

CURING LYME DISEASE WITH SAMENTO

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Lyme Disease was initially regarded as an uncommon illness caused by the spirochete *Borrelia burgdorferi* (Bb). The disease transmission was thought to be solely by the bite from a tick infected with this spirochete. The Bb spirochete is able to burrow into tendons, muscle cells, ligaments, and directly into organs. A classic bulls-eye rash is often visible in the early stage of the illness. Later in the illness the disease can afflict the heart, nervous system, joints and other organs. It is now realized that the disease can mimic amyotrophic lateral sclerosis, Parkinson's disease, multiple sclerosis, Bell's Palsy, reflex sympathetic dystrophy, neuritis, psychiatric illnesses such as schizophrenia, chronic fatigue, heart failure, angina, irregular heart rhythms, fibromyalgia, dermatitis, autoimmune diseases such as scleroderma and lupus, eye inflammatory reactions, sudden deafness, SIDS, ADD and hyperactivity, chronic pain and many other conditions.

Dr. Paul Fink, past president of the American Psychiatric Association, has acknowledged that Lyme Disease can mimic every psychiatric disorder in the Diagnostic Symptoms Manual IV. This includes attention deficit disorder (ADD), antisocial personality, panic attacks, anorexia nervosa, autism and Asperger's syndrome etc. It might be prudent in any person suddenly found to have psychiatric symptoms to obtain a Q-RIBb blood test to exclude Lyme Disease.

Biology professor, Lida Mattman, author of *Cell Wall Deficient Forms: Stealth Pathogens*, has been able to recover live spirochetes of Bb from mosquitos, fleas, mites, semen, urine, blood, and spinal fluid. A factor contributing to making Bb so dangerous is that it can survive and spread without having a cell wall (cell wall deficient CWD). Many valuable antibiotics kill bacteria by breaking down the cell wall. These antibiotics often prove ineffective against Bb.

Lyme Disease is now thought to be the fastest growing infectious disease in the world. There are believed to be at least 200,000 new cases each year in the U.S. and some experts think that as many as one in every 15 Americans is currently infected (20 million persons). Dr. Robert Rowen knows a family where the mother's infection spread to 5 of her 6 children[1] (FTN1) all of whom recovered with appropriate therapy. It is difficult to believe that these children were all bitten by ticks and seems more plausible that person to person spread within the family caused this problem. Bacteriologist, Dr. Lida Mattman, states "I'm convinced Lyme disease is transmissible from person to person". In 1995 Dr. Mattman obtained positive cultures for Bb from 43 of 47 persons with chronic illness. Only 1 of 23 control patients had a positive Bb culture. Dr. Mattman has subsequently recovered Bb spirochetes from 8 out of 8 cases of Parkinson's Disease, 41 cases of multiple sclerosis, 21 cases of amyotrophic lateral sclerosis and all tested cases of Alzheimer's Disease. The complete recovery of several patients with terminal amyotrophic lateral sclerosis after appropriate therapy shows the great importance of establishing the diagnosis of Lyme Disease.

Some very important information has recently become available about the spread and magnitude of the problem with Lyme Disease. The severity of the Lyme illness is related to the spirochete load in the patient. Few spirochetes produce mild or asymptomatic infection. A study from Switzerland in 1998 pointed out that only 12.5 % of patients testing positive for Bb had developed symptoms. A German boy developed Lyme arthritis 5 years after his tick bite. Often mycoplasmal infections remain without symptoms until the victim suffers a traumatic event (stress, injury, accident etc.) These stressing events enable the mycoplasma to begin consumption of cholesterol and symptoms may begin to present. The mechanism of this deterioration is thought to be suppression of the immune system secondary to stress.

Many patients with LD have concomitant infections with other parasites (Ehrlichia in white blood cells and Babesia in red blood cells) Some patients have all 3 parasites. Each requires a different therapy with Babesia being particularly difficult to eradicate. Recently, Artemisinin appears effective in Babesia infections. All co-infections must be eliminated .to obtain a successful result.

Dr. Joanne Whitaker relates that nearly every patient with Parkinson's Disease (PD). has tested positive for Bb. Dr. Louis Romero reports that 3 patients with PD are 99 % better after TOA-free Cat's Claw (Uncaria tomentosa- pentacyclic chemotype) therapy. When Dr. Mattman cultured 25 patients with fibromyalgia all subjects had positive cultures for the CWD Bb. which causes LD. She relates that Bb can be found in tears and could thus easily appear on the hands where touching could spread LD. Several families are now documented where nearly every family member is infected. How sick the individual patient becomes probably relates to their initial spirochete dose, immune system, detoxification capability and stress levels.

