

Lyme Disease

Revision Dates

Case Definition	June 2015
Reporting Requirements	June 2015
Remainder of the Guideline (i.e., Etiology to References sections inclusive)	June 2015

Case Definition

Confirmed Case

Clinical illness^[1] with a history of residence in, or visit to, an endemic area^[2] and with laboratory evidence of infection:

- Positive serologic test^a following the two-tier algorithm of EIA (ELISA) and Western Blot.

OR

Clinical evidence of illness^[1] with laboratory confirmation:

- Detection of *Borrelia burgdorferi*^a DNA by molecular diagnostic methods (i.e., PCR) from an appropriate clinical specimen (e.g., skin biopsy, synovial fluid or CSF).^[3]

OR

- Isolation of *B. burgdorferi*^a from an appropriate clinical specimen (e.g., skin biopsy, synovial fluid or CSF).^[3]

Probable Case

Clinical illness^[1] without a history of residence in, or visit to, an endemic area^[2] and with laboratory evidence of infection:

- Positive serologic test^a using the two-tier EIA and Western Blot algorithm.

OR

- Clinician-observed erythema migrans (EM) that occurred within the previous 12 months, without laboratory evidence, but with history of residence in, or visit to, an endemic area^[2].

Case Classification Summary

Case Classification	Exposure to an endemic area ^[2] required	Clinical evidence of illness ^[1] required	Laboratory Criteria	Testing ^a	MD Diagnosis Required?
Confirmed	Yes	Yes*	Yes	Two Step: 1. EIA Positive or Indeterminate 2. Western Blot (IgM and/or IgG) Positive	Yes
	No	Yes*	Yes	PCR or culture	Yes
Probable	No	Yes*	Yes	Two Step: 1. EIA Positive or Indeterminate 2. Western Blot (IgM and/or IgG) Positive	Yes
	Yes	Yes*	Not Required		Yes

*EM may be present or absent.

^a Includes the genospecies *B. burgdorferi*, *B. afzelii* and *B. garinii*.

^[1] **Clinical evidence of illness:** For purposes of surveillance, EM is defined as a round or oval expanding erythematous area of the skin greater than 5 cm in diameter and enlarging slowly over a period of several days to weeks. It appears 1 – 2 weeks (range 3 – 30 days) after infection and persists for up to 8 weeks. Some lesions are homogeneously erythematous, whereas others have prominent central clearing or a distinctive targetlike appearance. On the lower extremities, the lesion may be partially purpuric. Signs of acute or chronic inflammation are not prominent. There is usually little pain, itching, swelling, scaling, exudation or crusting, erosion or ulceration except that some inflammation associated with the tick bite itself may be present at the very centre of the lesion. **Note:** An erythematous skin lesion present while a tick vector is still attached or which has developed within 48 hours of detachment is most likely a tick bite hypersensitivity reaction (i.e., a non-infectious process), rather than EMs. Tick bite hypersensitivity reactions are usually <5 cm in largest diameter, sometimes have an urticarial appearance, and typically begin to disappear within 24 – 48 hours.

OR

Objective evidence of disseminated Lyme disease includes any of the following when an alternative explanation is not found:

- **Neurological:** Early neurological Lyme disease: acute peripheral nervous system involvement including radiculopathy, cranial neuropathy, and mononeuropathy multiplex (multifocal involvement of anatomically unrelated nerves), and central nervous system (CNS) involvement including lymphocytic meningitis and, rarely, encephalomyelitis (parenchymal inflammation of brain and/or spinal cord, with focal abnormalities). Late neurologic Lyme disease may present as encephalomyelitis, peripheral neuropathy, or encephalopathy.
- **Musculoskeletal:** Lyme arthritis is a monoarticular or oligoarticular form of arthritis most commonly involving the knee, but other large joints or the temporo-mandibular joint may be involved. Large effusions that are out of proportion to the pain are typical. Lyme arthritis is often intermittent if untreated, with episodes of joint inflammation spontaneously resolving after a few weeks to a few months. Persistent swelling of the same joint for 12 months or more is not a usual presentation.
- **Cardiac:** Cardiac involvement associated with Lyme disease includes intermittent atrioventricular heart block often involving the atrioventricular node, (although heart block may occur at multiple levels), and sometimes associated with myopericarditis. Carditis can occur in early stages of the disease.

^[2] An **endemic area** is defined as a locality in which ≥ 2 locally-acquired human cases have been reported OR a reproducing population of *Ixodes scapularis* or *I. pacificus* tick vectors is known to occur, which has been demonstrated by molecular methods to support transmission of *B. burgdorferi* at that site (See [Occurrence](#)).

^[3] Consult the Provincial Laboratory for Public Health (ProvLab) Microbiologist-on-Call. Refer to the ProvLab Guide to Services available at: www.provlab.ab.ca/testing.htm

Reporting Requirements

1. Physicians, Health Practitioners and others

Physicians, health practitioners and others listed in Section 22 of the *Public Health Act* shall notify the Medical Officer of Health (MOH) (or designate) of all confirmed and probable cases in the prescribed form by mail, fax or electronic transfer within 48 hours (two days).

