

Lyme Disease

The Ecology of a Complex System

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It's the Deer



MILLIONS OF PEOPLE IN NEW ENGLAND, NEW YORK, THE MID-Atlantic, and the Upper Midwest regions of the United States, not to mention many other localities in North America, Europe, and Asia, live in fear of contracting Lyme disease. They are aware that any time they picnic, hike, garden, walk the dog, or play catch they could encounter a tick and get seriously ill. The pervasive impression is that ticks are much more abundant than they used to be, that they are pretty much everywhere, and that deer are to blame because they are responsible for feeding the ticks and spreading them around. These notions are reinforced in virtually all accounts of Lyme disease and ticks provided by newspapers, television, and the Internet and are repeated in discussions with neighbors and friends. Deer are considered largely culpable for the sense of foreboding that can accompany each spring or summer foray from the house or apartment. The irritation at deer is only increased by their tendency to eat the flowers and shrubbery and dash out in front of cars. Many people are indignant that their towns or counties haven't done enough to manage deer and protect their health. In more and more of these towns, local people are organizing and pressuring governments to aggressively cull deer in order to reduce the Lyme disease threat.

Where did the notion that deer determine tick abundance and Lyme disease risk come from? What if it's wrong, or only partially right? What if culling deer—a very expensive and logistically challenging enterprise—does little or nothing to reduce the risk?

The notion that Lyme disease risk is closely tied to the abundance of deer arose from field studies that began shortly after the discoveries of the bacterial agent of Lyme disease and the involvement of ticks as vectors of these bacteria. The context of these studies was the hunt for the culprits—the *critical species*—involved in creating risk of exposure to Lyme disease. These were thorough and energetic studies, conducted with considerable urgency and scientific rigor. Such mission-oriented research can be a powerful weapon for fighting emerging infectious diseases, but the other side of this doubled-edged sword is the tendency to trim away complicating information before its importance can be evaluated. Perhaps it was the strong compulsion to provide specific information that could be used for disease prevention—in contrast to a quest for more basic information to support the understanding of general principles of disease risk and prevention—that led to this overly simplistic view of Lyme disease ecology.

White-tailed deer are considered “the definitive host of the [black-legged] tick” (Madhav et al. 2004), “the primary source of nourishment for gravid female *I. scapularis*” (Rand et al. 2004), “the primary reproductive stage host” (Rand et al. 2003), “the one indispensable piece in the LB [Lyme borreliosis] puzzle in North America” (Piesman 2002), and “the keystone host for adult *I. scapularis*” (Childs 2009). In his excellent book on the history of Lyme disease medical research, Jonathan Edlow (2003: 149) makes note of the ancient existence of Lyme spirochetes and asks the question: “If the Lyme spirochete had been around for so long, why did it begin to surface as a recognized medical entity only in the past few decades? This question can be answered in one word—deer.” Where did these conclusions come from, and what do they mean for Lyme disease ecology and prevention?

A flurry of research on what was then called *I. dammini* (see below) ensued in the late 1970s and 1980s to advance our understanding of risk factors for newly discovered Lyme disease and lay a foundation for preventive measures. Like other ixodid (hard) ticks, *I. dammini* was found to undergo two immature stages (larva and nymph) in addition to the adult stage (figure 8). At each stage, the tick takes a single blood meal from a vertebrate host to fuel transition to the next stage or, in the case of adults, to fuel reproduction. During these blood meals, the tick stays attached to the host’s skin for several days to about a week, steadily imbibing host blood. Newly hatched larvae seek a host in midsummer, and after their blood meal, they drop off the host and molt into nymphs, which then overwinter on the forest floor before seeking a host the next late spring or early summer. After taking their blood meal, nymphs molt into adults,

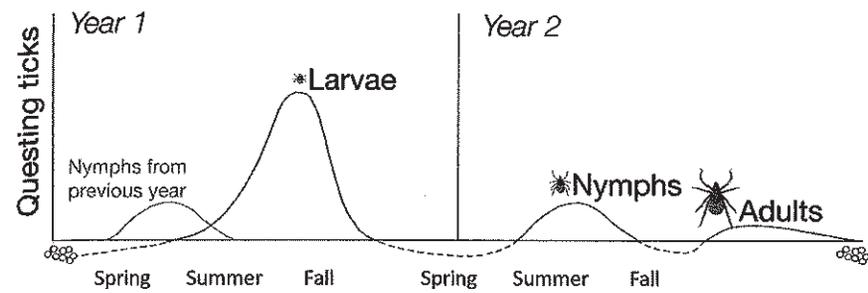


FIGURE 8. Basic life cycle of the blacklegged tick, *Ixodes scapularis*. This species was formerly and incorrectly called the “deer tick” (*Ixodes dammini*). Each year, a new cohort of larval ticks hatches from eggs in mid to late summer. After taking their single several-day-long blood meal from a vertebrate host, the larvae drop off, molt into nymphs, hunker down in the leaf litter or mineral soil, and overwinter on the forest floor. The next spring or early summer, these nymphs activate and seek a host (this is the stage mostly responsible for infecting humans and wildlife). After feeding on a host, the nymphs digest the blood meal, molt into the adult stage, and seek their final host in mid to late autumn. Thus, these ticks take three blood meals, once each as a larva, nymph, and adult, before reproducing and dying. Source: Brunner and Ostfeld (2008). Reprinted with permission.

