Parts rescued from web fragments by Sarah Vaughter – www.owndoc.com

Tom's personal story is at the end of this lecture of his.

More of his writings can be found at http://www.lymeneteurope.org/info/

Lecture Notes of Tom Grier: Tom Grier (Microbiologist from Minnesota) spoke at Lac Court Oreilles Convention Center in Hayward, WI. Tom's life work is to do further research and bring awareness of this illness to everyone.

Tom Grier is a microbiologist who contracted Lyme neuroborreliosis but was misdiagnosed as having Multiple sclerosis. Tom is an expert on Borrelia bacteria and considers it highly likely that MS is in fact a symptom of Lyme disease. He accused the medical establishment of corruption and ignorance.

Sadly, the only researchers and who are really knowledgeable are Lyme patients themselves. Øystein Brorson, Norwegian microbiologist, was also misdiagnosed with MS and he also thinks MS is in fact Lyme disease. Marie Kroun, a Danish GP also was forced to become a Lyme expert when the disease destroyed her career.

Introduction

Lyme disease was first recognized in Old Lyme CT in 1973 by two concerned mothers. Judy Mensch and Polly Murray felt there were too many diagnosed cases of Juvenile Rheumatoid Arthritis in their neighborhood children. Judy and Polly Murray who had backgrounds in public health collected over 200 local case histories and presented them to the CDC and CT health department.

Dr. Alan Steere M.D. investigated the local cases of JRA and coined the misnomer "Lyme Arthritis" in his 1975 publication that first described Lyme Borreliosis as an arthritic disorder.

Although the actual cause of Lyme Arthritis that was sometimes associated with a bull's-eye rash was not yet known, a treatment protocol of two weeks of tetracycline was already being recommended.

The infectious etiology of Lyme disease was not known until 1981 when Dr. Willy Burgdorfer PhD from Rocky Mt Labs isolated the new species of Borrelia bacteria from a tick from Shelter Island. The fact that "Lyme Disease" was caused by a spirochete should have been a real concern and everything we thought we knew about Lyme disease should have been reevaluated at that time.

Borrelia was a family of bacteria not only associated with relapses and antibiotic treatment failures, but also is part of the same family of bacteria that causes Tick-Bourne- Relapsing Fevers, a group of over 300 variant diseases that can be deadly within months (Borrelia duttonii and Borrelia crocidurai of Northeast Africa), or considered mild and often mislabeled as "self-limiting" such as Borrelia recurrentis found in the Southwest USA. (Ron Ferris of Canada was diagnosed with Borrelia recurrentis and was sick for years despite treatment right up until the day he died).

In 1982 the Lyme bacteria was isolated from the "Lyme Bull's-Eye rash" from patients from New England. With this new discovery causing so much excitement and demanding large sums of monies to investigate, no one wanted to admit that "Lyme" was just a new subclass of a larger world-wide disease spread by ticks that was well known for a century as tick-born-relapsing fevers.

We need to stop calling this disease Lyme disease and recognize that it is part of a worldwide problem called Borreliosis.

If we don't recognize Lyme disease as a larger worldwide health problem caused by bacteria that have a built in mechanism for variation, our health departments will define the disease out of existence.

Technically the MS/Lyme patient that recently died in Australia (a high ranking tennis player) may not have had Lyme disease even though he tested positive on two serology tests.

Why?

Because if he had Borrelia in the brain that causes relapsing fever and that variant borrelia lacks the OSP-A DNA sequence and we use a PCR osp-A test to test his brain tissues: he may test negative for osp-A, but still have Neuroborreliosis that is an osp-A deficient variant. The report would say it is not "Lyme Disease" but is any Borrelia in the brain acceptable? Ask his widow who had to fight this case to the Supreme Court just to get an autopsy. But what good is an autopsy if they look for a species unlikely to be in Australia? If a murderer kills you with a 44 caliber or a 38 caliber your still just as dead. But our CSI people looking at Lyme need to be doing a better job if all they are going to look for is Borrelia burgdorferi (stricto sensu)

Despite documented case histories of Lyme patients still being in the rash stage and yet progressing on to serious late stage complications while on tetracycline- class antibiotics, the use of cyclines for 2 weeks is still the recommended Lyme treatment. This has to change.

When Lyme was found to be caused by a spirochete, no serious reevaluation of treatment protocols ever took place. Instead major medical institutions and universities made a mad rush to register patents for Lyme tests, Lyme vaccines, and patents on the bacteria's DNA sequences for PCR testing.

But what no one did including the CDC and NIH, they didn't do pathology studies to see if treatments were successful.

"Today when Lyme patients are told that they are cured, and that their continuing symptoms must be caused by something else: I would like to know what is out there in nature that defies antibiotic treatment better than a spirochete? Also if we keep discovering new infectious disease causing organisms like anaplasmosis, why do we assume that a patient with continuing symptoms that still responds to antibiotics is "cured"? Are we so arrogant to think we have to keep our options limited only to diseases that only appear on a hospital lab-order test check-list?

Are we so sure Lyme is so easily curable?

Just one brain-autopsy that finds spirochetes post antibiotic treatment, can disprove the misguided position that a few weeks of antibiotics is sufficient to "cure" Lyme disease, and that work has already begun.

But my question is:

Who do we hold accountable? The answer has to be who ever it is that is withholding or restricting antibiotic treatment from symptomatic patients.

Who has signed their guidelines in the blood of dying Lyme patients?"

Early on doctors and scientists seemed to want to talk about Lyme disease in absolutes.

Absolutes that changed:

Lyme was absolutely transmitted by a new tick species called Ixodes dammini, then it was discovered that this new species was the same as Ixodes scapularis ticks which had been around centuries and had a wider range than we first thought that Ixodes dammini had. But in California three major mountain ranges away from New England, Lyme was transmitted by Ixodes pacificus. In Missouri the Lone Star Dog Tick had a

new Borrelia that caused Bull's-eye rashes and Lyme, in Europe it was the Sheep Tick, or the Sea Bird Tick that transmitted Lyme, and their host reservoirs were different from America's white footed mouse thought at one time the only reservoir of Borrelia burgdorferi and now just one of many rodents and possibly bird species.

