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The following is an excerpt from the Lyme Disease Survival Manual 1997.

Lyme Disease is a multi-system disease which can affect virtually every tissue and every organ of the human body. It is a disease which can be mild to some, and devastating to others. It can cripple and disable, or fog your mind. It can affect men, woman, and children, and even your family dog. (1-5,7-19) You may test negative for the disease, and still have it, or test positive and be symptom free. Some will get symptoms within days of a tick bite, while others may have it for years before they are even diagnosed. Some Lyme patients are told they have fibromyalgia, chronic fatigue syndrome, MS, or some other disease of unknown origin. (See abstracts of the 1996 International Lyme Conference) There are some studies which strongly support that the infection can be transmitted from mother to the unborn fetus, and may even cause still birth and has been implicated in some SIDs deaths. (MacDonald 20,45,52,53)

Why is Lyme disease such a mystery? Why does it mimic so many other disease? Why is it so difficult to detect? The reasons come from the microbiology of the bacteria that causes Lyme Disease.

Lyme disease is caused by a spiral shaped bacterium known as a spirochete. Diseases that are caused by spirochetes are notorious for being relapsing in nature, difficult to detect, and great imitators of other diseases. Syphilis, Tick-Borne Relapsing Fever, and Leptospirosis are other examples of spirochetal diseases. Lyme disease is caused by a bacteria called Borrelia burgdorferi, named after the man who isolated it from a Deer Tick in 1981, Dr. Willy Burgdorfer. The following is a tutorial to help explain away the mysteries of this bacteria, and why it causes so much controversy between patients and the medical community. (1)

The Structure of the Lyme Bacteria

The structure of the Lyme spirochete is unlike any other bacteria that has ever been studied before. It is one of the largest of the spirochetes (0.25 microns x 50 microns) It is as long, as a fine human hair is thick. Borrelia burgdorferi is a highly motile bacteria, it can swim extremely efficiently through both blood and tissue because of internal propulsion. It is propelled by an internal arrangement of flagella, bundled together, that runs the length of the bacteria from tip to tip. Like other Borrelia bacteria Borrelia burgdorferi has a three layer cell wall which helps determine the spiral shape of the bacteria. What makes this bacteria different from other species, is that it also has a clear gel-like coat of glyco-proteins which surround the bacteria. This extra layer is sometimes called the Slime Layer or S-layer. (See diagram 1) (45,46,59)

This means: This extra layer of glyco-proteins may act like a stealthy coat of armor that protects and hides the bacteria from the immune system. The human immune system uses proteins that are on the surface of the bacteria as markers, and sends attacking antibodies and killer T-cells to those markers, called outer surface protein antigens (OSP antigens). This nearly invisible layer is rarely seen in washed cultures, but can be seen regularly in tissue biopsies.(46)

The Lyme bacteria is different from other bacteria in its arrangement of DNA. Most bacteria have distinct chromosomes that are found floating around inside the cytoplasm. When the bacteria starts to divide and split in two, the chromosomes divide and the new copies of the chromosomes enter the new cell. The arrangement of DNA within Borrelia burgdorferi is radically different. It is arranged along the inside of the inner membrane. It looks something like a net embedded just underneath the skin of the bacteria. (46)

This means: We really don't understand the mechanisms of how Bb regulates its genetic material during its division.

Another unique feature to Borrelia burgdorferi are Blebs. This bacteria replicates specific genes, and inserts them into its own cell wall, and then pinches off that part of its cell membrane, and sends the bleb into the host. Why it does this we don't know. But we do know that these blebs can irritate our immune system. Dr. Claude Garon of Rocky Mountain Laboratories has shown that there is a precise mechanism that regulates the ratio of the different types of blebs that are shed. (46) In other bacteria the appearance of blebs often means the bacteria can share genetic information between themselves. We don't know if this is possible with Borrelia species. There have been reports of a granular form of Borrelia, which can grow to full size spirochetes, and reproduce. These granules are so small that they can be filtered and separated from live adult spirochetes by means of a micro-pore filter. (Stealth Pathogens Lida Mattman Ph.D. 66)

The division time of Borrelia burgdorferi is very long. Most other pathogens such as Streptococcus, or Staphylococcus, only take 20 minutes to double, the doubling time of Borrelia burgdorferi is usually estimated to be 12-24 hours. Since most antibiotics are cell wall agent inhibitors, they can only kill bacteria when the bacteria begins to divide and form new cell wall.(35,59-62)

This means: Since most antibiotics can only kill bacteria when they are dividing, a slow doubling time means less lethal exposure to antibiotics. Most bacteria are killed in 10-14 days of antibiotic. To get the same amount of lethal exposure during new cell wall formation of a Lyme spirochete, the antibiotic would have to be present 24 hours a day for 1 year and six months! Note: Antibiotics kill bacteria by binding to the bacteria's ribosomes, and interrupting the formation of cell wall proteins.