which seek a host in mid to late autumn (figure 9). Adult females engorged with host blood overwinter before depositing an egg mass in spring, from which the next generation of larvae emerge in mid summer. When free-living on the forest floor (not attached to a host), these ticks are poor at getting about—they’re able to crawl only a matter of meters. But they appear to be exquisitely sensitive to chemical and physical gradients, able to orient toward safe locations for overwintering and toward hosts emitting carbon dioxide and infrared radiation. Thus, the tick life cycle lasts two years and can involve three distinct vertebrate host species, with considerable time spent either inactive or seeking a host (*questing*) on the forest floor. The abundance and distribution of organisms with such complex life cycles could have been assumed to be determined by a complex suite of biotic and abiotic factors, including availability of several different hosts. However, even when first principles suggest that several factors are important in determining species abundances and how they change through time, such multifactorial approaches are rarely a part of initial research strategies (Lidicker 1991, Ostfeld 2008).

Several years before Lyme disease was recognized as a serious health threat, researchers began pursuing the ecology of another tick-transmitted disease that was attacking residents and visitors of Cape Cod and nearby islands such as Martha’s Vineyard and Nantucket (Spielman

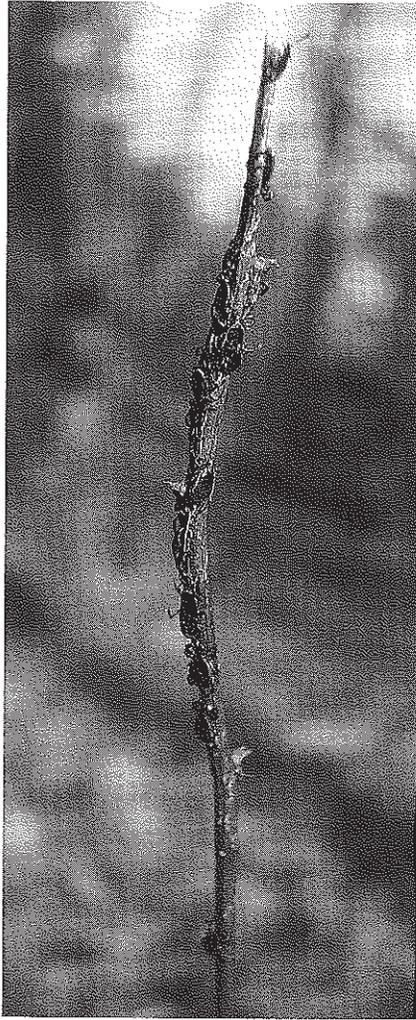


FIGURE 9. A group of adult blacklegged ticks in the act of *questing*—seeking a vertebrate host by climbing on forest-floor vegetation to a height of about 1 meter. As a potential host brushes against the vegetation, the ticks will grab hold of the animal and seek a site for attaching to the skin and drinking blood. Photograph by Michael Benjamin.

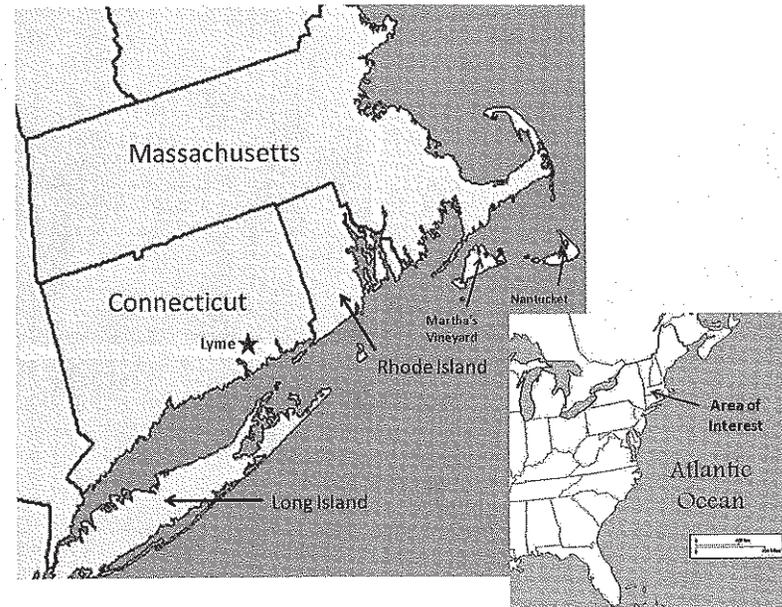


FIGURE 10. Map showing the coast of southern New England and adjacent New York, where Lyme disease first emerged in the 1970s. Cape Cod and the adjacent islands of Nantucket and Martha's Vineyard, the coast of Connecticut, and the north shore of Long Island were sites of particular importance as the seminal early studies of Lyme disease ecology got under way.

