Purple Paper

Lyme Disease in Canada: An Update on the Epidemiology

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1. Introduction

Lyme disease is the most common reported vector-borne disease in North America, and has been endemic in Canada since the early 1980s.[1] The Canadian range of Lyme disease is expanding, and it is currently endemic in areas of six provinces: British Columbia, Manitoba, Ontario, Quebec, New Brunswick, and Nova Scotia. Saskatchewan and Prince Edward Island have each documented a single recent case that is likely local. Diagnosis of Lyme disease requires an estimation of 'exposure', which is complicated by this patchwork distribution. Where formerly exposure could be defined by a history of residence or travel, now clinicians and public health officials need to estimate the risk for a Canadian locale to be endemic for Lyme disease. Since the limit for the tick vectors that transmits Lyme disease is shifting north effective monitoring is required to constantly reassess the tick's presence. in the near future most Canadians will probably live in regions where the tick has spread and Lyme disease will be endemic.

The purpose of this Purple paper is fourfold: 1) to review the current epidemiology of Lyme disease in Canada, 2) to summarize the vector ecology of Lyme disease and how this informs surveillance, 3) to discuss projections of the spread of Lyme disease, and 4) to identify other tick-borne diseases in Canada and describe their co-occurrence with *B. burgdorferi*. This Purple Paper has a companion, which outlines the diagnostic pathway for Lyme disease.

2. Epidemiology in Canada

Lyme disease is an illness caused by the spirochete bacterium *Borrelia burgdorferi.* It was originally identified as a tick borne infection in 1978 in the town of Lyme, Connecticut and the cause, *B. burgdorferi* was discovered in 1982. This bacterium occurs in nature as a harmless saprophyte in ticks of the genus *Ixodes* (in North America, mainly *I. scapularis* but also *I. pacificus* and possibly others) and is transmitted to a number of mammals and songbirds. In humans, infection by *B. burgdorferi* begins with a cutaneous phase marked by an acute febrile illness similar to influenza – low grade fever, arthralgia, myalgia, nausea – usually with the nearly pathognomonic bull's-eye rash.[2,3] If untreated, the bacteria can disseminates and localize in multiple organs. Early disseminated disease frequently manifests with neurologic (Bell's palsy, cranial neuritis, radiculoneuritis, meningitis) or cardiac (atrioventricular block, myopericarditis, pancarditis) symptoms; later disseminated disease produces the arthritis that is characteristic of Lyme[4] and further neurologic symptoms.[5] The course of the infection can be highly variable. Treatment is straightforward, a two week course of one of several antibiotics (doxycycline, amoxicillin, cefuroxime, ceftriaxone), and is most effective during the acute Lyme illness; as the disease progresses the chance of extended sequelae becomes greater, but these are probably the residual effects of inflammation. To date there is no definitive proof of continuing infection following treatment.[6,7,8]

In the United States ~34,000 confirmed cases were reported in 2011; however it is estimated that actual annual incidence exceeds 300,000.[9] There were 258 reported confirmed Lyme infections in Canada in 2011.[10] Table 1 compares the incidence of Lyme disease in Canada with American border states.

Table 1. Lyme disease incidence in Canada and the United States (cases/100,000, except where noted)

Canadian province	Incidence	American state	Incidence[11]
British Columbia[12]	less than 0.5	Washington	0.2
		Oregon	0.1
		Idaho	0.0
Alberta13 (average 1998-2012)	0.05	Montana	0.6
Saskatchewan[14] (historic total)	'occasional sporadic cases'	North Dakota	1.4
Manitoba15	0.64	Minnesota	16.9
		Wisconsin	23.9
		Michigan	0.8
		Ohio	0.4
Ontario (2010)[16]	0.72	New York	10.4
Quebec[17]	0.21	Vermont	61.7
New Brunswick[18] (average 2005-2012)	0.27	New Hampshire	75.9
Nova Scotia (2011)[19]	5.8	Maine	66.6

Table 2. Annual reported cases in endemic Provinces

Province[a]	# Annual cases[b]	% Indigenous infections[c]	Province	# Annual cases	% Indigenous infections	Province	# Annual cases	% Indigenous infections
British Columbia[20]	18 (2012)	50%	Ontario	95 (2010)	74%[21]	Prince Edward Island[22]	3 total cases	33%
Alberta	2	0%	Quebec	17 (2012)	22.5%[d,23]	Newfoundland[24]		0%
Saskatchewan	'occasional sporadic cases'	1 likely	New Brunswick (av. 2005-12)	2	unknown			
Manitoba	13 (2013)	69.5%[25]	Nova Scotia	35 (2011)	84%			

[a] Unless noted, sources are the same as the previous table.