Like other spirochetes, such as those that cause Syphilis, the Lyme spirochete can remain in the human body for years in a non-metabolic state. It is essentially in suspended animation, and since it does not metabolize in this state, antibiotics are not absorbed or effective. When the conditions are right, those bacteria that survive, can seed back into the blood stream and initiate a relapse. (59-62,70)

This means: Just because a person is symptom free for long lengths of time doesn't mean they aren't infected. It may be a matter of time. Whereas viral infections often impart a lifelong immunity, Lyme, like other bacterial infections, does not retain active immunity for long periods of time. People are often reinfected with Lyme. (96)

How does the Lyme bacteria travel from the bloodstream to other tissues? While we have known for a long time that the Lyme spirochete can show up in the brain, eyes, joints, skin, spleen, liver, GI tract, bladder, and other organs, we didn't understand the mechanism by which it could travel through capillaries and cell membranes. (Abstract 644) Then Dr. Mark

Klempner presented at the 1996 LDF International Lyme Conference an interesting paper that gave us part of the answer.

Many researchers have observed that the Lyme spirochete attaches to the human cells' tip first. It then wiggles and squirms until it enters the cell. What Dr. Klempner showed was that when the spirochete attached to the human host cell, it caused that cell to release digestive enzymes that would dissolve the cell, and allow the spirochete to go wherever it pleases. This is very economical to the bacteria to use our own cell's enzymes against us, because it does not need to carry the genes and enzymes around when it travels. Dr. Klempner also showed that the spirochete could enter cells such as the human fibroblast cell (The skin cell that makes scar tissue.) and hide. Here the pathogen was protected from the immune system, and could thrive without assault. More importantly, when these Bb-fibroblast cultures were incubated with 10 x the MIC for IV Rocephin, two thirds of the cultures still yielded live spirochetes after two weeks, and in later experiments for more than 30 days. If we can't kill it in a test tube at these high concentrations in four weeks, how can we hope to kill it in the human body? (22,48,79,80,)

This means: The infection can enter the tissue that is optimal for its survival, and it may evade the immune system and antibiotics by hiding inside certain types of cells.

Another interesting observation about this bacteria is how it interacts with our body's immune system; Dr. David Dorward of Rocky Mountain Labs made a video tape of how Borrelia burgdorferi acts when surrounded by B-cells. (The type of white blood cell that makes antibody.) The spirochete attached tip first, entered the B lymphocyte, multiplied and ruptured the cell. It repeated this process for three days until the B-cells were able to come to an equilibrium. A matter of concern was that some of the spirochetes were able to strip away part of the B-cell's membrane, and wear it like a cloak. (Dorward, Hulinska 1994 LDF Conference Vancouver BC)

This means: If this spirochete is evolved enough to attack our B-lymphocytes, then it may also be evolved in other ways that we do not yet understand. It is for certain that its ability to kill B-lymphocytes evolved as part of a defense mechanism to evade its own destruction. The observation that it can use the B-cell's own membrane as camouflage indicates that it may be able to go undetected by our immune system. The way our immune system is supposed to work is that it recognizes foreign invaders as being different from self, and attacks the infection.

Unfortunately, the immune system sometimes attacks our own cells. This is called autoimmune disease. If a foreign invader has a chemical structure similar to our own tissue antigens, our bodies sometimes make antibodies against our own tissues. In people with Lyme disease scientists have discovered auto-antibodies against our own tissues including nerve cells (axons), cardiolipid, myelin (also seen in MS), myelin basic protein (also seen in MS), and neurons (brain cells) (23,28,38-40,43,45,56,57,60,88)

When the immune system finds a foreign invader, it tags that invader in a number of ways. A cell called the macrophage can engulf the bacteria, and then communicate to other immune cells the exact description of the bacteria. Another cell might mark the cell with antibody which attracts killer T-cells. Some types of T-cells communicate to other cells what to attack, and regulate the immune assault. But sometimes the body can produce a type of antibody that doesn't attack or help. A blocking antibody will attach and coat the intruder, but it won't fix compliment, and it shields the bacteria from further immune recognition. In Lyme we have

seen quantities of IgG4 blocking antibody such as is seen in some parasitic infections. (Tom Schwann RML 92 LDF Conference) *Note: Compliment is a term used for a series of 18 + digestive proteins that are only activated by signals from our immune system, such as compliment fixing antibodies.

In order for the immune system to make an attacking antibody, the immune system must first find an antigen which it can attack. Unfortunately, as seen by freeze fracture electron microscope, photographs of the Lyme bacteria show that most of the antigens are on the inside of the inner membrane, and not on the outside. (60) This makes the bacteria less visible to the immune system and more difficult to attack. The most intriguing fact about Borrelia spirochetes is their well documented ability to change the shape of their surface antigens when they are attacked by the human immune system. When this occurs, it takes several weeks for the immune system to produce new antibodies. During this time the infection continues to divide and hide. (1,47,63,66)

It appears that Borrelia are able to change their surface antigens many times, and can do it quickly. In one study by Dr. Andrew Pachner MD, he infected mice with a single strain of Borrelia burgdorferi. After several weeks, he was able to isolate two slightly different forms of the bacteria. The bacteria from the bloodstream was attacked and killed by the mouse's immune sera, but the bacteria isolated from the mouse's brain was unaffected by the immune sera. The bacteria isolated from the mouse's brain had a new set of surface antigens. It appears that contact with the CNS caused the bacteria to change its appearance. Since the brain is isolated from the immune system and is an immune privileged site, the bacteria became its own separate strain. (47,97)

This means: Infections of the bloodstream may be different from the infections that are sequestered in the brain. While we continue to have active immunity in the bloodstream, the brain has no immune defenses except for circulating antibodies. So, if those circulating antibodies are ineffective to attack the bacteria in the brain, then the brain is left without any defenses, and the infection goes unabated.

