

## PESTS AND PUBLIC HEALTH

Arthropods are probably the most successful of all animals. They are found in every type of habitat and in all regions of the world. They feed on a wide variety of plant and animal materials and have been known as major causes of disease for centuries. Insects are referred to in the Bible as well as in Chinese, Greek, Roman and other historical writings. Insect-transmitted diseases have killed more soldiers than all military actions combined. Fleas, lice and mosquitoes infect humans and domesticated animals directly or indirectly with the organisms of many dangerous diseases. Arthropod stings or bites are occasionally fatal to humans. Their germ-laden saliva or contaminated bodies also are potential sources of disease transmission. Arthropods, especially insects, are vital players in the transmission of certain diseases to humans.

### I. VECTOR-HOST RELATIONSHIPS

In considering transmission of disease-causing organisms, it is important to understand the relationships between the **vector** (the disease-transmitting organism, for example, an insect), the disease **pathogen** (the infecting organism, for example, a virus) and the human or animal **host**. Pathogens that undergo changes in life stages within the vector before being transmitted to a host require the vector. Without the vector, the disease life cycle would be broken and the pathogen would die. Some diseases that require an insect host may require several individual hosts before completing pathogen transmission. In the various phases of the process, the hosts may be different species. Public-health personnel can better design and manage control programs for a particular problem by understanding how a disease is transmitted and the involvement of vectors.

**Myiasis.** Infestation of an animal, including humans, by living maggots deposited on the skin or in a wound by flies is termed “myiasis.” Some forms, such as bot flies, breed solely in this manner. Screwworm infestations of livestock and occasionally humans are examples of this phenomenon. In the U.S., the primary screwworm fly has been eradicated by integrated pest management (IPM) technology, but the less economically important secondary screwworm fly still invades the flesh of wild animals such as deer.

**Mechanical transmission of disease.** Mechanical transmission of disease pathogens occurs when a vector transports organisms, such as bacteria that cause dysentery, on its feet, body hairs and other body surfaces to the host. There is no multiplication or development of the pathogen within the vector’s body. For example, the house fly, *Musca domestica*, is a passive (mechanical) transmitter of bacillary dysentery. Mechanical transmission can be considered more or less accidental.

**Biological transmission of disease.** Disease-causing pathogens need help to move from one host to another. Many ticks and insects are important vectors of biologically transmitted diseases. The arthropod acquires the pathogen from one host. The pathogen then develops in the arthropod’s body and is transmitted to another host. Within the arthropod, the pathogen may or may not multiply. If the pathogen or **parasite** that causes the disease undergoes the sexual portion of its life cycle in a host, that host is the **primary** or **definitive host**, as in the mosquito that harbors malaria. For malaria, a human is the **intermediate host** in which the asexual stages of the parasite are found.

Modes of biological transmission:

- **Obligatory vectors.** Obligatory vectors of disease include those in which the pathogen develops from one life-cycle stage to another. Malaria is an example of a disease that must be taken up by a vector (mosquito) to develop from one stage to another. Otherwise, the malaria parasite would die. Mosquitoes are often obligatory vectors of diseases.
- **Reservoir hosts.** Reservoirs are defined as one or more host species that harbor a disease-causing pathogen over an extended period of time without showing symptoms of the disease. Disease transmission can occur when a vector feeds on a host that carries the pathogen then feeds on another, susceptible host. Birds are an example of reservoirs for mosquito-borne encephalitis, which can affect humans, horses or other **dead-end hosts** — those from which susceptible mosquitoes cannot acquire an infection. In many cases the disease is **endemic**, that is, it exists continually in reservoirs in a geographically defined area. Diseases that cycle in nature to nonreservoir hosts without involving humans are called **zoonoses**. When a disease outbreak occurs in these nonhuman animals, it is called an **epizootic**.
- **Transovarial transmission.** Some vectors pass the disease to their offspring through eggs. When the female is infected with the disease organism, her eggs become infected and the resulting **larvae** are infected with the disease. The subsequent adult stage is then infected and can transmit the pathogen without needing to feed on an infected host. This occurs with several mosquito-borne viruses and with *Rickettsia rickettsia* causing Rocky Mountain spotted fever. In that instance the tick and the mosquito serve both as reservoir and vector.

## II. POISON IRRITATION AND ALLERGY

Many insects, some spiders, scorpions and centipedes have developed poisoning mechanisms to use in self-defense or in paralyzing their prey. Stings and bites may be intensely painful and irritating to humans but seldom cause death. Probably the most dangerous is the bite of the black widow spider, *Latrodectus mactans*. The brown recluse spider, *Loxosceles reclusa*, may inflict a serious bite, often resulting in so much dead tissue that skin-grafting is needed. There have been deaths reported from both spiders. Stings of bees and wasps may be serious or even fatal to people highly allergic to their venoms.

Other insects that cause skin irritations to humans include the puss, io moth and saddleback caterpillars. These larvae have urticating hairs rather like those of the stinging nettle plant. Cantharidin, present in the blood of blister beetles, can cause painful blistering of the skin when the beetle is crushed on the skin surface. These are not insects of direct public-health concern but are often topics of public inquiry.

## III. THE DISEASES TRANSMITTED

### Mosquito-borne

Aside from the irritation and annoyance that mosquitoes inflict on humans and livestock alike, the threat of disease pathogen transmission is always present. Malaria, dengue, yellow fever and encephalitis are just a few major representatives of a long list of human diseases for which mosquitoes serve as vectors. Of these, several forms of encephalitis represent the greatest perennial threat in the U.S. Malaria

and dengue are occasionally imported by travelers, and yellow fever is now rare due to the availability of a highly effective vaccine.

The word “encephalitis” means an inflammation of the brain and possibly the spinal cord (encephalomyelitis). There are several possible causes for this inflammation, but one in particular involves mosquito-transmitted viruses, commonly referred to as arthropod-borne or **arboviruses**.

**St. Louis encephalitis (SLE).** This disease is caused by a virus that has a natural transmission cycle involving several species of wild birds and a few species of mosquitoes (Figure 2.1). The virus is briefly present in the blood of infected birds, and mosquitoes ingest the virus when taking a blood meal. After one to two weeks at summer temperatures, longer at cooler temperatures, the virus multiplies and spreads to the mosquito salivary glands. Then it is inoculated into the next host when the infected mosquito feeds.

Once infected, the mosquito can transmit the virus each time it takes, or attempts to take, a blood meal for the duration of its life. In the vertebrate host, usually a bird, the virus reproduces and must reach a certain concentration in the blood to infect other susceptible mosquito species. In most cases, this level is reached less than a week after the infective bite occurs and lasts for only three to four days, after which the virus in the host blood is reduced below the level required to infect the mosquito. To become infected and later transmit the virus, a mosquito must feed on the infected vertebrate during this three- to four-day period.