Length of Tick Attachment: A tick must be attached for at least 36-48 hours. WOW! How could you ever make that conclusion on the sparse veterinary and human data we have? In fact the data suggests this is not true. Studies that looked at improper removal of a tick showed much shorter attachments are possible.

What child removes a tick properly?

Dr Elizabeth Burgess DMV Madison: her work has been overlooked for decades. Her preliminary work showed that the Borrelia species of spirochete possessed some mechanism and ability to penetrate mucous membranes suggesting transmission in cattle could be through urine –to- mouth contact putting cattle at a risk besides just ticks. Dr Burgess's work veternary was harshly and unjustly criticized without investigation or inspection. A decade later we see that Borrelia is a champion at penetrating mammalian blood vessels and endothelial cells that line the blood vessels. How hard is it to imagine mucosa capillaries in cattle are exposed targets for Borrelia to penetrate on contact? Since the introduction of the veternary Lyme vaccines, we here little about entire herds of cattle and horses being infected.

Absolute treatment:

Two weeks of doxycycline is adequate despite persisting symptoms: I am curious: Has two weeks of tetracycline ever cured a case of acne???

Let YALE defend their own position:

A synopsis of this view point can be found in the Yale Medicine Report, May 15, 1996 in an article by Marc Woortman states that: (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, such as 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.

This article suggests that treating a symptomatic patient with a negative Lyme test with antibiotics, may be more harmful than treating Lyme disease based on a single positive test and late stage symptoms. It is clear that the Yale perspective on diagnosing Lyme disease is that late stage Lyme symptoms, including a swollen knee and Bell's Palsy, do not warrant antibiotic treatment despite a positive ELISA test! It is suggested that delaying treatment in asymptomatic Lyme patient with these late stage symptoms is better than risking antibiotics.

The statement from the second paragraph tells us the intentions, "Only if this [second] test proves positive should a course of antibiotic therapy begin."

This statement tells us that Yale puts more faith in serology tests than in a patient's symptoms, Even symptoms that include an expanding EM rash after a known tick bite, a swollen knee, Bell's Palsy, and a positive blood test must all be ignored because antibiotics are costly and could cause side effects! (Apparently no one told this to dermatologists who often prescribe tetracycline for acne for years at a time at a cost of about $10 a month.) I have to ask; What motivates these doctors to deny treatment to a symptomatic patient who has a rash and a positive ELISA serology?

What happens when a tick attaches?

This is the essence of the pathogenisis of Lyme disease: if you understand this concept of infection, you begin to understand why the conservative viewpoint of Lyme is causing latent morbidity and mortality.

In several mammal studies in the late 1980s, it was shown in many species including dogs that within hours of tick attachment that the Lyme organism is with every beat of the heart circulating through the entire body. The spirochete's motility allows it to position itself into the cracks and folds of a blood vessel wall.

Borrelia burgdorferi has a tropism or an attraction to attach to the endothelial cells lining blood vessels. Once the bacteria has attached it traps tissue plasminogen that converts to plasmin and this begins the process of inflammation. This irritation causes the endothelial cells to release digestive enzymes such as basement membrane laminase, hyaluronidase, lipases, proteases. White blood cells join in at the site releasing Metalo-Matrix- Protease that facilitates cell penetration, and Tumor Necrosis Factor Alpha, and Il-1, Il-6 and TGF beta, all which play a role in cell communications and begin inflammatory cascades to begin.

The net result is within 24-48 hours we can measure the breakdown of the blood-brain- barrier in dogs that peaks at 48 hours and lasts for up to 14 days! So are we really going to say that a tick has to be attached 48 hours? This animal model suggests that the infection is potentially already established within the brain. This study was done by tagging normal blood albumin with radioactive Iodine and tracking it into the CSF of the dogs. (1989 Immunological Methods of Borreliosis Cold Spring Harbor)

If the Borrelia bacteria which as a family has been known to be neurogenic and deadly since 1910 can penetrate blood vessels, then why do physicians who should know better still make public statements in the media that Lyme disease is not transmitted transplacentally to the fetus during pregnancy. Nine published fetal autopsies since 1987 suggest otherwise, so what is their agenda for pretending to be ignorant of the facts?

The heart and soul of the mechanism of infection, or the pathogenisis of Borrelia bacteria that cause Relapsing Fever and Lyme Disease is its ability to attach to the lining of blood vessels and cause gaps or holes to appear between the endothelial cells.

The endothelial cells themselves release digestive substances, as well as our own white blood cells releasing blood-born immune factors such as tissue plasminogen, TNF-alpha, IL-1, Il-6, histamines, vaso-active amines and MMP-9 that facilitates cell penetration through any and all blood vessels, but especially important is the immediate transit of *Borrelia burgdorferi* through the blood-brain-barrier.

Animal models including dogs and primates show conclusively that this is not just a random occurrence, but rather a very specific mechanism that facilitates both the immediate and long-term survival of Borrelia within mammalian systems. In dog-models, the uninfected dog's blood protein *albumin* was tagged with radioactive Iodine, and then traced using radio-detection of entering the brain and spinal-fluid. After infected ticks were allowed to feed on the dogs, this *"leaky-brain-effect"* took less than 24-48 hours to reach its full potential.