2. Laboratories

All laboratories, including regional laboratories and the Provincial Laboratory for Public Health (ProvLab), shall report all confirmed positive laboratory results by mail, fax or electronic transfer within 48 hours (two days) to the:

- Chief Medical Officer of Health (CMOH) (or designate),
- MOH (or designate) and
- Attending/ordering physician.

3. Alberta Health Services

- The MOH (or designate) where the case resides shall forward the preliminary Notifiable Disease Report (NDR) to the CMOH (or designate) of all confirmed and probable cases within two weeks of notification and the final NDR (amendment) within four weeks of notification.
- For out-of-zone Alberta resident cases
 - The MOH (or designate) first notified shall notify the MOH (or designate) of the zone where the client currently resides by mail, fax or electronic transfer and fax a copy of the positive laboratory report within 24 hours.
- For out-of-province and out-of-country non-Alberta resident cases
 - For non-Alberta residents, where the infection was likely acquired within Alberta, the MOH (or designate) of the zone shall forward the preliminary Notifiable Disease Report (NDR) of all confirmed cases to the CMOH (or designate) within seven days of notification and the final NDR (amendment) within two weeks of notification.
 - For non-Alberta residents, where the infection was likely acquired outside Alberta, the following information should be forwarded to the CMOH (or designate) by phone, fax or electronic transfer within 24 hours including:
 - name;
 - date of birth;
 - out-of-province health care number;
 - out-of-province address and phone number;
 - attending physician (locally and out-of-province) and;
 - positive laboratory report (faxed).

Etiology

The causative spirochete of Lyme disease in North America is solely *Borrelia (B.) burgdorferi sensu lato*, first identified in 1982 by Steere and colleagues.⁽¹⁾ Historically, there was published clinical evidence of Lyme disease in Europe, with the description of acrodermatitis chronica atrophicans (ACA) in 1883 by Buchwald^(2,3) and only much later in the United States in 1976, after clusters of children diagnosed with juvenile rheumatoid arthritis, subsequently found to have Lyme disease, were reported.⁽⁴⁾

Currently, 19 genospecies have been described within *B. burgdorferi sensu lato*; three of these have been definitively associated with disease in humans, namely *B. burgdorferi*, *garinii* and *afzelii*.⁽⁵⁾ In North America *B. burgdorferi sensu stricto* is the only genospecies, whereas in Europe, *B. burgdorferi sensu stricto*, *B. garinii* and *B. afzelii*⁽¹⁾ also occur with two recently described pathogens *B. spielmanii* and *B. bavariensis*⁽⁵⁾ associated with occasional infections. *B. garinii* and *afzelii* are also found in Asia.⁽⁶⁾

The spirochete produces a number of differentially expressed outer surface proteins, such as Osp A though F, which are key to its adaptation, survival and infection in many different mammalian and arthropod hosts.⁽⁶⁾

Clinical Presentation

Lyme disease is a multi-system inflammatory disease that ranges from asymptomatic or mild illness to chronic, debilitating illness and may manifest in three stages: early localized, early disseminated, and late persistent infection. A small proportion of infected individuals have no recognized illness or rash or manifest only non-specific symptoms making the clinical diagnosis of Lyme disease difficult.⁽¹⁾ In addition, manifestations of Lyme disease may depend upon the infecting *Borrelia* genospecies, which have been shown to have a predilection for specific organs and sites.⁽⁶⁾ For example, infections with *B. burgdorferi sensu lato* in Europe mainly result in localized EM whereas in the United States and Canada, disseminated illness, with chronic arthritis is a more frequent manifestation.⁽⁷⁾ Similarly, *B. afzelii* frequently causes the skin lesions of lymphadenitis benigna cutis and ACA,⁽¹⁾ whereas *B. garinii* gravitates towards the CNS causing chronic encephalomyelitis.⁽⁸⁾

In the absence of antibiotic treatment, the intact immune response will usually begin to control a disseminated infection and generalized systemic symptoms will decrease.⁽⁹⁾ However, the organism can survive for several years in isolated niches within the body subsequently causing relapsing arthritis, polyneuropathy or other systemic symptoms. Few cases have had relapses beyond five years.⁽¹⁰⁾

For a summary of manifestations by stages refer to [Annex 1: Stages and Manifestations of Lyme Disease](#).