Over 100 references, abstracts and diagrams are inserted into the text to support the statements in this chapter.

Another peculiar observation of these bacteria is seen inside the bacteria. When the genetic control mechanisms of this bacteria are inhibited with antibiotics known as DNA Gyrase Inhibitors (ciprofloxin) the bacteria start to produce bacterio-phage. A phage is a virus that specifically attacks bacteria. In this case there are two distinct forms. This means the Lyme bacteria at one time were attacked by viruses. It was able to suppress them, but the DNA to make the phage is still incorporated within the DNA of the bacteria. Perhaps activation of this phage could one day be beneficial to treating chronic Lyme patients? (JTBD 94)

What happens when the infection gets to the brain? In the case of Lyme disease, every animal model to date shows that the Lyme spirochete can go from the site of the bite to the brain in just a few days. (41,60, abstract 644) While we know these bacteria can break down individual cell membranes and capillaries, its entrance into the brain is too pronounced for such a localized effect. When the Lyme bacteria enters the human body, we react by producing several immune regulatory substances known as cytokines and lymphokines. Several of these act in concert to break down the blood brain barrier. (E.g. Il-6, Tumor Necrosis Factor-alpha, Il-1, Transforming Growth Factor-beta etc.) In addition to affecting the

blood brain barrier, these cytokines can make us feel ill, and give us fevers. (54,60,) (JID 1996:173, Jan)

Since the brain has no immune system, it prevents infection by limiting what can enter the brain. The capillary bed that surrounds the brain is so tight that not even white blood cells are allowed to enter. Many drugs can't enter either, making treatment of the brain especially hard. For the first ten days of a Lyme infection, the blood brain barrier is virtually nonexistent. This not only allows the Lyme bacteria to get in, but also immune cells that can cause inflammation of the brain. (41) *Note: The breakdown of Bb was shown to occur by tagging WBCs, albumin, and other substances known not to cross the BBB with radioactive Iodine. The CSF was tested, and then the animals were infected with Bb. Then the CSF was tested everyday for several weeks. The result: No cross over of Iodine in the control group, 100% crossover in the infected group for 10 days. The infection had the same result as injecting the radioactive iodine directly into the brain. (60)

When the human brain becomes inflamed, cells called macrophages respond by releasing a neuro-toxin called quinolinic acid. This toxin is also elevated in Parkinson's Disease, MS, ALS, and is responsible for the dementia that occurs in AIDS patients. What quinolinic acid does is stimulate neurons to repeatedly depolarize. This eventually causes the neurons to demyelinate and die. People with elevated quinolinic acid have short-term memory problems. (27,29-37,40-42,74,75, 82-84,87-90)

This means: If we think of all of our brain cells like telephone lines, we can visualize the problem. If all of the lines coming in are busy, we can't learn anything. If all of the lines going out are busy, we can't recall any memories. Our thinking process becomes impaired.

A second impairment to clear thinking that Lymies experience is the restriction of proper circulation within the blood vessels inside the brain. Using an instrument called the Single Photon Emission Computer Tomography scanner (SPECT scans), we are able to visualize the blood flow throughout the human brain in 3-D detail. What was seen in the brains of chronic neurological Lyme patients was an abnormal "swiss-cheese" pattern of blood flow. The cortical, or thinking region of the brain, was being deprived of good circulation; the occipital (eyesight) regions had an increase flow. This could help explain why most Lyme patients complain of poor concentration and overly sensitive eyes. (91)

Lyme Tests

There's a Lyme test, so what's the problem? There are several Lyme tests, but most of them are dependent on the body's ability to make antibody against this bacteria. As we have seen, this may be a problem. There is the S-layer protecting the bacteria; the surface antigens are not readily exposed; there may be a blocking antibody; the bacteria might be inside a human cell; the bacteria might be down regulating the immune system through cytokines; the bacteria might have altered its antigenic appearance to fool the immune system; the bacteria might be cloaked in B-cell membrane; the bacteria might be hiding in joints, tendons, white blood cells, skin cells or the brain. Remember, if even just one spirochete survives, it could cause a relapse. Then there is another problem - the tests that detect antibody can only detect free uncomplexed antibody. (23,25,55,70)

When an antibody is formed, it is meant to latch on to something and never let go until it is destroyed. Like a lock and key, antibodies fit their associated antigens. Once the antibody

attaches to the antigen it is no longer is a detectable antibody, because it has now become an antibody-antigen complex. This complex is not measurable using today's commercially available tests. Also, as the amount of antigen increases, the amount of antibody can decrease, because the antigen will trap out the available antibody and sequester it. So, a person who has a bad infection but is making a limited amount of antibody can be overwhelmed by antigen, thus making antibody detectable only if you can detect the complex.

