Akamushi,' carrier of Nippon River fever," *Kitasato Arch. Exper. Med.*, 1: 1–14.

52. Nagayo, M.; Miyagawa, Y.; Mitamura T.; Tamiya, T.; Sata, K.; Hazato, H.; and Imamura, A., 1931. "Ueber den Nachweis des Erregers der Tsutsugamushi-Krankheit, der *Rickettsia orientalis*," *Japanese J. Exper. Med.*, 9:87–150.

53. Hayashi, N., 1920. "Etiology of Tsutsugamushi disease," *J. Parasitol.*, 7:53–68.

54. Steinhaus, Edward A., 1946. *Insect Microbiology, an Account of the Microbes Associated with Insects and Ticks with Special Reference to the Biologic Relationships Involved.* Ithaca, N. Y.: Comstock Publishing Co., Inc. x + 763 pp. (250 figs.).

55. Philip, Cornelius B., 1948. "Tsutsugamushi disease (scrub typhus) in World War II," *J. Parasitol.*, 34:169–91.

56. ——, and Kohls, Glen M., 1948. "Mites and scrub typhus," *Proc. 4th. Internat. Cong. Trop. Med. and Malaria.* (Abstracts) Washington, D. C.

57. Wharton, G. W., 1947. "Studies on North American chiggers. I The "Akamushi" group," *J.Parasitol.*, 33:260–64.

58. Goldberger, J., and Schamberg, J. F., 1909. "Epidemic of an urticarioid dermatitis due to a small mite *(Pediculoides ventricosus)* in the straw of mattresses." U. S. Public Health Service, *Pub. Health Rep.*, 24:973–75.

59. Webster, F. M., 1910. *A Predaceous Mite Proves Noxious to Man.* Washington, D. C.: Dept. Agric., in Bur. Entomol. Circ., no. 118. 24 pp.

60. Hinman, E. H., and Kampmeier, R. H., 1934. "Intestinal acariasis due to *Tyroglyphus longior* Gervais," *Am. J. Trop. Med.*, 14:355–62.

61. Rebrassier, R. E., and Martin, E. D., 1932. "*Syringophilus bipectinatus*, a quill mite of poultry," *Science*, 76:128.

62. Sambon, L. W., 1922. "A synopsis of the family Linguatulidae," *J. Trop. Med.*, 25:188–206, and 391–428.

63. Heymons, R., 1935. *Pentastomida. Dr. H. G. Bronns. Klassen und Ordnungen des Thierreichs.* 5 Band: Arthropoda. iv. Abteilung Arachnoidea. 1 Buch Pentastomida. 268 pages (148 text figures).

64. Faust, E. C., 1927. "Linguatulids (Order Acarina) from man and other hosts in China," *Am. J. Trop. Med.*, 7:311–22 + 2 plates.

65. Hobmaier, A., and Hobmaier, M., 1940. "On the life-cycle of *Linguatula rhinaria*," *Am. J. Trop. Med.*, 20:199–210.

66. Hill, H. R., 1934. "The occurrence of linguatulids in Pythons," *Bull. South. Calif. Acad. Sc.*, 33:117–22.

CHAPTER XXII

VENOMOUS AND URTICARIAL ARTHROPODS

Insect venoms. Insect venoms, like other animal venoms, are toxic principles probably not greatly unlike the bacterial toxins, but about which we know comparatively little. Unlike many of the bacterial toxins which reach toxic proportions only after a period of elapsed time subsequent to the introduction of the infection into the body, the animal venoms take effect almost instantly, i.e., as soon as introduced and without incubation.

The venoms act in one or more ways when introduced into the body: (1) they may act directly on the blood corpuscles (*hemolytic*); (2) they may act directly on the nervous system, producing shock or inhibiting reflexes (*neurotoxic*); (3) they may produce an infiltration and congestion of blood (*hemorrhagic*) often in the vicinity of the wound or in deeper tissue, such as the mesenteries, etc. A given arthropod venom may act in one or more of these ways.

It is a matter of common observation verified by various investigators that repeated inoculation of minute or attenuated quantities of a venom may lead to a degree of immunity, so it is also with the venoms or poisons of bees, bedbugs, mosquitoes, fleas, conenoses, etc. Newcomers to a flea-infested community may suffer great misery until they have acquired an "immunity"; this may require many months and may never be acquired by some individuals. Promising results have been obtained by administering a course of immunization consisting of injections (six) of a flea antigen[1] [extract of *Pulex irritans* (75 per cent) and *Ctenocephalides canis* (25 per cent)] given subcutaneously one week apart.

In the ants, bees, and wasps (aculeate Hymenoptera) there are two poison-secreting glands, one of which is said by some authors to secrete *formic acid,* and the other an *alkaline* fluid. The combination of the two agents in certain proportions is believed to be necessary to produce lethal effect on other insects. Koszalka[2] points out that "bee venom is considered to be a complex organic compound, containing lecithin and an albumin-free sapotoxin, which produces histamine-like effects and has a potential hemolytic action."

The scorpion (an arachnid) secretes a large quantity of colorless acid-reacting liquid soluble in water and heavier than water. According to Calmette, less than 0.0005 gm from *Buthus afer* Leach will kill a white mouse in about two hours.

How the venom is introduced. Arthropod venoms are introduced into the body of man and other animals in one of three ways: (1) by *contact* e.g., by means of urticarial hairs of certain caterpillars, such as the brown-tail moth, *Nygmia phaeorrhoea* Don. [= *Euproctis chrysorrhoea* (Linn.)], producing a condition similar to nettling, or with vesicating fluids of the blister beetles (Meloidae), particularly *Lytta vesicatoria* (Linn.), resulting in a *vesicular dermatitis;* (2) by the *bite,* thrust of piercing mouth parts, as in the conenoses (Reduviidae), or penetration of the chelicerae of spiders; (3) by the *sting,* as in the bees and wasps (aculeate Hymenoptera) and the scorpion. The operation and structure of the sting of insects and arachnids differ considerably.

Stinging insects. The stinging insects belong to the order Hymenoptera, suborder Aculeata, the ants, bees, and wasps, in which the females of all species are provided with a specialized ovipositor known as a sting, more or less well developed for piercing the skin of higher animals or other insects. The sting is used either as an organ of defense or offense; in the latter case often to procure food for the young. The venom apparatus of the aculeate Hymenoptera, such as bees, wasps, hornets, and bumblebees, does not differ greatly in structure.

The principal aculeate Hymenoptera are divided into the following super-families, viz.: Formicoidea, the true ants; Sphecoidea, the digger wasps; Vespoidea, the true wasps; and Apoidea, the bees.

Morphology of bee sting. The sting of the worker bee is regarded as a specialized ovipositor; it originates from the seventh and eighth segments and lies between the oviduct and the rectum above. The darts of the sting follow the ventral line of the abdomen and are held in place by the sheath situated above, while the barbs of the darts point downward and outward. In a space above the sheath lie the fleshy palpi (Fig. 179). The delicate attachment between the sting and the organs of the abdomen accounts for the ease with which the sting is torn from the abdomen when the barbs become embedded after the darts are thrust into the skin. The sting can be easily extracted either by separating the segments of the abdomen from it by means of dissecting needles, or by squeezing the live bee between forceps, which causes it to protrude the sting. The sting can then be grasped with other forceps and drawn out. After extraction, the sting can be best examined when the parts are floated out in a few drops of glycerin. The sting may be divided into three parts, viz.: the piercing apparatus; the lateral plate and appendages; the poison sac and glands.

The piercing apparatus itself consists of three parts, one the so-called sheath, the other two lying within the sheath, and partially surrounded by it. In appearance the sheath is yellowish and translucent. The darts, which present concave surfaces to one another, are highly chitinous. The distal one-third of the dart possesses a series of sharp

FIG. 179. Sting of a honeybee, *Apis mellifera: a,* the two serrated darts; *b, b',* sting palpi; *c,* venom (poison) sac; *d,* venom gland; *e, e',* triangular plates or levers; *f, f',* semilunar plates or levers; *g, g',* lateral plates or levers; *h, h',* Y-shaped darts; *i, i',* points of attachment for darts to levers; *e, e', j, j',* points around which levers rotate; *k, k',* points of attachment for lever (*f, f'*). ×17.5.

barbs, whose shape has been aptly compared to the tip of a crochet needle. Cheshire states that each dart has from three to six barbs, other writers seem doubtful as to the number. Many darts have been carefully examined by the writer, but in no instance were less than *ten* barbs distinguished on the outer edge of each dart (Fig. 179). Several writers state that poison pores are to be found at the base of each dart, from which poison exudes. In this matter the writer agrees with Snodgrass, as he has failed to observe the exit of poison elsewhere than between the darts at their tip.

Proceeding upward on the dart from the tiny barbs, the darts are seen to form a "Y" as they lie within the sheath. The arms of the "Y" gradually bend laterally. Plates are attached to the upper edges of these laterally bent arms. One of the most remarkable portions of the darts is the poison valve with which each is provided. At the point of separation the darts each present a delicate cup-shaped valve, whose closed portion is directed downward toward the tip of the sting. This is formed of the same chitinous material as the darts, and each is free to move with the movement of the dart. In order to accommodate this enlargement of the darts, the sheath at this point expands to about five times its smallest diameter, which is at the tip of the sting. For at least one-third of its length the sheath at this portion is expanded into a symmetrical oblong body providing ample room for the movement of the darts and valves within.

A curious structure, said by many writers to be found on the sheath, consists of two delicate, but strong, chitinous tracks or guide rails on which the darts, correspondingly grooved, fit and move back and forth. Since the sheath does not sufficiently surround the darts to direct their course, this guide-rail system which Carlet has observed, and which is accepted by other authors, probably explains why the darts move smoothly and accurately within the sheath.

The *lateral appendages* are of three kinds: semilunar, triangular, and lateral, according to shape or position. Both the *semilunar and triangular plates* are attached to the bent ends of the Y-shaped darts. The triangular plates are attached to the arms of the darts almost at their extremities, while the semilunar ones are connected for about one-third of the distance from the ends of the arms. The apex of the triangle is attached to the extremity of the dart. The other two points are directed outward and downward, and serve as points of attachment for two elevated edges on the lateral plates which hang thus suspended. As they hang, half of their surface lies above and covers the dorsal surface of the semilunar plates just beneath them. Continuing in the same straight line with the semilunar plates and attached at their extremity to them, lie the fleshy *palpi* covered with delicate hairs.

The third set of structures which completes the sting are the *venom sac and glands.* In order to understand these it is necessary to know that Hymenoptera are divided into two groups, those which kill their prey by stinging, and those which only paralyze it. The former are the more complicated, for they possess two poison glands; the acid gland, which opens directly into the great poison sac, the larger of the two, and the other, the alkaline gland, which is comparatively small and is situated at the base of the poison sac. It is the combination of the acid and alkaline fluids from the two glands that results in the death of the attacked insect, or that causes the extreme pain and resulting reactions in humans.

The formic acid gland alone is found in those Hymenoptera which only paralyze their prey by their sting. This fact has led various observers to make chemical tests of both the formic acid and alkaline substance. The result, according to Carlet and others, has been to show that neither substance by itself is effective except to paralyze, but when combined the substances have deadly effects upon other insects. Carlet's experiments to prove this were made on houseflies and blowflies by injecting each substance singly and then introducing both into the body of a fly.

Operation of the sting. The sting may be observed in operation by confining the bee on its back and then prodding it until its sting is angrily thrust in and out. This process shows three things, viz.: that the sharp-pointed sheath always appears first when the thrust is made; second, that the darts inside the sheath work back and forth alternately and quite independently of the sheath or of one another; third, that the poison exudes in droplets from the tip of the sting between the darts. Cheshire (*Bees and Bee-Keeping*, London, 1886) states that

> The sheath has three uses: first, to open the wound; second, to act as an intermediate conduit for the poison; and third, to hold in accurate position the long-barbed darts. The sheath does not inclose the darts as a scabbard, but is cleft down the side which is below, when the sting points backward. The darts, as soon as their ugly barbs establish a hold, first one and then another drive back and forth by successive blows. These in turn are followed by the sheath, when the darts again plunge more deeply, until the murderous little tool is buried to the hilt. But these movements are the result of a muscular apparatus yet to be examined. The dovetail guide-rails of the sheath are continued far above its bulbous portion, and along with these the darts are also prolonged upward, still held to the guides by the grooved arrangement; but both guides and darts, in the upper part of their length, curve from each other like the arms of the Y, before mentioned, to points *C, C'* (Fig. 179) where the darts make attachment to two levers *(i, i')*. The levers, or plates, as they are called *(Kl and K'l")*, are provided with broad muscles, which terminate by attachment to the lower segments of the abdomen. These, by contraction, revolve the levers aforesaid round the points *f, f'*, so that without relative movement of rod and groove, the points *c, c'* approach each other. The arms of the Y straighten and shorten, so that the sheath and darts are driven from their hiding place together and the thrust is made by which the sheath produces its incision and fixture. The sides being symmetrical, we may, for simplicity's sake, concentrate our attention on one, say the left in the figure. A muscular contraction of a

broad strap joining *K* and *D* (the dart protractor) now revolves *k* on *l*, so that *a* is raised, by which clearly *c* is made to approach *d*; that is, the dart is sent forward, so that the barbs extend beyond the sheath and deepen the puncture. The other dart, and then the sheath, follow, in a sequence already explained, and which *G* is intended to make intelligible, representing the entrance of the sheath, *b* the advance of the barbs, and *c* the sheath in its second position. The barb retractor muscle is attached to the outer side of *i*, and by it *a* is depressed and the barbs lifted. These movements, following one another with remarkable rapidity, are entirely reflex, and may be continued long after the sting has been torn, as is usual, from the insect. By taking a bee under the microscope and forcing the sting into action, the sting movement will be seen to be kept up by continued impulses from the fifth abdominal ganglion and its multitudinous nerves, which penetrate every part of the sting mechanism and may be traced even into the darts. These facts will show why an abdomen separated many hours may be able to sting severely.