Stage 1 - Early (Localized) Infection

Within 3 – 32 days of a tick bite, a distinctive rash, EM, at the site of the tick bite occurs in about 70 – 80% of individuals.⁽¹¹⁾ Many individuals do not remember the tick bite.⁽⁶⁾ The EM expands slowly in an annular (ring shaped) manner, with central clearing and is generally about 5 cm in diameter. EM lesions can vary greatly in location, size and shape, have vesicular or necrotic areas in the centre, or only partial central clearing and can be confused with cellulitis.^(6,11) Central clearing of the erythema is more common in European cases than North American cases.^(12,13) This is likely because it is identified at earlier stages in North American cases. The rash can be hot to the touch and may be described as burning, itchy or painful.⁽⁶⁾

With or without EM, early symptoms may also include malaise, fatigue, fever, headache, stiff neck, myalgia, migratory arthralgias, and/or lymphadenopathy, possibly lasting several weeks or more in untreated persons.^(1,14)

Stage 2 – Early (Disseminated) Infection

The most commonly reported manifestations are multiple EMs.^(6,11) They may develop within several days to weeks of the onset of the initial EM and may be similar to but smaller than the primary lesion.⁽⁶⁾ These lesions reflect spirochetemia with cutaneous dissemination and usually fade within 3 – 4 weeks (range: 1 day to 14 months).⁽⁶⁾

Systemic symptoms such as fatigue and lethargy are often constant, while arthralgia, musculoskeletal pain, headache, encephalopathy, hepatitis and lymphadenopathy or splenomegaly may intermittently occur in approximately 18% of untreated individuals at this stage.^(6,15,16)

After several weeks to months, approximately 15% of untreated individuals will develop other symptoms of early disseminated illness including, palsies of the cranial nerves (Bell's palsy), meningitis, motor and sensory radiculoneuritis, cerebellar ataxia, myelitis and/or conjunctivitis.^(6,11)

Cardiac manifestations (e.g., arrhythmias, heart block and syncopal episodes due to impaired conduction to the atrioventricular node) may develop in up to 5% of untreated cases⁽¹⁷⁾ and may last 3 days to 6 weeks.⁽⁶⁾ Cardiac involvement is uncommon in children.⁽¹¹⁾

Stage 3 – Late (Persistent) Infection

The most commonly reported symptom in 60% of untreated individuals is relapsing arthritis that usually affects the large joints, especially the knees^(6,11,17) and may occur weeks to years (average 6 months) after the onset of EM.^(1,18) Attacks may last from a few weeks to months with periods of complete remission in between.⁽⁶⁾ Arthritis may occur without prior signs and symptoms of illness (including EM).⁽¹¹⁾ Chronic arthritis is uncommon in children who are treated with antimicrobial agents in the early stage of the disease.⁽⁶⁾

CNS manifestations may also occur including polyneuropathy, leukoencephalitis⁽⁶⁾ and encephalopathy, which may include such non-specific manifestations as sleep disturbance, behavioural changes and headaches.⁽¹⁸⁾

About 5% of untreated individuals may develop chronic neurological manifestations such as spinal radicular pain or distal paresthesias.⁽⁶⁾

ACA, described mainly in European Lyme disease cases, begins with red violaceous lesions that become sclerotic or atrophic. These lesions, which may be the presenting manifestation of the disease, may last for many years, and *B. burgdorferi* has been cultured from such lesions as much as 10 years after their onset in the untreated patient.⁽⁶⁾

Antibiotic-Refractory Lyme arthritis

While the majority of patients with Lyme arthritis respond to appropriate antibiotic treatment approximately 10% may have persistent joint inflammation for months or years after completion of treatment.^(18,19)

Post-Lyme Disease Syndrome⁽⁶⁾

A small percentage of patients complain of pain, and neurocognitive, or fatigue symptoms for months or years afterwards, despite resolution of the objective manifestations of the initial infection with antibiotic therapy.⁽²⁰⁾ Indistinguishable from chronic fatigue syndrome or fibromyalgia, these patients

tend to have more generalized or disabling symptoms: marked fatigue, severe headache, diffuse musculoskeletal pain, multiple symmetric tender points in characteristic locations, pain and stiffness in many joints, diffuse paresthesias, difficulty with concentration, or sleep disturbance.

Patients with these conditions lack evidence of joint inflammation; they have normal neurologic test results; and they usually have a greater degree of anxiety and depression. At the present time there is no evidence that persistent subjective symptoms after recommended courses of antibiotic therapy for Lyme disease are caused by active *B. burgdorferi* infection.^(20,21)

Diagnosis

The diagnosis of early Lyme disease should be based on the clinical picture and epidemiological information (i.e., history of exposure to ticks or travel to Lyme-endemic area).⁽²²⁾ In order to prevent long-term sequelae from Lyme disease, early treatment is the best option. Laboratory testing early in the disease process, prior to the formation of antibodies, will result in a higher number of false negative results (negative when the person is actually a case). A clinical diagnosis (i.e., no laboratory testing required) can be made early in the disease course if the characteristic skin rash (EM) is observed and a history of travel to, or living in, an endemic area is indicated.^(17,18) Consultation with an infectious disease specialist is recommended as differential diagnoses among Lyme arthritis, encephalopathy or polyneuropathy and other syndromes such as chronic fatigue or fibromyalgia is difficult and the management differs significantly. In addition, the Microbiologist-On-Call (MOC) at ProvLab should be consulted prior to specimen collection to determine how and when specimens should be collected.

