MILITARY PREVENTIVE MEDICINE: MOBILIZATION AND DEPLOYMENT
Volume 1

Section 3: Preparing for Deployment

Before these Marines arrived in Southwest Asia for the Persian Gulf War, an impressive amount of planning and coordination had been done by preventive medicine professionals on their behalf. The health threats facing these Marines had been assessed, plans had been made to counteract those threats, and systems had been put into place to monitor their health status during and after the deployment. These efforts were focused on keeping disease and nonbattle injuries to a minimum—and they worked.

Photograph: Courtesy of the Defense Visual Information Center, March Air Reserve Base, California. Image 47 on the CD-ROM "U.S. Forces in Desert Storm."

Chapter 21

ARTHROPODS OF MILITARY IMPORTANCE

RAJ K. GUPTA, PhD; LEON L. ROBERT JR., PhD; and PHILLIP G. LAWYER, PhD

R. K. Gupta; Colonel, MS, US Army; Research Area Director, Research Plans and Programs, US Army Medical Research and Development Command, Fort Detrick MD 21702-5012; formerly, Department of Entomology, Division of Communicable Diseases and Immunology, Walter Reed Army Institute of Research, Silver Spring, MD 20910-7500

L. L. Robert Jr.; Lieutenant Colonel, MS, US Army; Department of Preventive Medicine and Biometrics, Division of Tropical Public Health, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814

P. G. Lawyer; Colonel, MS, US Army, (Retired); Associate Professor, Department of Preventive Medicine and Biometrics, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814; formerly, Entomology Consultant, Office of US Army Surgeon General

INTRODUCTION

Until the latter half of the 20th century, military casualties due to disease and nonbattle injuries (DNBI) usually outnumbered those directly related to combat. Historically, arthropods have been a leading cause of DNBI. The direct effects of arthropods include tissue damage due to stings, bites, and exposure to vesicating fluids, infestation of tissue by the arthropods themselves, annoyance, and entomophobia (an uncontrolled fear of arthropods). The indirect effects on human health include disease transmission and allergic reactions due to bites and stings and to arthropod skins or emanations. Arthropods also destroy property and materiel used by the military, and there is even some concern that arthropods could potentially be used as biological weapons. The importance of naturally occurring arthropod-borne disease is well documented, as is the direct injury, annoyance and material damage caused by nuisance arthropods. During World War II, it is estimated that more than 24,000,000 man-days were lost due to arthropod associated disease and injury.[1]

Arthropods are of greatest importance to military operations when they act as vectors of disease. A vector is an organism that transmits a pathogen to a susceptible host. Arthropods serve as vectors in a number of different ways, from simple mechanical transmission of pathogenic organisms on the arthropod body, as when house flies carry dysentery bacilli from infected feces to food, to the more complicated process of biological transmission, where the pathogens must spend part of their life cycle in the body of the arthropod before humans can be infected. A fundamental activity of military medical entomologists is to establish the role that certain arthropod species or populations play in the transmission of a particular infectious disease to service members. There are primary and secondary vectors. Primary vectors are those that are mainly responsible for transmitting a pathogen to humans or animals; secondary vectors are those that play a supplementary role in transmission but would be unable to maintain disease transmission in the absence of the primary vector.[2] The criteria that must be satisfied to declare with reasonable certainty that a particular arthropod is a vector of human disease are:

- Demonstration that members of the suspected arthropod population commonly feed upon vertebrate hosts of the pathogen, or otherwise make effective contact with the hosts under natural conditions,
- Demonstration of a convincing biological association in time and space between the suspected vectors and clinical or subclinical infections in vertebrate hosts,
- Repeated demonstration that the suspected vectors, collected under natural conditions, harbor the identifiable, infective stage of the pathogen, and
- Demonstration of efficient transmission of the identifiable pathogen by the suspected vectors under controlled experimental conditions.[3]

To minimize adverse effects caused by arthropods, military entomologists must be able to identify vector and pest species accurately and then prescribe appropriate countermeasures. This begins with a basic knowledge of arthropod morphology and classification. Members of the phylum Arthropoda have segmented bodies with paired, segmented appendages, bilateral symmetry, a dorsal heart, a ventral nerve cord, and an exoskeleton. The arthropods are divided into five classes: Insecta (insects), Arachnida (mites, ticks, spiders and scorpions), Crustacea (crabs, lobsters, shrimps, water fleas), Chilopoda (centipedes), and Diplopoda (millipedes). Two of these classes, the Insecta and the Arachnida, are especially important to war fighters because of the potential effect they can have on military operations (Table 21-1). The other three classes are of minor significance. The identification of the arthropods by class, order, family, genus, and species is achieved by matching their morphological characteristics or features to couplets on a published dichotomous key. The class Insecta is the largest arthropod class and the one that includes the majority of the arthropods of military importance.

CLASS INSECTA

Insects can be differentiated from other arthropods by the presence of three distinct body regions (head, thorax and abdomen), and three pairs of legs (Figure 21-1). Insects are the only arthropods that possess wings and most, but not all, have them. Insects also have only one pair of antennae.

Insects transmit diseases either mechanically or biologically. Mechanical vectors include cockroaches and filth flies. These insects may pick up pathogens on their feet or other parts of their body

TABLE 21-1

SYSTEMATIC LIST OF ARTHROPODS NAMED. ALL FALL WITHIN THE PHYLUM ARTHROPODA.

Scientific name	Common name
Class Arachnida	Arachnids
Order Araneae	Spiders
Family Theridiidae	Comb-footed spiders
Lactrodectus mactans	Black widow spider
Family Loxoscelidae	Recluse spiders
Loxosceles reclusa	Brown recluse spider
Order Acari	Mites and Ticks
Family Trombiculidae	Chigger mites
Leptotrombidium deliense	
Leptotrombidium fletcheri	
Leptotrombidium pallidum	
Trombicula alfreddugesi	
Trombicula autumnalis	Harvest mite
Family Pyroglyphidae	House dust mites
Dermatophagoides farinae	American house dust mite
Dermatophagoides pteronyssinus	European house dust mite
Euroglyophus maynei	
Family Glycyphagidae	
Glycyphagus destructor	
Family Macronyssidae	Fowl mites
Ornithonyssus bacoti	Tropical rat mite
Ornithonyssus bursa	Tropical fowl mite
Ornithonyssus sylviarum	Northern fowl mite
Family Dermanyssidae	
Dermanyssus gallinae	Chicken mite
Liponyssoides sanguineus	House mouse mite
Pyemotes tritici	Straw itch mite
Family Sarcoptidae	Scab or itch mites
Sarcoptes scabiei	Scabies mite
Trixacarus sp.	
Family Demodicidae	Follicle mites
Demodex brevis	Lesser follicle mite
Demodex folliculorum	Follicle mite
Family Argasidae	Soft ticks
Ornithodoros hermsii	
Ornithodoros parkeri	
Ornithodoros turicata	Relapsing fever tick
Family Ixodidae	Hard ticks
Amblyomma americanum	Lone star tick
Amblyomma maculatum	Gulf Coast tick
Dermacentor andersoni	Rocky Mountain wood tick
Dermacentor variabilis	American dog tick
Ixodes dammini	Northeastern deer tick
Ixodes scapularis	Black-legged tick
Ixodes pacificus	Western black-legged tick
Ixodes persulcatus	
Ixodes ricinus	European castor bean tick
Class Insecta	Insects
Order Anoplura	Sucking lice
Family Pediculidae	Head and body lice
Pediulus humanus capitis	Human head louse
Pediculus humanus humanus	Human body louse
Family Pthiridae	Crab lice
Pthirus pubis	Human crab louse
Order Siphonaptera	Fleas
Family Pulicidae	
Xenopsylla cheopis	Oriental rat flea
Order Hemiptera	True bugs
Family Cimicidae	Bed bugs
Cimex hemipterus	Tropical bed bug
Cimex lectularius	Common bed bug
Family Triatomidae	Assassin bugs
Triatoma infestans	
Triatoma dimidiata	
Triatoma barberi	
Triatoma gerstaeckeri	
Triatoma proctata	
Triatoma sanguisuga	Blood sucking conenose
Panstrongylus megitus	
Rhodnius prolixus	
Order Hymenoptera	Bees, wasps, and ants
Family Apidae	Bees
Apis mellifera mellifera	European honey bee
Apis mellifera scutellata	Africanized honey bee
Family Formicidae	Fire ants
Solenopsis invicta	Imported fire ant

TABLE 21-1 *continued*

Scientific name	Common name
Order Diptera	True flies
Family Ceratopogonidae	Biting midges
Culicoides spp.	
Family Simullidae	Black flies
Simulium spp.	
Family Psychodidae	Sand flies
Brumtomyia spp.	
Chinius spp.	
Lutzomyia spp.	
Phlebotomus papatasi	
Sergentomyia spp.	
Werileya spp.	
Family Culicidae	Mosquitoes
Aedes aegypti	Yellow fever mosquito
Aedes albopictus	Asian tiger mosquito
Aedes fulvus	
Aedes leucocelaenus	
Aedes lineatopennis	
Aedes niveus	
Aedes poicilius	
Aedes polynesiensis	
Aedes scutellaris	
Aedes serratus	
Aedes sollicitans	Salt marsh mosquito
Aedes taeniorhynchus	
Aedes thelcter	
Aedes togoi	
Aedes tongae	
Aedes triseriatus	
Aedes vexans	Inland floodwater mosquito
Aedes vigilax	
Anopheles campestris	
Anopheles darlingi	
Anopheles donaldi	
Anopheles funestus	
Anopheles gambiae	
Anopheles minimus	
Anopheles punctulatus group	
Culex gelidus	
Culex neavei	
Culex pipiens quinquefasciatus	Southern house mosquito
Culex pseudovishnui	
Culex tarsalis	
Culex tritaeniorhynchus	
Culex vishnui	
Culiseta melanura	
Deinocerites spp.	
Eremapodites spp.	
Haemagogus spp.	
Mansonia annulata	
Mansonia annulifera	
Mansonia bonneae	
Mansonia dives	
Mansonia uniformis	
Psorophora columbiae	Dark rice field mosquito
Psorophora confinnis	
Psorophora discolor	
Psorophora ferox	
Sabethes chloropterus	
Family Glossinadae	Tsetse flies
Glossina morsitans	
Glossina palpalis	
Glossina swynnertoni	
Glossina tachinoides	
Family Tabanidae	Deer and horse flies
Chrysops dimidiata	
Chrysops distincitipennis	
Chrysops silacea	
Tabanus spp.	
Family Chloropidae	Eye gnats
Hippelates flavipes	
Family Muscidae	Filth flies
Fannia spp.	
Musca domesticus	Common house fly
Family Calliphoridae	Blow flies
Auchmeromyia luteola	Congo floor maggot
Calliphora spp.	also called a filth fly
Cordylobia anthropophaga	Human bot fly
Lucilia spp.	
Chrysomyia bessiana	
Cochliomyia hominivorax	
Family Oestridae	Robust bot flies
Dermatobia hominis	Torsalo
Family Sarcophagidae	Flesh flies
Sarcophaga spp.	

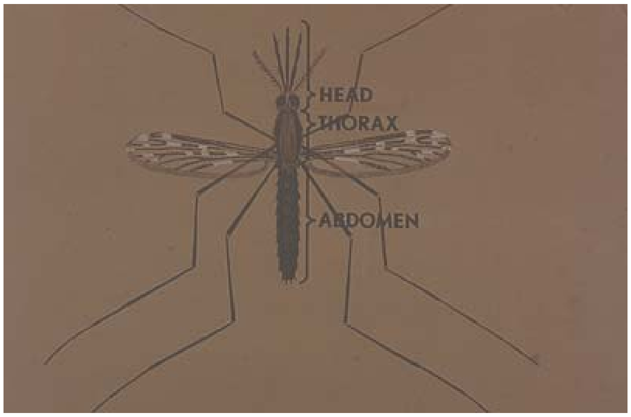

Fig 21-1. The three major divisions of an insect body are shown on this drawing of a mosquito.
Source: Letterman Army Institute of Research, Presidio of San Francisco, Calif.

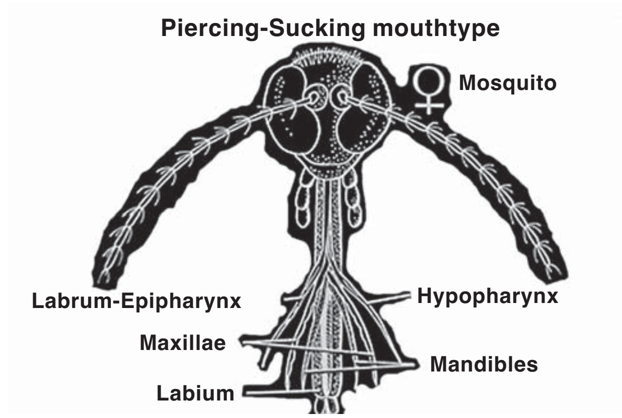

Fig 21-2. The sucking-piercing mouth parts of a female mosquito.
Source: Letterman Army Institute of Research, Presidio of San Francisco, Calif.

while feeding on fecal material or organic waste, or they may ingest the pathogens and later contaminate food consumed by the humans. Some examples of important mechanically transmitted diseases that have plagued military operations are typhoid fever, cholera, and dysentery. However, the insects that are of greatest importance to the military are those that serve as biological vectors of human pathogens. Pathogens transmitted in this manner must pass through part of their life cycle in the vector, where the pathogens multiply, change form, or do both before being passed on to a susceptible host. The host is usually infected via the bite of the vector or by having the excretions or body fluids of the vector rubbed into the skin.

Many of the vector insects discussed in this chapter are members of the order Diptera, the two-winged or true flies. Approximately 80,000 to 100,000 species in about 140 families have been described, and new ones are being added constantly. This order contains more insects involved in transmission of human and animal pathogens than any other. The Diptera are relatively small and soft-bodied insects; all winged members possess only one pair of wings and a pair of short, knob-like structures called halteres. Conspicuous compound eyes are usually present, and most species possess an additional three simple eyes, called ocelli, that are set in a triangle at the top of the head. The Dipteran mouthparts, while subject to great morphological variation, are all adapted for sucking fluids, as opposed to chewing solids, and are either piercing or nonpiercing. They can be divided into three subtypes as follows: (1) mosquito subtype (piercing/sucking), (2) horse fly subtype (piercing/cutting), and (3) house fly subtype (nonpiercing/sponging) (Figure 21-2). All Diptera are holometabolus, meaning they undergo complete metamorphosis, passing through egg, larva, and pupa stages before becoming adults. The larvae of most medically important Diptera species require high humidity and are aquatic, semi-aquatic, endoparasitic, or live in wet or moist terrestrial habitats. The mosquitoes, sand flies, black flies, horse flies, stable flies, midges and filth flies are examples of Diptera that are serious pests of humans and animals.[2]

Mosquitoes

Mosquitoes are the foremost nuisance insects and disease vectors in most regions of the world and the sole vectors of several pathogens that cause diseases of military importance, including malaria, yellow fever and dengue. They also play a significant role in transmitting filariasis and forms of viral encephalitis.[2,3] Mosquitoes belong to one of the more primitive families of Diptera, the Culicidae. The blood-sucking behavior of female mosquitoes and some other Dipterans predisposes them to acquiring pathogens and parasites from an infected host and depositing them in the skin of a susceptible host.