We can measure and observe this *leaky-brain-effect* in dogs, hamsters, rabbits, and primates within hours, and we can see and detect in many other animal models including guinea pigs, mice, hamsters, and rabbits the actual transit of Borrelia into the brain of these animals within days of tick-bite, yet our own USA

health-care experts are saying without equivocation that infected ticks have to be attached for at least 36-48 hours (YALE Medical Report, IDSA-Lyme Treatment Guidelines)

Why is there such an absolute dictatorship in our guidelines when we have direct animal studies since 1989 that suggest that not only does Borrelia bacteria penetrate blood vessels and enter the brain, but once the *blood-brain-barrier* closes up 10-14 days after initial infection; the sequestered bacterial infection within the brain is undetectable by serology tests.

Our current serology tests that detect antibodies to the Lyme bacteria; require at least 4-6 weeks after exposure to produce significant antibodies to the Lyme bacterium. By then the infection can be resting dormant and quiescently within the host's brain, undetected, undetectable, and creating changes within the brain that are subtle and perhaps for awhile negligible.

Consider these other short-comings of the current antibody based Lyme serology tests:

1. To create these tests we need a representative source of the wild bacteria as a source for specific antigens that can be used to detect the specific antibodies that patients produce as a result of an infection from their local area.

Since Borrelia bacteria are genetically equipped to change their antigenic appearance (strain variation) it is important to use tests that are designed using the best representation of the bacteria that is found in the local area. There can be tremendous variation in Borrelia isolates even those found within close proximity to each other. There are well over 1000 Borrelia isolates of Borrelia burgdorferi that are strain variations in the USA alone. This is not even counting the greater variation that we see if we look at other related geno-species such as Borrelia lonestarrii in Missouri, or Relapsing Fever Borrelia in the SW USA, or the genospecies Borrelia garinii and Borrelia afzelii found in Europe, or the dozens of other related bacteria in the world that cause Lyme-like or Relapsing-Fever-Like diseases caused by various variant strains of Borrelia bacteria. Once you see this global picture you can never look at Lyme as an isolated disease ever again. It is part of a global-pandemic called Borreliosis.

But the tests that have been chosen for us, and dictated that we use are not based on any Borrelia found in nature! Why?

Since Borrelia identity changes quickly by inserting variant plasmid genes into its larger linear chromosome, the bacteria will always have built in variation unless you eliminate plasmids.

(*Borrelia burgdorferi* has about 31 circular or linear plasmid-chromosomes that facilitate genetic variation, it is estimated that over 60 genes can insert in at least three different chromosome loci resulting in over sixty to the 3rd power variations in the bacteria or potentially over 200,000 possible variations that could be predicted based on what we currently know.)

This creates an economic and practical dilemma for manufactures of Lyme serology tests who want consistency and reproducibility without the expense of isolating local bacteria from local ticks and growing them in the lab which is very difficult, time consuming, inconsistent and expensive. For this reason manufacturers use a strain that was developed in a lab that resists variation.

Strain B-31 that was originally isolated from the NE USA ticks, and was created through high-passage selection until it remained consistent from division to division.

B-31 is never found in nature, and when B-31 tests were compared and tested by independent researchers in Madison WI, France, Austria, and United Kingdom, B-31 had short comings and never had the essential antibody detection that the tests developed from local wild-strains produced. One can make an argument for B-31 consistency, but never for its local strain selectivity.

What makes this discussion about what strain we use to make Lyme serology tests completely moot; is the one fact that we completely ignore in the United States:

Once Borrelia bacteria breach the brain's defenses, absolutely no Lyme serology test short of an autopsy can rule out infection within the human brain!

Here are some other considerations about Lyme test shortcomings:

1. Dr. Lori Bakken Madison WI tested 516 labs across the USA using Lyme ELISA tests, and found them seriously lacking and only about 50 % accurate in consistency of positive tests. She used triple paired identical blinded samples. This independent test illustrates the fallibility of the Lyme ELISA test yet incredibly the ELISA is demanded by so called experts and medical authorities to be used as one of two screening tests used for the diagnosis of Lyme disease.

(Bakken LL, Callister SM, Wand PJ, Schell RF. Interlaboratory Comparison of Test Results for the Detection of Lyme Disease by 516 Participants in the Wisconsin State Lab of Hygiene/College of American Pathologists Proficiency Testing Progrm. J Clin Microbiol 1997; Vol 35, No 3:537-543

Bakken LL, Case KL, Callister SM et al. Performance of 45 Laboratories participating in a proficiency testing program for Lyme disease serology. JAMA 1992;268:891-895

Now consider the second screening test: The Western Blot was once a useful tool for diagnosing Lyme disease when used properly, but the National Western Blot Criteria meeting held in Dearborn MI changed this test from somewhat useful to useless and the logic and science behind it is so poor we have to ask ourselves what **agenda** did the committee of state epidemiologists and concerned patent-owners have? Yes people and institutions who had conflicts of financial interest had input into the two-tiered system of diagnosis that we currently use.

The nearly arbitrary decision to eliminate species specific antibody-bands from the reporting of the Western Blot tests definitely made the Western-Blot test less accurate. This change in accuracy did not come about from changing the actual test but rather by enforcing a reporting-bureaucracy that made the test less sensitive. Make no mistake the labs that do this test still see the positive bands that are banned from reporting, but are legally unable to report them.

Then to further cloud the already muddy waters of accuracy it was decided that all laboratories across the USA have to report all Western Blots as either positive or negative and not report the essential bands.

Not reporting significant Western Blot Band is to a scientist, tantamount to saying:

There are no contaminates in your drinking water, so please don't waste your time testing the well water, and if you do test the waters and find something that we haven't reported, we have already deemed that the contaminates are unimportant and benign.

Well the contaminates (bands 31, and 34) aren't as benign as we are told. Let's look at the old Western Blot reporting criteria on 66 kids with a tick-bite and bull's-eye rash compared with the new reporting criteria. This is the same test and same patients, but we are now using the Dearborn MI "Dressler" criteria for Western Blot reporting.