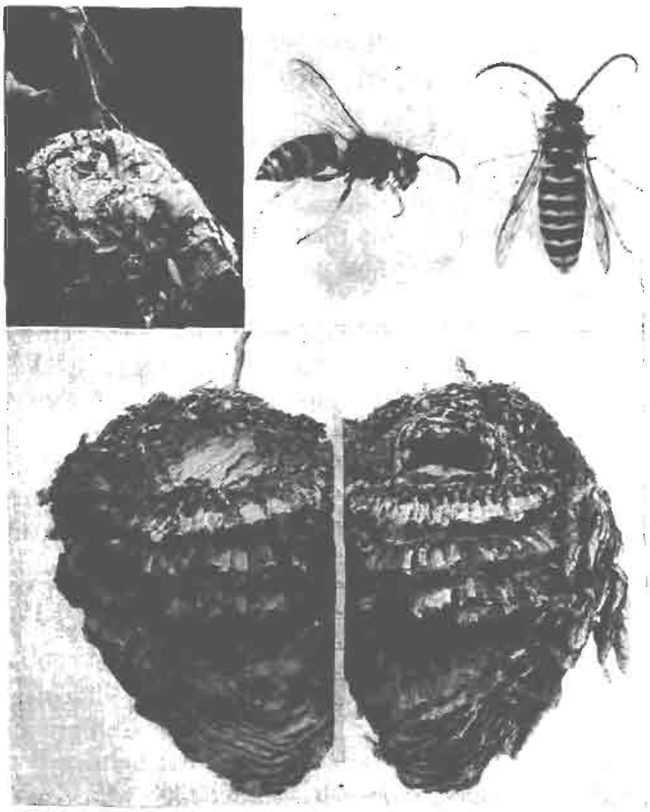
Reaction to bee stings. The painful effect of the sting of bees is probably not due to the simple thrusts of the barbs already described, but more particularly to the introduced venom. The severity is greatly increased in the case of multiple stings; however, one sting in a previously sensitized person or one inflicted at a vital point (e.g., a vein) may result fatally. Pawlowsky, according to Martini,[3] estimates 500 bee stings within a short time as a lethal dose for man. Tolerance to bee stings appears to be developed in beekeepers, long exposed to stings; however, this tolerance seems to be lost when exposure to stings is discontinued.

Allergic reactions are generally recognized as due to bee protein and pollen adhering to the bee. Death may result from anaphylactic shock.

Treatment for bee sting. If the sting remains in the wound, it must be removed quickly and carefully so as not to force additional venom into the puncture. Repeated and long-continued application of a bicarbonate of soda paste (soda with water enough to make a paste) is recommended. Eau de Javelle 1 to 100 is also recommended, as well as calcium chloride in water 1 to 60; application of ammonia or alcohol will also give some relief. If symptoms of envenomization arise, a physician should be consulted. Kozalka (see above) has recently (1949) reported rapid recovery from multiple bee stings attributable to the parenteral use of calcium gluconate.

Yellow jackets or hornets such as *Vespula* (= *Vespa*) *diabolica* (Sauss.), *Vespula* (= *Vespa*) *maculata* (Linn.), and *Vespula pennsylvanica* (Sauss.) are members of the family **Vespidae** (Order **Hymenoptera**). There are also several European species, among them *Vespa crabro* Linn., *Vespa vulgaris* Linn., and *Vespa germanica* Fabr. All of these species build **nests** (Fig. 180) of pulp made by masticating wood fiber. Nests are built in hollow trees, among the branches, under eaves of houses, and in holes in the ground. The somewhat elongate aerial nests may reach nine to ten inches in diameter.

The young fertilized queens hibernate throughout the winter and start the nest in the spring. In the control of these insects one should seek out the new nests under construction and destroy them by night



'IG. 180. Aerial nest of yellow jacket; side and top view of the insect, *Vespula diabolica;* and longitudinal section of nest below.

when the queen and her progeny are at home. The aerial nests may be cautiously and quickly enveloped at night with a tight drawstring bag and then soaked with kerosene, scalding water, or burned. The underground nests should be located by day and the entrance marked, then treated by pouring an ounce or two of carbon bisulfide into the hole at night and quickly pressing the entrance shut with the foot. An old-fashioned yet effective and safe way to handle a yellow jacket problem is to place a gallon jug, quarter filled with water, near the nest. As these insects investigate this new object, the buzzing sound echoing from the jug prompts them to enter, and as a result fall into the water and are drowned. In this way one by one the entire colony is lured into the jug (Melander).

The small nests with exposed cells made by such forms as *Polistes bellicosus* Cresson may be removed at night by quickly placing a tin can or box with open end over the nest and then slipping a sheet of tin between the surface and the open end of the can so that the nest is cut away. The surface from which the nest has been removed may be daubed with creosote to repel other female hornets from starting a nest on the same site.

Mud daubers or thread-waisted wasps such as *Sphex (Psammophila) luctuosa* Smith, *Sphex violaceipennis* Lepeletier, *Chalybion coeruleum* (Linn.), and *Pelopaeus californicus* Sauss., make their nests, usually quite small, of mud and provision them with insects and spiders to serve as food for the larvae. These mud nests may be removed as suggested above for *Polistes*.

Ants. The true ants belong to the family **Formicidae** (Order **Hymenoptera**), which comprises about 8,000 described species, divided into five or more subfamilies, e.g., Formicinae, Ponerinae, Dorylinae, Myrmicinae, and Dolichoderinae.

The subfamily Myrmicinae includes several formidable stinging species of pugnacious nature. Because of their numbers a mass attack may result seriously. Among the more dangerous species are: (1) the California or Mexican fire ant, *Solenopsis xyloni* var. *maniosa* Wheeler, the workers of which have a yellowish-red head and a black thorax and abdomen. They measure from 1.6 to 5.8 mm in length. (2) The Texas harvester or agricultural ant, *Pogonomyrmex barbatus* (F. Smith), in which the head, thorax, and legs are black and the abdomen red; the workers reach from 7 to 9 mm in length; (3) the California harvester ant, *Pogonomyrmex californicus* (Buckley), with a light rusty-red body, and legs somewhat more yellowish. These ants will readily attack humans and smaller animals. Young pigs may be killed by the stings of this species. A small pig may walk leisurely upon an ant mound and suddenly begin to kick and squeal, due to the terrific attack of ants rushing forth from the nest. The animals commonly topple over with legs outstretched and death may ensue.

Ants belonging to the subfamily Ponerinae also have well-developed stings and a potent venom. This is apparently particularly true of the Central and South American species, *Paraponera clavata* (Fabr.), which is common in high tropical rain forests. Weber[4] states that the ants of this species boil out of their nests in large numbers when disturbed and rush for the intruder. He states that the workers are fully an inch in length and blackish brown in color. The ant is greatly dreaded by the natives.

For the control of the fire ant, Mallis[5] recommends the use of a mixture of one part of carbon bisulfide and three parts of carbon tetrachloride injected into the nest openings by means of an oil can with an eight-inch curve-tip spout. A few drops of the liquid in each opening are sufficient. After treatment the openings must be covered. A large measure of control may be effected by applying kerosene to the nests, using a funnel or hollow rod to reach the deeper parts; potassium cyanide in liquid form may also be used, but great care must be exercised both in its preparation and application owing to its very poisonous nature.

FIG. 181. A velvet ant, Mutillidae, also known as a "cow killer." ×2.2.

Mutillid wasps. Among the less known stinging insects are the mutillid wasps belonging to the family **Mutillidae** (**Order Hymenoptera**). Members of this family are commonly known as velvet ants, woolly ants, cow killers, mule killers, etc. (Fig. 181). The mutillids are covered with a velvety pubescence; many are brightly colored with orange or red or yellow. The females are apterous, good runners, and may inflict a painful sting. They are parasites of bees and other wasps. There are very many species, some of the commoner forms measuring from ½ to 1 inch in length. Our knowledge concerning these interesting insects has been greatly advanced by Mickel.[6] A very common species in the central United States is *Sphaerophthalma occidentalis* (Linn.), a black species with a scarlet band. This species is very common on the beach sands of Lake Erie, causing barefoot bathers much distress.

Stinging Eypris. In 1927 von Geldern[7] reported a tiny wasp from Yolo County, California, identified as belonging to the genus *Eypris* which inflicts a severe sting.

The wasps appear in fairly great numbers in the fall after a warm spell and invade the house where they get into the bedding and clothing, and sting when brushed or crushed by clothing or sheets against the skin. . . . The sting is distinctly felt as a fairly sharp prick, decidedly less intense than a bee sting. . . . In the oldest and youngest child no further manifestations occur, but in the parents and second child a decided systemic disturbance follows. A few minutes after being stung, there is felt a numbness, often at the site of the sting, but at other times beginning at the finger tips. It remains localized for a few minutes and then gradually spreads and involves the entire body. In the mother there is an intense itching of the vulva and in the father an itching of the pubes. This is followed by a marked diarrhoea, not painful in the father, but resembling severe uterine cramps in the mother. The diarrhoea and cramps last for about ten minutes. The mother, who is an asthmatic, experiences no respiratory difficulty, but with the father, who has never had an attack of asthma, wheezing occurs occasionally. Accompanying these symptoms there is marked prostration, weakness and sweating. The duration of the attack is about half an hour. The second child becomes drowsy and is awakened with difficulty and wheezing occurs. He also recovers in about the same time as the parents.

Essig[8] in 1932 reported a number of instances of *Epyris* stings all from the same county in California as were the cases reported by von Geldern. The species of wasp was identified as *Epyris californicus* (Ashmead) belonging to the proctotrupid family Bethylidae. It measures barely over 5 mm in length and is black in color. Essig states, "concerning the life history and habits of this particular species, beyond its propensity for stinging, absolutely nothing is known."

BITING (PIERCING) INSECTS

Insects that pierce the skin with their mouth parts are usually normally bloodsuckers and the act of biting or piercing is simply a part of the act of food-getting. There are noteworthy exceptions, as later explained. The pain caused by the mechanical insertion of such tiny mouth parts would be, no doubt, relatively benign, particularly if only one or only a few individual insects were concerned in the attack; however, in perhaps every instance a venom of salivary origin is introduced. These venoms apparently differ among the various species, as evidenced by the resulting reactions, local and systemic, which are generally specific enough so that one who is experienced may be able to determine the cause, i.e., whether the offender was a bedbug, a flea, a mosquito, or a black fly (simuliid). Rubbing the bite with cotton saturated with chloroform or carbon tetrachloride will relieve the irritation.

To understand the operation of the bloodsucking mechanism of the various offending insects one should consult the previous chapter on mouth parts and the other chapters appropriate to this subject. The student will profit much by a careful study of "Zoönosen der Haut in wärmeren Ländern" by Martini (*loc. cit.*). In general the reactions are the result of both *mechanical* and *chemical* processes.

Some individuals apparently suffer no ill effect from bedbug bites, not even a swelling at the site of the bite; others react violently to even one bite. These differences in tolerance to a given species are not fully understood. Martini remarks that doubtless allergic processes play a role in these reactions.

Insects that inflict a very painful bite, such as the stable fly, *Stomoxys calcitrans* (Linn.), and most salt-marsh mosquitoes, e.g., *Aedes dorsalis* (Meig.), are apparently seldom vectors of disease, while species with benign bites, such as *Anopheles maculipennis* (Meig.), are commonly potent vectors. May it not be that in order to become a successful vector of disease, the arthropod must first modify the severity of its bite!

Conenoses or kissing bugs, belonging to the family **Reduviidae** (see Chapter VIII), commonly inflict painful "bites." Their mouth parts (see Chapter VI) are well adapted for piercing the skin of the host. Reduviids are essentially predaceous, attacking many species of insects, particularly soft-bodied forms from which they suck the body fluids. Attack upon humans is made principally, if not wholly, in self-defense. Persons picking up boards, sticks, or stones, etc., may accidentally pick up one of these insects; likewise in plucking a leaf or flower from a tree or other plant the fingers may close upon the insect as well, with the result that a very painful bite may be suffered.

The principal offenders are about 18 to 20 mm in length, and all bear a general resemblance to the illustrations, Figs. 40 and 41. Among the species inflicting painful bites are the following: *Reduvius personatus* (Linn.), known as the "kissing bug"; *Triatoma sanguisuga* (Lec.), the "bloodsucking conenose" or "big bedbug"; *Triatoma protracta* (Uhler), the "China bedbug"; and *Rasahus biguttatus* (Say), the "two-spotted corsair."

The symptoms produced by *Triatoma protracta* (Uhler), the usual offender in California, are described as follows: "In a few minutes after a bite the patient develops nausea, flushed face, palpitation of the heart, rapid breathing, rapid pulse, followed by profuse urticaria all over the body. The symptoms vary with individuals in their intensity."

The symptoms described for *Rasahus biguttatus* (Say) are as follows: "Next day the injured part shows a local cellutitis with a central spot; around this spot there frequently appears a bulbous vesicle about the size of a ten-cent piece and filled with a dark grumous fluid; a smaller ulcer forms underneath the vesicle, the necrotic area being generally limited to the central part, while the surrounding tissues are more or less swollen and somewhat painful."

Biting water bugs. The order **Hempitera** (see Chapter VIII) contains a number of families of aquatic forms several of which included biting species; among these are the families **Belostomatidae** and **Notonectidae.**

The giant water bugs *Lethocerus, Belostoma,* and *Benacus,* belonging to the family **Belostomatidae,** are among the largest of the water bugs, measuring 2½ to 4½ inches in length and possessing formidable beaks. They feed on other aquatic insects, also young frogs, fish, etc., and since they are winged and readily attracted to lights, they are commonly known as electric light bugs. They have been known to attack birds. Ewing[9] describes the effect of the bite as follows:

> . . . at 9:30 A.M. a giant water bug *Benacus griseus* (Say) was allowed to bite the back of the right index finger. The beak was left inserted for a few seconds. Immediately a burning sensation followed. Two minutes later the same bug was allowed to puncture the back of the left index finger for several seconds. A burning sensation was produced. Soon some swelling was noted, and a reddened area developed about the point of the puncture. Pain continued but diminished during the forenoon and by noon the reddened area had become reduced. By 1:30 P.M. a small red spot was all that was left at the puncture. . . . When *Benacus griseus* bites it emits a milky fluid from the tip of the beak, and the beak adheres to the skin after penetration, so that the skin is pulled up when the beak is withdrawn.