with larvae peaking in August (up to 467 per deer), nymphs peaking in June (up to 68 per deer), and adults most prevalent in November (up to 292 per deer). These researchers contrasted their results with those of the sparse prior literature on associations between ticks and their various hosts. This literature described immature *I. dammini* ticks as host specialists, being largely restricted to small rodents like mice and voles, and adult *I. dammini* as host generalists, being found on dogs, bears, foxes, skunks, opossums, raccoons, and other medium-sized and large mammals. Piesman and colleagues (1979) emphasized that their new findings suggested a very different scenario; the commonness of immature ticks on deer indicated that larval and nymphal abundances in fact might depend at least as much on deer as on small rodents, and the rarity of nondeer hosts on Nantucket indicated that adult ticks could be supported by deer alone (figure 11). This characterization of all tick life stages as closely tied to deer was highly influential and stimulated research on deer control as a method of reducing tick populations and the threat of tick-borne diseases.

1976, Spielman et al. 1979) (figure 10). Medical entomologists working on Nantucket in 1976 and 1977 recruited Massachusetts Fisheries and Game Division personnel to shoot white-tailed deer year-round, and they also examined many deer shot by recreational hunters during the late autumn hunting season. Careful inspections of deer carcasses revealed large numbers of *Ixodes* ticks on various parts of the skin (Piesman et al. 1979). All three active life stages of the ticks were found on



FIGURE 11. White-tailed deer, *Odocoileus virginianus*. Source: Myers et al. (2006). Reprinted with permission.

Strongly reinforcing this perception of deer as the critical host was the newly invented common (nonscientific) name “Dammin’s northeastern deer ixodid”—later shortened to “deer tick”—applied to what was thought to be a newly described species, *Ixodes dammini* (Spielman et al. 1979). Initially, the tick species responsible for transmitting both Lyme disease and babesiosis in coastal New England was identified as *Ixodes scapularis*, the blacklegged tick (Spielman 1976, Wallis et al. 1978, Burgdorfer et al. 1982). This species was first described and named back in 1821 and had been found over the ensuing decades to be a very widespread tick species with populations documented from Massachusetts to Florida and from Ontario and Minnesota to Texas (Keirans et al. 1996) (figure 12).

For a species with such an enormous geographic range, some degree of variation in physical appearance among populations is to be expected, and indeed, considerable variation in morphological features thought to be taxonomically important has been described (Oliver et al. 1993, Keirans et al. 1996). During the course of their Nantucket field studies of ticks and human babesiosis in the late 1970s, Spielman and colleagues (1979) became convinced that the northern populations of *I. scapularis* were sufficiently

Established* and reported** distribution of the Lyme disease vectors *Ixodes scapularis* (*I. dammini*) and *Ixodes pacificus*, by county, United States, 1907-1996

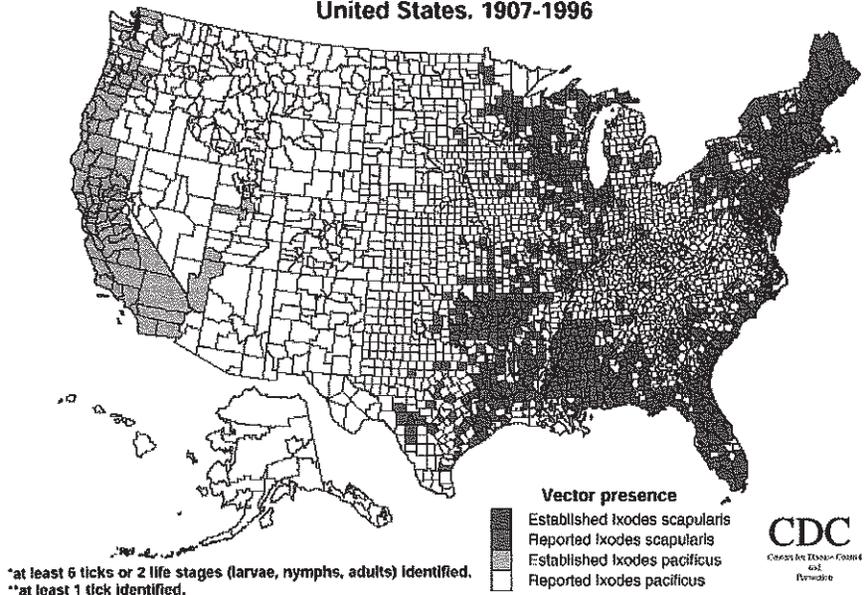


FIGURE 12. Counties in the contiguous United States in which the blacklegged tick, *Ixodes scapularis*, has been collected. Also shown are the western counties where its close relative, the western blacklegged tick, *I. pacificus*, has been collected. Source: Dennis et al. (1998). Reprinted with permission.

distinct from southern populations to warrant the description of a new species, *I. dammini*, that was distributed from New England and New York to Ontario, Wisconsin, and Minnesota. This conclusion was based on differences between northern and southern populations in size, shape, and position of spines (auriculae) on the exoskeleton and in the apparent preference of immatures in northern populations for feeding on small rodents, in contrast to southern populations, which appeared to prefer lizards. Ultimately, neither the morphological nor the behavioral characteristics that were claimed to support the designation of a new species were justified.