Western Blot and False Negatives in Children: *1995 Rheumatology Symposia Abstract # 1254 Dr. Paul Fawcett et al.* 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 under the previously mention YALE Criteria be denied treatment! That's a success rate of only 31 %.

66 Children with Bull's Eye rash Old W. Blot Criteria 100 % positive

New NIH Criteria 31% positive

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."

More issues with serology testing in Lyme:

1. The human body starts to make IgM antibody at 4-6 weeks after exposure to the pathogen, and does not make IgG antibodies for many months, yet some "Lyme Experts" want to eliminate IgM Western Blot reporting completely. This would almost certainly mean less early Lyme disease detection because most doctors who use "Two-Tiered" testing protocols will test within the first two months of tick bite and the negative Western Blots will demand that they not treat. *(See Yale treatment protocols above)*

D) The Lyme bacteria can hide almost immediately within the human body.

Without a large enough number of bacteria *(infection load)* that remains in the bloodstream for a sufficient time for the immune system to recognize the pathogen, the human immune response will be minimal or absent.

Intracellular localization of *Borrelia burgdorferi* within human endothelial cells. Ma Y, Sturrock A, Weis JJ. Infect Immun 1991 Feb; 59(2): 671-8. PMID:

Characterization of *Borrelia burgdorferi* invasion of cultured endothelial cells. Comstock LE, Thomas DD. Microb Pathog 1991 Feb; 10(2): 137-48. PMID:

Penetration of endothelial cell monolayers by *Borrelia burgdorferi*. Comstock LE, Thomas DD. Infect Immun 1989 May; 57(5): 1626-8. PMID:

Although the antibody tests would be negative possibly for years, the infection can still be alive and cause problems where it survives such as in the: joints, heart, inside endothelial cells, and inside the brain and more specifically inside brain neurons and glial cells. These bacteria cannot be detected with indirect methods like Lyme antibody test including ELISA and Western Blots, nor is it likely that DNA-PCR can detect these infections without heroic efforts to obtain proper sampling that goes far beyond just blood and urine. Time, money and expediency has forced doctors to use tests that are inadequate for the task of determining the worst possible scenario which is a persistent infection within the brain.

For the simple reason that most patients are not obviously or immediately affected by their neurological infection, the medical system has ignored these ticking time-bomb patients that are seronegative, and symptom free. But the neuro-lyme patients will pay a severe price for having doctors who refuse to go back and connect all the dots after these patients reappear in their offices with severe disabling symptoms.

Untreated and improperly treated tick bites can lead to patient disasters. Yet the treatment guidelines are so black and white that we have to now ask ourselves: Are we going to hold the users of these treatment guidelines accountable for their lack of any flexibility?

Patients are not paid to be experts in any disease, but when an entire medical community has limited all the options for sick patients both in diagnosis and treatment, then can we not hold these professionals to the same standards we would expect from a plumber? If the pipes leak, at least try and understand why?

Here is an example of unrealistic expectations from the medical community. In Valhalla New York a temporary Lyme treatment center was created that used the ELISA test to screen patients. Using this inadequate test it was determined that about 30 % of all walk in patients had Lyme disease. But here is what one of the coordinators had to say about it:

There is great hysteria about Lyme disease… less than a third of the patients who walked in to our center actually had Lyme disease.

Would we hold the same standards of accurate self-diagnosis to cancer patients, or heart patients? Do we publicly chastise patients walking into a sexually transmitted disease center and say***: "These people are wasting my time! Only a third of them have VD!!!"***

Why then is there a double standard for people who are losing their jobs, their marriages and quality of life who are just seeking answers. No wonder so many patients turn to alternative treatments. The options for Lyme disease patients to get diagnosed and aggressively treated in America is extremely limited and only getting worse every year!

Now consider this: Recently a Lyme disease expert stated nationally that there is no evidence of transplacental transfer of active infection from mother to fetus.

We have actually observed in culture Borrelia burgdorferi penetrating umbilical vein.

We also have nine case histories 1987-1989 that confirmed by either culture or direct tissue staining that in fact Borrelia burgdorferi does cross the placenta, and has caused still-births including infections within the fetal brain.

(See work and photo by Dr. Andrew Szycpanski Stony Brook Dept. of Pathology New York of Borrelia creating holes in umbilical vein.)

If I was a Obstetric Nurse or OB-GYN and told to repeat this factoid that Lyme does not cross the placenta as stated by our guiding experts on Lyme disease concerning pregnant patients, and then to also be forced by clinic administrations, insurance companies and peer pressure to rely on two-tiered testing, and follow published treatment guidelines that ignore our entire encyclopedia of knowledge on spirochetes, I would be worried! I would be worried that when the next fetal autopsy is done that I would be called to be accountable.

If Lyme disease patients have early undetectable neurological infections that resist current antibiotic treatment regimens, then why haven't we seen evidence of this?

First of all if you define treatment success by merely saying that the patient's Lyme tests are now negative after treatment, you will by virtue of incredibly bad science never see treatment failures. This is because eliminating the infection from the blood is not the same as eliminating it from the heart, brain and joints. But serologies will fail to detect these areas of sequested infection where the bacteria fails to stimulate antibody production.

Next you have to look at follow-up. If you do a study that compares doxycycline to IV ceftriaxone and the only symptom is a bull's-eye rash and your only determination of cure is the absence of rash and a negative ELISA test, and your only follow-up post treatment is two weeks. You will probably conclude that doxycycline is as effective as IV ceftriaxone, and insurance companies will smile and love you. (See M. Eckman)

Two things have been consistently true in nearly one dozen antibiotic treatment studies: The longer you treat the fewer relapses you have, and the sooner you treat after tick bite the better, and the longer you follow patients after treatment the higher the relapse rate will be. We have patients from Nantucket Island that were followed over five years after months of antibiotic treatment and still relapsed and it didn't matter if intravenous drugs were used. What was more important was How long you treated and how soon after tick bite you treated. Overall the relapse rate after 5 years approached 50 %, but to get all the facts you had to go to a Lyme Conference because this final relapse rate was never published and conveniently left out

How antibiotics work:

In most cases bacterial lethal exposure occurs only during cell division. For a spirochete like Borrelia that is a slow divider (24 hours under good conditions) to get the same lethal exposure during cell-wall synthesis as say treating strep bacteria, you would have to treat for one year and five months. Using the old microbiology formulas for tuberculosis from the 1950s, we would expect both TB and Lyme disease to require in many cases over one year of antibiotics including combination therapy. Well we learned our lesson with Tuberculosis but not yet with Lyme disease.

