

Public Health Pesticide Applicator Training Manual

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 or animal material 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. More soldiers have been killed by diseases transmitted by insects than by all those killed by all military actions combined. Fleas, lice and mosquitoes infect man and domesticated animals directly or indirectly with the organisms of many dangerous diseases. Arthropod stings or bites are occasionally fatal to humans, however, their germ-laden saliva or contaminated bodies are also potential sources of disease transmission. Arthropods, especially the insects, are vital players in the transmission of certain diseases to humans. This chapter is devoted to disease transmission relationships. Comments on the impact of animals of public health importance that are not implicated in the transmission of disease, yet are annoying and / or harmful are provided in chapters devoted to those relationships.

I. VECTOR - HOST INTER-RELATIONSHIPS

In considering transmission of disease-causing organisms, it is important to understand the relationships between the **vector** (for example, insect), the disease **pathogen** (for example, virus) and the **host** (humans or animals). The pathogen may or may not undergo different life stages while in the vector. In either case, the vector is the only means for the pathogen to pass from one host to another.

For those pathogens that undergo changes in life stages in the vector before being transmitted to humans, the vector is required for pathogen transmission. Without the vector, the disease life cycle would be broken and the pathogen would die. Some diseases requiring an insect host may require several hosts before completing the pathogen transmission. The hosts may be different species in the various phases of the process. By understanding how a disease is transmitted and the involvement of vectors in the transmission, public health personnel can better design and manage control programs for a particular problem.

Myiasis. Infestation of an animal, including man, 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 man are examples of this phenomenon. In the United States, the primary screwworm fly has been eradicated, 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 the vectors transport organisms, such as bacteria that cause dysentery, on their feet, body hairs, and other body surfaces to the host. There is no multiplication or development of the pathogen within the vector's body. The house fly, *Musca domestica*, is a passive (mechanical) transmitter of bacillary dysentery. Mechanical transmission can be considered as being more or less accidental. That is, the vector does not consciously seek out the disease organism or its location to feed and then transmit the disease to humans.

Biological transmission of disease. Disease causing pathogens need help in moving from one host to another. Many ticks and insects are important vectors of biologically transmitted diseases, where pathogen transmission occurs when an arthropod like a tick or mosquito acquires the pathogen from one

host, it develops in the arthropod's body and is transmitted to another host. Within the arthropod, the pathogen may multiply or may remain as it was first acquired. 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, man is the **intermediate host** in which the asexual stages of the parasite are found.

Obligatory vectors. Obligatory vectors of disease include those in which the pathogen will develop from one stage to another. Malaria is an example of a disease which must be taken up by a vector (mosquito) in order for the pathogen (malaria) 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 hosts that harbor a disease-causing pathogen over an extended period of time without showing symptoms of the disease. Disease transmission occurs when a vector feeds on a host that has the pathogen. After acquiring the pathogen, the vector then transmits it at a later date while feeding on another host. Birds are an example of reservoirs for mosquito-borne encephalitis which may affect humans and horses or other dead-end hosts that are not normally in the transmission cycle. 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 non-reservoir hosts without involving man are called **zoonoses**. When a disease outbreak occurs in these non-human animals, it is called an **epizootic**.

Some vectors pass the disease through eggs by **transovarial** transmission. This means that when the female is infected with the disease organism, her eggs become infected and the resulting larvae will be 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 *rickettsiae* causing Rocky Mountain Spotted Fever and with several mosquito-borne viruses. In this instance the tick and the mosquito serve both as reservoirs and vectors.

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. Even stings of bees and wasps may be serious or even fatal to persons 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 stinging nettle plant. Cantharidian, 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 enquiry.

