

Some excerpt from ELISA and Western Blot: Lies that can kill you?

<http://www.centuryinter.net/tjs11/bug/blot1.htm>

Eighty-five percent of seronegative patients who still had active disseminated infection had been treated within one month of tick bite. This means that early antibiotic treatment may make you test negative, but you still progress to develop encephalitis.

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Let me give you a local example in Duluth MN: A man phoned me and told me about his strange untreatable Rheumatoid arthritis, he mentioned he and his brother and his father were all avid sportsman from an area rife with Lyme disease. When he told me his brother had non-remitting MS [multiple sclerosis] and his father was in a nursing home with Alzheimer's, I started to see a pattern. Despite the different diagnosis's they all shared similar symptoms that were consistent with Lyme disease.

Shared Symptoms: Sensitivity to bright lights, migrating joint pain, head aches, muscle twitches, stiff crunchy neck, floaters in the eyes, heart palpitations and chest pains, depression, hearing disturbances, night sweats, strange burning skin sensations, sensitivity to noise, urinary frequency/urgency but decreased urinary volume and many more.

Both the sons in their 30s responded slowly but steadily to constant antibiotics, and neither one has either MS or arthritis. When they asked their father's physicians to test for or treat their father for Lyme disease the doctors became furious and refused to not only treat, but refused to even do a single Lyme test.

Despite the strong family history and symptoms and life-long exposure to infected ticks, the doctors refused to even entertain the possibility that the man dying in the nursing home of senile "Alzheimer's like" dementia could possibly have Lyme disease.

Why? What did the doctors have to lose with this patient?

I believe they were afraid of exactly what the autopsy tests showed! The boys wanted to be absolutely sure one way or another if their father had long standing untreated Lyme disease. After all TRUE ALZHEIMER'S MAY HAVE A GENETIC COMPONENT, BUT LYME DISEASE IS TREATABLE!

When their dad died they sent his brain to a pathologist who specializes in brain sectioning, and silver stain.

In virtually every cross section of the cerebral cortex spirochetes were found. Further they were consistent with *Borrelia burgdorferi* and even more astoundingly, in a serial cross section where several slides were made of the same brain cell and bacteria, a spirochete was found half in and out of a human neuron.

This is important because although we have seen in-vitro penetration of human fibroblast cells, macrophage, B-cells, and rodent neurons, we have never seen in-vivo evidence ever before of intracellular spirochetes in any human cell let alone human brain cells. This was a huge discovery.

When the son of this man tried to see the doctor to discuss the autopsy findings he flatly refused, and in fact became quite agitated and said that it meant nothing and meant he probably died of Syphilis!

The son and his entire family including his mother had Syphilis tests which all were negative and once again tried to see the doctor. Almost immediately a restraining order was placed against the patient's son.

He only wanted to know why the doctor didn't test for Syphilis if he thought it was Syphilis? Why hadn't father hadn't been given a Syphilis test or treated for syphilis? [See Jim Forris story LA Spotlight])

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- Can an infection be present in the body without the immune system making measurable antibodies?
- Once an infection has left the bloodstream, a patient may not make enough antibodies to test positive.
- Once the infection has found a safer place in the body to hide, it can avoid the immune system, and also avoid any antibiotics that are circulating mainly in the blood.

MECHANISMS OF IMMUNE ESCAPE:

- Bb can be coated by human blocking antibody and become invisible to killer immune cells.
- Bb can coat it self with B-cell membrane and cloak itself in human proteins.
- Bb can find places like inside joints and tendons where it is sequestered from the immune system and even antibiotics.
- It can go metabolically inactive.
- It can hide in the brain, heart, bladder, and possibly skin cells.
- It is motile so it seeks out survivable places.
- Bb may have another form that lacks cell wall, and therefor lacks many of the antigens the human immune system would use to attack.
- It may hide inside some human cells.
- Without the infection being in constant contact with the (blood-borne) immune system, the body shuts off antibody production. Antibody levels will fall even despite the fact that the infection is still sequestered deep in the body such as the brain, tendons, heart, nerves, bladder, eyes, and joints.

How do we know this?