[b] Confirmed cases for the most recent year available.

[c] This figure does not correspond to the same year as the # annual cases and often represents combined data from several years, or a provincial estimate that does not outline data boundaries.

[d] This is the historical average – recently, the proportion of endemic local infections has risen above 50%.

The focal nature of the reported cases and the ten-year incidence trend (cases/100,000) of Lyme disease across Nova Scotia's Regional Health Authorities is depicted in Figure 1.

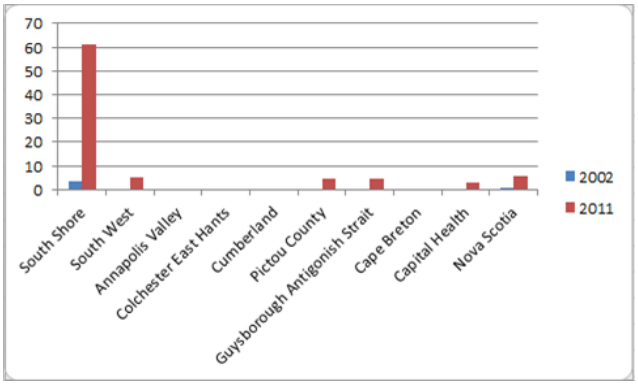

Figure 1. Change in distribution of reported Lyme disease, Nova Scotia.[19]

3. Vector Biology and Ecology

The causative agent of Lyme disease in North America, *B. burgdorferi,* is an obligate parasite that cycles between ticks of the genus *Ixodes* and small mammals or birds.[26] At the public health level, epidemiological tick surveillance identifies changing vector biology and ecology: it enables effective surveillance for *B. burgdorferi*, and predicts future trends.

Ixodes scapularis, the blacklegged tick, is the primary tick vector of *B. burgdorferi* in Central and Northeastern North America, and *Ixodes pacificus*, the western blacklegged tick, is the primary tick vector in the Pacific Northwest. Common hosts of *I. scapularis* include a wide range of mammals, lizards, and birds[27] – of these, competent vectors for *B. burgdorferi* include multiple species[28] of mouse, rat, shrew, chipmunks, squirrels, and at least nine species[e] of passerine birds[29]. The white-footed mouse (*Peromyscus leucopus*) is particularly important in anthropogenic habitats.[30,31] Humans, deer, and other large mammals are not often reservoirs for transmission of the *B. burgdorferi*.[27] White-tailed deer have a dual role in the natural prevalence of Lyme disease: they are an important host for adult ticks and contribute to denser tick populations; but at the same time they do not transmit Lyme disease, and may reduce infection in ticks through zooprophylaxis.[60]

A third tick, *Ixodes uriae* (the seabird tick) is a potential vector along the Atlantic seaboard. Ten of 61 *I. uriae* ticks from Gull Island, Newfoundland, were found to be positive for *B. garinii*, a vector of European Lyme borreliosis.[32] This could introduce *B. garinii* into Canadian mammalian populations,[33] and cause Lyme borreliosis in North America with significantly divergent symptoms.