This means: People who have the worst infections may have the lowest antibody titers, and test negative. Note: It takes four weeks from the tick bite to test positive.

There are two main categories of Lyme tests. The most common and least specific is the Enzyme Linked Immune Sera Assay or ELISA, the other is an Immuno Blot or Western Blot. The Western Blot essentially makes a map of the different antibodies we make to the bacteria. The map separates the antibodies by size and weight, and is reported in units called kilo daltons or kDa. For example, a Western Blot may report bands at 22, 25, 31, 34, 39, and 41 kDa. Each of these bands represents an antibody response to a specific protein found on the spirochete. The 41 band indicates an antibody to the flagella protein, and is non-specific. The 31-kDa band represents the OSP-A protein and is specific for Borrelia, as is the 34 band OSP-B and 25 kDa OSP-C.

In 1994, the NIH decided that there should be consistency between labs reporting Lyme Disease Western Blots, and that a specific reporting criteria should be established. This sounds good, but one could argue they made a bad situation worse. The consensus committee decided to set the standards for a positive test based on the number of bands that appear. Whereas every lab prior to the hearing had accepted bands 25, 31, and 34 as specific and significant, the NIH, without any clear reasoning, disqualified those bands from being reportable. The result was that what had been a fair good test had now become poor or even useless. (90)

How badly did the NIH bootstrap this test? The following is an analysis of the new guidelines presented as an abstract and lecture at the 1995 Rheumatology Conference in Texas. (1995 Rheumatology Symposia Abstract # 1254 Dr. Paul Fawcett et al.)

This was a study designed to test the recently proposed changes to Western Blot Interpretation. At the Second National Conference on Serological Testing for Lyme Disease, sponsored by the NIH, the committee proposed limiting the bands that could be reported in a Western Blot for diagnosis of Lyme Disease. An IgG Western Blot must have five or more of these bands: 18, 23,28, 30, 39, 41, 45, 58, 66, and 93 kDa. An IgM Western Blot must have two or more bands of the following three bands: 23, 39, 41. Conspicuously absent are the most important bands, 22, 25, 31 and 34, which include OSP-A, OSP-B and OSP-C antigens - the three most widely accepted and recognized antigens. These antigens are so immuno-reactive that they were the antigens chosen for human vaccine trials. Yet they are not considered important enough to include in the diagnostic criteria. Why?

This abstract showed that, under the old criteria, all of 66 pediatric patients with a history of a tick bite and Bull's Eye rash who were symptomatic were accepted as positive under the old Western Blot interpretation. Under the newly proposed criteria, only 20 were now considered positive. That means 46 children who were all symptomatic would probably be denied treatment! That's a success rate of only 31 %. The number of false positives under both criteria was ZERO %. * Note: A misconception about Western Blots is that they have as

many false positives as false negatives. This is not true. False positives are rare. The conclusion of the researchers was: "the proposed Western Blot Reporting Criteria are grossly inadequate, because it excluded 69% of the infected children."

We are told by manufacturers, health departments, and clinics that the Lyme ELISA tests are good and that they are useful, but in two blinded studies that tested laboratories accuracy, they failed miserably. In the latest study, 516 labs were tested. The overall result: 55% inaccurate! You are actually better off to flip a coin! (98, 99)

Repeatedly, there have been patients who are seronegative for antibodies, yet culture positive. Despite this, our medical community is dependent on these tests and relies upon them as though they were 100 % accurate. No matter how bad the tests are, as long as we have them doctors will use them. This is why doctor Samual Donta, M.D., called for a complete ban of the Lyme ELISA test at the 1996 LDF Lyme Conference. He found that, in some cases, Lyme ELISAs were more than 75 % inaccurate, yet it was relied upon as though it were the last word - and all too often it is.

The worst problem for chronic Lyme patients is that, after they are treated with antibiotics, they are told they are cured even if they have a recurrence of symptoms. There is a persistent dogma in medicine that 28 days of IV antibiotics cures all Lyme Disease. In fact, the ongoing six-year-old Nantucket Island Lyme Treatment Study showed IV antibiotics to have the highest relapse rate in late Lyme disease! This was because doctors put too much faith in IV antibiotics as being so powerful, that they did not follow up IV's with oral antibiotics. The key to treating late Lyme appears to be the length of antibiotic treatment, not the method. If IV's are followed up by six months or more of oral antibiotics, the relapse rate dropped to 13%. (Dr. Leslie Fein MD, MPH, Magnarelli MD, MPH 96 LDF Conference)

I have included in the references several published studies, case histories and abstracts that deal with culture positive patients who had been previously treated aggressively with antibiotics, often including intravenous antibiotics. Most of these cases are patients who are seronegative for any Lyme antibodies, yet are culture positive. If we are repeatedly culturing this bacteria out of patients who have been treated and who are negative by all other tests, we need to rethink our understanding of this disease! We need to treat symptoms, not tests; we need to recognize that, while Lyme is a treatable disease, in some cases it appears to be incurable. I would not like to be the doctor who under treats this disease, now knowing that relapses are potentially more dangerous than treating until the symptoms are gone. (4,6,42,49,67,68,70-96) (Lawrence C, Lipton RB, Lowy FD, and Coyle PK. Seronegative Chronic Relapsing Neuroborreliosis. European Neurology. 1995; 35(2): 113-117)

Too often, I have seen the word cured used in Lyme Disease Studies, only to find that the researchers have redefined the word cure to mean seronegative. Seronegativity is not synonymous with cure. The numerous culture positive cases in recent years should have negated that kind of logic years ago, and yet, in 1997, researchers are still publishing studies that use antibodies and PCR as the end point for cure. It's time to ask the patients one simple question: How are you feeling?