- Patients who have been repeatedly seronegative for antibodies, have been culture positive for the Lyme bacteria.
- Patients who have been aggressively treated with antibiotics have been culture positive for the Lyme bacteria.
- Despite repeated negative Lyme antibody tests these patients still had symptoms and still had active infection, symptoms that in most cases responded to extended antibiotic therapies. (See attached references)

Because the MEDICAL COMMUNITY HAS BY AND LARGE REFUSED TO ACCEPT A PATIENT'S SYMPTOMS AS PROOF OF INFECTION, and have continually based their diagnosis of Lyme disease on Lyme serologies, there has been an ever growing schism between so called "Chronic Lyme Patients", and a medical community that refuses to accept their claims as still having active infection post treatment. In many cases not only are serologies used to determine the diagnosis but the drop in antibodies is often used to indicate a biological cure. It has been the variable nature of the disease and its wide range of symptoms, and the reliance on unreliable tests that has given rise to two different camps concerning the diagnosis and treatment of Lyme disease. The evolution of these two opposed paradigms of diagnosis and treatment will be discussed in the next section.

PART TWO "The Need For A Post-Mortem Lyme Disease Study" by Tom Grier
"The Need For A Post-Mortem Lyme Study"

The medical community is unevenly divided into two opposing camps on three major issues concerning Lyme Disease:

- 1) What constitutes a proper diagnosis of Lyme disease?
- 2) What constitutes proper treatment for patients with Lyme disease who have symptoms that persist beyond four weeks of antibiotic therapy?
- 3) What role should Lyme tests play in both diagnosis and treatment?

The first camp, which I will call Camp A, represents the majority of the medical community and is spearheaded by researchers from Yale Medical, the American College of Physicians (ACP), and several other major medical institutions. In general terms, this camp believes that Lyme disease is best diagnosed through the use of two consecutive serology tests; the ELISA test followed by a confirming Western Blot. This is known as TWO-TIERED testing. (With very little opposition by the medical community, two-tiered testing has now become the diagnostic standard of most major medical centres.) Camp A also maintains that Lyme disease, despite the stage or severity, is usually cured with just a few weeks of oral antibiotics. (This is the by far the most popular position within the medical community and the health insurance industry at this time.). How does Camp A make a diagnosis of Lyme Disease? In the past a history of a tick bite followed by a bull's-eye skin rash or erythema migrans rash was diagnostic of the disease, but a diagnosis based on the rash and symptoms alone has come under increasing attack by several advocates of TWO TIERED testing including Yale

Medical (see Yale Medical Report) and the ACP.

A video training tape by the ACP is quite explicit in its portrayal of Lyme patients that in the absence of an erythema migrans (EM) rash, the diagnosis must be made by dual serologies and more than two weeks of antibiotics is almost always unnecessary. In one of the video scenarios, the tape suggests to treating physicians that patients who insist that they have persistent symptoms post-treatment should be referred to psychiatrists. The logic of this psychiatric referral stems from the premise that since antibiotics are accepted as curative, any persistence of symptoms has to be purely psychological. So if a patient doesn't feel better post treatment, send them to a shrink!

The second camp, often referred to as "LYME ADVOCATES," and which I will call Camp B, believes that most of the persistent symptoms post-antibiotic treatment are caused by persistent infection. This camp maintains that antibody serologies are poor at detecting a spirochetal bacterial infection that has sequestered in deep tissues and no longer found within the bloodstream. They believe spirochetes that have found sequestered, or privileged, sites tend to hide in the body and are poorly detected by any means. As proof of their position, this camp offers numerous studies which have shown persistence of *Borrelia* infection post-antibiotic treatment. Listed below are several of these published cases of persistent infection in humans and animals post-treatment as confirmed by either culture or tissue biopsy and stain: (For further information, please refer to the compendium of references to the persistence or relapse of Lyme disease at Lyme Links.

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- Neuroborreliosis: In the journal *Annals of Neurology* Vol. 38, No 4, 1995, there was a brief article by Dr. Andrew Pachner MD, Elizabeth Delaney BS, and Tim O'Neill DVM, Ph.D. The conclusion of the article was simple and concise: