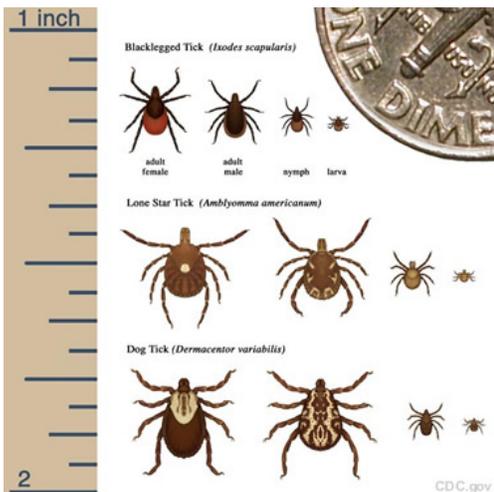


Ticks

TICKS. Just seeing or hearing the word is enough to make your skin crawl. And with good reason. Not just a blood-sucking nuisance, ticks transmit potentially bad diseases, including Lyme disease, Rocky Mountain spotted fever, relapsing fever, tularemia, tick-borne meningoencephalitis, Colorado tick fever, Crimean-Congo hemorrhagic fever, babesiosis, anaplasmosis (ehrlichiosis), and cytauxzoonosis. This handout covers those most common in the Northeast, mid-Appalachians and upper Midwest of the US.

Meet the Ticks

TICKS HAVE BEEN AROUND for 100 million years or so, so it's no surprise that there are almost 2000 species of them. But in North America, depending on how you count them, there are three or four main ticks of medical concern.



Ixodes scapularis, the **deer tick** or **black-legged tick** (or, in the upper midwest, **bear tick**), also known as *I dammini*, is a member of the *Ixodidae* family of hard-bodied ticks. It is the main vector for human Lyme disease. A very similar black-legged tick species in northern California and Oregon, *Ixodes pacificus*, also transmits Lyme disease. The **adult** black-legged tick is fairly large and easy to see, but most cases of early Lyme disease occur during the late spring and summer, when the nearly-invisible **nymph** is seeking a blood meal, and, 20% of ticks are in areas you can't see on yourself,¹ so it's common to get Lyme disease without noticing a tick bite.² Black-legged ticks also transmit anaplasmosis and babesiosis.

Dermacentor variabilis, the **American dog tick**, transmits Rocky Mountain Spotted Fever, tularemia and anaplasmosis.

Amblyomma americanum, the **lone star tick**, transmits STARI or Master's disease (see below), something very similar to Lyme Disease, found in the southern US.

Preventing Tick Attachment

ASSUMING YOU DON'T WANT TO WEAR a flea and tick collar, which doesn't work that well without a built-in fur coat, you may

want to investigate other methods to prevent tick attachment when outdoors.

There are a variety of chemicals that may be applied to the skin to prevent tick (and insect) bites. There is some information in the medical literature,³ but the best information comes from ongoing testing performed by Consumer Reports (<http://consumerreports.org>).

DEET is the most famous, and highly effective, but is mildly toxic, especially in kids,⁴ is greasy on the skin, and turns nylon and many plastics to a disgusting sticky goo. The American Academy of Pediatrics advises against using repellents with DEET concentrations higher than 30 percent on children, and Consumer Reports says that *nobody* needs anything more concentrated than 30%.

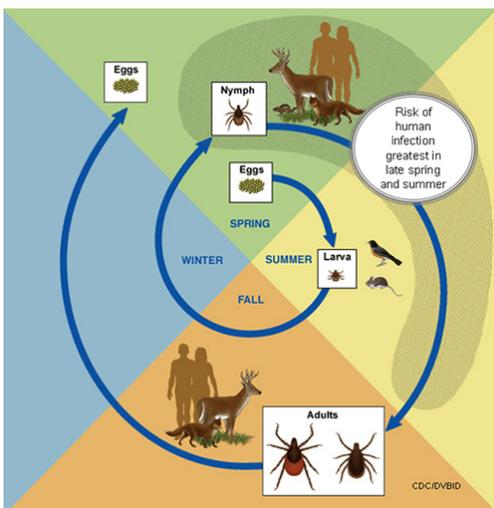
Picaridin (known outside the US as icaridin or KBR3023, tradename *Bayrepel*) is marketed as *Cutter Skinsations Ultra Light*, *Natrapel*, and *Avon Skin So Soft Bug Guard Plus*. It was invented by Bayer and is made by the Lanxess Corporation of Pittsburgh. It is much less greasy on the skin than DEET, doesn't dissolve nylon or plastics, and per the EPA has a low toxicity. It's important to choose a preparation with a vehicle that allows it to persist on the skin; the Cutter preparation lasts only 4.5 hours, whereas the Natrapel lasts for 8 hours. In one study of anopheles mosquitoes, picaridin was more effective than DEET.⁵

Repel is a mixture of lemon oil and eucalyptus, and, quite unlike other "herbal" repellents like *Burt's Bees All Natural Herbal* and *Bite Blocker Xtreme (organic)*, is virtually as effective as DEET.

Skin So Soft is a bath oil sold by Avon. In the 1990s, an urban legend arose that it repelled mosquitos and ticks. As with other urban legends about mosquitos (e.g., Bounce fabric softener, Vick's VapoRub, Lemon Joy detergent and extract of vanilla – see snopes.com for more on urban legends), it turned out to be quite false. Consumer Reports tested *Skin So Soft* and found it ineffective (*Consumer Reports*, Volume 58, No. 7, July, 1993, pages 451 - 454), as did a study by Fradin⁶ (protected against mosquito bites for only 10 minutes). However, the urban legend persisted, and Avon wasn't going to pass up a sales and marketing opportunity like his, so they did the smart thing: they offered a version called *Skin So Soft Bug Guard* with citronella in it, which unfortunately was no better than the original and quickly discontinued. So then they came out with a version containing picaridin, which *is* effective as described above, and then a version with:

IR3535 which is a newer repellent that is nice on the skin and doesn't dissolve your nylon clothes. It's highly effective against ticks for up to 12 hours at 20% concentration, though it's not as effective against mosquitos as picaridin or DEET.⁷ It's available as *Avon Skin So Soft Bug Guard Plus IR3535*, which, despite the confusingly similar name has IR3535 instead of *Bug Guard Plus's* Picaridin.

There is also an *Avon Skin So Soft Bug Guard Plus IR3535 Expedition* SPF 30 which, in addition to 20% IR3535 includes sunblock, a bad combination according to the CDC and Consumer Reports, because insect repellent should be applied sparingly (due to potential toxicity) and sunscreen should be applied liberally and frequently.



Bottom line: Consumer Reports (July 2010) tested and rated these as follows (higher scores better):

98	15-30% DEET
97	Repel
87	Natrapel
73	Avon Skin So Soft Bug Guard Plus IR3535
56	Bite Blocker
24	Burt's Bees

It's also possible to treat clothing with a "repellent." **Permethrin** doesn't really repel ticks, it actually kills them, though it's nontoxic to mammals. You can buy clothing pretreated with permethrin that will last through 70 washings, or treat clothes at home. It's highly effective, especially if you treat your socks with it.⁸

Removing Ticks

THERE ARE MANY WAYS to remove ticks, but only one is best. Some are bad. For example, smothering a tick with Vaseline or goosing it in the rear end with a hot, just-blown-out-match probably isn't such a good idea; not only is this poor at persuading the tick to let go, it may make it vomit into your skin. Twisting to the left or the right tends to rip the body off, leaving the head embedded in the skin (ticks glue their heads into your skin). Grabbing at the neck with a pair of blunt forceps and gently pulling until the jaws tire and the entire tick comes out is best.⁹⁻¹¹

There are a variety of devices that make this easier than using a pair of forceps: I particularly like the *Tick Key* and *Tick Nipper*, both available from REI stores or <http://rei.com>. Don't get devices that say they twist out the tick or use tape to try to suffocate the tick. But even with optimal removal, sometimes the head remains in the skin, and is likely to fester. And plenty of people come to the ED with embedded tick heads.

The best way I've found to get out an embedded tick head is to grab it with a pair of Adson (toothed) forceps, pull, then delicately cut around it with a #15 scalpel blade, then apply a Bandaid. One author recommends an elliptical incision followed by a suture (even for intact ticks)¹² but I prefer to excise very narrowly and shallowly then leave open to avoid infection. One author recommends a biopsy punch,¹³ but one's seldom available in the ED.

Lyme disease

Lyme disease is common here. Indeed, more cases are reported from Pennsylvania than any other state, though mostly from the southeast. And, according to the CDC, Lyme disease was the 6th most common Nationally Notifiable disease in 2008, and the most common tick-borne disease in the northern hemisphere.¹⁴ There were only 27 reported cases in Allegheny County for 2009 (latest statistics) but the CDC estimates that only about 10% of Lyme disease is reported, and incidence is increasing so a reasonable estimate is ~400 cases for Allegheny County in 2011. Going a bit east into the