In some species of birds the virus level that develops is much higher than in other species. Those with the higher levels are the most important sources of infection for mosquitoes. Also, some mosquito species are especially receptive and can become infected when feeding on blood with relatively low virus levels. These species are the most important in transmitting the virus to the vertebrates. The SLE virus does not multiply in some mosquito species and so is not transmitted by these species even if the mosquito has fed on an infected bird. Also, the virus does not develop to high enough levels in the blood of some dead-end vertebrates to infect even the most susceptible mosquito species that feed on these animals. Humans, nonhuman mammals and some bird species are dead-end hosts for SLE. There is no person-to-person transmission. Vertebrates, birds, humans or other mammals infected with SLE virus do not always exhibit disease symptoms. Birds, even those that develop high levels of virus in their blood, may not exhibit any symptoms.

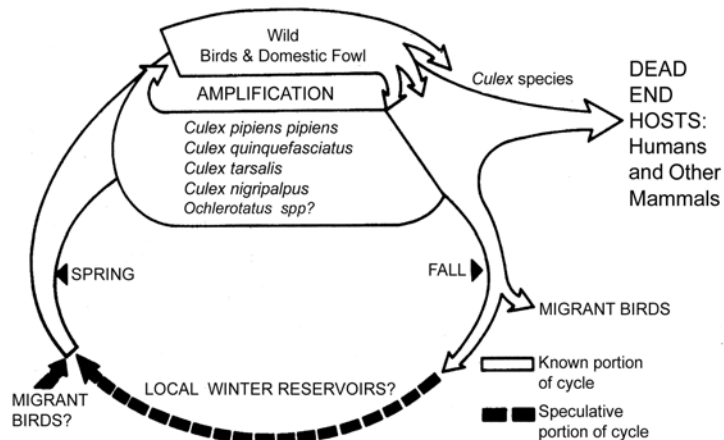


Figure 2.1. Life cycle of St. Louis encephalitis

The reaction of humans infected with SLE virus is quite variable and dependent on age, general health and other poorly understood characteristics. SLE affects the central nervous system. Infected humans may experience no noticeable symptoms, or symptoms may include the onset of fever, nausea and vomiting with severe headaches within five to seven days of infection. Severe symptoms may include temporary or permanent physical or mental disabilities, even death.

In general, the fatality rate is higher and the symptoms more severe in people over 50 years of age. In past epidemics, fatality rates among confirmed cases have ranged from 4 percent to 20 percent of infected individuals, with most deaths occurring in the older age groups. Studies conducted in epidemic areas indicate that for each person who developed symptoms of SLE virus infection (ranging from mild to severe), 80 to 800 infected people did not develop disease symptoms. The older age groups consistently exhibited the highest number and most severe symptoms of those infected.

In the eastern and midwestern U.S., epidemics of SLE have occurred in relatively densely populated urban and suburban areas where suitable bird and mosquito species live in close association with humans. For example, *Culex pipiens*, the northern house mosquito, is thought to be a primary vector of SLE in areas where there are many water-filled artificial containers that provide ideal breeding sites for mosquitoes amidst populations of birds such as sparrows and starlings that live and nest in or near human residences. Water-holding structures commonly associated with humans (storm-sewer catch basins, cesspools, sewage lagoons, open septic tanks, polluted drainage ditches and water runoff impoundments, failed septic systems, drain fields and effluent from sewage disposal plants) also can produce huge numbers of these mosquitoes during the warm months of the year. Rural areas may be highly susceptible to transmission when pastures and other extensive, temporary, freshwater breeding sites produce large populations of avian-feeding mosquitoes, for example, *Culex nigripalpus*.



Figure 2.2. Distribution map of St. Louis encephalitis in the U.S.

The probability of SLE transmission is in direct proportion to the number of SLE-virus-infected birds and the abundance of *Culex* mosquitoes. Mosquito agencies monitor virus activity by sampling the blood of wild birds or, more commonly, sentinel birds caged in the natural habitat. SLE outbreaks are most likely to occur from midsummer to early fall, with case detection continuing until cold weather reduces mosquito abundance. Outbreaks may be more closely related to water accumulation patterns than to total rainfall (for example, drought conditions cause polluted streams to pool and produce *Culex* mosquitoes). SLE occurs throughout the U.S. (Figure 2.2). More than 200 deaths were attributed to SLE in St. Louis alone in 1933. The number of reported cases ranged from a handful to more than 1,800 annually in the two decades following 1970.

In temperate zones, *Cx. pipiens* females overwinter in locations that are protected from weather extremes, such as outbuildings, attics, storm sewers and rodent burrows. All other stages are killed when exposed to freezing temperatures. After overwintering, females lay eggs in rafts of 40 to 500 on the surface of organically polluted water. At summer temperatures, eggs hatch in one to two days, and adult development is complete in eight to 10 days. In the cooler temperatures of spring and fall, this development might require two or more weeks. Mosquito breeding occurs throughout the warm months of the year, and populations typically increase throughout the summer, peaking in late August. Although this species feeds primarily on birds, there is some evidence that it may change its feeding habits in mid-August to September and feed on other animals, including humans. This, and favorable meteorological conditions, may explain in part why human outbreaks of SLE normally occur in late summer.

There is evidence that the virus can remain viable in overwintering *Cx. pipiens* adults and, presumably, be transmitted when they feed on susceptible vertebrates in the spring. Whether this is the primary overwintering mechanism is not yet known.

**Eastern equine encephalitis (EEE).** Eastern equine encephalitis is a viral infection maintained in nature by a bird-mosquito-bird cycle similar to that of SLE. It is distributed along the coastal states of the Gulf of Mexico and Atlantic seaboard and occasionally occurs in the Midwest (Figure 2.3).

Virus activity is confirmed by encephalitis monitoring programs conducted by health departments and mosquito-control districts. These agencies periodically collect blood samples from wild birds or sentinel flocks to detect antibodies produced after infection by the EEE virus. High antibody levels in young birds indicate recent circulation of the virus. Antibodies in older birds might be the result of infection the previous year or earlier. Periodic outbreaks among nonvaccinated equines also provide evidence of local EEE viral activity. Horses (equines) are involved as dead-end hosts in the cycle, along with humans. Vaccines are available for horses, and annual booster shots are recommended. There are no vaccines available for human use. EEE is more prevalent in horses and susceptible domesticated birds such as emus than in humans, often with fatal outcomes. Deaths due to EEE viral infections have been reported in red-winged blackbirds, house sparrows, whooping cranes and pheasants.



Figure 2.3. Distribution map of eastern equine encephalitis in the U.S.