Life Cycle

The immature stages of all mosquitoes develop only in water, but adaptation to and preference for particular types of water and water locations vary

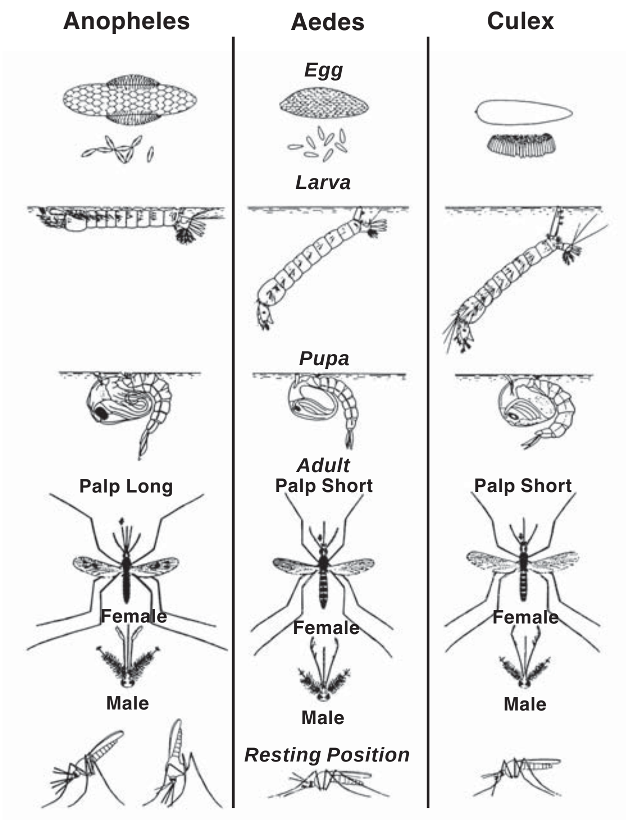

Fig. 21-3. Characteristics of anopheline and culicine mosquitoes.
Source: Centers for Disease Control and Prevention. *Vector-borne Disease Control Self-study Course 3013-G*. Manual G. *Mosquitoes of Public Health Importance and Their Control*. Atlanta: CDC; 1994: 14.

widely among species. The larvae are anatomically different from the adults, live in different habitats, and eat different types of food. Transformation to the adult stage takes place during a nonfeeding pupal stage (Figure 21-3).

Egg. Female mosquitoes may lay several hundred eggs, depositing them on the water's surface or on sites that will be flooded by water later. Each egg is encased in a protective shell. Depending on the species, eggs may be laid singly, in clusters, or in rafts.[2,4]

Larva. Mosquito larvae, also known as "wigglers," can be found in permanent ponds and marshes, temporary flood waters, or in water that has accumulated in tree holes, leaf axils, or other natural and artificial containers. They are very active and feed on minute aquatic animal and vegetable life and on decaying organic matter, coming to the surface to breathe through a respiratory siphon at the posterior end of the body. The larval stage includes four developmental instars. Mosquito larvae are affected by light and water conditions (eg, temperature, water movement), predators, and other living organisms in their habitat. When fully grown, a larva changes to a pupa.[2]

Pupa. The pupa, or "tumbler," is active but does not feed. This is the resting stage from which the adult is formed. The pupa rests on the surface of water unless disturbed, then it quickly moves towards the bottom. It breathes through two respiratory trumpets on the thorax. When the adult is ready to emerge, the pupal skin splits, and the adult pulls itself up and out of the floating skin, on which it rests until it is ready to fly.[2]

Adult. Adult mosquitoes are distinguishable from other Diptera by the long, filamentous antennae comprising 14 to 15 segments, a long proboscis adapted for blood sucking, and scales on the fringes of the wings and wing veins. The rounded head bears large compound eyes that almost touch in the middle. Males can usually be separated from the females by their bushy antennae. Adult mosquitoes display great diversity with regard to resting and oviposition habits, biting preferences, vector competence, dispersion, and flight range. Although breeding sites vary by species, mosquitoes can be divided into three major groups based on where they deposit their eggs: permanent water breeders, floodwater breeders, and artificial container or treehole breeders. Because each of these groups contains important vector species, breeding sites should always be considered when investigating a mosquito-borne disease outbreak or when implementing surveillance and control programs. *Anopheles* (including malaria vectors) and *Culex* mosquitoes usually select permanent bodies of water, such as swamps, lakes, ponds, streams and ditches. On the other hand, floodwater mosquitoes lay their eggs on the ground in depressions that are subject to flooding. During heavy rains or after snow melt, eggs deposited in these sites are inundated with water and hatch within minutes or hours. Included in the floodwater group are *Aedes vexans* (inland floodwater mosquito), *Aedes solicitans* (salt marsh mosquito), and *Psorophora columbiae* (dark rice field mosquitoes). Artificial container or treehole breeders include *Aedes aegypti* (the yellow fever mosquito), *Aedes albopictus* (the Asian tiger mosquito), *Aedes triseriatus*, and others.[2,5]

Both male and female mosquitoes feed on plant sugars, such as floral nectars, to obtain nourishment for basic metabolism and flight. In addition, most females will take a blood meal from birds, reptiles, amphibians, or mammals for egg development (Figure 21-4). Biting behavior may be very important

Fig. 21-4. A blood-engorged *Anopheles stephensi* mosquito, which transmits malaria in Asia.
Photograph: Courtesy of Ed Rowton, Walter Reed Army Institute of Research, Silver Spring, Md.

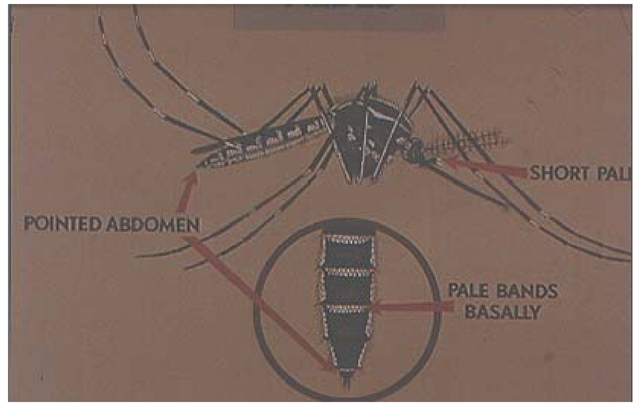

Fig. 21-5. Distinctive characteristics of *Aedes aegypti*
Source: Letterman Army Institute of Research, Presidio of San Francisco, Calif.

in the epidemiology of disease transmission. Many mosquito species bite humans to obtain their bloodmeals, and some feed on humans preferentially. Some will enter houses to bite humans (endophagic), while others bite their human hosts outside (exophagic). Other mosquitoes prefer to feed on non-human hosts, and many species do not bite people at all.[2]

Whereas wind-assisted dispersion records of up to 100 km have been recorded, in control programs and epidemiological studies it is usually safe to say that mosquitoes will not fly more than 2 km from their emergence site.[6] This is an important point to remember when surveying for potential vector breeding sites.

More than 3,000 mosquito species have been described. They are found throughout the world except in places that are permanently frozen. Three quarters of all mosquito species live in the tropics and subtropics, where warm, humid weather favors their rapid development. Mosquitoes may be found at elevations as high as 4,300 m above sea level, such as in Kashmir, India, and 1,160 m below sea level, as in the gold mines of south India.[2] Descriptions of the biology and physiology of mosquitoes can be found in Bates,[7] Christophers,[8] Clements,[9] Gillett,[10,11] and Horsfall.[12]

From a medical perspective, the three most important mosquito genera are *Anopheles*, *Aedes* and *Culex.* These are contained in two subfamilies, the Anophelinae (*Anopheles*) and the Culicinae (*Aedes* and *Culex*). The following characteristics serve to separate *Anopheles* mosquitoes from the other mosquito genera (Figure 21-5). *Anopheles* eggs are laid singly on the surface of the water and are boat-shaped, possessing a pair of lateral floats. *Aedes* lay their eggs singly on damp litter or moist areas near the water's edge. *Culex* deposit their eggs grouped in rafts of 100 or more on the water's surface. *Anopheles* larvae lack a breathing siphon and lie parallel to the surface. Culicines all have a prominent respiratory siphon at the posterior end of the body and are usually found suspended at a 45° angle beneath the water surface. When at rest and when feeding, adult *Anopheles* hold their bodies at a 45° to 75° angle to the surface, with the proboscis and abdomen in a straight line. *Aedes* and *Culex* rest and feed with the body held parallel to the surface. The most reliable way to distinguish between adult Anophelines and Culicinae is by examination of their heads. The *Anopheles* possesses palpi that are about as long as the proboscis, male palpi that are paddle-shaped at the tip, and an evenly rounded scutellum. The palpi of *Aedes* and *Culex* are much shorter than the proboscis.[6]

Parasitic Diseases Transmitted by Mosquitoes

Malaria. Malaria is the most important arthropod-borne disease of humans. Essentially a disease of the tropics and subtropics, it is present in 102 countries and infects up to 500 million people and causes 2.5 million deaths annually, primarily in the tropics.[13,14] In Africa, malaria is one of three infectious diseases that contribute most significantly to the burden of disease as estimated by DALY (disability adjusted life years).[15]

Malaria is caused by four species of protozoan parasites of the genus *Plasmodium: Plasmodium* *falciparum, P vivax, P ovale,* and *P malariae*. Although *P vivax* causes the most malaria infections worldwide, the most serious, and often fatal, form of malaria is caused by *P falciparum*. In many parts of the world, infected people may carry large numbers of parasites without showing signs or symptoms of the disease, thus serving as reservoirs of infection for blood-feeding mosquitoes. Malaria parasites enter the human host through the bite of an infected female *Anopheles* mosquito. The life cycle of the malaria parasite takes place in two separate cycles: one in the human or vertebrate host and the other in the mosquito. (See chapter 36 for details of the malaria parasite cycle.)

Malaria heads the list of arthropod-borne diseases of importance to the military. It has had tremendous impact on military operations in the past and continues to threaten the health and effectiveness of today's war fighters when they are deployed to malaria-endemic areas. In these areas, native human populations often appear healthy, but a large portion of the people may be carriers of or semi-immune to malaria to which they have been exposed repeatedly since birth. The malaria pathogens are maintained at low levels by the hosts' immune systems. These inapparent infections can be passed quickly to nonimmune newcomers by mosquitoes. US forces deployed to malarious areas lack immunity to the pathogen and if they are bitten by infected mosquitoes, they readily contract the disease. The results include loss of manpower, increased burden on the military health support system, and decreased unit morale. All of these factors reduce the commander's ability to execute the directed mission.

The malaria threat to force readiness that has confounded commanders in the past still confronts our forces today. In 1993, a number of Marines (106) and soldiers (70) in certain units participating in Operation Restore Hope in Somalia developed malaria.[16,17] The reasons for this, the largest outbreak of malaria in US military since the Vietnam War, are a complex mixture of incomplete medical intelligence regarding the malaria threat, lack of command emphasis on and compliance with personal protection measures, and the complex life cycle of the malaria parasite. Medical surveillance revealed that one half of all malaria and dengue cases during Operation Restore Hope occurred in a single Marine battalion located in the Baardera area. A subsequent investigation of these outbreaks found that the Marine commander did not enforce recommended countermeasures. Fortunately, the ill Marines recovered and the unit was not involved in any tactical engagements while in this weakened condition. A resurgence of malaria in South Korea in the late 1990s, particularly around the Demilitarized Zone, underscores malaria's importance to US military forces today.[18]

Filariasis. Lymphatic filariasis is another important mosquito-borne parasitic disease distributed throughout tropical and subtropical areas of the world. Currently, an estimated 146 million people are afflicted with this debilitating and disfiguring disease, which is caused by three species of filarial worms: *Wuchereria bancrofti, Brugia malayi*, and *Brugia timori*. *Wuchereria bancrofti* is the most widely distributed species, and its infections are the most prevalent.[19,20] An estimated 115 million people are infected with the parasite. It occurs in large sections of sub-Saharan Africa, parts of South and Central America and the Caribbean region, parts of India, China, Bangladesh, Burma, Thailand, Malaysia, Laos, Vietnam, Indonesia, the Philippines, Papua New Guinea, and island groups in the south Pacific Ocean. *Brugia malayi* has a more limited distribution, occurring in China, India, the Republic of Korea, and Southeast Asia, including Indonesia and the Philippines. *Brugia timori* is confined to certain southern islands of Indonesia in the Savu Sea. The number of people infected with the latter two species has not been estimated, although humans in endemic areas have infection rates as high as 30%.[2]

The worms may infest and block human lymphatic channels and cause enormous and debilitating swelling, resulting in much human suffering. Besides their physical and psychological impact, the filariases exact an enormous toll in terms of decreased economic production in nations where such decreases can be ill afforded. The filariases generally cause morbidity but not mortality in infected humans and so normally do not have an immediate effect on military operations. It can, however, cause a significant loss of personnel and resources in units stationed in endemic areas. In World War II, a survey of two units stationed in the South Pacific showed that they had infection rates of 65% and 55%. Both units were returned to the United States without having entered combat.[21]

The vector mosquito acquires the microfilariae from an infected person while obtaining a blood meal. The filariae shed their sac-like sheath and travel quickly through the midgut of the mosquito to the thorax where they develop in the large indirect flight muscles. After a number of internal changes and two molts, infective third-stage larvae develop. The infective larvae find their way into the hemocoele (body cavity) of the vector and eventually to the proboscis, from which they are transmitted to a new host during blood feeding.[22]

Depending on the species, vectors of filarial pathogens may be active during day or night. *Culex pipiens quinquefasciatus* is the main vector responsible for filarial transmission in urban areas; in rural areas, the vectors may be *Anopheles, Aedes,* or *Mansonia* species that inhabit the forests. The main vectors of *Wuchereria bancrofti* in the tropics are *An gambiae, An funestus, An darlingi, An minimus, An campestris, An punctulatus* group, *Aedes niveus, Ae poicilius, Ae polynesiensis, Ae tongae, Ae vigilax,* and *Culex pipiens quinquifasciatu*s. *Brugia malayi* occurs in rural populations in the Far East between 75° and 140° East longitude in small endemic foci. Specific known vectors of this pathogen are *Mansonia annulifera, M dives, M bonneae, M annulata, M uniformis, An campestris,* and *An donaldi.*[2]

Viral Diseases Transmitted by Mosquitoes

Approximately 100 arthropod-borne viruses (arboviruses) are known to produce disease in humans. The best-known examples of human arboviral pathogens are in the following genera and families: *Flavivirus* (Flaviviridae), *Bunyavirus* and *Phlebovirus* (Bunyaviridae) and *Alphavirus* (Togaviridae). The flaviviruses are either mosquito- or tick-borne, the bunyaviruses and other closely related viruses in the family Bunyaviridae may be transmitted by mosquitoes, ticks, sand flies, or midges; the phleboviruses are transmitted by mosquitoes and sand flies, and all but one of the alphaviruses are mosquito-borne. These pathogens cause infections in humans and livestock resulting in febrile illnesses ranging from mild discomfort to severe influenza-like symptoms and can cause encephalitis, encephalomyelitis, and hemorrhagic fevers. Mortality rates can be relatively high, especially in infections with central nervous system involvement or hemorrhagic symptoms.[2] Arboviral diseases are often characterized by sudden onset and periodic epidemics involving thousands of cases, which could seriously affect combat effectiveness in military operations. The following mosquito-borne viral diseases are discussed in order of relative medical significance.

