Tick Management Handbook

A integrated guide for homeowners, pest control operators, and public health officials for the prevention of tick-associated disease

Prepared by:

Kirby C. Stafford III
Chief Scientist
The Connecticut Agricultural Experiment Station, New Haven

Produced as part of the Connecticut community-based Lyme disease prevention projects in cooperation with the following Connecticut health agencies:

The Connecticut Department of Public Health
The Westport Weston Health District
The Torrington Area Health District
The Ledge Light Health District

Funding provided by

The Centers for Disease Control and Prevention

The Connecticut Agricultural Experiment Station

This handbook was developed as part of a community-based program for the prevention of tick-borne illness supported through a cooperative agreement with the Centers for Disease Control and Prevention (CDC). The CDC funded publication of this tick handbook. A series of tick and tick-associated disease information sheets first developed by Dr. Kirby Stafford at the Connecticut Agricultural Experiment Station in 1992 and updated and expanded periodically was the original basis for this handbook.

Acknowledgements

Thanks are given to Dr. Joseph Piesman (CDC, Fort Collins, Colorado), Dr. Peter J. Krause (University of Connecticut Health Center, Farmington, Connecticut), Carol Lemmon (CAES, retired), Bradford Robinson (Connecticut Department of Environmental Protection, Pesticide Management Division), Judith Nelson, Director, and the staff of the Westport Weston Health District (CT), Dr. Terry Schulze (NJ), Dr. Gary Maupin (CDC, retired), and Drs. Louis A. Magnarelli and John F. Anderson (CAES) for reviewing parts or all of this handbook. Their comments and suggestions were sincerely appreciated. Thanks are also extended to Vickie Bomba-Lewandoski (CAES) for publication and printing assistance.

Photo Credits

Many of the pictures and illustrations are those of the author or staff at the Connecticut Agricultural Experiment Station (CAES). Sources other than the author are numbered or otherwise noted in captions. Sincere thanks are given to the following for permission to use their photographs or illustrations and federal government sources are also gratefully acknowledged.

Pfizer Central Research (Groton Point Road, Groton, CT): 1, 6, 7, 8, 9, 11, 12, 20, 21, 23, 24, 25, 26, 32, 33, 34, 37, 47, 48.
Centers for Disease Control and Prevention: 13, 14, 16, 17, 19, 27, 28, 29, 30, 36, risk map.
United States Department of Agriculture: Cover (tick), tick morphology figure (adapted from Strickland et al. 1976), 39.
American Lyme Disease Foundation (Somers, NY): 3, 4, 10, 43.
Barnstable County Cooperative Extension (Massachusetts): 40.
Vector-borne Disease Laboratory, Maine Medical Center Research Institute (Portland, ME): 15.
Bayer Environmental Science (Montvale, NJ): 46.
United Industries (Spectrum Brands): 38.
Ric Felton (Goshen, CT; www.semguy.com): 35.
Jim Occi (Cranford, NJ): 5, 18, 45.
Lynne Rhodes (Old Saybrook, CT): 22, 23.
Steven A. Levy, DMV (Durham, CT): 31.
CAES: Jeffrey S. Ward, 2; Uma Ramakrishnan, 41, 42; and Jeffrey Fengler, 44.

Disclaimer

Published Summer 2004

Table of Contents

To these I must add the wood lice [ticks] with which the forests are so pestered that it is impossible to pass through a bush or to sit down, though the place be ever so pleasant, without having a whole swarm of them on your clothes.

Pehr Kalm, 18 May 1749
Raccoon [Swedesboro], New Jersey

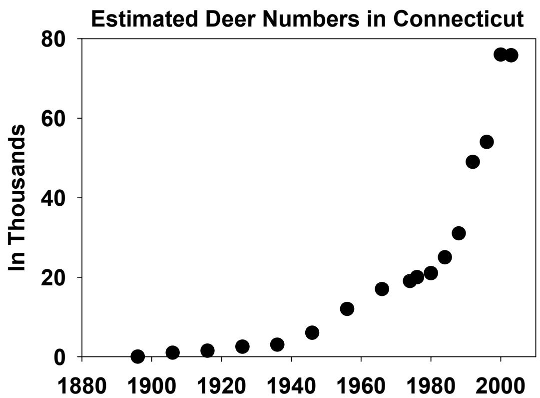
Introduction

Ticks have become an increasing problem to people and animals in the United States. Ticks are obligate blood-feeders that require an animal host to survive and reproduce. They feed on a wide variety of mammals, birds, reptiles, and even amphibians. While most ticks feed on specific host animals and are not considered to be of medical or veterinary importance, several ticks have a wide host range and attack people, pets, or livestock. Ticks can be a nuisance; their bites can cause irritation and, in the case of some ticks, paralysis. Severe infestations on animals can cause anemia, weight loss, and even death from the consumption of large quantities of blood. Ticks can also transmit many human and animal disease pathogens, which include viruses, bacteria, rickettsiae, and protozoa.

1

The association between ticks and disease was first demonstrated when Theobald Smith and Fred Kilbourne proved in 1893 that Texas cattle fever (cattle babesiosis) was caused by a protozoan transmitted by an infected tick. In the late 1800s, Rocky Mountain spotted fever was the first human tick-borne disease identified in the United States and for many years was the major tick-associated disease in this country. Although first recognized from the virulent cases in the Bitterroot Valley of Montana, it eventually became evident that most cases were distributed through the eastern United States. Lyme disease was first recognized as a distinct clinical entity from a group of arthritis patients in the area of Lyme, Connecticut, in 1975, although it became evident that this disease had an extensive history in Europe throughout the twentieth century. Today, Lyme disease is the leading arthropod-associated disease in the United States with over 23,000 human cases reported to the Centers for Disease Control and Prevention (CDC) in 2002. This may represent only about 10% of physician-diagnosed cases. Surveys have found that up to a quarter of residents in Lyme disease endemic areas have been diagnosed with the disease and that many residents perceive the disease as a serious or very serious problem. Without an effective intervention strategy, the steadily increasing trend in Lyme disease case reports is likely to continue.

In the northeastern United States, the emergence of Lyme disease can be linked to changing landscape patterns. A Swedish naturalist named Pehr Kalm recorded in his journal of his travels in the United States in 1748-1750 that ticks were abundant and



annoying. Over a century later in 1872, entomologist Asa Fitch noted that ticks were nearly or quite extinct along the route that Pehr Kalm had traveled. During this time, the land had been cleared for agriculture and white-tailed deer in many areas were drastically reduced or virtually eliminated due to habitat loss and unregulated hunting. With the reestablishment of forested habitat and animal hosts through the latter half of the twentieth century, ticks that may have survived on islands off the southern New England coast were able to increase and spread. The blacklegged tick, *Ixodes scapularis,* which is commonly known as the "deer" tick, and the principal vector for Lyme disease spirochetes, was present on Naushon Island, Massachusetts, in the 1920s and 1930s. Some *I. scapularis* from Montauk Point, Long Island, New York, that were collected in the late 1940s and early 1950s were found infected with Lyme disease bacteria. The risk of human infection increased through the 1960s and 1970s until the recognition of the disease from the cluster of cases in Lyme, Connecticut, in 1975. The rising incidence of Lyme disease is due to a number of factors including:

- Increased tick abundance
- Overabundant deer population
- Increased recognition of the disease
- Establishment of more residences in wooded areas
- Increased the potential for contact with ticks.

An estimated three quarters of all Lyme disease cases are acquired from ticks picked up during activities around the home. With the steady increase in the incidence and geographic spread of Lyme disease, there is a need for homeowners, public health officials, and the pest control industry to learn how to manage or control the tick problem. The withdrawal of the human Lyme disease vaccine (LYMErix™) has essentially brought the control of the disease back to managing tick bites and methods to suppress the local tick population or prevalence of pathogen infection in the ticks. The purpose of this handbook is to provide basic information on ticks and their biology, basic information on the diseases they carry, methods to reduce the risk of exposure to these parasites, and most importantly, information on how to reduce or manage tick populations, and therefore risk of disease, in the residential landscape.

Ticks: the foulest and nastiest creatures that be. Pliny the Elder, 23-79 A.D.

Ticks of the Northeastern United States

Ticks are not insects, but are arthropods more closely related to mites, spiders, scorpions, and daddy-long-legs. There are about 80 species of ticks in the United States (850 species worldwide). However, only about 12 or so in the U.S. are of major public health or veterinary concerns with a few others that occasionally attack humans. The ticks discussed in this handbook belong to the family Ixodidae or hard ticks. The principal hard ticks recovered from humans in the mid-Atlantic and northeastern United States are the blacklegged (i.e., deer) tick, *Ixodes scapularis*, the American dog tick, *Dermacentor variabilis*, and the lone star tick, *Amblyomma americanum*. Other tick species recorded as feeding on humans in the eastern U.S. include *Ixodes cookei*, *Ixodes dentatus*, and the brown dog tick, *Rhipicephalus sanguineus*. The Argasidae or soft ticks form the other major group of ticks. Soft ticks are generally nest inhabitants that are associated with rodents, birds, or bats. Several species of soft ticks attack humans and can transmit disease, mainly in western states, but are not the focus of this handbook. One species, *Carios* (*Ornithodoros) kelleyi*, a bat tick, has been recovered from states in the northeast to at least Connecticut.

Table 1. Important ticks of the northeastern states and some other major ticks of medical importance in the United States.

Tick	Common name	General distribution
Hard Ticks		
Ixodes scapularis	Blacklegged tick	Northeastern & mid-western United States
Ixodes pacificus	Western blacklegged tick	Pacific coast & parts Nevada, Arizona, Utah
Ixodes cookei	A woodchuck tick	Eastern United States & northeast Canada
Ixodes dentatus	A rabbit tick	Eastern United States
Amblyomma americanum	Lone star tick	Southeastern U.S., Texas to New York
Dermacentor variabilis	American dog tick	Eastern U.S. & west coast
Dermacentor andersoni	Rocky Mountain wood tick	Rocky Mountain states south to NM & AZ
Dermacentor albipictus	Winter tick	Canada, United States south to Central America
Rhipicephalus sanguineus	Brown dog tick	All U.S. and worldwide
Soft Ticks		
Ornithodoros species ticks	Relapsing fever ticks	Western United States
Carios kelleyi	A bat tick	Widespread in U.S., north to New York and Connecticut

Scientific Names and a Few Terms

The scientific name of ticks, like other organisms, is given in two parts: genus (capitalized, often abbreviated by the first letter, e.g. *I. scapularis*) and species (not capitalized) sometimes followed by the name of the person who described the organism (given in parenthesis if the genus name is later changed). The name Linneaus is abbreviated L. Common names like deer tick can vary regionally and some organisms may have no common name. The common names used in this guide follow those officially recognized by scientific societies. Several terms are used to define the cycles of animal, tick and pathogen.

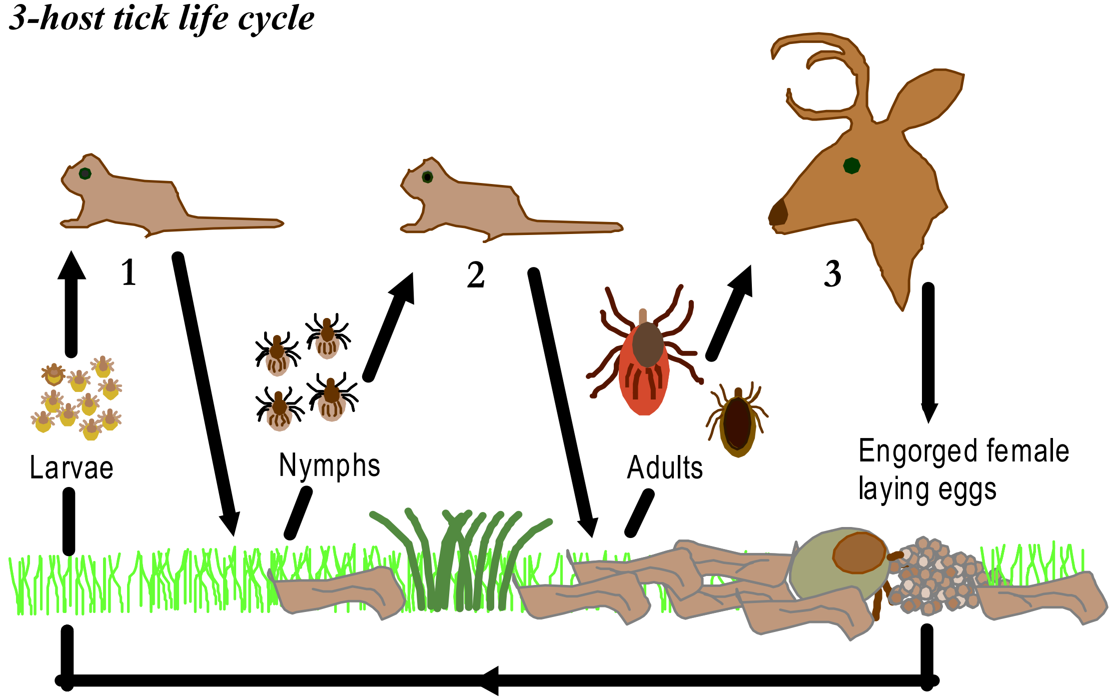
- Pathogen: the microorganism (i.e., virus, bacteria, rickettsia, protozoa, fungus) that may cause disease.

- Parasite: An animal that lives in or on a host for at least part of their life and benefits from the association at the expense of the host (from the Greek, literally para - beside and sitos - food).
- Vector: An insect or other arthropod, like a tick, that carries and transmits a disease pathogen. Diseases associated with pathogens transmitted by a vector are called vector-borne diseases.
- Host: An animal infected by a pathogen or infested with a parasite.
- Reservoir: An animal host that is capable of maintaining a pathogen and serving as a source of infection.
- Zoonoses: A disease caused by a pathogen that is maintained in vertebrate animals that can be transmitted naturally to humans or domestic animals by a vector or through other means (e.g. saliva, feces).
- Endemic disease: A disease that is established and present more or less continuously in a community.

Tick Biology and Behavior

Ticks are essentially mites that have become obligate blood-feeders, requiring a host animal for food and development. Ticks have four stages in their life cycle: egg, the 6-legged larva (seed ticks), and 8-legged nymph and adult (male or female). Larvae and nymphs change to the next stage after digesting a blood meal by molting or shedding the cuticle. Most of the ticks mentioned in this handbook have a 3-host life cycle, whereas each of the three active stages feed on a different individual host animal, taking a single blood meal. Larvae feed to repletion on one animal, drop to the ground and molt to a nymph. The nymphs must find and attach to another animal, engorge, drop to ground and molt to an adult. The adult tick feeds on a third animal. A replete or engorged (blood filled) female tick produces a single large batch of eggs and dies. Depending upon the species of tick, egg mass deposited can range roughly from 1,000 to 18,000 eggs.

3-host tick life cycle



The larvae and nymphs generally feed on small to medium-sized hosts, while adult ticks feed on larger animals. Some ticks may have a one-host (all stages staying and feeding on only one animal host before the female drops off) or other multi-host lifecycles. Depending upon the tick, the life cycle may be completed in 1, 2 or even 3 years, while a one-host tick may have more than one generation per year. Feeding for only a few days, the majority of the life of a tick is spent off the host in the environment either seeking a host, molting or simply passing through an inhospitable season (e.g., hot summers or cold winters). Soft ticks have a multi-host life cycle with multiple nymphal stages; each stage feeds briefly, and adults take multiple small blood meals, laying small egg batches after each feeding. As nest and cave dwellers, often with transient hosts, some argasid ticks may survive many years without a host. However, most hard ticks do not successfully find a host and perish within months or a year or two at best.

3

4

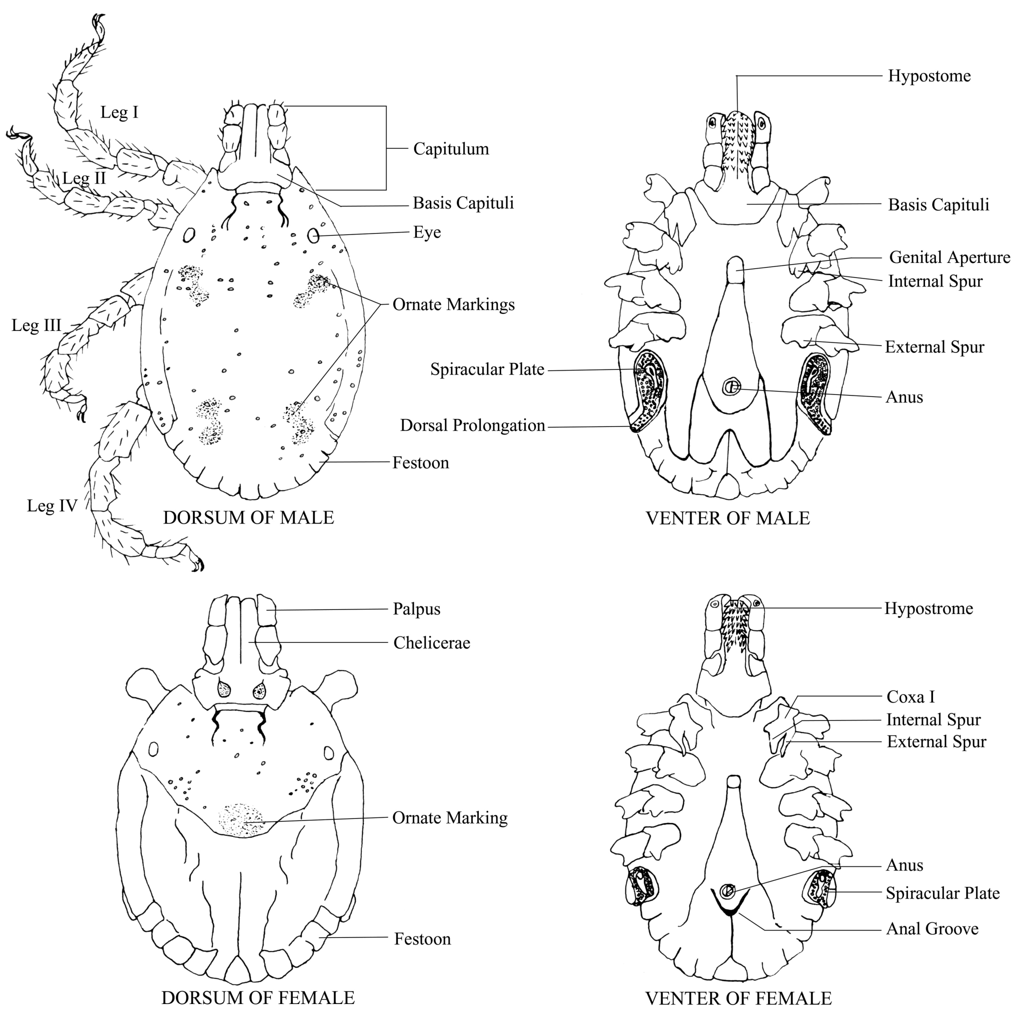
Larval ticks will be clustered on the egg mass after hatching and when ready to feed, ascend blades of grass or similar vegetation to await a host. Ticks assume a questing position by clinging to the leaf litter or vegetation with the back legs, and hold the first pair outstretched. Due to differences in susceptibility to desiccation and host preference, immature ticks generally remain in the low vegetation, while adult ticks may seek a host at a higher level in the vegetation. Ticks detect their hosts through several host odors (including carbon dioxide, ammonia, lactic acid, and other specific body odors), body heat, moisture, vibrations, and for some, visual cues like a shadow. When approached by a potential host, a tick becomes excited - waving the front legs in order to grab the passing host. Ticks cannot fly or jump; they must make direct contact with a host. Once on a host a tick may attach quickly or wander over the host for some time. Some ticks attach only or principally on certain areas like the ear or thin-skinned areas, while other species may attach almost anywhere on the host. Ticks feed slowly, remaining on the host for several to many days, until engorged with blood (see section on feeding in tick bite prevention). Male ticks feed intermittently, take small blood meals, and may remain on a host for weeks. For most ticks mating occurs on the host, as the male tick also requires a blood meal. However, male *Ixodes* ticks do not need to feed prior to mating and mating may occur on or off the host.

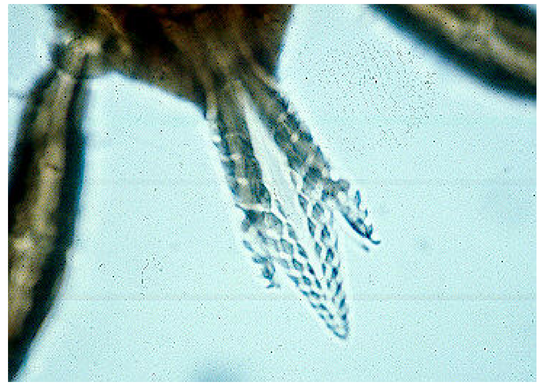
5

Tick Morphology

The body of a tick consists of a “false head” (the capitulum) and a thorax and abdomen fused into a single oval, flattened body. A larval tick has six legs, while nymphs and adults have eight legs present. The basal segment of the leg, the coxa, may have spurs that help in identification. An adult tick will have a genital aperture on the ventral surface, located roughly between the second pair of legs. The respiratory system is evident by spiracular plates located ventrolaterally behind the fourth pair of legs in the nymphs and adults. These plates may be oval, rounded, or comma-shaped. Hard ticks get their name from a tough dorsal shield or plate called the scutum present on all mobile stages of the tick. The scutum on the larva, nymph, and female tick covers the dorsal anterior third to half of the body. By contrast, the scutum on a male tick covers almost the entire dorsal surface and expansion during feeding is very limited. The scutum differs in shape and others characteristics (i.e., presence or absence of simple eyes) between tick genera. In some ticks, ornate or patterned markings may be present that can aid in identification. A distinct semicircular anal groove curves around the front of the anal opening in *Ixodes* ticks. In all other ticks, the anal groove is behind the anus or absent. Many ticks, but not *Ixodes*, have rectangular areas separated by grooves on the posterior margin of the tick body called festoons. Festoons, if present, may not be visible on fully engorged females. Argasid ticks are leathery, wrinkled and grayish in appearance. The capitulum of soft ticks is located on the underside of the body and cannot be seen from above.

Hypothetical Male and Female Ixodidae (Hard Ticks) with Key Characteristics Labeled





The capitulum in hard ticks is visible dorsally in all stages. The capitulum holds the mouthparts consisting of a base (basis capituli), two palps, paired chelicerae, and the median ventral hypostome, which is covered with denticles or recurved teeth. The shape of the basis capituli, length of the palps, number of denticles, and other characteristics of the mouthparts are used to help identify tick genera and species. While the adults of some common ticks can be easily identified with a little training because of distinctive markings or color, the identification of most ticks and the immature stages requires the services of a trained entomologist and the use of keys developed by tick taxonomists. These keys are designed to specifically identify adults, nymphs or larvae.

The Blacklegged Tick or "Deer" Tick, *Ixodes scapularis* Say

6

Above: left to right: larva, nymph, male and female *I. scapularis*. Below top: unfed and engorged female. Below bottom: male, female and engorged female with straight pin.

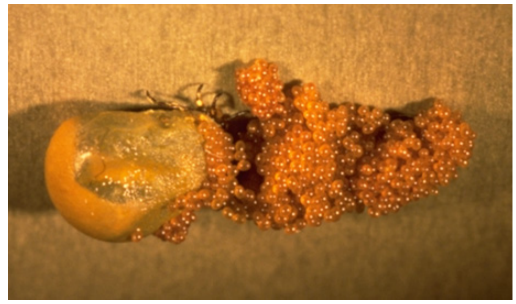
7

8

Blacklegged tick is the correct common name for the tick popularly known as the "deer" tick (the terms are not used together, it is not called the blacklegged deer tick). *Ixodes* (pronounced ix-zod-ease) *scapularis* transmits the causal agents of three diseases; Lyme disease, human babesiosis, and human anaplasmosis. The blacklegged tick is found from some southern portions of Canada and coastal Maine through the mid-Atlantic states into Maryland, Delaware and northern parts of Virginia and in several north central states, particularly Wisconsin and Minnesota, extending down through Illinois and into Indiana. This tick is also found throughout the southeastern United States west to southcentral Texas, Oklahoma, southern Missouri, and eastern Kansas. However, few *I. scapularis* in the southeast have been found infected with the bacterium that causes Lyme disease, the spirochete *Borrelia burgdorferi*. Therefore, the risk for Lyme disease from this tick in the southeastern United States is considered relatively low.

Unfed female *I. scapularis* have a reddish body and a dark brown dorsal scutum (plate) located behind the mouthparts. Length of the female tick from the tip of the palpi to the end of the body is about 3 to 3.7 mm (about 1/10 of an inch). Male *I. scapularis* are smaller (2 – 2.7 mm) than the female and are completely dark brown. Nymphs are 1.3 to 1.7 mm in length, while larvae are only 0.7 to 0.8 mm. Female blacklegged ticks become fairly large when engorged with blood and, consequently, are sometimes confused with engorged female American dog ticks.

Blacklegged ticks feed on a wide variety of mammals and birds, requiring 3-7 days to ingest the blood, depending on the stage of the tick. Larvae and nymphs of *I. scapularis* typically become infected with *B. burgdorferi* when they feed on a reservoir competent host. The white-footed mouse is the principal reservoir (source of infection) for *B. burgdorferi*, the protozoan agent of human babesisois, *Babesia microti*, and can serve as a reservoir for the agent of human granulocytic ehrlichiosis. Birds are also a major host for immature *I. scapularis* and have been implicated in the long-distance dispersal of ticks and *B. burgdorferi*. White-tailed deer, *Odocoileus virginianus* (Zimmerman), are the principal host for the adult stage of the tick, which feeds on a variety of medium- to large-sized mammalian hosts. An engorged female tick may typically lay around 2,000 eggs or more.



