INFECTIOUS DISEASE WARNING

Pandemic of Syphilis-Like Spirochete in U.S.A.

by Laurence Hecht

April 2—The fastest growing infectious disease in the U.S.A. is a spirochetal bacterial infection, similar in symptomology to tertiary syphilis. Untreated it becomes a progressive, degenerative disease that attacks the muscles, joints, central nervous system, heart and other organs, and finally the brain.

Infection is characterized by a wide variety of possible symptoms, including joint and muscle pain, fatigue, light sensitivity, mood disorders, inability to focus, memory loss, facial palsy, tremors, cardio-vascular complications, loss of control over limbs, seizures, and eventual cerebro-neural deterioration resembling the *general paresis* of late-stage syphilis. The causative agent is a spirochete-form bacteria, first identified in 1982 by the Swiss-born American microbiologist Willy Burgdorfer, and known as *Borrelia burgdorferi.* The spirochete is usually transmitted to humans by the bite of the black-legged deer tick.

Juvenile cases often begin with fatigue, joint pain, mood disorders, memory loss, and difficulties in school, and progress to such symptoms as locomotion problems, seizures, loss of speech, and sometimes, death. School districts in highly infected areas have reported as high as 15% infection rates among children.

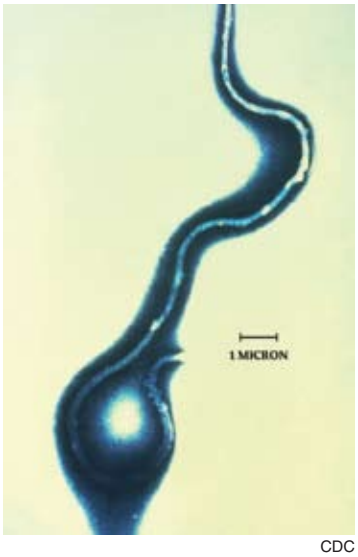
Experts estimate the rate of new infection at 200,000 cases per year in the U.S.A.[1] The greatest concentration of reported cases falls along an east-west band of temperate climate zone within about 200 miles north and south of the last glacial terminal moraine, in the Northeastern, Mid-Atlantic, and North Central states. This region is the natural habitat of the deer tick, *ixodes scapularis*. Within this general geographic area, the densest concentration falls within a 150-mile-wide band, running north to south along the East Coast of the United Sates, from southeastern Maine to northern Virginia. A second density of high incidence is found in Wisconsin and eastern Minnesota. Recently, the disease has been identified in the temperate zone regions of northern California and Washington State. Outside of the U.S.A., incidence is high in Scandinavia, Germany, Britain, northern and mid-latitude Europe, and the Eurasian steppes, reaching across to eastern Mongolia and northern China.

Disease History

The disease was identified in the U.S.A., in 1975-76, in Lyme, Conn. An alert citizen with some knowledge of epidemiology had noticed clusters of the same symptoms appearing on certain streets and within families in this eastern Connecticut shoreline community.[2] The same symptoms were soon identified in other parts of Connecticut, Massachusetts, New York, and New Jersey. Well before the discovery of the spirochete, physicians recognized the Lyme disease symptoms as

1. Because of frequent misdiagnosis, this expert estimate is about seven times greater than the number of presently reported annual cases. In 2007, 27,444 cases of Lyme disease were reported to the Centers for Disease Control. The largest proportion of cases is reported among persons aged 5 to 14 years, and 55 to 69 years, although all ages and both genders are susceptible.

2. Later research showed descriptions of the disease syndrome in German-language medical texts dating to the late 19th Century, and in some U.S. medical records.



CDC


USDA/Scott Bauer

Left: Photomicrograph of Borrelia burgdorferi*, the spirochete bacterium that causes Lyme disease; the spirochete lives in the gut of the tiny adult deer tick,* ixodes scapularis*; the tick in its nymphal stage attaches itself to a white-footed mouse,* Peromyscus leucopus*; and then, in maturity, the tick attaches to a larger mammal, commonly a deer in the U.S. Northeast.*


USDA/Scott Bauer


Virginia Dept. of Game and Inland Fisheries

similar to late-stage syphilis, and had some initial success treating it with the standard penicillin protocol used for tertiary syphilis. Later, other antibiotics, particularly Doxycycline and intravenous ceftriaxone (Rocephin), were found to be more effective. Today, these are used in conjunction with other antibiotics which target the bacteria in its alternative cyst form, and immuno-supportive therapy. Other novel treatments include vitamin D denial to starve the bacterial cell wall.

