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 and cytauxzoonosis.

The Ticks

There are many species of ticks. But in North America, there are four main types to be concerned with. ***Ixodes scapularis*** is known as the deer or **black-legged tick** (or, in the upper midwest, bear tick), and is a member of the *Ixodidae* family of hard-bodied ticks. It is the main vector for human Lyme disease. There is a related but very similar black-legged tick species, *Ixodes pacificus*, which is found on the west coast and also transmits Lyme disease. The adult blacklegged 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, so it’s common to get Lyme disease without noticing a tick bite.¹

*Dermacentor variabilis*, also known as the **American dog tick**, can transmit Rocky Mountain Spotted Fever, tularemia and anaplasmosis.

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.²

**DEET** is the most famous, and highly effective, but is mildly toxic, especially in kids,³ 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.⁴

**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 mosquitoes and ticks. As with other urban legends about mosquitoes (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 Fradin⁵ (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 mosquitoes.
as picaridin or DEET. 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):

<table>
<thead>
<tr>
<th>Score</th>
<th>Insect Repellent</th>
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<tbody>
<tr>
<td>98</td>
<td>15-30% DEET</td>
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<tr>
<td>97</td>
<td>Repel</td>
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<tr>
<td>87</td>
<td>Natrapel</td>
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<td>73</td>
<td><em>Avon Skin So Soft Bug Guard Plus IR3535</em></td>
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<tr>
<td>56</td>
<td>Bite Blocker</td>
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<tr>
<td>24</td>
<td>Burt’s Bees</td>
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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.

**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 and gently pulling until the jaws tire and the entire tick comes out is best.

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 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.

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. Let it bleed, then apply a Bandaid. One author recommends an elliptical incision followed by a suture (even for intact ticks) but I prefer to excise very narrowly and shallowly then leave open to avoid infection. One author recommends a biopsy punch, but one’s seldom available in the ED when I need it.

**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. 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, so a guess of 300 cases for Allegheny County in 2011 is appropriate. And going a bit east into the mountains Lyme disease is much more common. The vector is the common *deer* or *blacklegged tick* of the *Ixodes* (hard tick) genus, particularly *Ixodes scapularis*. A variant of Lyme disease with different characteristics and caused mostly by *Borrelia afzelii* occurs in Europe and Scandinavia.

There is great public discord between hunting groups and animal rights groups about the role of deer culling in controlling Lyme disease. But, there seems to be little correlation to deer populations.
Indeed, the population of the white-footed mouse, *Peromyscus leucopus*, seems to be more important than the number of deer. In the short term, mouse population and Lyme incidence is determined by the number of acorns two years prior. There is well-known phenomenon called a “mast year,” when oaks produce perhaps 10 times their normal yearly crop of acorns; these years occur irregularly, most recently in 2010 across the entire eastern US, and are thought to be linked to an oak reproductive of “predator saturation” and producing many more acorns than the squirrels, mice and deer can eat. The environmental trigger, or mode of communication among oak trees, is quite unknown. Other tree species also exhibit mast years.

Looking at the longer term, forest destruction and fragmentation (think: new strip malls and new housing developments) increases the white-footed mouse population, and is also thought to be a major cause of the current increase in 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. It fairly often has central clearing except a central red point or area. Some people (about a quarter) will also develop these red, hive-like rashes, sometimes with central clearing, in areas away from the initial bite. 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. 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. 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.

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 will develop low-grade fever, malaise, and myalgias, but cold symptoms or vomiting and diarrhea are rare. Some of these people do not have the classic rash, which makes diagnosis quite difficult, especially because blood tests have many false positives.

*Early disseminated infection* occurs when the causative Rickettsial organism, *Borrelia burgdorferi*, spreads through the blood. This may occur without evidence of early localized infection.

The classic picture here (better than any other pictures I’ve found) is of my daughter’s leg in 2005 when she was five years old, and the rash was misdiagnosed as hives by her pediatrician.

About half of those with erythema migrans have *B burgdorferi* in their blood. 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.

### Tests for Lyme disease

“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.”

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There are many tests for Lyme disease, but none are useful in the ED. Blood and skin biopsy cultures and PCRs are not performed in standard clinical laboratories. Serological tests can be performed as a baseline, but prompt treatment with an antibiotics may prevent the convalescent serology from turning positive.

There is now a national standard 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 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.

For other possible manifestations of Lyme disease, referral and outpatient workup is standard. For possible erythema migrans or later phases of Lyme disease, it may be appropriate to send some tests from the ED to aid the primary care physician if the emergency physician coordinates care with the PCP.

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 will have positive tests with no actual infective symptoms due to *Borrelia*. 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. 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. The causative organism, *Borrelia lonestari*, has been cultured, and the infection appears to respond to doxycycline.

**Lyme Controversy (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 diagnosing chronic Lyme Disease is difficult, and the correct treatment for chronic Lyme Disease – especially the length of treatment – is controversial.

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.22

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.23

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 (ICHHT): 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.24 One 30 year old woman died from an infected IV that had been in place for two years, for treatment of unsubstantiated Lyme disease.25 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.

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...26

My bottom line: if a patient presents 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.

**Treatment**

The 2011

One author recommends that pregnant patients with Lyme disease should be treated with 2 g of ceftriaxone IV daily for 14 days.27

**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.28,29

**Babesiosis**

*I. scapularis* 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. Babesiosis is diagnosed primarily by repeated thick and thin Giemsa smears, just like malaria. 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.

Tick paralysis

References