Although the use of both morphological and behavioral characteristics to infer taxonomy has a rich history, these types of characteristics are also known to be unreliable in many instances. For example, differences between northern and southern tick populations in host associations could be caused by a variety of differences not related to taxonomy. Lizards are scarce and patchily distributed in some parts of these northern states and provinces and nonexistent in others; therefore, the ticks' lack of “preference” for

lizards in these areas is not surprising. This leads to the question of how we ascertain which hosts ticks prefer to feed on. Rarely are ticks confronted with a choice between different host species that would allow their true preferences to be examined (but see Shaw et al. 2003). Instead, “preferences” are inferred from patterns of distribution of ticks on hosts. These patterns might be determined more by the relative availability of different host species in different areas than by innate preferences. (Interestingly, northern populations of these *Ixodes* ticks do in fact parasitize lizards where lizards occur [Giery and Ostfeld 2007].) Host associations also might be influenced by climate conditions that affect the specific locations where ticks dwell when seeking hosts. Surprisingly little research has been done on what preferences ticks might have for particular hosts and on what factors besides innate preferences affect the distribution of ticks on various host species that are available to them.

The conclusion that a new tick species had been discovered was rejected for a variety of reasons. Up until the 1970s, taxonomists relied primarily on morphological features to distinguish between related species. But taxonomists are fundamentally interested in separating species on the basis of their evolutionary, or *phylogenetic*, relationships, and morphological features can mislead scientists trying to infer these phylogenetic relationships. Many examples exist of organisms that cannot be distinguished morphologically, and so are lumped into the same species, that are later shown to consist of groups that are reproductively isolated from one another and therefore (by definition) distinct species. Similarly, many “species” are provisionally described on the basis of morphological differences from other related species but then are lumped into a single species when the groups are found not to be genetically distinct or reproductively isolated.

The dismantling of the status of *Ixodes dammini* as a distinct species began in 1993 with the publication of a comprehensive study by Oliver and colleagues (1993), who conducted laboratory mating experiments between “*I. dammini*” from Massachusetts and *I. scapularis* from Georgia. They found that these two species interbred readily and produced fertile offspring through at least three generations. In contrast, when either of these species was bred with another related *Ixodes* species—in this case *I. pacificus*—the mating resulted in hybrid sterility in the first generation. Oliver and colleagues also measured 65 different morphological characters in adults and nymphs of *I. dammini*, *I. scapularis*, and their “hybrid” offspring and found that statistical programs designed to discriminate between closely related taxonomic groups could not find distinguishing sets of characters and lumped the three groups together. Closely related

species often differ in the size and arrangement of their chromosomes and in their versions of particular enzymes and proteins (isozymes), but neither chromosomes nor isozymes of the groups differed. Later comparisons of the DNA sequences for both ribosomes and chromosomes showed that *I. dammini* was not distinct from *I. scapularis* and that there was no evidence of reproductive isolation (Wesson et al. 1993, Norris et al. 1996). Norris et al. (1996) and Keirans et al. (1996) provide various lines of genetic and morphological evidence that all ticks previously thought to be *I. dammini* are actually *I. scapularis*, and that *I. scapularis* consists of two lineages, one that occurs from Florida to North Carolina (the “southern clade”) and another that extends from Mississippi to New England (the “American clade”). The hypothesis that *Ixodes dammini* is a valid species has been discredited, and almost all scientists studying this species call it *Ixodes scapularis*. By the rules of zoological nomenclature, the common name “blacklegged tick” is the only correct vernacular name for this species.

But the 1979 description of the New England and New York ticks responsible for transmitting Lyme disease and human babesiosis as a new species, the deer tick *Ixodes dammini*, was accepted almost without dissent by tick biologists (*acarologists*), medical entomologists, epidemiologists, health care providers, and others. In the 14 years that elapsed before this taxonomy and associated names were invalidated, there was a veritable explosion of scientific research on these ticks, their hosts, and the pathogens they transmit. Much of the current understanding of the ecology and epidemiology of these diseases began during this period from the late 1970s to the early 1990s, when the tick was incorrectly named. Even today the invalidated common name “deer tick” persists in both the scientific and nonscientific literature. The persistence of this discredited name certainly helps perpetuate the notion that deer are the essential host for this tick.

The Science of the “Deer-Tick” Connection

Meanwhile, back in coastal New England in the 1980s, ticks were making many people ill, and scientists began to devise and evaluate means of controlling ticks. Given the pervasive view that deer were the critical host, these studies consisted of two basic approaches: (1) reducing, eliminating, or excluding deer from areas where ticks were abundant and disease risk high—an experimental approach; and (2) determining whether the abundance of ticks correlated with that of deer—a correlational approach. The first experimental reduction in deer density to assess impacts on ticks and

disease risk was conducted by Mark Wilson and colleagues in 1982 on Great Island, Cape Cod, Massachusetts. They initially intended to capture deer, tranquilize them, apply insecticide/acaricide or tick repellents to their skin, and release them. These efforts proved both impractical and highly stressful to the deer, so instead this research group employed state biologists to shoot deer. Before removal, this 240-ha island supported an estimated population of at least 30 deer, and the experimental hunt reduced the deer population by an estimated 70%. To assess the impact of deer reduction on ticks, the researchers live-trapped white-footed mice (*Peromyscus leucopus*) and counted the larval and nymphal ticks on them. The results of Wilson and colleagues' study were disconcerting, to say the least. The dramatic reduction in deer abundance did nothing to decrease the number of ticks on mice, and there was even a suggestion that that number might be increasing (Wilson et al. 1984).