The suspicion by medical entomologists that the vector for the spirochete was likely an arthropod, touched off the search which led to Willy Burgdorfer at the Colorado research lab of the CDC discovering the Lyme spirochete in the gut of the common deer tick (*Ixodes scapularis*). The life cycle of the tick commonly involves two co-vectors, a white-footed mouse (common field mouse) and a larger mammal. In the nymphal stage, the ticks are usually found attached to the white-footed mouse. On reaching maturity, they attach to a larger mammal, commonly deer. The deer tend to carry them to areas of human habitation where the tick might detach, and wait for a new blood meal, while attached to a branch, a leaf pile or high grass. This is thought to be the most common path of human infection. However, cases of *in utero* transmission have been documented in mothers infected with Lyme disease, often resulting in miscarriage of an infected fetus, or severely debilitated children. Human sexual transmission may also occur in some cases.

One of the widespread myths preventing earlier identification of Lyme infection is the belief that a bite by an infected tick will always lead to the characteristic red bull's eye rash, known as *erythema migrans*. Actually, only 60 to 70% of documented cases of Lyme infection have shown the bull's eye rash. However, because there is such a large number of undiagnosed or misdiagnosed cases, the actual percentage of infected people showing the rash is probably less than 50%. In the nymphal stage, the deer tick is only about the size of a poppy seed, but still capable of biting and infecting a human host. Infection by tiny attached nymphs, which subsequently drop off the body unnoticed, may be a major cause for undetected cases.

Treatment

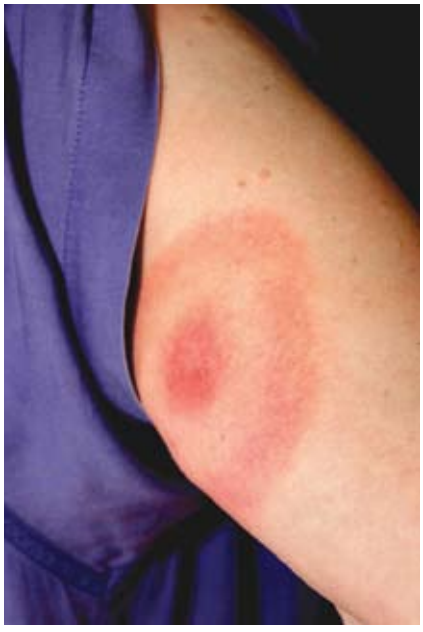
The spirochete is a helical-shaped bacteria that looks something like a drill bit under magnification. It has the

capability to dig into tissue, shedding surface antibodies as it penetrates. When the immune system mobilizes a response to the invader, the shedded antibodies which have attached to uninfected tissue are also targeted, producing more widespread inflammation.

If caught in the early stages, the infection can usually be stemmed by a short-term antibiotic treatment. However, if progressed to intermediate or late stage, treatment may take several years. As it is very difficult to know if the infection has been completely eliminated, often treatment is stopped after symptoms abate, but must be resumed as symptoms begin to reappear. With persistence, complete recovery is usually achieved. However, for those diagnosed in very late stage, full recovery may be impossible.

Response to antibiotics is not straightforward, as the spirochete will change into a cyst or L-form when under antibiotic attack. The patient will often experience an improvement, followed by a deterioration. An experienced physician is required to assess the changing symptomology, and alter therapies to target the changing forms and locations of the infectious organism.

A frequent response to antibiotic treatment for Lyme infection is the Jarisch-Herxhiemer reaction, named for two dermatologists involved in the development of the anti-syphilitic drug Salvarsan. They noted an initial increase in severity of symptoms, usually one to three days after commencement of treatment. The cause is thought to be the release of large quantities of toxins when the spirochetes die off.



CDC/James Gathany

The "bull's eye" rash is a common manifestation of Lyme disease. But 30-40% of reported cases do not show this pattern, contrary to widespread belief, and a large number of cases are undiagnosed or misdiagnosed.

The Lyme Scandal

In the course of the 1980s, victims of the spirochetal infection that had come to be known as Lyme disease were turning up in doctors' offices throughout the Northeast. A great number of the cases were late-stage infections presenting severe symptoms, especially among children. The average cost of treatment, including daily intravenous therapy, was $100,000 per case. Pressure from the insurance companies began to come down. New York's Empire Blue Cross was among the first insurers to begin routinely denying payment and terminating coverage of Lyme-infected families.

With documented conflict of interest, a faction within the Infectious Diseases Society of America launched an effort to deny long-term treatment. They did this by first inventing a new term for the late stage of the disease, *chronic* Lyme, and then declaring it nonexistent. If a patient did not respond to treatment within a prescribed time frame (usually 4 weeks), they were declared to be free of actual Lyme infection—even if proof of the continued presence of the spirochete in the body was presented. Instead, they were deemed to have *chronic* Lyme, as if there were also a different acute form of the disease.