Back swimmers belonging to the family **Notonectidae** may also inflict a painful bite. These predaceous bugs swim on their backs, hence the common name "back swimmers." The bite is nearly as severe as a bee sting.

Bloodsucking phytophagous bugs. Numerous instances of bloodsucking among phytophagous Hemiptera have been reported. Much information concerning these cases has been assembled by Usinger (1934, *loc. cit.*) and Myers[10] (1929).

Among the species exhibiting this fortuitous bloodsucking behavior are members of the following families: **Membracidae** (treehoppers) such as *Ceresa bubalus* (Fabr.); **Cicadellidae** (leafhoppers and sharpshooters) such as *Eutettix tenellus* (Bak.) and *Erythroneura comes* (Say); **Miridae,** such as *Irbisia solani* (Heid.) and *Sopidae marginata* Uhler; **Corizidae,** *Leptocoris trivittatus* (Say).

Usinger remarks that the change from the sucking of plant juices to bloodsucking at first appears to be very great. "However, upon comparison of the chemical constituents, it is found that in general the same elements are found in plants as in blood and often in very similar combinations although in very different proportions."

Thrips biting man. Thrips (Order **Thysanoptera**) are minute plant-feeding (sapsucking) insects (see Chapter VI for description of mouth parts); however, there have been numerous reports of their attacking man and of their ability to suck blood. Bailey[11] states that while working on experimental plots he experienced bites from the onion thrips, *Thrips tabaci* Lind. He felt slight pricks on the arms, face, and neck, both when perspiring and when not. He observed that the larvae (second instar) were more prone to bite than the adults and that the alimentary canal took on a reddish-brown appearance after feeding. Small pinkish dots appeared on the skin and disappeared in one to two days. No swelling occurred but there was a slight itching sensation. He had similar experiences with the pear thrips, *Taeniothrips inconsequens* (Uzel).

Several other species of thrips have been reported in a similar connection, e.g., *Heliothrips indicus* Bagnall, a cotton pest of the Sudan; *Thrips imaginis* Bagnall, reported for Australia; *Limothrips cerealium* Haliday, for Germany; *Gynaitkothrips uzeli* Zimmerman, for Algiers; and other species. It would appear that many species of thrips are thus involved and that this behavior is not restricted to any one species.

Urticarial hairs. The caterpillars of many species of **Lepidoptera,** (at least eight families) possess urticating hairs. Among the families which have urticarial larvae is the **Saturniidae** of which the genus *Hemileuca* is especially offensive. *Hemileuca oliviae* Cockerell, the range caterpillar, is reported to be a menace to cattlemen in New Mexico.[12] A rash known as the "brown-tail rash" is traceable to the caterpillar of the brown-tail moth (*Nygmia phaeorrhoea* Don.), a common and very destructive shade tree pest in Europe and in America, especially in New England. When the caterpillars of this species molt, myriads of tiny barbed hairs are shed with the skin. The cocoons of the pupated caterpillars as well as the adult moths possess these hairs. These hairs are blown about by the wind and coming in contact with the skin of the neck, face, hands, or other exposed parts of the body produce a very severe dermatitis. The hairs are hollow, and it has been shown by Tyzzer[13] that they contain a definite poisonous principle which is injected into the circulation by the sharp-pointed hair in contact with the skin, thus producing the rash. Ingestion of the hairs by swallowing or inhaling in breathing may cause serious internal disturbances.

Bishopp[14] describes the symptoms produced by contact with the "puss" caterpillar (*Megalopyge opercularis* S. & A.) as follows:

> Almost immediately after any portion of the body comes in contact with one of these caterpillars an intense burning pain is felt, described by some as similar to a severe nettle sting. This usually becomes worse accompanied by itching for several minutes and persists from 1 to 12 hours and sometimes longer. Almost immediately after a sting the area touched by the caterpillar shows minute raised whitish spots or papules which soon become red, followed by spreading of the inflammatory area for several inches and often accompanied by general swelling of the portion of the body stung. Stings on the wrist

have been followed by a swelling of the entire arm to almost double its normal size. A feeling of numbness which almost assumes the characteristics of paralysis accompanies the swelling. This is usually confined to the member attacked but may be generalized. Apparently stings on the neck are even worse, as the writer has one record of a man who was stung severely on the neck and completely incapacitated, being confined to the hospital for 6 days. These paralytic symptoms are often accompanied by nausea and sometimes by vomiting. The stings are especially severe among young children, who often develop considerable fever and nervous symptoms. These sometimes last for a day or two and are accompanied by nausea, especially during the first few hours. Usually within two or three hours after a sting, the reddened pimple-like swellings at the site assume the appearances of small vesicles or blisters. These usually persist for a few hours and then apparently harden through absorption, leaving a roughened area. In some instances the discoloration of the skin surrounding the point of attack is rather marked, varying from a deep red to almost black. The paralytic symptoms usually subside with the pain, but the local lesions often persist for several days.

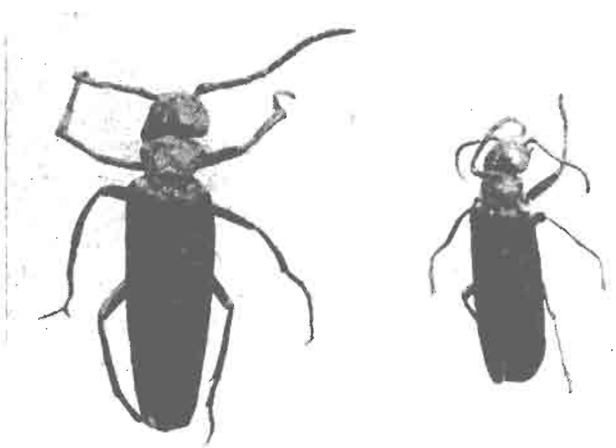
Students concerned with the subject of urticarial hairs will need to consult Weidner's[15] (1936) work which includes a comprehensive bibliography on poisonous insect hairs. Weidner lists the following families of Lepidoptera which include caterpillars with hairs causing skin irritation; namely, **Morphidae**, *Morpho hercules* Dalm.; **Arctiidae**, *Lithosia caniola* Hbn., *L. griseola* Hbn.; **Lymantriidae**, *Nygmia phaeorrhoea* Don., [= *Euproctis chrysorrhoea* (L.)], *Porthesia similis* Fuessly, and several others; **Thaumetopoeidae**, *Thaumetopoea pinivora* Tr., *Anaphe infracta* Wlsgh., and others; **Lasiocampidae**, *Macrothylacia rubi* (L.), *Dendrolimus pini* (L.), *Lasiocampa quercus* (L.), and others; **Noctuidae**, a few species occasionally cause irritation; **Nymphalidae**, larval hairs may pierce the skin, e.g., *Vanessa io* (L.) and *Hamadryas antiopa* (L.) (mourning cloak); **Saturniidae**, *Automeris io* (Fabr.) (Io moth), *Hemileuca maia* Drury (buck moth), and others; **Megalopygidae**, *Lagoa crispata* Pack. (white flannel moth), and others; **Limacodidae**, *Sibine stimulea* Clem. (the saddle-back caterpillar), and others.

Blister beetles. Blister beetles belong to the family **Meloidae** (**Cantharidae**) Order **Coleoptera** and are so designated because of their vesicating properties, i.e., the application of the pulverized bodies or even simple contact with the insect may produce a blistering of the skin.

The **Meloidae** (**Cantharidae**) are described by Comstock: "The blister beetles are of medium or large size. The body is comparatively soft; the head is broad, vertical and abruptly narrowed into a neck; the prothorax is narrower than the wing covers, which are soft and flexible; the legs are long and slender; the hind tarsi are four-jointed, and the fore and middle tarsi are five-jointed."

The blister beetles deposit their eggs on the ground; the larvae are active and feed, it is said, in some species on the eggs of locusts and solitary bees; others are predaceous. They undergo a number of changes not usual to insects, their development being termed "hypermetamorphosis." The adults are vegetable-feeding.

Spanish fly. The Spanish fly, *Lytta vesicatoria* (Linn.), is a European species of beetle found most abundantly during the early summer in Spain, southern France, and other parts of Europe (Fig. 182). It is golden green or bluish in color, and ranges from one-half to three-quarters of an inch in length. It makes its appearance quite suddenly in early summer, when it may be collected by the hundreds, clinging principally to such vegetation as ash, privet, and lilac. The peculiar hypermetamor-



A B

FIG. 182. The Spanish fly, *Lytta vesicatoria*; (*A*) female, (*B*) male.

phosis of these insects and the subterranean predaceous larval habits give to them some obscurity during their early development; the sudden appearance and equally sudden disappearance, owing to short adult life, give rise to the belief that they are migrating forms.

Cantharidin, a crystalline principle, the anhydrid of cantharidic acid, was isolated by Robiquet in 1812 from the Spanish fly, *Lytta vesicatoria* (Linn.). Cantharidin penetrates the epidermis quite readily and produces, even in very small quantity ($\frac{1}{10}$ mg) violent superficial irritation, resulting in vesication (blistering) in a few hours. Even when applied to the skin, cantharidin irritates the kidneys so that "fly blisters" are contraindicated in nephritis. It was formerly used as an aphordisiac, but its effects may be dangerous to life; hence its use for this purpose has been largely discontinued.

The collection and preparation of the beetles for medicinal purposes provides an occupation for many persons for a brief period. Collecting and preparing the insects requires special precautions owing to their vesicating properties. The best quality of cantharidin produced from the pulverized beetles is the result of special care in the drying, which must be gradual.

Other blister beetles, causing severe vesicular dermatitis in Africa, are *Mylabris nubica* de Marseul, *Epicauta tomentosa* Maeklin, and *Epicauta sapphirina* Maeklin, according to Chalmers and King.[16] Also *Paederus crebripunctatus* Epp. is reported to be a severe vesicating beetle of East Africa[17] affecting Europeans and Africans similarly, although less severely on habitually exposed parts of natives. The term "Nairobi eye" applies to the conjunctivitis caused when the juices of crushed beetles are rubbed into the eye. The active principle is cantharidin. Roberts and Tonking[17] (*loc. cit.*) recommend a cold compress of saturated solution of magnesium sulfate.

At least two species, *Sessinia collaris* (Sharp) and *Sessinia decolor* Fairm., belonging to the family **Oedomeridae,** cause severe blistering on some of the mid-Pacific Islands where they are called coconut beetles.[18] These beetles fairly swarm about the newly opened male flowers of the coconut where they feed on pollen. They are readily attracted by light. Coming in contact with one of these beetles causes a sharp momentary pain, like a burn from hot oil, but the large blister which forms in a few hours causes little pain.

SPIDERS

Class Arachnida—Order Araneida (Araneae)

General characteristics. Spiders are arachnids in which the prosoma is uniform, bearing not more than eight eyes, and joined to the opisthosoma by a pedicle. The opisthosoma is usually unsegmented and bears not more than four, usually three, pairs of spinnerets. There is no telson. The chelicerae are two-segmented, moderately large and unchelate, and contain a poison gland. The pedipalps are six-segmented, leg-like and tactile in function. The legs consist of seven segments; the tarsi have two or three claws. Respiration is by book lungs or trachea or, normally, both. The pedipalps of the male are modified as intromittent organs (Savory, *loc. cit.* p. 59).

Though spiders are universally feared, no doubt because of the knowledge that they are able to kill insects and other small animals by introducing a venom with the bite, it is nevertheless true that out of the more than two thousand genera in more than thirty families only a very few species are actually dangerous to man.

Tarantulas. The term *tarantula* was first applied to a European species, *Lycosa tarantula* (Linn.), a member of the family **Lycosidae** (wolf spiders). In Italy in the vicinity of Taranto there occurred a spider scare during the seventeenth century which gave rise to a condition known as "tarantism" resulting from the bite of this species of spider. To rid the body of the venom those bitten engaged in a frenzied dance known as the "tarantella."[19]

The following account of *tarantism* is from the *Cambridge Natural History*, Vol. 4, p. 361:

> The bite of the spider was supposed to induce a species of madness which found its expression—and its cure—in frantic and extravagant contortions of the body. If the dance was not sufficiently frenzied, death ensued. In the case of survivors, the symptoms were said to recur on the anniversary of the bite. Particular descriptions of music were supposed to incite the patient to the excessive exertion necessary for his relief; hence the name "Tarantella."
>
> In the middle ages epidemics of "tarantism" were of frequent occurrence and spread with alarming rapidity. They were seizures of an hysterical character, analogous to the ancient Bacchic dances, and quite unconnected with the venom of the spider from which they took their name. The condition of exaltation and frenzy was contagious, and would run through whole districts, with its subsequent relapse to a state of utter prostration and exhaustion. The evil reputation of the Tarantula appears to have exceedingly little basis in fact.

In the southwestern United States the term "tarantula" is applied to the very large spiders belonging to the family Aviculariidae, also known as "bird spiders." Many of these spiders measure about five inches in spread of legs. Among these is *Eurypelma californica* Ausserer; Baerg[20] reports that this species has been credited with prodigious jumping power (10 to 25 feet) and is greatly feared on account of its alleged poisonous nature.

> Many tests have been made with the poison of this tarantula. On white rats and guinea pigs both the bite and injections have been employed. The injections were made by grinding up both poison glands in distilled water, and also in physiological salt solution. On guinea pigs no serious effects have ever been observed. On white rats, one to two months old, four deaths have been observed. The bite and the injection proved equally fatal. As a rule the bite of this tarantula is not fatal to rats, but nevertheless it produces fairly definite symptoms. At first the rat runs about excitedly, and in a jumping and jerky manner. Then it becomes more quiet and appears to have considerable pain in the wounded leg. For much of the time the eyes are closed. In about four or five hours the rat shows evidence of recovery and in another hour it is normal.
>
> On myself I tried the bite of this tarantula twice, and subsequently I have been bitten by accident. The relatively dull fangs produce a pain that may be compared to that made by a pin prick. It lasts for only 15 to 30 minutes and is not accompanied by any inflammation or swelling.