Later assessments of the impacts of deer reduction on tick abundance showed somewhat different results. Wilson and colleagues continued to reduce the deer population on Great Island throughout the mid-1980s until it was less than one-tenth of its previous size. At this point, the average number of larval and nymphal ticks on white-footed mice, and the total estimated population of immature ticks on the mouse population, declined significantly (Wilson et al. 1988). However, tick populations did not approach extinction on the island. On another coastal New England island, Monhegan Island in Maine, Rand and colleagues (2004) used hunters to remove deer, achieving their goal of complete eradication in the spring of 1999. By 2001, abundance of larval and nymphal ticks on Norway rats (*Rattus norvegicus*), the only apparent host for immature ticks on the island, declined to near zero, and by 2002 host-seeking adult ticks collected from vegetation became very scarce. On Long Island, New York, Duffy and colleagues (1994) surveyed tick abundance in 22 natural areas, seven of which had no deer. They found significantly fewer immature ticks in the deer-free sites, but all sites had nymphs, and the average number of nymphs collected in deer-free areas—10 per hour spent sampling—was within the range seen in areas with rampant Lyme disease.

Complete eradication of deer might be possible on some islands, but on the mainland it is infeasible, because deer-free zones are quickly recolonized by deer from neighboring areas. Consequently, researchers in mainland sites have sought to reduce rather than eliminate deer herds. On the Crane Reservation of coastal Massachusetts, Deblinger and colleagues (1993) used hunters to reduce the deer population from about 350 in 1985 to about 50 in 1991. Their counts of numbers of immature ticks on small

mammals were initially encouraging, with larvae declining from about 21 per mouse before deer removal to about 10 per mouse after deer removal, and nymphs declining from about 3 to about 1.5 per mouse. But these reductions were only temporary, with numbers of both larvae and nymphs increasing in the early 1990s to levels similar to those measured before the deer reduction, despite the vastly reduced deer density at this time. On two sites in southern coastal Connecticut, Bridgeport and Bluff Point, Stafford and colleagues (2003) reduced deer density from more than 90 per square kilometer to about 15 to 30 per square kilometer. At Bridgeport, numbers of host-seeking nymphal ticks declined significantly after deer reduction, but numbers of host-seeking larvae fluctuated quite a bit, increasing in some years to near prehunt levels. At Bluff Point, the researchers found no significant correlation between abundance of host-seeking nymphs and abundance of deer, although abundance of larvae correlated with abundance of deer. At a suburban site in Somerset County, New Jersey, Jordan and colleagues (2007) assessed the impact of deer control by archery and shotgun hunters on tick populations. Hunters reduced the deer population by 47%, from about 46 to about 24 deer per square kilometer. However, abundances of both host-seeking larval and adult ticks at the culling sites were *greater* after deer reduction than before. Abundance of nymphs fluctuated with no apparent relation to deer culling. In addition, Jordan and colleagues assessed the impact of the deer reduction on numbers of Lyme disease cases reported to local health authorities and found no correlation between deer abundance and Lyme disease incidence in the township.

Logistically much easier and far less contentious than culling deer is excluding them from specified areas with deer-proof fencing. Several research groups have constructed deer exclosures to test the hypothesis that tick populations will be reduced where this host is excluded. The results of these studies have been striking in their inconsistency. In Westchester County, New York, one of two deer exclosures had a reduction in host-seeking nymphs compared to an unfenced reference site, whereas the other was not different from its reference site (Daniels and Fish 1995). In Lyme, Connecticut, deer exclosures reduced the abundance of host-seeking larvae and nymphs by more than 80% and about 50%, respectively (Stafford 1993). However, exclosures had no effect on abundance of host-seeking adult ticks, suggesting that the nymphs present in deer exclosures survived particularly well. In other cases, deer exclosures have strongly *increased* the abundance of host-seeking ticks of several species (including *I. scapularis*) compared to unfenced reference sites (Perkins et al. 2006). An intriguing synthesis of research on deer-exclosure impacts on

ticks showed that small (~ 1-hectare) exclosures consistently *increase* tick abundances, whereas medium-sized exclosures (2–4 hectares) have no impact, and only those larger than about 4 hectares reduce tick populations (Perkins et al. 2006). These authors suggested that the exclusion of deer causes ticks that would have fed on deer to feed on other hosts, particularly small rodents. In small exclosures, these rodents can easily import ticks from the edges of surrounding unfenced areas into the interior of the exclosure, whereas in larger deer-free zones, tick importation declines in the interior (Perkins et al. 2006). It is also possible that exclusion of deer improves survival probabilities for ticks by protecting vegetation from intense browsing and increasing shading and moisture. It is critical to note that the threshold deer-exclosure size of 1–2 hectares, or 2–4 acres, within which tick populations are likely to increase corresponds closely to the size of individual private properties that people are likely to surround with fences in order to reduce Lyme disease risk.