Currently, antibody detection and laboratory confirmation follows a two-step testing approach,⁽²³⁾ in keeping with the recommendations of the Public Health Agency of Canada (PHAC) and the United States Centers for Disease Control (CDC), to decrease the possibility of reporting false-positives as cases of confirmed infections.^(18,24,25)

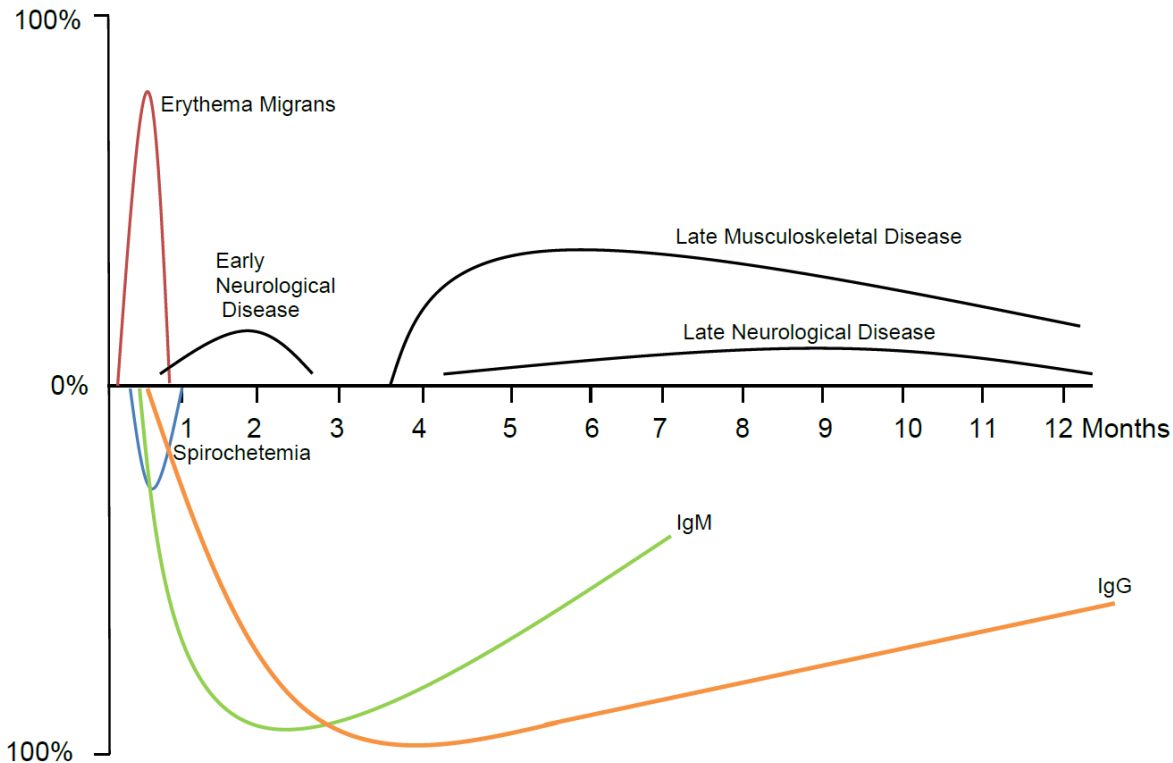
Untreated individuals, who remain seronegative, despite continuing symptoms for 6 – 8 weeks, are unlikely to have Lyme disease, and other potential diagnoses should be actively pursued.⁽²²⁾

Recommendations for Testing and Treatment Strategy:⁽²⁶⁾

Stage of Infection	Recommendation
Stage 1 (Acute): EM (seasonal occurrence, tick-endemic area)	Clinical diagnosis and empirical treatment may be considered (in consultation with an infectious disease specialist)
Stage 1 (Acute): EM (out of season, not a tick-endemic area)	EIA* - repeat in 4 weeks if negative; treatment at physician's discretion
Stage 2/3 (Early Disseminated /Late Infection) Characteristic neurological, cardiac or joint involvement	EIA* - consider PCR of synovial or spinal fluid
No objective findings	Testing or treatment not recommended
Persistent symptoms following recommended treatment	Testing or further treatment not recommended

*EIA with an approved-in-Canada kit and Western Blot confirmation

IgM antibodies generally appear within 2 – 4 weeks of EM onset and peak around six weeks. IgG antibodies appear within 4 – 6 weeks of EM onset and peak around 2 – 3 months. IgM antibodies usually decline to undetectable levels after 4 – 6 months, while IgG can remain detectable for prolonged periods despite treatment.^(27,28)



Adapted from Morrison⁽²⁹⁾

Enzyme Immunoassay

The first test of the two-step testing approach is screening for antibodies to *B. burgdorferi*, *B. afzelii* and *B. garinii* by C6 enzyme immunoassay (EIA). This test is performed at the ProvLab; however this assay cannot distinguish between the three genospecies. The C6 EIA that is used in Alberta is more sensitive (66.5% versus 35.2%, $p < 0.001$) than whole cell sonicate EIAs in patients with early Lyme disease (i.e., can detect antibodies earlier).^(30,31) In addition, the C6 peptide sequence is highly specific to *Borrelia* strains causing Lyme disease, further reducing the cross-reactivity to other infectious organisms seen with previous whole cell sonicate (WCS) EIAs.