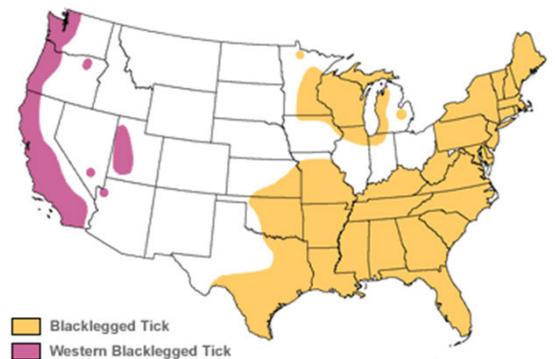
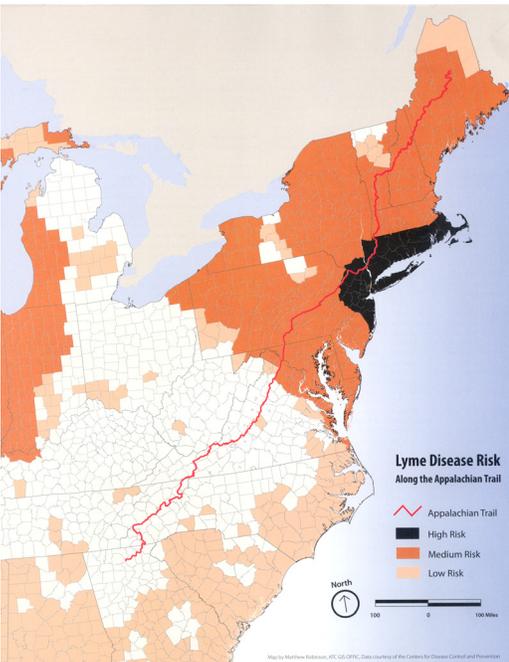
mountains Lyme disease is much more common. A variant of Lyme disease with different characteristics and caused mostly by *Borrelia afzelii* occurs in Europe and Scandinavia,¹⁵ where erythema migrans is more chronic and people sometimes get acrodermatitis chronica atrophicans (a "tissue paper-like cutaneous atrophy") and there are other *Borrelia*, carried by ticks of the same *Ixodes ricinus* complex to which American black-legged ticks belong, that cause similar borreliosis in eastern Europe and Asia.¹⁶

Lyme Ecology

THERE IS GREAT PUBLIC DISCORD between hunting groups and animal rights groups about the role of deer culling in controlling Lyme disease. However, the evidence shows that decreasing the deer population actually causes an *increase* in the incidence of Lyme disease, likely by increasing nymphal ticks feeding on mice.¹⁷

Indeed, the incidence of Lyme disease reflects the population of the white-footed mouse, *Peromyscus leucopus*. In the short term, mouse population and Lyme incidence is determined by the number of acorns two years prior.¹⁸ There is well-known phenomenon called a "mast year," when oaks across the continent produce perhaps 10 times their normal yearly crop of acorns. Mast years occur irregularly, most recently in 2010, and are thought to be linked to an oak reproductive strategy of "predator satiation": keeping production down to control the acorn-eating population, then suddenly producing many more acorns than the squirrels, mice and deer can eat. The environmental trigger, or mode of communication among oak trees, is totally unknown. Other tree species also exhibit mast years, but they are not synchronized with oaks. (And in fact, there are two different groups of oaks that mast on separate schedules, those with pointy leaves and those with rounded leaves; both masted in 2010, round more than pointy.) Based on this, we can expect many more cases of Lyme disease in the summer of 2012.

Looking at the longer term, forest destruction and fragmentation, from suburban sprawl with construction of new strip malls and housing developments, increases the white-footed mouse population, and is thought to be a major cause of the current increase in Lyme disease.¹⁸



Clinical Lyme Disease

IN THE 19TH CENTURY, syphilis was known as **The Great Imitator**, but in 21st century North America, that title has to go to Lyme disease. The incubation period ranges from days to years, and the manifestations are many and varied.

Early localized infection is characterized by **erythema migrans** (which used to be called **erythema chronica migrans** but it's not really that chronic). This is a red, raised hive-like patch which, unlike hives, is *not* migratory, and doesn't go away with SQ epi. It usually appears 3-30 days after the bite. A third of the time it has central clearing except a central red point or area, the other two-thirds of the time it has no central clearing; half of the time it will be on the lower extremities or buttocks.¹⁹ About 10% of the time erythema migrans can develop vesicles or blisters, and it can sometimes be itchy as well as tender, making it easy to confuse with poison ivy dermatitis or ringworm.²⁰ It tends to go away in about four weeks. A patch should be **>2 inches** (5 cm) in diameter to be considered erythema migrans; smaller patches may be just a local allergic reaction to the tick bite.²¹

About 80% of people with early localized Lyme disease will have erythema migrans. Which means it's hard to tell that those other 20% actually have Lyme disease.

Some with erythema migrans will develop low-grade fever, malaise, and myalgias, but cold symptoms or vomiting and diarrhea are rare. Some with early Lyme Disease have similar systemic symptoms but no rash, which makes diagnosis quite difficult, especially because blood tests have many false positives.

Early disseminated infection occurs when, days to weeks following the tick bite, the causative Rickettsial organism, *Borrelia burgdorferi*, spreads through the bloodstream to joints, heart, nervous system, and distant skin sites. This may occur without evidence of early localized infection.

Some people (a tenth to a quarter of those with a single lesion) will develop erythema migrans in areas away from the initial bite.^{19,21} These tend to be on the same extremity but may be anywhere; sometimes they are in places unusual for cellulitis or poison ivy (axilla, groin, popliteal fossa, back), which helps the diagnosis. The classic of multiple target lesions is of my daughter Laurel's leg in 2005 when she was five years old, and the rash was misdiagnosed as hives by her pediatrician. The other picture is the more common and more ambiguous erythema migrans; note the red dot in the center.

Most people recover from early Lyme disease spontaneously.

Late Lyme disease may occur after untreated erythema migrans (it usually goes away in about 4 weeks), or by infection with *B burgdorferi* without erythema migrans. The line between early disseminated infection and late Lyme disease is still unclear, some classifying neurological or cardiac manifestations as one or the other.

As we are concerned primarily with early Lyme disease, here is a high-level overview of this complex subject.

B burgdorferi has some tricks to prevent clearance by the immune system, which may result in persistent infection.²² In untreated patients, cultures up to 10 years later have been positive.²³ It is also likely that Lyme disease can trigger a variety of autoimmune sequelae, which persist after the elimination of all spirochetes, at least in the case of

Lyme arthritis.²⁴

About half of those with erythema migrans have *B burgdorferi* in their blood, and about half of those with untreated erythema migrans will go on to have arthritis, usually in a knee; one out of ten will have neurological symptoms, most commonly facial nerve palsy; and one out of twenty will have cardiac problems, most likely AV block. Treatment prevents these sequelae.¹⁹

Lyme arthritis is intermittent, usually in the knee or other large joints, but in about one out of ten, may become persistent. Due to presumed autoimmune mechanisms, this may persist after elimination of the spirochetes.²²

Neurological involvement in late Lyme Disease comes in two main forms. Most common is involvement of a single nerve, often the facial nerve: Bell's Palsy. But it's quite unclear if all those with Bell's Palsy in endemic areas need a workup for Lyme Disease, though there is a recent recommendation to obtain serological testing in endemic areas.²⁵ Long-term follow-up of those with Lyme Bell's Palsy shows excellent results from standard treatment.²⁶

A percentage of untreated patients will have an indolent lymphocytic encephalo-meningitis, with a variety of neurological and psychological features. These symptoms tend to spontaneously resolve in weeks or months. However, up to 5% of untreated patients may go on to chronic neuroborreliosis, which can be treated by a month of IV ceftriaxone.²⁷

Cardiac involvement in Lyme Disease is most commonly heart block, but may also present as acute myocarditis. (Chronic cardiomyopathy is rare in the US, but has been reported in Europe.)

Chronic Lyme disease, at least to a good approximation, does not exist. Some patients whose Lyme disease has been appropriately treated will have fatigue, musculoskeletal pain, difficulties with concentration and short-term memory, or some combination of these, which is classified as "post-Lyme disease symptoms," and if longer than 6 months, "post-Lyme disease syndrome."²⁸ However, Seltzer et al found out that, if you follow those with acute Lyme Disease for a long time, there is no more chronic fatigue, fibromyalgia or similar chronic problems than with matched controls.²⁹ See below for more on this controversial topic.

Tests for Lyme disease

MOST PATIENTS WITH EARLY LYME DISEASE, particularly if *B. burgdorferi* is still localized to the skin, do not yet have a humoral immune response to the spirochete.¹⁹ The diagnosis of erythema migrans is based on recognition of the characteristic appearance of the skin lesion in persons who live in or have recently traveled to regions in which Lyme disease is endemic.²¹

If you have a patient in the ED with what looks like it might be erythema migrans, there is no point in sending any tests. Blood and skin biopsy cultures are only performed in research laboratories. Serological tests can be performed as a baseline, but prompt treatment with an antibiotic (which is the standard of care) may prevent the convalescent serology from turning positive. If you clinically diagnose a patient with erythema migrans and treat appropriately, then subsequent negative serological tests do not mean that you misdiagnosed the patient, but more likely that you acted in time to prevent a detectable immune response.³⁰

For other possible manifestations of Lyme disease (carditis, neuritis, new heart block), admit or discharge for outpatient workup as seems appropriate. In such cases, it may be appropriate to send some tests from the ED to aid the admitting physician or follow-up physician.

There *are* national standards for serologic testing for Lyme disease. Guidelines established by the Association of State and Territorial Public Health Laboratory Directors and the U.S. Centers for Disease Control and Prevention (CDC) recommend the use of a two-test protocol for the serologic diagnosis





Quest Diagnostics Incorporated

QUEST DIAGNOSTICS INCORPORATED
CLIENT SERVICE: 412.329.7700

SPECIMEN INFORMATION
SPECIMEN: [REDACTED]
REGISTRATION: [REDACTED]
LAB REF NO: [REDACTED]

COLLECTED: [REDACTED]
RECEIVED: [REDACTED]
REPORTED: [REDACTED]

PATIENT INFORMATION

DOB: [REDACTED] Age: 22
GENDER: F

REPORT STATUS: Final

ORDERING PHYSICIAN: [REDACTED]
CLINIC INFORMATION: [REDACTED]
44181
MEDICAL EXPRESS FMCX
50 FREEPORT ROAD
SUITE 500
PITTSBURGH, PA 15215

