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REVIEW ARTICLE

Lyme Disease: What the Wilderness Provider Needs to Know

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Lyme disease is a multisystem tickborne illness caused by the spirochete *Borrelia burgdorferi* and is the most common vectorborne disease in the United States. Prognosis after initiation of appropriate antibiotic therapy is typically good if treated early. Wilderness providers caring for patients who live in or travel to high-incidence Lyme disease areas should be aware of the basic biology, epidemiology, clinical manifestations, and treatment of Lyme disease.

Key words: Lyme disease, tickborne disease, Lyme carditis, *Borrelia burgdorferi*, *Ixodes scapularis*, tick

Introduction

Vectorborne diseases, including those spread by ticks, pose a growing risk to outdoor enthusiasts. The 2013 Outdoor Participation Report estimated that nearly 50% of US residents age 6 and older participated in outdoor recreational activity in 2012, accounting for more than 12 billion outdoor excursions.[1] Twelve percent of all US residents reported hiking, 13% reported camping, and 19% reported jogging or trail running.[1] Among hikers there was an average of 18 outings annually with a cumulative 603 million hiking outings in 2012.[1] In the next 30 years, person-days spent hiking, backpacking, and camping are all expected to increase, doubling in some areas of the country.[2]

Lyme disease is a multisystem illness caused by the spirochete *Borrelia burgdorferi,* which is transmitted through the bite of certain species of ticks. The risk of exposure is greatest at the confluence of wooded and grassy areas, where contact between humans and infected ticks can be common. Although infection can be cured quickly with early and appropriate antibiotic therapy, rare deaths in young adults with cardiac involvement have been reported.[3] It is important for wilderness medical providers to understand that risk for transmission of Lyme disease may parallel trends in outdoor recreational activities. Wilderness providers should understand spirochete and vector biology, prevention practices, and treatment strategies to prevent, diagnose, and treat patients with Lyme disease.

Disclaimer: The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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HISTORY

Lyme disease was first described by Steere et al[4] in 1977 after an investigation of a cluster of arthritis cases in children living near Old Lyme, Connecticut, and in 1981 the causative spirochete *Borrelia burgdorferi* was discovered in nymphal *Ixodes scapularis* ticks.[5] Although the discovery of Lyme disease as a clinical entity is relatively recent, evaluation of museum specimens of ticks collected in the early 1900s from the northeastern United States identified spirochete-specific DNA sequences by polymerase chain reaction (PCR).[6,7] Since its discovery, the incidence and geographic distribution of reported Lyme disease cases has increased.[8] The spread of Lyme disease is attributed to changing land-use patterns and deer densities in the Northeast and Midwest, in addition to improved Lyme disease detection and reporting practices.[7–9]

CAUSATIVE AGENT

Borrelia burgdorferi, a member of the eubacterial phylum Spirochaetes, is a vigorously motile, 2-membrane, spiral-

shaped bacterium that has limited metabolic capabilities.[10,11] In Europe and Asia, there are 3 genospecies of *B burgdorferi* sensu lato that commonly cause human illness: *B burgdorferi* sensu stricto, *Borrelia garinii,* and *Borrelia afzelii.* In the United States, human infection is caused by *B burgdorferi* sensu stricto, hereafter referred to as simply *B burgdorferi.*

B burgdorferi has a small, linear chromosome containing at least 17 linear and circular plasmids.[10] Gene sequencing has not identified any lipopolysaccharide, toxin, or secretion system virulence factors.[10,11] Additionally, the spirochete has no genes for cellular biosynthetic reactions such as synthesis of amino acids, fatty acids, enzyme cofactors, or nucleotides, explaining why the organism is fastidious and difficult to culture in vitro.[10]