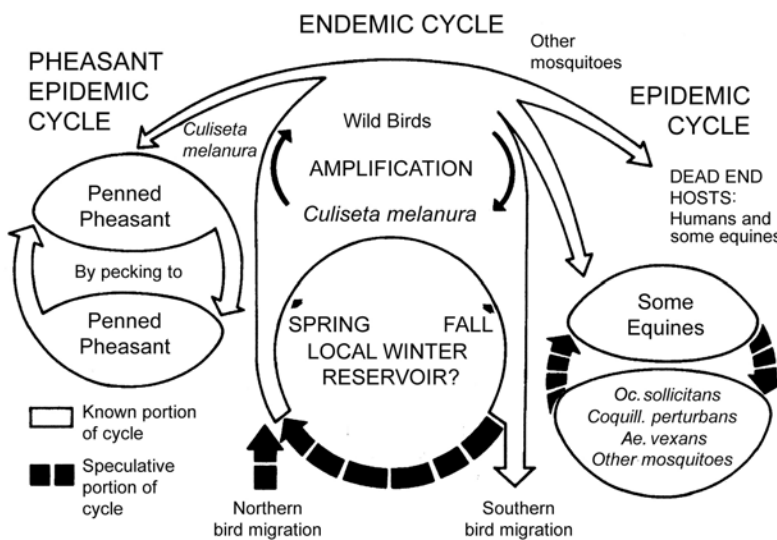


Figure 2.4. Life cycle of eastern equine encephalitis

Eastern equine encephalitis can infect people of any age, but young children and infants are particularly vulnerable. Seldom are more than 20 or 30 human cases reported nationally in any given year, although localized groupings of human cases do occur. Mortality rates in human infections can exceed 50 percent. In fatal cases, death usually occurs within 48 to 72 hours after the onset of illness. Children and infants who survive frequently are afflicted with varying degrees of mental retardation and residual paralysis.

There are a number of mosquito species, especially *Culiseta melanura*, capable of maintaining and transmitting EEE to birds in nature. Because *Cs. melanura* is relatively uncommon and seldom bites humans, other mosquitoes such as *Cs. inornata*, *Cx. quinquefasciatus*, *Aedes albopictus*, *Ae. vexans*, *Ochlerotatus sollicitans*, *Oc. infirmatus*, *Oc. atlanticus* and *Coquillettidia perturbans* are important in transmission from birds to humans and/or horses. Factors favoring infections in humans are the buildup of virus in wild-bird populations and a high density of adult mosquito vectors.

**Western equine encephalitis (WEE).** Western equine encephalitis is another common arboviral disease in the U.S. and is found mainly in the states west of the Mississippi River, Wisconsin and Illinois (Figure 2.5). Major, widespread outbreaks of WEE have occurred in the western part of the U.S. and

Canada. This disease occurs primarily in rural locations and has a natural transmission cycle that involves both mosquitoes and wild birds. Mortality rates generally range from 1 percent to 5 percent in humans but are considerably higher in horses. *Culex tarsalis* and *Oc. melanimon* are the most important vectors throughout the western U.S., and birds are the major host. East of the Mississippi River, *Cx. quinquefasciatus* is the suspected vector. As with EEE, there is a vaccine for horses, but not humans.

Western equine encephalitis can be a severe disease in humans and horses, but both humans and horses are dead-end hosts and are not involved in further transmission of the virus. As with SLE, individuals infected with WEE exhibit a wide range of responses to the infection, ranging from minor, transient symptoms to severe central-nervous-system disease. Infections are more severe in children than in adults. Initial symptoms of WEE may include fever, headache, vomiting and a stiff neck, then may progress to confusion, disorientation, stupor, coma and other evidence of severe central-nervous-system infection. Adults recover completely from this disease, although neurological problems may persist for some time. In children, however, about half of those infected suffer permanent neurological effects, including progressive mental retardation and varying degrees of physical and mental dysfunction.

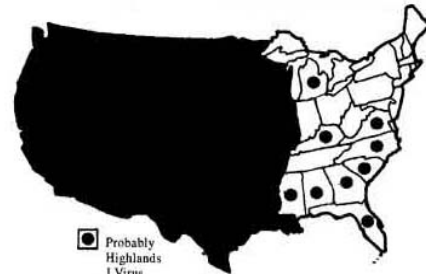


Figure 2.5. Distribution map of western equine encephalitis in the U.S.

Infected wild birds infect the mosquitoes that feed on them, but the WEE virus is present in the infected bird's blood for only three to four days in the amounts needed to infect feeding mosquitoes. Birds infected with WEE normally do not show any symptoms. Although several mosquito species are able to transmit this virus, *Cx. tarsalis* has been the major vector in past outbreaks of this disease. This species usually breeds in both permanent and temporary groundwater with high organic content, such as sewage effluent. In many areas of the West, the species is most numerous where agriculture irrigation practices result in water accumulation. This mosquito feeds readily on both birds and mammals, so it serves as the major vector in the natural transmission cycle as well as in transmitting WEE virus to humans and horses. As with SLE, it is not presently known how WEE virus is maintained in areas of the U.S. and Canada where adult mosquitoes are not active during the cold months of the year.

**California (CE), LaCrosse (LAC) and Jamestown Canyon (JC) encephalitis.** These viruses of the California serogroup viruses are reported sporadically, ranging from 30 to 160 cases annually. Most cases of LAC are reported from midwestern states — namely Ohio, Indiana and Wisconsin — in children under 16 years of age. The disease is less severe than SLE and EEE, with a mortality rate of about 0.4 percent, but infected children often experience learning disabilities or personality changes. The natural cycles differ from SLE, EEE and WEE in that natural hosts are small and medium-sized animals such as rabbits, hares, chipmunks and squirrels rather than birds. The vectors are mostly woodland mosquitoes, and most of the human cases have occurred in people who lived or worked in or near wooded areas. However, LAC virus also seems to be associated with container-breeding mosquitoes, such as *Oc. triseriatus*, that readily maintain it in a transovarial transmission cycle. These viruses have been isolated from a number of species of woodland mosquitoes, particularly *Oc. triseriatus*, *Oc. canadensis*, *Oc. melanimon*, *Oc. trivittatus*, *Oc. stimulans* and *Oc. atlanticus*.

**Venezuelan equine encephalitis (VEE).** Venezuelan equine encephalitis is a virus that was first recognized in South America. A number of subtypes exist, including the endemic Everglades virus (EV) in Florida, which is of low virulence and transmitted from wild rodents by *Culex* mosquitoes. Both humans and equines are affected by VEE, but unlike the other arboviral encephalitides, victims develop

high viremia, enabling mosquitoes to secure infective quantities of virus from either host and to subsequently transmit the disease to humans or horses without the involvement of birds. The disease is mild in humans, producing flulike symptoms, but is often fatal to horses. In the 1971 epidemic, more than 1,500 horses died of VEE in Texas, but no deaths were reported among the more than 100 suspected human cases. Equine vaccination, when combined with quarantine and appropriate mosquito-control measures, provides seasonal protection, but no VEE vaccine is available for humans.