Ixodes ticks have three life stages – larva, nymph, and adult – each feed on blood; and all three stages are capable of receiving or transmitting *B. burgdorferi*. Eggs hatch into larvae in late spring or summer, seek small mammal hosts and feed once; if successful, they moult into nymphs and remain quiescent through the winter. The next spring these nymphs seek and feed again; in the fall they moult into adults, and search out larger mammals, particularly deer, to feed on for a final time. After this they mate, lay their eggs, and die.[34]

Within this life cycle, tick nymphs are the primary transmitter of *B. burgdorferi* to humans.[35,36] Recently-hatched larvae become infected when they feed on an infected host; after moulting into nymphs they search out a second host, and it is this stage that returns the infection to a vertebrate host that may (many rodents, some birds) or may not (humans, white-tailed deer) be a competent vector. This completes the cycle of Lyme disease. The adult female tick can be infective but is less important for human transmission; being larger than the nymph, she is more likely to be found and removed.[37]

Ixodes ticks thrive in the following conditions,[38] apparently particularly where they are expanding into marginal territory:

[e] Bewick's wren, common yellowthroat, dark-eyed junco, Oregon junco, red-winged blackbird, song sparrow, Swainson's thrush, swamp sparrow, white-throated sparrow (Scott et al., 2012).

- humid leaf litter, particularly in deciduous forests
- forest patches
- lower elevation
- wetlands and their borders
- high populations of deer and small rodents

There is an association between several vectors of Lyme disease (e.g. white-footed mouse and eastern chipmunk) and mixed-use, quasi-urban, fragmented habitats.[39] These frequently support high densities of deer and rodents in close proximity to humans. The appearance and rapid spread of Lyme disease in New England over the past 40 years, for example, is likely related to the wide-scale abandonment of agriculture[40, 41] and the development of a patchy ecological landscape that combines suburbia with forest fragments. There is a debate[39] about the relative importance of deer and small rodents, especially *Peromyscus* species and shrews. Isolated patches as small as 50 hectares have been shown to sustain stable transmission of *B. burgdorferi* between ticks and hosts[42], and Lyme should not be considered only a rural infection.

4. Tick Surveillance

There are two levels of surveillance for Lyme disease. The first is for populations of *I. scapularis* (in British Columbia, *I. pacificus*). Finding a population of *I. scapularis* is a sign that Lyme disease will likely become endemic. The second stage is testing local *Ixodes* ticks for *B. burgdorferi*, which is a direct measurement of current risk.

Passive surveillance depends upon people submitting ticks for identification and testing. It can be highly effective, and is the primary method used in Alberta and Saskatchewan, where Lyme disease is not yet endemic. It requires a local specialist to separate *Ixodes* ticks from other species; relevant specimens are sent to a laboratory to be tested for *B. burgdorferi*.

Active surveillance is appropriate for areas that are suspected of becoming endemic. A white cloth is systematically dragged to pick up questing ticks; potential sites include leaf litter, grass, brushy areas and lawns. The ticks collected must then be picked off and identified; *Ixodes* ticks (nymphs and adults) would then be submitted for testing for *B. burgdorferi*. Tick behaviour is highly responsive to local seasonality and surveillance must be timed to coincide with questing activity of tick nymphs.

When submitting a tick for testing, at least 24 hours, and normally more, are required.

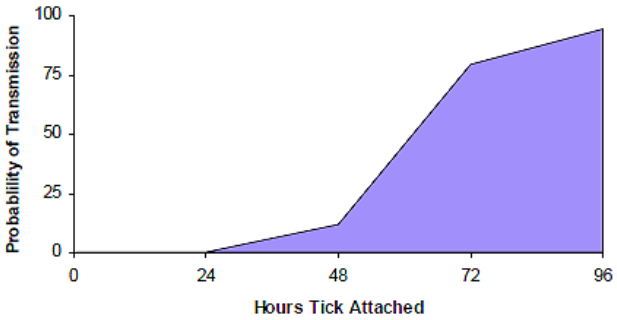

Figure 2. The probability of transmission increases as the tick remains attached (Ontario Ministry of Health and Long-Term Care, 2013).

There are several variables that can inform basic surveillance planning and local announcements. Simple enumeration of the population density of *I. scapularis* nymphs is an excellent indicator of human risk;[35] and as an adjunct, keeping records of the number of ticks submitted for identification/human population can identify emerging *I. scapularis* populations.[43] Large numbers of uninfected blackleg ticks are likely to represent an emerging population – this form of passive surveillance can provide a crowd-sourced data set on the respective movements of *I. scapularis* and *B. burgdorferi*.