Interestingly, whenever deer are eliminated, reduced by hunting, or excluded by fencing, the next several years sees an *increase* in the proportion of immature ticks that are infected with Lyme disease spirochetes (Rand et al. 2004, Perkins et al. 2006). Apparently, many of those immature ticks that would have fed on deer instead feed on other hosts, such as small mammals. Because deer are highly unlikely to transmit a spirochete infection to feeding ticks, but many small mammals are quite likely to transmit infection (more on this in chapter 4), the result is an increase in tick infection rates. Taking away deer, at least initially, removes the protective role they play in reducing tick infection (LoGiudice et al. 2003).

Even where deer populations have not been eliminated or reduced for experimental purposes, we know that deer populations naturally undergo ups and downs and vary from place to place, and one might expect that these changes might cause corresponding fluctuations in the tick population. On Long Island, New York, white-footed mice occupying forest sites that were used more intensively by deer were infested with more immature ticks than were those in sites used less intensively by deer (Wilson et al. 1990). Studies in coastal Maine showed that abundance of host-seeking adult ticks positively correlated with that of deer, and that these correlations occur both when many small sites are analyzed and when fewer, larger sites are examined (Rand et al. 2003). However, extensive, long-term studies in northern New Jersey (Schulze et al. 2001, Jordan and Schulze 2005) and southeastern New York (Ostfeld et al. 2006a) found no relationship between deer abundance and that of larval or nymphal ticks. In the latter case, deer abundance was estimated at six sites over a 13-year period.

Despite varying threefold among sites and years, deer abundance did not predict abundance of host-seeking nymphal ticks (Ostfeld et al. 2006a).

So, what do all these studies tell us about the relationship between deer and tick abundances? It is sometimes strong and sometimes weak or nonexistent. It can vary from place to place. The different tick life stages respond inconsistently, as do host-seeking ticks and those attached to rodent hosts. The relationship might depend on the starting density of deer or of ticks. It seems to depend on the size of deer-free zones. It seems to be stronger in coastal or island localities than inland. Given the variable results described above, it is hard to support the conclusion that deer density and tick density are tightly coupled. The long-held, entrenched notion that deer are “indispensable,” “primary,” “keystone,” and “definitive” needs to be replaced by a broader view of the factors responsible for regulating numbers of blacklegged ticks.

Why might the relationship between deer and tick abundance be so variable and sometimes weak or nonexistent? Ecologists expect species to be tightly coupled and their populations interdependent when one is a specialist on the other. For example, lynx are specialized predators on snowshoe hares; as hare populations go up, so too do lynx (after a time lag that corresponds to the lynx's generation time) (Elton 1966). But *Ixodes scapularis* is not a specialist on white-tailed deer. This tick species has been found on at least 125 species of North American vertebrates and is quite abundant on many of them (Keirans et al. 1996). Even the adult stage, which is frequently described as a specialist on deer, has been documented on 27 species of mammals (Keirans et al. 1996). Unfortunately, to my knowledge, no investigators have rigorously determined the relative abundance of adult blacklegged ticks on deer versus raccoons, opossums, skunks, foxes, and other common hosts on which they are regularly found. Ideally, such a study should be conducted by comparing among sites or among years in which deer abundance varies. When they seek a host, adult ticks climb understory vegetation to a height of roughly half a meter to a meter and grab hold of vertebrates that brush by (see figure 9). No one knows to what degree they are selective while questing—whether they avoid some hosts while favoring others or climb on the first host they encounter irrespective of species. If deer are the most abundant vertebrate in the size range that adult ticks encounter when questing, then more adult ticks might feed from deer than from other hosts. But if deer are scarce—whether from natural or anthropogenic causes—then more adult ticks might feed from these other nondeer hosts. Some of these hosts, such as opossums and raccoons, can reach abundances that exceed those of deer (see LoGiudice et al. 2003 and

references therein). It seems reasonable to conclude that one reason tick populations are not always closely tied to deer populations is because the ticks have other hosts that can support them.

Deer that are killed by hunters in November are often infested with several dozen to more than a thousand adult blacklegged ticks (Main et al. 1981, Wilson et al. 1985). The female ticks embed their mouth parts and imbibe deer blood for up to a week, expanding from the size of a sesame seed to that of a small jellybean. During this time, the males wander over the deer, mating with the immobile females while sometimes taking brief blood meals to fuel their meanderings. Females that finish their blood meal drop off the deer and overwinter on the forest floor before laying a mass of about 2,000 eggs the next spring or summer. So a quick, back-of-the-envelope calculation shows that if a typical deer feeds a total of 2,000 adult ticks in any given autumn (500 per week for four weeks), of which one-half (1,000) are females, and half of all the eggs laid by those female hatch, the result from that one deer will be one million larvae. It quickly becomes evident that only a very small number of deer is necessary to produce astronomical numbers of ticks. If we double deer density, we get two million larvae; quadruple, and it's four million. But we don't find millions of ticks—of any life stage—in an area the size of a deer's home range. We might find up to a few thousand larvae in an area this size no matter how abundant deer are. Is it important whether the initial number of newly hatched larvae is four million or only one million, if a maximum of a few thousand will survive to seek a host? The point here is that other factors besides deer abundance are certainly involved in regulating abundance of ticks. Moreover, whatever these other regulatory factors are, they are likely to become increasingly important as the abundance of deer increases. This is simply because, if a relatively low threshold of deer abundance saturates the environment with ticks, then increasing density of deer above this threshold will not increase tick survival. Interestingly, the leading models of tick populations suggest that deer abundance thresholds *are* critical (Mount et al. 1997, Van Buskirk and Ostfeld 1995).