Yellow Fever. Historically, yellow fever virus, a flavivirus, is probably the most important and most dangerous of the mosquito-borne viruses. It was first recognized in humans in the 17th century, but mosquito transmission of yellow fever virus was not demonstrated until the landmark studies by Major Walter Reed, Dr. Carlos Finlay, and their colleagues in Cuba in 1900.[23] Yellow fever has two transmission cycles, a sylvatic or jungle cycle and an urban cycle. The sylvatic cycle involving forest canopy mosquitoes as the vectors (*Haemagogus* species in Central and South America and *Aedes* species in Africa) and forest primates, mainly monkeys, as the hosts. The urban cycle involves *Ae aegypti* as the vector and humans as the hosts (see Figure 36-10). The urban cycle begins when humans become infected in the sylvatic cycle by entering forest habitats and being bitten by infected sylvatic vectors. People infected in this way return to their villages or cities, thereby initiating urban transmission. Yellow fever originated in Africa and was brought to the New World during the 1500s by slave trade ships. These ships maintained their own colonies of *Ae aegypti* in fresh-water storage containers. The virus and urban vector became established in the Caribbean region, on the east coast of South and Central America, and in the southern United States. During 1741, the British lost 20,000 to 27,000 men to an epidemic of "black vomit" while on an ill-fated expedition to conquer Mexico and Peru. The British and Spanish forces suffered heavy losses on Cuba in the 1760s, and the French lost 29,000 in 1802 while in Haiti and the Mississippi Valley.[3]

In 1900, a US Army Yellow Fever Commission under the direction of Major Walter Reed was established in Cuba, where its studies demonstrated that a filterable agent present in the blood of acute phase patients could be transmitted by *Ae aegypti* mosquitoes (Figure 21-6).[24] Jungle yellow fever in the Western Hemisphere is transmitted chiefly among monkeys, marmosets, and other animals and

Fig. 21-6. *Aedes aegypti*
Photograph: Courtesy of Colonel Raj Gupta, MS, US Army, and the Letterman Army Institute of Research, Presidio of San Francisco, Calif.

causes fatal infections in these animals. The reported vectors of the forest canopy are *Haemagogus* species, *Ae leucocelaenus, Sabethes chloropterus*, and possibly *Ae fulvus*.[25] Many cases of yellow fever have been reported in recent years from South America, the vast majority of these were in males over 15 years of age who were presumably infected while working in the jungle. In the last 30 years, *Ae aegypti* has reinfested Central and South America and brought with it the potential threat to transmit yellow fever in an urban cycle. Because of a very efficacious vaccine, yellow fever is no longer a threat to the US military.

Dengue. Dengue is a viral disease transmitted from person to person by mosquitoes throughout the tropics and subtropics. It is an acute, nonfatal disease of particular importance to military operations because of its rapid spread and its ability to incapacitate large numbers of personnel at critical periods. Dengue fever is caused by any one of four closely related dengue virus serotypes (dengue 1-4) of the Flaviviaridae family.[26] The virus is transmitted primarily by *Aedes* mosquitoes, particularly *Ae aegypti,* an urban species that lives in close association with humans, and is distributed throughout the tropics.[27] Secondary vectors include *Ae albopictus* (the Asian Tiger mosquito, which was introduced into the United States from Asia in the late 1980s), *Ae scutellaris,* and *Ae polynesiensis* (Figure 21-7).[2]

The first epidemics of dengue fever were reported simultaneously in 1779 from Egypt and Indonesia and in 1780 from the United States. The first recognized epidemic of dengue hemorrhagic fever occurred in Manila in 1953 and 1954.[28] In the ensuing 20 years, dengue hemorrhagic fever spread to other parts of southeast Asia, and today it is one of the leading causes of hospitalization and death among children in southeast Asia.[29] Dengue fever was not considered a public health problem in the Americas until the latter part of the 20th century. During the 1970s and 1980s, dengue serotypes 1, 2, and 4 were introduced into the Americas.[30] The increased incidence of dengue and dengue hemorrhagic fever worldwide has resulted in an increased number of cases being imported into the United States. Additional details can be found in chapter 36.

Japanese Encephalitis. Japanese encephalitis is

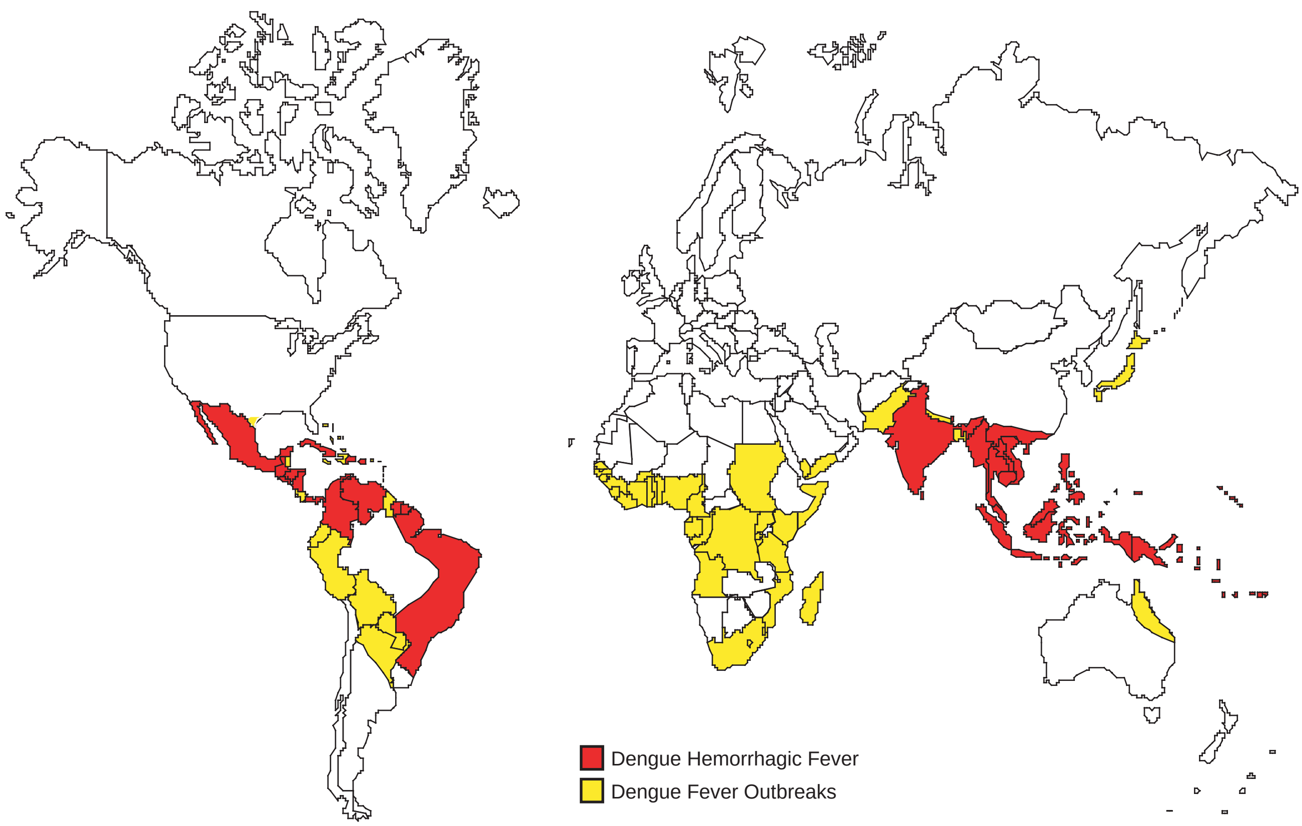

Fig. 21-7. The distribution of dengue fever outbreaks and dengue hemorrhagic fever. Map: Courtesy of Colonel David Vaughn, Walter Reed Army Institute of Research, Silver Spring, Md.

one of the major public health problems in Asia.[31] Outbreaks have been reported from Japan, Korea, Taiwan, Siberia, China, Okinawa, Thailand, Malaysia, and Singapore. They occur in warm weather in temperate regions and throughout the year in the tropics. Japanese encephalitis is principally a disease of rural agricultural areas, where vector mosquitoes breed in close association with pigs, birds, and ducks. Japanese encephalitis virus is a flavivirus and is maintained in mosquitoes and hosts other than humans. The principal vector of Japanese encephalitis is *Cx tritaeniorhynchus,* which feeds primarily on animals and birds. *Cx vishnui, Cx gelidus, Cx pseudovishnui,* and related species are the primary vectors in Thailand and India.[31] These mosquitoes breed in ground pools and especially in rice paddies flooded with water. Studies have shown that the number of Japanese encephalitis cases is directly proportional to the density of the vector when an epizootic in pigs is in progress.[32] Transovarial transmission has been reported in *Ae albopictus* and *Ae togoi* (Figure 21-8). There have been occasional isolations from Anopheles mosquitoes.[2]

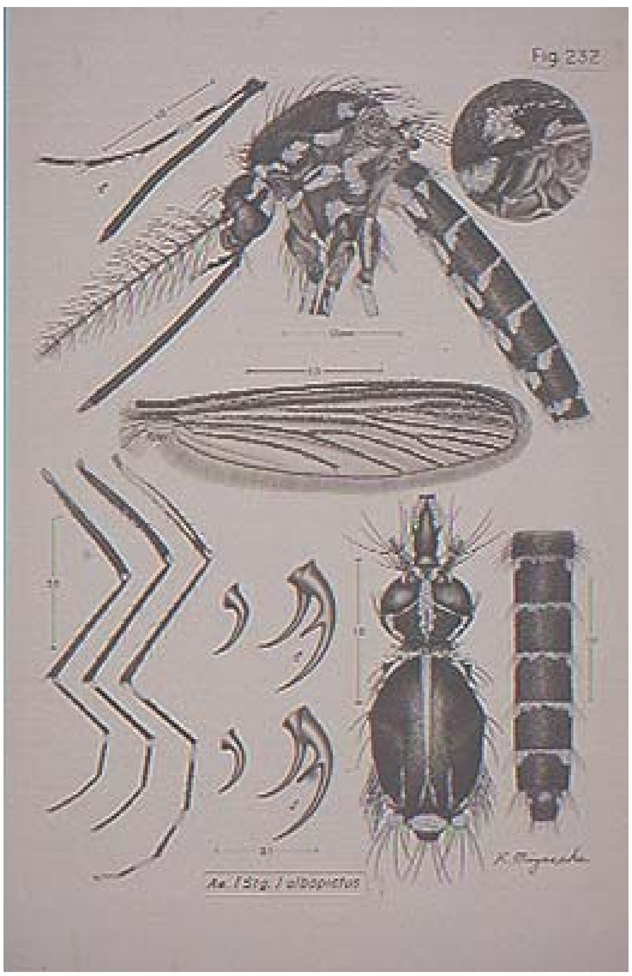

Fig. 21-8. Distinguishing features of *Aedes albopictus* Source: Walter Reed Biosystematics Unit, Department of Entomology, Walter Reed Army Institute of Research, Silver Spring, Md. Art by K. Miyasaka.

A vaccine affording excellent protection (> 90%) against Japanese encephalitis was licensed by the Food and Drug Administration in 1992 and is available for military personnel deploying to endemic areas. It is widely used in parts of Asia, such as South Korea.

West Nile Fever. West Nile fever (WNF) is probably one of the most common arbovirus infections of humans, considering its broad geographical distribution. It is still considered a disease of unknown origin, however, because of its infrequent epidemics. WNF virus, a flavivirus, has been implicated in outbreaks since 1950, but the largest known outbreak occurred in South Africa in 1974. Infection usually occurs in early childhood and produces only a mild febrile illness. In 1998, an outbreak of WNF occurred in New York City with 9 deaths.[33] WNF could have significant impact on military operations. For example, in an earlier outbreak in Israel, 636 cases of clinical disease were reported in a population of approximately 1,000 at a military camp.[34]

WNF virus has been isolated from humans and wild birds, but isolates also have been recovered from animals, including lemurs, chimpanzees, chickens, ducks, geese, horses, mules, donkeys, pigs, and cows. WNF virus has been isolated in 17 countries from three different zoo-geographic regions: the Palearctic, the Ethiopian, and the Oriental. The transmission activity of WNF virus indicates a seasonal pattern limited to summer months. All of the recorded outbreaks in Israel occurred from July through early September; reported outbreaks in South Africa occurred from December through April.[35]

In nature, WNF virus has been isolated from mosquitoes and ticks, but mosquitoes are considered the major vectors. Studies in Egypt, Israel, and South Africa have implicated *Cx univittatus* as the main vector in these countries.[35] *Cx neavei* has been identified in South Africa. *Cx vishnui* complex, containing *Cx tritaeniorhynchus, Cx vishnui,* and *Cx pseudovishnui,* has been implicated in India and Pakistan.

Rift Valley Fever. Rift Valley fever (RVF) is an acute, febrile, arthropod-borne yet contagious zoonotic disease caused by a bunyavirus of the genus *Phlebovirus*[36] (Table 21-2). Most arboviruses are associated with either a single species of mosquito or a closely related group of mosquitoes, except for RVF virus. RVF virus has been associated with numerous flood-water mosquito species, including members of the genera *Aedes*, *Culex*, *Anopheles*, and *Eremapodites*.[37] Other incriminated vectors include a biting midge (*Culicoides* species), a black fly (*Simulium* species), and a tick (*Rhipicephalus* species).[36]

RVF virus has been reported in 24 African countries. In 2000, it appeared for the first time outside of Africa when it caused a major epidemic in Saudi Arabia and Yemen.[38,39] A major epidemic and epizootic in Egypt from 1977 to 1979 caused an estimated 200,000 human infections and 598 reported deaths.[40] In May 1993, RVF virus was responsible for an outbreak in Aswan Governorate, Egypt.[41] RVF epizootics follow periods of excessive seasonal rain, which bring an increase in mosquito population. Outbreaks of RVF have occurred in areas of Africa as diverse as the dry low-rainfall climate of Egypt, the wet forested areas of Uganda and the Central African Republic, and the relatively dry, high veld areas of South Africa where winter temperatures frequently drop below freezing.