The author has also been bitten by this species with similar benign effects.

Sericopelma communis Cambr. is a large black species of "tarantula" common in the Panama Canal Zone, where it is generally feared. Baerg (*loc. cit.*) allowed a spider of this species to bite him on the finger. He allowed only one fang to puncture the skin. The finger felt numb in a few minutes, and in 10 minutes the pain was quite severe. There followed considerable swelling of the finger, hand, and wrist. After two hours Baerg put the hand in hot water for 30 minutes, when the pain and swelling subsided. A lame feeling in the small and third fingers remained for several days. Baerg concludes that although decidedly painful, the bite of this tarantula is probably not dangerous.

Atrax formidabilis Cambridge and *A. robustus* Cambridge are cited by Thorp and Woodson[19] (1945) as dangerous spiders in Australia. The bite may cause severe symptoms, and death may result. They are funnel-web spiders of fairly large size, up to 1½ inches in length excluding the legs. The fore part of the body is glossy ebony black, the black abdomen is covered with a velvety pile, while the under surface bears tufts and brushes of red hair.

DANGEROUS *Latrodectus* SPIDERS

Genus Latrodectus. Dangerous spiders belonging to the genus *Latrodectus* are found throughout the world. The genus belongs to the family **Theridiidae**, the comb-footed spiders. They have eight eyes and three tarsal claws; the hind pair of legs is comb-footed. They spin irregular webs in which the female spiders hang belly upward. In the United States at least two species are recognized; namely, *Latrodectus mactans* (Fabr.) and *L. geometricus* C. Koch; in Hawaii, *L. mactans* is common; in Mexico *Latrodectus mactans* is known as *arana capulina*, the "cherry spider," also as *chintatlahue;* in Central America it is known as *pallu* or *cassampulga;* in the West Indies *L. geometricus* and *L. curacaviensis* (Müller) are known as *cul rouge;* in South America *L. mactans* prevails in many parts to Argentina as does *L. geometricus;* in Europe there is *L. guttatus*, the *malmignatte*, also *L. congoblatus* Koch, the *falangio* in Italy, and *L. tredecimguttatus* (Rossi) (with 13 triangular or half-moon–shaped spots on the underside); in Russia, *L. lugubris* Motsch is known as the *karakurt* (the black wolf); in Africa *L. geometricus, L. concinnus* Cambridge, and *L. indistinctus* Cambridge are known as *knoppie spinnekop* (shoe-button spider); in Asia *L. lugubris* is widely distributed and is known as the *karakurt* (black wolf) among the Tartars; in Australia, *L. hasseltii* Thorell is common.

The black widow spider, Lactrodectus mactans (Fabr.), was first described from America by Fabricius in 1775, under the name *Aranea mactans*. Like many other species the specific name of this spider has many synonyms, among them the following: *Lathrodectus malmignathus* var. *tropica* van Haaselt, *Latrodectus perfidus* Walck., *L. insularis* Dahl., *L. datatus* C. Koch, *L. apicalis* Butler. Many common names are also applied to this spider, among them in addition to "black widow" are "hourglass spider," "shoe-button spider." "Pokomoo," a name used by the California Indians, who referred to this species as "a small black spider with a red spot under his belly."

FIG. 183. The black widow spider, *Latrodectus mactans*; (*upper left*) mature female with egg sac, (*upper right*) egg and first instar spiders, (*lower left*) second and third instar spiders, (*lower right*) fourth instar spiders.

The *adult female* is glossy black to sepia and densely clothed with short, almost microscopic hairs which give it a naked appearance. An irregular white stripe, a remnant of nymphal pattern, is sometimes present on the dorsal anterior margin of the abdomen. The characteristic crimson hourglass marking on the underside of the abdomen (Fig. 183), rarely altogether absent, varies among individuals from the distinct hourglass marking to a design comprising two or more distinct triangles or occasionally only an irregular longitudinal area. An occasional specimen has a crimson spot at the posterior end of abdomen on the dorsal side, just above the spinerets. The abdomen is globose and often likened to a shoe button. The average width of the abdomen is 6 millimeters, or ¼ inch; and the length over all (legs extended) is about 40 millimeter;

that is, about 1½ inches. The abdomen of gravid females often measures 9 by 13 millimeters (⅜ by ½ inch).

The color pattern of the adult male (Fig. 184), while exhibiting considerable variation, approaches that of the immature female spider. Occasional mature males are almost black but retain some of the abdominal markings of the immature form. The terminal segment of each palpus is shaped like a knob (black) at the front of the head and contains the ejaculatory sexual apparatus, a portion of which resembles a coiled watch spring. The abdomen measures about 3 millimeters, or ⅛ inch, in diameter, and the length over all is about 30 millimeters (about 1⅛ inches).

Distribution and habitat. This species, like most of the members of the genus, favors warmer climates, although it is abundant in some of the northern states of the United States. It has been reported in nearly every state in the United States. The distribution of this spider is re-

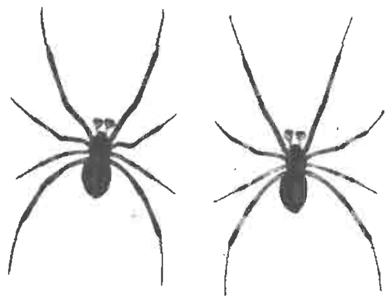


FIG. 184. Male black widow spider, *Latrodectus mactans*. Dorsal view (*left*), ventral view (*right*).

ported as ranging from Canada in North America to Tierra del Fuego in South America, including Mexico, Central America, Hawaii, and the West Indies; it has been taken at an altitude of 8,000 feet in Colorado.[21]

In its natural habitat the black widow spider is found with her web and egg sacs in protected darkened locations, such as vacated rodent burrows, under stones, logs, and long grass; in hollow stumps, and in brush piles. Convenient abode is found in darker corners of barns, stables, privies, pump houses, garages, fruit drying sheds, piles of boxes and crates, wood piles, stone piles, etc.

As a rule the females are not aggressive unless agitated or hungry. When guarding the egg sac the female is particularly prone to bite. Mature female spiders are present throughout the year but are more abundant in the late summer and early fall. Many females have reached maturity by that time, while a few are carried over from the brood of the previous year. The mature males live but a few weeks. The observations coincide with the incidence of poisonous bites by months, i.e., the majority of the spider-bite cases are recorded during July to October, inclusive.

Once a web is established in a suitable location, the female spends the rest of her life feeding on the prey ensnared in this crude but effective web and guarding such egg sacs as she may deposit.

Feeding habits. Whether the prey be a nocturnal moth, cricket, or domestic fly, the technique of capturing, killing, and finally sucking the fluids from the victim is very consistent. The spider depends largely upon vibrations of the web as an indication of a trespasser, or prospective meal. The coarse, permanent web is not particularly viscid in nature, but inadvertent insect visitors become temporarily entangled and in struggling to free themselves inform the owner of the presence. The spider always approaches the victim backwards, extending a freshly spun strand of viscid silk with either one or both hind legs, and attempts to tie down the thrashing appendages. If the captured prey appears particularly obstreperous, the spider ejects from the spinnerets large viscous droplets which dry quickly after the manner of rubber cement, and if the victim becomes entangled by these jets, escape is impossible. At about this point a lethal bite is usually administered. After being bitten the victim struggles violently, and in the course of a few minutes of progressively weaker tremors, dies. The body fluids are sucked from the trussed up victim at the leisure of the captor. After the meal is finished, all points of attachment between the remains of the prey and the web are cut loose, allowing it to drop from the web.

The diet of the black widow consists largely of insects of the locality, small spiders, and even centipedes and sow bugs. It is surprising how many insects an individual spider consumes during its lifetime. Accurate records kept of the food of isolated specimens have shown a total, in the life of an individual spider, as high as 250 houseflies, 33 vinegar flies (*Drosophila*), two crickets, and one small specimen of *Latrodectus mactans* (Fabr.). In considering the economic status of this spider its large diet of prevailing pests is a matter which should not be put aside lightly. It is interesting to note in connection with the diet that one individual (a male) was reared on a diet of its own species exclusively.

Mating habits. After molting the last time the male leaves its web and seeks a mate. In this active, wandering state the male makes no attempt to capture prey but will occasionally suck up a small amount of water or liquid food if the opportunity is offered. If fortunate in finding a likely mate, the male vibrates his abdomen rapidly, causing the entire web to vibrate; the female may produce reciprocating movements. Cautiously the male approaches and strokes the female with his forelegs. It is a dangerous game the suitor plays, for if the female is not ready for his advances, death may result. On the other hand, if the female accepts his advances, the wooing begins. If agreeable, the female remains quiet and allows herself to be spun up in a delicate web. Once the web is successfully spun the male effects coitus by applying the spring-like apparatus of either palpus to the female genital opening. Occasionally this is repeated. After coitus the female easily frees herself and in many instances ensnares and feeds upon her mate. The infrequent observance and recognition of the male of *Latrodectus mactans* (Fabr.), together with the mariticidal habit of the female, has given rise to the name of "black widow." However, if other food is immediately available, often the male is not killed and in due course of time dies a natural death. In the laboratory the males will readily mate a second time, but the females do not show such a tendency.

Comstock reports that in some male spiders the seminal fluid is transmitted from the sexual organs (in the abdomen), which lack any ejaculatory apparatus, to the palpi and is there stored for some time previous to mating. A delicate web appears to be spun upon which the fluid is emitted and then collected by the palpi. The process has been observed by the author in *L. mactans* (Fabr.).

Life history. The life history of the black widow spider from egg to maturity requires about four months under laboratory conditions with ample food.[22] The gravid female, when ready to deposit her eggs, forms a loosely woven cup of silk which hangs downward and while clinging inverted to its rim emits the eggs singly with rapid but regular upward flexures of the abdomen. The eggs, which appear to be forced into an expanding, gelatine-like film, gradually fill and adhere to the silken cup. The open end of the cup is then covered with loose strands of silk and the whole enclosed in a tough, watertight covering of silk. The entire process consumes from one to three hours. Shortly after the egg sac is completed, the film surrounding the eggs seems to evaporate, and the eggs are free to roll about within their envelope. Egg-laying usually takes place during the night.

In California these white or buff-colored egg sacs have been found suspended in webs out of doors from March to October, inclusive. Egg-laying may take place in the laboratory where food is plentiful throughout the year. The egg sacs (Fig. 183) measure from 12 to 15 millimeters (½ to ⅝ inch) in diameter, are usually oval in shape, and may contain from 25 to 917 (Lawson,[23] 1933) spherical eggs, each of which is about 1 millimeter (1/32 inch) in diameter. Females have been observed to spin from one to nine egg sacs a season. One spider under the writer's observation spun seven egg sacs; the eggs in the last one did not hatch. We have never observed egg deposition on the part of mature virgin females. Fertile, mature females in isolation have produced egg sacs in the fall, and surviving the winter, have produced additional egg sacs the following spring, both groups of eggs being fertile.

The time between the deposition of successive groups of eggs varies from about one week to about four months. The incubation period depends on the temperature, and at normal summer temperatures requires about 20 days in the interior of California, the observed extreme range being from 14 to 30 days. At a sustained temperature of $27^\circ \pm 1^\circ$ C the incubation period was about 30 days. The majority of the eggs usually hatch but not simultaneously. In the case of several egg sacs, each being formed by one female, the later lots appear to contain many sterile eggs.

The spiderlings after hatching spend some time—varying from four days in the summer to about one month in cooler weather—within the egg sac before emerging from one or more small holes which they make in the tightly woven envelope. The first molt, previous to which the spider cannot feed, occurs from one to two weeks after hatching. Usually the entire first instar (and sometimes the second) is spent within the egg sac, and at emergence the molted skins are left behind together with the egg remnants. There is a tendency on the part of the spiderlings to cluster for a few days after emerging from the egg sac, and cannibalism rules during this time. The spinnerets appear to be capable of functioning at the time of emergence, but the extremely delicate web is capable of holding only the smallest of prey, such as gnats, mosquitoes, and other tiny spiders. The mother if confined with her young will not feed upon them even though extremely hungry.

Shortly after emerging and after a brief period of clustering, the nymphal spiders disperse by means of nearly invisible strands of silk. For several weeks they move about in the vicinity of their birthplace and suffer a high mortality from other predaceous spiders, and as already stated, from their own species. When about one-third grown the female spiders establish themselves in some protected niche, construct small, loosely woven webs of their own, lacking in specific design, or, rarely, take possession of an abandoned funnel, sheet, or irregular web. Once settled they remain in the chosen lair, capturing progressively larger prey and extending the web as they approach maturity.

The number of molts that the black widow has varies, and the length of the intervening periods is even more inconstant, seemingly conditioned by the season and the amount of food assimilated. The average number of skins cast by the male is five. At optimum temperatures and with plenty of food this number is often reduced to three; under less favorable conditions, resulting in slower growth, a series of six skins may be shed. The sexes may be distinguished by the palpi, or feelers, which in the male are swollen or knob-like (Fig. 184), while in the female they are slender. Subsequent to acquiring this secondary sexual character the male molts once (sometimes twice) before attaining maturity, at which time the web is abandoned, and his search for a mate begins.

The female takes longer to mature and has an average of seven molts, with a range from six to eight. When preparing to molt, she eats nothing for several days. The old skin splits around the margin of the carapace, slips off the abdomen, and the spider then gradually pulls its legs free from its old sheaths, leaving the "ghost" of itself on or near the web. The entire process requires about an hour. The newly molted spider is rather delicate and usually remains at rest for a day or so after molting. Individuals occasionally die during the molting process.

The immature stages. First instar. The abdomen of the newly hatched spiderling is opalescent white with no markings. The cephalothorax and appendages are white to pale yellow; short hair covers the body, becoming dark at the tips of the legs. The tarsi, or last segment of the legs, have two notched claws and a supplementary third between. Eight simple eyes are on the anterior margin of the cephalothorax in two rows of four, and the anterior medians are comparatively dark.

Second instar. All eyes become darker and a black band extends down the center and around the margin of the carapace. Also the mouth parts and the appendages become darker as well as the margin of the sternum of the cephalothorax. A double row of black dots extends down the mid region of the dorsum of the abdomen which remains whitish. On the underside of the abdomen the white area takes on a broad hourglass design outlined by a dark brown border.

Third instar. From this stage to maturity a wide variation in color pattern occurs. Distinct lateral stripes begin to appear on the dorsum of the abdomen, in the region of the dots of the second instar. Intervening areas take on a pale greenish-yellow cast, and the legs acquire four black bands, and at each end of the patella, one near the center of the tibia, and one at the junction of the tibia and the metatarsus. The longitudinal white area on the underside of the abdomen becomes tinged with crimson.

Fourth instar. Dark stripes or bands become distinct and faintly bordered with buff. The spinnerets take on a mottled appearance. Black bands at the leg joints become more distinct.

Fifth instar. The central dorsal white stripe on the abdomen tends to be constricted at intervals and acquires a reddish tinge near the tip. All white areas become lightly colored with brown. Males usually mature at this point.

Sixth and seventh instars. Usually only the females go through these two stages. All coloration is much darker, and the more variable remaining white areas become more and more restricted. Often a series of reddish spots are formed along the middorsal region of the abdomen.

Eighth instar. Only the females pass through this stage, which is often difficult to distinguish from the mature form. They are usually all black or sepia with the exception of the characteristic crimson markings and an occasional white band on the anterior margin of abdomen.

Longevity. The length of life of individual spiders, as one might expect depends on such factors as food supply, natural enemies, including man, etc. Under optimum conditions of food, temperature, humidity, protection, etc., the complete life cycle from egg to maturity requires at least four months. Spiderlings emerging from eggs laid in July and hatched in August will, of course, pass the winter in an immature stage which thus materially extends the length of time required to complete the life cycle. Activity on the part of both the spiders and the insect prey is greatly reduced during the winter months and thus largely accounts for the retardation in development. When a brood emerges in late spring or early summer, the females generally reach maturity before cold weather sets in, but egg laying is held over until the following spring, and hence the life cycle is extended over a complete year. Mature males have not been found overwintering.

Under laboratory conditions a few females have lived through the second and third summers, giving a life span of nearly two years.

Arachnidism (spider bite). While spiders in general have been considered poisonous, though largely erroneously, for centuries, the group to which the black widow belongs in particular has been classed as poisonous for little more than a century. Many of the early reports of spider bite traceable to the black widow came from the southern states and from 1889 to 1894 were frequently mentioned in *Insect Life* (Riley and Howard, 1889–94). After the rapid increase in the population of California during the latter part of the nineteenth century, reports of poisonous spider bites began to be received from this state. In 1932 Bogen[24] listed a total of 380 cases from 18 states, of which 250 were from California.

The chain of symptoms resulting from the bite of the black widow spider is so striking that once recognized there is little danger of confusing it with that of other venomous forms or with an acute abdominal condition. Cases of arachnidism, or spider-bite poisoning, suggest numerous acute abdominal conditions such as a ruptured peptic ulcer, acute appendicitis, renal colic, enteritis, food poisoning, etc.

The bite itself (similar to a pinprick) is not always felt, and often there is but little evidence of a lesion. However, a slight local swelling and two tiny red spots may occur, and local redness is usually in evidence at the point of attack.

Pain, usually in the region of the bite, is felt almost immediately and increases in intensity, reaching its maximum in one to three hours and generally continuing for 12 to 48 hours, gradually subsiding. Rigidity and spasm of most of the larger muscle groups of the body (particularly those of the abdomen) are constant symptoms. The abdominal muscles become "board-like," but local tenderness as in appendicitis is almost always absent. There is a slight rise in body temperature, increased blood pressure, a definite leucocytosis, and usually an increase in the pressure of the spinal fluid There is usually profuse perspiration and often a tendency to nausea. The degree with which these symptoms are present varies in individual cases, and other symptoms such as chills, urinary retention, constipation, hyperactive reflexes, priapism, and a burning sensation of the skin are frequently reported.

Baerg,[25] who permitted himself to be bitten (basal joint of the third finger of the left hand) by the black widow spider, when reporting on the effect of the bite, states:

> Referring briefly to some of the general effects of the case, I would say that the sharp pain in the finger, or rather in the left hand, was the most prominent feature. Very nearly as unpleasant was the aching pain which was most violent in the thick muscles of the lower part of the back, and present in almost all the muscles of the shoulders, chest, and legs. There was no marked tendency towards profuse perspiration. I sweated heavily only when I first went to bed, and later after each one of the hot baths. I covered up well after these baths in order to bring about sweating, and I believe that it aided in recovery. There was no evidence of constipation. One dose of magnesium citrate brought fairly prompt results. On the day I left the hospital I took a second dose in order to facilitate recovery as much as possible.

Baerg's physician, Dr. E. F. Ellis, added the following note:

> The subjective symptoms in Mr. Baerg's case have been very graphically described by him. The objective symptoms would indicate, as observed by me, that there is a very marked phagocytosis locally around the area of the spider bite. The toxicity of the bite was such that the phagocytes very shortly offered no resistance to the systemic invasion of the poison. The poison in my opinion was partly transmitted through the blood stream and partly through the nerve trunk which in this case was the median nerve. Strange to say in this particular instance the patient had a marked vasomotor disturbance on the flexor side of the forearm, as was evidenced by a narrow strip something like an inch in width, extending up almost to the elbow in which there was very marked diaphoresis. This was present during the first 24 hours after the bite. The toxicity was also manifested by vasomotor changes in the lumbar muscles and muscles of the extremities, and in all the large joints of the body, as was shown by intermittent pains and symptoms similar to intermittent claudication. There seems also to be a disposition, on his part, to unload very slowly, by elimination, the products of poison. More so than is the case with bites of any of the snakes including the rattler that I have observed."

Clinical case records. The following case records from the Woodland (California) Clinic are typical of arachnidism:

Case No. 1

Age: 41 Sex: Male Nationality: Dutch
Occupation: laborer. .Date admitted: 9-3-31. .Date dismissed: 9-8-31

9-3-31. About 8:30 P.M. on September 3 patient was taken with a sharp pain in the glans penis as he was sitting on the stool of an open privy. In about

15 to 20 minutes the pain spread to the abdomen, hips, and thighs, the pain being steady, deep, and dull, and so severe that the patient doubled up and rolled on the floor. His abdomen got hard with the onset of the pain. In about two hours pain had developed in the back. He was given two hypodermics (morphine) on entry which relieved him some but stated that he slept only one-half hour during night. Heat was applied to legs and back. Around 4 this morning (9-4-31) the pain seemed to shift to legs (below knees) and feet. In addition to type of pain described in the abdomen, he had an intense burning sensation of skin, worse on soles of the feet. Patient has slight pain in chest, no appetite, and feels weak and shaky all over. Patient unable to urinate.

9-4-31. Patient now has pain in back and legs. Sweating profusely. Pulse, 80. Abdomen absolutely rigid—"board-like." Legs flaccid, neck not stiff. Examination of penis reveals old scars, no recent lesion seen. Impression—arachnidism.

9-5-31. Pain practically gone. Voiding and eating well.

9-7-31. Free of pain. Except for weakness, feels well.

9-8-31. Free of pain. Feels well. Dismissed.

Chart shows patient's temperature fluctuated but not abnormally, pulse somewhat above normal, and respiration normal.

CASE NO. 2

Age: 24 Sex: Male Nationality: American

Occupation: laborer. .Date admitted: 6-17-32. .Date dismissed: 6-31-32

6-17-32. About 9 P.M. last evening patient retired and after getting in bed something dropped on his right shoulder. After turning on the light he discovered a large jet-black spider which he killed. About midnight he noted a tightness in his abdomen and a rather severe lumbar backache. Went to a local physician who gave him a hypodermic about 2 A.M. Abdominal tenseness and pain increased and patient began to note leg pain.

6-17-32. Patient admitted at 3:30 A.M. and examined. Eight hundred cubic centimeters of 5 per cent glucose in normal saline given intravenously after which pulse, which was thready, became fuller and stronger. At 4:45 A.M. considerable back pain and given morphine sulphate (¼ grain).

6-18-32. Abdomen softer. Considerable pain in feet. Profuse perspiration—now apparently out of danger.

6-20-32. Abdomen soft, eyelid edema gone. Considerable discomfort in both feet. Still perspiring but reduced.

6-31-32. Patient discharged. Fingernails look blue.

Chart shows patient's temperature to be slightly above normal, pulse fluctuating, and respiration normal.

Of 37 persons with cases of arachnidism treated at the Woodland (California) Clinic during a period of 10 years, about one-half were bitten on the genitalia while using a privy.[26] Four of the patients were females and the remainder males, the majority of whom were laborers and farmers between the ages of twenty and fifty. Five school children were among those treated. The temperature and pulse of the patients were usually slightly above normal, and the respiration normal. Morphine sulphate (¼ grain) was employed in 25 cases and other drugs such as codeine, amidopyrine, atropine, hyoscine, etc., in the remainder. Although spider-bite cases were admitted to this hospital every month in the year except December and January, the greatest number were admitted during June, July, and August. There was no correlation between the time of day and the bites. The spider was seen in 11 cases. None of the patients reported any after effects on the follow-up inquiry.

Only two deaths were reported in these records, one (6/25/24) of a male 78 years of age, with heart and syphilitic complications, and the other (7/13/25) also of a male (an itinerant) aged 44 years who died four days after being bitten. Post-mortem examination of the latter patient showed a ballooned intestine, and he was recorded as dying of a paralytic ileus resulting from a black widow spider bite.

Venom apparatus. Spiders are equipped with a poison apparatus for purposes of killing their prey. The venom apparatus of *Latrodectus*

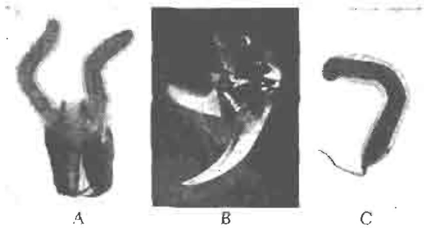


FIG. 185 (*A*) chelicerae and venom glands of female black widow spider, *Latrodectus mactans;* (*B*) separated chelicera; and (*C*) a freed gland. (Photograph by Charles Ladenheim.)

mactans (Fabr.) consists of two poison sacs each with an attached duct leading into the heavily chitinized chelicerae (horny fangs) from which the poison is expelled at the time of biting (Fig. 185). The venom apparatus is present in both the male and the female spider. In the male the venom is primarily of use in the immature stages. The mature male does not attack prey, and the poison apparatus appears to become inactive with maturity and remains small in size. In the case of the female spider the venom apparatus increases in size and strength with maturity. The large quantity of the venom present in the poison glands of the female black widow spider makes her presence perilous. The poison glands of the mature female average about 0.40 mm in diameter and 2.70 mm in length, and in the mature male they average 0.16 mm in diameter and 0.66 mm in length.

The poison glands are located in the cephalothorax extending posteriorly about two-thirds of the way to the junction of the cephalothorax with the abdomen. The long narrow glands curve outwardly around the *fovea media,* that is, the indentation in the center of the cephalothorax for muscular attachment; this is due to the position of the sucking stomach directly below. If the glands were over the sucking stomach they would be squeezed together every time the sucking stomach filled with food and cause a loss of venom. In the female (all stages) the glands are opaque and filled with venom, while in the mature male they are rather translucent but according to experimental evidence contain sufficient venom to produce symptoms of arachnidism in white mice.

Nature of the venom. There is good evidence showing that the so-called poison glands are not glandular in nature, but function as absorptive organs which take up the poisonous constituents from the body fluid of the spider. Sachs (1902) and Kobert in 1901 and 1906, according to Bogen, isolated from the spider body a specific poisonous principle named "arachnolysin" which they claim has a hemolytic effect on the blood of various animals [see also Hall and Vogelsang[27] (1932)]. Experimental evidence indicates that the venom of *L. mactans* acts primarily as a neurotoxin.[28] Spider poison is not limited to the poison glands but is also carried in the body fluids as pointed out by Sachs and by Kobert.

Blyth and Blyth[29] also state that the poison of *Latrodectus lugubris*,

> . . . is not only found in special glands, but is also diffused through the body. . . . it is a generic type of poison of spiders; the active principle is neither a glucoside, acid, nor an alkaloid. It does not dialyse, and drying destroys its activity; it has the characters of a toxalbumin, and has much similarity to the action of ricin and abrin. The "Kara-Kurt" poison dissolves the coloring matter of the red blood corpuscles even with a dilution of 1:127,000; it has a paralyzing effect on the heart, either due to action on the motor ganglia, or possibly a direct action on the muscle itself. The blood pressure sinks, the walls of the smallest arteries and capillaries become so changed as to allow the transudation of the blood and serum, producing punctiform hemorrhages and edema. This is best seen in the lungs. . . . The poison also has a paralyzing action on the central nervous system, but it is not clear whether this action is primary, or whether it depends on the circulation troubles.
>
> The fatal dose of the poison, injected subcutaneously or intravenously, is extremely small. Cats are killed by quantities equal to 0.2 to 0.35 milligram per kilogram body weight. Repeated injections of nonfatal doses confer immunity.

Kellogg[30] (1915) states that high temperatures destroy the action of extracted black widow spider venom. It can withstand a temperature of 56° C (132° F) for 40 minutes, but it is wholly destroyed if heated for 45 minutes at 70° C (158° F). The venom cannot be crystallized, since it is destroyed by desiccation. The best means of preserving the extracted venom is in glycerin, where it can be kept for several months. Eating black widow spiders caused no harm to white mice, guinea pigs, nor a monkey; neither did canned spinach cooked with black widow spider in it.[31]

Treatment. When bitten by the black widow spider, the patient should be treated with local antiseptics, such as tincture of iodine, at the point of injury to prevent secondary infection, and should be kept as quiet as possible; a physician summoned at once. Since, among other properties, the venom appears to be neurotoxic and its effect little short of instantaneous, first-aid measures for snake bite are of little value.

Medical records, according to Bogen (1926, 1932), list more than 75 different remedies and, of all these, three seem to be outstanding as palliatives; namely, spinal puncture, intravenous injections of magnesium sulphate, and intramuscular administration of convalescent serum when given within eight hours.

Gilbert and Stewart[32] (1935) point out that, as "the toxin directly stimulates the myoneural junctions or that it acts on the nerve endings, to find a type of therapy which would have a direct depressant effect upon these structures would be ideal." Because calcium apparently depresses the neuromuscular junctions, Gilbert and Stewart selected calcium salts for the treatment of black widow spider bite. They report on this treatment as follows, "We found that intravenous injections of 10 per cent calcium chloride gave instantaneous and prolonged relief of the pain and at the same time produced immediate relaxation of the muscle spasm so commonly seen in these patients. However, calcium chloride is not given thus without considerable danger. Its necrotic action on tissue outside a vein is only too well known. This danger is greatly magnified when its use is attempted in the treatment of children. Therefore, calcium gluconate (10 cc. of 10 per cent solution, intravenously), which does not have this objectionable feature, was used and found to produce equally as spectacular results as the calcium chloride. The intramuscular route, advisable for children, gave relief within a minute's time. Calcium lactate orally was ineffective as far as determined, probably because of its incomplete and slow absorption." Halter and Kuzell[26] also report similar immediate relief, usually in about one minute, following intravenous treatment of adult males with both calcium chloride and calcium gluconate (10 cc of 10 per cent). In some cases, these authors suggest, a repeat may be necessary at intervals of one to two hours for four or five times.

As Bogen (*loc. cit.*, 1932) states, in part, " . . . despite its severe symptoms arachnidism is in the majority of cases a self-limited condition, and generally clears up spontaneously within a few days."

Control. The black widow spider is frequently found in garages, basements, in living quarters, in old outbuildings (particularly privies, barns, pump houses, stables), and woodpiles. Frequent disturbance of spider webs with a broom or brush is suggested, and the spiders should be killed in any convenient way. Workers in dried fruit industries find numerous black widow spiders under and in the drying trays when handling these. The use of gloves is suggested in this instance. Out-of-doors the spider may be found in vacant lots and open fields and on hill-sides, building their webs in rock piles, heaps of rubbish, under logs, under projecting banks, in deserted squirrel or rabbit holes, under low wooden or concrete bridges, culverts, etc.

Since the egg sacs are conspicuous and are not carried about by the spider, they may be readily collected and destroyed. Great care should be exercised when collecting egg sacs because the female spider guards the sac closely and is particularly vicious at that time. The public should be encouraged to collect and burn or otherwise *destroy* the egg sacs of the black widow spider. Where accessible, the adult spider can be brushed from its web with a broom or stick and stamped upon, or a suitable fly spray may be used to knock the spider to the floor where it should then be crushed. The use of a blow torch when no hazard is involved is effective in the destruction of eggs and spiders as well. Because of the danger from spider bite when using privies in rural sections, it is suggested that the undersides of the seats and corners of the box be painted well with creosote. Privies should be properly constructed to exclude spiders and notably flies on which they feed. Thorough and liberal spraying of all parts particularly under the seats with DDT fly spray, benzene hexachloride, chlordane, or the like is strongly urged.

The adult female black widow spider is extremely difficult to kill with the usual fly sprays which act for the most part as moderate repellents and at best only render the spider temporarily paralyzed. It is of interest to note that a good kill of black widow spiders has been achieved incidental to the use of DDT spray in fly control operations in stables and dairies. Chlordane and benzene hexachloride sprays are reported to be fairly effective. Pyrethrum fly spray fortified with an organic thiocyanate (Lethane 384) is a useful contact spray in the household.[33] Corners in garages, outbuildings, basements, etc., may be sprayed with a penetrating creosote which has some repellent effect against spiders.

Natural enemies. Under natural conditions the black widow spider is held at least moderately in check by its natural enemies. Among the various spider-hunting wasps and mud daubers there appear to be certain species which completely ignore this spider, as for example the yellow-marked mud dauber, *Sceliphron caementarius* Drury, while other species of mud daubers store the black widow spider in their nests; thus Irving and Hinman[34] discovered that the blue mud dauber, *Chalybion cyaneum* (Klug), captures great numbers of this spider. They found 285 black widow spiders had been stored in 15 nests, an average of 19 per nest. The large San Diegan alligator lizard, *Gerrhonotus multicarinatus webii* (Baird), in southern California, has been suggested by Cowles[35] as probably an important factor in cutting down the incidence of this spider on his property. Cowles points out that the elimination of cats is essential because they will eat this lizard. Egg predators, such as the scelionid wasp, *Baeus latrodecti* Dozier, and the chloropid fly, *Pseudogaurax signata* (Loew) (*Gaurax araneae* Coq.), play an important role.

The chloropid fly, *Pseudogaurax signata* (Loew), can be reared successfully in captivity as proved by George Elwood Jenks, who has indicated his success to the author by correspondence and has beautifully illustrated the work of this interesting fly in the August, 1936, number of *Popular Science Monthly*. The larvae of this fly lie free in the egg sac and completely consume the eggs of the spider. The fly deposits its glistening white eggs on the outside of the spider's egg sac. The larvae hatch in five or six days and gain entrance into the sac by pushing their way through the fibers. The length of the larval stage is eight to nine days, according to Kaston and Jenks,[36] and the pupal stage requires 11 to 12 days.

With protection afforded by man-made structures which exclude the natural enemies, the spider thrives and multiplies rapidly.

Other spiders. Instead of being objects of admiration because of their beauty, many of the beautiful garden spiders, orb weavers, are greatly feared. These spiders are harmless and what is more, they are beneficial because they feed on insects which may be harmful to the garden. Two common garden spiders are *Miranda aurantia* (Lucas), the golden orb weaver, and *Argiope argentata* (Fabr.), the silvered orb weaver; both of these construct beautiful geometrical webs.[37] They belong to the family **Argiopidae.**

The remarkable trap-door spiders represented by the California species, *Bothriocyrtum californicum* Cambridge, and the eastern trap-door spider, *Pacylomerus audouini* Lucas, belong to the family **Aviculariidae** already mentioned as including the tarantulas. The trap-door spiders are perfectly harmless. (Passmore.[38])

The burrowing spider, *Brachythele longitarsus* Simon, commonly causes consternation in the home when winter rains and cold weather drive it undercover. Like the trap-door spider, which it resembles, it is quite harmless.

Steatoda borealis (Hentz), belonging to the family **Theridiidae,** resembles the black widow spider rather closely but is slightly smaller; the color is sepia and it bears no red markings. The egg sac is small and loosely woven. It is a harmless species.

The banana spider, *Heteropoda venatoria* Koch, belongs to the family **Heteropodidae** and is frequently brought into northern fruit markets in bunches of bananas or other produce from the tropics. These rather large (spread of 3½ inches), long-legged, dark-brown spiders are commonly mistaken for tarantulas. The bite of this species is painful and at times may cause severe envenomization.

In the house as well as out of doors one commonly encounters the small jumping spiders, belonging to the family Attidae. Members of the genus *Phidippus* may be seen stalking houseflies on the windows of the dining room or kitchen. These spiders have been known to bite, but so far as the author is aware, the effects are benign.

The "pruning spider," *Gliptocranium gasteracanthoides* Nicolet, is reported by Escomel[39] to be a particularly venomous species in Peru.

SCORPIONS

Class Arachnida, Order Scorpionida

General characteristics. Scorpions are easily recognized by their more or less crab-like appearance but particularly by the long fleshy five-segmented tail-like postabdomen terminating in a bulbous sac and prominent sting (Fig. 186). The pedipalps are greatly enlarged, and the last two segments form strong chelae or pincers. The true jaws, chelicerae, are small and partly concealed from above by the front edge of the carapace. There are four pairs of terminally clawed legs.

The cephalothorax bears a pair of conspicuous eyes near the mid-dorsal line (median eyes), and several smaller ocelli in groups of from two to five, on the lateral margins (lateral eyes). Some species are eyeless. Scorpions breathe by means of lung books. They are ovoviviparous, and when the young are born they are carried attached by their pincers to the body of the mother. Although the sexes are very similar in appearance, the males have a longer cauda and broader chelae. An excellent account of the morphology as well as characters used in classification of scorpions is given by Moreno[40] in "Scorpiologia Cubana."

Scorpions are found most commonly in warmer climates. They are nocturnal, remaining hidden during the day beneath loose stones, loose bark of fallen trees, boards, piles of lumber, floors of outbuildings, and debris; some bury themselves in sand or loose earth. They feed upon large insects and spiders, which they seize with their chelae and sting with their powerful sting, which is thrust forward over the scorpion's back.

Scorpion sting. The "aculeus" or sting of the scorpion is situated terminally on the final bulbous segment. This segment contains a pair of venom glands, which are separated by a muscular septum. From the glands are given off fine efferent ducts opening at the apex of the sting (Pawlowsky[41]). The sting curves downward when the "tail" is extended but upward and forward when the scorpion poises for attack or defense, the entire tail-like postabdomen being curved dorsally and forward. The victim is struck quickly and repeatedly, the thrust being made quite close to the front of the carapace.

The *venom* is a transparent liquid, acid in reaction. The toxic principle is said to be a neurotoxin. "It also has a lecithide which haemolyzes

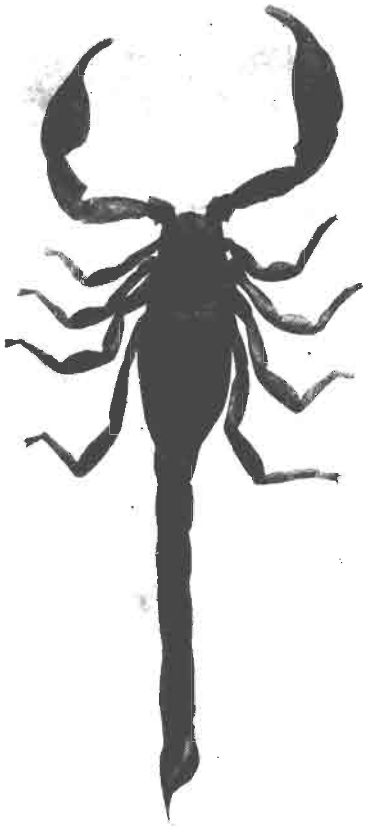


FIG. 186. A scorpion, *Hadrurus hirsutus.* ×.6.

nucleated as well as nonnucleated blood corpuscles. . . . Although the sting of a scorpion is very painful, the poison as a rule does not produce general symptoms in adults, but in children under five years of age the sting frequently causes death," according to Waterman,[42] whose notes on the subject of scorpion poisoning should be consulted by physicians concerned with this matter. Waterman states that the "diagnosis is generally easy if a history of the sting is obtained, a slow full pulse easily compressible, with rapid respirations, a pulse respiration ratio of 3:1, 2:1, or 1:1; salivation, vomiting, glycosuria and epigastric pain and tenderness—a characteristic picture of scorpion sting." The puncture made by the aculeus may be visible.

The symptoms caused by the sting of the Durango scorpion, *Centruroides suffusus* Pocock [*Centrurus gracilis* (Latreille)], are described by Baerg (*loc. cit.*, 1939) as follows:

Immediately following the sharp pain produced by the sting is a feeling of numbness or drowsiness, then there is an itching sensation in the nose, mouth and throat that makes the victim distort the face, rub nose and mouth, and sneeze. There is at first an excessive production of saliva; this and a curious feeling that is described as the sensation of a ball of hair in the throat, induce the victim to swallow as rapidly as possible. The tongue is sluggish, so that communication is often by signs. The muscles of the lower jaw are contracted so that it is difficult, or impossible, to give medicine through the mouth. There is a disorder of movements in arms and legs. The temperature rises rapidly to 104° or 104.8° F., the salivary secretion now diminishes and there is a scarcity of urine. The senses of touch and sight are affected, objects appear large on touching them, hair feels rigid, face feels bulky, a veil seems to be interposed between the eyes and various objects, strong light is unpleasant to patients. Luminous objects, such as a candle, are surrounded by a red circle. Frequently there is a pronounced strabismus. There may be a hemorrhage of the stomach, intestine and lungs. The convulsions come in waves and increase in severity for an hour and a half to two hours, or in severe cases until a fatal result. When the case ends in death, respiration stops a full minute before the pulse ceases to beat. When the patient survives for three hours he is usually considered out of danger; yet death may occur six to eight hours after the patient was stung. It is then probably due to nervous exhaustion following the long periods of convulsions.

Even though stung by a presumably innocuous scorpion the person bitten should be taken to a physician if the bite has been inflicted on the face, the back of the neck or the genitalia. Ordinary scorpion stings may be given first-aid treatment according to Stahnke,[43] as follows, "A piece of ice should be applied as soon as possible against the site of the sting; then more ice should be finely crushed and placed in water, using more ice than water. With a piece of ice against the site of the sting place the hand in the ice mixture. . . . Where a large dose of venom has been received the hand should be kept in this mixture for about two hours . . . a large ice pack of finely crushed ice in a thin cloth may be placed over the site of the sting." The function of the ice is to decrease absorption or to localize the venom; it has no effect on the venom itself.

The order Scorpionida is divided into six or seven families, depending upon the author. These are **Scorpionidae, Buthidae, Vaejovidae, Chac-**

tidae, Ischnuridae, Bothriuridae, and **Chaerilidae.** According to Ewing,[44] four of these families occur in the United States and northern Mexico, and are separated as follows:

A.[1] Sternum subpentagonal, with sides almost parallel.
- B.[1] Membrane at base of last tarsal segment of most of the legs with a single spur; postabdomen frequently reduced................ **Scorpionidae**
- B.[2] Membrane at base of last tarsal segment of most of the legs with two spurs.
  - C.[1] Only two ocelli on each lateral margin of carapace........ **Chactidae**
  - C.[2] With three to five ocelli on each lateral margin of carapace **Vaejovidae**

A.[2] Sternum triangular, the sides being strongly convergent anteriorly; membrane at the base of last tarsal segment of most of the legs with two unbranched spurs; fixed arm of chelicerae without ventral tooth **Buthidae**

Over 300 species of scorpions are known. Of the 40 species found in the United States, according to Stahnke (*loc. cit.*) only two are dangerously virulent to man (see below).

Family Buthidae. This is the most widely distributed of the families of scorpions and is distinguished from the other American families by the shape of the sternum which is triangular, the sides being strongly convergent anteriorly (Ewing). To this family belong *Centruroides suffusus* Pocock, the so-called Durango scorpion, the common scorpion of the State of Durango, as well as of other neighboring states in Mexico and portions of Arizona. During the period from 1890 to 1926, according to Baerg (1929, *loc. cit.*), " . . . a period of 36 years, there have been 1,608 deaths" from scorpion stings in Durango. The majority of victims are children from one to seven years. The symptoms caused by the sting of this scorpion are described above.

Centruroides sculpturatus Ewing, said by Stahnke (*loc. cit.*) to be confined at present to Arizona, is a small species, about 2½ inches in length; it is generally of a solid yellow-straw color. It is a dangerously virulent species and is reported to be abundant.

Centruroides gertschi Stahnke, so far as is known, according to Stahnke (*loc. cit.*), is also confined to Arizona but is much less abundant than *C. sculpturatus*. It is about the same size as the latter but has two irregular black stripes down its entire back; its basic color is yellow. This is also a dangerous species.

Centruroides vittatus (Say) [*C. carolinianus* (Wood)] is the common striped scorpion of the United States. It is widely distributed, having been reported from Georgia, Florida, Kansas, Texas, Arkansas, Louisiana, New Mexico, and South Carolina. Concerning the sting of this species, Ewing (1928, *loc. cit.*) writes, "The writer has induced this species to sting him and has observed the effects of its sting on others. At the time of the stinging there is a sharp pain, but this soon subsides. A small swollen area, or wheal, usually develops about the puncture point. This soon disappears. There are no permanent effects of the sting reported for the species as far as known to the writer." Stahnke (*loc. cit.*) also regards this species as relatively harmless.

Centruroides nigrescens (Pocock) is known as the black scorpion. It is reported from Texas and Mexico. It measures 10 cm in length and is a dark chestnut-brown to jet-black color.

Centruroides californicus (Girard) is the widely distributed striped scorpion of California. It resembles *C. vittatus* (Say) very closely.

Buthus quinquestriatus Hemprich and Ehrenberg is a common Egyptian and North African species, more especially in upper Egypt, according to Wilson,[45] who states that it is of a sandy-yellow color tending to brown and measures about 10 cm in length. He also states that it is undoubtedly the commonest species in that region and is generally believed to be the most dangerous; it is frequently found in houses and is the species in all probability giving rise to the numerous cases of scorpion sting said to be most commonly fatal in upper Egypt. Shulov[46] reports having traced four fatal cases in children between the ages of 6 and 13 in Palestine.

Family Scorpionidae. According to Ewing this family is well represented in Central America and the tropical regions of the Old World, but is poorly represented in the United States.

Diplocentrus whitei (Gerv.) is a very dark reddish-brown scorpion from 5 to 7 cm in length. This species has been taken on the Mojave Desert, California. Ewing writes that he has been inclined to associate serious scorpion stings with this species, because of the descriptions given by persons living near the Mexican boundary who have had experience with cases of severe scorpion stings. On the other hand, Baerg (*loc. cit.*) states that several punctures from two scorpions failed to produce any appreciable effect on him except the slight pain of the puncture.

Family Chactidae. This family differs from all other North American scorpion families in having only two occeli on each side of the carapace.

Broteas alleni (Wood) is a small dark-brown species measuring about 3 cm in length. It has been taken in lower California and at Fort Tejon, California.

Family Vaejovidae. This is the best represented family in North and Central America. It contains a number of very large species.

Hadrurus hirsutus (Wood) (Fig. 186) is the giant hairy scorpion, our largest species, measuring 11 to 12 cm in length. The body is dark yellowish and hairy. It is found in the southwestern United States and northern Mexico. The writer has taken this species in the Imperial Valley, California.

Hadrurus aztecus Pocock is the Mexican hairy scorpion and closely resembles *H. hirsutus* (Wood).

Vaejovis spinigerus Wood is the stripe-tailed scorpion. It has four longitudinal dark stripes on the underside of the "tail." It measures from 5 to 8 cm in length. It is a typical desert species, occurring in rocky waste places of Texas, New Mexico, and California, where it is common. Baerg (*loc. cit.*) reports that its sting caused only a slight pain which disappeared in less than half an hour; its poison had no appreciable effect on white rats.

Vaejovis boreus (Girard) is the northern scorpion occurring in North and South Dakota, Idaho, Wyoming, Nebraska, and Montana. It is a dark, yellowish-brown, unmarked species measuring from 3.5 to 5 cm in length. Its sting, though painful, is benign in effect.

Uroctonus mordax Thorell is the mordant scorpion, a dark-brown medium-sized Pacific coast species. It is the commonest species in the San Francisco Bay region. Its sting is about as painful as that of a yellow jacket, but causes as a rule less swelling; the effects soon disappear. It occurs under loose rocks, beneath bark of fallen trees, under rubbish, tent floors, and the like.

Scorpion control. The scorpion hazard on premises may be largely reduced by the elimination of favorable hiding places, such as boards, loose rocks, rubbish, platforms, and the like. Creosote sprays have a substantial repellent effect, but unless a clear solution can be obtained, may not be desirable about the yard. In some localities where scorpions are abundant and dangerous a bounty has been paid. Thus Baerg (*loc. cit.*) reports that in 1928 (May 1 to July 31) 12,941 scorpions were collected in and near the city of Durango by scorpion collectors (alacraneros), and a bounty of two and one-half cents for the females and two cents for the males was paid. Baerg also states that Dr. Brachetti advises that the "powder of chrysanthemum will drive scorpions away, or even kill them, and so does creoline—a dilute solution of creoline to be sprinkled on the floors and on the flower beds about the house."

WHIP SCORPIONS

Class Arachnida, Order Pedipalpida

Characteristics of Pedipalpida. The Pedipalpida are tropical and subtropical arachnids although very unevenly distributed. They are said to be absent from Europe and North Africa (Savory). The term "whip scorpion" is applied to the Family **Thelyphonidae** because the terminal end of the abdomen is provided with a long, slender, many-segmented appendage (Fig. 187).

The giant whip scorpion, *Mastigoproctus giganteus* (Lucas), occurs in Florida where, according to Ewing, (1928 *loc. cit.,*) it is found on the ground under various litter and under logs, boards, and lumber lying on moist ground. It feeds on almost all kinds of larger insects and other

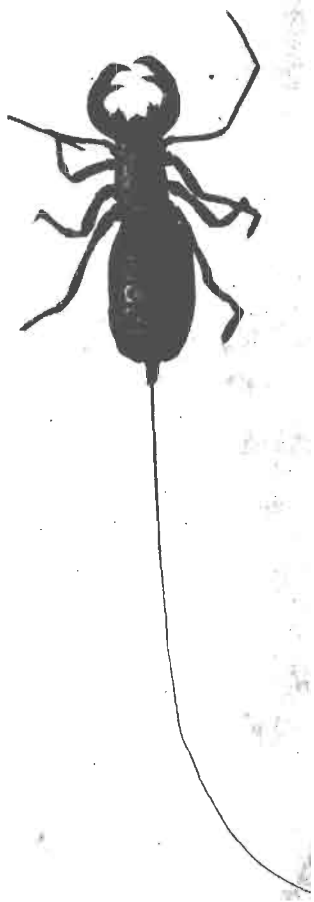


FIG. 187. Whip scorpion (Pedipalpida) *Mastigoproctus giganteus*. ×.8.

arthropods if not too hard or too active. A closely related species, if not identical, occurs in southern California, mainly in sandy desert places where it burrows in sand under debris. It is commonly regarded as poisonous, although it cannot sting but may bite. The writer has found that many persons living in the arid parts of California fear this creature very much, but knows of no evidence to justify this fear. Ewing states that on no occasion was there more than a trivial mechanical effect from the bite, similar to that of a slight pinprick. He states that when handled it gives off a repellent fluid which has the odor of vinegar. This fluid may possibly produce some irritation to persons with a tender skin.

SOLPUGIDS

Class Arachnida, Order Solpugida

Characteristics of the Solpugida. The solpugids (Fig. 188), commonly known as "sun spiders" and "wind scorpions," are in general appearance spider-like, although there is no pedicle; they are very hairy, largely nocturnal, occurring mainly in desert, tropical, and subtropical regions.

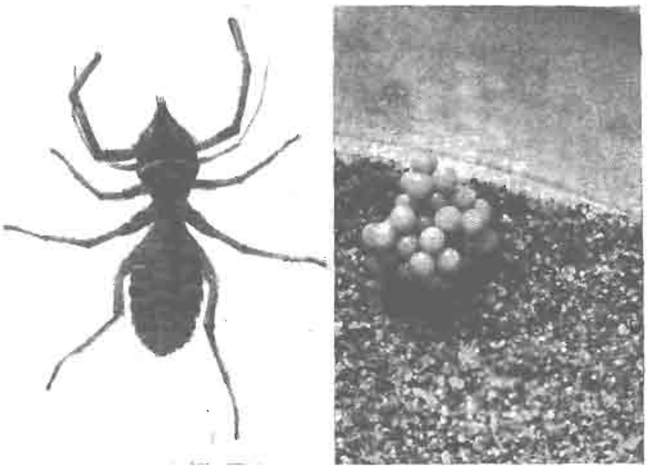


FIG. 188. (*Left*) a solpugid, commonly called "sun spider"; (*right*) eggs.

They are common in many parts of California and have been reported as far north as Nebraska. The chelicerae are large and powerful and are two-segmented. The second segment is movable and articulates in such fashion as to work in a more or less vertical plane. Food is crushed to a pulp, the fluid is swallowed, and the hard parts are ejected. The first pair of legs are used as tactile organs. Respiration is tracheate. They are commonly but erroneously regarded as exceedingly venomous. The writer has been told that the presence of one of these animals in a watering trough would result in the death of any animal drinking from the same. There is evidently not the slightest foundation for this belief. Although these animals are able to inflict a painful bite by means of their powerful jaws, the effect is fleeting. Poison glands are absent.

There are said to be only twelve species in the United States, all but one belonging to the two genera, *Eremobates*, e.g., *Eremobates formi-*

carius (Koch) and *E. formidabilis* (Simon); and *Ammotrecha*, e.g., *Ammotrecha limbata* (Lucas).

VENOMOUS TICKS

Class Arachnida, Order Acarina

Ticks belonging to both familes, **Ixodidae** and **Argasidae**, may cause local as well as systemic disturbances by their bite (see Chapter XX).

Ordinarily little or no injury results from the mere bite of an ixodine tick; the writer has known of *Dermacentor occidentalis* Neumann and *Dermacentor variabilis* (Say) to remain attached to a person for days without causing great inconvenience and occasionally quite unobserved by the host. However, Nuttall (1911, *loc. cit.*) records a number of cases cited by other authors in which the bite of *Ixodes ricinus* (Linn.) has caused serious consequences, notably a case described by Johannessen of a "boy where the tick's body was removed but the capitulum remained embedded in the skin at the back of the head. Swelling followed at the point of injury, accompanied by headaches, stiffening and cramps in the muscles of one side, partial loss of memory and polyuria; the pupils became dilated, etc. The boy made a slow recovery." The bite of *Ixodes pacificus* commonly results in more or less marked systemic disturbances as observed by the author in California.

Quite a number of species belonging to the family **Argasidae** are known to cause more or less serious consequences by their bites alone, notably *Ornithodoros moubata* (Murray), *O. coriaceus* Koch, *O. talaje* (G.-M.), and *O. turicata* (Dugès).

Ornithodoros moubata (Murray) has been reported repeatedly as causing marked disturbances by its bite. Wellman, as quoted by Nuttall and Warburton (1908, *loc. cit.*, p. 98), "states that the bite is very painful, the swelling and irritation (especially in Europeans) not subsiding for days. The wheals are hard, raised and swell most disagreeably if scratched, and this even a week after being bitten. The bite of young ticks (nymphae) is said by the natives to be more severe than that of the adults."

Ornithodoros coriaceus Koch. This species (Fig. 189) occurs commonly in the more mountainous coastal counties of California, having been first described from Mexico, where it is known as the "tlalaja." In California it is known as the "pajaroello." The writer has collected it on Mount Hamilton where it flourishes in the deer beds among the low scrub oaks (*Quercus dumosa*). The following description of the species is a translation by Nuttall from the original:

Shaped like the sole of a shoe, thick margined, roughly shagreened, yellowish-earthly color, spotted rusty-red, legs toothed dorsally. Length 9.3 mm. Body about twice as long as wide, width fairly uniform, indented on the sides, pointed above the mouth parts, rounded posteriorly, a thick turned-up border all around; the whole surface above and below thickly granulated like fish skin (shagreen), the granules flat above, consequently, the whole leathery, on the back unequal folds and grooves. Beneath in the front of the body a deep groove

FIG. 189. Showing *Ornithodoros coriaceus* just backing away from her eggs recently deposited in the sand. Note protective coloration of the tick. ×5.

running to the stigmata and on the inner protrusion the rather large round quite clearly marked eyes. The coxae gradually thicken toward the distal extremity and are somewhat bent; the other articles somewhat compressed and clearly notched or round toothed. The whole surface, above and below, dirty yellowish-earthy color, rusty-red spots irregularly distributed throughout. Capitulum and palps light yellow. Legs gray-brown. Female.

The "pajaroello" is more feared than the rattlesnake by persons living where it exists, and many harrowing tales are told regarding the loss of an arm or leg, or even death resulting from its bite. Much of this is, of course, gross exaggeration. However, the following is an account of two bites which a former student suffered some years ago in July. He experienced a sharp pain on the left arm and upon rolling up his sleeve discovered a large tick, partly engorged, attached to the upper arm in front. He dislodged the tick and sucked the lesion. The lesion when first discovered showed a small dark purple ring surrounding a bright red spot, the point of attachment. The discoloration disappeared in a short time, but the arm was "highly irritable for two or three days and at the point of attachment a minute clear scab formed." The tick proved to be a "pajaroello."