Relapse or Failure %

Logigian (1990) 37% After 6 months, 10 of 27 patients treated relapsed or failed treatment. 17 (63%) improved, 6 (22 percent) improved, then relapsed, 4 (15%) had no response."

Pfister (1991) 37% 33 patients with neuroborreliosis treated. After a mean of 8.1 months, 10 of 27 were symptomatic and borrelia persisted in the CSF of one patient:

Asch (1994) 28% 3.2 years after initial treatment: 28% relapsed with major organ involvement; 18% were reinfected. Persistent symptoms of arthralgia, arthritis, cardiac or neurologic involvement, were present in 114 (53%) patients."

Shadick (1994) 26% 10 of the 38 patients …relapsed within 1 year of treatment and had had repeated antibiotic treatment."

Shadick (1999) >37% 69 of 184 previously treated patients (37%) reported a previous relapse.

Treib (1998) >50% After 4.2 years, more than ½ of 44 treated patients with clinical signs of neuroborreliosis and specific intrathecal antibody production were symptomatic.

Valesova (1996) 38% At 36 months, 10 of 26 had relapsed or progressed: complete response or marked improvement in 19, relapse in 6, and new symptoms in 4.

Also in modern Lyme disease mouse model; the infection appears to have disappeared, but ticks that feed on the mice can then infect other mice. We might be looking for spirals, but that doesn't mean that's what we will find in every case.

Spirochetes are masters at morphing and changing forms. It helps them survive or another way of putting it; it contributes to relapses occurring even after aggressive antibiotic therapy.

What these early MS researchers found was amazing.

First most isolated the bacteria from the human MS lesions, but just like Syphilis, they found it was only possible to keep them alive in animal models.

Culturing Borrelia in 1911 was just not yet possible.

Once the organism was introduced to various animal models, it was often and many times re-isolated from the brains of the animals and reintroduced to new uninfected animals with exactly the same results.

The bacteria found its way to the brain of the animals, and the brain tissue could cause infection in uninfected animals.

The research became so established that the researchers often communicated with each other and commonly referred to the organism as “The MS Spirochete” which was eventually named Myela phethora or “myelin loving” by Dr. Gabriel Steiner from Germany.

Dr. Steiner was the most fastidious and persistent of all the MS/spirochete researchers, and wrote several position papers on the position, that MS was caused by an unidentified species thought most likely to be in the Borrelia family of spirochetes.

Steiner transferred the MS agent to many animals including monkeys. He created a better silver-stain, which we still use, today and is called Steiner-Silver-Stain.

When things got dicey for Jewish scientists in Germany in the mid 1930s, Steiner fled Germany and resettled in Ann Arbor, Michigan.

Steiner did not publish again for over a decade, and was amazed that America had nearly no knowledge of the European spirochete model of MS, so he wrote an article in 1952 called: “The Pathogenic Role of Spirochetes in the Etiology of Acute Plaques in MS”.

What Steiner found in American MS patients was the same as other parts of the world. MS lesions sometimes contained spirochetes that could infect animal models.

Compare below the photo of a spirochete from the lesion of a German MS patient in 1922, compared to the spirochete isolated from an American MS patient in 1952 from Michigan.

His work was completely corroborated by an American scientist Dr. Rachael Ichelson, who worked in public health in Philadelphia for 40 years.

If we look back and do a quick review of the lecture so far, we see some important points that keep repeating themselves in all stages and aspects of Lyme disease.

This is because of their fundamental importance in the disease process. To understand and make sense of the end stages of Lyme disease, we have to understand the fundamentals.

My Lyme misdiagnosed as MS

By Tom Grier, M.S.

In 1978 something strange happened to me that I now believe was the harbinger of more ill fortunes to come. In the winter of 1978, I was training for a marathon foot race. It was being held early in the summer, so I had to train all winter by running on a small sixteenth-mile indoor track. The running track was deep in the bowels of an old campus building that was built in the late 1800s. It had dim lighting and a funny smell, and was always heated to about 85 degrees. I was constantly being asked by the track coach to relocate so that the track and field team could train. When Spring approached, I was only too glad to run in the mud and grime of the local woods. I enjoyed splashing through puddles and traversing streams, celebrating the joy of being young. In an average week, I would run more than 60 miles. I ran two marathons that summer, and continued to run in the woods through the changing leaves, right up until the snow became impassable. During this time, I was taking graduate school courses in immulogy and microbiology at the medical school in Duluth. It was while I was in the microbiology lab that I saw the first of many personal medical conundrums which would follow.

I pricked my finger and placed a drop of my blood on a slide. I did a routine blood smear which allowed me to do a white blood cell count and a differential ratio of the various cell types. I was shocked when I found I had 7% eosinophils. A normal count would be 1%. In a patient with extreme allergies 3-4 % would be normal, but 7% was quite high. I had an instructor repeat the smear, and she found it to be even higher. There were some discussions of a rare fatal blood disorder, perhaps a parasitic disease, but we all opted for the more conservative explanation of allergies. Even though I hadn't had so much as a sniffle or sneeze all year, we all concluded that my spring time allergies were kicking in. We also ignored the fact that my neutrophil count was down, and my overall WBC count was low.