RVF virus may be maintained transovarially in flood-water mosquitoes, as demonstrated by the recovery of RVF virus from both male and female

TABLE 21-2

ARTHROPODS FOUND NATURALLY INFECTED WITH RIFT VALLEY FEVER VIRUS

Species	Locality
Aedes	
aedimorphus	
cumminsii	Burkina Faso 1983
dalzieli	Kenya 1982 Senegal 1975, 1983
dentatus	Zimbabwe 1969
tarsalis	Uganda 1955
durbanensis	Kenya 1937
Neomelaniconion	
circumluteolus	Uganda 1955 South Africa
lineatopennis	Zimbabwe 1969 South Africa 1975 Kenya 1982, 1984
palpalis	Central African Republic 1969
Ochlerotatus	
caballus	South Africa 1953
juppi	South Africa 1978
Stegomyia	
africanus	Uganda 1956
dendrophilus	Uganda 1948
furcifer	Burkina Faso 1983
Anopheles	
anopheles	
coustani	Zimbabwe 1969
coustani and *fusicolor*	Madagascar 1979
Cellia	
pauliani and *squamosus*	Madagascar 1979
christyi	Kenya 1982
pharoensis	Kenya 1982
Culex	
antennatus	Kenya 1982 Nigeria 1967, 1970
antennatus, *annulioris* gp, *simpsoni*, and *vansomereni*	Madagascar 1979
antennatus, *simpsoni*, and *vansomereni*	Kenya 1982 Madagascar 1979
theileri	Kenya 1982 South Africa 1953, 1970, 1975 Zimbabwe 1969
zombaensis	Kenya 1982
Eumelanomyia	
rubinotus	Kenya 1982
Eretmapodites	
quinquevittatus spp.	South Africa 1971 Uganda 1948
Coquillettidia	
fuscopennata	Uganda 1960
grandidieri	Madagascar 1979
Mansonia	
uniformis	Madagascar 1979
Mansonioides	
africana	Uganda 1959, 1968 Central African Republic 1969
uniformis	Uganda 1960
Other Diptera	
Culicoides spp.	Nigeria 1967
Simulium	South Africa 1953

Reprinted with permission from: Meegan JM, Bailey CL. Rift Valley fever. In: Monath TP, ed. *The Arboviruses: Epidemiology and Ecology*. Vol 4. Boca Raton, Fla: CRC Press; 1989: 51–76.

Ae lineatopennis collected from larvae in the field and reared to adults in the laboratory.[42]

The Equine Encephalitides. The Togaviridae include two genera, only one of which, *Alphavirus* (or Group A virus) is arthropod-borne. All but one of the 15 *Alphavirus* species are transmitted by mosquitoes.[3] The major Alphaviruses transmitted by mosquitoes are eastern equine encephalitis (EEE) virus, Venezuelan equine encephalitis (VEE) virus, and western equine encephalitis (WEE) virus.

EEE virus has been isolated in equine and human cases from Canada to Argentina along the eastern seaboard of the Americas and in the Caribbean region, southeast Asia, and eastern Europe. The main vectors associated with transmission of EEE are *Culiseta melanura, Ae taeniorhynchus, Ae sollicitans,* and *Ae vexans.* EEE virus generally causes a severe and often fatal encephalitis; its mortality ranges from 30% to 60%. Between 1961 and 1985, however, only 99 human cases were reported.[43]

EEE virus was first isolated in 1933 from the brains of horses and in 1938 was identified as causing an epidemic in Massachusetts. The viruses isolated from outside the New World have not been associated with human or equine cases. *Culex pipiens* is known to circulate this virus in bird–mosquito enzootic cycles in Europe.[44] The ecology is poorly understood, and the mechanism of spread to humans remains speculative. Research investigations suggest that EEE virus is maintained in North America in an enzootic bird–mosquito cycle. The main enzootic vector is a fresh water swamp mosquito, *Cs melanura*, which rarely bites horses or humans. Epidemics in humans and equines occur when the amount of virus in hosts living in swamp environments becomes high and it is carried by mosquitoes to birds and mammals outside of the swamp environment (Figure 21-9).

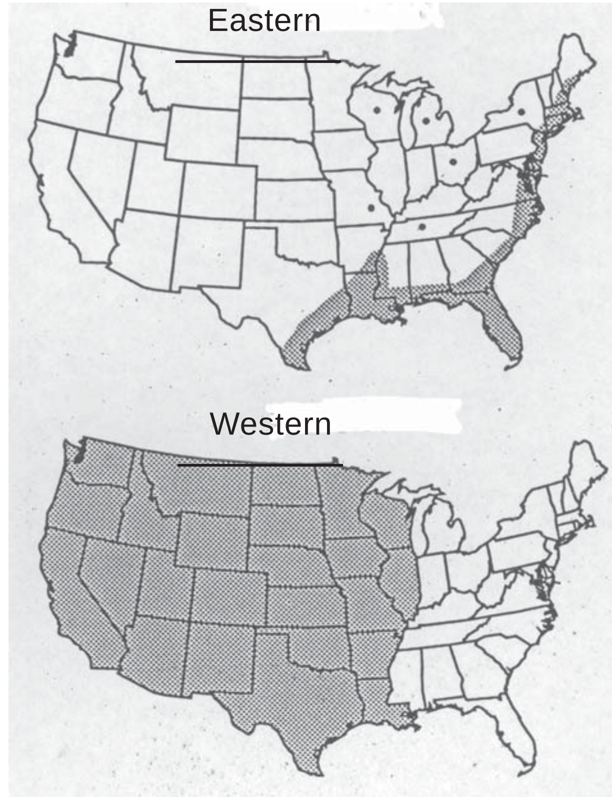

Fig. 21-9. The distribution of eastern and western equine encephalitis in the United States.
• Sporadic or focal infection reported
Source: Centers for Disease Control and Prevention. *Vector-borne Disease Control Self-study Course 3013-G*. Manual G. *Mosquitoes of Public Health Importance and their Control.* Atlanta: CDC; 1994: 4.

VEE is caused by a number of serotypes and causes fatalities in horses, mules, donkeys, and humans. In 1971, an outbreak of the 1B serotype of VEE virus occurred in Texas in association with a similar outbreak in Mexico.[45] Primary vectors during this outbreak in South Texas were *Psorophora columbiae, Ps discolor, Ae sollicitans,* and *Ae thelcter.* Other species of the genera *Culex, Anopheles, Mansonia,* and *Deinocerites* were also found infected in Texas and may be important vectors in other countries. Twenty major outbreaks of VEE between 1935 and 1971 have occurred in 11 countries but have mainly been centered in Colombia and Venezuela. VEE virus isolations from mosquitoes have mainly involved *Ae serratus, Ae taeniorhynchus, Ps confinnis,* and *Ps ferox.* A study[46] showed that VEE virus in *Ae aegypti* invaded many of the mosquito's tissues, with the highest levels in the salivary glands.

WEE is found in the United States west of the Mississippi River and in Wisconsin, Illinois, and Canada. It is also present along the eastern seaboard of North and South America and in eastern Europe. There were many major outbreaks in horses in the 1930s, with thousands of cases and many deaths. The largest human epidemic, involving approximately 3,000 cases, occurred in the western United States in 1941. Another large epidemic was reported in 1990 in the central valley of California.[47]

Culex and *Culiseta* mosquitoes are known vectors of this virus. In western endemic areas, *Cx tarsalis* is the most important vector of WEE because of its suitability for propagating and retaining WEE virus[48] (Figure 21-10). WEE has been isolated from *Cs melanura* in the eastern United States.[49] Isolations

Fig. 21-10. *Culex tarsalis*, a major vector of western equine encephalitis.
Source: The Ken Gray Image Collection, Oregon State University Department of Entomology, Corvallis, Ore.

Fig. 21-11. *Phlebotomus papatasi*, a phlebotomine sand fly vector of Old World cutaneous leishmaniasis.
Photograph: Courtesy of Ed Rowton, Walter Reed Army Institute of Research, Silver Spring, Md.

of WEE virus have also been made from many other species of mosquitoes and from birds.[50]

Flies

Sand Flies

Sand flies are Diptera of the family Psychodidae. Nonbiting moth flies in the genus Psychoda are also in this family, but only the sand flies are medically important. Sand flies occur mainly in the tropics and subtropics, with a few species ranging into temperate zones of the northern (to 50° N) and southern (to 40° S) hemispheres. Distribution is limited to areas that have temperatures above 15.6°C for at least three months of the year. There are no sand flies in New Zealand or on Pacific Islands. The genera *Phlebotomus, Chinius* (represented by a single species in China), and *Sergentomyia* occur in the Old World and the genera *Brumtomyia, Warileya*, and *Lutzomyia* occur in the New World. Anthropophilic sand flies in the Old World are distributed mostly in the subtropics, with a few human-biters south of the Sahara and none in Southeast Asia. In the New World, they are limited mainly to the tropics.[3]

Characteristics. Sand flies are small (2–4 mm in length), delicate flies with long, thin legs and narrow, pointed wings with parallel venation (Figure 21-11). They are small enough to pass through the mesh of a standard mosquito net. At rest, the wings are held erect over the abdomen at a 45° angle. The proboscis of the sand fly is short, and the antennae are long. Only the females bite and suck blood, thus acting as disease vectors. The bite of the female sand fly is typically sharp and painful and often causes considerable irritation. Extensive reviews of the biology and vector associations of New World[51] and Old World[52] sand flies have been published. There are over 700 species of sand flies that are found in a wide range of habitats.

Female sand flies require vertebrate blood for maturation of their eggs. Some species feed once between ovipositions; others may take multiple blood meals during a single oviposition cycle. Most human-biters feed at dusk and during the evening, but some species will bite during the daytime as well if they are disturbed in their resting site. Windless or nearly windless, dark conditions may suddenly induce great numbers of sand flies to seek hosts. The majority of anthropophagic sand flies are also exophagic, biting persons outside their houses. However, some species are endophagic, readily entering human dwellings, where they bite the occupants and either leave or rest inside.

The type of resting site used by adult sand flies varies according to species, availability of microhabitat, season, and amount of moisture present. In the New World, most species prefer the tropical rain forest. The forest has many microhabitats used by resting sand flies, such as on the underside of leaves; on or under tree bark, on tree trunks, and in animal burrows; in hollow trees; and in rock crevices and caves. In the Old World, sand flies tend to breed and rest in drier habitats. They can be found associated with termite mounds, cracks and fissures in the soil, animal burrows, piles of rubble, cracks in stone or brick walls, and caves. In peridomestic situations in both the New and Old World they may be found resting on walls inside human or animal dwellings.

Diseases Transmitted by Sand Flies

The Leishmaniases. The public health impact of the leishmaniases has been grossly underestimated due mainly to lack of public awareness. Only during the last 2 decades has it become increasingly apparent that most forms of leishmaniasis are much more prevalent than previously expected.[53] The leishmaniases are endemic on four continents in 22 New World and 66 Old World countries; 16 are developed countries, 72 are developing countries, and 13 are among the least-developed countries. Major epidemics of visceral leishmaniasis are in progress in Brazil, India, and the Sudan. In Bihar State in eastern India, it is believed that 200,000-250,000 people contracted the disease in 1993. In southern Sudan 15,000 cases were treated and 100,000 deaths occurred in a 5-year period (1991-1996). Cutaneous leishmaniasis is on the rise in many countries with epidemics ongoing in newly settled areas of the Amazon basin, North Africa and the Middle East.[53,54]

Among the more than 500,000 service members deployed to the Persian Gulf region during the Persian Gulf War, 28 cases of leishmaniasis were identified in personnel after their return from the theater of operations. Of these, 11 were identified as viscerotropic, and 17 presented with cutaneous lesions.[55] The causative agent of the visceral form, the protozoa *Leishmania tropica*, was previously thought to cause only cutaneous disease.[56]

Although not a war stopper, leishmaniasis is a persistent health threat to US military personnel because they are deployed or conduct military exercises in locations where the disease is endemic and its overall potential to compromise mission objectives is significant (Figure 21-12). Patients seen at Walter Reed Army Medical Center, Washington, DC, have included both active duty and reserve service members from all three branches of the US military, as well as personnel temporarily or permanently assigned to endemic areas. The number of units represented by this relatively small number of patients suggests that many more service members were at risk.[57]

Fig. 21-12. The geographic distribution of leishmaniasis. Courtesy of Colonel Raj Gupta, MS, US Army, and the Walter Reed Army Institute of Research, Washington, DC.

Fig. 21-13. Verruga peruana lesions on a child in Peru. This extreme case resolved after treatment.
Photograph: Courtesy of Richard Andre, Uniformed Services University of the Health Sciences, Bethesda, Md.

Bartonellosis. Bartonellosis (Carrion's disease), also believed to be transmitted by sand flies, is caused by the bacterium *Bartonella bacilliformis* and only occurs in rural areas in the Andes mountains of South America. The disease causes two distinct clinical manifestations in humans: Oroya fever and verruga peruana. The former condition is characterized by severe hemolytic anemia, joint pains, pallor, fever, and jaundice. Mortality rates range as high as 40% if untreated. *Salmonella* infections are serious concomitant complications of Oroya fever.

The other, less life-threatening form of bartonellosis, verruga peruana, is named for the warty appearance of the skin of infected persons (Figure 21-13). These lesions usually develop following recovery from Oroya fever and may last for up to a year. The number and appearance of the painless nodules varies considerably. *Lutzomyia* sand flies are the suspected vectors of both forms of this disease.[58] Humans are thought to be the only known host.

Sand Fly Fever. Viruses in three families and three genera are transmitted to humans and other vertebrates by sand flies. Symptoms of human illness usually resemble influenza, with fever, retro-orbital pain, myalgia, and malaise. Complete recovery within a week is the norm, but encephalitis has been reported in at least two patients infected with vesiculovirus. Sand fly fever (also known as 3-day fever or papataci fever), the best known example, occurs in the Mediterranean region, the Middle East, Pakistan, and northern India where *Phlebotomus* vectors exist. Stiff neck and limbs, red face, severe headache, and painful neck muscles may occur. Long-lasting immunity is conferred after the first attack. The disease has been considered of military importance because up to 75% of nonimmune adults arriving in an endemic area can be affected.[59]

In the Mediterranean area, the sand fly *Phlebotomus papatasi* is the suspected vector of sand fly fever in humans. Although little if anything is known about transmission cycles, it is suspected that vertebrates, rather than insects, serve as reservoirs of the viruses.[2]

Black Flies

Black flies are blood-sucking Diptera of both public health and economic importance. Besides being disease carriers, they are annoying to humans and domestic animals because of their biting and crawling habits. Black flies belong to the family Simuliidae, which contains several genera but only one of which, *Simulium,* is an important human-biting fly. These are small, stoutly built, hump-backed flies. Only the female feeds on blood and is a daytime feeder. These flies have a worldwide distribution where suitable conditions exist; there must be well-oxygenated water for the immature stages to develop. The larvae feed by filtering particles from the water and brushing microorganisms into the mouth using a pair of prominent mouth brushes. Adults emerge from the water, often attacking in large numbers. In the tropics, there are multiple generations each year. The mouthparts of the female are short and broad. When feeding, they tend to stab the tissue and wait for the ruptured capillaries to ooze blood rather than sucking neatly like mosquitoes do. *Simulium* bites cause considerable pain and irritation and frequently become infected (Figure 21-14). The punctum heals by forming a characteristic black scab.