So, let's say hypothetically you are bitten by an infected tick, you get a rash, you get sick, and you have a positive test. So you get 2-4 weeks of antibiotics and you get better, but then you get sick again. No problem! You go back to your doctor, and he says, "Well, we'd better give you another Lyme test" - and its negative. Why? Even though you have an active infection,

the antibiotics cleared that infection from your blood stream. That is where your immune system is. The rest of the pathogens are hiding from the immune system inside your joints, your tendons, and your brain. Only now you don't have antibiotics to fight the infection, or any antibodies!

In a study by Dr. Musher, M.D., he looked at incompletely treated Tertiary Syphilis patients, and compared them to those Tertiary Syphilis patients who never got antibiotics. He found that the incompletely treated group went into dementia faster.

Why? Because they had no natural immunity left. Their ability to make sufficient antibody was diminished, because the antibiotics eliminated the stimulus from the blood stream, but the infection was still hidden in the brain! (35,61,62,65,74,83)

Conclusion: Lyme is an extremely complex disease that can cause long term chronic infections. Patients can be seronegative, yet culture positive (even after aggressive antibiotic therapy). The infection enters the brain early in the infection (within days). The sequestered bacteria within the CNS can be so different from the initial infection that serum antibodies are ineffective. Incomplete antibiotic treatment of Lyme Encephalitis can harm the patient.

Addendum 1:

The Final Note: Recently, there have been some very large educational institutions that have maintained a strong position on Lyme disease being easily detected and treated, with a high degree of success. Let me site two such examples:

First, several state health departments, including the Minnesota State Department of Health, have received mailings from Yale University pertaining to Lyme Disease. *(Reference: The Yale Medicine Special Report by Marc Wortman, 1996 Yale Medicine Magazine pp.1-15, May 15th, 1996)* This report was sent to several health departments throughout the United States who had received a CDC grant to initiate a patient education program on Lyme disease.

In the table of contents,Yale describes themselves as being the "Lyme Dream Team". The "dream team" then goes on to recommend what to do, "If you are bitten by a Deer Tick....".*(Excerpts from page 11, Yale Medicine, May 15th, 1996)*

- If you suspect the tick was attached for at least 36 hours, observe the site of the bite for development of the characteristic skin rash, erythema chronica migrans, (sic) usually a circular red patch, or expanding "bull's eye", that appears between three days and one month after the bite. Not all rashes at the site of the bite are due to Lyme disease. Allergic reactions to tick saliva are common. Preventative antibiotic treatment is not necessary, is costly, and may cause side effects.
- If symptoms of later-stage Lyme disease develop - arthritic swelling of a joint, most often the knee, or facial nerve palsy - have a test done. If the test is positive, have a more precise test done. Only if this test proves positive should a course of antibiotic therapy begin. Expect some symptoms to linger up to three months. No further antibiotic treatment is necessary.

Once the Minnesota State Health Department became aware of this passage, the reprint was pulled from their educational literature sent to Health Department Lyme Education Trainers. (A program developed from a CDC grant.) The advice in this excerpt is in direct conflict with

the Minnesota Guidelines for the Treatment of Lyme Disease. More importantly, in my opinion, it is bad advice that is potentially dangerous to patients!

The passage inferrs that, if you have a tick attached for 35 hours, you couldn't get Lyme. If you do have a tick bite and a rash, there is no need to seek treatment - don't go to the doctor unless you have late symptoms. The symptoms the Yale medical "Dream Team" deems as important enough to go to the doctor are a severely swollen knee, or Bell's Palsy. Even if you have these symptoms, but your ELISA Lyme test is negative, you can't have Lyme and should not seek treatment. If the ELISA is positive, you need to have a second confirmatory Western Blot Lyme test. If this test is negative, you can't have Lyme disease!

Let's review this medical advice:You are bitten by a deer tick, it is attached for 35 hours, you get a rash at the site of the bite, you develop late stage symptoms, you have a positive ELISA Lyme test, but you shouldn't pursue treatment if a second test is negative!! Apparently, antibiotic therapy is more dangerous than having late stage disease!

If your doctor wants to take on the medical/legal risk of not treating a positive tick-bite rash, symptoms, and a positive test, then he is a very brave soul. In my opinion he would be at risk of malpractice and could be held accountable for any irreparable harm that occurs due to refusal to treat late stage Lyme symptoms with antibiotics.

This article by Yale is an example of two erroneous prevailing attitudes within the medical community about Lyme disease. First, don't treat seronegative Lyme disease (even apparently in presence of a rash, and late state symptoms, post tick bite), the test are accurate. Second, once you treat with antibiotics, even despite the persistence of symptoms, the patient is cured. The trouble with relying on these two absolutes in Lyme disease is that they are quickly dismissed by any case of seronegative Lyme, or a single patient that is culture positive post-antibiotic treatment.