A week later I got shingles on one side of my body. Why? I was in the best shape of my life. I only had a mild fever and a mild case of shingles, but why did I get an outbreak of herpes zoster at age 24? Normally, shingles is a condition elderly people get when their immune systems are compromised. About this time I also had a case of nonspecific urethritis; a burning sensation when I urinated. I was treated with two weeks of tetracycline and it went away, and so did my Shingles. I really gave very little thought to these events, and it was only years later that I would learn of a disease called Human Granulocytic Ehrlichiosis (HGE), which I now believe I had contracted from a tick bite I received during all the training I was doing in the woods. What I didn't know then was that I probably had a second tick-borne disease that would not even be isolated for another three years - Lyme Disease!

I had always been able to function on just a few hours sleep, but by 1980 I was sleeping a lot, and when I was awake I was tired, depressed, fatigued, and constantly distracted. It seemed I could not get myself to concentrate on any one project for long. I had transferred graduate schools, to the University of Washington. There, I lived in a cabin in the woods where I had to chop my own wood for heat. The cool, humid weather was perfect for ticks, and I was bitten many times. In 1980, tick-borne diseases in America were an anomaly, so I never gave a thought that I could have ever contracted anything serious from a tick bite. Even if I did, the symptoms would certainly manifest themselves within a few days of the bite, so tick-borne diseases never entered my thinking. I spent a summer camping all through Europe, and once again the concern of tick bites never entered my thinking.

The doctoral program in Geriatrics I had enrolled in was unexpectedly moved from the College of Biological Sciences to the College of Sociology. I quit school and went to work, with the idea I would go back to graduate school the following fall. I eventually found myself in the pharmaceutical business as a salesman. During the next decade, thoughts of school faded as I continued to suffer from unexplained depression and fatigue. I started to gain weight, and had what felt like a continual hangover that lasted from the time I awoke until I slept. When I did sleep, it was a tortured sleep where I would toss and turn and tear at my covers. I despised warmth and craved cold. My bed in the morning would look like a war zone.

Needless to say, my personal relationships were suffering. It was now 1984. I had moved back to Duluth, Minnesota, and I continued to experience an ever increasing array of symptoms. I now regularly had sharp shooting pains in my chest that felt like ice picks. I had heart palpitations; my es were sensitive to bright lights; I had manic mood swings, where I would be awake all night reading or playing piano. This was usually followed by weeks of wanting to do nothing but stay in bed.

Yet, all through these years I always had one faculty I could depend upon, and that was my brain. If I read something I knew it. I remembered it, and I could quote it almost word for word. During one of my manic episodes, it was nothing to read an entire book or two in a single afternoon. I was a walking encyclopedia of knowledge, but I was still unable to pick up on what was wrong with me.

Then, around Thanksgiving of 1990, I had an episode I could not ignore. By this time I was severely obese. I was tired all of the time, and I now had to put all my efforts into my job just to keep up. I was the top salesman in the country, and yet I could not find the time to answer my own mail. I was using all my weekends and all of my vacation time to just to keep up. I kept thinking that I was going through a bad patch and things would go back to normal. I would lose some weight and things would be fine. I'd catch up on work and start to enjoy my prosperity. As a top salesman, I was now being sent all over the country to sales conventions to help promote our drugs, and to give training seminars to fellow salesmen. I was even asked if I wanted to become a trainer. A few years earlier this would have been a wonderful perk and a great honor, but now it was torture.

I went to New Orleans, Las Vegas, Orlando, New York, Colorado Springs, Philadelphia, Cleveland, and Chicago. Each and every time it was torture. I wanted to get through the day as quickly as possible, so I could go to bed and sleep. I skipped cocktail parties; I skipped awards banquets; I even skipped important dinners with company executives - all so that I could just get those extra few hours of sleep I would need to get me through the next day. At national sales conferences, while three thousand of my fellow employees were at dinner and seminars, I would sneak back to my hotel room and sleep. Still, I was considered the top salesman in the region overall, and top in the country for our two newest products. I felt like a complete fraud.

While returning from a regional sales conference, I got lost and confused while changing planes in the Chicago Airport. I was sitting at the wrong gate for an hour, waiting for my plane to be called. Everything around me looked strange. The people sounded like cackling geese. Everyone looked like they were in fast motion, like someone had sped up the projector. Every time I turned, I was dizzy and disoriented. I was sweating, and completely lost. I had to get to a completely different end of the terminal. Because of the metal in my suspenders, when I went through the metal detectors I set them off. Here was a sweating, nervous, completely disoriented man trying desperately to get to his connecting flight, talking what was must have sounded like gibberish to the security guards, about a flight that had left more than an hour earlier. I was finally personally escorted to a sales agent, and had to spend the next several hours trying to collect my wits.

The following week, I had bouts of disorientation that would come and go. My heart palpitations were getting worse. That summer, I thought a camping trip in the Wisconsin Dells would give me some rest and perspective. I returned to work even worse than before and farther behind. By the time Thanksgiving arrived in 1990, I was only going through the motions of working at all. I now had severe joint pain in my ankles; I had an unmistakable pressure building up in my head; I was getting lost driving to places that I had been to hundreds of times. I went to the doctor many times, and each time it was some vague "...lets wait and see?" kind of attitude. I was now sleeping almost 18 hours a day, and sweating so profusely that I often soaked through my sheets and into the mattress. During this time, I was the head of our local Pharmaceutical Salesman Association, and I had to debate a panel of doctors on the topic of what benefit pharmaceutical salesmen served the medical community. It was a hostile debate on the ctor's turf. I wasn't worried though, because I slept up to about an hour before, debated for two hours, and went home and slept another 12 hours straight. The few hours that I spent awake were now like hazy dreams to me. Sleep was my only refuge. I now cared about nothing else.