II. THE DISEASES TRANSMITTED

Mosquito-borne

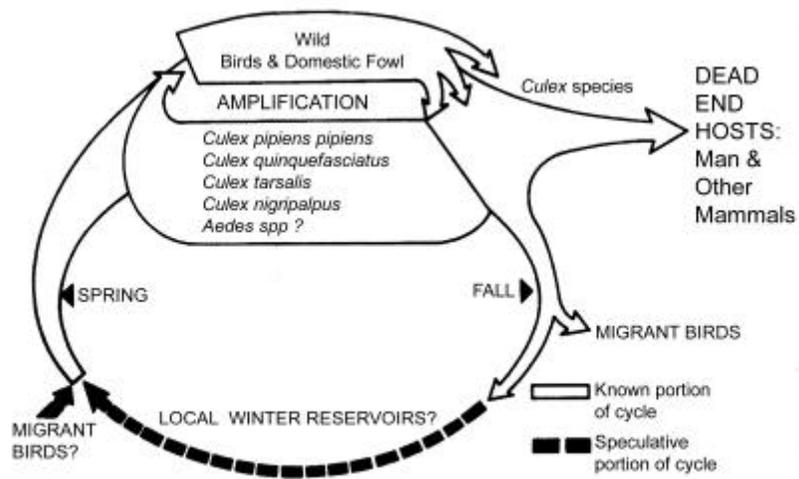
Aside from the irritation and annoyance that mosquitoes inflict on humans and livestock alike, the threat of disease pathogen transmission is always present. Malaria, dengue, yellow fever, and encephalitis are just a few major representatives of a long list of human diseases for which mosquitoes serve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

United States, whereas malaria and dengue are occasionally imported by travelers, and yellow fever is now quite 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 *Culex* mosquitoes. 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, and 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 again 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.



Life cycle of St. Louis encephalitis

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 refractory mosquito species and so is not transmitted even though the mosquito has fed on an infected bird. Also, the virus does not develop to high enough levels in the blood of some vertebrates to infect even the most susceptible mosquito species that feed on these animals. These vertebrates are called "dead-end hosts" because the virus cannot be acquired from them by susceptible mosquitoes and transmitted to other vertebrates. Humans, non-human 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.

The reaction of humans infected with SLE virus is quite variable and dependent on age, general health and other poorly understood characteristics. Infected humans may experience subclinical (inapparent) symptoms or the onset of fever, nausea, and vomiting with severe headaches within five to seven days, and severe central nervous symptoms that may produce temporary or permanent physical or mental disabilities, or even death. In general, the fatality rate is higher and the symptoms more severe in people over 50 years of age. In past epidemics, the fatality rates have ranged from four to 20 percent with most deaths occurring in the older age groups. Studies conducted in areas where epidemics have occurred indicate that for each person who developed symptoms of SLE virus infection (ranging from mild to severe) from 80 to 800 people had been infected with the virus but did not develop disease symptoms. The older age groups consistently exhibited the highest number and most severe symptoms of those infected during these epidemics.



Distribution map of St. Louis encephalitis in U.S.

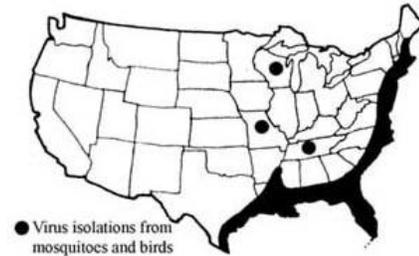
In eastern and midwestern United States, epidemics of SLE have occurred in relatively densely-populated urban and suburban areas in which suitable bird and mosquito species live in close association with humans. *Culex pipiens*, the northern house mosquito, is thought to be a primary vector of SLE in areas where there are (1) many water-filled artificial containers that provide ideal breeding sites for mosquitoes, and (2) populations of birds, such as sparrows and starlings, that live and nest in or near human residences. Water-holding structures commonly associated with humans (for example, 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: But rural areas are also highly susceptible to transmission because pastures and other extensive temporary fresh-water breeding sites can produce large populations of non-domestic avian-feeding mosquitoes, e.g., *Culex nigropalpus*.

The probability of SLE transmission occurring 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 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) and are most likely to occur from mid-summer to early fall, with case detection continuing until cold weather reduces mosquito abundance. SLE occurs throughout the United States, the number of reported cases ranging from a handful to over 1800 annually in the two decades following 1970, and over 200 deaths were attributed to SLE in St Louis alone in 1933.