I would not want my reputation and credential dependent upon such a flimsy foundation. If one example of seronegative Lyme exists, or culture positive Lyme post treatment, it refutes everything these institutions have insisted is true. (See two such references, Lawrence & Masters.)

Why do these erroneous beliefs persist? Let me site my second example of large educational institutions disseminating information to doctors that supports these beliefs. The American College of Physicians and Surgeons offers a teaching seminar to doctors on how to diagnose and treat Lyme disease. Included is a VHS video tape that has several vignettes of doctors dealing with patients with potential Lyme disease. In every case, there is either a dependence on Lyme testing - or, once the patient has been treated, they are no longer capable of sustaining infection.

In my opinion, the absence of information about the accuracy of Lyme testing, the incidence of sero-negative Lyme, and the possibility of relapse post treatment, indicated to me that the tape is designed to give doctors a method of dumping Lyme patients. In no case presented is sustained treatment advocated, suggested, or advised. If symptoms persist, the patient has either post-Lyme Syndrome, or needs further testing, including a psychological work up, to find some other cause for the patient's symptoms.

In my opinion, the advice on the ACP video is based on two wrong conclusions: First, that serological Lyme tests are accurate. Second, that active infection cannot persist post-antibiotic treatment - therefore treating relapses with further antibiotics is unnecessary. (Unless supported by further serum antibody testing.) In every case that is presented, the doctor is given an opportunity to give up on his patient before considering sustained antibiotics to treat the persisting symptoms. It is a wonder that any of the patients are diagnosed at all, considering the apparent lack of recognition of even the most common Lyme disease symptoms.

Nowhere on the tape do I hear doctors asking about common and frequent symptoms of Lyme disease, such as: stiff, crunchy neck, visual complaints, heart palpitations, muscle twitches (especially in the face), fatigue, depression, urinary frequency, and memory problems. The only symptoms the educators seemed concerned about was a history of a deer tick bite, a bull's eye rash, and swollen joints. How can you make a clinical diagnosis if you don't know the most basic of symptoms? The truth is, there is no effort made to make a clinical diagnosis, except by an erythema rash. What is distressing about using the rash as diagnosis is that, in the Vanderhoof study of over 1000 chronic Lyme patients, it took on average 5.3 doctors to diagnose Lyme even in the presence of a bull's eye rash. This same study showed that a delay in treatment of more than a few months led to a much higher incidence of relapse, or chronic symptoms.

The key to dumping a Lyme patient is how to write the patient's chart to support a non-Lyme diagnosis. In no instance is the patient ever asked how they are feeling, nor is the patient's response to antibiotics ever to be considered as the end point of treatment. Once again, the clinical picture is ignored in favor of either serology, or a blind belief that all Lyme patients are cured with a few weeks of treatment. But I as you what brought the patient to the doctor? Symptoms! So, if the cure does not alleviate the symptoms, what good has the short course of antibiotics done? I can understand why treatment is discontinued in patients who feel cured, but when they are still sick it seems unconscionable. In fact, I find that the very same doctors who advocate short term treatment in Lyme disease, rarely seem to follow the same protocol when they or a family member gets sick.

I once had a discussion with an Internal Medicine Specialist, whom I've known for ten years, about the length of treatment for his Lyme patients. He was very vocal about how most Lyme is really Ehrlichiosis, and that all Lyme patients get two weeks of doxycycline, period - no exceptions. Except one. When I met him at an airport that summer, I came to learn his son had been bitten by a tick that spring. As a precaution, he kept his son on amoxicillin for three months, even though he had no symptoms or rash! Yet his late stage Lyme patients still receive two weeks of doxycycline.

Apparently the treatment for the proverbial goose is different than for the gander! It is always difficult to refute large educational institutions, and any individual doctor who tries may be jeopardizing his career. An equally difficult battle is getting doctors who have adopted these false tenets as absolutes to change their minds. It is a difficult thing for a doctor to admit that his or her paradigm of diagnosis and treatment is flawed and possibly harmful. The revelation that perhaps hundreds of patients should have received extended therapy could be an unsettling realization for many doctors.

The first hurdle is having to go against large teaching institutions who have the full support of the NIH, CDC, or AMA.Then, there is the second issue of having to confront previous

patients.What does a doctor say to a patient who really had a treatable illness instead of MS? Any new change in diagnosis and treatment is a scary proposition for most doctors if it means confronting failures. Few want to be the vanguard force in leading that crusade, but the ones who do support their patients are heroes.

While large institutions continue to disseminate their opinions and view points to millions, it is a much smaller audience that the opposition can address. Right now, doctors have to win their battles one patient at a time, and in doing so they face persecution and criticism.

Treating Lyme patient is not a profitable endeavor. Lyme patients take too much time, require lots of counseling and education, and often continue to return with symptoms despite aggressive treatment. The physician who treats Lyme disease aggressively enters into a controversial area of medicine. It is simply easier to avoid Lyme patients than treat them, because, in treating them, they have to go against all the major medical institutions who have declared only their treatment protocols are acceptable. But let's look at the real area of interest that large institutions have in not treating Lyme disease - profit!