White-tailed deer have an enormous geographic range (Kays and Wilson 2002) that encompasses that of the blacklegged tick (figure 13). But deer were extirpated, or nearly so, over much of their range in the late 1800s to early 1900s as a result of rampant deforestation of the landscape and overhunting. Some deer survived, particularly in the southern United States, and deer populations elsewhere were reestablished by translocations (McShea et al. 1997). Over much of their eastern range, deer populations have increased in numbers and expanded during the twentieth century.

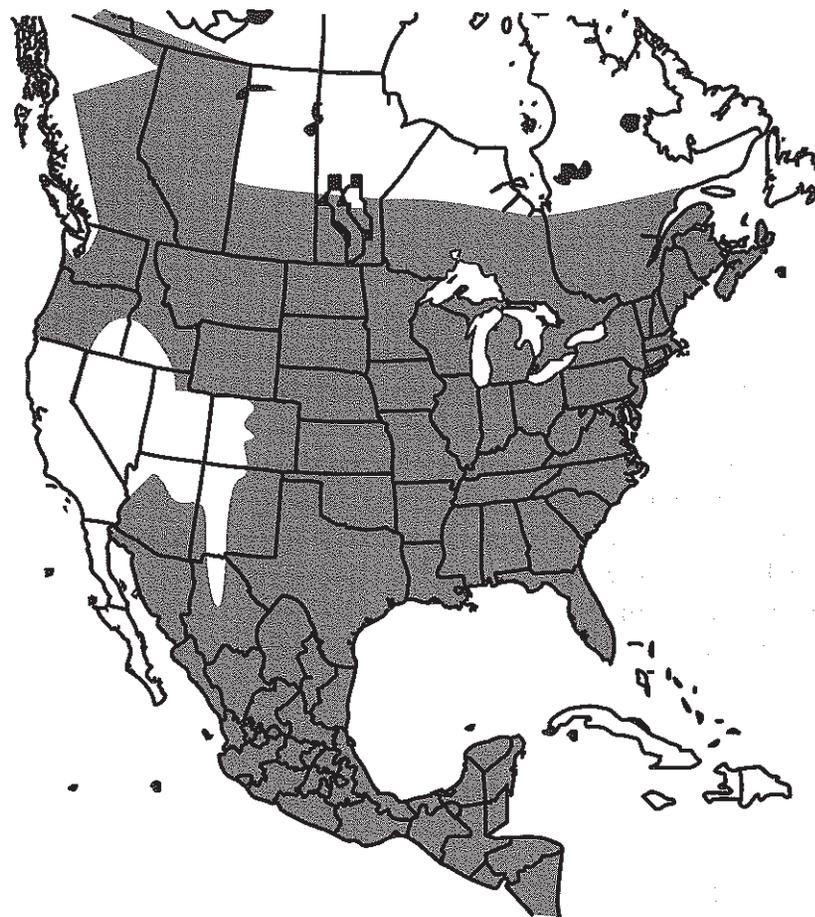


FIGURE 13. Geographic range of the white-tailed deer in North America. *Source:* Kays and Wilson (2002). Reprinted with permission.

When food is abundant, female deer can reproduce at two years of age and produce one or two fawns every year until they die. The ability of deer populations to grow quickly and expand in range has been attributed to regrowth of forests, combined with habitat fragmentation that created ideal habitat (juxtaposed forests and fields), plus the eradication of large predators such as cougars and wolves (McShea et al. 1997). It is dogma that the emergence of Lyme disease in the 1970s and 1980s was caused by the reestablishment of abundant deer populations after twentieth-century reforestation and predator decimation in the eastern United States (Spielman et al. 1985, Lane et al. 1991, Barbour and Fish 1993, among many others). The widely accepted scenario for the emergence of Lyme disease is

that northern populations of blacklegged ticks survived in small refuges where the forest had never been completely cleared and thus deer had not been extirpated. The north shore of Long Island has been postulated as the main refuge (Barbour and Fish 1993, among many others). All around this refuge the forest regrew, deer habitat increased in quality, and deer reoccupied the landscape, setting the stage for ticks and Lyme disease to invade. Locations nearer to Long Island (for example, Lyme, Connecticut, which is just across Long Island Sound) were invaded earlier than those farther away.