Transmission of the disease has been clearly documented after bites by fleas, mites mosquitoes and ticks. There is compelling evidence that Lyme disease (LD) can be spread by sexual and congenital transfer. One physician has cared for 5000 children with LD. 240 of these children were born with the disease. Dr. Charles Ray Jones, the leading pediatric specialist on Lyme Disease, has found 12 breast fed children who have developed LD. Miscarriage, premature births, stillborn, birth defects, and transplacental infection of the fetus have all been reported. Studies at the Univ. of Vienna have found Bb in urine and breast milk of LD mothers.

Researchers at the Univ. of Wisconsin have reported that dairy cattle can be infected with Bb hence milk could be contaminated. Bb can also be transmitted to lab animals by oral intake such as food.

The Sacramento, California blood bank believes that LD can be spread by blood transfusions. The CDC (Center for Disease Control) in Atlanta, Georgia states that their data indicates that Bb can survive without detection by the blood processing techniques used for transfusions in the U.S.

Lyme Disease is the fastest growing epidemic in the world. LD is grossly underreported so there may be far more than the 200,000 cases reported annually in the U.S. Dr. Harvey and Salvato estimate that 1 billion persons in the world may be infected with LD. LD is thought to be a contributing factor in 50 % of patients who have chronic illness.

Dr. Joanne Whitaker, a Lyme disease victim from childhood, has developed a reliable test for the presence of Lyme disease. This test looks for the Bb organism, not antibodies, and is able to identify the cell wall deficient (CWD) form of the spirochete as well as the actual Bb organism. The test is called Q-RIBb which stands for quantitative rapid identification of Bb. Dr. Lida Mattman has confirmed that Dr. Whitaker's test is sensitive because there has been a 100 % correlation between a positive culture of Bb by Dr. Mattman's lab and a positive Q-RIBb test from Dr. Whitaker's Laboratory.

Case Reports Illustrating The Critical Importance Of Establishing The Diagnosis Of Lyme Disease

Case 1 Larry Powers, a former Mr. America in 1962, became ill with the symptoms of Parkinson's Disease in 1990. Sinemet therapy was taken for eight years but he gradually became worse. He became confined to a wheel chair and required help with eating. After learning that Lyme Disease might be causing his symptoms of PD he started taking TAO free cat's claw (Uncaria tomentosa-pentacyclic chemotype). Within three weeks he was out of his wheelchair and fishing for 100 pound tarpon.

Case 2 Tom Coffey at age 34 developed diplopia, severe hypertension uncontrolled by drugs, and impaired balance. A diagnosis of amyotrophic lateral sclerosis was made. Surgery was performed to

correct the diplopia. By June 2001 he was unable to swallow saliva and feeding tube nutrition was begun. His weight had fallen by 100 pounds. Nutritional support from the tube feedings produced slow resolution of the swallowing problem. Consultation with a Lyme expert uncovered the history of a bulls-eye rash after a tick bite. Therapy with Rocephin led to complete recovery.

Case 3 A young male college student developed such severe cognitive difficulties he was forced to drop out of school. A RIBb test was positive for LD and he resumed a normal life after receiving 4 months of antibiotic therapy...

What Causes Neurone Death In Amyotrophic Lateral Sclerosis ALS?

One of the most insidious mimics for Lyme disease is ALS. The neurotoxins released by the Bb organism are capable of causing neurologic dysfunction in the central nervous system that produces symptoms typical of amyotrophic lateral sclerosis. The pathological hallmark of ALS is motor neurone degeneration and death.

Research performed by Dr. Harold Clark and Dr. Garth Nicholson and coordinated by Donald W. Scott[2] (FTN2) has resulted in a breakthrough in our understanding of amyotrophic lateral sclerosis.