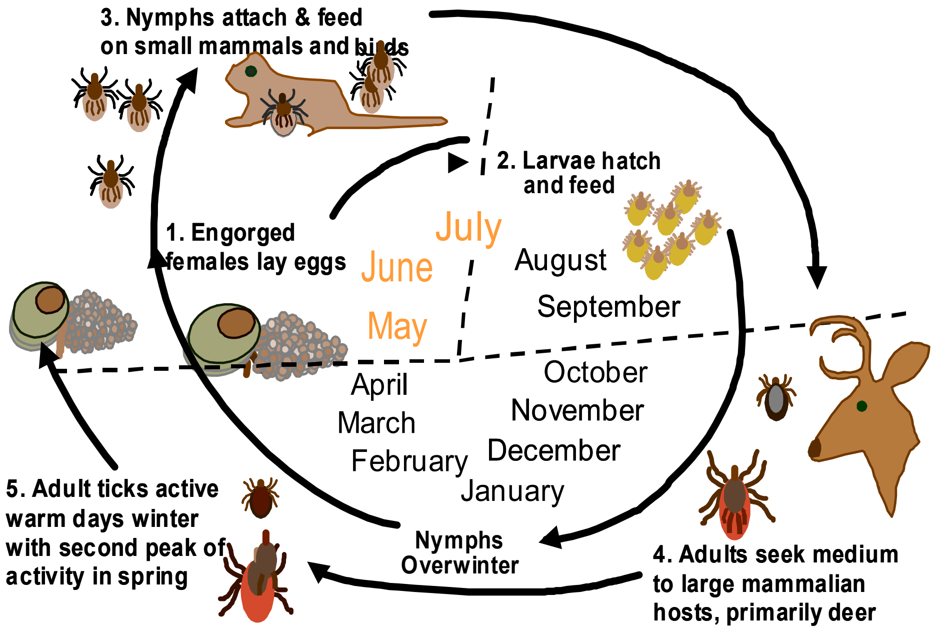
Below clockwise from top left: Nymphal *I. scapularis* in the hand, close-up of an *I. scapularis* nymph (fingerlike projections of the tick mid-gut where the Lyme spirochetes are found are visible through the tick cuticle), female and nymph *I. scapularis* on finger, and nymphal *I. scapularis* on finger.

9

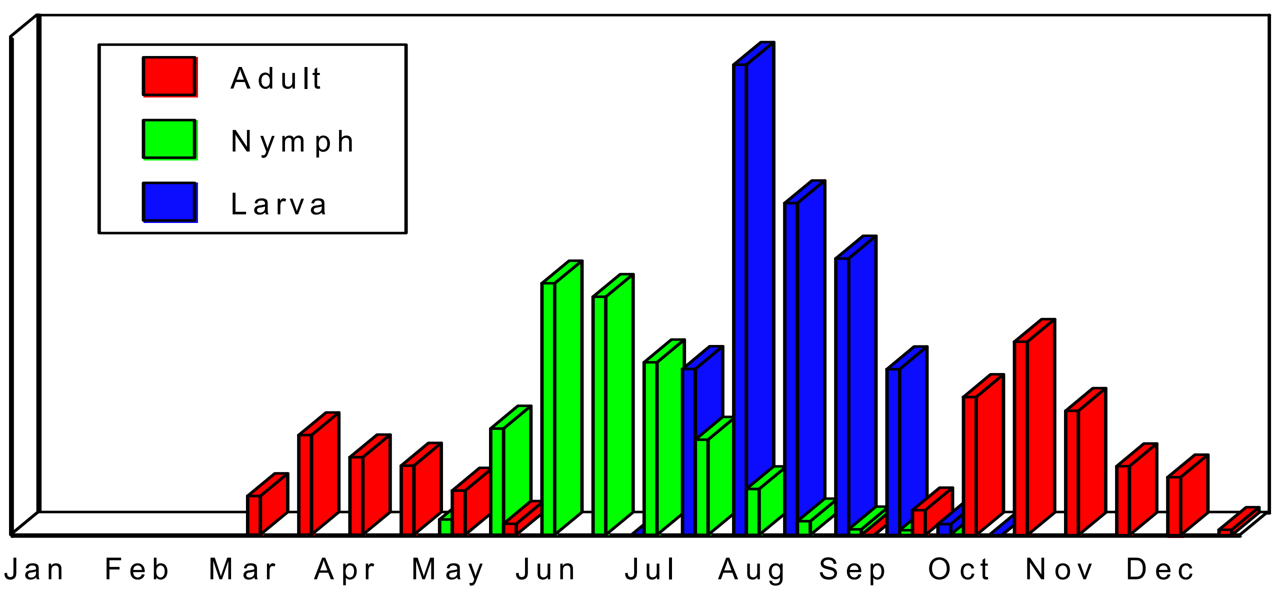
10

The Lyme disease spirochete in northern states is maintained, in part, by the two-year life cycle of the tick. Seasonally, the nymphs precede larvae and infect a new generation of animal hosts. Larvae active later in the summer then become infected when feeding on reservoir host animals. Adults of *I. scapularis* are more commonly infected with *B. burgdorferi* than the nymphs because the tick has had two opportunities to become infected, once as a larva and once as a nymph.

Two-year Life Cycle for *Ixodes scapularis*.



Seasonal activity of *I. scapularis* adults, nymphs, and larvae.



The American Dog Tick, *Dermacentor variabilis* (Say)

The American dog tick, *Dermacentor variabilis*, is the primary vector of the causal agent of Rocky Mountain spotted fever in the eastern United States and is also a vector for the agent of tularemia. This tick does not transmit Lyme disease spirochetes and recent studies have indicated that it is not a vector for the agent of human granulocytic ehrlichiosis. The American dog tick, known by some people as the wood tick, is one of the most widely distributed and common ticks in the eastern and central United States, found from Nova Scotia to the Gulf Coast as far west as Texas, Kansas and the Dakotas. It is also found in parts of California, Oregon, eastern Washington, and northern Idaho. Only adults of the American dog tick feed on people and their pets – records of nymphs from humans are rare. The Rocky Mountain Wood tick, *Dermacentor andersoni*, is found in western North America from British Columbia and Saskatchewan south through North Dakota to northern New Mexico and Arizona and California. This tick is the vector for Rocky Mountain spotted fever and Colorado tick fever in western Canada and the northwestern United States.

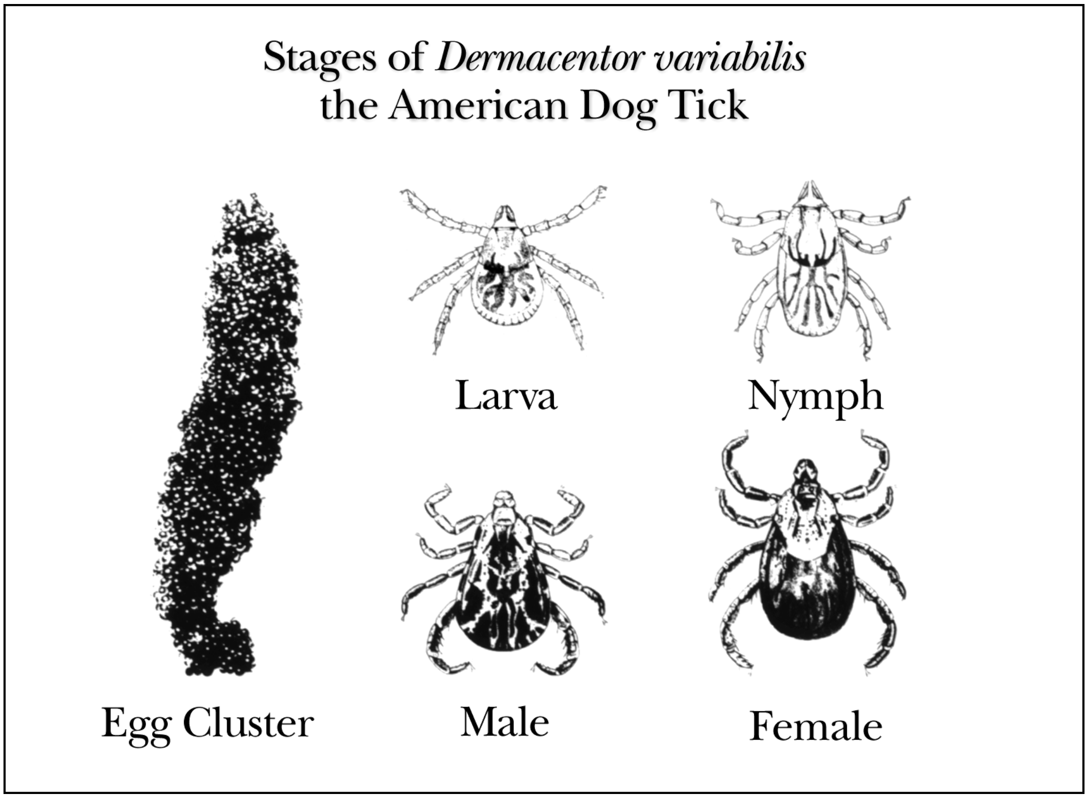
11

12

13

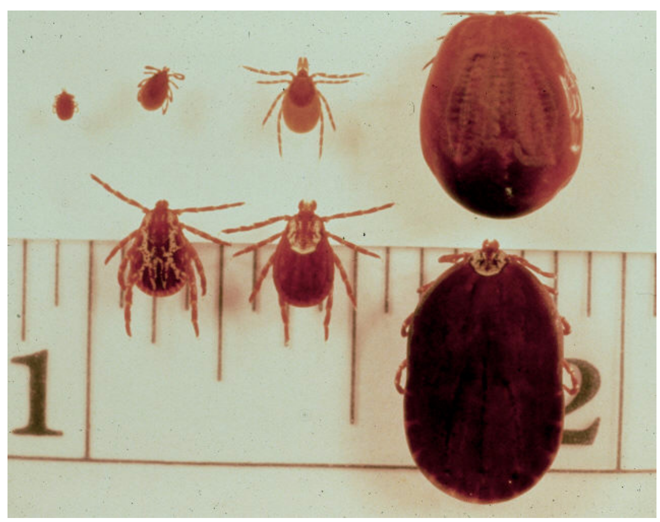
Adult American dog ticks are reddish brown in color with silvery-gray or whitish markings on the back or upper body. They are almost 6.4 mm (¼ inch) in length. The palps are short. The ornate marking are on the scutum of the female and on the male extend over the entire back. Female ticks increase dramatically in size as they obtain their blood meal from a host animal. Fully engorged females may reach ½ inch in length (13 mm long by 10 mm wide) and resemble a dark pinto bean. Male ticks do not change notably in size as they feed. The scutum or plate does not change in size and the white markings are readily visible on a blood-fed tick. Adult dog ticks can be distinguished from adult *I. scapularis* by their larger size and the white markings on the dorsal scutum. In the northeast, adults of both tick species are active during the spring.

Dogs are the preferred hosts of adult ticks, but they also feed readily on other medium to large mammals. These include opossums, raccoons, skunks, fox, coyote, bobcat, squirrel, cattle, sheep, horses and people. Larvae and nymphs of the American dog tick feed on meadow voles (*Microtus pennsylvanicus*), white-footed mice (*Peromyscus leucopus*), and other rodents. In New Jersey, adult ticks are active from mid-March to mid-August. In Connecticut and Massachusetts, adults become active about mid-April to early May, peak in June, and may remain a nuisance until mid-August. Mating occurs on the host. A female tick will feed for 10-12 days. Once she is engorged with blood, she drops off the host, and may deposit about 3,000 to 7,000 eggs (average around 5,000). Males continue to ingest small amounts of blood from the host. In the northeast, the American dog tick probably requires 2 years to complete its life cycle as opposed to one year in the southern parts of its range. American dog ticks can live for extended periods without feeding, more than two years to almost three years, if suitable hosts are not available. Larvae, nymphs, and adults may live up to 540, 584, and 1,053 days, respectively, although typically survival will be much less.



14

American dog ticks are most numerous along roadsides, paths, old fields, marshy areas and trails in brushy woodlands or meadows with tall grass or weeds. Meadow voles are found in fields, pastures, fresh and saltwater marshes and meadows, borders of streams and lakes, and open and wooded swamps. Consequently, large numbers of American dog ticks may be encountered in these areas. People or their pets may bring these ticks from outdoors into the home, where they can survive for many days. However, the tick will not become established indoors. The Brown dog tick (page 13) is the species that may cause household infestations.



15

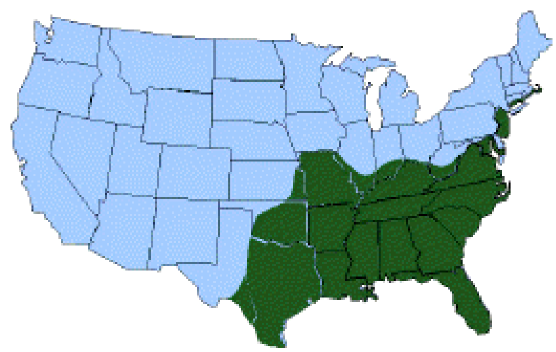
Comparison between the blacklegged tick and American dog tick (above). Top row left to right: nymph, male, female, and engorged female *I. scapularis*. Note engorged female is nearly as large as the engorged female American dog tick. Bottom row left to right: male, female, and engorged female *D. variabilis*. Note the white markings on the scutum of *D. variabilis* can help distinguish between the two engorged ticks (ruler is marked in 1/16 inch intervals between the 1 and 2 inch mark).

The Lone Star Tick, *Amblyomma americanum* (L.)

16

The lone star tick, *Amblyomma americanum*, is named from the conspicuous spot on the end of the scutum of the female tick. This tick is the vector for *Ehrlichia chaffeensis*, the agent of human monocyctic ehrlichiosis (HME). The tick does not transmit the Lyme disease bacterium, *B. burgdorferi*, but has been linked with a Lyme-like illness with a rash and other symptoms resembling Lyme disease called southern tick-associated rash illness or STARI. Possibly caused by another species of spirochete, attempts to culture the organism from skin biopsies at the rash or obtain serological evidence of Lyme disease from affected patients have not been successful thus far. A new spirochete, *B. lonestari*, has been described from lone star ticks based on a DNA analysis and has recently been cultured from ticks. It has been detected in both a tick and associated rash, but it is yet not clear if it is the agent of the Lyme-like illness.

The lone star tick is widely distributed through the southeastern United States as far west as Texas and north to southern parts of Iowa, Illinois, Indiana, Ohio, and Pennsylvania. Along the Atlantic coast, its northern range extends to New Jersey and Long Island, New York, and it is also abundant on Prudence Island, Rhode Island. Lone star tick populations in Connecticut are sparse, but these ticks are occasionally recovered from residents, mainly in coastal communities in Fairfield and New Haven Counties.



Approximate distribution of *A. americanum* shown in green shaded area.

Lone star ticks are reddish brown in color and about 3 to 4 mm long. The palps of *Amblyomma* ticks are long. Female ticks have a conspicuous spot on the end of the scutum. Male ticks have faint white markings at the edge of the body. Nymphs are more circular in shape than *I. scapularis* nymphs and reddish in tint. Adults are active in the spring, while nymphs are active from April through the mid-summer. Larvae are active in the late summer and early fall.

The lone star tick has a wide host range, feeding on virtually any mammal. All stages will feed on people. On wild hosts, feeding occurs principally in and on the ears and the head. The bite of this tick can be painful because of the long mouthparts and attached ticks can cause great irritation. All stages are active during the summer months. Female ticks can deposit 1,000 to 8,000 eggs with an average of around 3,000 eggs. Deer and other large to medium sized animals are hosts for the adults and nymphs. Heavy infestations of this tick have been known to result in blindness and death of fawns of white-tailed deer. In some localities, this tick may also be known as the “deer” tick. Larvae and nymphs commonly feed on large and medium-sized mammalian hosts. Larval ticks also feed on many species of birds. Rodents do not appear to be important hosts for immature *A. americanum*.

Other Ticks

Ixodes cookei Packard

Ixodes cookei, sometimes referred to as the “woodchuck tick”, is found throughout the eastern half of the United States and Canada. It is a primarily a parasite of medium-sized mammals such as woodchucks, opossums, raccoons, skunks, and foxes. In a New York study, it was the second most abundant tick on medium-sized mammals behind *I. scapularis*. All stages of *I. cookei* will feed on humans, though reports in southern New England and New York are uncommon. It appears to be a more frequent human parasite in northern New England and Ontario, Canada. After the American dog tick, *I. cookei* was the second most common tick removed from humans in Maine from 1989-1990 (*I. scapularis* was third). Lyme disease spirochetes have been detected in this tick, but laboratory studies suggest it is not a good vector for *B. burgdorferi*. However, *I. cookei* is the principal vector for Powassan virus, which can cause severe or fatal human encephalitis.

Brown Dog Tick, *Rhipicephalus sanguineus* (Latreille)

The brown dog tick or kennel tick, *Rhipicephalus sanguineus*, is a three-host tick found almost worldwide and throughout the United States. The tick is more abundant in the southern states. This is the only species of this genus in the U.S. Domestic dogs are the principal host for all three stages of the tick, especially in the United States, although the tick feeds on other hosts in other parts of the world. Adult ticks feed mainly inside the ears, head and neck, and between the toes, while the immature stages feed almost anywhere, including the neck, legs, chest, and belly. People may occasionally be attacked.

This tick is closely associated with yards, homes, kennels and small animal hospitals where dogs are present, particularly in pet bedding areas. In the North, this tick is found almost exclusively indoors. Brown dog ticks may be observed crawling around baseboards, up the walls or other vertical surfaces of infested homes seeking protected areas, such as cracks, crevices, spaces between walls or wallpaper, to molt or lay eggs. A female tick can deposit between 360 to 3,000 eggs. Under favorable conditions, the life cycle can be completed in about two months. This tick is the vector for canine ehrlichiosis (*Ehrlichia canis*) and canine babesiosis (*Babesia canis* or *Babesia gibsoni*). The brown dog tick is a vector for Boutonneuse fever in Europe and Africa.

Winter Tick, *Dermacentor albipictus* (Packard)

The winter tick, *Dermacentor albipictus*, is a one-host tick found commonly on moose (*Alces alces*), elk (*Cervus elaphus*), and deer. Hunters will encounter this tick (as well as *I. scapularis*) on harvested deer, moose, and elk during the hunting season. Heavy tick infestations can cause anemia and other problems and death of the animal. Larval ticks infest animals in the fall and then develop into nymphs and adults without leaving the host. Engorged females will drop off the host animal in the spring. This tick is broadly distributed from Canada to Central America. This tick will occasionally feed on humans.

Western Blacklegged Tick, *Ixodes pacificus* Cooley and Kohls

Although outside the scope of this handbook, readers should note that the western blacklegged tick, *Ixodes pacificus*, is the principal vector for Lyme disease to humans in the western United States. It looks just like the blacklegged tick in the east and only a specialist could tell them apart. It is found along the Pacific Coast in the western half of Washington and Oregon, almost all of California, and in parts of Utah, Arizona, and New Mexico. Infection rates with *B. burgdorferi* are generally low, 5-6% or less, because many of the immature *I. pacificus* ticks feed on the western fence lizard (*Sceloporus occidentalis*), a reservoir incompetent host for *B. burgdorferi* whose blood also contains a borreliacidal factor that destroys spirochetes in *I. pacificus* nymphs. Several rodents (mainly woodrats) and a nest dwelling tick, *I. spinipalpis*, maintain the enzootic cycle of Lyme disease in California and other western states.

Carios (Ornithodoros) kelleyi Cooley and Kohls

This tick feeds on bats and is found in homes, bat colonies, and other areas where bats may be found. It may occasionally bite humans whose dwellings are infested by bats. Distributed throughout the U.S., records from the northeast include Pennsylvania, New York, and Connecticut.

17

Imported ticks

Travelers abroad have found exotic ticks on themselves after returning to the United States. Other ticks may be imported on pets and other animals. Some of these ticks are potential vectors of pathogens of domestic livestock and introduction and establishment of these ticks would have serious consequences for the livestock industry. For humans, there are a number of bacterial and rickettsial pathogens and encephalitis and hemorrhagic fever viruses carried by ticks in Europe, Asia, Africa and Australia. For example, cases of boutonneuse fever, also called Mediterranean spotted fever, have occurred in travelers returning to the U.S. Boutonneuse fever is distributed through Africa, countries around the Mediterranean, southern Europe, and India. Other spotted fever diseases are African tick-bite fever, Siberian tick typhus, and Queensland tick typhus.

Several tick-borne encephalitis viruses, as well as Lyme disease spirochetes, are transmitted by *Ixodes ricinus* ticks in the British Isles, central and Eastern Europe, and Russia and by *Ixodes persulcatus* from central Europe, Russia, parts of China, and Japan. The following ticks have been documented from traveler's returning to the northeast (destination, origin): *Amblyomma cajennense* (CT, Jamaica), *A. hebraeum* (CT, South Africa), *A. variegatum* (NY, Kenya), *Rhipicephalus simus* (CT, Kenya), *Dermacentor auratus* (ME, Nepal), and *Hyaloma marginatum* (CT, Greece). The Connecticut travelers returning from South Africa and Kenya were physician diagnosed with boutonneuse fever. Tick bite prevention measures should be taken by travelers to potentially tick infested areas abroad. Physicians should consider exotic tick-associated diseases in the differential diagnosis for a patient with a travel with a travel history outside the United States.

18

Amblyomma hebraeum, one exotic species that has been imported into the U.S. Found throughout southern Africa, it is a vector for *Rickettsia conori*, the agent of boutonneuse fever. (J. Occi).

Louse Flies of Deer May Be Confused with Ticks

These flies are tick-like, blood-feeding parasitic flies (family Hippoboscidae), which may be confused with true ticks. The adult flies are dorsally flattened like a tick, with short legs. Several species are common parasites of white-tailed deer in the U.S. and are frequently seen by hunters or others in close association with deer. One species, *Lipoptena cervi* is known as the "deer ked" and was imported from Europe. It occasionally will bite humans. Other "deer keds" are native to the U.S. The female fly retains the larvae, nourishing them internally, and then lays mature larvae, which promptly pupate. The hippoboscid flies associated with deer have wings when they emerge, but lose them once they find a host.

Tick-Associated Diseases

There are at least eleven recognized human diseases associated with ticks in the United States, seven or eight of which occur in the mid-Atlantic or northeastern states. Each of the diseases is highlighted in this section of the handbook. The greatest attention is given to Lyme disease, ehrlichiosis, and babesisois. Although each is a zoonotic vector-borne disease, not all are caused by an infectious agent or are exclusively tick transmitted. A toxin causes tick paralysis, tularemia can be transmitted through contaminated animal tissue or other materials, and babesisois and ehrlichiosis can be transmitted perinatally and through blood transfusion. Tick associations with other pathogens like *Bartonella* or *Mycoplasma* are not yet clearly defined. The causative pathogens transmitted to humans by the tick vector are maintained in a reservoir host. *Ixodes* ticks can be infected with more than one agent and co-transmission and infection can occur. Alternatively, multiple infections can occur from multiple tick bites. In a Connecticut and Minnesota study, 20% of Lyme disease patients also had serological evidence of exposure to another tick-borne agent.

Table 2. Tick-associated diseases in the United States.

Disease	Pathogen or causal agent	Tick Vector
Babesiosis	*Babesia microti, Babesia* spp.	*I. scapularis, I. pacificus*
Colorado tick fever	CTF virus (Retoviridae)	*D. andersoni*
Ehrlichiosis, monocytic	*Ehrlichia chaffeensis*	*A. americanum*
Ehrlichiosis, granulocytic	*Anaplasma phagocytophilum*	*I. scapularis, I. pacificus*
Lyme disease	*Borrelia burgdorferi*	*I. scapularis, I. pacificus*
Southern rash illness	*Borrelia lonestari* (?)	*A. americanum*
Powassan encephalitis	Powassan virus	*I. cookei*
Rocky Mountain spotted fever	*Rickettsia rickettsia*	*D. variabilis, D. andersoni*
Tick-borne Relapsing Fever	*Borrelia* species	*Ornithodoros* species ticks
Tularemia	*Franciscella tularensis*	*D. variabilis, A. americanum*, others
Tick paralysis	Toxin	*D. variabilis, D. andersoni*

Lyme disease, monocytic and granulocytic ehrlichiosis, Rocky Mountain spotted fever, and tularemia are nationally reportable diseases. The amount and quality of surveillance data provided to state health departments and then to CDC is quite variable. Most surveillance is passive, dependent upon physician reporting. Most diseases are greatly underreported. Active surveillance or laboratory-based reporting may also exist in some states or areas. Case reports are based on a standardized surveillance case definition, which is not meant to be the basis for diagnosis. An increase in case reports can represent a true increase in disease or increased awareness of the disease and increased reporting. Conversely, a decrease may represent a change in reporting or a lack of reporting, rather than a true decrease in the incidence of disease. Nevertheless, surveillance case reports generally provide valuable long-term tracking of disease trends and may be useful for targeting intervention strategies.

Lyme Disease

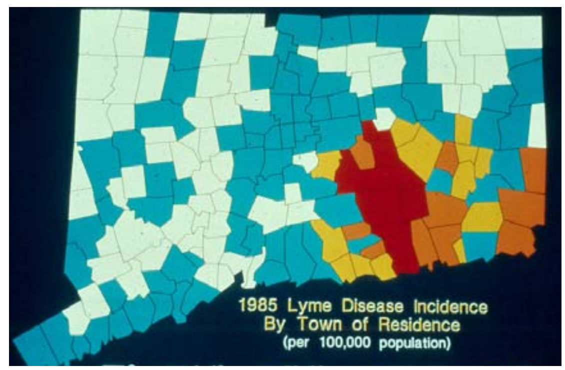
Lyme disease is the leading arthropod-associated disease in the United States and is caused by the spirochete *Borrelia burgdorferi*, a corkscrew-shaped bacterium. It is associated with the bite of certain *Ixodes* ticks, particularly the blacklegged tick, *I. scapularis*, in the northeastern and north-central United States and the western blacklegged tick, *Ixodes pacificus*, on the Pacific Coast. Other *Ixodes* ticks spread the disease in Europe and Asia. The disease has been reported from 49 states, as well as parts of Canada, and across Europe and Asia.