The parasitic round worm, *Onchocerca volvulus,* which causes onchocerciasis, is transmitted by *Simulium* species. Of the 18 million people infected with this parasite, more than 95% live in Africa.[60] The clinical manifestations of the disease include dermal nodules and lymphatic and systemic complications due to vessel blockage by the microfilariae. This causes severe itching, papule formation in the skin, and thickening and loosening of the skin, which subsequently hangs in folds. The most severe complications are onchocercal lesions of the eye, which may lead to blindness ("river blindness"). This affects 40,000 new victims each year. The 1990s, however, saw a decline in the prevalence of onchocerciasis infection and morbidity due to the remarkable success of vector control programs in West Africa.

Fig. 21-14. A black fly (*Simulium* sp).
Photograph: Courtesy of Colonel Philip Lawyer, MS, US Army.

Black flies also act as mechanical vectors of myxomatosis and certain arboviruses, such as Venezuelan equine encephalitis. However, this mode of transmission is normally of minor importance.

Tsetse Flies

There are at least 23 species of tsetse flies belonging to the genus *Glossina.*[61] These flies are found only on the continent of Africa south of the Sahara desert and infest an area of approximately 12 million square kilometers. They are larger than a house fly and can easily be recognized by the wings, which extend beyond the abdomen when the fly is at rest and lie one over the other like a crossed pair of scissors. The wings also have characteristic venation, with a discal medial cell shaped like a hatchet or cleaver (Figure 21-15). Both sexes of tsetse suck blood and feed

Fig 21-15. The tsetse fly (*Glossina morsitans*) after taking a blood meal.
Photograph: Courtesy of Ed Rowton, Walter Reed Army Institute of Research, Silver Spring, Md.

exclusively by day.

Two groups of *Glossina* are involved in the transmission of African trypanosomiasis or "sleeping sickness" to humans. Other species transmit the trypanosome causing nagana in cattle. The riverine areas of West Africa, mainly the Niger River and Congo River basins, are the habitat of *G palpalis,* vector of the chronic Gambian form of the disease caused by *Trypanosoma brucei gambiense.* Further inland, where the rivers shrink into pools in the dry season, the vector is the smaller species *G tachinoides.*[2,3]

In the dry bush country of East Africa, species of tsetse occur that do not require the humid conditions of river bank vegetation. *G morsitans* is found in wooded savanna where rocks and trees provide the shade that protects the fly from desiccation. *G swynnertoni* inhabits much more open terrain where the only shade is provided by large animals. Both species are associated with the Rhodesian form of the trypanosome found in wild game and infective to humans, *T brucei rhodiesiense.*

Deer Flies and Horse Flies

Although of only minor importance to military operations, deer flies and horse flies can be a nuisance in localized areas. Deer flies (genus *Chrysops)* and horse flies (genus *Tabanus)* are the two most common genera in the family Tabanidae. They are robust flies varying in length from 5 to 30 mm and have clear or mottled wings. The bite of female deer flies and horse flies can cause a severe reaction in a sensitized host, with considerable edematous swelling, pain, and irritation (Figure 21-16).

Species of *Chrysops* known as mango flies, *C silacea* and *C dimidiata* in tropical west Africa (from Sierra Leone to Ghana and Nigeria) and *C distinctipennis* in central Africa (southern Sudan and Uganda), transmit the filarial worm, *Loa loa*, which causes loiasis in humans. The microfilariae are taken up by the female fly from the peripheral blood of an infected human host during the day. The parasite is passed to the susceptible host during a subsequent blood meal. Adult worms live under the skin and migrate around the body, causing allergic "Calabar" swellings and abscesses at the site of the dead worms. The adult worm becomes visible to the patient if it crosses the anterior chamber of the eye, but it does not cause serious ocular damage. Occasionally, worms invade the brain, causing meningoencephalitis. Some tabanids, particularly certain species of *Chrysops*, are known to serve as mechanical vectors of tularemia, anthrax, and possibly other diseases.[62]

a b

Fig. 21-16. (**a**) a horse fly (*Tabanus punctifer*) and (**b**) a deer fly (*Chrysops discalis*).
Source: The Ken Gray Image Collection, Oregon State University Department of Entomology, Corvallis, Ore.

Filth Flies

The term filth fly is generally applied to flies that live in close association with humans (synanthropes) and which feed readily on human feces and other decomposing organic matter, as well as food intended for human consumption.[63] These flies are of medical importance because they act as mechanical vectors of pathogenic organisms, especially those causing enteric diseases.

The fly genera *Musca, Fannia*, and *Calliphora* all contain important filth fly species. These flies are medium-to-large, burly flies with conspicuous compound eyes and a well-developed thorax and abdomen (Figure 21-17). They lay eggs on feces or other decaying organic matter. The maggot-like larvae subsequently develop and consume the organic matter. The adult fly consumes only liquid food. The fly regurgitates fluid from its crop onto a food source to liquefy the material, and then sucks it up.

Throughout history, military personnel have suspected the association between flies and enteric

Fig. 21-17. A female filth fly (*Musca domestica*).
Source: The Ken Gray Image Collection, Oregon State University Department of Entomology, Corvallis, Ore.

diseases. They have observed that a rise in enteric disease follows a rise in fly populations. Greenburg discusses evidence that *Shigella, Salmonella*, infectious hepatitis virus, and typhoid fever virus are transmitted and spread by a variety of filth fly species. Effective fly control programs have demonstrated on many occasions that fly control can reduce the incidence of enteric disease.[63] However, there are many examples that caution against an exclusively entomological approach to the epidemiology of enteric diseases. There are many potential vehicles for transmission of such diseases, and their importance depends in part on the habits and lifestyle of the people involved. Thus, field sanitation and personal hygiene are as important today as they always have been.

Eye Gnats

Hippelates eye gnats of the family Chloropidae are an important group of synanthropes that frequent the face and eyes, as well as the mucous and sebaceous secretions and the wounds, pus, and blood of humans. Certain species have long been suspected as possible vectors of conjunctivitis and pinkeye or sore eye of humans and animals.[63] In Jamaica, *Hippelates flavipes*, which feeds avidly on humans, has been implicated in yaws transmission.

Adult eye gnats fly throughout the day. These flies are nonbiting but may produce minute incisions with their labellar spines. They feed on exudates of sores and cuts and around natural orifices.

Blow Flies, Bot Flies, and Flesh Flies

Myiasis is the term given to an infestation of living tissue by dipterous larvae. Some species of fly are

a

b

Fig. 21-18. A warblelike lesion produced by the larva of the human bot fly, *Dermatobia hominis*, is shown in photograph (**a**). A *Dermatobia hominis* larva is shown in photograph (**b**). The adult fly lays her eggs on the abdomen of a blood-sucking insect, such as a mosquito (and sometimes a tick). When the mosquito bites a host, the proximity of the *Dermatobia* eggs to the host causes the eggs to hatch and the bot larvae burrow into the skin.
Photographs: (a) Courtesy of Colonel Philip Lawyer, MS, US Army. (b) Armed Forces Institute of Pathology, Washington, DC.

Fig. 21-19. *Calliphora vicina*, the flesh fly.
Source: The Ken Gray Image Collection, Oregon State University Department of Entomology, Corvallis, Ore.

unable to develop through their immature stages except on living tissue. This condition is known as specific or obligatory myiasis and is restricted to tropical species. Examples include the Old World screwworm (*Chrysomyia bessiana*), the tumbu fly *(Cordylobia anthropophaga)* and the Congo floor maggot *(Auchmeromyia luteola)* in Africa, and the New World screwworm (*Cochliomyia hominivorax*) and the human bot fly *(Dermatobia hominis)* in Central and South America. Specific myiasis occurs when fly larvae invade the skin. The infection presents as multiple painful blind boils in which secondary infection may occur (Figure 21-18). The head of the boil shows the black spiracles of the fly larvae. Treatment involves the suffocation of the larvae with an oily ointment, after which they can be removed with forceps; care must be taken to ensure that the larvae are removed intact.[5] Another method that seems effective and less likely to result in infection or other complications is commonly used in endemic areas of South and Central America. A slab of raw meat is placed over the boil and held there overnight. When the fly larva surfaces to breathe, it crawls between the surface host's skin and the meat, or it may burrow into the slab of meat and can then be removed. The presence of larvae can be psychologically distressing, but they are not usually life threatening.

Some larvae of *Calliphora, Lucilia*, and *Sarcophaga* (the flesh fly) species that normally develop on decaying organic matter will readily feed on wounds or other live, damaged, or contaminated tissue (Figure 21-19). This condition is known as semi-specific or facultative myiasis and can sometimes occur in the anal and genital regions. Accidental myiasis occurs when fly eggs or larvae are swallowed in food, and it results in intestinal disturbances.

Fleas

Fleas are small, wingless insects that can be important vectors of disease and often are serious pests. Their bodies are flattened laterally, are usually brown, heavily sclerotized, and generally possess bristles. The legs are well developed for jumping. The oriental rat flea, *Xenopsylla cheopis,* found on commensal rats in many parts of the world, is the most important vector of bubonic plague and murine typhus. The plague bacilli and murine typhus rickettsiae are ordinarily acquired by the flea during feeding on infected rats. Then the pathogens may be transmitted to humans in the absence of the flea's normal hosts, as when the rats are dying of plague in an epizootic. Plague exists in a sylvatic form in widespread parts of the world, and many of the 2,000 species of fleas are vectors of this disease.[2] Fleas can be very annoying, and their bites may produce itching and dermatitis in sensitive individuals.[5]

There are identification keys for most common fleas of public health importance. Fleas are generally discussed by families, which are based on the fleas' morphological characteristics and their ability to serve as vectors. The families are Ceratophyllidae, Leptopsyllidae, Pulicidae, and Tungidae. Family Pulicidae includes a significant number of species that are pests of humans, domestic fowl, and pets; are vectors of the plague pathogen; and are putative vectors of murine typhus to humans.

Fleas have piercing and sucking mouthparts and feed exclusively on blood. They lay their eggs on or among the hairs or plumage of the host or on debris on the ground. Eggs hatch into yellowish white, maggot-like larvae. The flea larva usually has one or two rows of sparse but well-developed bristles on most of its segments. The larva usually lives in the nest of the host and feeds on host-associated organic debris including food particles, dried skin, dried blood, or excreta. Larvae of some medically important species undergo three molts in 2 to 3 weeks. It may take a few months in other species. The larva spins a silken cocoon around itself, thus entering the pupal stage. The pupa develops into an adult in a few days. Adult emergence from the cocoon may be triggered by vibrations resulting from host movements. Adults may live for weeks or months, sometimes even without food. If environmental conditions are unfavorable, or if hosts are not available, developing adult fleas may remain inactive within the cocoon for extended periods.

Lice

Lice are small (1-4 mm long), wingless insects with elongated or discoidal bodies that are compressed or flattened dorso-ventrally. Three kinds of bloodsucking lice infest humans: body lice, head lice, and crab (pubic) lice. The head louse and body louse are somewhat elongated and similar in appearance, but the head louse is the smaller of the two. The crab louse is much smaller and has a nearly round, hairy body. The life cycles of these three species vary slightly, but all take place on the body or clothing of the host. The body louse confines its feeding to the host's body and remains on the clothing next to the skin. The head louse lives in the hair of the head and the crab louse among the hairs of the pubic region. The crab louse can also be found on the hairs of the legs, the chest, the armpits, and occasionally the beard and eyebrows.

The genera *Pediculus* and *Pthirus* contain the lice that normally infest humans (Figure 21-20). *Pediculus humanus,* containing both the head louse (*P humanus capitis*) and body louse (*P humanus humanus*—no longer *P humanus corporis*) of humans, is one of the three or four species included in genus *Pediculus.* Ferris[64] has discussed the nomenclature in detail. The genus *Pthirus* includes the crab louse, *Pthirus pubis.*

The most important member of this group is the human body louse. Historically, it has had a more profound effect on the history of humanity than has any other insect.[65] Lice have always been associated with wars because they thrive when sanitary conditions are poor and human populations are homeless and dislocated.

Head Lice

The head louse is gray but tends to resemble the color of the hair of the host. Head lice remain mostly

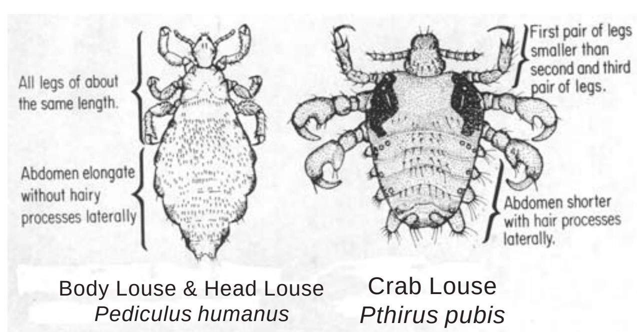

Fig. 21-20. The body louse, head louse, and crab louse. Source: Centers for Disease Control and Prevention. *Vector-borne Disease Control Self-study Course 3013-G*. Atlanta: CDC; 1994: 11.

on the scalp. Head lice have been pests of humankind throughout history and pose no health threat to infested persons. However, the infestation may lead to embarrassment and social ostracism. Head lice are small (1–3 mm long), elongate, wingless insects that occur on the head and around the ears and occiput, but heavy infestations may move on to other parts of the body. The female lays the eggs on the hair. The eggs are glued to the hair and take about 5 to 10 days to hatch. The nymphal stage includes three molts before changing into the adult stage. The entire life cycle takes about 3 weeks.

Body Lice

The body louse looks almost identical to the head louse, but it usually stays on clothing and makes contact with the body while feeding. In heavy infestations, some lice may remain on the human body even when all clothing has been removed. Eggs are deposited in the seams of clothing. The incubation period varies from 5 to 7 days when eggs are laid near the body; it is longer at lower temperatures. After hatching, the young lice begin to suck blood at once and feed frequently throughout the nymphal stage. Like the head lice, the body lice nymphs go through three molts before becoming adults and the egg-to-egg cycle takes about 3 weeks. The optimum temperature for development is similar to normal human body temperature. The louse does not leave the body unless the body becomes too cold (death) or too hot (high fever). New louse infestations mainly occur during contact with louse-infested persons or their clothing.

Crab Lice

Crab lice are only found on humans and die within 24 to 48 hours if forced to leave the host.[66] The eggs are laid mainly on the coarse hairs of the pubic area. Nymphs pass through three molts before becoming adults. The total life cycle takes about 3 to 4 weeks. Pubic lice are transmitted from person to person, most often by sexual contact.

Diseases Transmitted by Lice

Three diseases of public health importance that are associated with lice are louse-borne (epidemic) typhus, trench fever, and epidemic relapsing fever. *Pediculus humanus humanus* is probably the sole vector of the organisms that cause these diseases. Louse-borne (epidemic) typhus has been recognized since ancient times and occurs mainly in Europe; the African highlands, especially Burundi, Rwanda, and Ethiopia; Asia; and the higher altitudes of Mexico, Central America, and South America.[2] The causative organism is *Rickettsia prowazekii.*

Trench fever is a nonfatal disease, and the causative agent is *Bartonella quintana*. This organism multiplies freely in the lumen of the louse's digestive tract and is not pathogenic to the louse. Epidemic relapsing fever has occurred in many parts of the world. The pathogen is a spirochete, *Borrelia recurrentis.* The insect can acquire the pathogen by a single feeding on an infected person but cannot directly pass it on to a second person. It is transmitted by crushing the louse and so releasing its infected hemolymph onto the skin.[67]

Bugs

The true bugs may be winged or wingless, but they always have a proboscis or beak suitable for piercing and sucking that is attached anteriorly and kept flexed under the head when not in use. The two bugs of medical importance belong to the families Cimicidae (bedbugs) and Reduviidae (assassin and kissing bugs).