White-tailed deer populations correlate with *I. scapularis* populations. Population measures of small rodents can be particularly useful, and one line of research found that two measurements best predicted local risk: populations of white-footed mice and chipmunks the previous year, and acorn density from two years previous.[44] GIS tools that take into account vegetation, soil type, ground

moisture, and other variables can be useful as well.[45]

Piesman[42] emphasizes possibly the most important factor for local management of surveillance, control, and health promotion – the strong association of *I. scapularis* with leaf litter.

5. Current Distribution in Canada and Potential for Change

'Endemicity' of Lyme disease in an area is an important element of diagnosis – knowledge of this lets the clinician estimate a patient's exposure and treat some clinical cases presumptively. PHAC defines an endemic site as follows:[46]

A confirmed Lyme disease endemic area is defined as a locality where active surveillance has detected i) reproducing populations of the tick vector as confirmed by the presence of all three stages (larva, nymph and adult) on resident animals or in the environment for at least 2 consecutive years; and ii) the agent of Lyme disease (B. burgdorferi) in ticks and/or wild animal hosts collected from the locality as evidenced by culture or molecular method, (specific PCR) or immunofluorescent antibody staining (IFA).

A suspected Lyme disease endemic area is a locality where active field surveillance has revealed the presence of multiple ticks at one or more visits suggesting that the tick vector is becoming established, and where B. burgdorferi has been detected in ticks or animals collected from the site.

The actual definition of an endemic area is left up to provincial authorities. There are two approaches that can be taken.

1. Site-based. This approach defines any site which meets PHAC criteria as endemic.
2. Region-based. This approach assumes that surveillance is partial, that tick populations expand and contract without observation, and that the presence of a single population carrying *B. burgdorferi* indicates the likelihood of other populations.

Nova Scotia offers an excellent illustration of how these two approaches differ. As noted in a recent provincial report:[19]

The first endemic tick populations in Nova Scotia were identified in Lunenburg County in 2003, in the localities of Garden Lots, Blue Rocks, First Peninsula, and Heckman's Island. The second endemic area, Admiral's Cove Park in Bedford, Halifax County, was identified in 2006. In 2008, Gunning Cove, Shelburne County, was declared an endemic area. In August 2010, areas around Melmerby Beach, Egerton, Kings Head, and Pine Tree within Pictou County were declared endemic. The fifth endemic area to be named in Nova Scotia was Gavelton, Yarmouth County, in December 2011.

A site-based definition would restrict exposure around Halifax to Admiral's Cove Park.

If a less strict definition of 'endemic' is adopted, then human 'exposure' can be estimated by behaviour within a broader region, not visitations to specifically defined areas. This is the approach that Nova Scotia has adopted; its map of public health areas covers the entire Capital region, indicating that a broad range of activities in this area is a clinical indication of exposure. The value of this is in heightened suspicion for Lyme disease in clinicians; the drawback is, presumably, an increase in testing of people who are negative. Whether it is worthwhile depends on local conditions. For example, this approach has basically been adopted for the Montérégie region south of Montreal, where

a large number of incipient *I. scapularis* populations have been documented and tick distribution and infection is a fluctuating mosaic; PHAC's report is '5 known endemic areas', and gives no more detailed information.[46] There is, however, no map that reflects this.

Finally, a reliance on defined endemic sites can be misleading. *Ixodes* ticks are continually being carried by their hosts deep into Canada; *I. scapularis* has been identified from every province, in one instance from Slave Lake, Alberta, a distance of 1760 kilometers from the nearest recognized population of ticks.[47] An estimated 45 million to 175 million *I. scapularis* ticks enter Canada in this manner every year and approximately 12%-15% of these adventitiously dispersed ticks are positive for *B. burgdorferi.*[48, 49] There are at least two possible human infections from this mechanism, in Saskatchewan and Prince Edward Island.