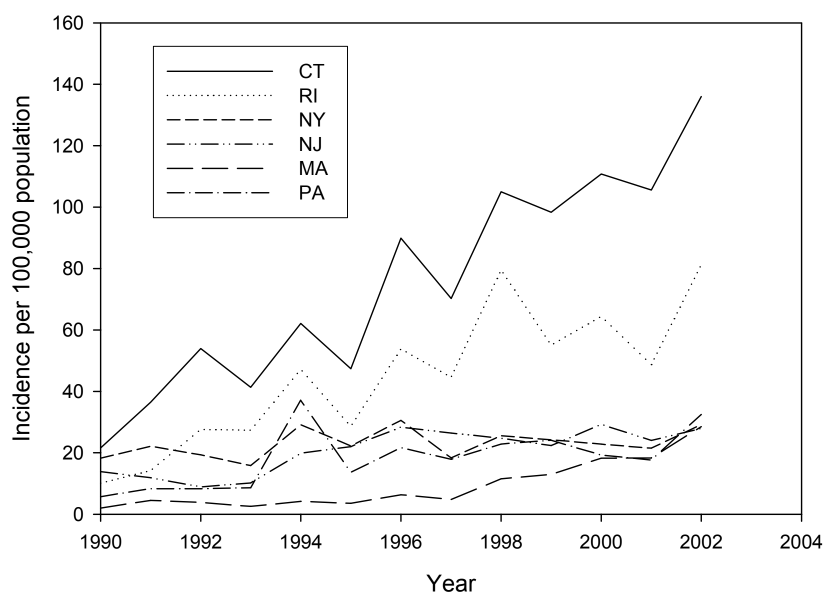
Lyme disease was first recognized as a distinct clinical entity in a group of arthritis patients from the area of Lyme, Connecticut in 1975. In 1981, Dr. Willy Burgdorfer discovered spirochetes in the mid-gut of some *I. scapularis* ticks from Long Island, New York and the bacteria were later named after him. A Lyme disease testing program by the Connecticut Agricultural Experiment Station and Connecticut Department of Public Health found the greatest prevalence in Connecticut in 1984 and 1985 was in towns east of the Connecticut River (map below right). The distribution of the tick and the risk of disease have since expanded dramatically (see map next page). Nationally, 17,739 human cases were reported in 2000, 17,029 cases were reported in 2001 and 23,763 cases were reported in 2002. Twelve states accounted for 95% of reported cases. In order of incidence in 2002 they were Connecticut, Rhode Island, Pennsylvania, New York, Massachusetts, New Jersey, Delaware, New Hampshire, Wisconsin, Minnesota, Maine, and Maryland. Lyme disease is underreported and these numbers may represent only 10-20% of diagnosed cases.

19

The spirochete *Borrelia burgdorferi* (CDC)

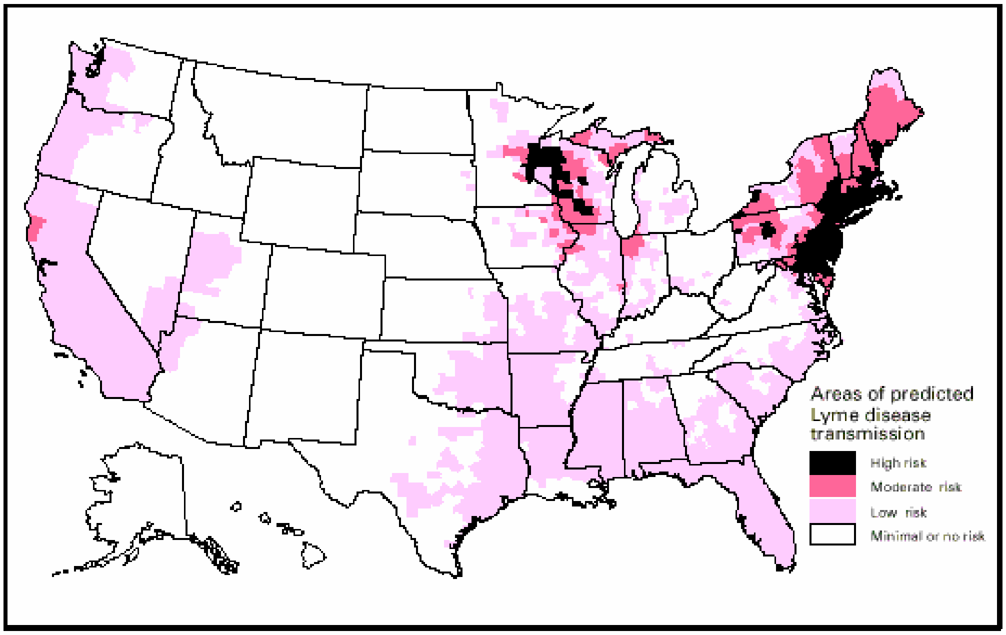
National statistics are available through the CDC website, www.cdc.gov and local statistics may be available through state public health departments or on their websites. Lyme disease affects all age groups, but the greatest incidence of disease has been in children under 14 and adults over 40 years of age. In most cases, Lyme disease symptom onset occurs during the summer months when the nymphal stage of the blacklegged tick is active (see prevention).





Incidence of Lyme disease per 100,000 population by selected northeastern states, 1990-2002. Connecticut and Rhode Island have had the highest incidence of disease, while New York, Connecticut, Pennsylvania, and New Jersey have had the largest number of reported cases.

National Lyme disease risk map with four categories of risk



Note: This map demonstrates an approximate distribution of predicted Lyme disease risk in the United States. The true relative risk in any given county compared with other counties might differ from that shown here and might change from year to year. Risk categories are defined in the accompanying text. Information on risk distribution within states and counties is best obtained from state and local public health authorities.

Clinical signs and symptoms of Lyme disease

Lyme disease is a multisystem disorder with diverse cutaneous, arthritic, neurologic, cardiac, and occasional ocular manifestations. Symptoms that occur within days or weeks following the tick bite reflect localized or early-disseminated infection. Late manifestations become apparent months or years after infection. The major signs and symptoms provided below do not cover all those associated with infection by *B. burgodorferi*. Those who want additional information can consult the literature provided in the bibliography.

Localized infection

20

- Lyme disease is characterized in the majority of patients (70-90%) by an expanding red rash at the site of the tick bite called erythema migrans (or EM). Therefore, the rash serves as a clinical marker for early disease, although the presence of a rash may go unrecognized.
- Erythema migrans may appear within 3 to 30 days (typically 8 or 9 days) after the tick bite. The rash gradually expands over a period of days to a week or more at a rate of ½ to ¾ inch per day and should not be confused with the transient reaction to a tick bite.
- Rashes vary in size and shape, and may occur anywhere on the body, although common sites are the thigh, groin, trunk, and axilla. Many rashes reach about 6 inches in diameter, but some can expand to 8-16 inches or more. The CDC surveillance case definition

specifies that an EM rash must be 2.5 inches or greater in diameter (this definition should not be used as diagnostic criteria).

- An EM may be warm to the touch, but it is usually not painful and is rarely itchy. Swelling, blistering, scabbing or central clearing occur occasionally. The "bull's-eye" appearance usually is noted in less than half the cases and is characteristic of older rashes. The EM will resolve spontaneously without treatment.
- Mild nonspecific systemic symptoms may be associated with the rash in about 80% of cases and include fatigue, muscle and joint pain, headache, fever, chills, and stiff neck. These flu-like symptoms may occasionally occur in the absence of an identified rash and be identified as 'summer flu.' Respiratory or gastrointestinal complaints may occur, but are infrequent.


21


22

Below: Lyme rash (EM) 5 days (left) and 10 days (right) on antibiotic treatment. The rash on the left is the same as above. The rash right is the same EM illustrated on the previous page.

23



24

Early disseminated infection

Lyme disease spirochetes disseminate from the tick bite site through the skin, lymph, or blood to various organ systems, particularly skin, joint, nervous or cardiac tissue. Signs and symptoms may be intermittent, migratory and changing. Nonspecific viral-like symptoms generally mark early-disseminated infection and up to a fourth of patients may develop multiple secondary rashes. Days or weeks after the bite of an infected tick, migratory joint and muscle pain (also brief, intermittent arthritic attacks), debilitating malaise and fatigue, neurologic or cardiac problems may occur. The symptoms appear to be from an inflammatory response to active infection. Multiple EM, headache, fatigue, and joint pain are the most common clinical manifestations noted in early-disseminated disease in children. Multiple components of the nervous system can be affected by *B. burgdorferi*. Early neurologic symptoms develop in about 15% of untreated patients and these can include Bell's palsy (paralysis of facial muscles), meningitis (fever, stiff neck, and severe headache), and radiculoneuropathy (pain in affected nerves and nerve roots, can be sharp and jabbing or deep). Children present less often with facial palsy and more commonly with fever, muscle and joint pain, and arthritis (primarily the knee). Carditis (various degrees of heart block) and rhythm abnormalities may occur in 8% or less of patients. Ocular manifestations may include conjunctivitis and other inflammatory eye problems. Antibodies to *B. burgdorferi* are usually detectable in tests during these manifestations.

25

Late disseminated infection

A year or more after the bite of an infected tick, symptoms of persistent infection in untreated or inadequately treated individuals may include numbness or tingling of the extremities, sensory loss, weakness, diminished reflexes, disturbances in memory, mood or sleep, cognitive function deficits, and an intermittent chronic arthritis (typically swelling and pain of the large joints, especially the knee). Approximately 50-60% of untreated individuals develop arthritis and about 10% of these will progress to chronic arthritis. Attacks of arthritis may last weeks to months with remissions and relapses over a period of several years.

The course and severity of Lyme disease is variable. Mild symptoms may go unrecognized or undiagnosed and some individuals may be asymptomatic (no early illness). The EM rash or subsequent arthritic, cardiac or nervous system problems may be the first or only sign of Lyme disease. Most symptoms eventually disappear, even without treatment, although resolution may take months to over a year. The disease can also be chronic and debilitating with occasional permanent damage to nerves or joints, but is rarely, if ever, fatal. Chronic Lyme disease or post-Lyme disease syndrome is a controversial and unclear constellation of symptoms related to or triggered by infection with *B. burgdorferi*. Disease persistence might be due to a slowly resolving infection, residual tissue damage, inflammation from remains of dead spirochetes, immune-mediated reactions in the absence of the spirochete, co-infection with other tick-borne pathogens, or an alternative disease process that is confused with Lyme disease.

Diagnosis and treatment of Lyme disease

26

A physician should be consulted if Lyme disease is suspected. In the absence of an EM rash, Lyme disease may be difficult to diagnose because its symptoms and signs vary among individuals and can be similar to those of many other diseases. Conversely, other arthritic or neurologic diseases may be misdiagnosed as Lyme disease. Lyme disease is probably both over-diagnosed and under-diagnosed with some patients without Lyme disease convinced they have it and other patients with the disease being told they do not have it. A diagnosis of Lyme disease is based primarily on objective clinical findings. A blood test to detect antibodies to Lyme disease spirochetes (serological testing) can aid or support the diagnosis of the disease. Antibodies to *Borrelia* antigens (parts of the bacteria recognized by the immune system) can usually be detected 3-4 weeks after infection. These tests are not reliable enough to be used as the sole criterion for a diagnosis, however, especially during the early stages of the disease. Tests can give false-negative and false-positive results. Newer tests are more specific, greatly reducing false positive reactions. Reliability of the test improves dramatically in the later stages of the disease as serological reactivity increases, although inaccurate results may still occur. Patients with acute or chronic neurologic or arthritic Lyme disease almost always have elevated antibody levels.

Two stage serological testing for Lyme disease is suggested by many public health organizations:

- Stage One: A relatively sensitive first test by enzyme-linked immunosorbent assay (ELISA) or indirect fluorescent antibody (IFA) test. If negative, no further testing is done. Testing at the time of the Lyme disease rash is unnecessary as many will be negative. Antibiotic treatment early in infection may abrogate the antibody response. An ELISA test provides a quantitative measure antibody levels (measurable color reaction) and for rapid testing of large numbers of samples. An ELISA test measures the reaction to all the antigens in disrupted *Borrelia* or to recombinant antigens, but does not allow identification of which antigens are being bound by antibody and can yield false positives from cross-reactive antibodies. ELISA tests using the C6 peptide of the VslE protein, another protein in *B. burgdorferi* that elicits a strong response by the immune system, may be as sensitive and selective as the two stage testing procedure.
- Stage Two: If the first test is positive or equivocal, a more specific Western immunoblot test is performed to simultaneously demonstrate an antibody response to several *B. burgdorferi* antigens (i.e., proteins recognized by the immune system), which show up as bands on the blot. The Lyme disease spirochete has numerous immunogenic proteins including outer surface proteins (OspA, OspB, and OspC), the 41 kDa antigen on the flagellum, and at least 9 other prominent antigens. The Western blot is labor intensive and requires a subjective interpretation of the results. Although there is an established criterion for a positive blot, there is some disagreement on the number and specific "bands" required for a positive test.

Lyme disease can be treated with one of several antibiotics, including, doxycycline, amoxicillin, cefuroxime axetil, penicillin, ceftriaxone, or cefotaxime. The standard course of treatment is for 14-28 days, depending upon clinical manifestation and drug, though a physician may elect a longer course of treatment. Patients treated in the early stages of the disease usually recover rapidly and completely with no subsequent complications. Oral antibiotics are effective in treating most cases of Lyme disease.

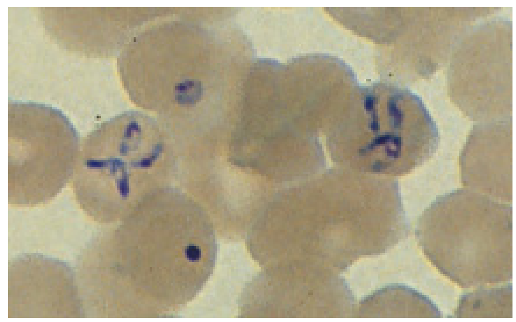
Intravenous antibiotics are indicated for central nervous system involvement and for recurrent arthritis. Full recovery is likely for patients treated in the later stages of the disease but resolution of some symptoms may take weeks even with appropriate treatment. Persistence of some symptoms and inability to determine if the bacteria are eliminated can make decisions on the length of treatment difficult. Courses of antibiotics may have health consequences due to the disruption of the normal intestinal bacteria, allergic reactions, increased sun sensitivity (with doxycycline), gall bladder problems (with ceftriaxone), and infection risks with catheters (extended intravenous antibiotics). Treatment failure may result from incorrect treatment, long delay before treatment, misdiagnosis (not Lyme disease), poor treatment compliance by the patient (did not finish the full course of antibiotics), and infection or co-infection with other tick-borne agents (i.e., *Babesia* or *Anaplasma*). Concurrent babesiosis or ehrlichiosis should be considered in patients with a flu-like illness, particularly fever, chills, and headache, that fails to respond to antibiotic therapy for *Borrelia*. Immunity is insufficient to prevent new infections of Lyme disease with subsequent tick bites that require another course of treatment. Antibody levels generally will decline after treatment, although they may persist for months in some patients after symptoms have resolved.

Southern Tick-Associated Rash Illness (STARI)

A Lyme-like rash has been noted following the bite of the lone star tick, *A. americanum*, in south central and southeastern states and given the name Southern tick-associated rash illness (STARI). The rash is indistinguishable from the rash caused by *B. burgdorferi*. Little is known about this illness. While spirochetes have been observed in about 1-3% of lone star ticks, the bacteria cannot be cultured in the media used for *B. burgdorferi*. A spirochete named *Borrelia lonestari* has been identified in *A. americanum* by DNA analysis and has recently been cultured in tick cell lines.

Human Babesiosis

Human babesiosis is a malaria-like illness that is caused by protozoa found in the red blood cells of many wild and domestic animals. Babesiosis is caused by *Babesia microti* in the northeast and upper mid-west United States. *Babesia microti* is a parasite of white-footed mice, as well as voles, shrews, and chipmunks. Other species or variants of *Babesia* are associated with human disease in other parts of the United States (i.e., California and Missouri), Europe, and Asia. Human babesiosis has been recognized since the early 1970's in parts of Massachusetts (particularly Nantucket Island), Block Island, Rhode Island, and the eastern parts of Long Island, New York. Most cases in Rhode Island are reported from the southern coastal regions. The first Connecticut case of human babesiosis was reported from Stonington in 1988 and the majority of cases continue to be reported from the southeastern portion of that state, although recent evidence indicates that the organism has become more widely distributed in the state. The number of confirmed cases has increased in New Jersey in recent years, which may represent increased risk or increased awareness. The disease is reportable in only a few states. The number of reported cases is probably only a small fraction of clinically diagnosed cases with many other subclinical or mild cases going undetected and unreported. Nevertheless, the distribution and number of cases of babesiosis appears to be increasing.


27

Babesia microti in red blood cells (CDC).

Table 3. Number of reported human cases of babesiosis in select northeastern states, 1997-2001 (compiled from state health department web sites or reports).

State	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
CT	31	45	40	52	56	69				
RI	2	6	18	35	27					
MA	19	66	51	8	18					
NY	26	107	61	72	-					
NJ	3	7	3	15	19					

The white-footed mouse is the primary reservoir for *B. microti*, which is transmitted by *I. scapularis*. While data on the prevalence of infection in *P. leucopus* and particularly in *I. scapularis* is limited to a few studies, babesial parasites have been observed in up to 41% of mice and over 90% can be serologically positive in endemic areas. Infection in mice may be life long. Infections in ticks generally appear to be lower than with *B. burgdorferi*, but in highly endemic areas tick infection by *Babesia* species may be equally prevalent. Maintenance of the parasite seems to require moderate to high tick densities. Most human cases occur during the summer months when nymphs of the blacklegged tick are active. *Babesia* can also be transmitted through blood transfusions from asymptomatic donors.

White-footed mouse with *I. scapularis* ticks.

Both the mouse (or other reservoir competent rodent host such as the meadow vole) and the blacklegged tick are required to complete different aspects of the *Babesia* lifecycle. Larval or nymphal ticks acquire the babesial parasites when feeding on an infected mouse. In the tick gut, male and female gametes unite to form zygotes. Subsequently a stage of the parasite reaches the salivary glands and become dormant until the tick feeds again. The parasite is passed to the next stage of the tick (transstadial transmission). Upon tick attachment, infectious sporozoites are formed and shed in the saliva of the tick. It may require as many as 54 hours of attachment before transmission occurs. Adult *I. scapularis* also can transmit the parasite. During transmission, the sporozoites enter red blood cells, reproduce asexually, and emerge to invade new cells, destroying the infected cells in the process and causing the symptoms associated with babesiosis. Introduction of *B. microti* into another mouse perpetuates the cycle. A female tick does not transmit this parasite to her eggs (transovarial transmission).

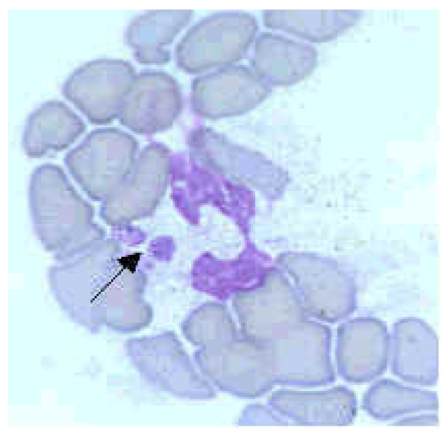
The clinical presentation of human infection ranges from subclinical to mild flu-like illness, to severe life-threatening disease. Infection often is accompanied by no symptoms or only mild flu-like symptoms in healthy children and younger adults. The disease can be severe or fatal in the elderly, the immune suppressed (HIV infection or use of immunosuppressive drugs), and people without spleens. The greatest incidence of severe disease occurs in those older than 40 years of age. Symptoms of babesiosis include fever, fatigue, chills, sweats, headache, and muscle pain beginning 1-6 weeks after the tick bite. Gastrointestinal symptoms (nausea, vomiting, diarrhea, abdominal pain), respiratory symptoms (cough, shortness of breath), weight loss, dark urine, and splenomegaly also may occur. Complications such as acute respiratory failure, congestive heart failure and renal failure have been associated with severe anemia and high levels of infected cells (parasitemia). Up to 80% of red blood cells can be infected in a splenectomized patient, although 1-2% parasitemia is typical in those with intact spleens. Illness may last weeks to months and recovery can take many months. Co-infection with *B. microti* and *B. burgdorferi* can result in overlapping clinical symptoms, a more severe illness, and a longer recovery than either disease alone.

A specific diagnosis of babesiosis can be made by detection of the parasites in Giemsa-stained blood smears and confirmed serologically by an indirect fluorescent antibody (IFA) test. A complete blood count (CBC) is useful in detecting the hemolytic anemia and/or thrombocytopenia (decrease in blood platelets) suggestive of babesiosis. Elevated liver function tests may be present. The parasite can also be detected by polymerase chain reaction (PCR) assay. The drugs of choice in the treatment of babesiosis are oral clindamycin plus quinine sulfate or a combination of oral azithromycin and atovaquone. Adverse effects (i.e., tinnitus, vertigo, lower blood pressure, gastrointestinal upset) are commonly associated with clindamycin and quinine use and some patients cannot tolerate the treatment. The combination of azithromycin and atovaquone is better tolerated. Severely ill patients should be given intravenous clindamycin and quinine and an exchange blood transfusion. Following drug treatment, the parasites usually are eliminated and there is no recurrence of disease. In immunocompromised individuals, parasitemia may persist for months and possibly years following recovery from illness and relapse may occur. Currently, individuals who have ever been diagnosed with babesisois are excluded from donating blood.

Human Ehrlichiosis

The Ehrlichiae are a group of bacteria with several genera and species known to cause disease in dogs, cattle, sheep, goats, horses and humans. These bacteria invade different types of white blood cells (leucocytes) and the disease is often named from the primary type of infected blood cell, including granulocytes or monocytes. Veterinarians have known about canine ehrlichiosis, caused by *E. canis* and transmitted by the Brown dog tick since 1935. Two principal forms of ehrlichiosis in humans currently are recognized in the United States. Human monocytic ehrlichiosis (HME) is caused by *Ehrlichia chaffeensis*. Human granulocytic ehrlichiosis (HGE) is caused by *Anaplasma phagocytophilum* (some cases by *Ehrlichia ewingii*) and accounts for about two-thirds of all ehrlichiosis cases in the U.S. Surveillance for ehrlichiosis in most states is sparse. Ehrlichiosis was added to the national list of reportable diseases in 1999. In Connecticut, there were 544 confirmed cases of HGE reported from 1995-2002. Cases were distributed across all eight Connecticut counties. In New York, both HGE and HME have been reported mainly from the lower Hudson River Valley and eastern Long Island.

Human granulocytic ehrlichiosis was first described from patients in Wisconsin and Minnesota in 1994. The blacklegged tick is the principal vector for HGE (or technically Anaplasmosis) in the northeastern and upper mid-western states. Therefore, most cases of HGE have been reported from states where Lyme disease is highly endemic, particularly Connecticut, New York, and parts of Minnesota and Wisconsin. The western blacklegged tick is the vector in northern California. Laboratory studies indicate transmission can occur within 24 hours of tick attachment and possibly within 8 hours. A single tick has been demonstrated to simultaneously transmit both *B. burgdorferi* and *A. phagocytophilum*.

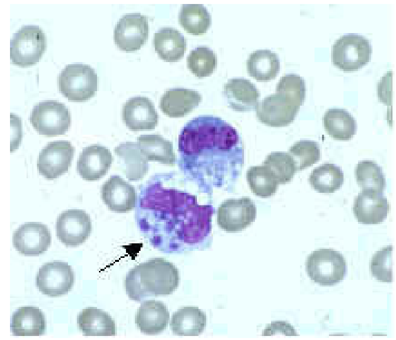


28

Morulae of *A. phagocytophilum* in cytoplasm of neutrophil (CDC).

Human monocytic ehrlichiosis, caused by *E. chaffeensis*, was first recognized in the United States in 1986 in a patient who was bitten by a tick in Arkansas. Lone star ticks are the vector for *E. chaffeensis* in south central and southeastern regions of the country where most cases of HME occur. The DNA of *E. chaffeensis* has been detected in lone star ticks from Connecticut and Rhode Island, so cases of HME may occur in southern New England.

Most cases of ehrlichiosis occur in May, June, or July with 80-90% of cases occurring between April and September. This corresponds to the activity of nymphal *I. scapularis* and adult lone star ticks. Symptoms for both types of ehrlichiosis are non-specific and include fever, headache, muscle pain, nausea, vomiting, and malaise. Initial symptoms appear 5-10 days after the tick bite. Illness may be mild, moderate or severe. Some cases require hospitalization and there have been fatalities. A rash is uncommon in adults, but a rash has been observed in many HME cases in children. Most patients show a decrease in their white blood cell (leukopenia) and blood platelet (thrombocytopenia) counts and an increase in liver enzymes. The number of clinical cases increases with age. The highest rates have been observed for patients 50 years of age or older. Severe cases and fatalities have been reported across all age groups. HME has been confused with Rocky Mountain spotted fever (RMSF). There are no absolute clinical criteria to distinguish Human monocytic ehrlichiosis from RMSF although patients with HME are much less likely to have a rash (10-15 percent) and are more likely to be leukopenic. A diagnosis of ehrlichiosis should be considered for patients with a flu-like febrile illness and possible exposure to *I. scapularis*. Co-infections by the agents of HGE and Lyme disease have been reported and may result in more severe disease. A diagnosis of ehrlichiosis can be confirmed by a serological test, observing the organism in white-blood cells, culturing the organism, or amplification of the DNA of the ehrlichia organism by polymerase chain reaction (PCR). Tests may be negative in the early stages of disease and are more reliable in specimens obtained during the 3 third week of illness. The drug of choice for the treatment of ehrlichiosis is doxycycline (tetracycline may also be used) and should be started upon suspicion or clinical diagnosis of ehrlichiosis. Response to antibiotic therapy is rapid with fever subsiding in 24-72 hours. The use of doxycycline in children under 8 years of age is generally not recommended because it may stain the permanent teeth, but could be used in severe cases. Rifampin has been used successfully when doxycycline cannot be used.

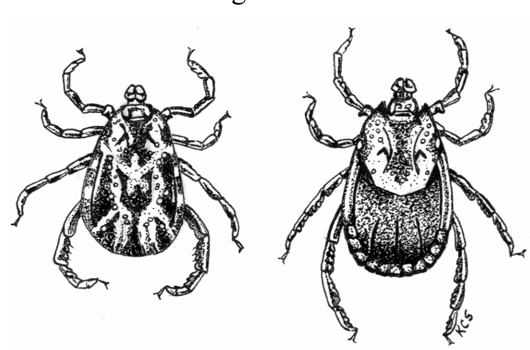


29

Morulae of *E. chaffeensis* in cytoplasm of monocyte (CDC).

Rocky Mountain Spotted Fever

Rocky Mountain spotted fever (RMSF) is caused by *Rickettsia rickettsii,* a type of bacterium that occurs throughout the continental United States, southern Canada, Mexico and Central America and parts of South America. Although the disease was first recognized in 1896 from virulent cases in Idaho and Montana, the name is somewhat misleading as only a small proportion of current cases are reported from the Rocky Mountain region. In the U.S., most cases of RMSF occur in the South Atlantic and West Central states. North Carolina and Oklahoma have the highest rates of RMSF accounting for 35% of the total cases reported to the CDC during 1993-1996. The majority of RMSF cases are associated with the American dog tick. In the western U.S., the vector is the Rocky Mountain wood tick, *D. andersoni*.