Test Name	In Range	Out of Range	Reference Range	Lab
Lyme Disease, EIA w/REFL WB Lyme Disease Screen		1.19 H	LESS THAN 0.91 INDEX	U
INDEX			INTERPRETATION	
< OR = 0.90			NEGATIVE	
0.91 - 1.09			EQUIVOCAL	
> OR = 1.10			POSITIVE	

DOE TO RECOMMENDATIONS BY THE FOOD AND DRUG ADMINISTRATION (FDA), ALL POSITIVE OR EQUIVOCAL BORRELIA BURGORDFERI ANTIBODY EIA (SCREENING) TESTS WILL BE FOLLOWED BY THE WESTERN BLOT. THE SCREENING TEST FOR B. BURGORDFERI HAS A LOW PREDICTIVE VALUE FOR A NEGATIVE RESULT WHEN USED TO DETECT EARLY INFECTION AND A LOW PREDICTIVE VALUE FOR A POSITIVE RESULT WHEN EXPOSURE HISTORY, SYMPTOMS, AND CLINICAL FINDINGS ARE NOT CONSISTENT WITH Lyme DISEASE. POSITIVE OR EQUIVOCAL RESULTS SHOULD NOT BE INTERPRETED AS TRUE POSITIVES UNTIL A SECOND-STEP TESTING OF THE SPECIMEN IS DONE USING A METHOD THAT IS MORE SPECIFIC FOR ANTIBODIES TO B. BURGORDFERI (E.G. WESTERN BLOT).

Test Name	In Range	Out of Range	Reference Range	Lab
Lyme Disease AB (IGG, IGM) WB Lyme Disease (IGG) WB				U
18 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
23 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
28 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
30 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
39 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
41 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
45 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
58 KD (IGG) BAND	NONREACTIVE	REACTIVE	NONREACTIVE	
48 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	
93 KD (IGG) BAND	NONREACTIVE		NONREACTIVE	

STERNARD, ANNA - A050912Q

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Quest Diagnostics Incorporated

QUEST DIAGNOSTICS INCORPORATED

COLLECTED: [REDACTED]
REPORTED: [REDACTED]

PATIENT INFORMATION

DOB: [REDACTED] Age: [REDACTED]
GENDER: F
ID: [REDACTED]

REPORT STATUS: Final

ORDERING PHYSICIAN: [REDACTED]
CLINIC INFORMATION: [REDACTED]

Test Name	In Range	Out of Range	Reference Range	Lab
Lyme Disease AB (IGG, IGM) WB (Continued) Lyme Disease (IGM) WB				U
23 KD (IGM) BAND	NONREACTIVE	REACTIVE	NONREACTIVE	
39 KD (IGM) BAND	NONREACTIVE		NONREACTIVE	
41 KD (IGM) BAND	NONREACTIVE		NONREACTIVE	

Performing Laboratory Information:

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of Lyme disease. The two-test protocol relies on a sensitive but nonspecific screening test, and if the screening test is positive, followed by specific immunoglobulin M (IgM) and/or IgG immunoblotting (IB), depending on the date of disease onset.³¹ The test to order is “Lyme Disease Antibodies, Including Reflex to Western Blot on Positives.”

One problem with Lyme disease testing is that many people have had Lyme disease in the past without knowing it, have recovered without sequelae, and even 10-20 years later, some will have positive IgM as well as IgG titres with no actual infective symptoms due to *Borrelia*.^{32,33}

Another is that there are cases of what look for all the world like Lyme disease, particularly in the southeast states and the Caribbean, but serologic tests are negative, making an as-yet-discovered *Borrelia* likely.^{34,35} At least a portion of this Lyme variant is vectored by the lone star tick, *Amblyomma americanum*, and has been named southern tick-associated rash illness (STARI) or Masters' disease after the physician who first reported this.³⁶ The causative organism, *Borrelia lonestari*, has been cultured,³⁷ and the infection appears to respond to doxycycline.³⁸⁻⁴⁰

Treatment

IN VIVO TESTING CONFIRMS that *B burgdorferi* is sensitive to tetracycline and doxycycline, amoxicillin, cefuroxime axetil (e.g., Cefitin), penicillin and ceftriaxone.^{41,42}

A 2006 Clinical Guideline by the Infectious Diseases Society of America discusses prophylaxis and monitoring of those with tick bites:

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) (BI) when all of the following circumstances exist: (a) the attached tick can be reliably identified as an adult or nymphal *I. scapularis* tick that is estimated to have been attached for >36 h on the basis of the degree of engorgement of the tick with blood or of certainty about the time of exposure to the tick; (b) prophylaxis can be started within 72 h of the time that the tick was removed; (c) ecologic information indicates that the local rate of infection of these ticks with *B. burgdorferi* is ≥20%; and (d)

doxycycline treatment is not contraindicated. ... Infection of ≥20% of ticks with *B. burgdorferi* generally occurs in parts of New England, in parts of the mid-Atlantic States [eastern and south-central but not western Pennsylvania], and in parts of Minnesota and Wisconsin, but not in most other locations in the United States. ... Doxycycline is relatively contraindicated in pregnant women and children <8 years old.