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, these 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 development to adult is completed in eight to 10 days, whereas 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 reaching their peak in late August. This species feeds primarily on birds, but there is some evidence that it may change its feeding habits from 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.

It is not known how SLE virus is maintained in nature during the winter months when vector mosquitoes do not actively feed and animal infections are not detected. There is some 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 SLE. It is distributed along the coastal states of the Gulf of Mexico and Atlantic Seaboard and occasionally occurs in the Midwest.

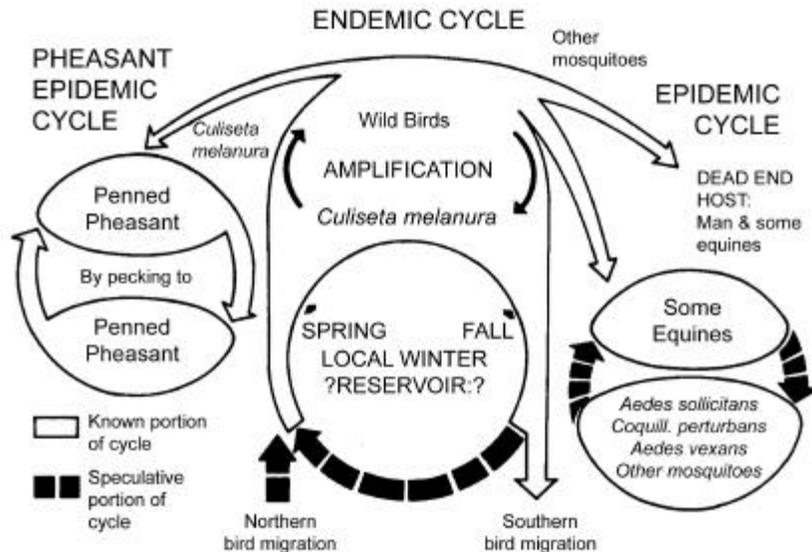


Distribution map of Eastern equine encephalitis in U.S.

Virus activity is confirmed by encephalitis monitoring programs conducted by mosquito control districts. These agencies periodically capture wild birds or maintain sentinel chicken flocks and collect blood samples for laboratory analysis to detect antibodies that were produced after they were infected by the EEE virus. High antibody levels in young birds indicate recent circulation of the virus, whereas antibodies in older birds might be the result of infection the previous year or earlier. Periodic outbreaks among non-vaccinated equines also provide evidence of local EEE viral activity. Equines are involved as "dead-end" hosts in the cycle along with man. 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, like emus, than in man and often produces fatal outcomes. Deaths due to EEE viral infections have been reported in red-winged blackbirds, house sparrows, whooping cranes and pheasants.

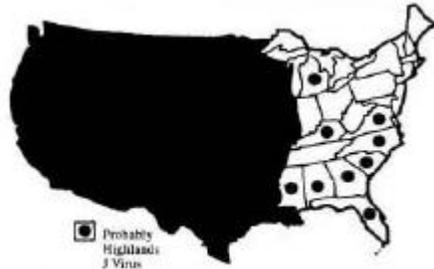
EEE can infect persons of any age, but young children and infants are particularly vulnerable to the disease. Seldom are more than 20 or 30 human cases reported nationally in any given year, although localized foci of human cases do occur. Mortality rates in human infections may exceed 50 percent. In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

There are a number of mosquito species, especially *Culiseta melanura*, that are capable of maintaining and transmitting EEE to birds in nature. Since *Cs. melanura* is relatively uncommon and seldom bites man, other mosquitoes such as *Cs. inornata*, *Cx. quinquefasciatus* (southern



Life Cycle of Eastern equine encephalitis

house mosquito), *Aedes albopictus* (Asian tiger mosquito), *Ae. sollicitans* (tan salt marsh mosquito), *Ae. vexans*, *Ae. infirmatus*, *Ae. atlanticus*, and *Coquillettidia perturbans* are important in transmission from birds to man and/or equines. Factors favoring infections in man are the buildup of virus in wild-bird populations and a high density of adult mosquito vectors.