RMSF is relatively uncommon in New England. Between 1997 and 2002, based on figures in the CDC's Morbidity and Mortality Weekly Report (MMWR), approximately 3,520 human cases were reported in the United States, of which 28 (less than one percent) were from New England. More cases of RMSF are reported from the mid-Atlantic states, but these still accounted for only 6.7% of the total. Few ticks are infected. Scientists at the Connecticut Agricultural Experiment

Station found that less than 1% of 3,000 American dog ticks examined in Connecticut had spotted fever-group organisms, and not all spotted fever group rickettsiae are infectious to humans.

Table 4. Number of reported human cases of Rocky Mountain spotted fever in northeastern states, 1997-2002. (Data compiled from MMWR and/or state health department web sites; 2002 numbers provisional. One case was reported from New Hampshire in 2001).

State	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
CT	3	2	0	0	0	0	3			
RI	1	0	4	0	0	4				
MA	1	0	2	2	2	6				
NY	14	13	14	9	4	18				
NJ	9	12	7	12	9	2				
PA	16	13	18	25	20	20				
US total	409	365	579	495	695	961				

Children are particularly at risk for RMSF with two-thirds of the cases in patients under 15 years of age. Like Lyme disease, the highest rate in children is in the 5 to 9 year old age group. Symptoms usually appear within 2 to 9 days after a tick bite. Patients experience a variety of symptoms including sudden fever (90%), severe headache (89%), muscle pain (83%), and rash (78%). The rash may begin as small, pink, non-itchy spots (macules) and then develop into the spotted (petechial) rash characteristic of RMSF. The rash may include the palms (50%) and soles of the feet. The rash may not be present or faint when a physician initially examines a patient. Some patients (10-15%) never develop a rash. The classic spotted rash of RMSF appears after about six days or later. Prompt antibiotic treatment with doxycycline, tetracycline, or chloramphenicol for suspected cases is important because RMSF is fatal in 15-20% of untreated cases. Delays in diagnosis because of the absence of the rash or no knowledge of a tick bite could be dangerous. RMSF is made more severe with inadvertent use of sulfonamides. In recent years, about 1-4% of cases in the U.S. have been fatal. A clinical diagnosis may be confirmed serologically or by PCR, but antibodies may not yet be present when a patient is seen by a physician early in the illness.

Below: Examples of spotted fever rash (CDC). Left to right: early (macular) rash on sole of foot, late (petechial) rash on palm and forearm, and rash on hand of a child.

Tick Paralysis

A toxin produced by certain *Dermacentor* ticks during feeding can cause a progressive, ascending paralysis, which is reversed upon removal of the tick. Recovery is usually complete. Paralysis begins in the extremities of the body with a loss of coordination and inability to walk. It progresses to the face with corresponding slurred speech, and finally shallow, irregular breathing. Failure to remove the tick can result in death by respiratory failure. Cases appear more frequently in young girls with long hair where the tick is more easily overlooked. Most cases of tick paralysis are caused by the Rocky Mountain wood tick (*Dermacentor andersoni*) in northwestern states. The American dog tick has also been known to cause tick paralysis.

Tularemia

The bacterium, *Francisella tularensis*, that causes tularemia (Rabbit Fever or Deer Fly Fever) is transmitted by the bite of several species of ticks or bites from deer flies. Ticks associated with tularemia include the American dog tick, *D. variabilis*; lone star tick, *A. americanum*; and Rocky Mountain wood tick, *D. andersoni*. Most cases occur during the summer (May-August), when arthropod transmission is common. The disease also may be contracted while handling infected dead animals (particularly while skinning rabbits), eating under cooked infected meat, or by an animal bite, drinking contaminated water, inhaling contaminated dust, or having contact with contaminated materials. Natural reservoirs of infection include rabbits, hares, voles, mice, water rats, and squirrels. Tularemia was removed from the list of reportable diseases after 1994, but was reinstated in January 2000 because of its potential as a bioterrorism agent.

Tularemia occurs throughout the United States. There have been fewer than 200 cases reported each year during the first half of the 1990s and again in 2000 and 2001. Most cases have been reported from the central states of Missouri, Arkansas, and Oklahoma. With the exception of outbreaks of pneumonic tularemia on Martha's Vineyard that appear related to gardening, landscaping or mowing activities that may have stirred up contaminated dust, reports of this disease are not common in New England, although sporadic cases and outbreaks may occur. There have been pneumonic cases resulting from accidentally running over a rabbit with a lawnmower.

30

All persons are susceptible to tularemia. The clinical symptoms of tularemia depend upon the route of infection. With infection by a tick, an indolent ulcer often occurs at the site of the bite with occasional swelling of the regional lymph nodes. Fever is the most commonly reported symptom. Diagnosis usually is made clinically and confirmed by an antibody test. Antimicrobials with demonstrable clinical activity against *F. tularensis* include the fluorinated quinolones such as ciprofloxacin as well as streptomycin and gentamicin. While tetracycline or chloramphenicol also may be used, they are less effective and relapses occur more frequently.

Powassan Encephalitis

Powassan virus, a *Flavivirus*, is the sole member of the tick-borne encephalitis (TBE) group present in North America. The disease is named after a town in Ontario, Canada where it was first isolated and described from a fatal case of encephalitis in 1958. Documented cases of Powassan encephalitis (POW) are rare, but the disease may be more common than previously realized. While there were only 27 known cases in North America between 1958-1998 (mainly in Canada and New York state), four additional cases were identified in Maine and Vermont from 1999-2001 as a result of increased testing for West Nile virus. The ages of these recent New England cases ranged from 25 to 70 years. Previously, the latest recognized symptomatic cases occurred in New York in 1978 and Massachusetts in 1994. POW presents as meningitis or meningoencephalitis progressing to encephalitis with fever, convulsions, headache, disorientation, lethargy, with partial coma and paralysis in some patients. The disease has a fatality rate of 10-15% and may result in severe long-term disability in the survivors. The principal tick vector appears to be *Ixodes cookei* with cases occurring from May through October.

Patients generally have a history of tick bite, or a history of exposure to tick habitat or exposure to hosts such as squirrels, skunks, or woodchucks. The blacklegged tick is a competent vector of Powassan virus in the laboratory. A virus very closely related to and apparently a separate subtype of the Powassan virus has been isolated from *I. scapularis*, but the prevalence and public health significance of this virus is unknown.

Tick-borne Relapsing Fever

Soft ticks of the genus *Ornithodoros* transmit relapsing fever, caused by *Borrelia hermsi*, or a group of tick-adapted species of the spirochete. Disease is characterized by cycles of high fever and is treated with antibiotics. Relapsing fever ticks are found in rodent burrows, nests, and caves through the western United States. They can live for many years without feeding. Human cases are often associated with people staying in shelters or cabins infested with these ticks. Relapsing fever may be a risk for northeastern residents vacationing or visiting the western U.S..

Colorado Tick Fever

Colorado tick fever, caused by a virus, occurs in mountainous areas of the western United States and Canada. There are 200-400 cases each year. Scientists believe cases are underreported. The virus is transmitted by female Rocky Mountain wood ticks. Symptoms begin with an acute high fever, often followed by a brief remission, and another bout of fever lasting 2-3 days. Other symptoms included severe headache, chills, fatigue, and muscle pain. Illness may be mild to severe, but is self-limited and is not fatal. Treatment is symptomatic. Recovery occurs over several weeks but occasionally may take months.

Bartonella Infection

The genus *Bartonella* includes at least 16 species of vector-associated, blood-borne bacteria that infect a wide variety of domestic and wild animals, including rodents. Several are known human pathogens. For example, *Bartonella henselae*, the agent of cat scratch disease, is transmitted to cats by fleas and generally to humans by bites or scratches from infected cats. The DNA of various *Bartonella* have been found in ticks, including *I. scapularis* and *I. pacificus*, clearly ingested during feeding, but the ability of ticks to transmit these bacteria in the laboratory or field still needs to be determined.

Lyme Disease in Domestic and Companion Animals

Swollen joints in a dog with Lyme disease (31).

Domestic animals (dogs, cats, horses, cows, and goats) can become infected with Lyme disease bacteria and develop clinical disease. Lameness and swollen joints, fever, lymph node enlargement, reduced appetite, and a reluctance to move are the usual symptoms in these animals. Disease is more common in dogs and relatively less frequent in cats. Most dogs in a Lyme disease endemic area will eventually become infected (based on positive serology) due to their high exposure to ticks and some will develop disease each year. Limb and joint arthritis is the most frequent symptom in canine Lyme disease; cardiac, neurological, ophthalmic, and a unique renal involvement is less common. Lyme nephritis in dogs often results in the death of the animal, even with aggressive treatment. Animals are treated with antibiotics (tetracycline or penicillin-group) and nonsteroidal anti-inflammatory drugs for symptomatic relief. Most dogs respond dramatically to antibiotic treatment within days and will make a complete recovery. Chronic disease appears rare and a lack of response to therapy may suggest another diagnosis. Other disease processes, which should be ruled out, include rheumatoid arthritis, infectious arthritides, and other tick-borne diseases such as Rocky Mountain spotted fever and ehrlichiosis. However, studies have shown infection and antibody titers may persist in dogs after efficacious treatment. It is not clear if a reoccurrence of disease is due to another tick exposure or from the initial infection. Some data suggests that treatment in the absence of clinical disease for seropositive dogs or those with a history of tick bite may be indicated. Prevention of disease in companion animals is covered in the host management section.

Additional sources of information about tick-associated diseases

The Centers for Disease Control and Prevention, National Center for Infectious Diseases, Division of Vector-Borne Infectious Diseases, P.O. Box 2087, Fort Collins, Colorado, 80522 and Division of Viral and Rickettsial Diseases, 1600 Clifton Road, NE, MS G-13, Atlanta, Georgia 30333. The CDC-NCID web site (www.cdc.gov/ncidod/index.htm) provides details on the natural history, epidemiology, signs & symptoms, diagnosis, treatment, prevention & control for several zoonotic diseases.

State health departments can provide information on the incidence of Lyme disease and other tick-borne illnesses in the state. There is usually a division or department that handles Lyme disease and other vector-borne diseases. Statistics are sometimes available on a department's web site.

Lyme disease foundations or groups can provide information or patient support. These include the American Lyme Disease Foundation, Inc. (ALDF), Mill Pond Offices, 293 Route 100, Somers, New York 10589 (telephone 914-277-6970, fax 914-277-6974, e-mail: inquire@aldf.com, web site: www.aldf.com) and the Lyme Disease Foundation (LDF), One Financial Plaza, Hartford, CT 06103 (telephone 860-525-2000, hotline 800-886-LYME, e-mail: Lymefind@aol.com, website: www.Lyme.org).

Personal Protection

Tick Bite Prevention

Personal protection behaviors, including avoidance and reduction of time spent in tick-infested habitats, using protective clothing and tick repellents, checking the entire body for ticks, and promptly removing attached ticks before transmission of *Borrelia* spirochetes can occur, can be very effective in preventing Lyme disease. However, surveys and the continuing incidence of disease suggest that few people practice these measures with sufficient regularity. Preventive measures are often considered inconvenient and, in the summer, uncomfortable. Despite the efficiency of tick repellents, particularly with DEET applied to skin and permethrin applied to clothing, they are under-utilized.

Checking for ticks and prompt removal of attached ticks is probably the most important and effective method of preventing infection!

Important points to consider in tick bite prevention and checking for ticks include:

Tick Behavior

- Most Lyme disease cases are associated with the bite of the nymphal stage of the blacklegged tick, of which 10-36% may be infected with Lyme disease spirochetes.
- Nymphal blacklegged ticks are very small (about the size of a pinhead), difficult to spot, and are active during the late spring and summer months when human outdoor activity is greatest. The majority (about 75%) of Lyme disease cases are associated with activities (play, yard or garden work) around the home.
- Adult blacklegged ticks are active in the fall, warmer days in the winter, and in the spring when outdoor activity and exposure is more limited. They are larger, easier to spot, and therefore associated with fewer cases of Lyme disease (even though infection rates are higher).
- Ticks do not jump, fly or drop from trees, but grasp passing hosts from the leaf litter, tips of grass, etc. Most ticks are probably picked up on the lower legs and then crawl up the body seeking a place to feed. Adult ticks will, however, seek a host (i.e., deer) in the shrub layer several feet above the ground.
- Children 5-13 years of age are particularly at risk for tick bites and Lyme disease as playing outdoors has been identified as a high-risk activity. Take notice of the proximity of woodland edge or mixed grassy and brushy areas from public and private recreational areas and playing fields. While ticks are unlikely to be encountered in open fields, children chasing balls off the field or cutting through woods to school may be entering a high-risk tick area.
- Pets can bring ticks into the home, resulting in a tick bite without the person being outdoors. A veterinarian can suggest methods to protect your pets. Engorged blacklegged ticks dropping off a pet will not survive or lay eggs in the house, as the air is generally too dry.

32

33

Prevention

- Wear light-colored clothing with long pants tucked into socks to make ticks easier to detect and keep them on the outside of the clothes. Unfortunately, surveys show the majority of individuals never tuck their pants into their socks when entering tick-infested areas. It is unclear just how effective this prevention measure is without the addition of a repellent. Larval and nymphal ticks may penetrate a coarse weave sock. Do not wear open-toed shoes or sandals.
- Use a DEET or permethrin-based mosquito and tick repellent, which can substantially increase the level of protection (see section on repellents). This approach may be particularly useful when working in the yard, clearing leaves, and doing other landscaping activity with a high risk of tick exposure. A separate set of work or gardening clothes can be set aside for use with the permethrin-based clothing tick repellents.
- When hiking, keep to the center of trails to minimize contact with adjacent vegetation.
- Unattached ticks brought in on clothing can potentially result in a later tick bite. Blacklegged ticks can survive for many days in the home depending upon the humidity. In the laboratory, nymphal *I. scapularis* can survive for over 6 months at 93-100% relative humidity (RH), but over half will die in less than 4 days at 65% RH (RH in modern homes is generally <65%). On returning home, remove, wash and dry the clothing. Many blacklegged ticks and lone star ticks can survive a warm or hot water wash, but they cannot withstand one hour in a hot dryer.
- Carefully inspect the entire body and remove any attached ticks (see below). Ticks may feed anywhere on the body. Tick bites are usually painless and, consequently, most people will be unaware that they have an attached tick without a careful check. Also, carefully inspect children and pets. A hypersensitivity reaction to tick bite may aid detection in a few individuals, but most people will be unaware a tick is attached and feeding.

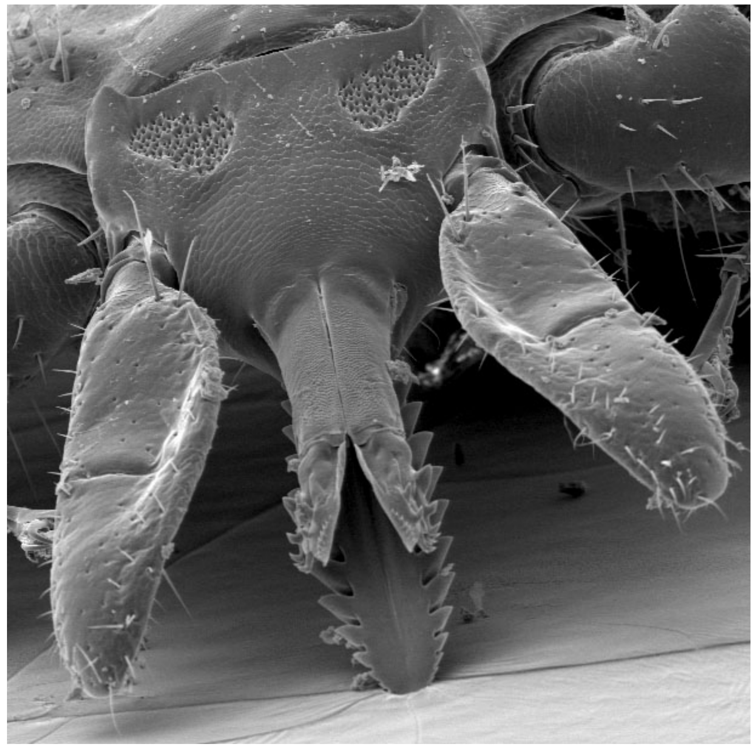
34

Transmission

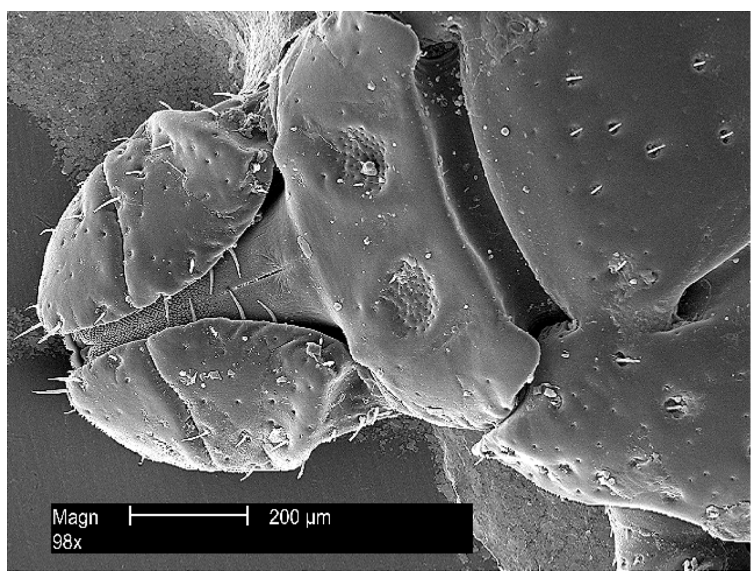
- It takes 36-48 hours or more for transmission of *B. burgdorferi* or *B. microti* to occur from an attached tick and not all ticks are infected. Therefore, a tick bite does not necessarily mean a person will get infected. Prompt removal of an attached tick will reduce the chance of infection. However, transmission of the agent of ehrlichiosis can occur within 24 hours.
- Lyme disease may result from an unrecognized tick rather than the tick that was detected and removed, as the primary Lyme disease rash is sometimes found at a different location than the detected tick. It is not unusual to have more than one tick attached at one time.
- In some areas, tick-testing services for the presence of Lyme spirochetes may be available from a government or commercial laboratory. The detection of spirochetes in a tick does not necessarily indicate transmission and an estimate of risk is difficult without a measure or estimate of length of attachment.

How a Tick Bites and Feeds

The term tick bite may be misleading as ticks do not bite and depart or feed rapidly like a mosquito. Ticks attach and feed gradually over a period of several to many days. Once a tick has found a suitable place to feed, it grasps the skin, tilts the body at a 45-60° angle, and begins to cut into the skin with the paired chelicerae. The palps splay outwards on the skin surface. After the chelicerae and hypostome penetrate the skin, they become encased in "cement" secreted by the tick. The cement serves to hold the mouthparts in place while the tick feeds. Mouthparts on larval and nymphal ticks are small with less penetration and produce a smaller host reaction. Adult *Ixodes* and *Amblyomma* ticks have long mouthparts that can reach the subdermal layer of skin, produce a larger reaction, and make the tick harder to remove. Insertion of the mouthparts often takes around 10-30 minutes, but can take longer (1-2 hours). The reaction to a feeding tick may make the tick appear imbedded, but only the slender mouthparts actually penetrate the skin.



35



36

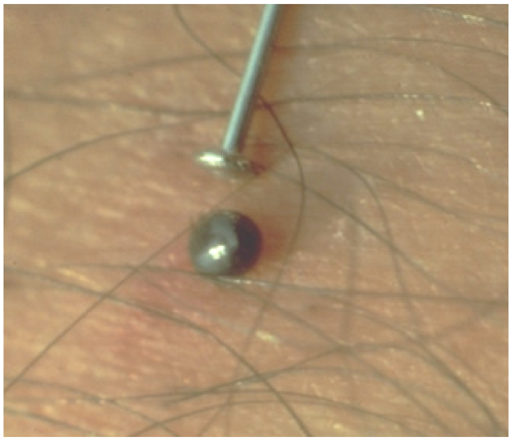
Scanning electron micrographs of the mouthparts of the blacklegged tick (top) and American dog tick (bottom). On the top picture the two palps are spread apart showing the upper two chelicerae and the lower hypostome bracketing the oral cavity.

Physical and enzymatic rupture of tissue creates a lesion or cavity under the skin from which blood is imbibed. A variety of pharmacologically active compounds that aid the feeding process and possibly increase pathogen transmission are introduced in the tick's saliva (e.g., blood platelet aggregation inhibitors, anticoagulants, anti-inflammatory and immunosuppressive agents, enzymes, and vasodilators to increase blood flow). Feeding is not continuous and most of the blood meal is taken up during the last 12-24 hours of feeding. The body weight of a feeding female tick can increase 80-120 times. Male ticks are intermittent feeders, take smaller amounts of blood, and do not change appreciably in size (male *I. scapularis* do not need to feed and are rarely found attached).

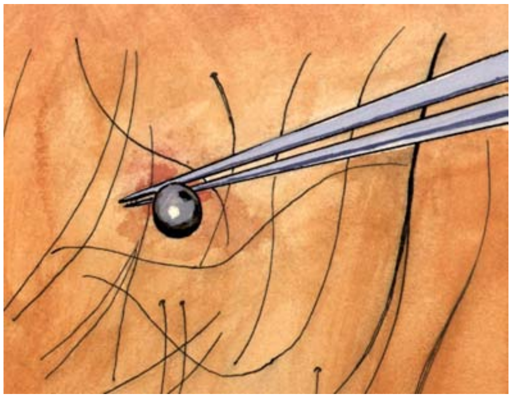
The probability of transmission of Lyme disease spirochetes increases the longer an infected tick is attached (0% at 24 hours, 12% at 48 hours, 79% at 72 hours, and 94% at 96 hours in one recent study). The estimated average time for attachment before detection and removal was 30 hours for nymphs and 10 hours for adult ticks, nymphal ticks were twice as likely as adult ticks to be partially engorged.

Tick Removal

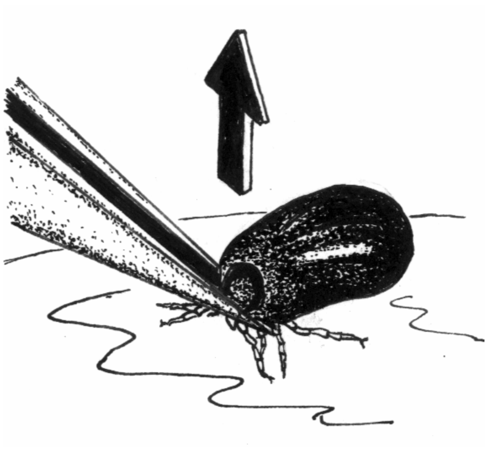
To remove a tick, use thin-tipped tweezers or forceps to grasp the tick as close to the skin surface as possible. Pull the tick straight upward with steady even pressure. This should remove the tick with the mouthparts intact. Commercial tick removal devices have been shown to vary widely in their efficacy for removing nymphal blacklegged ticks: some worked in every attempt, some failed in every attempt, some were in between. Tick removal devices that have been shown to successfully remove *I. scapularis* nymphs attached for 48 hours in all attempts in a recent study include #4 forceps, Original Tick Kit (Tick Kit, Inc.), Pick-Tick (Encepur, Chiron), Pro-Tick Remedy (SCS, Ltd.), and the Nick Nipper (Josyln Designs, Inc.). Squeezing the tick will not increase the risk of infection.



37



The mouthparts of larval and nymphal ticks will seldom be left in the skin. With proper removal, they usually come out intact. Adult ticks are more difficult to remove intact because of the the longer mouthparts. If the mouthparts break off, it will not change the chance of getting Lyme disease. Spirochetes in the mouthparts or cement plug, and therefore the feeding lesion, means the tick was removed too late and transmission has already occurred. Other methods of tick removal (e.g. petroleum jelly to suffocate the tick, heat from matches to make the tick back out or gasoline or other chemicals) are not effective.



After removing the tick:

- Disinfect the area with alcohol or other skin disinfectant; a topical antibiotic may also be applied.
- Save the tick for reference and in some cases testing (if available). A live tick can be placed in a crush proof container with a blade of grass to keep it alive (a sealable plastic bag will also work). A small plastic vial is best.

Dead ticks are tested by DNA methods and should be held dry in a crush proof container. For long-term storage, ticks are held in 70-80% ethyl alcohol (rubbing alcohol will work). Avoid placing ticks in black film containers or using cellophane tape to mount the tick to paper, a note card or a slide if it needs to be identified or tested. Ticks under cellophane tape are difficult to handle. If the tick is removed by a health professional, ask to keep the tick for future reference or testing.

- Note the site and date of the bite.
- Watch for signs and symptoms of Lyme disease or consider prophylactic treatment if tick is engorged or infected (see below). Watch for evidence of secondary infection.

Localized tick bite reactions develop rapidly and can sometimes resemble a small Lyme disease rash, but these transient reactions generally disappear in 24-48 hours and do not continue to expand like an EM rash. Mouthparts left in the skin may cause irritation as the body attempts to absorb or reject the foreign tick tissue (analogous to a minute splinter that is difficult to remove) with a slight risk of secondary bacterial infection. A foreign body granuloma may persist for weeks, especially if the mouthparts remain. A physician should be consulted if there is evidence of infection.

Tick Bite Prophylaxis

The prophylactic use of antibiotics following a tick bite has not generally been recommended by most medical authorities in the U.S. as the chance of Lyme disease from a known tick bite with *I. scapularis* appears low (< 5%; 0% with flat ticks, 10% with engorged ticks in one study). Only 14-32% of patients diagnosed with Lyme disease remember a feeding tick.

Factors against prophylactic (preventative) treatment:

- The infection status and degree of engorgement of the tick, and therefore the risk of infection, are generally not known. Routine testing of ticks attached <24-36 hours is not necessary.
- Not infrequently, ticks submitted for identification or testing turn out not to be a tick (i.e., a scab, beetle, spider , etc.) or a tick that is not a vector for Lyme disease (better training of physicians or clinic staff to recognize major tick species is important).
- There may be a risk of an adverse reaction to the antibiotic.

Factors in favor of prophylactic (preventative) treatment:

- A single 200 mg dose of doxycycline within 72 hours following a tick bite can prevent Lyme disease. A single dose is less likely to stain teeth in children or produce adverse reactions.
- For a partly or fully engorged blacklegged tick, the risk of infection may be high. It can equal the prevalence of infection in the tick, which can be as high as 36% for a nymphal tick and greater than 60% for an adult tick.
- If a tick is infected (determined by testing at a competent laboratory) and the tick is engorged, infection by Lyme disease spirochetes is highly likely and treatment may be seriously considered. However, results from tick testing may not be available in time for prophylactic treatment or Lyme disease symptoms may already be evident.