Persons who develop a skin lesion or viral infection-like illness within 1 month after removing an attached tick should promptly seek medical attention to assess the possibility of having acquired a tickborne infection.³²

The standard treatments for erythema migrans, or early disseminated Lyme disease, including facial nerve palsy, mild arthritis or carditis, include doxycycline 100 mg PO BID, amoxicillin 500 mg PO TID, or cefuroxime axetil 500 mg PO BID, or equivalent pediatric doses, for 14-21 days. Doxycycline is better in that it also covers anaplasmosis, which can be transmitted by the same ticks that transmit Lyme disease and STARI/Master's disease.

Doxycycline should be avoided in kids and pregnant women. The other problem I have with doxycycline is that I seem to see people with erythema migrans right before a summer vacation at the beach, and doxycycline is known to cause photodermatitis. There is evidence that a 10-day course is as good as a 20 day course,⁴³ so I prescribe 14 days rather than 21.

An important note: if you aren't sure if what you see is cellulitis or erythema migrans, do not treat solely with cephalexin (e.g., Keflex) or trimethoprim-sulfamethoxazole (e.g., Bactrim); they are not effective against *B burgdorferi*.⁴⁴⁻⁴⁶ So if you see “cellulitis” that's not improving with one of these antibiotics, consider treating for erythema migrans.

More serious CNS disease (e.g., meningitis, encephalitis), or more serious arthritis or carditis, including those with a PR interval of greater than 0.3 second, are treated with ceftriaxone 2 g IV daily, cefotaxime 2 g IV Q8H, or penicillin G 18-24 million units IV daily either continuous or divided Q4H, also for 14-21 days.^{21,22}

One author recommends that any pregnant patient with any Lyme disease should be treated with 2 g of ceftriaxone IV daily for 14 days.⁴⁷

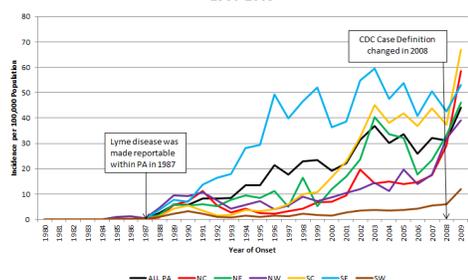
Lyme Controversy and Quackery (especially in Pennsylvania)

LYME DISEASE IS CONTROVERSIAL. Don't worry, the diagnosis and treatment of acute Lyme disease in the ED is quite straightforward, but “chronic Lyme disease” is a whole different can of worms. Why? Because there are many people who are convinced, against all evidence, that their [pick one: a. chronic fatigue b. fibromyalgia c. memory loss] is “chronic Lyme disease.” And, according to Cooper, writing in the Pediatric Infectious Disease Journal in 2004, the Internet is a vast wasteland of Lyme disease misinformation.⁴⁸

In 2005, concerns about inappropriate laboratory testing prompted the CDC and FDA to issue a warning about commercial laboratories that conduct testing for Lyme disease by using assays whose accuracy and clinical usefulness have not been adequately established. These tests include urine antigen tests, immunofluorescent staining for cell wall-deficient forms of *Borrelia burgdorferi*, and lymphocyte transformation tests. In addition, some laboratories perform polymerase chain reaction tests for *B. burgdorferi* DNA on inappropriate specimens such as blood and urine or interpret Western blots using criteria that have not been validated and published in peer-reviewed scientific literature.⁴⁹

Sigal writes: *The diagnosis should be based on symptoms and the probability of exposure to the Lyme spirochete. Laboratory evaluation is appropriate for patients who have arthritic, neurologic, or cardiac symptoms associated with Lyme disease, but it is not appropriate in patients who have nonspecific symptoms, such as those of chronic fatigue syndrome or fibromyalgia.*⁵⁰

Incidence of Lyme Disease in Pennsylvania by Region 1980-2009



A great variety of treatments, including outright quackery and malpractice, have evolved for the treatment of self-diagnosed “chronic Lyme disease,” often without a history of positive Lyme serology. These include but are not limited to:

- colloidal silver;
- intracellular hyperthermia therapy (ICHT): taking 2,4-dinitrophenol (DNP) to “rev up mitochondria” and cause intracellular hyperthermia, despite the fact that DNP, when tried as a diet medication back in the 1930s, killed people;
- rife machines (electromagnetic devices that “sync their frequencies” to the spirochetes);
- hydrogen peroxide injections, and
- infecting oneself with malaria.