No profit in treating Lyme patients - treatment is a low yield return. Today's clinics thrive on high profit, low overhead procedures. It is much more profitable and less risky to do a fifteen minute $600 EMG test for carpal tunnel syndrome than it is to encourage Lyme patients to spend an hour in an exam. The net reward per time spent is too small. Low overhead, low risk, high dollar return is why hospitals focus on programs to combat smoking, weight loss, drug rehabilitation, repetitive strain disorders, depression, birthing centers, diabetes education, etc. How often do you see hospitals seeking out quadriplegics, MS patients, ALS, and other chronic disabling diseases? Many of these programs have to be government subsidized before they are profitable enough for institutions to take them on.

Every major medical research facility in the last few years has been focused on two areas of research: vaccines and tests. While only 15,000 patients a year are reported as having Lyme disease (CDC figures), hundreds of thousands are tested. By one institution's own figures, they give 100 tests for every case they treat. Amazingly, Olmstead County in Minnesota has yet to report a single case of Lyme disease to the CDC. This means they have made their money by testing, not treating. (Most tests are unnecessary, because Lyme disease, according to the NIH, is a clinical diagnosis made on the basis of symptoms.) Perhaps millions of people will be vaccinated with the new vaccines. So, whoever owns that concession stands to make a fortune.

Addendum 2:

It used to be a dogma that you either publish or perish, but now it's apply for patents or perish. The last four major announcements of "new Lyme tests" were released as press releases before a single study was published in any peer review journal as to the real effectiveness of the tests. Institutions now compete with each other for patents on tests. The real money is to own the test that is going to be the new standard in medicine. This is why they publicize the slightest advancement even before they publish. They want the business before they are forced to compare their product to the competition. None of these institutions want to tell you how bad their test is they only want to tell you how much better it is than the competition's.

Let me give you an example: the University of Minnesota tried to develop a new PCR test for early Lyme detection. The PCR test is only as good as the primers1 you use, and, if you won

the patent with bad primers, you are stuck. You either buy the rights to use someone else's primers, or you use what you have. So, what do you do if you have lousy test results? You don't compare them to the competition, but rather with an easier standard. In this case, the University didn't compare their test to other PCR tests, but instead compared it to culturing. The abstract stated that patients with tick bite and bull's-eye rash were only successfully cultured 4% of the time, but the PCR was 18% positive therefore, the PCR test was four times more useful than culturing. The trouble with this comparison is that most labs can get 80% culture success, thus the researchers are setting arbitrary standards to make the test look good. This PCR test is actually four times less accurate than culturing, if the culturing is done by a competent lab.

What nobody seems to question is that the bull's-eye rash indicates 100% of the test subjects were infected, and that this PCR test only detected 18 out of every 100 tested! But no one wanted to summarize the conclusion that their test was poor, and worse than the competition. The newspaper headline read "New Lyme Test More Accurate in Early Lyme." More accurate than what??? It's all in how you express your conclusion. This study, of course, was never published, because peer review journals would have rejected it. Instead, an abstract was presented at a rheumatology conference, and a press release issued on what the press called ".a new and better Lyme test."

The trouble is that it's all hype, yet many doctors will read the headline and think that this is a better test just because it is new. The fact is that most new tests aren't better, they just represent new patents, and have better press. Recently, the Minneapolis Tribune published a press release from the U of MN on a new, FDA approved PCR test for Lyme that used synovial fluid from an enlarged knee. In the article, it gave the cost of the test, where to send it, and the labs telephone number for people to make arrangements for sending in samples. What the article implied was this was the best and newest Lyme test available. What the article failed to do was compare it to any other test, or to emphasize that most press releases of this type are less than altruistic. The patients have become secondary in the business of Lyme disease. The patients are tested, vaccinated, and sent out the door in a rush to maintain rapid turnover. Complicated patients that require treatment slows this process down, and decreases profits.

The days of doctors going on house calls are a thing of the past. It's not the doctor's fault, though. Monthly administrative meetings within most large clinics look at profit and loss statements just as though medicine were any other business. Administrators address issues of income and expenses, just like any other corporation. If patents on tests are profitable, that's what you do. If house calls aren't, then that's what you stop. The formula is maximum amount of money per hour versus least expenses per hour, with the least possible risk. The net result is: tests are profitable, vaccines are profitable, and treating Lyme patients is not profitable.

So, how do you justify not treating? You create treatment protocols that fit the needs of the clinic and not the patient. Then, you put the entire weight of the institution behind these protocols, and intimidate everyone who disagrees. Let's put these protocols to the test. If one sero-negative, culture-positive patient exits post-antibiotic treatment, their protocols crumble. It is interesting that, while most published studies supporting Lyme as a relapsing disease of active infection are either position papers, opinion/editorial pieces, or are based on that institution's own Lyme serology tests! When an institution uses its own serology tests as an endpoint for cure, the fox is definitely watching the hen house.