No VEE activity has been reported in the U.S. since December 1971, although 500,000 mosquitoes, 9,000 wildlife blood samples and 1,500 equine blood samples were tested by various agencies in 1972. The VEE virus failed to become established in 1971 in the U.S., perhaps because of massive, timely and targeted mosquito-control activities. In fact, this was the largest coordinated aerial application for mosquito control ever recorded in this country. But the threat of reintroduction remains because the virus resides in neighboring countries to the south, particularly Mexico.

On the basis of virus isolation studies conducted during the 1971 VEE epidemic, numbers of species of mosquitoes have become suspected vectors. *Psorophora columbiae*, *Oc. sollicitans* and *Ps. discolor* were incriminated, and it is suspected that other species of *Aedes* (*Ae. albopictus*, for example) and *Ochlerotatus*, and certain species of *Culex*, *Anopheles* and *Coquillettidia* could also transmit VEE virus. Mechanical transmission by biting insects other than mosquitoes and direct transmission by contact (bridle bits) or aerosols (sneezing) are also considered possible means of infection during the height of an epidemic.

**West Nile virus.** West Nile virus is very similar to SLE in many respects but was unknown in the Americas until 1999, when it appeared in the northeastern U.S. More than 60 clinical human cases occurred in the New York City area in late summer, while subsequent serosurveys suggested that there actually had been several thousand subclinical human infections following a widespread die-off of American crows in the region. The virus, previously found in parts of Eurasia and Africa, is found in birds and other vertebrates and is thought to be transmitted to humans primarily by *Culex* mosquitoes. In 2001, the virus spread throughout the eastern U.S. and crossed the Mississippi River, causing widespread fatality among horses and eight deaths among the 57 recorded human cases. For every recognized human case, an estimated 100 to 200 infections went undiagnosed because of mild or nonexistent symptoms.

**Dengue.** Dengue is a viral disease, commonly called “breakbone fever.” It is often characterized by severe headache, pain behind the eyes, high fever, backache, pain in the joints and a severe rash, with convalescence that may require several weeks. These symptoms generally occur five to six days after an infected mosquito has bitten a susceptible person. In uncomplicated cases, death rarely occurs. Four strains of dengue virus are recognized, each of which produces lifelong immunity against the infecting virus. Exposure to infection by a second strain of dengue virus in an already immune individual may result in a more severe form of dengue known as dengue hemorrhagic fever (DHF) with accompanying dengue shock syndrome (DSS). Increased incidence of DHF has been experienced in the Western Hemisphere in the last 20 years, with outbreaks occurring in the Caribbean region.

Dengue epidemics were common in the 1800s and early 1900s in the U.S. Before 1980, the last major epidemic of dengue in the continental U.S. occurred in Louisiana in 1945 with 62 confirmed cases, but authorities estimated that there were probably several hundred unapparent and/or unreported cases. In 1980, Texas recorded 23 locally acquired cases of dengue, and new cases occurred sporadically until 1999, when 18 cases were reported. Although dengue is not endemic now in most of the U.S., potential vectors are present in the southern U.S., and the virus is commonly imported in people entering the country from endemic areas of the tropics. Conditions for dengue transmission are present, but it is

unlikely that this would occur at the levels observed at the turn of the last century, when hundreds or thousands of cases are estimated to have occurred in some major cities.

Dengue is transmitted in the U.S. by *Ae. aegypti* in a mosquito-human-mosquito cycle. Other potential vectors are *Ae. albopictus* and *Oc. japonicus*. These **peridomestic** species are found in close association with humans, breeding in natural and artificial water-holding containers around dwellings. After biting an infected person, the female mosquito requires eight to 10 days for viral development before it is capable of transmitting the virus to humans. Once infected, the mosquito remains infective for the remainder of its life. Presently, there is no vaccine available to prevent dengue infection.

**Yellow fever (YF).** Yellow fever is caused by a virus closely related to the dengue virus. In fact, YF infections produce denguelike symptoms in humans, but the effects of YF are normally much more severe. The fatality rate may reach 50 percent or more in epidemics. The virus is presently found in Africa and South America. It is absent from the continental U.S., where the last epidemic of YF occurred in New Orleans in 1905.

As with dengue, the YF virus is transmitted in urban areas by *Ae. aegypti*, with *Ae. albopictus* and *Oc. japonicus* as potential vectors as well. Unlike dengue, however, the probability of YF virus reaching the U.S. is low. This may be due, in part, to the fact that YF, along with cholera, smallpox and plague, is quarantinable. This means that the Centers for Disease Control and Prevention in Atlanta, Ga., are continually monitoring outbreaks of YF in the Western hemisphere. Yellow fever vaccines are used to prevent disease in humans traveling to YF-endemic areas. They are also required of people moving from YF-endemic areas to YF-free areas where *Ae. aegypti* is present.

**Malaria.** On a worldwide basis, malaria remains the most important human disease transmitted by mosquitoes. It is estimated that there are 400 million human cases of malaria in the world (mostly in Asia and Africa), with more than 2 million human deaths annually. Most of those who die are children under 10 years of age. In Africa, more than one in every 20 children die from malaria. It is believed that malaria was introduced into North America during colonial days. Tens of thousands of cases occurred in the U.S. before the 1930s, but there are no reliable statistics available for the period. It is estimated that as many as a million cases may have occurred in the U.S. during the Civil War. In the 1930s, approximately 100,000 cases were reported annually; however, in the early 1940s, the number of cases reduced dramatically due to the work of public-health agencies using DDT during and after World War II and the Tennessee Valley Authority (TVA) source-reduction program.

Since the late 1950s, a few hundred to a few thousand new cases have been recorded annually, almost entirely attributable to travelers and military returnees from malarious areas. These cases were contracted outside the U.S., but symptoms appeared following re-entry into the country. Until recently, rarely has secondary infection been contracted in the U.S. However, there are periodically documented cases of human malaria being transmitted in the U.S. from infected hosts to uninfected hosts by indigenous mosquitoes. Antimalarial prophylaxis is effective, and most nonresistant forms of malaria are responsive to treatment.

Human malaria is caused by any of four species of *Plasmodium*, a protozoan parasite that causes fever, chills, sweating and headache. If not treated, it may cause shock, renal failure, acute encephalitis, coma and death. The disease is transmitted by several species of *Anopheles* mosquitoes. In the eastern U.S., some members of the *An. quadrimaculatus* species complex are important vectors. Another species, *An. crucians*, is a vector also but probably to a lesser degree. In the West, the major vectors are *An. hermsi* and *An. freeborni*. These species are widespread and are most abundant from April to September.