A less overtly quackish treatment is prolonged treatment with antibiotics, particularly IV ceftriaxone, which has resulted in biliary problems including likely iatrogenic cholecystitis leading to cholecystectomy.⁵¹ One 30 year old woman died from an infected IV that had been in place for two years, for treatment of unsubstantiated Lyme disease.⁵² Some people with fibromyalgia or chronic fatigue and positive Lyme titres (or even without such titres) are so desperate for antibiotics that, when they can’t find doctors to prescribe them, there are reports in the popular press (see quackwatch.org) they resort to veterinary antibiotics. And some of these antibiotic-seekers are quite politically active, and since they have such a hard time finding doctors to prescribe their antibiotics (LLMDs or “Lyme-Literate MDs”), they have introduced a bill into the Pennsylvania Senate, SB 210 (www.legis.state.pa.us), in the

2011-12 session, which would ensure insurance coverage for prolonged courses of antibiotics and prevent any misconduct hearings or actions by the Board of Medicine against those accused of inappropriately prescribing prolonged antibiotics for presumed Lyme disease. Some LLMDs are treating problems such as autism, multiple sclerosis and amyotrophic lateral sclerosis with long-term antibiotics on the assumption that they are really forms of Lyme disease. And the chief of rheumatology at Tufts has been harassed, stalked, and threatened by patients and patient advocacy groups demanding that he endorse long-term antibiotic treatment for “chronic Lyme disease” – to the point where he had to be assigned security guards.⁵³ There have been many media “exposés” of how mainstream doctors are ignoring the needs of “chronic Lyme disease” patients for prolonged antibiotics, so the public perception seems to align, not with mainstream medicine, but more with the quacks.²⁸

Lightfoot et al concluded that for most patients with a positive Lyme antibody titer and only symptoms of fatigue or nonspecific muscle pains, the risks and costs of intravenous antibiotic therapy exceed the benefits. *In areas endemic for Lyme disease, the incidence of false-positive serologic results in patients with nonspecific myalgia or fatigue exceeds by four to one the incidence of true-positive results...*⁵⁴ And Klempner showed, in placebo-controlled trials, that such patients do not benefit from ninety days of antibiotic.⁵⁵

Quackwatch.org gives the following bullets as their bottom line, which I heartily endorse:

- Lyme disease, when diagnosed early, is readily treatable with oral antibiotics.
- Positive antibody tests, by themselves, do not provide a sufficient basis for diagnosing Lyme disease. The diagnosis should be based on the overall clinical picture, including medical history and physical findings.
- Negative antibody testing after the first few weeks strongly suggests that the patient does not have Lyme disease.
- Many patients with chronic, nonspecific symptoms (such as headaches, fatigue, muscle aches, mental confusion, or sleep disturbances) mistakenly believe they have Lyme disease.
- Intravenous antibiotic therapy, when given appropriately, should not last more than a month. It should not be given unless oral antibiotic therapy has failed and persistent active infection has been demonstrated by culture, biopsy, or other bacteriologic technique.
- Malariotherapy, intracellular hyperthermia therapy, hyperbaric oxygen therapy, colloidal silver, dietary supplements, and herbs are not appropriate measures for treating Lyme disease. Doctors who recommend them should be avoided.

My bottom line: if a patient presents to the ED with likely or even possible erythema migrans, I treat with a short course of antibiotics. If they are complaining of possible chronic Lyme Disease, I refer to a reputable PCP without ordering any sort of testing and without ordering any antibiotics. End of story.

Other Tick-Associated Diseases

Anaplasmosis (Ehrlichiosis)

THE SAME IXODES TICKS that transmit *B. burgdorferi* may be infected with and transmit *Anaplasma phagocytophilum* (previously referred to as *Ehrlichia phagocytophila*), which causes human granulocytic **anaplasmosis** (previously called **ehrlichiosis**). In the upper midwest (Wisconsin) about one in ten of patients with early Lyme disease will also have anaplasmosis, but it’s not as frequent in New England. The treatment of choice is doxycycline, and there are strong recommendations for empiric treatment pending the results of PCR testing.^{56,57}

Babesiosis

ISCAPULARIS TICKS may also carry the malaria-like *Babesia microti*, which causes **babesiosis** (“malaria of the Northeast” “Montauk Malaria”). The disease is most common in coastal southern New England, particularly eastern Long Island (Montauk is the town at the far east end of Long Island), Fire Island, Nantucket Island and Martha’s Vineyard, and in these areas, about one in ten patients with Lyme disease also have babesiosis. Babesiosis has been reported in other areas of New England and the northern midwest.

Clinical clues to babesiosis (or early Lyme disease complicated by babesiosis) include an unusual severity of symptoms, including fatigue, headache, sweats, chills, anorexia, emotional lability, nausea, conjunctivitis, and splenomegaly, elevated LFTs, and hemolytic anemia as well as thrombocytopenia. Babesiosis is diagnosed primarily by repeated thick and thin Giemsa smears, just like malaria.³² A history of a blood transfusion or travel to the endemic area within the past nine weeks combined with the above history should raise suspicion of babesiosis.