Repellents

DEET

The primary active ingredient in most insect/tick repellents today is DEET (N,N-diethyl-3-methylbenzamide, also known as N,N-diethyl-m-toluamide). DEET is the most effective, broad-spectrum repellent ever discovered, effective against mosquitoes, biting flies, chiggers, fleas and ticks. The U.S. Environmental Protection Agency (EPA) estimates that over one-third of the U.S. population will use a DEET-based product. There are approximately 230 products containing DEET registered with the EPA (e.g., *Cutter*, *Off*, *Repel, Muskol, Ben's, Sawyer, and others*). Products range in concentration from 5% to 100% DEET and are available as an aerosol can, pump spray bottle, stick, lotion, cream, or towelette for application to skin or clothing. The duration of activity increases with the concentration used.

There are few firm guidelines on the concentration a consumer should use. The effectiveness of DEET on the skin is influenced by the concentration of DEET, absorption through the skin, evaporation, sweating, air temperature, wind, abrasion of the treated surface by rubbing or washing and the arthropod for which protection is desired. A recent study found that a repellent with 23.8% DEET provided an average of 302 minutes of protection against mosquitoes. Higher concentrations generally provide longer protection, but increasing the concentration does not proportionally increase protection time. Several controlled-release or extended-release DEET formulations have been developed which decrease skin absorption and increase protection time. These products may provide longer protection similar to products with a higher concentration of DEET. All active ingredients and their concentrations are listed on the product label.

DEET and ticks

DEET will repel ticks and decrease the chances of tick bite, but depending upon the concentration, it may not provide total protection against the blacklegged tick. Not all products with DEET are labeled for ticks. Little is known about the effectiveness of different concentrations of DEET against *I. scapularis*. Concentrations of DEET that might discourage tick attachment may not deter a tick from walking across the skin to unexposed and untreated areas. Some protection against tick attachment appears to come from the oily or creamy nature of some products. When applied to clothes, 30% and 20% DEET was found to be 92% and 86% effective against *I. scapularis*, respectively, but skin applications were reported to be only 75 to 87% effective against crawling ticks in a second study. For blacklegged ticks, DEET concentrations around 30 to 40% will probably be most effective for general use. A recent evaluation of repellent products by Consumer Reports found a 33% DEET cream-based formulation was effective against nymphal *I. scapularis* for at least 9 hours, while 100% DEET kept ticks off for up to 4 hours. Lower concentrations of DEET were also found repellent. The effectiveness of various concentrations of DEET against *I. scapularis*, especially higher (>50%) and lower (<20%) concentrations, needs to be examined more closely under natural use conditions.

Safe Use of DEET

DEET has been used by many millions of Americans for 40 years and the incidence of adverse reactions is low. The Environmental Protection Agency (EPA) conducted a review of DEET and believes that normal use of DEET does not present a health concern to the general population when used according to label directions (Reregistration Decision document available from the EPA). Some allergic, toxic, and neurological reactions to DEET have been reported in medical literature, but toxic encephalopathic reactions are rare. Reported adverse reactions appear to have involved high concentrations of DEET, over application of product contrary to label directions, or ingestion of product. Repeated applications have occasionally produced tingling, mild irritation or contact dermatitis. Important points in the safe use of DEET include:

- Follow the directions and precautions given on the repellent label.
- Apply DEET sparingly to exposed skin, and spray on clothing when possible.
- Do not use DEET under clothing or over cuts, wounds, or irritated skin.
- Use the lowest concentration necessary for protection and estimated time of needed protection. Minimize the use of higher concentrations on the skin. Lower concentrations, such as 10% DEET, will provide approximately 2 hours of protection against mosquitoes (but may be less effective against ticks), while a concentration of 24% will provide about 5 hours of protection against mosquitoes.
- A concentration of DEET up to 30% for adults and children over 2 years of age is the maximum concentration currently recommended by the American Academy of Pediatrics (AAP).
- The AAP does not recommend the use of DEET on children under 2 months of age. Apply sparingly to small children.
- AAP precautions suggest DEET should not be used in a product that combines the repellent with a sunscreen as sunscreens are often reapplied periodically. DEET is not water-soluble and will last many hours. Reapplications of DEET may increase the possibility of a toxic reaction to DEET.
- Apply the product to a child yourself. Repellent on a child's hands can end up in the eyes or mouth.
- Wash the hands with soap and water after applying DEET.
- People with certain skin conditions should be cautious about the use of DEET.
- Wash off the repellent with soap and water when returning indoors.
- DEET generally won't harm cotton, wool or nylon. DEET can damage some synthetic fabrics (acetate, rayon and spandex), plastics (watch crystals and eyeglass frames), and car and furniture finishes.
- If you suspect a reaction to DEET (or any other repellent), stop using the product, wash the treated skin, and call a poison control center or contact your physician.

Permethrin-based Repellents

Several products contain 0.5% permethrin (e.g., *Duranon Tick Repellent, Repel Permanone, Sawyer's Permethrin Tick Repellent, Sawyer's Clothing Insect Repellent,* and others), which is for use only on clothing or other fabrics such as mosquito netting or tents. A synthetic pyrethroid insecticide rather than a true repellent, permethrin works primarily by killing ticks on contact with the clothes and can provide high levels of protection against ticks (and mosquitoes). Permethrin is available as an aerosol spray or pump, mainly in lawn and garden centers or sports stores. Permethrin has a relatively low mammalian toxicity, is poorly absorbed through the skin and is quickly metabolized and excreted by the body, although the EPA does list it as a potential carcinogen. Permethrin can cause mild skin and eye irritation, but reactions appear uncommon. Important points in the safe use of a permethrin repellent include:

38

- Follow the directions and precautions given on the repellent label.
- Apply to CLOTHING ONLY. Do not apply to skin. Immediately wash with soap and water if you get material on the skin.
- Do not apply to clothing while it is being worn. Apply before you put the clothing on.
- Apply in a well-ventilated area outdoors protected from the wind.
- Lightly moisten the fabric, do not saturate. Allow drying for 2 hours (4 in humid conditions).

- Allow clothing to dry prior to before wearing.
- Do not treat the clothing more than once every two weeks. Launder treated clothing at least once before retreating.

If you suspect a reaction to DEET, permethrin, or any other repellent, stop using the product, wash the treated skin, and call a poison control center or contact your physician.

Botanical and Other Repellents

Botanical products generally provide less protection time against mosquitoes than DEET or permethrin and, though information is limited, are likely to be even less effective against ticks. Many of these products are not labeled for ticks and do not make tick protection claims. Non-DEET products may contain compounds like IR3535 (ethyl butylacetylaminopropionate), or botanical oils such as 0.05% to 15% citronella, 2% soybean oil, or some other plant oil (i.e. eucalyptus, peppermint, lemongrass, geranium or cedar). Consumer Reports found that IR3535 repelled *I. scapularis* nymphs for 3-4 hours, and among plant oils tested, only the soybean oil product offered reasonable protection against mosquitoes (it is not labeled for ticks). Botanical repellents, even if they might reduce tick attachment, probably will not stop a tick from walking across the skin to an unprotected area. Avon's Skin-So-Soft Bath Oil, a widely used folklore mosquito repellent protects against mosquito bites for less than 10 minutes and is unlikely to deter ticks.

Medical and safety information about the active ingredients in an insect repellent is available from:

National Pesticide Information Center by telephone (1-800-858-7378) from 6:30 a.m. to 4:30 p.m. Pacific Standard Time or 9:30 a.m. to 7:30 p.m. Eastern Standard Time, 7 days week, except holidays.

Human Lyme disease vaccine

The Food and Drug Administration (FDA) approved a human Lyme disease vaccine, LYMErix™ (GlaxoSmithKline), which contained recombinant outer-surface protein A (OspA) of *B. burgdorferi*, in December 1998. However, the manufacturer took the vaccine off the market in February 2002 because of declining sales. In clinical trials, vaccine efficacy was 49% after 2 doses for those with definite Lyme disease and 76% after the third dose. Protection in an immunized individual was provided when levels of antibody to OspA in the blood were high enough to neutralize the spirochetes inside a feeding tick before transmission occurred. Protection in vaccinated individuals will wane after a year or two, so protection against Lyme disease in previously vaccinated people will be low to nonexistent. No human Lyme disease vaccine is currently available in the U.S. at this time.

Integrated Tick Management

Integrated pest management (IPM) basically involves the selection and use of several methods to reduce, rather than eliminate, a pest population with expected ecological, economic, and sociological costs and benefits. For ticks, this may involve the use of landscape practices to reduce tick and host animal habitat adjacent to the home, management or treatment of host animals, targeted applications of least-toxic pesticides to high-risk tick habitat – all in conjunction with tick checks and other personal protective measures to either reduce the number of infected ticks and number of tick bites. The ultimate goal, of course, is to reduce the number of human cases of disease as much as possible with the resources available. A decision has to be made on how much one is willing to spend and what ecological impact one is willing to tolerate to reduce the risk of a tick-borne illness. An integrated management approach does not necessarily preclude the use of pesticides, for example, but seeks to use chemicals effectively and responsibly in order to minimize and reduce exposure and use. Research and computer models have shown that pesticides are the most effective way to reduce ticks, particularly when combined with landscaping changes that decrease tick habitat in often-used areas of your yard.

Tick Distribution and Creating a Tick Safe Zone in the Residential Landscape

Tick abundance is related to landscape features of the suburban residential environment that provide a suitable environment for the tick and its animal hosts, particularly white-tailed deer and white-footed mice. While there is a lot of variation in tick numbers between homes, larger properties are more likely to harbor ticks because they are more likely to have woodlots. The blacklegged tick is found mainly in densely wooded areas (67% of total sampled) and ecotone (22%), which is unmaintained transitional edge habitat between woodlands and open areas. Fewer ticks are found in ornamental vegetation (9%) and lawn (2%). Within the lawn, most of the ticks (82%) are located within 3 yards of the lawn perimeter particularly along woodlands, stonewalls, or ornamental plantings. Tick abundance in manicured lawns is also influenced by the amount of canopy vegetation and shade. Groundcover vegetation can harbor ticks. Woodland paths also may have a high number of ticks, especially adults, along the adjacent grass and bushes.

39

The lawn perimeter, brushy areas and groundcover vegetation, and most importantly, the woods, form the high-risk tick zone. The idea for residential tick management is to create a tick managed area around your home that encompasses the portions of the yard that your family uses most frequently. This includes walkways, areas used for recreation, play, eating or entertainment, the mailbox, storage areas and gardens. Tick management strategies are summarized in Table 3 and some actions to consider in an integrated management approach are listed under Table 3.

Table 3. Tick management strategies for the control of *Ixodes scapularis*.

Personal protection	Tick-bite prevention, tick checks and tick removal.
Landscape management	Vegetative modifications to render the environment less suitable for tick survival and for tick hosts.
Management of host abundance	Exclusion of hosts by fencing and host reduction by management of the habitat.
Host-targeted acaricides	Treatment of white-footed mice, chipmunks or deer through passive topical application devices.
Area application acaricides	Spraying chemical insecticides to control ticks.
Biological control	Use of fungal pathogens as biopesticides to control ticks (not yet available at the time this was written, see section on biological control).

- Keep grass mowed.
- Remove leaf litter, brush and weeds at the edge of the lawn.
- Restrict the use of groundcover, such as pachysandra in areas frequented by family and roaming pets.
- Remove brush and leaves around stonewalls and wood piles.
- Discourage rodent activity. Cleanup and seal stonewalls and small openings around the home.
- Move firewood piles and bird feeders away from the house (see section on small mammals and birds).
- Manage pet activity, keep dogs and cats out of the woods to reduce ticks brought back into the home.
- Use plantings that do not attract deer or exclude deer through various types of fencing.
- Move children's swing sets and sand boxes away from the woodland edge and place them on a wood chip or mulch type foundation.
- Trim tree branches and shrubs around the lawn edge to let in more sunlight.
- Adopt hardscape and xeriscape (dryer or less water demanding) landscaping techniques with gravel pathways and mulches. Create a 3-foot or wider wood chip, mulch, or gravel border between lawn and woods or stonewalls.
- Consider areas with decking, tile, gravel and border or container plantings in areas by the house or frequently traveled.
- Widen woodland trails.
- Consider host products to kill ticks on deer or rodent hosts.
- Consider a pesticide application as a targeted barrier treatment.

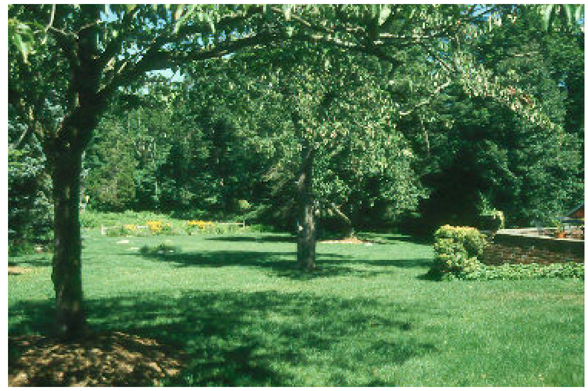
Landscape management

40

Residential landscapes are designed for a variety of aesthetic or environmental reasons and “tickscape” practices can be a part of the landscape in Lyme disease endemic areas. Landscape modifications can create an environment unattractive to primary tick hosts and may decrease the abundance of ticks that are present in parts of the yard. Fewer ticks have been found on well-maintained lawns, except on areas adjacent to woodlands, stonewalls, or heavy groundcover and ornamental vegetation. This section provides some ideas on how to incorporate tick management into the landscape. Clearing leaf litter and woodchip barriers have been documented to help reduce ticks on the lawn. However, landscape practices to create a lower risk tick zone will not directly eliminate many ticks and you may need to consider integrating other tick control practices into the overall program. Landscape work may also be expensive, not acceptable to some residents, and must be done by residents on their own property. In computer simulations of a hypothetical community of 10,000 individuals, a 90% habitat reduction on lawns, 80% habitat reduction in ecotone, and 10% reduction in forested areas by nearly half the residents resulted in the prevention of only a moderate number of Lyme disease cases in comparison to the application of acaricides or treatment or removal of deer. Landscape management alone may not reduce disease incidence, as the undetected bite of only one infected tick is required for transmission of *B. burgdorferi*.

Woodland edge and leaf litter are high-risk areas for nymphal blacklegged ticks!

In most cases, alterations will be made to an existing landscape, although landscape architects and designers should also incorporate tick safe landscaping concepts into major renovations or new construction. There are several basic interrelated concepts in modifying the landscape to create an area with fewer ticks and environmentally acceptable management practices.



- Open up to direct solar exposure the part of the landscape used or traveled frequently by family members to reduce tick and small mammal habitat and cover. Bright, sunny areas are less likely to harbor ticks.
- Isolate areas used by the family or public (i.e., lawns, play areas, recreational or ball fields) from tick habitat or tick hot spots (i.e., woods, dense vegetation, groundcover, stonewalls).
- Use hardscape and xeriscape landscaping (i.e., brick, paving, decking, gravel, container plantings, low water requirement plantings) in areas immediately around the house that are frequently used.
- In cases where environmentally acceptable alternatives to large tracts of open lawn or only small lawn areas are desired, consider butterfly gardens, vegetable gardens, formal herb gardens, colonial style gardens, wildflower meadows and hardscapes. Elimination of woodland and all wildlife habitats is not necessary or environmentally desirable. Some evidence suggests a lack of biodiversity and a landscape that specifically favors deer and mice increases tick abundance and transmission of *B. burgdorferi*. The key factor appears to be the presence and abundance of deer.

Reducing tick habitat

Altering the landscape to increase sunlight and lower humidity may render an area less hospitable to ticks. Management of the habitat should focus on the areas frequently used by the family, not necessarily the entire property. To reduce ticks adjacent to homes, prune trees, mow the lawn, remove leaf litter accumulations around the house and lawn perimeter, and cut grass, weeds, and brush along edges of the lawn, stonewalls, and driveways. Plants can be pruned to provide open space between the ground and base of the plant. Individual shade trees, with the exception of fruit trees like crab apple that are attractive to deer, and small ornamental stands in the open lawn will probably not contribute to the tick numbers unless surrounded by groundcover.

A. Yard before landscape intervention.

B. Yard after landscape intervention.

Ticks also may be found in groundcover such as *Pachysandra*. Restrict the use of groundcovers to less frequently used areas of the yard. Clean up the vegetation around or even seal stonewalls near the house. The removal of leaf litter has been shown to reduce the number of *I. scapularis* nymphs on some properties. Mowing and removing cover vegetation around the house also will discourage rodent hosts. Leaf litter and other plant debris can be raked or blown out from under shrubs and bushes (use tick protection measures while handling leaf litter). Composting or removal by appropriate bagging is acceptable method of disposing of leaf litter. Leaves should not be simply moved to another part of the property. Some communities will compost collected leaves and provide the compost to residents for free or a nominal charge.

Move swing sets and playground areas out or away from the woodland edge!

Play activity can be a high-risk activity for tick exposure and children have some of the highest rates of Lyme disease.

The use of hardscapes, mulches, and xericape landscaping techniques can help reduce tick habitat and isolate parts of the yard from tick hot spots. Hardscapes refer to nonliving features of the landscape like patios, decks, and paths. Mulches are used to suppress weeds and help retain soil moisture, but can also help reduce tick movement. In the laboratory, untreated landscape landscape stones and pinebark woodchips have been shown to deter tick movement and around homes, a three-foot wide or broader woodchip barrier may help reduce tick abundance on the lawn, although results have been found to vary widely from home to home and from year to year depending upon other factors (i.e. density of woods, amount of shade, initial tick densities). Mulches are often organic materials like bark chunks or shredded bark, but can also be small stones or gravel. Wood chip and tree bark, gravel, or similar landscaping materials between woods or stonewalls and lawn as a buffer or barrier can help reduce the number of ticks on the lawn and delineate the tick zone. Quality of the landscape material may also influence results as wood chips from chipped trees, especially if it contains leaves, quickly degrade and may soon be no different than leaf litter. Properly maintained each year, the barrier may allow fewer ticks to migrate from the woodlands into the lawn. It also serves as a reminder that people who cross the barrier may be at higher risk of getting ticks. The application of a barrier or buffer will be easiest where there is a sharp delineation between the woods and lawn. A pesticide application can be focused on the landscape barrier or buffer zone to increase the effectiveness of the barrier. Move swing sets and sandboxes away from the woodland edges and place on a covering of smooth bark, mulch or other suitable material.

Xeriscaping is the application of water conserving landscape practices. This approach reduces habitat cover; helps isolate frequently used areas, can provide an attractive focal area in the yard or garden and reduce maintenance and water, fertilizer, and chemical use. Many drought resistant plants are also deer resistant. Landscapes can incorporate formal or informal designs around play, eating, or pool areas. Landscape materials such as laid brick, wood decking, stone paving, raked gravel or pea gravel (set down slightly from bordering bricks, stone, or paved areas), and concrete (exposed aggregate can provide varying attractive colors and textures and edged with brick or tile) can be used to create a patio and paths. Gravel can be laid over a layer of crushed stone covered with black plastic to discourage weed growth. Some plantings can be in raised beds or containers.

Management of Host Animals

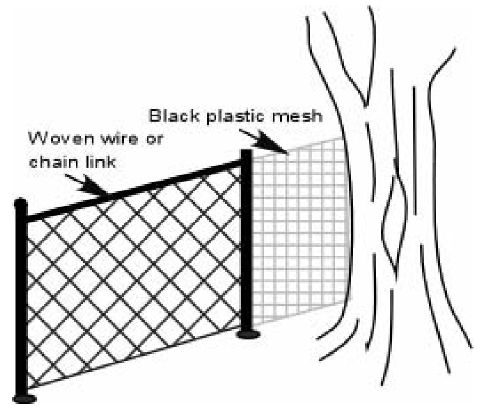
Food and shelter are essential requisites for wildlife. The residential landscape can be particularly attractive to white-tailed deer and conducive to mice, both important hosts in the prevalence of ticks and Lyme disease. One component of a tick management strategy is managing deer and small rodent activity in your yard. Some landscaping practices discussed in the previous section can also help manage key animals in the landscape. Stonewalls, woodpiles, and dense vegetation can harbor rodents.

White-tailed Deer, *Odocoilius virginianus* (Zimmerman)

41

In the northeast from New Jersey to Maine, the deer population is estimated at 1,918,000 animals. In Connecticut, the number of deer has increased from about 12 in 1896 to over 76,000 today. Overabundance of deer is associated with problems such as deer/vehicle collisions, agricultural damage, lack of forest regeneration, detrimental impacts on other wildlife (especially birds), damage to residential landscapes, and the rising incidence of Lyme disease. The fault is not in the animal. Who has not appreciated the thrill of a glimpse of these animals in the meadow or grazing in our landscapes? The problem is in their numbers. There only need be fewer of them. Mature, shaded forests with poor forage and browse support low densities of deer and fewer ticks. A mosaic of light fragmented woodland and woodland edges, clearings and abundant shrubs, berries, grass, and forbs and a lack of predators are ideal for deer. Fencing out deer can allow greater landscape options favorable to other wildlife.

The abundance and distribution of *I. scapularis* has been directly related to the size of the deer population. It has been estimated that over 90% of adult ticks feed on deer. Therefore, deer are key to the reproductive success of the tick. Deer transport blood-engorged female ticks into the property where they can lay thousands of eggs, increasing the number of larval ticks available to feed on small animals. Reservoir incompetent, deer do not infect feeding ticks with Lyme disease bacteria. Larvae of *I. scapularis* pick up the spirochetes when they feed on small animals, especially mice, which are reservoir competent hosts. Island or peninsular communities with extremely high deer densities have superabundant tick populations. Conversely, islands without deer do not appear to support *I. scapularis* or *B. burgdorferi*. Deer management options include deer fencing, repellents, and deer resistant landscape plantings. Dogs also may help deter deer, but to be effective the animal may have to be active both day and night, something a family pet may not do.

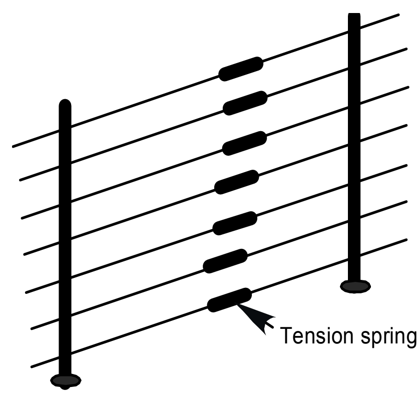


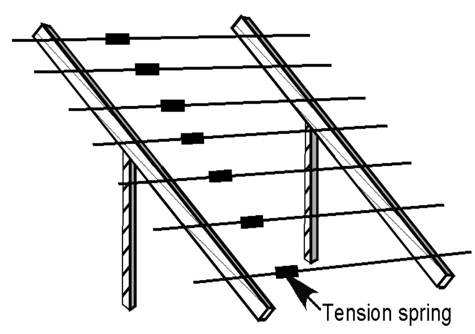
Deer Fencing: Fencing is the most effective method to control access by deer to a property. Fences can keep deer from large garden beds or small to moderate sized home lots. The exclusion of deer from areas of 15 to 18 acres with a slant high-tensile electric fence was shown to reduce the abundance of *I. scapularis* nymphs by as much as 84% and larval ticks by 100% approximately 70 yards or greater inside the fence. Fencing of smaller areas also may be beneficial, but tick management practices within the enclosure and the use of an insecticide at the fence perimeter may also be needed. A deer fence does not stop small animal movement and tick movement. Barrier fencing can be used to protect individual trees, shrubs or other plantings from deer.

There are many types of deer fences and selection will depend upon deer pressure, area to be protected, and site characteristics. The most common choice in a fence is a plastic or wire mesh vertical fence. An electric fence is another option. A number of companies specialize in providing deer fencing and can provide the fencing materials or install the fence. However, many communities have local restrictions or ordinances on the type and height of fencing allowed – check with your local authorities.

42

Non-electric fence – The fence may be vertical or three-dimensional. A vertical fence requires the least space and a wide variety of fence materials and designs are available. Increasingly, a black polypropylene plastic fence-like mesh or steel mesh is being used instead of a chain-link for vertical fences because of reduced cost, low maintenance, long life, and near invisibility, an attractive feature in the residential landscape. The plastic material comes in rolls of various lengths and 7.5 feet wide and can be fastened to existing trees or several different types of posts. White flags should be attached at around 4 feet to signal the presence of the fence. While deer can jump a vertical fence of eight feet from a standing position, they rarely do so and are more likely to try and push under fencing. Proper anchoring or staking of the fence along the ground is important. Single or multiple electric strands also can be placed along the top of a vertical wire or mesh fence. Another option is a slant deer fence set at an angle of 45 degrees for use in areas with moderate to high deer densities, but it requires more space (about 6 feet of horizontal space). Deer cannot clear both the height and width of the fence and often find themselves under the top outer wire. Solid 5- to 6-foot fences are also effective. Access gates, driveway gates (can be remotely controlled in more expensive systems), or in ground driveway deer grates (similar to cattle guards) will be needed to completely enclose the area and still allow owner and vehicle access.





Electric fence – An electric fence requires maintenance, proper grounding, and may not be appropriate in many residential settings. A vertical or slant seven-strand, high-tensile electric fence is very effective for larger areas where deer densities are high.

Deer Repellents: The use of deer repellents may reduce damage to plants and help defer the animals elsewhere, but by itself will not impact tick abundance unless deer consistently avoid the property entirely. Repellent performance is highly variable depending upon the product (most are either odor or taste-based), rain, frequency of application, and the availability of other food sources for deer. Nevertheless, some repellents are fairly effective with low to moderate deer densities.

Deer Resistant Plantings: Substituting less palatable landscape plants may discourage browsing around the home, reduce damage to ornamental plants and may help make the yard less attractive to deer, though deer will also readily graze on lawns. The use of deer resistant plantings may have no impact on ticks unless deer consistently avoid the property and the use of these plants specifically as part of tick management has not been examined. It simply seems to make sense to make your yard and plantings less attractive to deer.