Serologic (EIA) test sensitivity is low during the first several weeks of infection and usually may remain negative in persons treated early with antibiotics.^(1,11,18) Approximately 16% of individuals with EM will test positive during the first week of illness.^(32,33) Sensitivity increases to ~50% as individuals progress to the convalescent stage 2 – 4 weeks later. After 4 weeks, and with the presence of other symptoms (e.g., arthritis, acute neurologic or cardiac abnormalities), EIA test sensitivity increases to >99%. Cross-reacting antibodies may cause false-positive reactions on EIA in persons with other spirochetal infections (e.g., syphilis, leptospirosis, and relapsing fever), viral infections (e.g., Epstein-Barr virus, varicella), immunodeficiency (e.g., HIV) and autoimmune diseases (e.g., systemic lupus erythematosus and rheumatoid arthritis).^(1,31,34-39)

In some individuals, IgM responses may persist for many years after treatment,⁽⁴⁰⁾ therefore a positive IgM response should not be interpreted as recent infection or reinfection unless the appropriate

clinical picture is also present.⁽⁶⁾ If patients with resolved past or asymptomatic Lyme infection have symptoms caused by another illness, the danger is that the current symptoms may be attributed incorrectly to Lyme disease.

Sera that screen as reactive (positive) or equivocal/indeterminate are referred to the National Microbiology Laboratory (NML), Winnipeg, for the second test of the two-step: confirmation by western blot (WB) testing, and when appropriate, for the species identity. Results are generally available within 2 weeks of referral. Sera that test negative in the screening assay performed at ProvLab are not referred to NML.⁽⁴¹⁾

Western Blot

The WB test has high specificity and should not be performed without a reactive (positive) or equivocal/indeterminate EIA result because of the increased risk of false positive results.⁽¹⁷⁾ Infrequently, false-positive IgM Western Blot tests can also occur for unknown reasons. If signs and symptoms have been present for 30 days or less, both IgM and IgG Western blot testing can be performed; if signs and symptoms have been present for more than 30 days, only IgG Western blot testing should be performed.

Travel history is obligatory as the Western Blot assay for *B. garinii* and *afzelii* is only performed if travel outside of North America is provided: there is no or minimal serologic cross-reactivity between these three genospecies by the individual Western blot assays in the acute stage of infection (refer to NML guide to services).⁽⁴¹⁾

Molecular Detection

Polymerase chain reaction (PCR) testing has been used to detect *B. burgdorferi* DNA in a variety of samples. Skin lesions have the highest yield, followed by a much lower rate in joint fluids⁽¹¹⁾ and CSF⁽¹³⁾ of serologically positive patients. Testing is available from the NML by special request, and the ProvLab Microbiologist-On-Call (MOC) must be contacted prior to submission of samples.⁽⁴¹⁾

Urine Antigen Detection

Results obtained from the Lyme urine antigen test have been unreliable and are not recommended in the diagnosis of Lyme disease.⁽⁶⁾

Culture

Isolating *B. burgdorferi* from EM lesions, joints, blood and CSF via culture is possible but results are not available in a timely manner and therefore lack clinical usefulness.⁽¹⁸⁾

Epidemiology

Reservoir

The survival and spread of *B. burgdorferi* depends on the availability of a suitable tick vector as ticks are the primary means by which the bacteria can move from one habitat to another. Movement of the organism into new geographic areas requires the presence of both suitable vectors and hosts. Infected hosts can move the disease into areas with uninfected vectors and vice versa.⁽⁴²⁾ Two species of Ixodes ticks act as the primary reservoirs for Lyme disease in Canada: *Ixodes scapularis* (blacklegged tick) in the east and *Ixodes pacificus* (western blacklegged tick) on the west coast.⁽⁴³⁾ The primary vector of Lyme disease in Europe is the sheep tick, *I. ricinus*, and in Asia is the taiga tick, *I. persulcatus*.⁽⁶⁾

In North America, the nymphal stage of *I. scapularis* is most active in biting small mammals and humans in late spring and summer. The adult stage is more active in biting large mammals such as deer, elk and moose which allow it to survive over the winter.⁽⁶⁾

In Europe and Asia, the preferred host of *I. ricinus* is largely debated however, small rodents are important reservoirs for *B. afzelii* and birds are important for *B. garinii*.⁽⁴⁴⁾

Ixodes ticks have been found to also carry other parasites such as *Theileria microti* and *Anaplasma phagocytophilum*, which cause the human diseases babesiosis and anaplasmosis, respectively.