Babesiosis is treated with atovaquone and azithromycin.⁵⁸

Rocky Mountain Spotted Fever

ROCKY MOUNTAIN SPOTTED FEVER (RMSF) is a misnomer, it’s actually common in the eastern US than in the Rocky Mountains. It’s caused by *Rickettsia rickettsii*, and transmitted by a variety of ticks, most commonly the dog tick. Classic early symptoms include fever, nausea and vomiting, so are quite nonspecific. Later symptoms often include abdominal pain and diarrhea and arthralgias.

THE GENERAL ASSEMBLY OF PENNSYLVANIA

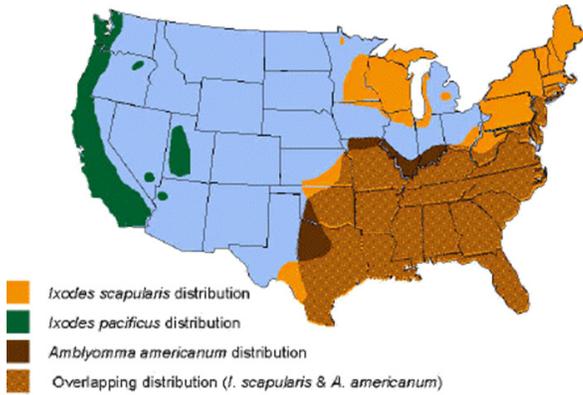
SENATE BILL No. 210 Session of 2011

INTRODUCED BY GREENLEAF, ARGALL, TARTAGLIONE, BROWNE, RAFFERTY, ERICKSON, COSTA, WAUGH, FOWIARA, BOSCOLO, SMUCKER, PICCOLA, BAKER AND McILHINNEY, JANUARY 31, 2011.

REFERRED TO BANKING AND INSURANCE, JANUARY 31, 2011.

AN ACT

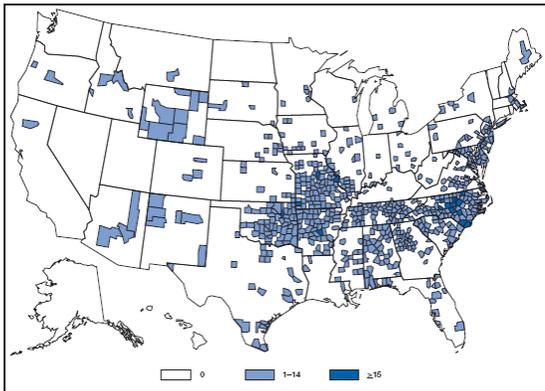
- 1 Establishing a task force on Lyme disease and related maladies;
- 2 and providing for powers and duties of the task force, the
- 3 Department of Health, the Department of Conservation and
- 4 Natural Resources and the Pennsylvania Game Commission to
- 5 execute prevention and education strategies and ensure
- 6 patient access to appropriate care and treatment.
- 7 The General Assembly of the Commonwealth of Pennsylvania
- 8 hereby enacts as follows:
- 9 Section 1. Short title.
- 10 This act shall be known and may be cited as the Lyme and
- 11 Related Tick-Borne Disease Education, Prevention and Treatment
- 12 Act.
- 13 Section 2. Findings.
- 14 The General Assembly finds that:
- 15 (1) Lyme disease and other tick-borne diseases are
- 16 carried primarily by ticks and pose a serious threat to the
- 17 health and quality of life of many citizens of this
- 18 Commonwealth.



Anaplasmosis Endemic Area



Babesiosis Endemic Area



RMSF, 2009 (CDC)

The maculopapular and later petechial rash of Rocky Mountain spotted fever doesn't usually appear until days 2-5, and it occurs in only one-third to two-thirds of patients with RMSF. In half to two-thirds of those with a rash it appears on the palms and soles and then moves toward the torso.

The diagnosis of RMSF is clinical – and based on the severity of the illness (it used to have a 30% mortality rate) it's recommended that one treat based on suspicion.

Treatment is doxycycline or chloramphenicol.⁵⁹⁻⁶¹

The American Academy of Pediatrics has identified doxycycline as the drug of choice for treating presumed or confirmed RMSF and ehrlichial infections in children of any age.⁶²

Tick paralysis

TICK PARALYSIS IS CAUSED by a neurotoxin secreted in the saliva of certain ticks and is a well-known cause of illness death in livestock and pets. In North America, tick paralysis is caused by the **Rocky Mountain wood tick** (*Dermacentor andersoni*) and the **American dog tick** (*Dermacentor variabilis*). Tick paralysis mimics, and is often mistaken for, Guillain-Barre syndrome with an ascending paralysis that is fatal in about one out of ten cases – yet quickly curable by simply detecting and removing the tick. It is likely that it is only caused by engorged and egg-laden female ticks. Other than

the effects it causes, and that it is usually secreted from days 5-7 after attachment, little is known about the toxin. The tick is usually found on the scalp, and during the months April to June, when ticks are feeding.⁶³

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