Lyme Disease:
Dispelling the Myth

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Fact box

In 2002, there were more than 20,000 cases of Lyme disease in the U.S.[1]

From 1981 to the end of 1998, 127 human cases were reported in Ontario patients with no history of out-of-province travel.[2]

British Columbia has approximately six cases/year and last year in Nova Scotia, Lyme disease was diagnosed in two patients thought to have acquired it near the town of Lunenburg, where deer ticks are now endemic.[3]

Lyme disease was first discovered near Lyme, Connecticut in the 1970s. Since then, the area of endemnicity has gradually expanded, as have the number of cases.

This multisystemic infection is due to *Borrelia burgdorferi*, a fastidious bacterium that has adapted to life in both its mammal hosts and in tick vector.

Ixodes scapularis (also known as black legged or deer ticks) are the principal vector in Central and Eastern North America; *Ixodes pacificus* in British Columbia. On the East coast, the white-footed mouse is the preferred host of both the larval and nymphal stages of the tick and white tail deer are the preferred hosts for the adults.

Humans represent an incidental host and are not involved in the life cycle of the spirochete.

The ticks have three stages in their life cycle:

- larvae,
- nymphs and
- adults.

Luke's Lyme

Luke, 37, presents to his Halifax family doctor with subjective joint complaints and easy fatigueability. Four years earlier, he lived in New England, where Lyme disease was known to be endemic. Luke's family doctor reluctantly performs a screening antibiotic test, which is negative.

For more on Luke, see p.75.

Both nymphs and adults may contain *B. burgdorferi* and most infections occur in the spring and early fall.

What are the clinical features?

Once bitten by an infected tick, the spirochetes migrate outward, producing an enlarg-

Cover Photograph: Deer Tick (Getty Images®)

Truth & Rumour:

Rumour: Lyme is highly endemic and most Canadian cases are undiagnosed.

Truth: Even if half of Lyme cases were missed each year in Canada, this is still a very rare disease.

Rumour: Early onset (within 48 hours) localized erythema represents EM.

Truth: It is an immune response to tick salivary proteins.

Rumour: False negative screening tests are common.

Truth: Almost all immunocompetent individuals with untreated Lyme will develop antibodies. However, rare patients may not develop antibodies if treated very early.

Rumour: Infections require long-term antibiotic therapy and may be incurable.

Truth: There have been very few well-documented cases of treatment failures. Early Lyme is readily treatable with three weeks of amoxicillin or doxycycline; for later stages, courses longer than four weeks duration are seldom required.

Rumour: Long-term prognosis is poor.

Truth: Vast majority of patients with Lyme respond completely to treatment. Persisting symptoms are often those not attributable to Lyme in the first place.

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ing erythematous margin called erythema migrans (EM). EM is typically seen between three and 30 days after a tick bite. The primary infection may be associated with:

- fevers,
- chills,
- headaches and
- nonspecific muscle and joint aches and pains.

The organism spreads hematogenously to other organs with particular tropism for the nervous system, atrioventricular node and joints.

Approximately one-quarter of individuals with Lyme disease have no history of EM. Other manifestations include:

- unilateral facial nerve paralysis,
- aseptic meningitis,
- heart block and
- arrhythmias.

When the persistent phase of Lyme disease develops, it's most often as oligoarticular arthritis, involving larger joints. Later, nervous system complaints may include memory loss, mood changes and difficulty with concentration or sleep. Some patients may complain primarily of fatigue.

How do you diagnosis it?

Laboratory testing for Lyme disease is problematic. For practical purposes, serology is the preferred method of diagnosis. Although enzyme immunoassay (EIA) based screening

tests are sensitive, they suffer from a lack of specificity. Laboratories should confirm positive screening tests with a Western Immunoblot.

As case presentations suggest, Western Blots may also have nonspecific banding patterns and results may be difficult to interpret. When the pretest likelihood is low (*e.g.*, patients living in non-endemic areas or with nonspecific symptoms), the predictive value of positive, unconfirmed screening test or an atypical Western Blot is very low.

Because Web sites are replete with Lyme misinformation, many patients are difficult to reassure. Negative screening tests are often followed by demands for Western Blot testing. Equivocal or negative tests will not reassure many such patients.

References
1. Morbidity and Mortality Weekly Report, 2003; 52(31):741-50.
2. http://www.bccdc.org
3. First Isolation Of Lyme Disease Spirochete, Borrelia Burgdorferi From Blacklegged Tick, Ixodes Scapularis, Collected At Rondeau Provincial Park, Ontario, Canadian Communicable Diseases Report 26-06, 2000.

Further references available—contact *The Canadian Journal of CME* at **cme@sta.ca**.

More on Luke

At the further insistence of the patient, serum is sent to a reference laboratory in the U.S. for Western Blot testing. The Western Blot for immunoglobuline (IgG) antibodies is negative; the IgM antibody test has several positive bands.

The patient is aware of issues around Lyme test sensitivity and specificity and now wishes to be tested for ehrlichiosis and babesiosis and to be treated for Lyme disease.

The low pre-test likelihood and incongruous serology testing suggests Luke almost certainly does not have Lyme disease.

Canadian physicians are likely to see many more cases like Luke's than genuine cases of Lyme disease.