The second bite took place two weeks later while he was seated in a thicket of willows (the first bite had occurred while he was riding over

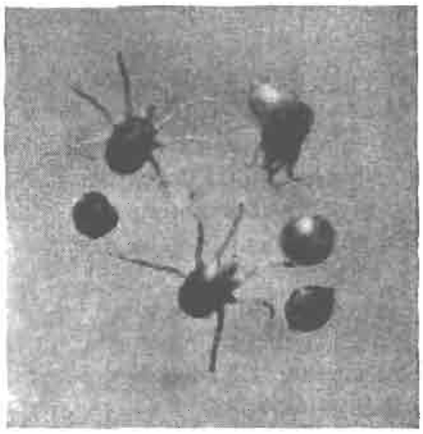


FIG. 190. Egg of *Ornithodoros coriaceus*, larvae in the act of emerging, and two fully emerged individuals. ×14.

a brush-grown hill), and in this case the sharp pain involved the left leg. An almost fully engorged tick (again a pajaroello) measuring about three-quarters of an inch in length and about one-half inch in width was removed from just above the shin. Once more a bright red spot was visible at the point of attachment, surrounded by an irregular purple ring about three-quarters of an inch in diameter. In about an hour the leg began to swell in the vicinity of the lesion, and in about three hours the entire lower leg was tremendously swollen. The coloration about the point of attachment had widened considerably, was puffy, and a clear lymph exuded freely from the lesion. The young man lanced the wound, causing the blood to flow freely, and treated it with crystals of potassium permanganate, binding the leg with cotton and gauze. During the following night he reports experiencing a generally disagreeable feeling, the entire lower leg "irritable and numb." On the following day the bite on the arm became "irritable" again and was treated as had been the leg as he feared bad results. For several weeks both lesions exuded a clear lymph from beneath an "oily-looking, transparent, red mottled scab" which remained in evidence for two or three months.

Life history of Ornithodoros coriaceus Koch. The "pajaroello" deposits large plum-colored spherical eggs (Fig. 190). In the laboratory these are deposited on sand in slight depressions. There are commonly four to seven layings at intervals of from several days to several weeks during the months of May to July, inclusive (as early as February under laboratory conditions), and the female is known to deposit eggs for at least two successive seasons. The greatest number of eggs observed at one laying was 802, with a total of 1,158 for one season. The incubation period at a maintained temperature of from 24° to 26° C was from 19 to 29 days, with an average of about 22 days.

The larvae (Fig. 190) are very active, scattering quickly and attaching readily to a host, particularly rabbits in the laboratory. Experimentally the human may also serve as a larval host. The ear of a rabbit apparently affords a most satisfactory point for attachment. The larva remains attached to the host for a period of about seven days, becoming quite globular and much enlarged. Under favorable conditions the larval stage lasts 10 to 12 days.

Sexually differentiated ticks appeared after the fourth molt. Others did not become sexually differentiated with five molts. There may be six or seven nymphal stages. Ordinarily the tick molts once for each engorgement, but there may be two molts between feedings. Smith[47] reports that reared females began ovipositing four to six months after the first feeding. He records the adult longevity of reared, fed ticks as ranging from about eight months to more than three years and seven months for males and from 13 months to more than five years for a female.

CENTIPEDES AND MILLIPEDES

Class Myriapoda

Myriapoda. The Myriapoda are worm-like animals with separate head, possessing antennae, and many fairly similar segments, each possessing one or two pairs of segmented appendages. Like the insects they are tracheated and for the most part terrestrial.

The class Myriapoda is divided into four or five orders, among which are **Chilopoda**, the centipedes, with only one pair of appendages to each segment; and the **Diplopoda**, the millipedes, with two pairs of appendages to each segment. They inhabit moist situations and feed principally on decaying vegetable matter. An example is *Julus nemorensis* Koch, a so-called "thousand-legged worm" (Fig. 8b).

Centipedes. The **Chilopoda** have only one pair of appendages to each segment which are widely separated at the bases, the antennae are many-jointed, the genital pore is located on the terminal body segment. The larger species, at least, are predaceous, feeding mainly on insects. Notwithstanding the confusing abundance of walking appendages, the centipedes crawl very rapidly.

The centipedes are provided with powerful poison claws located immediately ventral to the mouth and connected by means of a hollow tube with large poison glands. The first pair of legs which terminate in these claws are bent in position to form maxillipeds.

The larger centipedes (Fig. 191) are commonly regarded as venomous and are generally much feared. Large insects are quickly killed when the poison claws close upon them. It is true that the larger species belonging to the genus *Scolopendra* and even the smaller house centipede, *Cermatia* (= *Scutigera*) *forceps* (Raf.), are able to pierce the skin

FIG. 191. A venomous centipede, *Scolopendra heros.* ×.66.

with their poison claws and cause severe pain with same swelling at the site of the bite. Cases are reported showing a double reddish streak on the skin when the centipede had crawled. This condition may be caused by the dragging sharp tips of the terminal pair of legs. Among the several formidable-appearing species of the genus *Scolopendra* are *S. heros* Gir. of the southern United States, *S. polymorpha* Wood of the southwestern states and Mexico, *S. viridis* Say, a Mexican species, and *S. sumichrasti* Sauss. from the Canal Zone. All of these species may reach a length of from five to six inches. Many of the centipedes are markedly phosphorescent, notably *Geophilus electricus* (Linn.) and *G. phosphoreus* Gervais.

Millipedes. The **Diplopoda** are commonly separated into two groups depending upon the presence or absence of repugnatorial glands. In the **Chilognatha** these glands are present and are capable of producing irritating effects (Burt[48]). Certain large tropical species, such as *Rhinocricus* spp., are able to squirt irritating fluids a distance of 18 to 33 inches. Halstad and Ryckman[49] (1949) report a conclusively proven case of vesicular dermatitis in Montemorelos, Nuevo Leon, Mexico, caused by a diplopod of the genus *Orthoporus.*

BIBLIOGRAPHY

1. Cherney, L. S.; Wheeler, C. M.; and Reed, Alfred C.; 1939. "Flea-antigen in prevention of flea bites," *Am. J. Trop. Med.*, 19:327–32.

2. Koszalka, M. F., 1949. "Multiple bee stings with hemoglobinuria and recovery: Report of a case," *Bull. U. S. Army Med. Dept.*, 9:212–17.

3. Martini, E., 1932. *Zoönosen der Haut in wärmeren Ländern,* aus Handbuch der Haut- und Geschlechtskrankheiten (12:575–707). Berlin: Julius Springer. 133 pp.

4. Weber, Neal A., 1937. "The sting of an ant," *Am. J. Trop. Med.*, 17: 765–68.

5. Mallis, Arnold, 1938. "The California fire ant and its control," *Pan-Pacific Entomol.*, 14:87–91.

6. Mickel, C. E., 1928. *Biological and Taxonomic Investigations on the Mutillid Wasp.* Washington, D. C.: Smithsonian Institution, in U. S. Nat. *Museum* Bull., no. 143, 352 pp.

7. von Geldern, Chas. E., 1927. "Systemic effects following the sting of a species of *Epyris*," *Science*, 65:302–03.

8. Essig, E. O., 1932. "A small insect which stings severely," *Science*, 75:242–43.

9. Ewing, H. E., 1928. "Observations on the habits and the injury caused by the bites and stings of some common North American arthropods," *Am. J. Trop. Med.*, 8:39–62.

10. Myers, J. G., 1929. "Facultative bloodsucking in phytophagous Hemiptera," *Parasitiology*, 21:472–80.

11. Bailey, S. F., 1936. "Thrips attacking man," *Canad. Entomologist*, 68:95–98.

12. Caffrey, D. J., 1918. "Notes on the poisonous urticating spines of *Hemileuca oliviae* larvae," *J. Econ. Entomol.*, 11:363–67.

13. Tyzzer, E. E., 1907. *The Pathology of the Brown-tail Moth Dermatitis.* Second Report, Supt. for Suppressing the Gypsy and Brown-tail Moths. pp. 154–68.

14. Bishopp, F. C., 1923. *The Puss Caterpillar and the Effects of its Sting on Man.* Washington, D. C.: Dept. Agric., in Dept. Circ., no. 288. 14 pp.

15. Weidner, Herbert, 1936. "Beiträge zu einer Monographie der Raupen mit Gifthaaren," *Zeits. f. Angewandte Entomol.*, 23:432–84.

16. Chalmers, A. J., and King, H. H., 1917. "Blister beetles as a public nuisance," *New Orleans M. & S. J.*, 70:445–55.

17. Roberts J. I., and Tonking, H. D., 1935. "Notes on an East African vesicant beetle, *Paedurus crebripunctatus* Epp.," *Ann. Trop. Med.*, 29:415–20.

18. Herms, W. B., 1925. "Entomological observations on Fanning and Washington Islands," *Pan-Pacific Entomologist*, 2:49–54.

19. Thorp, Raymond W., and Woodson, Weldon D., 1945. *Black Widow, America's Most Poisonous Spider.* Chapel Hill: Univ. North Carolina Press, xi + 222 pp.

20. Baerg, W. J., 1929. "Some poisonous arthropods of North and Central America," Tr. 4th Internat. Cong. Entomol., Ithaca, 1928, 2:418–38.

21. D'Amour, F. E.; Becker, F. E.; and Van Riper, W., 1936. "The black widow spider," *Quart. Rev. Biol.*, 2:123–60.

22. Herms, W. B.; Bailey, S. F.; and McIvor, Barbara; 1935. *The Black*

Widow Spider. Berkeley: Univ. Calif., in Agric. Exper. Sta. Bull., no. 591. 30 pp.

23. Lawson, P. B., 1933. "Notes on the life history of the hour-glass spider," *Ann. Entomolog. Soc. Amer.*, **26**:568–74.

24. Bogen, E., 1932 . "Poisonous spider bites: newer developments in our knowledge of arachnidism," *Ann. Int. Med.*, **6**:375–88.

25. Baerg, W. J., 1923. "The effects of the bite of *Latrodectus mactans*. Fabr., *J. Parasitol.*, **9**:161–69.

26. Halter, B. L., and Kuzell, W. C., 1943. "Black widow spider bites in the adult male," *Mil. Surgeon*, **92**:427–32.

27. Hall, W. W., and Vogelsang, W. A., 1932. "Spider poisoning: A study of the toxin of the black widow spider *(Latrodectus mactans)*," *U. S. Naval Medical Bull.*, **30**:471–78.

28. Vellard, J., 1936. *Le venin des Araignées*. Monographies Inst. Pasteur. Paris. 311 pp.

29. Blyth, A. W., and Blyth, M. W., 1920. *Poisons: Their effects and detection*. 5th ed., London: Chas. Griffin Co. 745 pp.

30. Kellogg, V. L., 1915. "Spider poison," *J. Parasitol.*, **1**:107–12.

31. Herms, W. B.; McIvor, Barbara C.; and Ladenheim, Charles; 1940. "The effects of ingestion of black widow spiders with canned food." *J. Econ. Entomol.*, **33**:550–54.

32. Gilbert, E. W., and Stewart, C. M., 1935. "Effective treatment of arachnidism by calcium salts, a preliminary report," *Am. J. Med. Sc.*, **189**: 532–36.

33. Herms, William B., 1947. "The black widow spider menace," *Pest Control and Sanitation*, **2**:8–11.

34. Irving, W. G., and Hinman, E. H., 1935. "The blue mud-dauber as a predator of the black widow spider," *Science*, **82**:395–96.

35. Cowles, R. B., 1937. "The San Diegan alligator lizard and the black widow spider," *Science*, **85**:99–100.

36. Kaston, B. J., and Jenks, G. E., 1937. "Dipterous parasites of spider egg sacs," *Bull. Brooklyn Entomol. Soc.*, **32**:160–65.

37. Ewing, H. E., 1933. "Afield with the spiders," *National Geographic Magazine*, **64**:163–94.

38. Passmore, Lee, 1933. "California trap-door spider performs engineering marvels," *National Geographic Magazine*, **64**:195–211.

39. Escomel, E., 1917. "The *Latrodectus mactans* and the *Gliptocranium gasteracanthoides* in the department of Arequipa, Peru," *Tr. Am. Soc. Trop. Med.*, **2**:95–108.

40. Moreno, Abelardo, 1940. "Scorpiologia Cubana," *Rev. Universidad Habana*, nos. 23, 26 and 27. 75 pp.

41. Pawlowsky, E. N., 1924. "Studies on the organization and development of scorpions," *Quart. J. Micr. Sc.*, **68**:615–40 (3 plates).

42. Waterman, J. A., 1938. "Some notes on scorpion poisoning in Trinidad," *Tr. Roy. Soc. Trop. Med. & Hyg.*, **31**:607–24.

43. Stahnke, Herbert L., 1944. "Scorpions of the United States," *Turtox News*, **22**:20–22.

44. Ewing, H. E., 1928. "The scorpions of the western part of the United States, with note on those occurring in northern Mexico," *Proc. U. S.* Nat. *Museum,* 73 (Art. 9): 1–24 (2 plates).

45. Wilson, W. H., 1904. "On the venom of scorpions," *Records Egyptian Govt. School Medicine,* 2:1–44 (3 plates).

46. Shulov, A., 1939. "The venom of the scorpion *Buthus quinquestriatus* and the preparation of an anti-serum," *Tr. Royal Soc. Trop. Med. & Hyg.*, 33:253–56.

47. Smith, Carroll N., 1944. "The life history of the tick *Ornithodoros coriaceus* Koch (Argasidae)," *Ann. Entomolog. Soc. Amer.*, 37:325–35.

48. Burt, E., 1947. "Exudate from millipedes, with particular reference to its injurious effects," *Trop. Dis. Bull.*, 44:7–12.

49. Halstead, B. W., and Ryckman, R. E., 1949. "Injurious effects from contacts with millipedes" (in manuscript).

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