**Dog heartworm.** Dog heartworm (*Dirofilaria immitis*, a filarial worm) is a serious disease for all breeds of dogs in temperate and tropical climates. Infection rates in some states have been reported to be as high as 80 percent in dogs over 2.5 years old, and almost 100 percent in dogs over 5 years old. The infection rate in cats is sufficient to be a cause of veterinary concern. Humans can also become infected, but the worms are unable to develop or mature because humans are not their natural hosts. Wild canids, such as foxes and coyotes, probably serve as **enzootic reservoirs**. Several genera of mosquitoes can transmit the parasite to dogs (*Aedes*, *Anopheles*, *Culex*, *Ochlerotatus* and *Psorophora*, for example). In nature, *Cx. quinquefasciatus* is considered to be the most important vector. Other incriminated vectors of dog heartworm include *Ae. albopictus*, *Ae. vexans*, *Oc. sierrensis*, *Oc. taeniorhynchus* and *Cx. salinarius*.

The life cycle of the dog heartworm (Figure 2.6) involves two hosts: mosquitoes and dogs. Mosquitoes ingest the immature worms called **microfilariae** while taking blood from dogs. These immature worms develop through three larval stages in the mosquito in nine to 14 days. After they have developed into infective or third-stage larvae, the microfilariae are transmitted via the mosquito's mouthparts to a dog when the mosquito feeds again. The larvae grow and migrate through the host's subcutaneous tissues and large blood vessels and eventually enter the right ventricle of the heart — thus the name “dog heartworm.” In the heart, the larvae grow into adult worms measuring about 11 inches for the females and 6 inches for the male. The adult female lays no eggs but produces microfilariae that circulate in the blood, completing the cycle.

In areas where dog-heartworm infection is prevalent, dog owners should provide mosquito-proof sleeping quarters for their animals. Additionally, the owners should seek the assistance of a veterinarian to administer a program of chemical prophylaxis. Highly effective preventative medication is readily available and easily administered.

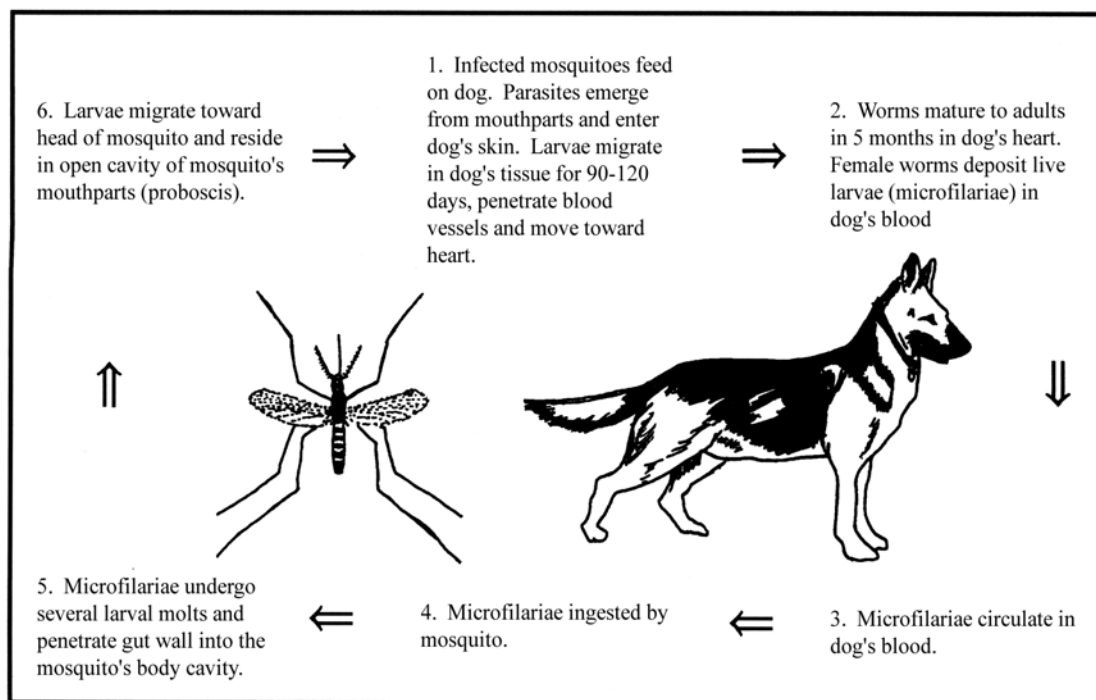


Figure 2.6. Life cycle of the dog heartworm

**Filariasis.** Although not currently present in the U.S., filariasis is endemic in some islands of the Caribbean and is undoubtedly imported occasionally from endemic areas in the Western Hemisphere, Africa and Asia. Transmitted among humans by *Mansonia*, *Culex*, *Anopheles*, *Ochlerotatus* and *Aedes* species, the filarial nematode parasites (*Wuchereria bancrofti* and *Brugia malayi*) live in the human lymphatic system. They cause extreme enlargement of soft tissues, called elephantiasis. Mosquitoes pick up the microfilariae by feeding on infective humans. After developing through the larval stages, the infective (third-stage) larvae leave the mosquito and enter the human host as the female mosquito penetrates the skin in search of blood. Filariasis represents a minimal threat to subtropical areas in the U.S.

**Acquired immune deficiency syndrome (AIDS).** When the AIDS virus was initially discovered in humans, one of the first questions asked of public-health officials was: “Is the virus that causes AIDS transmitted by bloodsucking insects?” There have been exhaustive laboratory tests conducted by the Centers for Disease Control and Prevention and other similar agencies around the world using a wide range of blood-feeding insects and arthropods in attempting to answer this question. Currently, there is no scientifically based, credible evidence to suggest or imply that mosquitoes or other biting arthropods can transmit the virus to humans. Is that enough evidence to say that it is impossible for insects to transmit the virus to humans, either biologically or mechanically (for example, infected blood on the mouthparts)? While that question may not have been fully answered to the satisfaction of some members of the general public, it is safe to say that the involvement of blood-feeding insects and other arthropods is biologically insignificant, if, in fact, it occurs at all.

#### **Tick-borne**

From a public-health standpoint, ticks are important as vectors of disease to humans. Many species are quite resistant to environmental stresses and may live for many years. They have a wide range of vertebrate hosts. Large urban populations in the U.S. are becoming increasingly at-risk for tick-borne diseases. Humans are increasingly closer to potentially pathogen-infected ticks because of reversion of farmland to scrub vegetation, continuous incorporation of rural land into urban population centers, frequent travel to rural areas for recreation and vacations, and increasing wildlife populations (hosts for tick-borne disease) in both rural and urban areas.