Actually, they just had Lyme disease, but the imposition of an artificial time frame for treatment required a new term of art. Those who insisted that they still had the infection, now called *chronic* Lyme, were said to be making it up. Many were deemed to have mental disorders, and referred to psychiatrists. Patients who organized protest groups against this mistreatment were declared part of a cult.

Dr. Allen Steere, a one-time schoolmate of mine at Columbia College, played a key role in carrying out this fraud against the infected. Trained as a rheumatologist, Steere had been assigned by Yale University and the Centers for Disease Control in Atlanta (CDC) to investigate the Lyme, Conn. infections that resident Polly Murray had discovered. Steere first distinguished himself by denying the existence of an insect vector, and then holding to his prejudice long after Burgdorfer's discovery of the spirochete in the deer tick. In Steere's pet theory, Lyme was an autoimmune disease like rheumatoid arthritis. Later, when the evidence for the spirochete had become indisputable, Steere proposed that so-called *chronic Lyme* was an autoimmune disease.

Even after the spirochete had been isolated in biopsies and fluid samples from these patients with the nerve to claim they were still sick, Steere and his corrupted cohorts held to the absurd denial.

Later, when an antibody test protocol was developed, it was used to squeeze out more Lyme victims from medical insurance coverage. The antibody tests had been developed for screening, and not clinical diagnostic purposes. Their combined sensitivity was sufficient to give the CDC or other monitoring agencies a rough idea of Lyme incidence. However, too many false negatives and positives slipped through to be useful as a method for clinical diagnosis. Although it was well known that many patients infected with Lyme would show up as negative on these tests, the CDC authorized a protocol for interpretation of the antibody tests that is used in most doctors' offices today. If you are not positive on five bands of a limited assay of antibody response to the *Borrelia* spirochete, you will likely be denied any treatment for Lyme by your insurance company.

Doctors who opposed this cruel mistreatment of their patients were ruthlessly targeted. In 2000, a nationally recognized Lyme specialist, Dr. Joseph Burrascano, who had treated over 40,000 patients, was brought before the New York State Office of Professional Medical Conduct. He was charged with mistreating nine patients, all of whom supported the doctor. In 2002, the review board in this highly politicized case found Burrascano had committed professional misconduct in negligently treating two of the patients.

In 2006, North Carolina Lyme specialist Joseph Jemsek was charged with unprofessional conduct, and placed on a one-year suspension, which allowed him to continue practicing on Lyme patients only if he adhered to the restrictive criteria of the CDC.

The same year, Charles Ray Jones, a 76-year-old pediatrician, who had treated 10,000 children with Lyme disease, was brought before the Connecticut Medical Board. He was charged with prescribing antibiotics over the phone to a child who was the subject of a custody case. The case excited such popular outrage in a state teeming with Lyme infection, that the State Attorney General Richard Blumenthal launched a counter-investigation. He charged that the Infectious Diseases Society of America had violated antitrust laws in setting their restrictive guidelines for diagnosing and treating Lyme disease. Blumenthal charged that the IDSA panel "locked out competing points of view." His office had received numerous complaints of denial of insurance from families of Lyme-infected children and adults. In December 2007, at age 78, Dr. Jones was fined $10,000 and placed on two years probation by the medical board, but the decision was stayed on appeal. In 2008, the Department of Public Health brought new charges against the doctor, who is a hero to thousands of parents and children.

The message of the show trials is clear. Doctors who prescribe longer courses of antibiotic treatment, or take other aggressive measures required for cure, may be subject to the same treatment as Burrascano, Jemsek, Jones, and 50 other physicians who have been investigated or sanctioned by state medical boards. Ironically, long courses of doxycycline, the most commonly used antibiotic in Lyme treatment, are routinely prescribed for acne.

Connecticut Attorney General Blumenthal's antitrust investigation won a partial victory in April 2008, when the Infectious Diseases Society of America (IDSA) agreed, out of court, to convene a new independent review panel. Blumenthal said that he had proof of "undisclosed financial interests held by several of the most powerful IDSA panelists," which, however, were not made public. According to the Lyme Disease Association, a patients' adocacy group, the majority of individuals involved in the IDSA guidelines development process held direct or indirect commercial interests related to Lyme vaccines, patents, and/or test kits.

Meanwhile, the IDSA-dictated treatment guidelines which restrict antibiotic treatment for Lyme infection to 28 days remain in effect.

An added factor contributing to the failure respecting Lyme and other emerging diseases, was the 1980 court decision which permitted the patenting of biological materials. As a result of the ruling, researchers in government labs had a financial incentive to hoard their findings, and go private with them, in hopes of striking it rich with a new vaccine, drug or other invention. Willy Burgdorfer donated his discovery, but many others jealously guarded theirs, preventing collaborative research in this and many other areas in hopes of later profit.

Misdiagnosis, Co-infection, Prevention

The official denial, and pressure against doctors, has led to the situation that the vast majority of Lyme cases are unreported or misdiagnosed. A great number of cases later definitively identified by Lyme specialists,

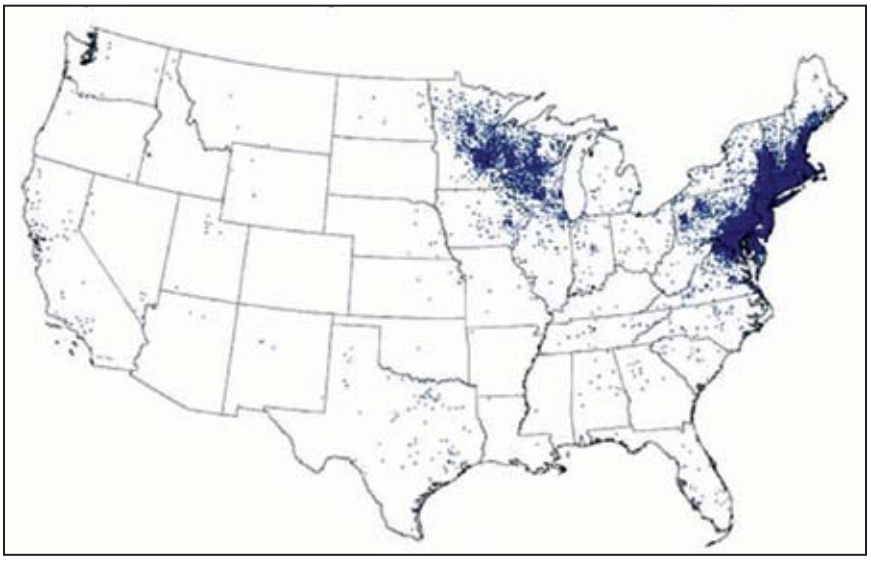
were initially misdiagnosed, often by several other doctors. Among the most common misdiagnoses reported by Lyme specialists have been: chronic fatigue syndrome, fibromyalgia, Epstein-Barre virus, multiple sclerosis, and Krohn's disease. One of the greatest complaints of patients later cured by Lyme specialists was their having been bounced around from doctor to doctor, sometimes 15 or more, before getting a proper diagnosis.

In many cases of Lyme there is co-infection with other agents present in the tick. Babesiosis is a malaria-like infection caused by a protozoan parasite that invades the red blood cells. Infection may be asymptomatic or may include severe symptoms including high fever, anemia, chills, and shaking. The most common strain in the U.S. is *Babesia microti*. First seen in Lyme cases in the Cape Cod, Mass. area, it has spread as far south as Pennsylvania, Maryland, and Virginia. Unlike the malaria plasmodium, the babesia parasite does not reproduce outside the red blood cell, and is not as deadly. Other common co-infections from Lyme-infected ticks include bartonella, Ehrlichiosis, and Mycoplasma bacteria.

Avoidance of ticks is the best preventive measure against infection. Although the deer tick is the primary vector for Lyme, there is some evidence suggesting other species may be capable of transmitting the spirochete. Ticks are commonly found attached to tall grass, bushes and the lower limbs of trees, leaf piles, stone walls, and any other place where moisture is present. The presence of deer is a warning sign that Lyme-infected ticks are likely to be near. To avoid exposure in such places, wear long pants and long sleeves, and light-colored clothing, and spray clothing with 25% DEET insecticide. Tuck your pants into socks, and examine yourself after exposure. Shower and use a brush on parts of the body that you can't see. The time it takes for the tick to attach is uncertain, but may be greater than 12 hours.

Lyme disease has probably been present for millennia. The most likely explanation for its explosive increase recently, is the growth in size of the deer population in proximity to human habitation. Deer are found in areas where open lands meet forest and wooded areas. The recent decades' pattern of reduction of agricultural land area, and growth of suburban developments, has increased the areas where exposure is likely for a large number of people. Some estimate that the deer population has increased by four-fold since the early 19th Century.

FIGURE 1
Reported Cases of Lyme Disease, United States, 2007



Centers for Disease Control

One dot is placed randomly within county of residence for each reported case.

Reduction in deer population below an average of 10 per square mile has been shown to greatly reduce the likelihood of Lyme exposure. Deer population density must be examined on a local scale, however. In some counties, the overall density may fall below 10 per square mile, but in suburban and semi-rural portions, deer herds may abound. These are the locales where high rates of Lyme infection in the human population are invariably found. In such areas, there is no reason to maintain restrictions on deer hunting, except where the discharge of weapons may endanger public safety. The threat of Lyme infection to the human population far outweighs other concerns.

Link to Alzheimer's and MS

As it is an ill wind that blows no good, new disease outbreaks usually lead to new breakthroughs in medical research. One of the most exciting is the possible explanation for some forms of Alzheimer's disease. Recent

laboratory evidence suggests that the *borrelia* spirochete may be the causative agent in many cases of Alzheimer's disease. The cyst form of the *borrelia* spirochete is identical in size and shape to the "amyloid plaques" found by Alois Alzheimer in the brains of the degenerative dementia cases now described as Alzheimer's. By molecular interrogation of Alzheimer brain tissues, Lyme researcher Dr. Alan MacDonald has identified the antibody signature of *Borrelia burgdorferi* in 7 of 10 brain samples of Alzheimer's victims received from the Harvard University Brain Bank. In another brain sample, taken from a case of Alzheimer's which appeared eight years after infection with *Borrelia burgdorferi*, MacDonald found that the topographic distribution of the antibody response coincided with the Alzheimer plaques.

There is also a suggestion that other neurodegenerative diseases of unknown etiology, including multiple sclerosis (MS), ALS (Lou Gehrig's disease), and Parkinson's disease may, in some cases, be the result of infection with the *borrelia* spirochete. MS is also a disease of temperate climates. In the United States, the incidence of MS is nearly double above the 37th parallel, approximately the latitude dividing Virginia from North Carolina, and northern from southern California. Incidence by state is unavailable because the CDC has not yet established statewide reporting for the disease. However, localized correlations of incidence of ALS with Lyme disease have been noted. These associations merit further investigation.

L-Form Bacteria, and the Next Phase of Medicine

A related area of broader significance is the discovery of new types of morphologically changed forms of common bacteria never before seen. Improvements in microscopy and biochemical techniques employing antibody reactions and DNA analysis, have led to the discovery that many commonly known bacteria pass through a phase in their life cycle in which they change into a very small form lacking a cell wall. These cell-wall deficient bacteria, known as *L-forms*, may be the infectious agent in many diseases not previously recognized as having a bacterial cause. They are already proven to be a part of the life cycle of the bacteria responsible for some of the most common and deadly infectious diseases.

In recent studies, cell-wall-deficient, L-form bacteria have been found, *in vitro*, among various species of bacteria treated with beta-lactam antibiotics, a class that includes penicillin. The penicillin does not merely select for L-forms, as prescribed by the usual theory of antibiotic resistance, but actually induces their growth. Over 50 different species of bacteria which demonstrate L-forms have already been identified, and experts believe that most bacterial species can probably be converted into L-forms when treated with antibiotics that inhibit cell-wall formation.[3]

The L-forms are smaller than viruses or fungal particles, and may evade detection by hiding inside the cells of the immune system. They may infect many types of cells, but seem to prefer the white blood cells known as macrophages, which are responsible for removal of necrotic tissue and other debris, and as a first-line defense for the immune system. Infection of the macrophages and other cells allows the L-forms to circulate to the rest of the body. They are able to take control of the host's genetic material to create proteins which enhance their own survival against attack.

The L-form bacteria cause an inflammatory response of the immune system, and are implicated as the infectious agent in many diseases that are still classified as immune-mediated and non-infectious, such as lupus rheumatoid arthritis and Crohn's disease, according to Stanford University immunologist and microbiologist Dr. David Relman.[4]

Some of the species of L-form bacteria that have been implicated in diseases which are already recognized as of bacterial nature, include *Bacillus anthracis* (anthrax), *Treponema pallidum* (syphilis), *Mycobacterium tuberculosis* (the most common form of tuberculosis), *Helicobacter pylori* (gastric ulcers and stomach cancer) *i, Rickettsia prowazekii* (typhus), and *Borrelia burgdorferi* (Lyme).[5]

Outside of cells, L-form bacteria have been shown to grow into long, thin filaments known as biofilm which possess a protective protein sheath. Large bodies or colonies of L-forms have been observed in the laboratory.

There is hope that further research into these previously unrecognized bacterial forms will lead to the cure and control of many previously unconquerable diseases, including cancers.

3. Amy Proal, *Understanding L-form Bacteria,* http://bacteriality.com/2007/08/15/l-forms/

4. Cited in Proal, *Ibid.*

5. *Ibid.*