6. Future Increases

Research suggests that in recent years the most significant predictor of change in the incidence of Lyme disease in the United States is latitude;[50] southern states have seen a decrease and northern states an increase in incidence. This is entirely congruent with the recent spread of *I. scapularis* into Canada, and the establishment of breeding populations and local transmission of *B. burgdorferi*. The presence of this tick is the simplest measure for Lyme disease risk; today it is found in southern Manitoba, along a long discontinuous line stretching from southern Ontario to Montérégie, two sites in New Brunswick, and a substantial area of Nova Scotia. Its territory is approaching all the major population centres between Windsor and Halifax. One study estimates that the frontier of *I. scapularis* in Eastern Canada is expanding at 46 km/year.[51] This is independent of *B. burgdorferi* introduction; but it is predicted that by 2020 80% of the population of Eastern Canada (including Manitoba) will be living in a region supporting established populations of *Ixodes* ticks.

Ticks can be introduced in anomalous populations by bird dispersal or carried over the ground by land mammals.[52] Lyme disease establishment occurs in two phases. During the first, *I. scapularis* ticks establish a breeding population; this can be highly focal. Given an endemic population of tick hosts, the likelihood of *B. burgdorferi* eventually being introduced is quite high; the constant 'rain' of ticks from birds provides a founder source for the bacteria, and potential mammalian vectors are common in Canada. It is estimated that in eastern Canada the normal lag between the establishment of an *I. scapularis* population and the introduction of *B. burgdorferi* will be ~5 years, and in central Canada ~3 years.[53]

In British Columbia the opportunity for expansion is less, given that the endemic areas already cover the main population centres: Vancouver and the Lower Mainland, Victoria and southern Vancouver Island[12]. Beyond the short-term prospectus, Brownstein et. al.[54] estimate that by the 2080s northern Ontario and northern Manitoba might be suitable habitat for *I. scapularis*.

7. Similar Tick-borne Diseases

Five distinct tick-borne illnesses are transmitted by *I. scapularis* in the northeastern United States: Lyme disease (*Borrelia burgdorferi*), babesiosis (*Babesia microti*), human granulocytic anaplasmosis (*Anaplasma phagocytophilum*), deer tick virus encephalitis, and *Borrelia miyamotoi* meningoencephalitis.[55] This section will examine the co-occurrence of these and other non-Lyme tick-borne illnesses.

Babesiosis – Babesiosis is transmitted by *I. scapularis* and is caused by the protozoan parasite *Babesia microti* (rarely other *Babesia* species). Its distribution in North America largely coincides with

that of Lyme disease. In one study between 2.9% and 6.2% of blacklegged ticks were infected with both diseases.[56,57] It produces general flu-like symptoms, but not an erythema migrans. Unlike Lyme disease, babesiosis can be transmitted vertically in both humans and tick vectors, and also through blood donation. It is a more severe infection than Lyme disease and particularly in splenectomized patients, can be fatal.

Human Granulocytic Anaplasmosis – HGA is transmitted by *I. scapularis* and *I. pacificus* in North America, and its distribution tracks that of Lyme disease at a much lower incidence.[58] It causes flu-like symptoms that are typically more severe than in early Lyme disease, plus thrombocytopenia, leukopenia, and increased liver enzymes; HGA has no distinctive symptom and requires laboratory confirmation for diagnosis.[59] This zoonosis is becoming established in southwestern Quebec[60]. Between 2.3% and 10% of patients presenting with erythema migrans (acute Lyme disease) are co-infected with HGA.[61] Treatment for HGA is the same as for Lyme disease.

Other Borrelia species, North America – Tick-borne relapsing fever (TBRF) is caused by several non-Lyme *Borrelia* species: *B. hermsii*, *B. parkerii*, and *B. turicatae*. TBRF is an unusual infection in Canada and found largely in British Columbia, and seems to be associated with staying in rodent-infested cabins. Its symptoms are general, similar to Lyme disease without the bull's-eye rash, and can be marked by febrile periods of ~3 days with an interval of 7 days. TBRF is carried by various species of the soft tick, *Ornithodoros.*[58]

B. miyamotoi, only recently shown to infect humans, has now been documented from all regions of the United States where Lyme disease occurs, including the West Coast.[62] It can be mistaken for HGA[58] or Lyme disease. Its prevalence in ticks is one eighth that of Lyme disease.[63]