Ticks are highly successful **ectoparasites** and are efficient at transmitting several diseases, in part because they are persistent bloodsuckers. They attach and feed for long periods of time, which allows the pathogen to enter the host and extends the time and distance that the ticks are transported by the host. In addition, many species have a wide host range. They have a high reproduction potential and lay several thousand eggs.

**Colorado tick fever (CTF).** Transmitted in areas above 1,525 meters in the mountainous regions of the West, CTF occurs annually — mostly in Colorado. The viral, denguelike disease has an incubation period of three to six days, followed by fever, chills, severe headaches, sore throat, nausea, etc. In the internal organs of humans, CTF virus can develop into encephalitis and hepatitis in rare cases. Usually a mild illness, it sometimes takes several weeks for complete recovery. Mortality is rare, occurring in less than 0.2 percent of the cases.

Victims are usually involved in recreational activities in the mountain forests, rivers and valleys. The primary vector is the Rocky Mountain wood tick, *Dermacentor andersoni*. The primary reservoirs are mountain squirrels, chipmunks and mice. Adult and nymphal tick activity from March to November coincides with human CTF cases.

**Rocky Mountain spotted fever (RMSF).** The American dog tick (*D. variabilis*) and the Rocky Mountain wood tick (*D. andersoni*), found in the Rocky Mountain states, Nevada, eastern California, Oregon and Washington, transmit the rickettsial pathogens that cause RMSF. The disease is also called tick fever, spotted fever, tick typhus, tick-borne typhus fever, black fever and several other common names. The lone star tick, *Amblyomma americanum*, can transmit RMSF, but it is not as important a vector as the two species of *Dermacentor*. At least four to six hours elapse after the tick begins feeding before pathogen transmission begins. If ticks are removed during this noninfective period, infection will not occur unless abraded skin is contaminated with crushed tick tissues or feces. The causal organism is *Rickettsia rickettsia*, which is found most often in the mid-Atlantic and south-central states but is endemic throughout the continental U.S. Ticks are the primary reservoirs, maintaining the disease through passage from stage to stage and transovarial transmission.

Symptoms of RMSF appear three to 12 days after initial tick contact. There is a sudden onset of symptoms, which includes fever, chills, severe headaches and general aches and pains. A reddish-purple-black rash may occur on the bottoms of the feet, ankles, palms of the hands, or characteristically on the wrists or forearms a few days after infection. The rash may spread to the trunk, neck and face. Aching in the lower back and headaches around the head and eyes will often occur. Victims feel very tired and can run fevers of 104EF to 106EF. Less-obvious symptoms may not be noticed. Laboratory blood tests can assist diagnosis in questionable cases. Early treatment using antibiotics is most successful. If left untreated, the patient may be highly agitated, develop insomnia, become delirious or go into a coma. Most fatalities can be attributed to a delay in seeking medical attention.

**Lyme disease.** The most prevalent vector-borne disease of humans in the U.S. is Lyme disease (about 15,000 cases annually), named after Lyme, Conn., where cases were first reported in 1976. Caused by a spirochete-type bacterium, *Borrelia burgdorferi*, the disease is transmitted primarily by the blacklegged tick (*Ixodes scapularis*, previously known as *I. damini*) in the East and *Ixodes pacificus* in the West. Transmission usually occurs in the nymphal stage, which is most active in late spring and summer. Rodents are the main reservoir from which larval ticks acquire the spirochete, which is passed **transstadially** to nymphs and adults. White-tailed deer are the primary hosts of the adult ticks in nature but are incompetent reservoirs of the parasite. A vaccine is available for people in high-risk situations.

This serious disease is expected to increase. There are no easy or effective area control measures that state or federal agencies can perform. Children, who are at highest risk, encounter infected ticks in camps and parks, on hikes, or at play in areas where deer and mice abound, and are less diligent about searching for ticks on themselves than are adults. Also at increased risk of exposure are adults whose occupations place them in tick habitat (farmers; outdoor maintenance workers; and park, forestry and military personnel), and members of the general public who hike, camp, hunt, participate in outdoor recreational sports or live in areas of preferred tick and host habitat.

Symptoms vary and may mimic other diseases; many cases go undiagnosed. The first indication of a potential infection may be the discovery of an attached tick. Disease transmission has been reported not to occur for an estimated 10 to 12 hours or even longer after feeding begins. If the tick is located and removed within that time, no infection will occur. A common early symptom is a red, expanding, ringlike lesion developing at the site of a tick bite within two to 32 days. The clear center often is the site of the bite. The rash may burn or itch. Technically, this rash is called erythema cronicum migrans (ECM), which is not uncommon to find at multiple sites. It disappears within three weeks but can recur. Other skin symptoms may be hives, redness of cheeks under eyes and swelling of eyelids with reddening of the whites of the eyes. Flulike symptoms may accompany the skin symptoms — for example, high fever, headache, stiff neck, fatigue, sore throat and swollen glands.

A second set of symptoms occurs in untreated patients four to six weeks after transmission. More than half of untreated victims experience arthritis intermittently or chronically in the large joints (primarily knees, elbows and wrists). A few (10 percent to 27 percent) experience neurological effects, including severe headache, stiff neck, facial paralysis, weakness and pain of the chest or extremities. These symptoms may persist for weeks. In 6 percent to 10 percent of the cases, heart block may occur. Early antibiotic therapy reduces the risk of subsequent severe arthritic, neurologic or cardiac complications developing days to years later. Dogs can also acquire Lyme disease when they forage in tick habitat. In fact, diagnosis of the disease in dogs in an area is a harbinger of human cases to follow. Symptoms in dogs include sluggishness and lameness.

Endemic areas in the U.S. include the East Coast from Massachusetts to Virginia, Pennsylvania, Wisconsin, Illinois, the upper peninsula of Michigan, northern California, Oregon and southern Washington. The distribution of Lyme disease in the U.S. is strongly linked to the distribution of the vectors.

**Ehrlichiosis.** Human monocytic ehrlichiosis (HME, caused by *Ehrlichia chaffeensis*) and human granulocytic ehrlichiosis (HGE, of unknown origin) are the two forms of ehrlichiosis found in the U.S. Ehrlichiosis occurs in the Southeast, south-central and mid-Atlantic regions. These infections, first recognized in 1990, range from mild illness to life-threatening or fatal disease. Ehrlichiosis is similar to RMSF with high fever, headache and nausea but has a lower incidence of rash. Severe and fatal complications may ensue, including kidney and respiratory failure.