In March of 1991, I was diagnosed with chronic atrial fibrillation. This was the first solid diagnosis I had received in the past year. My doctor investigated low blood sugar, high blood sugar, Wolf-Parkinson-White Syndrome, Sick Sinus Syndrome, Menier's Syndrome, Sinus Infection, anxiety panic attacks, viral encephalitis, meningitis and finally, Multiple Sclerosis. By April of 1991, I now had a constant severe pressure in my head. When I turned my head, the pressure would migrate from the top of my head to the back of my head. My vision was now reduced to a circle directly in front of my eyes, and my peripheral vision was just a blurry swirling mess of lights and images. When I would move my head, there was a disturbing gurgle as I heard bubbles move around inside my head. I found myself for hours in a trance like state almost catatonic, and severe body jerks would awaken me. These jerks were so severe that I had to catch myself to keep from falling. Then, one day I did fall. Everything was a blurry mess. was in the middle of the street floundering in front of oncoming traffic. I checked myself in at the local hospital where I was being worked up for Multiple Sclerosis.

While I was in the hospital, my family doctor went on vacation and the neurologist on duty took over my MS work up. As she walked into my room for the very first time, she picked up my chart and immediately asked what the results of my Lyme tests were? I told her in slurred speech that no one had ever mentioned Lyme disease. She said, "Well, we'll complete the MS work up, but it will all come back negative."

Negative? Why? "Because you have classic late stage Lyme." Even before I had a scheduled spinal tap and MRI, she had ordered IV Rocephin. What was it she saw in my chart that in but a few minutes she could diagnose Lyme Disease? Had not others read my chart and investigated my case for nearly a year? Where a team of specialists could only reluctantly come up with MS after nearly a year of tests, this neurologist was confident in five minutes that I had Lyme disease?

I felt relieved that I had a treatable disease, but I was angry that it was not recognized earlier! Had I not given my doctors everything they needed to make the diagnosis? Did I not patiently submit to tens of thousands of dollars of tests? Did my doctors not have every marvel of medical science at their beckon call? Did I not agree to go to half a dozen different specialist so that they could work as a team to diagnose me? What had I done wrong that allowed them to miss a simple diagnosis of Lyme disease? The answer was evident when my family doctor came back three days later and visited me at my bedside in the hospital. The first thing he said almost apologetically was "Who'd of ever thought it would be a Zebra Disease like Lyme?".

A Zebra Disease! Meaning it was as unlikely to find a Zebra in the local woods as it would be to find Lyme disease. After all, you don't look for malaria in Alaska, and apparently my doctor didn't look for Lyme in Minnesota! Yet, by 1991, hadn't Lyme become an established local pathogen? Surely a newly discovered tick-borne disease would cause doctors to learn all they could about this disease. This was my assumption, but it would be almost a year later when certain pieces of this puzzle would start to fit together.

I was too sick to do anything now except concentrate on getting better. I was told in a matter-of-fact manner that all Lyme was easily treated in 21 days, and that I would soon be back to work. Twenty one days later, I was sicker than before treatment. Every muscle in my body twitched and pulsated, and the pressure in my head was far worse. I could only sit up in bed for a few minutes before the pressure got worse, and I would pass out. I was hallucinating both visually and auditory. I heard phones ring when there were none. I saw shadows twist into menacing shapes. I heard voices talking. At night, I saw flashing lights fill my vision, and my ears were constantly buzzing with static and ringing. I felt for the first time that I may be truly going mad. I had to sleep with headphones on, playing classical music to drown out the noise in my own head. To make matters worse, my Neurologist who had been so sure about Lyme disease, now seemed completely unsure. She wanted to stop my antibiotic treatment because I wasn't "cured" yet. In fact, in many ways I was worse. "What about herxheimer reactions?", I said. "Those only last a few days", she replied.

I had to fight for treatment every single week. My heart was worse; my brain was shot; my mind was a hopeless jumble of uncontrolled thoughts - images and sounds that haunted me. It was as if several minds had been merged into one, and there was no way to sort the images. During this time, I lost my disability

status and was now without a job, living on my savings. It seemed that my whole life was out of control, yet doctors had no empathy because ,despite it all, my verbal skills were quite good. The skill by which I earned a living was now my enemy. Every word I articulated expressed a cogent, coherent, lucid individual. I thought that, by clearly expressing my symptoms in a rational manner, I would give doctors some scientific insight to this disease. I was wrong! I learned something that, in all of the years I had called on doctors as a salesmen, I had failed to observe. Most doctors have almost no scientific curiosities about new diseases, and always want the short story, one paragraph or less. How do Iiagnose it? How do I treat it? Anything that isn't black and white in medicine is dangerous territory, as far as the doctor is concerned. I was alive, I could walk, I could talk. "So", the doctors asked, " What's the problem?"

Doctors I had called on for ten years were now distancing themselves from me, because I was a threat to their paradigm of thinking about Lyme. I was pointing out things about Lyme disease that they had never known. I had read several hundred journal articles, and I thought sharing this knowledge would be met with open arms. I soon realized that I was a threat to doctors because where they had made one mistake, they had probably made hundreds. Ignorance was not only bliss; it was safer. The maxim of the new medical community was: Unknown conditions were medical-legal time bombs best left to other doctors to defuse. My journey had brought me to an unsavory conclusion. Some of the same people I went to school with - the same people I taught as a graduate student - were playing it safe. They were in essence cowards, unwilling to risk a shred of security to advance their knowledge of a disease that was threatening the lives of their patients. There was safety in numbers, and until their peers were treating Lyme dease aggressively, they were not about to be the lone maverick.