Deer Tick Virus Encephalitis – Powassan virus is the only North American member of the deer tick virus complex. In North America it has two lineages, one carried by *I. scapularis* ticks (DTV lineage) and one carried by *I. cookei* and *I. marxi* (POW lineage). Evidence suggests it has expanded its range since 1977.[64] It is rare in humans and the initial symptoms are nonspecific but it is a more severe infection than Lyme with frequent l progression to life threatening neurological symptoms.[58]

European Lyme borreliosis – 85,000 cases of Lyme borreliosis are reported yearly in Europe. Countries with a notable incidence include Austria, Slovakia, Germany, Bulgaria, France, the Czech Republic, and areas that border the Baltic Sea; however, cases are reported from nearly every European country. As in North America, this disease is spread by *Ixodes* ticks that prefer deciduous woodlands with high humidity.[65]

European tick-borne borreliosis is a cluster of diseases, caused by several species of *Borrelia*; primarily *B. afzelli* and *B. garinii*, but also *B. burgdorferi* and possibly *B. bavariensis.* [66] Their symptoms are variable. When European borreliosis is suspected serological testing with the C6 Elisa either as a stand-alone test[67] or as the second tier following a first tier WCS Elisa,[68] is preferred.

Ehrlichiosis – Ehrlichiosis presents with features similar to HGA, but it is transmitted by *Amblyomma americanum,* the Lone Star tick and found primarily in the South and Southwestern United States.[58] Three species of *Ehrlichia*, *E. ewingii*, *E. chaffeensis and E. muris cause human illness*. The Lone-Star tick appears to be expanding its presence and has been found in southern Ontario but little is known about the epidemiology of this tick.

STARI (Southern Tick-Associated Rash Illness) – STARI is an illness caused by an unknown organism (possibly *Borrelia lonestari*), associated with the bite of the Lone-Star tick; [69] it produces a classic bull's-eye rash but rarely other symptoms such as fever, headache, fatigue, muscle pain[70]. No treatment has been demonstrated to be efficacious, and there is no evidence of long-term sequelae. STARI's principal

clinical significance is its propensity to cause a false-positive diagnosis of Lyme disease. An analysis of different treatment strategies suggests that whenever a patient presents with an EM rash and possible exposure to both LD and STARI, presumptive treatment for Lyme disease is indicated.[71]

Wormser et al.[72] reported the following clinical differences between LD and STARI:

- STARI patients experience symptoms other than EM less often (19% vs. 76%)
- The STARI EM rash is more likely to be circular in shape, and to have central clearing (76% vs. 22%)
- STARI patients less likely to have multiple lesions, and the lesions are smaller (6-10 cm vs. 6-28 cm)

STARI should be considered in the differential whenever a patient presents with erythema migrans and a history of exposure south of Virginia. Virginia and Maryland are currently the two states with the greatest overlap between STARI and Lyme disease.

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Prevention and Treatment of Tick Bites

Lyme disease prevention essentially means either preventing tick bites or extracting ticks within 24 hours of being bitten. More advanced interventions are possible, but have not been evaluated. The following measures are considered effective:

- wear protective clothing: long sleeves, long pants, boots, shirts tucked into pants and pants tucked into socks
- check clothing and exposed skin for ticks every two or three hours, and for a week afterwards
- wear clothes treated with permethrin, and using a repellant containing PMD or DEET (this practice may be most realistic for outdoor workers)
- if bitten, remove ticks properly
- if bitten, monitor for rash, facial palsy, headache or flu-like symptoms, or arthralgia
- if bitten in a location in which Lyme disease is endemic, preserve the tick and consult your GP or public health office about the appropriateness of prophylactic antibiotics.

There is good evidence that most people resist adopting these interventions. They are technically effective (e.g. repellant use; treated clothing,′ mass spraying) but their use in health promotion is not based in strong evidence.

Public health measures should recognize that risk is not evenly distributed and that people *often do not comply* with the recommendations for avoiding ticks, but that educating people on checking for ticks seems to be accepted. Tailoring messages for people who are most likely to be exposed to ticks has been found valuable. For example people who spend a great deal of time outdoors are at risk, but do not need general information on how to remove ticks; instead, they should receive information on the symptoms of Lyme disease and how to avoid tick habitats.

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