HME transmission occurs primarily from May through July, apparently associated with the American dog tick, *D. variabilis*; the lone star tick, *A. americanum*; and the blacklegged tick, *I. scapularis*. White-tailed deer are natural reservoir hosts for *E. chaffeensis* and may serve as a source of infection for *A. americanum* in all life stages. *Ixodes scapularis* is a suspected vector of HGE.

**Tick-borne relapsing fever.** Tick-borne relapsing fever is caused by a spirochete in the genus *Borrelia*. Some specialists view it as a tick-adapted strain of the louse-borne relapsing fever spirochete *B. recurrentis*. It is found in the western U.S. and Canada, generally limited to remote, undisturbed areas. Victims suffer high fever, throbbing headache, nausea and digestive upset a few days after the appearance of an itchy scab at the site of the bite. After the illness subsides, untreated individuals may experience up to 10 relapses with similar symptoms.

The vector in mountainous areas above 900 meters is *Ornithodoros hermsi*, and at lower altitudes, *O. turicata*. The infection is highly localized and often associated with rustic mountain cabins or remote caves where, in the absence of humans, ticks are active and all stages transmit the disease to small mammals, mostly rodents. The disease persists in the ticks, and the rodents serve as a natural source of infection.

**Babesiosis.** Human babesiosis is an uncommon infection caused by several species of *Babesia*, blood parasites known as piroplasms. In humans, *Babesia* infection follows a clinical course similar to malaria but without periodicity and usually self-limiting. It can become life-threatening in elderly people. The pathogen, *B. microti*, is transmitted during the summer months by nymphs of the blacklegged tick, *I. scapularis*, in the Northeast and midwestern U.S. Other species may be involved. The reservoirs identified to date are the white-footed mouse and meadow vole.

**Tick paralysis.** Tick paralysis is caused by the toxins injected as ticks feed. Several ticks are responsible for this malady in the U.S., including *D. andersoni*, *D. variabilis*, *A. americanum*, *A.*

*maculatum* and *I. scapularis*. Generally, only female ticks cause paralysis and they must be attached for several (four to seven) days before they begin secreting the toxins in their saliva. In humans, symptoms usually begin in the legs with muscle weakness and progress to the trunk, back and chest, where they can lead to respiratory failure. Ultimately, the victim may be unable to sit up or move arms or legs, and chewing and swallowing may become difficult. The condition progresses rapidly, and death may occur within 24 to 48 hours after the onset of symptoms. Symptoms usually resolve themselves within hours or days after removal of the tick, which is often at the nape of the neck.

**Tularemia.** Tularemia, or rabbit fever, is a **zoonosis** caused by the bacterium *Francisella tularensis*. The disease is characterized by sudden onset of fever, chills, headache, sore muscles and fatigue, and the severity of illness is variable. Modes of transmission to humans are varied: inoculation of skin or mucous tissues while skinning or performing necropsies of animals; exposure to contaminated particles; bites of ticks, deer flies and mosquitoes. The organism has been isolated from at least 13 species of ticks (one *Amblyomma*, five *Dermacentor*, two *Haemaphysalis* and five *Ixodes*), but the three major vectors appear to be *A. americanum*, *D. andersoni* and *D. variabilis*. Cases occur year-round but are concentrated during the fall and winter hunting seasons and in summer when people are outdoors and ticks or other vectors are abundant.

#### Louse-borne

**Epidemic typhus and louse-borne relapsing fever.** Historically, the disease typhus, transmitted by body lice (*Pediculus humanus*), was common where people were confined together and could not wash or delouse their clothing. This disease became epidemic within confined populations such as cities under siege or armies limited to trenches or on the move and unable to delouse their clothes. Typhus is a fatal disease and was so pervasive that it often determined who was victorious and who was defeated in wartime. Widespread louse epidemics ceased being a problem when DDT dust became available during World War II. Although body lice became resistant to DDT when it was intensively and repeatedly used, other synthetic pesticides were found to work as well. Neither epidemic typhus nor louse-borne relapsing fever are found in the U.S., so these diseases are not addressed further in this manual.

#### Flea-borne

**Plague.** Ground squirrels, wood rats, sage brush voles, meadow mice, deer mice, rabbits, hares and prairie dogs can support infestations of fleas infected with sylvatic (zoonotic) plague, which can cause **bubonic** plague in humans by infective bite. Rodent nests in and near buildings occupied by humans or pets are potential sources of these vectors. The reservoir of **murine** plague is domestic rats. The primary vector is the oriental rat flea, *Xenopsylla cheopis*, which transmits the plague bacteria, *Yersinia pestis*, from rodent to rodent through its bite, and it will bite humans. The bacteria multiply in the flea's crop and stomach where they may form an obstruction. When the flea attempts to feed, the blood cannot pass beyond the blockage and becomes contaminated with the plague bacilli. The contaminated blood is then regurgitated into the wound caused by the flea bite.

Bubonic plague is endemic throughout the western U.S. Once contracted, direct spread of the infection through bubonic human patients with secondary lung involvement is apt to lead to manifestations of primary pneumonic plague. The Great Plague of London killed half the city's population. The Black Death of Europe in the 14<sup>th</sup> century lasted 50 years and killed 25 million people. In the first quarter of the 20<sup>th</sup> century, an estimated 11 million people died in Asia from plague. Although no major urban outbreak of plague has occurred since 1924, this is not a disease of the past. In the bubonic form of plague, symptoms include the sudden onset of fever with painful swelling of the lymph nodes. If the infection spreads to the lungs (pneumonic plague), it produces pneumonia that is highly contagious

and often fatal. As suburbia expands into undeveloped areas, wild rodents can transmit the disease to urban rats. An outbreak of urban plague could occur in the U.S.

**Murine typhus.** Murine typhus occurs in California and in southeastern and Gulf Coast states. It is a relatively mild disease in humans. As with plague, murine typhus is transmitted from rodent to rodent and rodent to humans by the oriental rat flea. In this case, however, the disease organism (*Rickettsia typhi*) enters the bloodstream when feces of infected fleas are scratched into a flea-bite wound. Symptoms may include fever, severe headache and rash.

#### Snail-borne

**Swimmers' itch.** Adult blood flukes, called **schistosomes**, live as parasites in the tissues of mammals and birds, usually those associated with ponds, lakes and streams. They produce tiny (0.03-inch), free-swimming larvae called **cercariae**, which are an intermediate stage in the life cycle of blood flukes. Swimmers' itch is caused by the penetration of these cercariae into the skin of a human. When cercariae accidentally come in contact with an unsuitable host, such as humans, they may penetrate into, but not through, the skin. As the cercariae enter the skin a temporary, prickly, itching sensation occurs, sometimes followed by a general inflammation of the area affected. This condition usually subsides quickly, leaving the victim with minute red spots — and very little itching. The cercariae die within 24 hours after penetration, but the body's allergic reaction to these organisms may cause severe itching at each point of entrance. Several hours after penetration, intense itching occurs, together with the development of raised red spots similar to pimples. These may become larger and be accompanied by general swelling, especially when aggravated by unrestrained scratching. The victim is usually most uncomfortable during the second, third and fourth days following exposure. A week after contact all symptoms have normally disappeared except in extreme cases, but small red spots may persist for some time. The condition is associated with several different species of snail (*Stagnicola*, *Lymnaea*, *Stagnales*, etc.).