TRANSMISSION

Transmission of Lyme disease to humans occurs through a bite of certain species of infected *Ixodes* ticks. Ticks are arthropods, not insects, and are closely related to mites and spiders.[12] The vectors for Lyme disease in North America are *I scapularis* (the black-legged) tick in the Northeast and Midwest, and *Ixodes pacificus* (the western black-legged) tick along the Pacific coast.[13] These ticks are flattened dorsoventrally and oval in shape with a capitulum or "false head" extending forward from the body (Figure 1). Distinguishing male from female ticks can be done based on scutum (the hard chitinous plate on the anterior dorsal surface of hard-bodied ticks) size and appearance, but is of little clinical value as both sexes can transmit Lyme disease.[12]

Humans are not part of the natural life cycle of the spirochete. Human infection only occurs when humans enter areas where ticks, infected reservoir hosts, and deer coexist at a level high enough to support a dynamic enzootic cycle (Figure 2). Larval ticks hatch in the spring from eggs laid the previous fall; they are uninfected as the spirochete is not transmitted transovarially.[14] After several days of maturation, the larval ticks climb up foliage and begin to "quest" for hosts (Figure 3), taking their first blood meal in the late summer, typically from small rodents, birds, and deer.[12] This blood meal lasts for approximately 3 days, after which the larva will drop to the ground to digest the meal.[15] This is the first time that the ticks can become infected, most commonly from an infected rodent. It is important to note that although deer can provide the blood meal for the tick, deer are immune to infection and do not transmit the spirochete to the larva.

The larval ticks molt into nymphal ticks that quest again, searching for a blood meal; humans can be incidentally fed on during this time. If the nymph was infected as a larva, then the infected nymph can transmit the spirochete. Nymphal ticks typically feed during the late spring and early summer, with meals lasting approximately 5 days, after which the nymph disengages and falls to the ground.[15] Although both nymph and adult ticks can transmit the spirochete, nymphs are believed to be the principal source of human infection owing to their greater abundance and smaller size, which makes detection more difficult.

The nymph molts to become an adult tick that feeds in the fall, most commonly on deer, although humans can also serve as blood meal sources. Again, the spirochete can be transmitted transstadially from nymph to adult. Adult female ticks consume more than male ticks, reaching more than 200 times their prefeed body weight.[15] Adult ticks feed for approximately 1 week and then drop to the ground.[13,15] If the adult tick feeds on humans, spirochetes can be transmitted. After feeding, adult female ticks can lay approximately 1000 to 10,000 eggs before dying.[12]

PATHOGENESIS

To maintain the enzootic cycle, spirochetes must be viable in both mammalian and tick hosts. There are

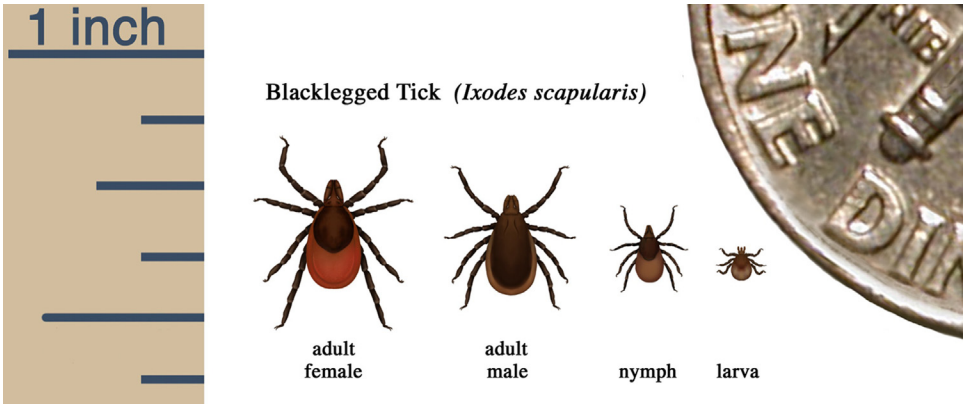

Figure 1. *Ixodes scapularis* life forms. Dime is provided for size reference (figure courtesy of CDC).