Other tick species, such *I. angustus*, have been or are being put forth as capable of being a reservoir for or transmitting *B. burgdorferi*, however more field research is needed in this area.⁽⁴⁵⁾

Transmission

Lyme disease is a tickborne disease. Infection is transmitted most often through the bite of infected nymphs. Transmission does not occur between infected female ticks and their eggs. In order to transmit disease, the tick must have its mouthparts buried in the skin for between 36 – 72 hours.^(1,6,46) Vector suitability results from molecular and biological factors rather than the number of hosts; Alberta has many ticks that are not competent Lyme disease vectors.

Lyme disease is not transmitted from person-to-person except as noted here. The bacterium has been found in breast milk⁽⁴⁷⁾ and stored blood from blood donations in the United States⁽⁶⁾ resulting in a theoretical remote risk. Transplacental transmission resulting in extremely rare fetal death has been documented.⁽⁴⁸⁾

Incubation Period

The incubation period from infection to the onset of EM is typically 7 – 14 days, but may be as short as 3 days and as long as 30 days.^(46,49)

Period of Communicability

There has been no conclusive evidence of person-to-person transmission.⁽¹⁾

Host Susceptibility

It is believed that susceptibility is universal. Re-infection has occurred in those previously treated with antibiotics for early Lyme disease.⁽⁶⁾ Infection with Lyme disease does not necessarily produce lifelong immunity.^(50,51)

Occurrence

General

Lyme disease occurs mainly in temperate regions of the Northern Hemisphere including Canada, United States, Europe, Russia, China and Japan.⁽⁶⁾ Lyme disease has also been reported in some African⁽⁵²⁾ and South American countries.⁽⁵³⁾ The disease is typically acquired in the spring and summer when ticks are most active.

Lyme disease is the most commonly reported vector-borne infection in the United States, focused along the upper Atlantic coast, the upper Midwest, and on the West Coast.⁽⁵⁴⁾ Over 30,000 cases are reported in the United States each year.⁽⁵⁵⁾ Potential factors contributing to this increase include growing populations of deer that support the tick vector, increased

residential development of wooded areas, tick dispersal to new areas, improved disease recognition, and enhanced reporting.

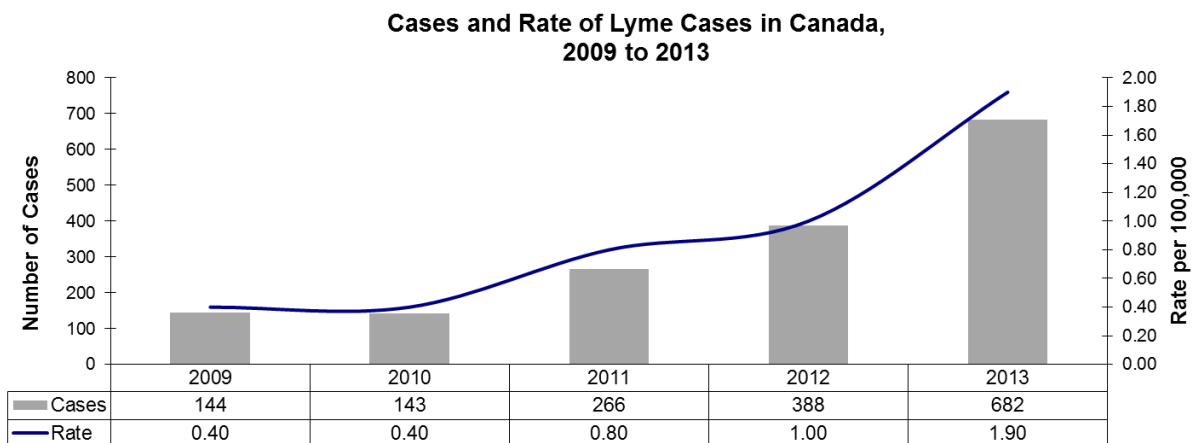
B. burgdorferi is the predominant strain in North America. *B. afzelii* and *B. garinii* cause most European cases of Lyme disease, although *B. burgdorferi* is also present in Europe.⁽⁵⁶⁾

For more information on endemic locations refer to:

- [United States Centers for Disease Control](#)
- [European Centers for Disease Control](#)

Canada

Lyme disease was made nationally notifiable in Canada in 2009. The number and rate of Lyme disease cases has been increasing in Canada. The rate in 2013 (1.9 cases per 100,000) was over 4 times higher than the rate in 2009 (0.4 cases per 100,000).

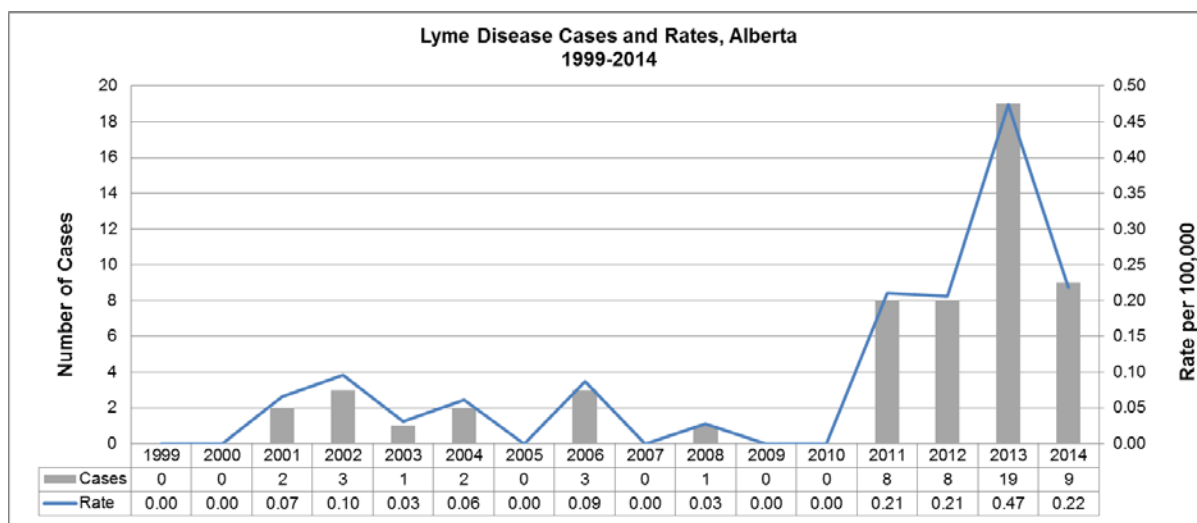


Source: National Lyme Disease Surveillance in Canada, 2009-2012. Available at: <http://www.healthycanadians.gc.ca/diseases-conditions-maladies-affections/disease-maladie/lyme/report-rapport-2009-2012-eng.php> and Canadian Notifiable Disease Surveillance System Tables, 2013: National rate and count (per 100,000 population) of reported cases by age group and sex (Unpublished data).

Established *I. scapularis* populations (endemic areas) have been identified in southern Ontario, Nova Scotia, New Brunswick, southeastern Manitoba and *I. pacificus* in parts of southern British Columbia.⁽⁴³⁾ The prevalence of *B. burgdorferi* in *I. pacificus* is much lower than in *I. scapularis*.^(43,57-59) In addition, up to 12% of bird-borne “adventitious” nymph ticks that survive and moult into adults may be infected with *B. burgdorferi*.⁽⁶⁰⁾

Alberta

Sixty-three cases of Lyme disease were reported in Alberta between 1991 and 2014.⁽⁶¹⁾ All cases were acquired outside of Alberta.



Alberta has an enhanced tick surveillance program and people are encouraged to “submit-a-tick” found on themselves, their pets or the environment for testing. This program will help Alberta Health better understand the risk of acquiring Lyme disease in Alberta. Ticks are speciated at the Parasitology Laboratory at Alberta Agriculture and Forestry. Any blacklegged ticks speciated are tested for *B.burgdorferi*. To date no supporting evidence for the establishment of the major Lyme disease vectors (*I. scapularis* and *I. pacificus*) in Alberta has been found. More information can be found on the Alberta Health [website](#).

Physicians can submit ticks to the ProVLab for speciation if it is required to assist in the diagnosis of their patients. Any blacklegged ticks found are then sent to the National Microbiology Laboratory to be tested for *B. burgdorferi*. Refer to the ProVLab’s [Guide to Services](#).

Key Investigation

Single Case/Household Cluster

- Determine history of recent tick exposure. Risk factors include:
 - travel to a known endemic area;
 - residential exposure during property maintenance, recreation, and leisure activities in known endemic areas;
 - living or working in areas surrounded by woods or overgrown brush infested by known tick vectors;
 - participating in recreational activities such as hiking, camping, fishing, and hunting in tick habitat; and
 - engaging in outdoor occupations such as landscaping, brush clearing, forestry, and wildlife and parks management in endemic areas.
- Determine presence or history of EM-like rash or other clinical symptoms.
- Identify others who may have been exposed to the same source.

Control

Treatment of a Case

- Treatment with an appropriate antibiotic may be started prior to laboratory confirmation if clinical exam and exposure history are suggestive of Lyme disease. Consultation with an infectious disease specialist is recommended.

Management of Contacts

- Lyme disease is not transmitted from person-to-person, however, it may be prudent to identify others who may have been exposed to the same source (e.g., endemic location) so they can be educated in order to monitor for the signs and symptoms of Lyme disease.
- Infants born to women infected with Lyme disease while pregnant should be assessed by an infectious disease specialist.

Post-Exposure Prophylaxis after a Tick Bite

- A single dose of doxycycline may be offered to adult patients (200 mg dose) and to children >8 years of age (4 mg/kg up to a maximum dose of 200 mg) when all of the following circumstances exist:⁽⁶²⁻⁶⁴⁾
 - the attached tick can be reliably identified as an *I. scapularis* tick that is estimated to have been attached for over 36 hours on the basis of the degree of engorgement of the tick with blood or of knowledge about the time of exposure to the tick;
 - prophylaxis can be started within 72 hours of the time that the tick was removed;
 - ecologic information indicates that the local rate of infection of these ticks with *B. burgdorferi* is >20% (e.g., known endemic areas; refer to [Occurrence](#)); and
 - doxycycline treatment is not contraindicated (e.g., pregnant, < 8 years of age).

Preventive Measures

- A Lyme disease vaccine is no longer available. The vaccine manufacturer discontinued production in 2002 after anecdotal reports of joint reactions associated with vaccination, accompanied by lawsuits, resulted in insufficient consumer demand and sales.⁽¹⁾
- Educate the public about ways to reduce transmission of tick-borne diseases, including:
 - removing brush and leaf litter from around the home, and
 - creating a buffer zone of wood chips or gravel between forest and lawn around the home.
- Avoid tick-infested areas when possible (see [Occurrence](#)).
- Use personal protective measures to minimize exposure and reduce the risk of tick bites.
 - Wear long sleeved shirts and long pants that are tight at the wrist and ankles or tucked into gloves or socks.
 - Light coloured clothing can aid in the detection of ticks that have not yet attached.
 - A hat should be worn where contact with vegetation cannot be avoided such as in dense woods, high grasses, or thickets.
 - Apply insect repellents containing 20 – 30% N,N-diethyl-3-methylbenzamide (DEET) (adults only) or up to 20% Picardin (anyone over 6 months of age) to the skin.⁽⁶⁵⁾
 - For children age 2 – 12 years, insect repellent with <10% DEET may be used up to 3 times per day and once per day for children 6 months to 2 years of age. DEET is not recommended for use on children under 6 months of age.
 - Apply permethrin to tents, pant legs, and sleeves. Do not apply directly to skin.
- If working or playing in a tick-infested area, daily inspection and prompt removal can prevent transmission of disease.
 - Removal of ticks within 24 hours of attachment usually prevents transmission of *B. burgdorferi*.^(57,66)
 - Ticks often attach to moist or hairy areas of the body such as the groin, axillae, neck or head.
 - In small children, ticks may be found on the head and neck, which are uncommon places for them to attach in adults.
 - Pets that spend time in tick-infested areas require daily inspection and prompt removal of ticks.
- Remove any attached ticks carefully without crushing.

- Grasp gently with tweezers as close to its mouth as possible (the part sticking into the skin).
- Slowly pull the tick straight out without jerking or twisting.
- Check the bite area daily for at least two weeks.
- If a red rash appears seek medical attention.
- Protect hands with gloves, cloth or tissue when removing ticks from humans or animals.
- Wash hands thoroughly following tick removal.

Superseded

ANNEX 1: Stages and Manifestions of Lyme Disease⁽⁶⁾

Body System[†]	Early Infection		Late Infection
	Localized Stage 1	Disseminated Stage 2	Persistent Stage 3
Skin	Erythema migrans (EM) – with or without central clearing	Secondary annular lesions	Acrodermatitis chronica atrophicans
		Malar rash	Localized scleroderma-like lesions
		Diffuse erythema or urticaria	
		Evanescent lesions	
		Lymphocytoma	
Musculoskeletal		Migratory pain in joints, tendons, bursae, muscle, bone	Prolonged arthritis attacks
		Brief arthritis attacks	Chronic arthritis
		Myositis[‡]	Peripheral enthesopathy
		Osteomyelitis[‡]	Periostitis or joint subluxations below acrodermatitis
		Panniculitis[‡]	
Neurologic		Meningitis	Chronic encephalomyelitis
		Cranial neuritis, facial palsy	Spastic parapareses
		Motor or sensory radiculoneuritis	Ataxic gait
		Subtle encephalitis	Subtle mental disorders
		Mononeuritis multiplex	Chronic axonal polyradiculopathy
		Pseudotumor cerebri	
		Myelitis[‡]	
		Cerebellar ataxia[‡]	
Lymphatic	Regional lymphadenopathy	Regional or generalized lymphadenopathy	
		Splenomegaly	
Heart		Atrioventricular nodal block	
		Myopericarditis	
		Pancarditis	
Eyes		Conjunctivitis	Keratitis
		Iritis[‡]	
		Choroiditis[‡]	
		Retinal hemorrhage or detachment[‡]	
		Panophthalmitis[‡]	
Liver		Mild or recurrent hepatitis	
Respiratory		Nonexudative sore throat	
		Nonproductive cough	
Kidney		Microscopic hematuria or proteinuria	
Genitourinary		Orchitis[‡]	
Constitutional systems	Minor	Severe malaise and fatigue	Fatigue

From Steere AC. Lyme disease. *N Engl J Med.* 1989;321:586.

*The staging system provides a guideline for the expected timing of the different manifestations of the illness, but this may vary in an individual case.

†The systems are listed from the most to the least commonly affected.

‡Because the inclusion of these manifestations is based on one or a few cases, they should be considered possible but not proven manifestations of Lyme disease.

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