#### Rodent-borne

**Hantavirus pulmonary syndrome (HPS).** A relatively recent entry (1993) for public-health concerns, HPS is increasingly evident throughout the U.S. The hantavirus fatality rate in humans ranges from 10 percent to 55 percent. Symptoms appear one to three weeks after exposure to the virus, occasionally up to six weeks. Fever and aching large muscles occur in all cases; abdominal pain is present in about half of the cases; and coughing, shortness of breath, dizziness and chills also may occur. Shortness of breath is a symptom that appears later due to the filling of the lungs with fluid. Pneumonia is the most frequent misdiagnosis of HPS. Hemorrhagic fever is common. There is no cure for HPS, but early detection and medical attention increase the probability of survival and recovery.

HPS is a respiratory illness associated with breathing air contaminated with rodent urine and feces contaminated with hantavirus particles. One hantavirus species, Sin Nombre virus, has been associated with the largest proportion of HPS cases. The deer mouse is the primary reservoir. But several species of hantavirus exist and are associated with other rodent species. The virus is passed in the feces, urine and saliva of infected rodents. Being bitten by an infected rodent is also a possible route of transmission. Biting, along with grooming, probably helps perpetuate the virus in deer mice. Person-to-person transmission has not been observed, and health-care workers who have cared for HPS patients have not become infected. There do not appear to be differences in susceptibility due to age or sex. Exposure to susceptible rodents or tightly enclosed areas of infestation increases the chance of contracting the disease. Spring and summer are the seasons when most cases occur, due to increased outdoor exposure.

**Dermatitis.** The house mouse easily adapts to life with people. It thrives in a wide range of climatic conditions in a great variety of habitats, feeding on most human food and reproducing at a remarkable rate. Dermatitis caused by the bites of mites has been associated with house mouse infestations. The uncomfortable skin irritation and itching can affect children and adults. Mites may spread through all of a mouse-infested house or apartment during particular times of the year, and the dermatitis is frequently blamed on other causes (heat rash, allergies, fleas and the like).

**Leptospirosis (Weil's disease).** The house mouse can be a major carrier of leptospirosis, although human cases are more commonly caused by rats. Human cases of this disease are seldom fatal. The disease organisms are spread from rat urine into water or food, and they enter humans through mucous membranes or minute cuts and abrasions of the skin. The disease may be so mild as to be unnoticed or may cause mild aches, pains and fever. More serious cases, often referred to as Weil's disease, can result in high fever, jaundice, aseptic meningitis, acute kidney failure, internal bleeding and occasionally death.

**Meningitis.** Lymphocytic choriomeningitis is a viral infection of house mice that may be transmitted to people (mainly to children) through contaminated food or dust.

#### Other

**Typhoid fever.** House flies transmit many human enteric diseases such as dysentery, cholera and typhoid fever. Sometimes the organisms are carried on the fly's **tarsi** or body hairs, and frequently they are regurgitated onto food when the fly attempts to liquefy it for ingestion. Because the house fly has a wide flight range and varied food tastes, and because the female is naturally attracted to filth where she can lay her eggs, the presence of flies in dining facilities or homes is dangerous.

## IV. IMAGINARY PEST INFESTATIONS

Imagination is the ability to form a mental image to experience something that is not present. Everyone experiences an occasional itch that feels like crawling insects. A look confirms that either an insect is present or the mental image was not real. These unreal feelings can be troubling. Concern that the cause of an itch cannot be seen and may be a microscopic parasite can be overwhelming. This idea affects some people so strongly that it inhibits their ability to function. Imaginary insect-related problems can be separated into three groupings: entomophobia, contagious hysteria and delusions of parasitism.

**Entomophobia.** Taken alone, entomophobia can be defined as an admitted fear of insects. This does not mean a fear of imaginary insects but an exaggerated, illogical, unexplained fear of actual insects. A fear of insects occurs to a minor extent with a majority of people. In an extreme form, when the fear inhibits normal functioning, professional counseling is needed. Group treatment has been found to be very successful. Entomophobes rarely are problems for pest-control technicians, but entomophobic people can be expected to demand preventive pesticide applications that are both unwarranted and undesirable. The term "entomophobia" is used sometimes generically to include all imaginary insect-related categories.

**Contagious hysteria.** As the name implies, imaginary pest infestations sometimes upset a group of people at the same time. This hysteria can be passed along or accepted by others. Contagious hysteria often occurs in an office work force. Factors usually connected with the hysteria include crowded conditions, overtime work, excessively detailed or boring tasks, changing climate, changing seasons, paper handling, perceived unfairness of working conditions caused by physical arrangements in the work space, etc. Classically, a few individuals, including a leader or spokesperson, begin feeling bites and

discover rashes and other skin eruptions. These individuals identify certain portions of rooms where the pests are common and demand control. Supervisors usually do not believe there is a pest problem because they are usually unaffected by the contributing conditions, but they may be recruited as pressure for results mounts. When careful inspection for mites, psocids and other pests fails to reveal culprits, premises should be checked for allergens and irritants such as insect parts, fiberglass fragments, static electricity, volatile materials, etc. Pesticide application in these cases should be avoided. Sticky traps can be used for monitoring. After taking the appropriate steps to review all possibilities, simply demonstrating a concern and continued level of alertness often will be an adequate solution.

**Delusory parasitosis.** A condition in which an individual has delusions of parasitism is an extremely emotional and sensitive situation. An inspection of the problematic environment and an examination of specimens alleged to be the pest or parasites affirm or contradict the occurrence of an infestation to the technician but rarely to the client. Often people affected by these delusions will have been referred from one or several physicians to dermatologists, psychiatrists, entomologists, health departments and pest-control companies ad infinitum. The amount of time that must be expended by each consultant soon becomes excessive, and the patient experiences repeated rejections of one type or another — not to mention strain due to expenditure of time and money. In any of these situations, it is possible that the individual has a medically treatable condition. There have been cases in which drug abuse or contraindicated drug prescriptions for patients being treated for several health problems elicited such manifestations. The fact is, there is little that can be done by anyone but a physician with experience in handling cases of delusory parasitosis. Be honest in answering questions, do not agree to see pests that are not there, and do not apply pesticides in these situations. Remind the client that pest management is conducted only when active pest infestations have been identified and evaluated.

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