Assassin and Kissing Bugs

These bugs are commonly called cone nose bugs because of their elongate (cone-shaped) head (Figure 21-21). Most Reduviids "assassinate" or kill other insects, but a small group of reduviids belonging to subfamily Triatominae exclusively feeds on the blood of vertebrates.[2] They are also called kissing bugs because occasionally they take the blood meal from around the lips of the host. The bites of these bugs are

Fig. 21-21. An assassin or kissing bug (Family Reduviidae), which is the vector of American trypanosomiasis. Photograph: Courtesy of Ed Rowton, Walter Reed Army Institute of Research, Silver Spring, Md.

usually painless but may cause urticaria if the host becomes sensitive to the injected saliva. Chagas' disease, or American trypanosomiasis, is one of the most important arthropod-borne diseases in tropical America; it is caused by *Trypanosoma cruzi*, which is transmitted by various species of Triatominae. The four principal vectors of Chagas' disease in Central and South America are *Panstrongylus megitus, Rhodnius prolixus, Triatoma infestans,* and *Triatoma dimidiata*. The most important vector in Mexico is *T barberi*, while *T gerstaeckeri* and *T proctata* are important species in the southwestern United States, and *T sanguisuga* is found throughout the United States.[68] The victims of this disease are mainly humans, dogs, and to a varying extent other domestic and wild animals. The infection mainly occurs in rural areas of the tropics and subtropics, but may occur in suburban and urban areas around poorly constructed houses.

Both sexes of triatomid bugs bite, and they take their blood meal at night and hide in any cracks or crevices when not feeding.[3] The pearl-like eggs are laid singly around the adult habitat. The incubation period varies, depending on the species and temperature. The nymphs typically go through five developmental instars. Nymphs camouflage themselves with dust particles or other debris. In temperate regions, some species overwinter in the egg stage, others as adults, and still others as nymphs.

Bedbugs

Bedbugs have been associated with humans for centuries. Bedbugs have been occasionally found infected with anthrax, plague, and typhus disease organisms, but they are not considered an important vector of these diseases.[5] They are extremely annoying; their bites produce small hard swellings or wheals that are often confused with flea bites. The bedbug's principal medical importance is the itching and inflammation associated with its bite. Two bedbug species attack humans: *Cimex lectularius* (in temperate regions) and *Cimex hemipterus* (in tropical areas).[2]

Bedbugs are dorso-ventrally flattened, reddish brown, wingless insects approximately 5 mm long. They lay eggs in wall cracks, furniture, bedding, and other sheltered places. The eggs hatch in about 6 days, and if a suitable host is available, the young bugs begin feeding on blood. Immature bedbugs look like adults except they are yellowish white. The nymphs mature into adults in 30 to 45 days. Adults live for 6 to 8 months and may survive for several days without food. The adults lack hind wings, and the forewings are reduced to two small pads.

Stinging Insects

Allergic reactions to insect stings are a common and often a serious medical problem: estimates of anaphylaxis in the general population range from 0.3% to 3%.[69] Insects that sting are members of the order Hymenoptera. There are two major subgroups: vespids (eg, yellow jacket, hornet, wasp) and apids (eg, honeybee, bumblebee); ants belong to a third subgroup. In most parts of the United States, yellow jackets are the principle cause of allergic reactions to insect stings.

The stinging apparatus originates in the abdomen of the female insect. It consists of a sac containing venom attached to a barbed stinger. A sting occurs when the stinger is inserted into tissue, the sac contracts, and venom is deposited into tissue.

Bees and Wasps

Although Africanized honeybees *(Apis mellifera scutellata),* the so-called "killer bees," have received much publicity, their venom is no more allergenic or toxic than that of European honeybees (*Apis mellifera mellifera*).[69] However, Africanized bees are much more easily provoked and more aggressive than European honeybees. This behavior can lead to massive stinging incidents. These bees are expected to keep moving northward in the United States, although they do not survive well in colder areas.

Reaction to honeybee stings range from slight pain and swelling to much more serious symptoms, including anaphylaxis. In the United States, deaths from all hymenopterous insects (bees, wasps, yellow jackets, and ants) average between 40 and 50 per year.[70] A single bee sting is seldom fatal unless its victim is hypersensitive and has a severe allergic reaction. All persons should know whether or not they are hypersensitive to bee and wasp stings (Figure 21-

Fig. 21-22. A wasp. Photograph: Courtesy of Ed Rowton, Walter Reed Army Institute of Research, Silver Spring, Md.

22). Hypersensitive persons should carry insect sting kits when frequenting an area where interaction with bees and wasps may occur.

Honeybees are often encountered in swarms, which can remain in an area a few hours or days. Bee swarms are only temporary and will move away as soon as the bees find a new location to colonize. However, situations may occur that require the immediate elimination of bee swarms. Swarms may be eliminated with approved insecticides or by using high-volume sprays of a 5% solution of liquid dishwashing soap.[70]

Fire Ants

The fire ant, *Solenopsis invicta,* is a nonwinged hymenopteran. It is responsible for an ever-increasing number of allergic reactions in the United States and throughout its range in North and South America. With its jaws, the fire ant attaches itself to a person. It then pivots around its head and stings with the abdominal stinger at multiple sites in a circular pattern. Within 24 hours, a sterile pustule develops that is diagnostic of a fire ant sting.

CLASS ARACHNIDA

The class Arachnida includes such diverse forms as ticks, mites, spiders, and scorpions and is found almost exclusively in terrestrial habitats throughout the temperate and tropical regions of the world. The most important of the arachnids—ticks, mites, and spiders—lack distinct body segmentation; scorpions, pseudoscorpions, and a few others are obviously segmented. The body is divided into two parts, the cephalothorax (comprosed of the combined head and thorax) and the abdomen. In ticks and mites, the cephalothorax and abdomen are strongly fused, giving the body a sac-like form. These arthropods have four pairs of legs, at least in the adult stage; larval ticks and mites have only three pairs of legs. None of the arachnids possess antennae or wings. The mouthparts lack mandibles and usually consist of a pair of piercing chelicereae and the pedipalpi, and in ticks and some mites, a hypostome. In general, arachnids are predatory or parasitic, although many mites are plant feeders or scavengers.

Like many of the medically important insects, ticks and some mites are important as mechanical and biological vectors of bacteria, rickettsia, viruses, and protozoa, which they transmit to humans and other animals by their bites.

Ticks and Mites

Ticks

There are three families of ticks, of which two contain species capable of transmitting pathogens to humans: the hard ticks (family Ixodidae) and the soft ticks (family Argasidae) (Figure 21-23). Hard ticks are

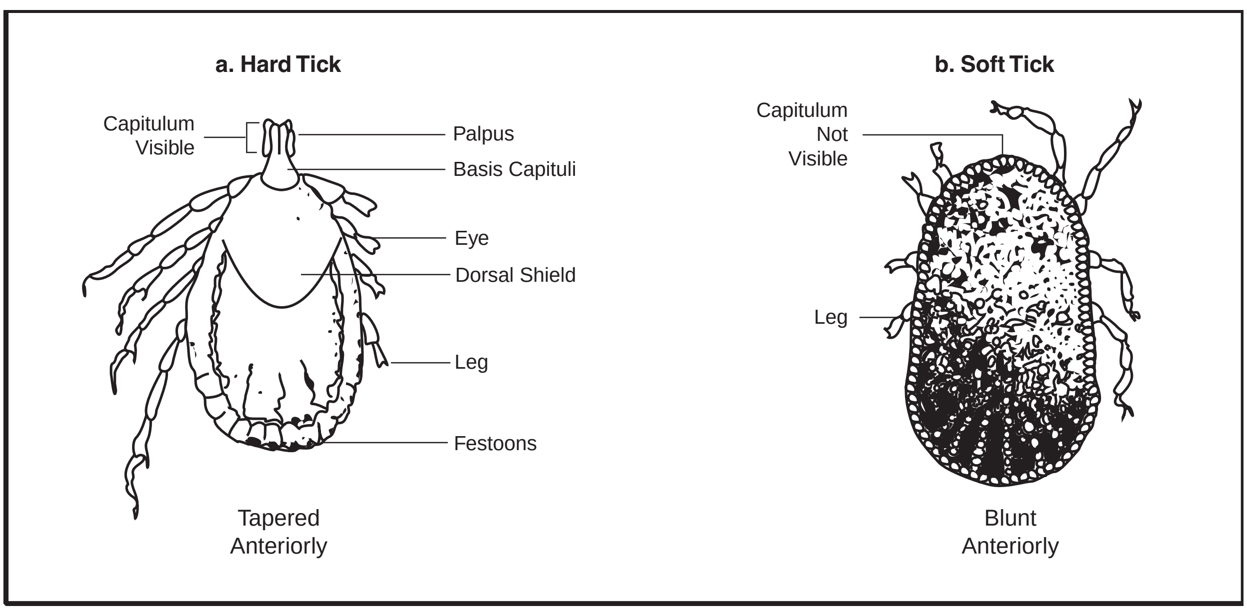

Fig. 21-23. The characteristics of female hard and soft ticks.
Source: Centers for Disease Control and Prevention. *Vector-borne Disease Control Self-study Course 3013-G*. Atlanta: CDC; 1994: 14.

oval with a sac-like body and vary in length from 2 to 10 mm when unfed, although the engorged female may be at least twice this size. They possess a hard dorsal plate called a scutum (hence the name "hard ticks"), and their mouthparts point anteriorly and are visible from above. Soft ticks are leathery and nonscutate. They are about the same size as the hard ticks. Their mouthparts point downward and are not visible from above.

Ticks feed by using their mouthparts to cut a small hole in the host epidermis; they insert the hypostome into the cut, thereby attaching themselves to the host. Blood flow is presumably maintained with the aid of an anticoagulant from the tick's salivary glands. Some hard ticks also secrete a "cement" to secure their attachment to the host. Blood-fed female ticks are capable of enormous expansion.

In the United States, more vector-borne diseases are transmitted by ticks than by any other arthropod.[71] Documented cases of ticks and tick-borne diseases affecting military personnel are increasing.[72] Ticks generally affect military operations in two ways: (1) directly, by tick bite and the accompanying psychological stress, and (2) indirectly, by disease transmission. Various bacteria, rickettsiae, viruses, and protozoa are transmitted to people via tick bites.

Lyme Disease. Lyme disease or Lyme borreliosis has emerged as the most common vector-borne disease in the United States. It also occurs in Eurasia and Australia. In the United States, 16,273 cases were reported in 1999[73] and nearly 50,000 cases were reported from 1982 through 1992.[74] Most human infections of *Borrelia burgdorferi* in the United States occur during the months of May through August, when tick activity is at its peak.

The causative agent of Lyme disease is the spirochete *B burgdorferi,* and it is transmitted by Ixodes ticks.[75] *Ixodes scapularis* (previously known as *I dammini*) (Figure 21-24) in the East and upper Midwest and *I pacificus* in the West are the primary vectors of this disease in the United States. *Ixodes ricinus* is the vector in eastern, central, and western Europe and *I persulcatus* in Japan, eastern Russia, and China.[76]

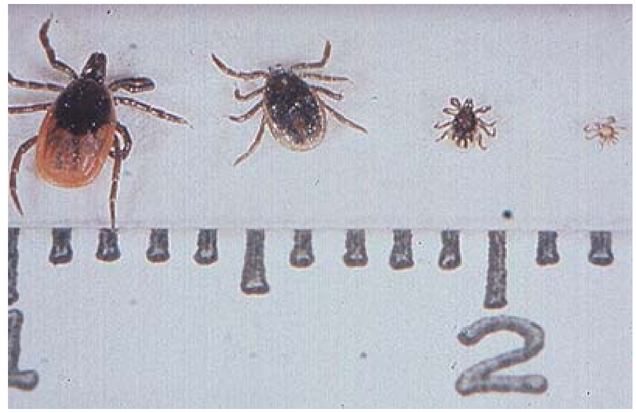

Fig. 21-24. Various life stages of the *Ixodes scapularis* tick. The larva has six legs and the nymph and adult have eight. The scale is in millimeters.
Photograph: Courtesy of Colonel Philip Lawyer, MS, US Army.

Tick-borne Relapsing Fever. Tick-borne relapsing fever is caused by *Borrelia* species other than *B burgdorferi* and is the one significant disease transmitted by soft ticks. In the United States, the geographic distribution of this disease is limited to remote, undisturbed natural areas in the West. The tick *Ornithodoros hermsi* transmits *B hermsi* in forested mountain areas, generally above an elevation of 900 m. In semi-arid plains areas, *Borrelia turicatae* is transmitted by *O turicata* ticks and *B parkeri* is transmitted by *O parkeri* ticks.[77] In general, only sporadic cases of tick-borne relapsing fever are diagnosed, but isolated epidemics have occurred in such places as the Grand Canyon.[78]

Tick-borne relapsing fever has an incubation period of 4 to 18 days, after which the illness characteristically begins abruptly with high fever, chills, tachycardia, headache, myalgias, arthralgias, abdominal pain, and malaise. If the disease is untreated, the primary fever breaks in 3 to 6 days and is followed by an afebrile interval of approximately 8 days. Without treatment, 3 to 5 relapses typically occur. The severity of illness generally decreases with each relapse. Death is rare and limited mainly to infants and the elderly. Antibiotics are an effective treatment.