Like many historical reconstructions, this scenario is probably impossible to evaluate rigorously. It seems plausible but also raises more questions than it answers. For example, deer and blacklegged ticks apparently survived the deforestation of the eighteenth and nineteenth centuries in many other locations in the southeastern, northeastern, south-central, and midwestern United States; why have these areas not seen similar invasions of Lyme disease? Why is there no correspondence at these large spatial scales between deer abundance and Lyme disease cases (figures 14 and 15)? Why was southern Connecticut invaded by Lyme disease in the 1970s, when deer had apparently been abundant for decades? More generally, why is there a long delay between the reestablishment of dense deer populations

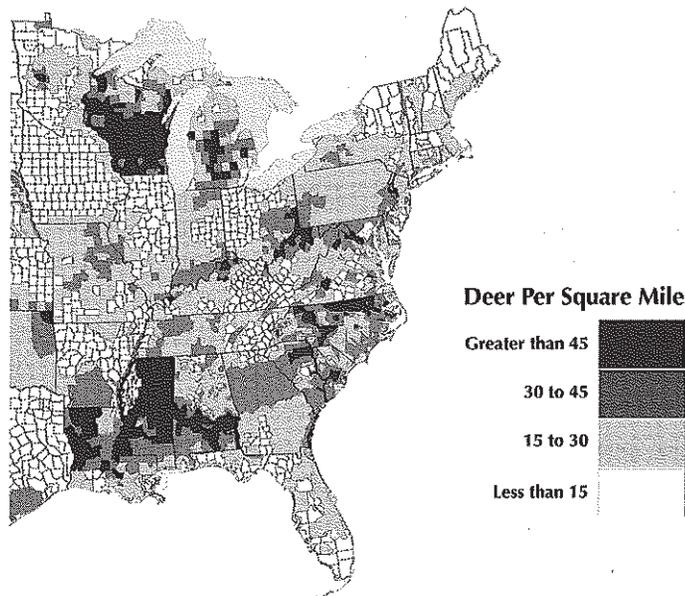


FIGURE 14. Distribution of deer population abundances in the United States. *Source:* The Quality Deer Management Association http://www.i-maps.com/Qdma/frame/default1024_ie.asp?C=48449&LinkID=0&NID=0&cmd=map&TL=100000&GL=010100

Annual Rate* of Lyme Disease by county of residence United States 1992 - 2006

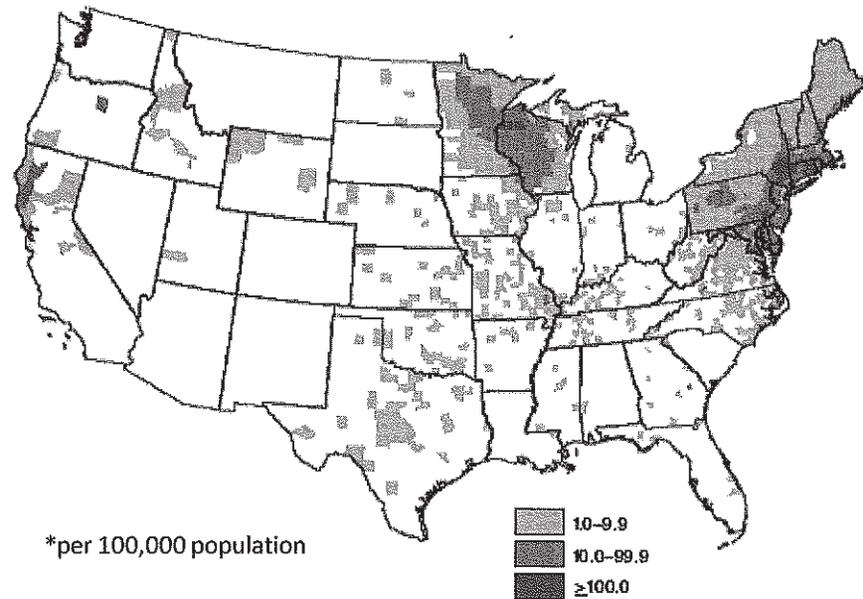


FIGURE 15. Distribution of Lyme disease incidence rates as reported to the United States Centers for Disease Control and Prevention by state health departments from 1996 to 2002. *Source:* CDC (2008). Reprinted with permission.

and that of Lyme disease, and why do these lag times appear to vary so dramatically from place to place? Perhaps, as discussed above, a particular threshold of deer density is necessary before tick populations and Lyme disease can be perpetuated, and the time lags reflect differences in when they exceed this threshold in different areas. But this notion does not by itself explain why ticks and Lyme disease don't occur in many areas with deer herds at least as abundant as in Lyme-endemic areas. Perhaps a threshold level of deer abundance is necessary but not sufficient; one also needs to have a massive influx of ticks—perhaps imported on migrating birds—to get the tick population off and running. But this by itself can't explain why Lyme disease is so rare in parts of the country with abundant deer and ticks. Perhaps it is necessary to have a threshold number of deer to support ticks, plus a source of tick importation, plus abundant wildlife species that feed the immature stages and are efficient at transmitting the Lyme disease spirochete to ticks (more about this in chapter 4). Notice that the scenario is getting increasingly removed from the notion that only one word—deer—