Distribution map of Western equine encephalitis in U.S.

Western Equine Encephalitis (WEE). Western equine encephalitis is another common arboviral disease in the United States and is found mainly in the states west of the Mississippi River, Wisconsin and Illinois. Major, widespread outbreaks of WEE have occurred in the western parts of the United States and Canada. This disease occurs primarily in rural locations and has a natural transmission cycle that involves both mosquitoes and wild birds. Human mortality rates generally range from one to five percent, but considerably higher in horses. *Culex tarsalis* and *Aedes melanion* are the most important vectors throughout the

western United States and, as with EEE, birds are the major host. East of the Mississippi River, *Cx. quinquefasciatus* is the suspected vector. As in EEE, a vaccine for horses is available, but no vaccine is available for humans.

Western equine encephalitis can produce severe disease in both humans and horses, but both of these 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 infections. 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.

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 the feeding mosquito. 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 ground water with high organic content, such as sewage effluent. In many areas of the West, the species is most numerous where improper agriculture irrigation practices result in water accumulations. This mosquito feeds readily on both birds and mammals, so it serves as the major vector in the natural transmission cycle as well as transmitting WEE virus to humans and horses. As with SLE, it is not presently known how this virus is maintained in areas of the United States 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 the 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%, 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 *Aedes*

mosquitoes, and most of the human cases have occurred in people who lived or worked in or near wooded areas. However, LAC virus seems also to be associated with container breeding mosquitoes, such as *Ae. triseriatus*, which readily maintain LAC virus in a transovarial transmission cycle. These viruses have been isolated from a number of species of woodland mosquitoes, particularly *Ae. triseriatus*, *Ae. canadensis*, *Ae. melanimon*, *Ae. trivittatus*, *Ae. stimulans* and *Ae. atlanticus*.

Venezuelan Equine Encephalitis (VEE). Venezuelan equine encephalitis is a virus that was first recognized in South America. But 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 either humans or equines without the involvement of birds. The disease is mild in humans, producing flu-like symptoms, but is often fatal to equines. In the 1971 epidemic of the 1B type, more than 1,500 horses died of VEE in Texas, but no human death was reported among the over 100 suspected human cases. Equine vaccination, when combined with appropriate mosquito control measures and equine quarantine, provides seasonal protection, but no VEE vaccine is available for humans.

No VEE activity has been reported in the United States 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 re-introduction 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, a number of species of mosquitoes have become suspect vectors. *Psorophora columbiae*, *Ae. sollicitans*, and *Ps. discolor* were incriminated, and it is suspected that other species of *Aedes* (e.g., *Ae. albopictus*) 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 United States. Over 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 sub-clinical 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 transmitted to humans primarily by *Culex* mosquitoes. At this writing (2000) public health authorities are carefully monitoring birds, mosquitoes and human encephalitis cases along the entire eastern seaboard from Maine to Texas to determine if the virus has become established in the U.S.

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. However, four strains of dengue virus are recognized, each of which produces life long immunity against the infecting virus in humans after infection. 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 (DDS). Increased dengue hemorrhagic fever

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 1800's and early 1900's in the United States. 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 that were unapparent and/or unreported cases. However, in 1980, Texas recorded 23 locally acquired cases of dengue and new cases have occurred sporadically until 1999 when 18 cases were reported. Although not endemic now in most of the United States, potential dengue vectors are present in the southern United States and the virus is commonly imported in persons 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-man-mosquito cycle. This species is 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 transmitting the virus to man. Once infected, the mosquito remains infective for the remainder of her life. Presently, there is no vaccine available to prevent dengue infections.

Yellow Fever (YF). Yellow fever is caused by a virus closely related to the dengue virus. In fact, YF infections produce dengue-like symptoms in humans; however, the effects of YF are normally much more severe. The fatality rate may reach 50% 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*. 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 are quarantinable diseases. This means that the Centers for Disease Control and Prevention in Atlanta, Georgia, are continually monitoring outbreaks of YF in the Western hemisphere. Yellow fever vaccines are used to prevent disease in humans going to YF endemic areas. It is also required of people moving from YF endemic areas to YF-free areas where *Ae. aegypti* is present.

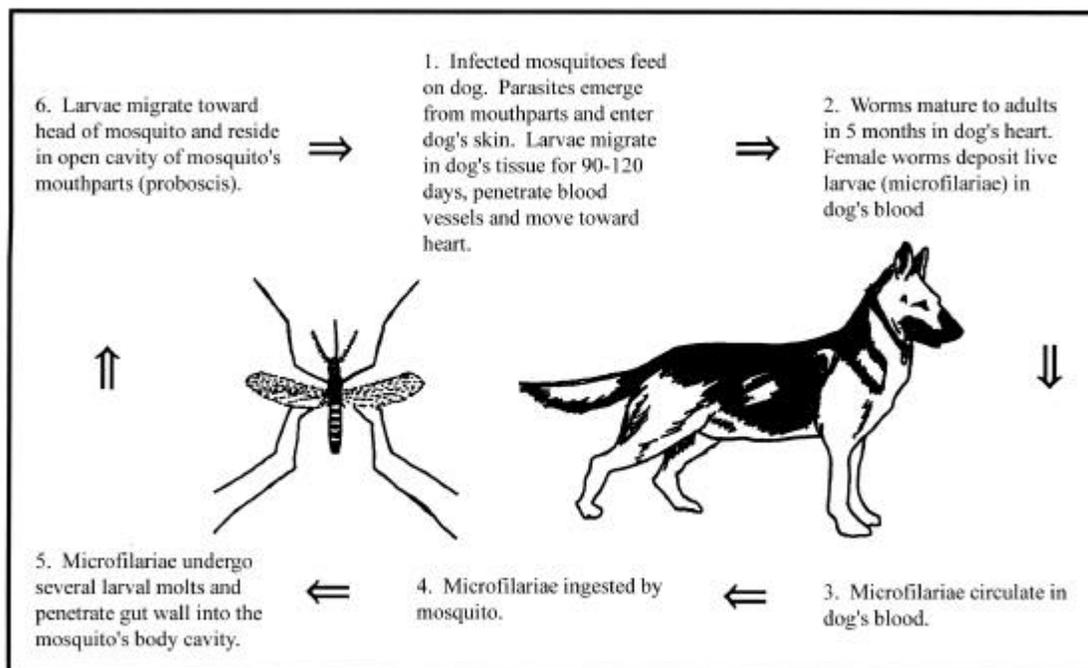
Malaria. On a worldwide basis, malaria remains the most important human disease transmitted by mosquitoes. It is estimated that there are 400 million human cases of malaria in the world (mostly in Asia and Africa), with over two million human deaths annually. Most of the deaths are children under 10 years of age. In Africa, more than one in every 20 children die from malaria. It is believed that malaria was introduced into the North American continent 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. However, in the 1930s, approximately 100,000 cases were reported annually; in the early 1940s, the number of cases was reduced dramatically due to the work of public health agencies using DDT during and after World War II and to 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 there been secondary infection 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. Anti-malarial prophylaxis is effective and most non-resistant 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 through 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% in dogs over two-and-a-half years old, and almost 100% in dogs over five years old. Man can also become infected, but the worms are unable to develop or mature because humans are not their natural hosts. Several genera of mosquitoes can transmit the parasite to dogs (e.g., *Aedes*, *Anopheles*, *Culex* and *Psorophora*). However, in nature, *Cx. quinquefasciatus* is considered to be the most important vector. Other incriminated vectors of dog heart worms include *Ae. albopictus*, *Ae. sierrensis*, *Ae. taeniorhynchus*, *Ae. vexans* and *Cx. salinarius*.



The life cycle of the dog heartworm involves two factors: 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.

Filariasis. Although not currently present in the United States, 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 from man to man by *Mansonia*, *Culex*, *Anopheles* 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 United States.

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 mechanical (i.e., 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. The large urban population in the United States is becoming increasingly at risk to 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 wildlife populations - hosts for tick-borne disease - inhabit 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 that allow the pathogen to enter the host and that extend the time and distance that the ticks are transported by the host. In addition, many species have a wide host range. They have a tremendous reproduction potential and lay several thousand eggs.

Colorado tick fever (CTF). Transmitted in areas above 1525 m in the mountainous areas of the West, CTF occurs annually - mostly in Colorado. The viral dengue-like disease is caused by a *coltivirus* in the family Reoviridae. After an incubation period of three to six days, the onset of illness includes 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% 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 (*Dermacentor 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 parochial 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 non-infective 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 continental United States. Ticks are the primary reservoirs, maintaining the disease by 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 that include fever, chills, severe headaches and general aches and pains. A reddish-purple-black rash may occur on the bottom of the foot, ankles, palms of the hand, characteristically on the wrists or forearms a few days after infection. The rash may spread to the trunk, neck, and face. Often aching in the lower back and headaches around the head and eyes will also occur. Victims feel very tired and can run fevers of 104 to 106° F. Less obvious symptoms may not be noticed. Laboratory blood tests can be done to 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, Connecticut 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 *Ix. damini*) in the East and *Ixodes pacificus* in the West - usually in the nymphal stage, which is most active in the 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 host of the adult ticks in nature, but are incompetent reservoirs of the parasite. A vaccine is available for use by persons in high risk situations.

This serious disease can be 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, 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 does not occur for an estimated 10 to 12 hours 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 ring-like 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. Flu-like 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. Over half of untreated victims experience arthritis intermittently or chronically in the large joints (primarily knees, elbows, and wrists). A few (10 to 27 %) 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 six 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 United States 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 United States is strongly linked to the distribution of the vectors.

Ehrlichiosis (HGE). Human granulocytic ehrlichiosis is one of two forms of ehrlichiosis found in the United States, first recognized in 1994. The infections, caused by *Ehrlichia chaffeensis* or HGE, range from mild illness to severe life-threatening or fatal disease. It is similar to RMSF with high fever, headache and nausea but with a lower incidence of rash. Severe and fatal complications may ensue, including renal and respiratory failure.

Transmission occurs primarily from May through July and appears to be 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 history stages.

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 western United States 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 over 900 m 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, but can become life-threatening in elderly persons. The pathogen, *B. microti*, is transmitted during the summer months by nymphs of the blacklegged tick, *Ixodes scapularis*, in the northeast and midwestern United States. 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 United States, 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 toxin in their saliva. In humans, symptoms usually begin in the legs with muscle weakness and progresses to the trunk, back and chest, where it 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 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 (1 *Amblyomma*, 5 *Dermacentor*, 2 *Haemaphysalis*, 5 *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, more than wounds of war, 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. However, neither epidemic typhus nor louse-borne relapsing fever are found in the United States, 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 that 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 bacterium, *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 can not pass beyond the blockage and becomes contaminated with the plague bacilli. The contaminated blood is then regurgitated back into the wound caused by the flea bite.

Bubonic plague is endemic throughout western United States. 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 of the city's population. The Black Death of Europe in the 14th century lasted 50 years and killed 25 million people. In the first quarter of this 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 United States.

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 United States. The hantavirus fatality rate in humans range from 10 to 55%. 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 may also occur. Shortness of breath is a symptom which 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 increases 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 (Weil's disease), 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 virus 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 these organisms are carried on the fly's tarsi or body hairs, and frequently they are regurgitated onto food when the fly attempts to liquify 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.

III. 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, help from professional counseling is needed. Group treatment has been found to be very successful. Entomophobes rarely are problems for pest control technicians, but entomophobic persons 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 workforce. 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 fruit flies 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 physician(s) to a dermatologist, to a psychiatrist, to entomologists, to health departments, to 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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