Tularemia. Tularemia is caused by the bacillus *Francisella tularensis.* The most important reservoirs for *F tularensis* are rabbits, hares, and ticks. The many modes of transmission include tick bite, direct contact with infected animal tissues, inhalation of aerosolized organisms, ingestion of contaminated meat or water, and bites of infected mammals, deer flies, or mosquitoes.[77] The three major tick vectors of tularemia in the United States are *Amblyomma americanum* (Figure 21-25) in the southeastern and south central United States, *Dermacentor andersoni* in the West, and *D variabilis* in many parts of the country. Tick bites account for about 50% of the tularemia cases in the United States. The incubation period of this disease is usually 3 to 5 days. The severity of tick-borne tularemia is highly variable and ranges from mild, afebrile, self-limited disease to cases of fulminating septic shock.[79]

Rocky Mountain Spotted Fever. Rocky Mountain spotted fever is caused by *Rickettsia rickettsia*

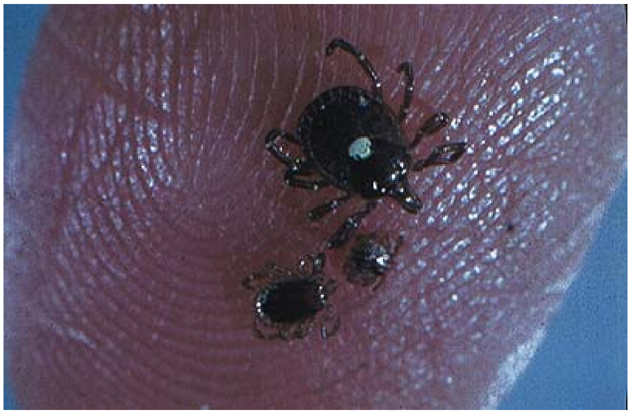

Fig. 21-25. *Amblyomma americanum*, the tick that transmits tularemia and human monocytic ehrlichiosis. The large tick at the top of the slide with the white spot is the female, the nymph is in the middle, and male is at the bottom. Photograph: Courtesy of James Occi, Biological Sciences, 195 University Avenue, Newark, NJ 07102.

and is transmitted by *Dermacentor variabilis* (Figure 21-26) in the eastern United States and *D andersoni* in the western United States. Although Rocky Mountain spotted fever is widely distributed across the United States, most infections are now acquired in the south Atlantic coastal region and the western and south-central states.[80] Three hundred sixty-five cases were reported in the United States in 1998.[81] This disease, if properly diagnosed, is easily treated with appropriate antimicrobial agents. Death is usually associated with missed or delayed diagnosis and mistreatment.

Fig. 21-26. *Dermacentor variabilis*, the dog tick. Photograph: Courtesy of Ed Rowton, Walter Reed Army Institute of Research, Silver Spring, Md.

Human Monocytic Ehrlichiosis. *Ehrlichia chaffeensis* is the sole causative agent of human monocytic ehrlichiosis in the United States.[82] This disease was first described in 1987 in a middle-aged man in Arkansas.[83] An outbreak of this disease also occurred in an Army Reserve unit.[84] Most of the 250 reported cases have occurred in the central and south Atlantic states. Recently, the ticks *D variabilis* and *Am americanum* have been identified as potential vectors.[77]

Ehrlichiosis generally presents as a nonspecific febrile illness that resembles Rocky Mountain spotted fever without the rash. Approximately 90% of patients have a history of a tick bite in the 3-week period preceding the onset of illness. Characteristic clinical features include high fever and headache, and other common symptoms include malaise, nausea, vomiting, myalgia, and anorexia. Acute complications, including acute renal failure, encephalopathy, respiratory failure, and death, are rare. Tetracycline (or its analogues) is an effective treatment.

Colorado Tick Fever. Colorado tick fever ("mountain fever") is caused by an RNA virus and is presumably transmitted by the tick *D andersoni.* Ground squirrels are the main vertebrate reservoir for this disease. The disease occurs in 11 Rocky Mountain states.[85] Most human infections are caused by tick bites, but the virus has also been transmitted by blood transfusion.[77]

Babesiosis. Babesiosis is a malaria-like illness caused by a protozoan parasite that invades erythrocytes. *Babesia microti* has been implicated in babesiosis acquired in the northeastern United States, and *B equi* is thought to be the causative agent of the disease in California.

Babesiosis is usually transmitted by the bite of *I scapularis* and *I pacificus* ticks. The white-footed mouse, *Peromyscus leucopus,* is the principal reservoir for *B microti* in the northeastern United States.

Tick Paralysis. Five tick species *(D andersoni, D variabilis, Am americanum, Am maculatum,* and *I scapularis)* can cause tick paralysis in humans. A neurotoxin that is produced in the tick's salivary glands is believed to cause the paralysis.[86] This toxin, which is usually transmitted by an engorged, gravid female tick, either blocks the release of acetylcholine at the synapse or inhibits motor-stimulus conduction.[77]

Diagnosis is made by finding an embedded tick, usually on the scalp. After removal of the tick, symptoms generally resolve within several hours or days. If untreated, tick paralysis can be fatal.

Boutonneuse Fever. An outbreak of boutonneuse fever in US Army personnel deployed to Botswana was reported in 1996.[87] More than 30% of the 169 deployed soldiers sought medical attention for boutonneuse fever symptoms. Subsequent serological tests indicated that 39 soldiers had boutonneuse fever. This disease is caused by *Rickettsia conori* and other rickettsia species and is believed to be transmitted by ticks. This disease has previously been reported from travelers returning from Morocco, Kenya, Botswana, and South Africa.

Mites

There are many free-living and predaceous mites, but some groups and families are exclusively parasitic. More than 200 families of mites are recognized in the entomological literature, but only a few contain species that affect humans. Some mites are vectors of organisms that cause human disease, and others cause dermatitis and allergic reactions in humans. Most species of mites are so small that they are barely visible to the naked eye. Their life cycles are often short (2 to 3 weeks), so mites can increase their numbers very rapidly under favorable conditions. For simplicity, the mites discussed here are grouped into five categories: chigger mites, house-dust mites, human biting mites, scabies mites, and follicle mites.[5]

Chigger Mites. Larvae of mites belonging to the family Trombiculidae are called "chigger mites" (Figure 21-27). There are approximately 20 different species in the family that are known to cause dermatitis or transmit diseases. The larval body is less than 1 mm long and more or less round. As adults, they are about 1 mm long, oval or, more often, hour-glass shaped. They are most often bright red. Larvae are six-legged and the only parasitic form. The adults and nymphs are eight-legged and free-living. Chigger larvae do not burrow into the skin or feed primarily on blood. The formation of a feeding tube, or stylostome, at the site of chigger attachment is characteristic of chigger attack. It is presumably the action of the mite's digestive fluid that causes the attachment site to itch after a few hours.

Fig. 21-27. Chiggers, which are trombiculid mites, are vectors of scrub typhus.
Source: Walter Reed Army Institute of Research, Silver Spring, Md.

The life cycle of chigger mites is very complex and includes six stages.[88] The female lays eggs singly on soil or litter, and the eggs hatch in about a week, exposing the six-legged larvae. The larval stage is the only parasitic stage. Larvae may attach themselves to many species of vertebrates. Engorged larvae leave the host and pass through a quiescent stage before they become nymphs and finally adults. The nymphs and adults are eight-legged and free living; they feed on insect eggs and other small soil invertebrates. The total life cycle takes about 60 days.

From a public health perspective, chiggers deserve attention because they are known to cause dermatitis and, more important, serve as vectors of scrub typhus. Chigger mites are found worldwide. Among the chigger mites that cause dermatitis is the European species *Trombicula autumnalis,* also known as the harvest mite, and *T alfreddugesi,* which occurs in the United States and parts of Central and South America. The chigger mite species that transmit scrub typhus, *Leptotrombidium deliense* and *L fletcheri*, are prevalent from New Guinea and the coastal fringe of Queensland, Australia, through the Philippines and China and westward through Southeast Asia to Pakistan.[2] *L pallidum* is reported as the vector of scrub typhus in parts of Japan, Korea, and the Primorye region of the former USSR.

Scrub typhus has caused significant casualties in Asian-Pacific military operations.[89] The American 6th Army in World War II lost more than 150,000 man-days during their operations in and around Schouten Islands and Sansapor beach head in Netherlands New Guinea.[90] The causal organism is *Rickettsia tsutsugamushi*. The disease has an incubation period of 6 to 21 days and is discussed in detail in Chapter 36.

House Dust Mites. House dust mites, in the family Pyroglyphidae, do not feed directly on living tissue but can be found in skin dander, stored food products, furniture, debris in household carpets, and areas that provide a variety of organic materials. Mites in the genus *Dermatophagoides* have been associated with house dust allergy and climate allergy.[91] The European house dust mite, *D pteronyssinus,* was implicated as an allergenic component of house dust to which most asthmatics are sensitive and that elicits the strongest responses.[2,92] The American house dust mites *D farinae, Euroglyphus maynei*, and *Glycyphagus destructor* have also been reported to cause allergies.[93]

Adult mites are plump and have well developed chelicerae and suckers. Their color varies from white to light tan. The life cycle takes about a month to complete, and adults may survive up to 2 months under optimum conditions. They are most abundant in home environments that are warm with high humidity.[94]

Biting Mites. The majority of the human-biting mites belong to the families Dennanyssidae, Macronyssidae, and Sarcoptidae. They are generally ectoparasites of poultry, wild birds, and rodents but may also attack humans, causing skin disorders and discomfort. The more medically important mites are the scabies mite and the hair follicle mite, which are discussed separately.

The tropical rat mite, *Ornithonyssus bacoti*, has been associated with debilitation, retarded growth, and high mortality in colonies of research mice. When the rats die or abandon their nests, the mites can travel considerable distances and bite humans, causing a sharp itching pain that may lead to development of dermatitis in sensitive individuals. The tropical fowl mite, *Ornithonyssus bursa,* is primarily an ectoparasite of poultry (it cannot exist for more than 10 days apart from its avian host), but it may bite humans. The bite causes only slight, temporary irritation.[2] *Ornithonyssus sylviarum,* the northern fowl mite, is a widespread parasite of poultry in New Zealand and Australia. The crawling of these mites is known to cause itching in personnel working on heavily infested farms. The chicken mite, *Dermanyssus gallinae,* commonly found on domestic fowl, pigeons, English sparrows, starlings and other birds, is considered one of the most common species causing dermatitis in humans, especially those working in poultry houses and on farms.

The house mouse mite, *Liponyssoides sanguineus,* is primarily an ectoparasite of mice but has been reported to feed on rats and other rodents and will readily attack humans. This mite is known to transmit the rickettsial pox pathogen to humans. The etiologic agent is *Rickettsia akari*, which is primarily found in the house mouse, *Mus muscularis,* but may be transmitted to humans by accident.

The straw itch mite, *Pyemotes tritici*, belongs to the family Pyemotidae. It commonly attacks a variety of stored-grain insects and is highly toxic to humans. Dermatitis associated with *P tritici* is known as straw, hay, or grain itch. The infestation generally occurs after sleeping on straw mattresses, working in grain fields during harvesting, or coming in contact with various grains or materials infested with the mites.

Scabies Mites. The scabies mites belong to the family Sarcoptidae. The family includes the genera *Sarcoptes, Notoedres*, and *Trixacarus*, each producing a particular type of dermatosis (Figure 21-28). *Sarcoptes scabiei* causes scabies, also known as 7-year itch or Norwegian itch. The mite most often is found on skin between the fingers, at the bend of knees and elbows, on the penis, on the breasts, and on the shoulder blades. Rash and itching are not experienced in newly infested persons for up to a month after infestation. The rash and itching are directly associated with the burrowing of mites into the skin. Human scabies occurs worldwide.

Follicle Mites. Follicle mites, *Demodex folliculorum* and *Demodex brevis*, live in hair follicles and sebaceous glands, respectively.[95] They are mainly found around the eyelids, nose, and other facial areas. Most commonly, the infestation is benign but may result in the loss of eye lashes or in granulomatous

Fig. 21-28. A scabies mite.
Photograph: Courtesy of Richard G. Robbins, PhD, Defense Pest Management Information Analysis Center, Armed Forces Pest Management Board.

Fig. 21-29. The ventral view of the follicle mite *Demodex folliculorum*.
Source: Uniformed Services University of the Health Sciences, Bethesda, Md.

Fig. 21-30. The brown recluse spider (*Loxosceles reclusa*)
Photograph: Courtesy of Ed Rowton, Walter Reed Army Institute of Research, Silver Spring, Md.

acne.[96] The follicle mite is an elongated species (Figure 21-29).

Spiders and Scorpions

There are several thousand species of spiders worldwide and the size range is considerable, the smallest being a few millimeters in length and the largest having a leg span of up to 20 cm. Spiders use their mouthparts to capture their prey and sometimes cause paralysis by injecting venom. The venom of a few species is particularly toxic to humans.

Large, hairy tarantulas found in many tropical and Mediterranean countries are rarely dangerous, although their large size is commonly frightening. In contrast, the small widow spiders (ie, *Lactrodectus* species, which includes *L mactans*, the black widow spider) found in most temperate, tropical, and subtropical regions, can be particularly toxic and sometimes fatal. Other species (eg, the brown recluse spider, *Loxosceles reclusa*, Figure 21-30) can cause considerable necrosis at the site of the bite. The bite of almost any large spider, although not necessarily poisonous, may be contaminated with pathogenic bacteria and may result in extensive and dramatic secondary infection.

Scorpions are found in many parts of the tropics and warm temperate regions. They are found in both humid and arid areas. Adults have eight legs, large pedipalps, and a flexible tail with a needle-like stinger. They typically exhibit cryptic behavior, sheltering under rocks, logs, and other debris. All scorpions inject a paralyzing poison into their prey and feed on the fluid content. All these animals should be presumed to be dangerous and are to be avoided.

SUMMARY

Arthropods and the diseases they transmit have had devastating effects on US and foreign military forces in the past and there is great potential for these effects to be felt in the future. Arthropod-borne disease and injury can severely affect training and can render entire units temporarily or permanently ineffective. Deployed military personnel are particularly vulnerable to arthropods and often are immunologically naïve to the diseases they carry. One of the maxims of war is to know your enemy. This applies to arthropod enemies as well at to human enemies. The more preventive medicine professionals know regarding arthropods of military medical importance, the more likely they are to be able to implement countermeasures to protect service members.

Acknowledgment

The authors wish to thank Major (retired) Louis Rutledge, Dr. (Captain) Pollie Rueda, Dr. Michael Turell, Dr. Curtis Hayes, Colonel (retired) William Bancroft, Lieutenant Colonel Jeffrey Gambel, and Colonel (retired) Don Johnson for their insightful review of the chapter. Dr. Edgar Rowton, Dr. Richard Wilkerson, and Medical Audio-Visual Services personnel from the Letterman Army Institute of Research and the Walter Reed Army Institute of Research were very helpful with photographs and illustrations.

REFERENCES

1. Peterson RKD. Insects, disease, and military history. *Am Entomologist*. 1995;41:147–161.
2. Harwood RF, James MT, eds. *Entomology in Human and Animal Health*. 7th ed. New York: MacMillan Publishing Co; 1979.
3. Eldridge BF, Edman JD, eds. *Medical Entomology: A Textbook on Public Health and Veterinary Problems Caused by Arthropods.* Boston, Mass: Kluwer Academic Publishers; 2000.
4. Clements AN. *Development, Nutrition and Reproduction*. Vol 1. In: *The Biology of Mosquitoes*. London: Chapman and Hall; 1992.
5. Goddard J. *Physician's Guide to Arthropods of Medical Importance*. 2nd ed. Boca Raton, Fla: CRC Press; 1996.
6. Service MW. *Medical Entomology for Students*. London: Chapman and Hall; 1996.
7. Bates M. *The Natural History of Mosquitoes*. New York: MacMillan Publishing; 1949.
8. Christophers SE. Aedes aegypti *(L.) the Yellow Fever Mosquito: its Life HistoryBionomics, and Structure*. London: Cambridge University Press; 1960.
9. Clements AN. *The Physiology of Mosquitoes*. Oxford: Pergamon Press; 1963.
10. Gillett JD. *Mosquitoes*. London: Weidendield and Nicolson; 1971.
11. Gillett JD. *The Mosquito: Its Life, Activities, and Impact on Human Affairs*. Garden City, NY: Doubleday; 1972.
12. Horsfall WR. *Mosquitoes: Their Behavior and Relation to Disease*. New York: Ronald Press; 1955.
13. Oaks SC Jr, Mitchell VS, Pearson GW, Carpenter CCJ, eds. *Malaria: Obstacles and Opportunities*. Washington, DC: Institute of Medicine, National Academy Press; 1991.
14. World Health Organization. World malaria situation in 1992, I. *Weekly Epidemiol Rec.* 1994;69:309–314.
15. World Bank. *World Development Report 1993: Investing in Health*. New York: Oxford University Press; 1993.
16. Newton JRA, Schnepf GA, Wallace MR, Lobel HO, Kennedy CA, Oldfield EC III. Malaria in US Marines returning from Somalia. *JAMA*. 1994;272:397–399.
17. Centers for Disease Control and Prevention. Malaria among US military personnel returning from Somalia, 1993. *MMWR*. 1993;42:524–526.
18. Feighner BH, Pak SI, Novakoski WL, Kelsey LL, Strickman D. Reemergence of *Plasmodium vivax* malaria in the Republic of Korea. *Emerg Infect Dis*. 1998;4(2):295–297.