No plant is completely browse resistant and susceptibility depends upon deer density, food availability, and food preferences, which can vary regionally. Plant selection will depend partly upon the type of terrain you have: a sunny, moist yard, a dry, sunny garden, a dry shady garden, or a wet, shady yard, proximity to streams or ponds and effect desired (e.g., fragrance, foliage color, seasonal color, showy borders, etc.). Use of native shrubs and trees is encouraged and the use of invasive plantings is discouraged. Non-native invasive plants, some of which are very resistant to deer browse damage, can crowd out natives. Examples include Japanese barberry, multiflora rose, Asiatic bittersweet, and several non-native honeysuckles. Lists of invasive species and alternative plantings are usually available from state agencies, universities, or environmental groups in each state.

A rating of deer browse damage to many plants was compiled at the Connecticut Agricultural Experiment Station (CAES) from a survey of Connecticut gardeners. A comprehensive list of the survey results with plants ranging from very susceptible to highly resistant to browse damage is available in CAES Station Bulletin 968 (online at www.caes.state.ct.us). Information is also available on deer resistant plantings and deer proofing from a variety of books and handouts. Many nurseries and garden centers can provide a suggested list of deer resistant plantings.

Groundcovers like pachysandra and myrtle, while browse resistant, have been found to harbor ticks and may not be the most appropriate choice near heavily used areas around the house, porch, or mailbox. In general, ornamental grasses and ferns are browse resistant and may be good choices in sunny and moist shady areas, respectively. A number of medicinal herb varieties, ornamental herbs, and butterfly garden plants are resistant to deer browse. The most browse resistant plantings should be placed at the edges and entrances of the property and the most browse susceptible plants closer to the house or areas frequented by people and pets. Susceptible plants can be surrounded by less palatable species. Clean up fruits and other produce from under trees or crop plants. While eliminating cover like mixed tall grass and brush may help discourage deer from bedding near the home, deer will bed wherever they consider it safe – even open lawn. In a study of tick egg-laying, female ticks from deer were found to survive in field bedding areas and lay eggs from which larvae successfully hatched. However, larval survival in the field was shorter than in the woods and they are less likely to be picked up by a mouse host.

Deer Reduction and Management

Some communities have explored the reduction of deer through regulated hunting or controlled hunts to reduce problems associated with deer overabundance. The incremental removal and virtual elimination of deer has been shown to substantially reduce tick abundance, but observational studies and computer models suggest deer densities must be reduced to very low levels (possibly as low as 8 deer per square mile or less) to interrupt the transmission of Lyme disease. In comparison, typical suburban deer densities along coastal Connecticut have been around 30-60 deer per square mile. With the exception of some islands or peninsulas, the need for such a drastic reduction in deer population to achieve satisfactory control levels may render this strategy unrealistic in many areas. Conversely, unregulated deer populations may lead to steadily increasing tick populations. It is not clear if *I. scapularis* can be maintained on medium-sized animal hosts in the absence of deer. Adult ticks also feed on opossums, raccoons, skunks, foxes, dogs and other animals. However, tick densities may be low enough to interrupt the enzootic cycle and transmission of *B. burgdorferi* to humans.

Lethal management options for deer are effective, though controversial, while the use of anti-fertility agents is experimental and labor intensive. A community that wishes to implement a deer management program, especially in densely populated urban and suburban areas must deal with hunting restrictions, real or perceived safety or liability concerns, and conflicting attitudes on managing wildlife. Since most land in the northeast is privately held, homeowner views and hunter access are important to deer management. Any deer population control program would require an initial reduction phase to lower high densities of deer and a maintenance phase to keep the deer population at the desired targeted level. Deer capacity for reproduction is high and deer herds can potentially double in size in one year. Management would be an ongoing process.

Host-Targeted Chemical Tick Control for White-tailed deer

43

The U.S. Department of Agriculture has developed a new experimental approach for the application of topical acaricides to white-tailed deer to kill ticks feeding on the deer. It consists of a feeding station with four paint rollers that hold the pesticide. Deer self treat as they brush against the rollers when they feed. These 4-posters were evaluated in the northeastern United States for the control of the blacklegged tick, having performed well in a trial against lone star ticks on deer in Texas. Computer models indicated that 95% control of *I. scapularis* on 90% of a local deer population could dramatically reduce the tick population in a treated area over a period of several years. While usage of the devices is generally high (> 90%), utilization of the devices by deer was extremely low when alternative food sources were available (i.e., acorns). The treatment of deer with 2% amitraz reduced tick abundance in the treated communities by around 64-69% by the fifth and sixth year in comparison with untreated areas. The use of 10% permethrin resulted in a 91-100% reduction of larval, nymphal, and adult questing ticks in sampled plots. According to computer simulations, this approach, in principal, could provide the greatest reduction in Lyme disease with the least direct community involvement (i.e. number of direct participating households).

The American Lyme Disease Foundation (Somers, NY) holds the license to the product's patent and works with Dandux Outdoors (Ellicott City, MD) for manufacturing the device. The U.S. Environmental Protection Agency has registered 10% permethrin as a restricted use, ready to use tickicide (Y-TEX® 4-Poster™ Tickicide, Y-TEX Corporation, Cody, WY) for application to deer via the 4-poster device to control *I. scapularis* and *A. americanum*. The 4-posters are to be placed as far as possible, but in no case less than 100 yards from any home, apartment, playground, or other place children might be present without adult supervision. States may have more restrictive requirements than the federal label. At the time of this writing, state pesticide registrations have been obtained in 33 states including Connecticut, Massachusetts, Rhode Island, New Jersey, Maine, New Hampshire, Vermont, Maryland, Delaware, Michigan, and Minnesota. Approvals or regulations for use by state wildlife officials are pending or under review. The use of the 4-poster will probably make the most sense as part of a neighborhood or community coordinated program to reduce ticks and the risk of Lyme disease, managed under state use regulations, combined with some form of a deer management program.

Small Mammals and Birds

Rodents and birds can infect ticks with *B. burgdorferi* and transport ticks onto your property. The importance of these animals in the dynamics of Lyme disease depends on the abundance of the animal host, number of ticks feeding on the host, and the host's ability to infect feeding ticks with the Lyme disease spirochete (i.e., the reservoir potential). In other words, what animals are contributing infected ticks to your property? Some animals may have a lot of ticks, but not be able to infect them with spirochetes.

Rodents

While different rodent and bird species may predominate in certain years and locations, white-footed mice, *Peromyscus leucopus*, are generally the most abundant and efficient animal reservoir for the Lyme disease bacteria. They contribute more infected ticks than eastern chipmunks or meadow voles. White-footed mice also are a reservoir for the causal agents of ehrlichiosis and babesisois. Over 90% of white-footed mice will be infected with *B. burgdorferi* in many areas and up to half have been found to carry all three pathogens in some areas. In one study, a single mouse was estimated to infect as many ticks as 12 chipmunks or 221 voles. Meadow voles, *Microtus pennsylvanicus*, are most abundant in fields, pastures, orchards, which harbor few *I. scapularis*. Although they harbor fewer ticks, short-tailed shrews, *Blarina brevicauda*, with their high reservoir potential, may contribute to the maintenance of both *B. burgdorferi* and *B. microti* in some areas, especially when mouse numbers are low. By contrast, squirrels have a lower Lyme disease reservoir potential. One study indicated that squirrels might reduce or dilute the number of infected ticks in the landscape.

Note in the second picture of the white-footed mouse the engorged larval ticks feeding on the ears and around the eyes of the animal. Larval ticks become infected with B. burgdorferi and other pathogens while feeding on an infected mouse or chipmunk.

White-footed Mouse
Peromyscus leucopus (Rafinesque)

44

The white-footed mouse is the principal animal carrying the pathogens that cause Lyme disease, human anaplasmosis (i.e., ehrlichiosis) and human babesisois. White-footed mice are found throughout most eastern and Midwestern United States, except in Florida and northern Maine. This mouse is difficult to distinguish from the deer mouse, *P. maniculatus*.

White-footed mice have a home range of generally 0.1 to 0.5 acre, sometimes larger. This woodland and brushy area dwelling animal nests in stonewalls, tree cavities, abandoned bird or squirrel nests, under stumps, logs, and may readily enter and nest in buildings, especially during the winter months. Mice may line the nest with fur, feathers or shredded cloth. These nocturnal animals are omnivorous and feed on acorns, seeds (including newly planted gardens), fruits, insects, snails, tender young plants, and carrion.

Mouse densities usually are around 1-10 per acre but can be higher (15 per acre) and may go relatively unnoticed until they enter homes that are not rodent proof. Breeding spring through fall, a female mouse typically has 3-4 young after a gestation period of 22-25 days. The mice reach sexual maturity in 6-7 weeks. There are no ticks on the mice during the winter and, inside buildings, they do not pose a risk for the transmission of Lyme disease. Folded hardware cloth (1/4-inch mesh) may be used to exclude mice from buildings, flowerbeds, and garden plots. Cleaning up small black droppings and urine-contaminated areas in confined areas can pose a risk for hantavirus disease.

Reduction of small mammal abundance should focus mainly on reducing mouse habitat near homes and encouraging predators like foxes, snakes, hawks, and owls, and weasels, to name a few. However, predators require large territories of several square miles. Although not quantified, this author has noticed mouse populations drop dramatically (based on trapping success) with resultant drops in the tick population at sample sites where a fox family or snakes have taken up residence in or near the stone walls. Mice have relatively small home ranges. Dense vegetation and ground cover plants like pachysandra adjacent to homes provide cover for rodents as they forage for food. Shaded stonewalls overgrown with grass and brush can harbor many mice and chipmunks.

Eastern Chipmunk
Tamias striatus L.

Eastern chipmunks are found in most states east of the Mississippi River, except along the southeastern coastal region. They are often the second most important rodent in the maintenance of Lyme disease and can be the principal reservoir in some areas. Solitary by habit and active during the day, chipmunks feed on seeds, grains, fruits, nuts, bulbs, mushrooms, insects, carrion and may prey on young birds and eggs. They can climb trees to gather seeds, fruit and nuts and store food throughout the year. They hibernate during the winter, but may become active for brief periods on sunny warm days. Requiring ample vegetative cover, chipmunks are found in deciduous woodlands with undergrowth, old logs, stonewalls, and in brushlands. Their home range is small, typically less than 100 yards in diameter and females defend a 50-yard radius around the home. A small (2 inch), inconspicuous entrance leads to a complex burrow system. There are typically 2 to 4 chipmunks per acre, but densities may be higher with adequate food and cover. There are 1 or 2 litters each year. Hardware cloth (1/4-inch mesh) may be used to exclude chipmunks from buildings and flowerbeds.

A fitted stonewall is unlikely to harbor rodents and ticks like the old stonewall with leaf litter and other vegetative cover.

Birds

Birds are frequent hosts for immature stages of the blacklegged tick. At a woodland residence, 26% of birds were infested with ticks and 94% were *I. scapularis*. While some bird species can infect feeding ticks with *B. burgdorferi* (i.e., American robin, veery, grackle, common yellowthroat, Carolina wren, house wren), other species (i.e., gray catbird, woodthrush) do not. Due to variability in bird species composition, population, habitat preferences, reservoir competence and feeder activity, it is unclear how many ticks (much less those infected with spirochetes) most birds actually contribute to a typical residential landscape. One early study found that American robins, a reservoir competent bird, were likely contributors to the nymphal tick population found in some suburban residential landscapes. Unlike mice, however, reservoir competency in robins declines after 2 months. A recent study suggested most birds probably contribute few infected ticks and may actually dilute pathogen transmission, at least in comparison to mice. Bird feeders in landscaped areas like mowed lawns were not found to be a risk factor for Lyme disease, probably because the habitat does not favor tick survival and seed feeding birds that frequent feeders in the summer do not deposit many ticks. However, higher tick abundance has been noted where feeders were installed at or beyond the lawn edge in wooded habitat suitable for tick survival and rodent activity (Gary Maupin, CDC retired, personal observation). Adult ticks, which are active in the fall, winter and spring months, do not feed on birds.

It is unknown what impact summer or winter fruit bearing trees and shrubs for birds has on the prevalence of ticks as related to mouse and chipmunk activity, as seeds and fruits can also serve as a food source for these animals.

Many berry plants, however, are important to fall migrants and the berries are quickly consumed. Deer resistant bird favorites include bayberry (*Myrica pensylvanica*) and Virginia creeper (*Parthenocissus*) and highbush blueberry (*Vaccinium corymbosum* – produces summer berries); cedars and certain holly cultivars, however, are subject to heavy deer browsing. Common winterberry (*Ilex verticillata*) is also fairly susceptible to heavy deer browse damage. It requires both female and male plants to produce winterberries for birds. Native viburnums will suffer only occasional to minimal damage

45

Note the ticks feeding around the eyes of this veery (J. Occi).

from deer and are good bird plants. Japanese barberry (*Berberis thunbergii*) is considered invasive.

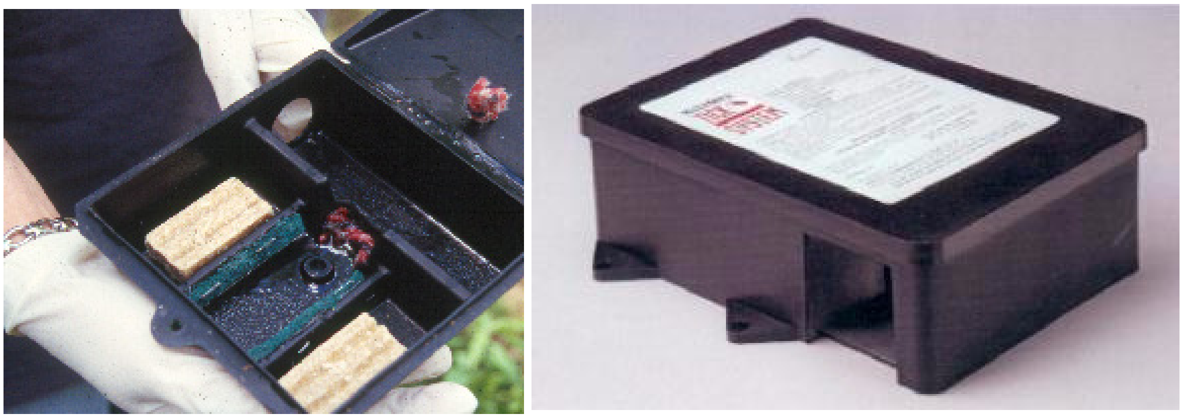
Possible small animal and bird management strategies include:

- Keep potential mouse nesting sites in stonewalls and woodpiles near the residence free of brush, high grass, weeds, and leaf litter.
- Seal or rework stonewalls near or under the home to reduce harborage.
- Move firewood away from the house.
- Place the birdhouses and feeders away from the house, but it is unknown if this will decrease risk of exposure to ticks. Clean up spilled feed (spilled bird feed can also attract mice).
- Set up bird feeders in late fall and winter when natural foods are scarce (and the immature stages of *I. scapularis* are not present on birds).
- Seal foundations. For example a garden shed on cement blocks can harbor raccoons, skunks, or woodchucks. This can be avoided through a proper foundation or use of hardware cloth buried at least two feet beneath the ground. A poorly sealed building or old garden shed can harbor mice.

Host-Targeted Chemical Tick Control for Rodents

The first rodent-targeted product was a cardboard tube of cottonballs treated with the insecticide permethrin (Damminix®). The product is aimed at larvae and nymphs of *I. scapularis* feeding on white-footed mice. The effectiveness of this product is dependent upon the mice collecting the cotton as nesting material from cardboard tubes distributed throughout the mouse habitat. Studies in Connecticut and New York state failed to show any reduction in the number of infected, host-seeking *I. scapularis* nymphs when this product was used for a three year period in woodland and residential areas of about 4 acres or less. Lack of control may be due to failure by the mice in some areas to collect the cotton or the presence of alternative tick hosts, such as chipmunks, an important secondary tick host and spirochete reservoir. Reductions in tick numbers were reported in a Massachusetts study with the treatment of one 18-acre tract.

Another approach, using bait boxes for the topical treatment of rodents with fipronil, was first evaluated for the control of *I. scapularis* on wild white-footed mice on an island community in Connecticut and then subsequently at residential locations in Connecticut, New Jersey, New York and Massachusetts. Fipronil is the active ingredient in topical or spray flea and tick control products (Frontline®). In the laboratory, a single topical application to a mouse can kill all ticks on the animal for up to 7 weeks. In the island community trial, the prevalence of infection of *B. burgdorferi* in the mice dropped dramatically after one year and nymphal tick populations were substantially reduced after only two years of use. A commercial version called the Maxforce® Tick Management System (Bayer Environmental Science, Montvale, NJ) received EPA registration July 2003 and is available through licensed pesticide applicators. The rodent bait box is one alternative to area applied sprays or efforts to reduce the small mammal population. The device consists of a sealed, ready to use, child resistant box containing nontoxic food blocks and an applicator wick impregrated with 0.70% fipronil. This device treats both white-footed mice and Eastern chipmunks as they pass through the box to forage on the food attractant. While high levels of immature tick control may be obtained by treating single, isolated properties, a number of adjacent homeowners may have to use this approach for optimum impact. The impact of the boxes on the tick population accumulates over time. There is no affect on the existing host-seeking tick population the first year the boxes are placed, so a pesticide application may be a consideration in the initial year if the Maxforce Tick Management System is used.



46

A. **B.**

The pictures show an open rodent box used in the initial experimental trials in Connecticut with two non-toxic bait blocks and a yarn wick treated with fipronil (A) and the Maxforce® TMS rodent bait box (B).

Prevention of Tick-Associated Disease in Companion Animals

47

Engorged female *I. scapularis* on a domestic cat.

The prevention of Lyme disease and other tick-associated disease in dogs relies on avoiding tick habitat, reducing ticks on the animal, daily tick checks, and use of one of the canine Lyme disease vaccines available (whole-cell killed bacterin or recombinant outer surface protein A of *B. burgdorferi* - OspA). Electronic fencing systems can help confine a pet in an area where the animal is less likely to pick up ticks or where other tick control measures have been implemented. If the pet is not allowed to freely roam into the wooded areas, it is less likely to pick up ticks. Animals can carry ticks into the home. However, studies to determine whether pet owners may be at increased risk of Lyme disease have been inconclusive. Ticks, once attached or fed, will not seek another host. Dogs and cats should be checked daily for ticks, but the immature stages may be virtually impossible to detect on longhair or dark-hair animals. Outdoor activities with animals also may increase the exposure of pet owners to ticks and their habitat.

48

A veterinarian should be consulted about the prevention and treatment of Lyme disease in your animals. A variety of products can repel and/or kill ticks on the animal. Some are available over the counter (OTC) and others only through your veterinarian. Chemical products to protect dogs from ticks are available as spot-ons, sprays, collars, powders, and dips. Ingredients include several insecticides such as pyrethrin, permethrin, amitraz, or fipronil (see section on chemical control). Some products are combined with an insect growth regulator to help control flea eggs. Follow label directions to minimize the chances for an adverse reaction to the product in your pet and do not combine products without the advice of your veterinarian. Different products can contain the same or similar ingredients, which could result in an overdose of the animal.

Although the risk of clinical disease is low, the canine Lyme vaccines can provide high levels of protection for dogs living in or traveling to Lyme disease endemic areas with a likely exposure to ticks. Depending upon the vaccine, an initial dose can be given as early as 9 or 12 weeks of age with a second required dose several weeks later. An annual booster is recommended.

Backyard Wildlife Programs and Environmentally Friendly Lawns

With increased environmental awareness, the focus for some residents has been to provide a more natural or organic landscape, with reduced inputs of energy, water, pesticides, fertilizer and labor, and provide increased wildlife habitat. Some shrubs and other plants are selected for their wildlife value due to the berries, fruit and cover they provide for birds and small mammals. Many resources are available to help create backyard wildlife habitats. How can the desire to have a more natural, environmentally friendly habitat be balanced with the need to reduce contact with animals carrying ticks and the creation of a tick safe zone? The presence of deer and rodents will result in the presence of ticks. Little information is available on how to integrate these two different objectives. Open lawns harbor fewer ticks and wildlife that carry potentially infected ticks. There is some evidence that increased animal diversity can actually reduce the rate of transmission of tick-associated disease, resulting in fewer infected ticks, although ticks are still present. The fragmented woodland and ecotone environment of suburbia favors the deer, mice, and chipmunks most involved in the maintenance and transmission of ticks and tick-associated diseases. Mixed ecotone with uncut grass, wildflower and shrubby vegetation, especially adjacent to woodlands is good deer, mouse and tick habitat.

Little is known about relative tick densities in various alternative landscapes to turf like wildflower meadows, gardens, and butterfly gardens. It is not known what specific plants or plant groupings may be associated with more or fewer ticks or if it makes that much of a difference. Some plants used in butterfly gardens are attractive to deer, while most herbs are highly resistant to deer browsing. Fencing against deer will allow greater landscape flexibility. While data is limited, meadows appear to harbor few blacklegged ticks except along the edge with woodlands, dense vegetation and stonewall. If a property is large enough, a separate wildlife and tick-managed zone could possibly be maintained. The objective of a tick management program is to discourage activity of several key tick hosts and create a physical and/or chemical barrier between woodland habitat and areas the family uses most frequently.

Area-Wide Chemical Control of Ticks

Insecticides, or as termed for ticks, acaricides, are the most effective way to reduce ticks, particularly when combined with the landscaping changes to decrease tick habitat reviewed earlier in this handbook. They provide consistent control, are relatively easy to apply, and are relatively inexpensive. Only small amounts of an acaricide applied at the right time of year are necessary. Chemical intervention should focus on early control of nymphal *I. scapularis* ticks, the stage most likely to transmit Lyme disease, by spraying once in May or early June. A fall application in October may be used to control adult blacklegged ticks (or in the spring if no fall application was made). Targeting lawn and woodland edges and perimeter areas near tick "hot-spots" or along the "tick zone" can minimize exposure. Some general points to consider if you spray for ticks:

- Applications can be made by the homeowner or by a commercial applicator.
- Spray once in the late spring or early summer for control of *I. scapularis* nymphs. For American dog ticks, an application can be made anytime after the adults emerge in the spring.
- A single application of most ornamental-turf insecticides will provide 85-90% or better control with some residual activity so multiple applications are rarely necessary. Some organic pesticide products are less effective, breakdown rapidly, and multiple applications may be required.
- Treat tick habitat only.
  Spray areas where the lawn meets the woods, stonewalls, or ornamental plantings. Spray several yards into bordering woodlands, area of greatest tick density. Spray groundcover vegetation like *Pachysandra* near the home or walkways. Spray perimeter of areas of the yard often used by people (play areas, gardens, outside storage areas, walkways or paths to neighbors or mailboxes). Avoid herb, vegetable, and butterfly gardens.
- In parks and school athletic fields, restrict any applications to high-risk tick habitat. Spraying of open fields and lawns is not necessary.
- Use a product specifically labeled for controlling ticks. Some products are packaged as fertilizer-pesticide mixtures or mixtures of different pesticides (e.g., herbicide and insecticide).

Acaricides Used for Tick Control

There are several factors that will influence the selection of a specific chemical product. All pesticides sold must be registered with the U.S. Environmental Protection Agency (EPA) and the appropriate state pesticide agency for use within that state.

- The product must be labeled for area-wide tick control (see Table 3). Some products are General Use Pesticides and others are classified as Restricted Use Pesticides for commercial use only, available only to licensed applicators. Some products are labeled for brown dog ticks only or for ticks on surfaces, indoors, as a building foundation or perimeter treatment and are not labeled for use on ornamentals or turf. Check the label and ask for assistance. A licensed commercial applicator often will have a preferred acaricide that is used most frequently.
- The toxicity and environmental impact of the chemical. Chemicals differ in their toxicity to humans, wildlife, aquatic organisms and beneficial insects. While some general information is provided in this handbook, more detailed information can be obtained from sources listed at the end of chemical control section.
- The type of formulation and method of application. Both liquid and granular formulations have been reported effective against *I. scapularis* with somewhat better control usually obtained with liquid formulations. Sufficient spray volume and pressure should be used for thorough coverage and penetration of the vegetation and leaf litter. A small hand pump sprayer is unlikely to provide the coverage needed for good tick control and, at a minimum, some type of garden hose sprayer is suggested. A homeowner who wishes to apply a granular material with a fertilizer spreader for tick control may not be able to treat woodland margins effectively and the product may be labeled for lawn use only.
- Effectiveness in controlling ticks. Blacklegged ticks and American dog ticks are readily killed by almost all ornamental and turf insecticides labeled for tick control. With the withdrawal of the organophosphate insecticides chlorpyrifos and diazinon from residential use (the U.S. Environmental Protection Agency has cancelled registration of these compounds for residential area-wide use), the synthetic pyrethroid insecticides are the most commonly used tick control agents. Pyrethroids are particularly effective at rates 6-45 times less than the now cancelled organophosphate insecticides and the carbamate insecticide carbaryl. In the laboratory, nymphal *I. scapularis* crawling on landscape stones treated with pyrethrin-based desiccants and insecticidal soaps suffered high (> 88%) mortality. However, natural pyrethrin with the synergist piperonyl butoxide provided limited tick control in the residential landscape in several trials. By contrast, synergized pyrethrin was more effective when combined with insecticidal soap or as part of a silicon dioxide (from diatomaceous earth) product. Silicon dioxide acts as a desiccant. Thorough coverage appears particularly important with pyrethrin and insecticidal soap products. With the exception of a desiccant, there is little residual activity. At least two applications may be required.

Table 4. Acaricides with products labeled for the control of ticks in the residential landscape.

Chemical	Some brand or common names*	Chemical type and usage
Bifenthrin	Talstar® Ortho® product	Pyrethroid insecticide. Available as liquid and granular formulations. Products available for homeowner use and commercial applicators.
Carbaryl	Sevin®	Carbamate insecticide. A common garden insecticide for homeowner use, some products are for commercial use only.
Cyfluthrin	Tempo® Powerforce™	Pyrethroid insecticide. Available for commercial and homeowner use with concentrates and ready to spray (RTS) products.
Deltramethrin	Suspend® DeltaGard® G	A pyrethroid insecticide for commercial applicators.
lambda-cyhalothrin	Scimitar® Demand®	A pyrethroid insecticide for commercial applicators.
Permethrin	Astro® Ortho® products Bonide® products Tengard® SFR Others	Pyrethroid insecticide. There are concentrates and ready to spray (RTS) products. Most are for homeowner use, a few are for commercial use only.
Pyrethrin	Pyrenone® Kicker® Organic Solutions All Crop Commercial & Agricultural Multipurpose Insecticide®	Natural pyrethrins with the synergist piperonyl butoxide (PBO) or insecticidal soap provide limited tick control. A combination of pyrethrin and PBO with either insecticidal soap or silicon dioxide (from diatomaceous earth) was found effective against ticks in one trial.