When hundreds of Lyme patients in a community all started complaining of the same persisting symptoms post-antibiotic treatment, eventually the lower ranks of doctors started reporting this to the infectious disease specialists. The infectious disease specialists responded by saying persistent symptoms were post-Lyme Syndrome, or psychosomatic disorder. The family practitioners were placated. If the Infectious disease doctors weren't worried, why should they do anything different? Then, more Lyme patients came forward, and the infectious disease experts were placed in an adversarial position. They had ignored the dozens of studies and case histories showing culture positive patients post-treatment. They are now in a position of either admitting they are wrong, and chronic relapsing seronegative Lyme Disease really does exist, or they can talk in vague generalities, always falling back on the old dogma: All infected patients are antibody positive. This is the medical loop hole that allows them to pass on treang complicated patients, and skirt all controversy. " We tested them; they were negative; end of story. Go away!"

But ask yourselves this: Currently, there is a 4.2 million-dollar NIH study to investigate the cause of Chronic Relapsing Lyme Disease. Much of this study's conclusion will be dependent upon using antibody serologies for determining diagnosis and relapse. If there is even one single study or accepted case history that proves a seronegative patient can still be culture positive for Borrelia burgdorferi, then isn't the entire basis of using any antibody serologies flawed? If the study is flawed in the beginning, the entire conclusion will be baseless. This is exactly how we got off on the wrong foot in the beginning. We depended upon serologies to indicate the endpoint and cure. We didn't know better then, but we should certainly know better now! So what's going on? Is it denial? A cover-up? Ignorance? Arrogance? The inability for egos to yield to the truth? "There are none so blind, as those who will not see." I think it is a combination of all of these things.

What keeps me devoted to bringing Lyme Disease research to the attention of doctors isn't just the daily reminder of my own symptoms, but something that occurred to an acquaintance of mine. At the same time I was tentatively diagnosed with MS, I was told about a pharmacist I used to call on who was diagnosed with what I was told was myasthenia gravis. It was a year later when I learned it wasn't MG he was diagnosed with, but MS. I promptly went over to see him at his house, and I was shocked at what I had seen. What once was a strapping 250-pound outdoors man was now a shriveled 100 pound invalid. He couldn't walk; he could only use one arm; saliva dripped from his mouth and his speech was almost indiscernible. I did a symptom check list, only to find that he had several tick bites, a history of rashes, and over half of the Lyme symptoms on Burrascano's symptom checklist.

Since his doctor, a man I had known for more than ten years, was clearly not informed about Lyme Disease, the family asked him to consult with a Lyme specialist - a young family practice doctor that was barely out of her residency. The elder physician went to the patient's house, and at the families request took several blood and urine samples, as well as cerebrospinal fluid, which were all to be sent to Mayo clinic, Igenix labs, and Marshfield Clinic for analysis In the meantime, the patient was placed on 500 mg. amoxicillin three times a day. He immediately burst into fever and sweats, and had difficulty swallowing. Although the family doctor was informed that this was a favorable and predictable Herxheimer reaction, the medication was stopped. While waiting for the results of the tests, the patient died of respiratory collapse. When the results never came, I went to the clinic lab director; a person I knew for years from working with her on the John Bear Grease Dog Sled Race. Renee' told me that all t samples were destroyed the same day they were collected. The doctor told her "...we don't send tests out of house." This meant that there was never any intention to find the truth about the patient's true condition.

What the physician didn't know was that the family had kept several of the urine samples in the freezer. Upon learning what the doctor had done, they authorized the Lyme specialist to send a sample in for analysis. It was determined there were particles of Borrelia burgdorferi in the urine. The particles were at such a high concentration that it was one of the highest positives ever seen. The remaining samples are still frozen, but the family has no interest in pursuing malpractice or medical negligence against a family friend. To me, it was a premeditated act on the physician's part to cover up the fact that he had misdiagnosed a treatable disease. Perhaps he felt it was a waste of time to even do the tests, but he indicated to the family that the samples would be drawn and sent to the various labs as requested. Yet, within minutes of returning to his clinic, he had the lab manager destroy all of the samples!

It now became apparent to me that, not only did most doctors not know much about Lyme disease, they didn't want to know! Many other things also became apparent to me, but the biggest revelation, was that doctors were unwilling or unable to say: "I was wrong, I made a mistake." Either it was their egos unable to humble themselves, or it was a fear of medical malpractice that could end their career. In either case, they were ignorant of Lyme disease and had put too much faith in tests, and not enough faith in their patients.

Since then, I have seen and heard many tragic Lyme disease stories - some of them ending in wheel chairs, and still others in death. The line in the sand that now divides the Lyme-aware from those who treat Lyme like a strep throat, is now so deep it is a chasm that I fear has become too large to build a bridge across. I am now convinced that it will take a public statement by the AMA and NIH to overcome the fear that doctors have for treating Lyme disease aggressively. There will have to be a national paradigm shift for doctors to feel safe in treating chronic relapsing Lyme disease without the fear that their own peers are questioning what they are doing. We must also legislate to eliminate third party provider's influence, such as HMO groups that pay a bonus to groups of physicians for holding down medical costs. This makes the doctors police their own peers in order to receive large bonuses. We need to eliminate insurance companies from being able to influence physician treatment decisions. Even now, Blue Cross of Minnesota is challenging a law which states that only the doctor, and not the insurance provider, can decide on the length of treatment. Their challenge is: The patient is seronegative, and therefore cannot have an active infection, so they won't pay for IV Rocephin. Yet they are willing to pay for less expensive oral antibiotics. Is it really a belief she is not actively infected, or is it actually a cost containment policy? All patients want is access to treatment!

The argument that antibiotics are overused and cause super infections is also flawed logic. It was underuse of antibiotics that caused antibiotic resistant forms of tuberculosis to evolve. The director of the CDC during the sixties was recently quoted in Discover Magazine as saying: "We didn't know we had a slow dividing bacteria that could hide in the body and sequester itself away from antibiotic treatment. If we knew then what we know now, we would have blasted tuberculosis with whatever it took to eradicate the infection off the surface of the earth. If we ever see those conditions again we will be ready!" Will we?