19. Otteson EA. Filarial infections. *Infect Dis Clin N Am.* 1993;7:619–633.

20. Michael E, Bundy DAP. Global mapping of lymphatic filariasis. *Parasitol Today.* 13:472–476.

21. Swartzwelder JC. Filariasis bancrofti. Coates JB Fr, Hoff EC, Hoff PM, eds. *Communicable Diseases: Arthropodborne Diseases Other Than Malaria*. Vol 7. In: *Preventive Medicine in World War II*. Washington, DC: Office of the Surgeon General, Department of the Army; 1964.

22. Zeilke E. Studies on the mechanism of filarial transmission by mosquitoes. *Z Tropenmed Parasitol*. 1973;24:32–35.

23. Sosa O, Carlos J. Finlay and yellow fever: A discovery. *Bull Entomol Soc Am*. 1989:35:23–25.

24. Reed W, Carroll J, Agramonte A, Lazear JW. Etiology of yellow fever, preliminary note. *Philadelphia Med J*. 1900;6:790.

25. Kumm HW, Novis O. Mosquito studies on Ilha de Margio, Para, Brazil. *Am J Hyg*. 1938:27:498.

26. Gubler DJ. Dengue. In: TP Monath, ed. *Epidemiology of Arthropod-borne Viral Diseases*. Vol 2. Boca Raton, Fla: CRC Press; 1988.

27. Gubler DJ, Trent DW. Emergence of epidemic dengue/dengue hemorrhagic fever as a public health problem in the Americas. *Infect Agents Dis*. 1993;2383–2393.

28. Hanimon W, Rudnick A, Sather G, Rogers KD, Morse LJ. New hemorrhagic fevers of children in the Philippines and Thailand. *Trans Assoc Am Physicians*. 1960;73:140–155.

29. World Health Organization. *Dengue Haemorrhagic Fever: Diagnosis, Treatment and Control*. Geneva: WHO Press; 1986.

30. Trent DW, Manske CL, Fos GE, Chu MC, Kliks SA, Monath TP. The molecular epidemiology of dengue viruses: Genetic variation and microevolution. In: Kurstak E, Marusyk RG, Murphy FA, Van Regenmortel MH, eds. *Virus Variability, Epidemiology, and Control*. Vol 2. In: *Applied Virology Research*. New York: Plenum Press; 1990.

31. Burke DS, Leake CJ. Japanese encephalitis: In: Monath TP, ed. *The Arboviruses: Epidemiology and Ecology*. Vol 3. Boca Raton, Fla: CRC Press; 1988: 63–92.

32. Fukumi H, Hayashi K, Mifune K, Shichijo A, Matsuo S, Omori N. Ecology of Japanese encephalitis virus in Japan, I: mosquito and pig infection with the virus in relation to human incidence. *Trop Med*. 1975;17:97–110.

33. Update: West Nile Virus activity—Eastern United States, 2000. *MMWR*. 2000;49: 1044–1047.

34. Klingberg MA, Jasinska-Klingberg W, Goldblum N. Certain aspects of the epidemiology and distribution of immunity of West Nile virus in Israel. *Proc 6th Intl Congr Trop Med*. 1959:5:132.

35. Hayes CG. West Nile fever. In: Monath TP, ed. *The Arboviruses: Epidemiology and Ecology*. Vol 5. Boca Raton, Fla: CRC Press; 1989.

36. Meegan JM, Bailey CL. Rift Valley fever. In: Monath TP, ed. *The Arboviruses: Epidemiology and Ecology*. Vol 4. Boca Raton, Fla: CRC Press; 1988:51–76.

37. Turell MJ, Presley SM, Gad AM, et al. Vector competence of Egyptian mosquitoes for Rift Valley fever virus. *Am J Trop Med Hyg*. 1996;54:136–139.

38. Update: Outbreak of Rift Valley fever—Saudi Arabia, August–November, 2000. *MMWR*. 2000;49:982–985.

39. Outbreak of Rift Valley fever—Yemen, August–Ocotber, 2000. *MMWR*. 2000;49:1065–1066.

40. Laughlin LW, Meegan JM, Strausbaugh LJ, Morens DM, Watten RH. Epidemic Rift Valley fever in Egypt: observations on the spectrum of human illness. *Trans R Soc Trop Med Hyg*. 1979;73:630–633.

41. Arthur RR, el-Sharkaway MS, Cope SE, et al. Recurrence of Rift Valley fever in Egypt. *Lancet*. 1993;342:1149–1150.

42. Linthicum KJ, Davies FG, Kairo A, Bailey CL. Rift Valley fever virus: isolations from Diptera collected during an inter-epizootic period in Kenya. *J Hyg (Lond).* 1985;95:197–209.

43. Morris CD. Eastern equine encephalomyelitis. In: Monath TP, ed. *The Arboviruses: Epidemiology and Ecology*. Vol 3. Boca Raton, Fla: CRC Press; 1988.

44. Ising E. Zoological aspects of the epidemiology of EEE in Europe. *Angew Zool*. 1975;62:419–434.

45. Zehner RB, Dean PB, Sudia WD, Calisher CH, Sather GE, Parker RL. Venezuelan equine encephalitis epidemic in Texas, 1971. *Health Surv Rep*. 1974;89:278–282.

46. Larsen JR, Ashley RF. Demonstration of Venezuelan equine encephalomyelitis virus in tissues of *Aedes aegypti. Am J Trop Med Hyg*. 1971;20:754–760.

47. Reeves WC. Epidemiology and control of mosquito-born arboviruses in California. *Cal Mosq Vector Control Assoc*. 1990;XIV:508.

48. Thomas LA. Distribution of the virus of western equine encephalomyelitis in the mosquito vector, *Culex tarsalis. Am J Hyg*. 1963;78:150–165.

49. Saugstad ES, Dalrymple JM, Eldridge BF. Ecology of arboviruses in a Maryland freshwater swamp, I: population dynamics and habitat distribution of potential mosquito vectors. *Am J Epidemiol*. 1972;96:114–116.

50. Reisen WK, Monath TP. Western equine encephalitis: In: Monath TP, ed. *The Arboviruses: Epidemiology and Ecology*. Vol 3. Boca Raton, Fla: CRC Press; 1988.

51. Young DG, Duncan MA. *Guide to the Identification and Geographic Distribution of Lutzomyia Sand Flies in Mexico, the West Indies, Central and South America (Diptera: Psychodidae)*. Gainesville, Fla: Associated Publishers; 1994.

52. Killick-Kendrick R. Phlebotomine vectors of the leishmaniases: a review. *Med Vet Entomol*. 1990;4:1–24.

53. Desjeux P. Leishmaniasis, public health aspects and control. *Clinic. Dermatol.* 1996; 14:417–423.

54. Magill AJ. Epidemiology of the leishmaniases. *Clinic. Dermatol.* 1995; 13:505–523.

55. Kreutzer RD, Grogl M, Neva FA, Fryauff DJ, Magill AJ, Aleman-Munoz MM. Identification and genetic comparison of leishmanial parasites causing viscerotropic and cutaneous disease in soldiers returning from Operation Desert Storm. *Am J Trop Med Hyg*. 1993;49:357–363.

56. Magill AJ, Grogi M, Gasser RA Jr, Sun W, Oster CN. Visceral infection caused by *Leishmania tropica* in veterans of Operation Desert Storm. *N Engl J Med*. 1993;328:1383–1387.

57. Martin S, Gambel J, Jackson J, et al. Leishmaniasis in the United States military. *Mil Med*. 1998;163:801–807.

58. Hertig M. Phlebotomus and Carrion's disease. *Am J Trop Med*. 1942;22(Suppl):1–80.

59. Lane RP. Sandflies. In: Lane RP, Crosskey RW, eds. *Medical Insects and Arachnids*. London: Chapman and Hall; 1993.

60. Greenberg B. *Flies and Diseases*. Vol 1. In: *Ecology, Classification, and Biotic Associations*. Princeton, NJ: Princeton University Press; 1971.

61. World Health Organization. *Control and Surveillance of African Trypanosomiasis: Report of a WHO Expert Committee*. Geneva: WHO; 1998. WHO Technical Report Series No. 881.

62. Lehane MJ. *Biology of Blood-sucking Insects*. London: Harper Collins Academic; 1991.

63. Greenberg B. *Flies and Diseases*. Vol 2. *Ecology, Classification, and Biotic Association*. Princeton University Press, NJ; 1973.

64. Ferris GF. *Contributions Toward a Monograph of the Sucking Lice*. Stanford, Calif: Stanford University; 1935.

65. Zinsser H. *Rats, Lice and History*. New York: Bantam Books; 1967.

66. Fisher I, Morton RS. *Phthirus pubis* infestation. *Br J Vener Dis*. 1970;46:326–329.

67. Kettle DS. *Medical and Veterinary Entomology*. New York: John Wiley and Sons; 1987.

68. Ochs DE, Hnlica VS, Moser DR, Smith JH, Kirchhoff LV. Postmortem diagnosis of authochthonous acute chagasic myocarditis by polymerase chain reaction amplification of a species-specific DNA sequence of *Trypanosoma cruzi. Am J Trop Med Hyg*. 1996;54:526–529.

69. Reisman RE. Insect stings. *N Engl J Med*. 1994;331:523–527.

70. Armed Forces Pest Management Board. *Bee Resource Manual with emphasis on the Africanized Honey Bee*. Washington, DC: AFPMB; 1995. Technical Memorandum No. 34.

71. Centers for Disease Control. Lyme Disease—United States, 1987 and 1988. *MMWR*. 1989;38:668–672.

72. Goodard J. *Ticks and Tickborne Diseases Affecting Military Personnel*. San Antonio, Tex: Brooks Air Force Base; 1989. USAFSAMSR-89-2.

73. Centers for Disease Control and Prevention. Lyme disease—United States, 1999. *MMWR*. 2001;50:181–185.

74. Centers for Disease Control and Prevention. Lyme disease—United States, 1991–1992. *MMWR*. 1993;42:345–348.

75. Burgdorfer W, Barbour AG, Hayes SF, Benach JL, Grunwaldt E, Davis JP. Lyme disease—a tick bome spirochetosis? *Science*. 1982;216:1317–1319.

76. US Department of Defense. Lyme disease: Vector surveillance and control. Washington, DC: Armed Forces Pest Management Board; 1990. Technical Information Memorandum No. 26.

77. Spach DH, Liles WC, Campbell GL, Quick RE, Anderson DE Jr, Fritsche TR. Tick-borne diseases in the United States. *N Engl J Med*. 1993;329:936–947.

78. Horton JM, Blaser MJ. The spectrum of relapsing fever in the Rocky Mountains. *Arch Intern Med*. 1985;145:871–875.

79. Evans ME, Gregory DW, Schaffner W, McGee ZA. Tularemia: A 30-year experience with 88 cases. *Medicine (Baltimore).* 1985;64:251–269.

80. Centers for Disease Control. Lyme disease—United States, 1987 and 1988. *MMWR*. 1990;38:668–672.

81. Centers of Disease Control and Prevention. Summary of notifiable diseases, United States, 1998. *MMWR*. 1999;47:1–92.

82. Anderson BE, Dawson JE, Jones DC, Wilson KH. *Ehrlichia chaffeensis,* a new species associated with human ehrlichiosis. *J Clin Microbiol*. 1991;29:2838–2842.

83. Maeda K, Markowitz N, Hawley RC, Ristic M, McDade JE. Human infection with *Ehrlichia canis*, a leukocyte rickettsia. *N Engl J Med*. 1987;316:853–856.

84. Petersen LR, Sawyer LA, Fishbein DB, et al. An outbreak of ehrlichiosis in members of an Army Reserve unit exposed to ticks. *J Infect Dis*. 1989;159:562–568.

85. Emmons RW. An overview of Colorado tick fever. *Prog Clin Biol Res*. 1985;178:47–52.

86. Kaire GH. Isolation of tick paralysis toxin from *Ixodes holocyclus. Toxicon*. 1966;4:91–97.

87. Smoak BL, McClain B, Brundage JF, et al. An outbreak of spotted fever rickettsiosis in U.S. Army troops deployed to Botswana. *J Emerging Infect Dis*. 1996;2:217–221.

88. Sasa M. Biology of chiggers. *Ann Rev Entomol*. 1961;6:221–244.

89. Philip CB. Tsutsugamushi disease (scrub typhus) in World War II. *J Parasitol*. 1948;34:169–191.

90. Philip CB. Scrub typhus and scrub itch. Coates JB Fr, Hoff EC, Hoff PM, eds. *Communicable Diseases: Arthropodborne Diseases Other than Malaria*. Vol 7. In: *Preventive Medicine in World War II*. Washington, DC: Office of the Surgeon General, Department of the Army; 1964.

91. Lang JD, Chariet LD, Mulla MS. Bibliography (1864–1974) of house dust mites *Dertnatophagoides* spp. (Acarina: Pyroglyphidae), and human allergy. *J Sci Biol*. 1976;2:62–83.

92. Fain A. Le genre Dermatophagoides Bogdanov 1864, son importance dans les allergies respiratoires et cutainees chez l'homme (Psoroptidae: Sarcoptiforines). *Acarologia*. 1967;9:179–225.

93. Wharton GW. House dust mites. *J Med Entomol*. 1976;12:577–621.

94. Murton JJ, Madden JL. Observations on the biology, behavior and ecology of the house dust mite, *Dermatophagoides pteronyssinus* in Tasmania. *J Aust Entomol Soc*. 1977;16:281.

95. Desch C, Nutting WB. *Demodex folliculorum* (Simon) and *D. brevis akbulatova* of man: redistribution and reevaluation. *J Parasitol*. 1972;58:169–177.

96. Grosshans EM, Kremer M, Maleville J. [*Demodex folliculorum* and the histogenesis of granulomatous rosacea.] *Hautarzt*. 1974;25:166–177.