Serology cannot determine cure. EVER! Yet it is still being done! The American College of Physicians and Surgeons has recently published a newsletter, "The ACP Initiative on Lyme Disease, Vol. 1, Issue 1". Which sites a recently published paper that uses serology as an end point for cure, and has a short period of a few months as a follow up. At no time were the patients symptoms assessed as the basis of successful treatment. Instead, serologies were used as the determinant (Reference: Cetriaxone (IV Rocephin), compared with doxycycline for the treatment of Lyme disease. Dattwyler RJ, Luft BJ, Kunkel MJ, Finkel MF, Wormser GP, Rush TJ, Grunwaldt, et al New England J. of Med. 3373(5):289:294 July 31 1997.) The conclusion of this paper was that a short course of doxycycline (the least expensive drug known for treating Lyme disease) is as effective as a short course of "costly" IV Rocephin.

It is quite a bone to throw to the health insurance industry by saying all Lyme patient can now be treated a short period of time with the least expensive drug, but is it true? Let's examine this article and premise: At the 1993 LDF Conference, a study was presented by Dr. Daniel Cameron, MD. In his study of more than 40 nursing home patients, he found that the relapse rate for IV Rocephin for four weeks was 25%, but the relapse rate for doxycycline was 87%. The difference in this study was that the follow up was 13 months not three months.

In a six year, ongoing study using the population of Nantucket Island, there was an interesting statistic that occurred involving the use of IV Rocephin. Since the entire population of 5000+ on the island went to only four doctors, it was easy to do long term followups on patients who were treated for Lyme disease. What was found was IV Rocephin had the highest rate of relapse, unless followed up for several moths with oral antibiotics. This was because the short duration of four weeks of treatment was inadequate to prevent relapse. This was why 57% of these patients had documentable relapses.

So, any current study that compares short-term doxycycline success with IV Rocephin is comparing two inadequate treatments to each other. Yet, the conclusion does not talk about total effectiveness it simply states the two drugs are equally effective (or ineffective). By not doing long filially to determine overall relapse rate, the New England Journal study makes doxycycline look good.

The key to the Nantucket Island study, spotting the high incidence of relapse, was in the length of the followup. The longer the followup, the higher the relapse rate. Some have said that this high relapse rate may be due to reinfection, but subsequent animal models have shown this to be otherwise.

At the 1997 LDF conference, a study was presented using naïve beagles as subjects. In this study, three groups of six beagles were studied. One group of six was infected; using infected ticks, and treated with four weeks of amoxicillin. Another group was infected and treated with a double dose of doxycycline for four weeks.

The third group was the control. In the doxycycline treated group, at three months post-treatment, it appeared that 100% were cured. But, at two years at autopsy, five of the six (5/6) beagles were shown to have active infection, or complete relapse. The key to uncovering the high incidence of relapse was a long, two-year followup period. The current study cited by the ACP totally ignored this experience showing the necessity of long followup periods, and the fallibility of antibody serologies used as end points to treatment, or as a measure of affecting a cure.

A more basic study showing the inadequacy of doxycycline goes back to 1989, in an abstract from Austria. Here, the researcher incubated a live culture of Borrelia burgdorferi with doxycycline for two weeks. The culture appeared to be dead, as both motility and reproduction had ceased. The culture did not have the appearance, however, of the amoxicillin treated culture, which was filled with Lysed cells. So, using micropore filters, the researcher filtered doxycycline treated cultures, and separated the intact Borrelia from the supernatant. He then washed them, and placed the filtrate back into fresh culture media. Over two thirds of the cultures reactivated, becoming motile and beginning to reproduce. It appeared that doxycycline immobilized the bacteria by interrupting protein syntheses and metabolism. This pushed the cells into a non-metabolic state. Since the doubling rate is often used as a means of determining if the cells are alive, it was assumed that the cultures were dead, when they were in fact just dormant.

The most recent New England Journal study is deeply flawed, yet it will have an immediate impact on the use of IV Rocephin. The design of this study has ignored previous studies that show a long-term followup is needed. It ignores the fact that the use of antibody serology cannot be used as the endpoint for treatment, or for determining cure. It does not use adequate methods to document the presence of live bacteria. The study does not use the patient's symptoms as a basis of cure. So, I ask, who has something to gain from this kind of study?

It seems very coincidental that, in the past, paid medical advisors on Lyme disease for the insurance industry produce study after study showing that, in the short term, doxycycline is as effective as other more expensive drugs. Once again, the basis of this study is a dependence on serologies, and short-term treatments leading to total cure. There are dozens of studies, case histories and abstracts, which document sero-negative.

Lyme, and culture positive Lyme post-treatment. Yet, these studies are being ignored. Today, major health institutions have backed themselves into a corner, and are using their influences, economic resources, and authority to make their view point the only viewpoint.

But do these physicians that disseminate this anti-Lyme point of view really believe what they are promoting, or are they just defending a position they have taken because they don't what to admit they have taken a position which may prove to be wrong? The answer is in the fact that most of these researchers have chosen to completely ignore studies documenting active infection post-treatment, or sero-negative Lyme disease. Instead, they only accept their own studies, using their own antibody tests as endpoints for cure.

1. A PCR primer is a short piece of specific DNA that primes the test to only amplify any matching DNA.

2. Naive means uninfected animals.

Complications of Lyme Disease: Morbidity and Mortality

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