*Active ingredients and brand names frequently change as new products are registered and others discontinued. New formulations for homeowner use may become available. Mention of a product is for information purposes only and does not constitute an endorsement by the Connecticut Agricultural Experiment Station.

Homeowner Application of Acaricides

One option is for the homeowner to make the pesticide application. Anyone applying pesticides to their own property should be familiar with how to read a pesticide label, how to correctly mix the pesticide, and follow the listed precautions in handling and applying the material. The pesticide label provides information on the active chemical ingredients, formulation, pests and sites for which it can be legally used, directions for use, precautions, hazards to humans, wildlife and the environment, and first aid instructions. Always read and follow pesticide label directions and precautions. It is a violation of federal law to use a pesticide in a manner inconsistent with the label. The label will provide an indication of how hazardous a pesticide is by the signal word on the label. Signal words are based on the EPA toxicity class and must be included on pesticide labels.

- Danger-Poison means highly toxic or poisonous through oral or dermal exposure
- Danger means highly toxic, but may include severe skin or eye irritants

- Warning means moderately toxic or hazardous
- Caution means slightly toxic or hazardous
- No signal word means practically nontoxic

Not all brands of a particular pesticide chemical will be labeled for area tick control. Some products may be for application in or on building and their immediate surroundings. Check the label. Homeowner products come in three forms.

- Ready-to-use (RTU) is premixed and applied directly from the existing container. They are used for spot treatments, treatments of individual plants, or treatment of small areas. Some RTU products, for example, are used to control dog ticks indoors or around a dog's bedding. Ready-to-spray (RTS) products are used for treating larger areas. The container attaches directly to a garden hose for automatic mixing of the water with the concentrate. For example, a ready spray of 2.5% permethrin or 0.75% cyfluthrin is available as a hose end sprayer for the control of *I. scapularis* and will cover about 5,000 square feet.
- Concentrates require mixing the product with water and using your own sprayer (pump-up style, hose-end style, or other type sprayer). Homeowner products may contain carbaryl, cyfluthrin, or permethrin.
- Granules are designed for lawn applications with a hand held or broadcast spreader. The chemical is usually released with addition of water, so granules generally must be watered in. Granules for tick control on the lawn may contain bifenthrin or carbaryl.

Appropriate protective gear as directed on the label should be used when applying pesticides. Surveys have shown many individuals fail to take precautions while applying pesticides. Store pesticides in a cool, dry, secure place. Keep them out of the reach of children. An EPA survey found 85% of households had at least one pesticide on the property and 47% with young children (under age 6) stored them within reach of the child. Keep a pesticide in its original container; do not store diluted spray. Either use up the product or properly depose of leftover product through a community household hazardous waste program. Pesticides should never be poured down the sink or toilet. Empty containers should be triple rinsed and placed in the trash. For more information on handling, applying, storing and deposing of pesticides, readers may refer to the EPA's Citizen's Guide to Pest Control and Pesticide Safety (available at www.epa.gov).

Commercial Application of Acaricides

Another option is to have a licensed commercial pesticide applicator apply the acaricide. Most companies offering tick control services are lawn care, landscape, or tree care companies, but may include some pest control operators (PCOs) in some states, depending upon what licenses the operator has obtained. A survey of commercial applicators in Connecticut in the mid-1990s found that about 16% offered tick control services. The application of pesticides for tick control comprised less than 5% of their business for most companies. Nevertheless, most companies reported that tick control business had increased and a few companies have specialized solely in providing tick control. A follow-up survey by the author in 1999 indicated that 53% were now offering tick control services. A number of companies provide organically oriented pest management services.

A company offering commercial application of pesticides must be registered with the state or states in which they conduct business. A pesticide license is required for the commercial application of pesticides or the application of restricted use materials in the area. There must be at least one commercial supervisory pesticide applicator certified in the type of application being made. In Connecticut, for example, a license for ornamental and turf application from the Department of Environmental Protection is required for applying pesticides for tick control in the landscape. Some tree service companies (arborists) also treat for ticks. Although arborists are tested and licensed by the state specifically for arboriculture services, they must also possess an ornamental and turf license to spray for ticks. Consumers should employ individuals who are licensed to spray for ticks and may request to see the license or license number or check with the agency responsible for the state pesticide program to see if the firms are properly registered and licensed. A commercial company should provide a consumer the name of the pesticide product to be used, the active ingredient in the product, the reentry period (the time before family members can safely reenter the treated area), and the form of the pesticide and type of equipment to be used. In most states, companies are required to provide copies of the label and material safety data sheets (MSDS). With this information, additional information can be obtained over the Internet, from local Cooperative Extension offices, state agencies and pesticide alternative groups. Tips on hiring an applicator are available from EPA's Citizen's Guide to Pest Control and Pesticide Safety (available at www.epa.gov). Some general guidelines about a pesticide application that homeowners and commercial applicators should be aware of include:

- Many states (including all New England states, New York, New Jersey, Pennsylvania) have notification laws that require customers or adjacent residents receive written notice prior to an urban pesticide application. Usually this notification is provided only to those who request it through a registry.
- Pesticides should not be applied on windy days (greater than 10 mph) to avoid drift to non-target areas.
- Before the spraying, the windows and doors of the home should be closed.
- Pesticides should be kept away from plants and play areas that you do not want treated. Most tick control pesticides are for ornamental and turf use only and are not labeled for use on plants meant for human consumption. Most of these chemicals are toxic to bees and should not be applied to areas with foraging bees.
- Pesticides should not be applied near (within 25 feet) wetlands (i.e. lakes, reservoirs, rivers, streams, marshes, ponds, estuaries, and commercial fish farm ponds) or near (within 100 feet) coastal marshes or streams. Even organic pesticides are toxic to fish and aquatic invertebrates.
- Family members and pets, especially cats, should be kept off the treated area for 12-24 hours or other specified reentry interval following the treatment (generally until a spray thoroughly dries).
- Do not water the lawn after the application of a pesticide to avoid run off (there are a few exceptions with some granular products which must be watered in). Do not apply within 24 hours of rain to avoid run-off. Once the pesticide has dried, however, some materials bind tightly to the soil or vegetation and do not readily move or wash off. They will breakdown with exposure to sunlight and soil microbes.
- Avoid pesticide applications near a wellhead. The shaft of the well should be tightly sealed and the well water source should be isolated from surface water source. Most acaricides used for tick control are water insoluble and pose little risk to wells by leaching through the soil, but direct exposure should be avoided.
- Many states (including all New England states, New York, New Jersey, Pennsylvania) have laws that require signs to be posted after an urban treatment is made.

An Acaricide Primer

The purpose of this section is to serve as a reference for some basic, general material on the major classes of chemicals used in tick control. More detailed information is available from the EPA, the Cooperative Extension Service, state pesticide agencies, and independent groups, particularly over the Internet. Some sources of information are listed at the end of this section. Acaricides belong to a variety of chemical classes, which differ in their chemistry, mode of action, toxicology, and environmental impacts. They also contain "inert ingredients," chemicals that carry or enhance the application or effectiveness of the active ingredient (i.e., the actual acaricide). A variety of pesticides are also used in products to control ectoparasites on pets. Some pet care products are available over the counter and others through a veterinarian.

- **Organophosphates.** There were two organophosphate insecticides commonly used for area-wide tick control, chlorpyrifos (i.e., Dursban) and diazinon. The EPA has cancelled the residential use and some agricultural uses of chlorpyrifos and has cancelled the registration of diazinon for lawn, garden, and other residential outdoor use. Residential applications accounted for nearly 75% of the use of diazinon. Products with these chemicals are no longer used for tick control.
- **Carbamates.** Carbaryl (Sevin®) is the carbamate used in the control of ticks. Carbaryl is a broad-spectrum compound used for a wide variety of pests on the lawn, on pets, and in the home. Carbaryl in animals is readily broken down and excreted. It does not appear to cause reproductive, birth, mutagenic, or carcinogenic effects under normal circumstances, but it is a suspected endocrine disrupter. Carbaryl is extremely toxic to bees and beneficial insects, is moderately toxic to fish, but is relatively nontoxic to birds.
- **Pyrethrins.** Pyrethrum is a natural insecticide extracted from certain chrysanthemum plants. Natural pyrethrins are a group of six compounds that form the insecticidal constituents of the natural pyrethrum, which is highly unstable in light and air. Natural pyrethrins are considered knockdown agents because they rapidly paralyze insects, but many insects can detoxify the compound and recover. Therefore, pyrethrins are sometimes combined with a synergist. A synergist is a compound that enhances the toxicity of an insecticide, but is not an insecticide itself. The most common synergist used with pyrethrin is piperonyl butoxide, which inhibits the enzymes that breakdown pyrethrin. Pyrethrins also may be combined with insecticidal soaps, spreader sticker agents, silicon dioxide (desiccant) and other agents to enhance the effectiveness of the product. Pyrethrins have little residual effect, being quickly broken down by exposure to light, moisture, and air.
- **Pyrethroids.** Synthetic pyrethroids are derivatives of the natural compounds, chemically modified to increase toxicity and stability. Most of the chemicals used for area-wide tick control are pyrethroids. The pyrethroids are less volatile than the natural compounds and photostable, which provides some residual activity and greater insecticidal activity. Both pyrethrins and pyrethroids are highly toxic to fish and other aquatic organisms, but generally are much less toxic to mammals, birds and other wildlife. Pyrethroids can be skin and eye irritants. Many concentrated pyrethroid formulations are restricted to commercial use by licensed applicators because of their potential impact on aquatic organisms. However, low concentration, ready-to-use products are available for homeowner use.
- **Inert ingredients**. They may be solvents, propellants, spreaders, stickers, wetting agents, or carriers for the active pesticide chemical. Because these compounds are not the active chemical, they are labeled "inert ingredients" or sometimes "other ingredients". These compounds often make up the major part of a pesticide formulation. In some cases, the

inert ingredients may be more toxic than the active ingredient. A few examples of inerts include napththalene, petroleum distillates, and the organic solvents xylene and toluene.

- **Acaricides for control of ticks on pets**. Carbaryl and the pyrethroid permethrin are used in several flea and tick control products for dogs. Studies have indicated that use of permethrin products (i.e., K9 Advantix™, Kiltix®) can prevent the transmission of *B. burgdorferi* and *A. phagocytophilum*. Both are topical products applied to spots along or on the back of the animal. They are not for use on cats, as cats are particularly susceptible to pyrethrin poisoning. Fipronil, a phenypyrazole, is the only commercial insecticide of this chemical type. Formulated pet products are available as a spray or topical spot application (Frontline®, Frontline® Top Spot™, Frontline® Plus) for long-term control of fleas and ticks on dogs and cats. It is the material used in the Maxforce® TMS rodent bait box. Fipronil dissolves in the oils on the skin, spreads over the body, and collects in sebaceous glands and hair follicles for long-term reapplication. It is not affected by bathing or water immersion. Skin irritation may occur. Fleas are killed from 1-3 months, while ticks are killed for about a month. Trizapentadiene or formanidene compounds include one currently used material, amitraz. In livestock, it is used to control ticks, mites, and lice. It is not a skin irritant, is not readily absorbed into tissue, and degrades rapidly in the environment. Amitraz is used in a tick prevention collar for dogs (Preventic®), and one study indicated it could prevent transmission of *B. burgdorferi*. An amitraz product was one of the compounds initially evaluated for the topical treatment of deer to control *I. scapularis*.

Additional sources of information about pesticides

Environmental Protection Agency (EPA) Public Information Center (telephone 202-260-2080), National Center for Environmental Publications and Information (telephone 513-489-8190), EPA booklets or the EPA web site (www.epa.gov).

National Pesticide Information Center (NPIC) (formerly the National Pesticide Telecommunications Network) is a cooperative effort of Oregon State University and the U.S. Environmental Protection Agency (EPA). The toll-free service is staffed 6:30 am – 4:30 pm Pacific time (9:30 a.m. – 7:30 p.m. Eastern time) 7 days week, except holidays (telephone 1-800-858-7378). Information provided by the NPIC includes pesticide information, information of recognizing and managing pesticide poisonings, safety information, health and environmental effects, referrals for investigation of pesticide incidents and emergency treatment information, and cleanup and disposal procedures. Pesticide related fact sheets and other information are available at the web site (http://npic.orst.edu/). Their address is NPIC, Oregon State University, 33 Weniger Hall, Corvallis, Oregon 97331-6502.

Extension Toxicology Network (EXTOXNET) is a cooperative effort of University of California-Davis, Oregon State University, Michigan State University, Cornell University, and the University of Idaho. Primary files are maintained and archived at Oregon State University. Pesticide Information Profiles (PIPs) and Toxicology Information Briefs (TIBs) provide information on pesticide trade names, regulatory status, acute and chronic toxicological effects, signs and symptoms of poisoning, ecological effects and environmental fate, physical properties, manufacturer, and references (http://ace.orst.edu/info/extoxnet/).

State pesticide regulatory agencies can provide information on the laws and regulations governing the application of insecticides, certification of pesticide applicators, and which products are registered for use in the state. Depending upon the state the agency may be associated with the state Department of Agriculture, Consumer Protection, or Environmental Protection.

Organic Landcare Practices

Standards for organic land care practices for design and maintenance of ecological landscapes have been developed and published by the Connecticut and Massachusetts chapters of the Northeast Organic Farming Association (NOFA). Tick IPM practices are covered under pest and wildlife management guidelines. Practices that are preferred to manage ticks would include personal protection measures, making the environment unsuitable for the pest (i.e., landscape modifications), deer resistant plantings (natives recommended), fencing against deer, and herbal-based deer repellents. The use of arthropod pathogens like entomopathogenic fungi (fungi that kill insects), diatomaceous earth, insecticidal soaps and botanical insecticides are allowed under the standards. However, botanicals cannot be formulated with aromatic petroleum distillates. Ammonia or hot sauce based deer repellents are allowed. Prohibited under the organic standards are all synthetic insecticides and piperonyl butoxide as an insecticide synergist, rodenticides containing warfarin, predator urine (due to collection practices), and products containing sewage sludge (e.g., Milorganite).

Biological Control of Ticks

Ticks have relatively few natural enemies, but the use of predators, parasites, and pathogens has been examined for tick control. Tick predation is difficult to document and observations are sporadic. Most arthropod predators are non-specific, opportunistic feeders and probably have little impact on ticks. Anecdotal reports suggested that guinea-fowl or chickens may consume ticks and impact local tick abundance. However, there is no good evidence to support this and turkey foraging was not found to reduce the local density of adult ticks. A minute parasitic wasp, *Ixodiphagus hookeri*, parasitizes blacklegged ticks in a few areas of New England with superabundant deer and tick populations. However, studies indicate that the usefulness of this wasp to control *I. scapularis* is very limited. Insect parasitic nematodes have been studied as possible biological control agents. Engorged female *I. scapularis* are susceptible to certain types of nematodes, but these nematodes are too sensitive to the colder autumn temperatures when the ticks are present. The application of entomopathogenic fungi, however, is a promising approach for controlling ticks. Several fungi have been shown pathogenic to *I. scapularis*. A perimeter treatment of existing commercial formulations of the fungus *Beauveria bassiana* and with *Metarhizium anisopliae* at residential sites has been shown to control *I. scapularis* in small experimental trials. The EPA has approved *M. anisopliae* for residential outdoor grub and tick control (Tick-Ex™, an oil formulation, and Taenure™, a granular formulation; Earth BioSciences, Glastonbury, CT). At the time of this writing, additional trials and commercial development are in progress. Entomopathogenic fungi, applied like a traditional pesticide, may be an option in tick management programs, and an oil-free formulation could meet organic standards.

Lyme disease can be a preventable disease!

Surveys have consistently shown most residents in Lyme disease endemic areas consider the disease an important or very important issue that poses a high risk to members of their family. A few precautions and the management of infected ticks in the residential or recreational landscape can substantially reduce the risk of Lyme disease and other tick-associated illnesses. Prompt recognition of infection and treatment can prevent more serious manifestations of disease. While education is important to preventing or mitigating disease, landscape and host management practices combined with the judicious use of acaricides can provide excellent tick control with minimal risk or impact to the environment.

Selected Bibliography and References

Ticks and Tick Ecology

Anderson, J. F., and L. A. Magnarelli. 1999. Enzootiology of *Borrelia burgdorferi* in the northeastern and northcentral United States, pp. 385-389. *In* G. R. Needham, R. Mitchell, D. J. Horn and W. C. Welborne [eds.], Acarology IX: Volume 2, Symposia. Ohio Biological Survey, Columbus.

Anderson, J. F. 1988. Mammalian and avian reservoirs for *Borrelia burgdorferi* [Review]. Ann. N.Y. Acad. Sci. 539: 180-191.

Balashov, Y. S. 1972. Bloodsucking ticks (Ixodoidea): Vectors of diseases of man and animals. Misc. Pub. Entomol Soc. Am. 8: 159-376.

Battaly, G. R., and D. Fish. 1993. Relative importance of bird species as hosts for immature *Ixodes dammini* (Acari: Ixodidae) in a suburban residential landscape of southern New York state. J. Med. Entomol. 30: 740-747.

Durden, Lance A. and James E. Keirans. 1996. Nymphs of the Genus *Ixodes* (Acari: Ixodidae) of the United States: Taxonomy, Identification Key, Distribution, Hosts, and Medical/Veterinary Importance. Entomological Society of America, Lanham, MD. 95 pp.

Donahue, J. G., J. Piesman, and A. Spielman. 1987. Reservoir competence of white-footed mice for Lyme disease spirochetes. Am. J. Trop. Med. Hyg. 36: 92-96.

Drummond, Roger. 1998. Ticks and What You Can Do About Them, 2nd ed. Wilderness Press. 74 pp.

Ginsberg, Howard S. 1993. Ecology and Environmental Management of Lyme Disease. Rutgers University Press. 224 pp.

IJdo, J. W., C. Wu, L. A. Magnarelli, K. C. S. III, J. F. Anderson, and E. Fikrig. 2000. Detection of *Ehrlichia chaffeensis* DNA in *Amblyomma americanum* ticks in Connecticut and Rhode Island. J. Clin. Microbiol. 38: 4655-4656.

IJdo, J. W., J. I. Meek, M. L. Cartter, L. A. Magnarelli, C. Wu, S. W. Tenuta, E. Fikrig, and R. W. Ryder. 2000. The emergence of another tickborne infection in the 12-town area around Lyme, Connecticut. J. Infect. Dis. 181: 1388-1893.

Keirans, J. E., and L. Durden. 2001. Invasion: Exotic ticks (Acari: Ixodidae) imported into the United States. A review and new records. J. Med. Entomol. 38: 850-861.

Keirans, James E. and Taina R. Litwak. 1989. Pictorial Key to the Adults of Hard Ticks, Family Ixodidae (Ixodida: Ixodoidea), East of the Mississippi. Journal of Medical Entomology. 16(5): pp 435-448.

Keirans, James E. and Lance E. Durden. 1998. Illustrated Key to Nymphs of the Tick Genus *Amblyomma* (Acari: Ixodidae) Found in the United States. Journal of Medical Entomology. 35(4): pp. 489-495.

Lane, R. S., J. Piesman, and W. Burgdorfer. 1991. Lyme borreliosis: Relation of its causative agent to its vectors and host in North America and Europe. Ann. Rev. Entomol. 36: 587-609.

LoGiudice, K., R. S. Ostfeld, K. A. Schmidt, and F. Keesing. 2003. The ecology of infectious disease: Effects of host diversity and community composition on Lyme disease risk. Proc. Nat. Acad. Sci (USA).

Mather, T. N., M. L. Wilson, S. I. Moore, J. M. C. Ribeiro, and A. Spielman. 1989. Comparing the relative potential of rodents as reservoirs of the Lyme disease spirochete (*Borrelia burgdorferi*). Am. J. Epidemiol. 130: 143-150.

Mather, T. N., S. R. Telford III, A. B. MacLachlan, and A. Spielman. 1989. Incompetence of catbirds as reservoirs for the Lyme disease spirochete (*Borrelia burgdorferi*). J. Parasitol. 75: 66-69.

McDaniel, Burruss. 1979. How to Know the Mites and Ticks. Wm. C. Brown Company Publishers. Dubuque, Iowa. 335 pp. (Out-of-Print)

Oliver Jr., J. H., M. R. Owsley, H. J. Hutcheson, A. M. James, C. Chen, W. S. Irby, E. M. Dotson, and D. K. McLain. 1993. Conspecficity of the ticks *Ixodes scapularis* and *I. dammini* (Acari: Ixodidae). J. Med. Entomol. 30: 54-63.

Sonenshine, Daniel E. 1991. Biology of Ticks, Volume 1. Oxford University Press. Oxford. 447 pp.

Sonenshine, Daniel E. 1993. Biology of Ticks, Volume 2. Oxford University Press. Oxford. 465 pp.

Smith, Carroll N., Moses M. Cole, and Harry K Gouck. Biology and Control of the American Dog Tick. USDA Technical Bull. No. 905, January 1946.

Smith, R. P., Jr., E. H. Lacombe, R. W. Rand, and R. Dearborn. 1992. Diversity of tick species biting humans in an emerging area for Lyme disease. Am. J. Public Hlth. 82: 66-69.

Stafford III, K. C., R. F. Massung, L. A. Magnarelli, J. W. IJdo, and J. F. Anderson. 1999. Infection with agents of human granulocytic ehrlichiosis, Lyme disease, and babesiosis in wild white-footed mice (*Peromyscus leucopus*) in Connecticut. J. Clin. Microbiol. 37: 2887-2892.

StaffordIII, K. C., V. C. Bladen, and L. A. Magnarelli. 1995. Ticks (Acari: Ixodidae) infesting wild birds (Aves) and white-footed mice in Lyme, CT. J. Med. Entomol. 32: 453-466.

Stafford III, K. C. 1994. Survival of immature *Ixodes scapularis* (Acari: Ixodidae) at different relative humidities. J. Med. Entomol. 31: 310-314.

Stafford III, K. C. 1993. The epizootiology of Lyme disease. Northeast Wildlife 50: 181-189.

Strickland, R.K., R.R. Gerrish, J.L. Hourrigan, and G.O. Schubert. 1976. Ticks of Veterinary Importance. USDA, HPHIS, Agric. Handb. No. 485.

Telford III, S. R., T. N. Mather, G. H. Adler, and A. Spielman. 1990. Short-tailed shrews as reservoirs of the agents of Lyme disease and human babesiosis. J. Parasitol. 76: 681-683.

Telford III, S. R., T. N. Mather, S. I. Moore, M. L. Wilson, and A. Spielman. 1988. Incompetence of deer as reserviors of the Lyme disease spirochete. Am. J. Trop. Med. Hyg. 39: 105–109.

Yuval, B., and A. Spielman. 1990. Duration and regulation of the developmental cycle of *Ixodes dammini* (Acari: Ixodidae). J. Med. Entomol. 27: 196-201.

Tick-Associated Diseases

Anderson, J. F., and L. A. Magnarelli. 1994. Lyme disease: A tick-associated disease originally described in Europe, but named after a town in Connecticut. Am. Entomol. 40: 217-227.

Bakken, J. S., J. S. Dumler, S. Chen, M. R. Eckman, L. L. V. Etta, and D. H. Walker. 1994. Human granulocytic ehrlichiosis in the upper midwest United States: A new species emerging? J. Am. Med. Assoc. 272: 212-218.

Benach, Jorge L. and Edward M. Bosler, eds. 1988. Lyme Disease and Related Disorders. Annals of the New York Academy of Sciences. Volume 539.

Barbour, Alan G. 1996. Lyme Disease: The Cause, the Cure, the Controversy. The John Hopkins University Press. 258 pp.

Barbour, A. G., and D. Fish. 1993. The biological and social phenomenon of Lyme disease. Science (Washington, D.C.) 260: 1610-1616.

Breitschwerdt, E. B., and D. L. Kordick. 2000. Bartonella infection in animals: Carriership, reservoir potential, pathogenicity, and zoonotic potential for human infection. Clin. Microbiol. Rev. 13: 428-438.

Dumler, J. S., and J. S. Bakken. 1995. Ehrlichial disease of humans: emerging tick-borne infections. Clin. Infect. Dis. 20: 1102-1110.

Edlow, J. A. 2003. Bull's Eye: Unraveling the medical mystery of Lyme disease. Yale University Press, New Haven.

Eskow, E., R. V. Rao, and E. Mordechai. 2001. Concurrent infection of the central nervous system by *Borrelia burgdorferi* and *Bartonella henselae*: evidence for a novel tick-borne disease complex. Arch. Neurol. 58: 1357-1367.

Centers for Disease Control and Prevention. 2004. Lyme disease – United States, 2001-2002. MMWR 53:365-369

Centers for Disease Control and Prevention. 2002. Lyme disease - United States, 2000. MMWR 51: 29-31.

Centers for Disease Control and Prevention. 2001. Outbreak of powassan encephalitis - Maine and Vermont, 1999-2001. MMWR 50: 761-764.

Centers for Disease Control and Prevention. 2002. Tularemia - United States, 1990-2000. MMWR 51: 181-184.

Centers for Disease Control and Prevention. 1995. Recommendations for test performance and interpretation form the second national conference on serologic diagnosis of Lyme disease. MMWR 44: 590-591.

Chang, Y. F., M. J. Appel, R. H. Jacobson, S. J. Shin, P. Harpending, R. Straubinger, L. A. Patrican, H. Mohammed, and B. A. Summers. 1995. Recombinant OspA protects dogs against infection and disease caused by *Borrelia burgdorferi*. Infect. Immun. 63: 3543-3549.

Dennis, D. T., and E. B. Hayes. 2002. Epidemiology of Lyme Borreliosis, pp. 251-280. *In* J. S. Gray, O. Kahl, R. S. Lane and G. Stanek [eds.], Lyme Borreliosis: Biology, Epidemiology and Control. CABI Publishing, Wallingford, Oxon, UK.

Ebel, G. D., A. Spielman, and S. R. Telford III. 2001. Phylogeny of North American Powassan virus. J. Gen. Virol. 82: 1657-1665.

Gerber, M. A., E. D. Shapiro, G. S. Burke, V. J. Parcells, and G. L. Bell. 1996. Lyme disease in children in southeastern Connecticut. New Eng. J. Med. 335: 1270-1274.

Gholam, B. I., S. Puksa, and J. P. Provias. 1999. Powassan encephalitis: a case report with neuropathology and literature review. Can. Med. Assoc. J. 161: 1419-1422.

Gray, Jeremy, Olaf Kahl, Robert S. Lane, and Gerold Stanek. 2002. Lyme Borreliosis: Biology, Epidemiology, and Control. CABI Publishing, Oxon, UK. 347 pp.

Herwaldt, B. L., P. C. McGovern, M. P. Gerwel, R. M. Easton, and R. R. MacGregor. 2003. Endemic babesiosis in another eastern state: New Jersey. Emerg. Infect. Dis. 9: 184-188.

Hu, R., and K. E. Hyland. 1997. Human babesiosis in the United States: a review with emphasis on its tick vector. Syst. Appl. Acarol. 2: 3-16.

Kjemtrup, A. M., and P. A. Conrad. 2000. Human babesiosis: an emerging tick-borne disease. Int. J. Parasitol. 30: 1323-1337.

Klempner, M. S., L. T. Hu, J. Evans, C. H. Schmid, G. M. Johnson, R. P. Trevino, D. Norton, L. Levy, D. Wall, J. McCall, M. Kosinski, and A. Weinstein. 2001. Two controlled trials of antibiotic treatment in patients with persistent symptoms and a history of Lyme disease. N. Engl. J. Med. 345: 85-92.

Krause, P. J. 2002. Babesiosis. Med. Clin. North Am. 86: 361-373.

Krause, P. and H. M. Feder, Jr. 1994. Lyme disease and babesiosis. Adv. Pediatric Infect. Dis. 9: 183-209.

Krause, P. J., K. McKay, C. A. Thompson, V. K. Sikand, R. Lentz, T. Lepore, L. Closter, D. Christianson, S. R. Telford III, D. A. Persing, J. D. Radolf, and A. Spielman. 2002. Disease-specific diagnosis of coinfecting tickborne zoonoses: babesiosis, human granulocytic ehrlichiosis, and Lyme disease. Clin. Infect. Dis. 34: 1184-1191.

Krause, P. J., T. Lepore, V. K. Sikand, J. Gadbaw, G. Burke, S. R. Telford III, P. Brassard, D. Pearl, J. Azlanzadeh, D. Christianson, D. McGrath, and A. Spielman. 2000. Atovaquone and azithromycin for the treatment of babesiosis. N. Engl. J. Med. 343: 1454-1458.

Krause, P. J., S. R. Telford III, A. Spielman, V. Sikand, R. Ryan, D. Christianson, G. Burke, P. Brassard, R. Pollack, J. Peck, and D. H. Persing. 1996. Concurrent Lyme disease and babesiosis: Evidence for increased severity and duration of illness. J. Am. Med. Assoc. 275: 1657-1660.

Levy, S. A., S. W. Barthold, D. M. Dombach, and T. L. Wasmoen. 1993. Canine Lyme borreliosis. Comendium (Small Animal) 15: 833-846.

Magnarelli, L. A., J. W. IJdo, J. F. Anderson, S. J. Padula, R. A. Flavell, and E. Fikrig. 1998. Human exposure to a granulocytic Ehrlichia and other tick-borne agents in Connecticut. J. Clin. Microbiol. 36: 2823-2827.

Magnarelli, L. A., J. S. Dumler, J. F. Anderson, R. C. Johnson, and E. Fikrig. 1995. Coexistence of antibodies to tick-borne pathogens of babesiosis, ehrlichiosis, and Lyme disease in human sera. J. Clin. Microbiol. 33: 3054-3057.

Meek, J. I., C. L. Roberts, J. E. V. Smith, and M. L. Cartter. 1996. Underreporting of Lyme disease by Connecticut physicians, 1992. J. Pub. Hlth. Man. 2: 61-65.

Murray, Polly. 1996. The Widening Circle: A Lyme Disease Pioneer Tells Her Story. St. Martin's Press. 321 pp.

Piesman, J., and A. Spielman. 1980. Human babesiosis on Nantucket Island prevalence of *Babesia microti* in ticks. Am. Soc. Trop. Med. Hyg. 29: 742-746.

Rahn, D. W., and M. W. Felz. 1998. Lyme disease update. Current approach to early, disseminated, and late disease. Postgrad. Med. 103: 51-54, 57-59, 63-64.

Rahn, Daniel W. and Janine Evans, eds. 1998. Lyme Disease. American College of Physicians. Philadelphia, PA. 245 pp. + color plates.

Schoen, R. T. 1989. Treatment of Lyme disease. Connecticut Medicine 53: 335-337.

Scoles, G. A., M. Papero, L. Beati, and D. Fish. 2001. A relapsing fever group spirochete transmitted by *Ixodes scapularis* ticks. Vector Borne Zoonotic Dis. 1: 21-34.

Sigal, L. H. [ed.] 1995. A symposium: National clinical conference on Lyme disease. Am. J. Med. 98 (suppl 4A): 1S-90S.

Sonenshine, Daniel E. and Thomas N. Mather. Eds. 1994. Ecological Dynamics of Tick-Borne Zoonoses. Oxford University Press. Oxford. 447 pp.

Stafford, K. C., III, M. L. Cartter, L. A. Magnarelli, S. Ertel, and P. A. Mshar. 1998. Temporal correlations between tick abundance and prevalence of ticks infested with *Borrelia burgdorferi* and increasing incidence of Lyme disease. J. Clin. Microbiol. 36: 1240-1244.

Steere, A. C. 2003. Duration of antibiotic therapy for Lyme disease [editorial]. Ann. Intern. Med. 138: 761-762.

Steere, A. C. 2001. Lyme disease. N. Engl. J. Med. 345: 115-125.

Steere, A. C., S. E. Malawista, D. R. Snydman, R. E. Shope, W. A. Andiman, M. R. Ross, and F. M. Steele. 1977. Lyme arthritis: an epidemic of oligoarticular arthritis in children and adults in three Connecticut communities. Arth. and Rheum. 20: 7-17.

Straubinger, R. K., B. A. Summers, Y. F. Chang, and M. J. Appel. 1997. Persistence of *Borrelia burgdorferi* in experimentally infected dogs after antibiotic treatment. J. Clin. Microbiol. 35: 111-116.

Telford, S. R., P. M. Armstrong, P. Katavolos, I. Foppa, G. A. S., M. L. Wilson, and A. Spielman. 1997. A new tick-borne encephalitis-like virus infecting New England deer ticks, *Ixodes dammini*. Emerging Infect. Dis. 3: 165-170.

Treadwell, T. A., R. C. Holman, M. J. Clarke, J. W. Krebs, C. D. Paddock, and J. E. Childs. 2001. Rocky mountain spotted fever in the United States, 1993-1996. Am. J. Trop. Med. Hyg. 63: 21-26.

Trock, D. H., J. E. Craft, and D. W. Rahn. 1989. Clinical manifestations of Lyme disease in the United States. Connecticut Medicine 53: 327-330.

Vanderhoof-Forschner, Karen. 1997. Everything You Need to Know About Lyme Disease and Other Tick-Borne Disorders. John Wiley & Sons, Inc. New York. 237 pp.

Varela, A. S., M. P. Luttrell, E. W. Howerth, V. A. Moore, W. R. Davidson, D. E. Stallknecht, S. E. Little. 2004. First culture isolation of *Borrelia lonestari*, putative agent of southern tick-associated rash illness. J. Clin. Microbiol. 42:1163-1169.

Walker, D. H., and J. S. Dumler. 1996. Emergence of ehrlichioses as human health problems. Emerging Infect. Dis. 2: 18-29.

Wormser, G. P., R. Ramanathan, J. Nowakowski, D. McKenna, D. Holmgren, P. Visintainer, R. Dornbush, B. Singh, and R. B. Nadelman. 2003. Duration of antibiotic therapy for early Lyme disease: A randomized, double-blind, placebo-controlled trial. Ann. Intern. Med. 138: 697-704.

Wormser, G. P., R. B. Nadelman, R. J. Dattwyler, D. T. Dennis, E. D. Shapiro, A. C. Steere, T. J. Rush, D. W. Rahn, P. K. Coyle, D. A. Persing, D. Fish, and B. J. Luft. 2000. Practice guidelines for the treatment of Lyme disease. Clin. Infect. Dis. 31: S1-S14.

Personal Protection, Repellents, Risk and Transmission

Cartter, M. L., T. A. Farley, H. A. Ardito, and J. L. Hadler. 1989. Lyme disease prevention - knowledge, beliefs, and behaviors among high school students in an endemic area. Connecticut Medicine 53: 354-356.

Consumers Union. 2000. Buzz Off! Consumer Reports June: 14-17.

Costello, C. M., A. C. Steere, R. E. Pinkerton, and J. H. M. Feder. 1989. A prospective study of tick bites in an endemic area for Lyme disease. J. Infect. Dis. 159: 136-139.

Couch, P., and C. E. Johnson. 1992. Prevention of Lyme disease. Am. J. Hosp. Pharm. 49: 1164-1173.

des Vignes, F., J. Piesman, R. Heffernan, T. L. Schulze, K. C. Stafford III, and D. Fish. 2001. Effect of tick removal on transmission of *Borrelia burgdorferi* and *Ehrlichia phagocytophila* by *Ixodes scapularis* nymphs. J. Infect. Dis. 183: 773-778.

Falco, R. C. 1996. Duration of tick bites in a Lyme disease-endemic area. Am. J. Epidemiol. 143: 187-192.

Falco, R. C., and D. Fish. 1989. Potential for exposure to tick bites in recreational parks in a Lyme disease endemic area. Amer. J. Public Health. 79: 12-15.

Fradin, M. S., and J. F. Day. 2002. Comparative efficacy of insect repellents against mosquito bites. JAMA 347: 13-18.

Levin, M. L., and D. Fish. 2000. Acquistion of coinfection and simultaneous transmission of *Borrelia burgdorferi* and *Ehrlichia phagocytophila* by *Ixodes scapularis*. Infect. Immun. 68: 2183-2186.

Medical Letter Advisory Board. 1985. Insect repellents. Med. Lett. Drugs-Ther. 27: 62-64.

Nadelman, R. B., J. Nowakowski, D. Fish, R. C. Falco, K. Freeman, D. McKenna, P. Welch, R. Marcus, M. E. Aguero-Rosenfeld, D. T. Dennis, and G. P. Wormser. 2001. Prophylaxis with single-dose doxycycline for the prevention of Lyme disease after an *Ixodes scapularis* tick bite. N. Engl. J. Med. 345: 79-84.

Osimitz, T. G., and R. H. Grothaus. 1995. The present safety assessment of deet. J. Am. Mosquito Cont. Assoc. 11: 274-278.

Piesman, J., and E. B. Hayes. 2003. How can we prevent Lyme disease? N. Engl. J. Med. 348: 2424-2430.

Piesman, J. 2002. Ecology of Borrelia burgdorferi sensu lato in North America, pp. 223-250. *In* J. S. Gray, O. Kahl, R. S. Lane and G. Stanek [eds.], Lyme Borreliosis: Biology, Epidemiology and Control. CABI Publishing, Wallingford, Oxon, UK.

Piesman, J., and M. C. Dolan. 2002. Protection against Lyme disease spirochete transmission provided by prompt removal of nymphal *Ixodes scapularis* (Acari: Ixodidae). J. Med. Entomol. 39: 509-512.

Piesman, J., B. S. Schneider, and N. S. Zeidner. 2001. Use of quantitative PCR to measure density of *Borrelia burgdorferi* in the midgut and salivary glands of feeding tick vectors. J. Clin. Microbiol. 39: 4145-4148.

Piesman, J., T. N. Mather, R. J. Sinsky, and A. Spielman. 1987. Duration of tick attachment and *Borrelia burgdorferi* transmission. J. Clin. Microbiol. 25: 557-558.

Schreck, C. E., D. Fish, and T. P. McGovern. 1995. Activity of repellents applied to skin for protection against *Amblyomma americanum* and *Ixodes scapularis* ticks (Acari: Ixodidae). J. Am. Mosq. Cont. Assoc. 11: 136-140.

Schreck, C. E., E. L. Snoddy, and A. Spielman. 1986. Pressurized sprays of permethrin or deet on military clothing for personal protection against *Ixodes dammini* (Acari:Ixodidae). J. Med. Entomol. 23: 396–399.

Shadick, N. A., L. H. Daltroy, C. B. Phillips, U. S. Lang, and M. H. Lang. 1997. Determinants of tick-avoidance behaviors in an endemic area for Lyme disease. Am. J. Prev. Med. 13: 265-270.

Shapiro, E. D., M. A. Gerber, N. B. Holabird, A. T. Berg, H. M. Feder, G. L. Bell, P. N. Rys, and D. H. Persing. 1992. A controlled trial of antimicrobial prophylaxis for Lyme disease after deer-tick bites. N. Engl. J. Med. 327: 1769-1773.

Sood, S. K., M. B. Salzman, B. J. Johnson, C. M. Happ, K. Feig, L. Carmondy, L. G. Rubin, E. Hilton, and J. Piesman. 1997. Duration of tick attachment as a predictor of the risk of Lyme disease in an area in which Lyme disease is endemic. J. Infect. Dis. 175: 996-999.

Warshafsky, S., J. Nowakowski, R. B. Nadelman, R. S. Kamer, S. J. Peterson, and G. P. Wormser. 1996. Efficacy of antibiotic prophylaxis for prevention of Lyme disease: a meta-analysis. J. Gen. Intern. Med. 11: 329-333.

Tick Distribution, Landscape and Host Management

Adler Jr., Bill. 1999. Outwitting Deer. The Lyons Press, New York, New York. 177 pp.

Anderson, J. F., R. C. Johnson, L. A. Magnarelli, F. W. Hyde, and J. E. Myers. 1987. Prevalence of *Borrelia burgdorferi* and *Babesia microti* in mice on islands inhabited by whitetailed deer. Appl. Environ. Microbiol. 53: 892-894.

Borman, F. H., D. Balmori, and G. T. Geballe. 2001. Redesigning the American Lawn: A Search for Environmental Harmony. 2nd edition. Yale University Press. 178 pp.

Carroll, M. C., H. S. Ginsberg, K. E. Hyland, and R. Hu. 1992. Distribution of *Ixodes dammini* (Acari: Ixodidae) in residential lawns on Prudence Island, Rhode Island. J. Med. Entomol. 29: 1052-1055.

Daniels, T. J., D. Fish, and I. Schwartz. 1993. Reduced abundance of *Ixodes scapularis* (Acari: Ixodidae) and Lyme disease risk by deer exclusion. J. Med. Entomol. 30: 1043-1049.

Deblinger, R. D., M. L. Wilson, D. W. Rimmer, and A. Spielman. 1993. Reduced abundance of immature *Ixodes dammini* (Acari: Ixodidae) following incremental removal of deer. J. Med. Entomol. 30: 144-150.

DeNicola, Anthony J., Kurt C. VerCauteren, Paul D. Curtis, and Scott E. Hygnstrom. 2000. Managing White-tailed Deer in Suburban Environments: A Technical Guide. Cornell Cooperative Extension, the Wildlife Society, and Northeast Wildlife Damage Research and Outreach Cooperative. 52 pp.

Ellingwood, Mark R. and Suzanne L Caturano. 1988. An Evaluation of Deer Management Options. Publication No. DR-11. CT Department of Environmental Protection. 16 pp. (revised and reformatted, NH Fish & Game Dept., 1996).

Falco, R. C., and D. Fish. 1988. Prevalence of *Ixodes dammini* near the homes of Lyme disease patients in Westchester County, New York. Am. J. Epidemiol. 127: 826-830.

Hart, Rhonda Massingham. 1997. Deer Proofing Your Yard & Garden. Storey Books. Pownal, Vermont. 155 pp.

Hygnstrom, Scott E., Robert M. Timm, and Gary E. Larson, eds. 1994. Prevention and Control of Wildlife Damage. University of Nebraska Cooperative Extension, USDA-APHIS-Wildlife Services, and Great Plains Agricultural Council Wildlife Committee.

Levy, S. A., B. A. Lissman, and C. M. Ficke. 1993. Performance of a *Borrelia burgdorferi* bacterin in borreliosis-endemic areas. J. Am. Vet. Med. Assoc. 202: 1834-1838.

Lewis, A. 1997. Butterfly Gardens: Luring Nature's Lovelist Pollinators to Your Yard. Brooklyn Botanic Garden, Inc., Brooklyn, NY.

Maupin, G. O., D. Fish, J. Zultowsky, E. G. Campos, and J. Piesman. 1991. Landscape ecology of Lyme disease in a residential area of Westchester County, New York. Am. J. Epidemiol. 133: 1105-1113.

McDonald Jr., John E., Mark R. Ellingwood, and Gary M. Vecellio. 1998. Case Studies in Controlled Deer Hunting. Northeast Deer Technical Committee. 16 p. Available from your state wildlife management agency.

Solberg, V. B., J. A. Miller, T. Hadfield, R. Burge, J. M. Schech, and J. M. Pound. 2003. Control of *Ixodes scapularis* (Acari: Ixodidae) with topical self-application of permethrin by white-tailed deer inhabiting NASA, Beltsville, Maryland. J. Vector. Ecol. 28: 117-134.

Stafford III, K. C. 2001. An increasing deer population is linked to the rising incidence of Lyme disease. Frontiers Plant Sci. 53: 3-4.

Stafford III, K. C. 1993. Reduced abundance of *Ixodes scapularis* (Acari: Ixodidae) with exclusion of deer by electric fencing. J. Med. Entomol. 30: 986-996.

Stafford, K. C., III, and L. A. Magnarelli. 1993. Spatial and temporal patterns of *Ixodes scapularis* (Acari: Ixodidae) in southcentral Connecticut. J. Med. Entomol. 30: 762-771.

Tekulski, M. 1985. The Butterfly Garden. The Harvard Common Press, Harvard, MA.

Ward, J.S. Limiting Deer Browse Damage to Landscape Plants. Connecticut Agricultural Experiment Station Bulletin 968, November 2000.

Wilson, M. L., S. R. Telford III, J. Piesman, and A. Spielman. 1988. Reduced abundance of immature *Ixodes dammini* (Acari: Ixodidae) following elimination of deer. J. Med. Entomol. 25: 224-228.

Wilson, M. L. 1986. Reduced abundance of adult *Ixodes dammini* (Acari: Ixodidae) following destruction of vegetation. J. Econ. Entomol. 79: 693-696.

Tick IPM and Chemical Control

Addiss, S. S., N. O. Alderman, D. R. Brown, and J. Wargo. 1999. A survey of private drinking water wells for lawn and tree care pesticides in a Connecticut town. Environmental & Human Health, Inc., North Haven, CT.

Allan, S. A., and L. A. Patrican. 1995. Reduction of immature *Ixodes scapularis* (Acari: Ixodidae) in woods by application of desiccant and insecticidal soap formulations. J. Med. Entomol. 32: 16-20.

Curran, K. L., D. Fish, and J. Piesman. 1993. Reduction of nymphal *Ixodes dammini* (Acari: Ixodidae) in a residential suburban landscape by area application of insecticides. J. Med. Entomol. 30: 107-113.

Deblinger, R. D., M. L. Wilson, D. W. Rimmer, and A. Spielman. 1993. Reduced abundance of immature *Ixodes dammini* (Acari: Ixodidae) following incremental removal of deer. J. Med. Entomol. 30: 144-150.

Dolan, M.C., G. O. Maupin, B. S. Schneider, C. Denatale, N. Hamon, C. Cole, N. S. Zeidner, and K. C. Stafford III. 2004. Control of immature *Ixodes scapularis* (Acari: Ixodidae) on rodent reservoirs of *Borrelia burgdorferi* in a residential community of southeastern Connecticut. J. Med. Entomol. In Press.

Elfassy, O. J., F. W. Goodman, S. A. Levy, and L. L. Carter. 2001. Efficacy of an amitraz-impregnated collar in preventing transmission of *Borrelia burgdorferi* by adult *Ixodes scapularis* to dogs. J. Amer. Vet. Med. Assoc. 219: 185-189.

Fish, D. 1995. Environmental risk and prevention of Lyme disease. Am. J. Med. 98 (suppl 4A): 2S-9S.

Hansen, Michael. 1992. Pest Control for Home and Garden: The Safest and Most Effective Methods for You and the Environment. Consumer Reports Books. Yonkers, New York. 304 pp. (out-of-print)

Hayes, E. B., G. O. Maupin, G. A. Mount, and J. Piesman. 1999. Assessing the prevention effectiveness of local Lyme disease control. J. Public Health Mgt. Practice 5: 84-92.

Kamrin, Michael A. Editor. 1997. Pesticide Profiles: Toxicity, Environmental Impact, and Fate. CRC Press. 676 pp.

Mount, G. A., D. G. Haile, and E. Daniels. 1997. Simulation of management strategies for the blacklegged tick (Acari: Ixodidae) and the Lyme disease spirochete, *Borrelia burgdorferi*. J. Med. Entomol. 34: 672-683.

Olkowski, William, Shelia Daar, Helga Olkowski. 1991. Common Sense Pest Control. The Taunton Press. 715 pp.

Ostfeld, R. S., and D. N. Lewis. 1999. Experimental studies of interactions between wild turkeys and black-legged ticks. J. Vector. Ecol. 24: 182-186.

Patrican, L. A., and S. A. Allan. 1995. Laboratory evaluation of desiccants and insecticidal soap applied to various substrates to control the deer tick *Ixodes scapularis*. Med. Vet. Entomol. 9: 293-299.

Patrican, L. A., and S. A. Allan. 1995. Application of desiccant and insecticidal soap treatments to control *Ixodes scapularis* (Acari: Ixodidae) nymphs and adults in a hyperendemic woodland site. J. Med. Entomol. 32: 859-863.

Pound, J. M., J. A. Miller, and J. E. George. 2000. Efficacy of amitraz applied to white-tailed deer by the '4-poster' topical treatment device in controlling free-living lone star ticks (Acari: Ixodidae). J. Med. Entomol. 37: 878-884.

Pound, J. M., J. A. Miller, J. E. George, and C. A. LeMeilleur. 2000. The '4-Poster' passive tropical treatment device to apply acaricide for controlling ticks (Acari: Ixodidae) feeding on white-tailed deer. J. Med. Entomol. 37: 588-594.

Schulze, T. L., R. A. Jordan, R. W. Hung, R. C. Taylor, D. Markowski, and M. S. Chomsky. 2001. Efficacy of granular deltamethrin against *Ixodes scapularis* and *Amblyomma americanum* (Acari: Ixodidae) nymphs. J. Med. Entomol. 38: 344-346.

Schulze, T. L., R. A. Jordan, and R. W. Hung. 1995. Suppression of subadult *Ixodes scapularis* (Acari: Ixodidae) following removal of leaf litter. J. Med. Entomol. 32: 730-733.

Solberg, V. B., K. Neidhardt, M. R. Sardelis, F. J. Hoffman, R. Stevenson, L. R. Boobar, and H. J. Harlan. 1992. Field evaluation of two formulations of cyfluthrin for control of *Ixodes dammini* and *Amblyomma americanum* (Acari: Ixodidae). J. Med. Entomol. 29: 634-638.

Stafford, K. C., III, A. J. DeNicola, and H. J. Kilpatrick. 2003. Reduced abundance of *Ixodes scapularis* (Acari: Ixodidae) and the tick parasitoid *Ixodiphagus hookeri* (Hymenoptera: Encyrtidae) with reduction of white-tailed deer. J. Med. Entomol. In press.

Stafford III, K. C. 2002. Environmental management for Lyme borreliosis, pp. 368. In J. Gray [ed.], Lyme Borreliosis: Biology and Control. CABI Publishing, Oxon, UK.

Stafford III, K. C. 1997. Pesticide use by licensed applicators for the control of *Ixodes scapularis* (Acari: Ixodidae) in Connecticut. J. Med. Entomol. 34: 552-558.

Stafford III, K. C. 1991. Effectiveness of carbaryl applications for the control of *Ixodes dammini* (Acari: Ixodidae) nymphs in an endemic residential area. J. Med. Entomol. 28: 32-36.

Stafford III, K. C.. 1989. Lyme disease prevention: Personal protection and prospects for tick control. Conn. Med. 53: 347-351.

Wargo, J., N. O. Alderman, and L. Wargo. 2003. Risks from lawn-care pesticides including inadequate packaging and labeling, pp. 96. Environmental and Human Health, Inc., North Haven, CT.

Wilson, M. L., and R. D. Deblinger. 1993. Vector management to reduce the risk of Lyme disease, pp. 126-156. *In* H. S. Ginsberg [ed.], Ecology and Environmental Management of Lyme Disease. Rutgers University Press, New Brunswick, N.J.

Zhioua, E., M. Browning, P. W. Johnson, H. S. Ginsberg, and R. A. LeBrun. 1997. Pathogenicity of the entomopathogenic fungus *Metarhizum anisopliae* (Deuteromycetes) to *Ixodes scapularis* (Acari: Ixodidae). J. Parasitol. 83: 815-818.

About the Author

Dr. Kirby Stafford is a medical-veterinary entomologist whose research focuses on the ecology and control of the blacklegged tick. He received his B.S. in entomology and M.S. in veterinary entomology from Colorado State University and Kansas State University, respectively, and his Ph.D. in medical/veterinary entomology from Texas A&M University in 1985. After working at Penn State on a poultry pest management project, he joined the Connecticut Agricultural Experiment Station in 1987. Dr. Stafford became Chief Scientist and Head of the Department of Forestry and Horticulture in 1997.

The Nation's First State Agricultural Experiment Station

The Connecticut Agricultural Experiment Station is a state-supported scientific research institution dedicated to improving the food, health, environment and well-being of Connecticut's citizens since 1875. The Connecticut Agricultural Experiment Station investigates the growth of plants and studies their pests, insects, ticks, soil and water quality, and food safety, and performs analyses for state agencies. Station staff registers and inspect nurseries, certify honeybee colonies, and inspect thousands of individual plants or other regulated material being shipped into or from Connecticut.

The Experiment Station first opened its doors in a laboratory in Wesleyan University in Middletown in October 1875. It was moved to Yale University in 1877 and to its current location in New Haven in 1882. Today, the Experiment Station is composed of one administrative and six scientific departments with around 100 scientists, technicians, and support staff. The Experiment Station also operates a 75-acre research farm in Hamden and a farm at its Valley Laboratory in Windsor, Connecticut.

Among many information sheets and publications, The Experiment Station's web page (www.caes.state.ct.us) features this handbook and an extensive electronic Plant Pest Handbook, which covers diseases, insects, cultural and nematode problems of Connecticut. plants.

The Connecticut Agricultural Experiment Station

Putting Science to Work for Society

Founded 1875

This handbook is available in electronic format at www.caes.state.ct.us

---

Printed by Printing Plus, Portland, Connecticut