Mycoplasma were discovered in 1898. These are living particles of bacterial nucleic acid which do not have a cell wall. In 1971 Rottem et al[3] (FTN3) learned that most species of mycoplasma were absolutely dependent for their growth on the consumption of pre-formed sterols including cholesterol obtained from animal and human host cells. These mycoplasma live harmlessly in host cells until they are stimulated to activity by a stressing traumatic event (bullet wound, bad fall, injury from accident etc.). The growth of the mycoplasma consumes the cell's cholesterol resulting in death of the affected cell. Mycoplasma have been identified in ALS using high resolution blood morphology. In the November 9, 2001 issue of Science Dr. Daniel Mauch[4] (FTN4) et al revealed that the glial cells surrounding the motor neurone supply the extra cholesterol needed to repair and replace aging synapses. If the repair does not properly occur the motor neurone cells proceed to die from overwork. Glial cells are also heavily involved in gathering, processing and storing glutamate. Elevations in glutamate have been found in brain tissue in ALS.

A mycoplasma species, probably fermentans, which was harmlessly sequestered in a glial cell becomes aroused by some traumatic stressful event. This mycoplasma then consumes the glial cholesterol which makes up 40% of the glial cell membrane causing rupture and death of the glial cell. The death of these glial cells releases large amounts of glutamate which becomes elevated in brain tissue. Within the neurone some of the excess glutamate accesses a urea molecule. The urea molecule gives up an ammonia ion which converts a glutamate molecule into less dangerous glutamine. This leaves the former urea molecule as a cyanate ion which damages the motor neurone's mitochondria. One of the consequences of the damaged mitochondria is a decrease in the energy output available to the neurone. This produces the severe weakness and fatigue seen in patients with ALS. If the mitochondrial injury is severe the neurone dies. The death of motor neurones stops message delivery to muscle cells leading to atrophy of muscle tissue a universal finding in ALS.

This avid consumption of cholesterol may also contribute to the endocrine dysfunction seen in ALS because it decreases the amount of cholesterol available to produce estrogen, testosterone, progesterone, hydrocortisone, and aldosterone. Patients with ALS, fibromyalgia, and chronic fatigue syndrome often have hypothalamic dysfunction which may result in adrenal insufficiency, hypothyroidism, and gonadal failure.

Lyme Disease frequently exhibits neurologic abnormalities because the Bb neurotoxins are drawn to the fatty tissue found in the brain and peripheral nerves. As a consequence sudden deafness, Bells palsy, Parkinson's Disease, Multiple Sclerosis, reflex sympathetic dystrophy, peripheral neuritis, chronic pain, and a multitude of other neurologic disorders may appear.

The Influence of Toxins from Bb On The Symptoms and Course of Lyme Disease

Autopsy examinations of young persons (30s) dying from what appeared to be Parkinson's disease PD have frequently failed to confirm the basal ganglion damage that would be expected in the classic PD seen in the elderly. Some patients with illnesses of many years duration misdiagnosed as Amyotrophic Lateral Sclerosis, Multiple Sclerosis, and Parkinson's Disease have made incredible recoveries within periods as short as 24 to 72 hours when placed on TOA-Free Uncaria tomentosa (Cat's Claw) for LD.. This rapid response could not rationally be attributed to improved immune function or bacteriocidal effects on spirochetes. Bb is known to produce a group of neurotoxins. The most sensible explanation for this recovery lies in turning off or blocking the neurotoxic effects of Bb on the lipid containing structures that the Bb neurotoxins are attracted to (central nervous system, peripheral nerves, muscles, joints etc.). This sudden improvement appears to be the result of blockage and inhibition of the neurotoxins[5] (FTN5). The most important example of a "Biotoxin Illness" appears to be Lyme Disease[6] (FTN6). Patients with symptoms of Parkinson's Disease at a young age caused by neurotoxins would not be expected to show permanent structural destruction in the basal ganglia. These neurotoxins probably act at specific sites such as neurotransmitters-pre- and-post synaptic membranes, altering dopamine, serotonin, GABA, and acetylcholine molecules, thereby blocking surface membrane receptors of various kinds which would interfere with the proper action of enzymes, coenzymes and hormones. This is only one of the damaging mechanisms of action of the neurotoxins.

The TOA free form of cat's claw (Samento) may have three direct beneficial effects in humans with LD:

Immune modulation (correcting immune dysfunction)

Direct broad spectrum anti-microbial effect on spirochetes. Quinovic acid glycosides found in TAO-free cat's claw are similar to the quinilones widely used as antibiotics.

Blocking the adverse neurotoxic effects on cells, enzymes, and hormones

Whether the serious lack of energy and fatigue seen in LD are similar to the cyanate[7] (FTN7) induced damage to the mitochondria's ability to produce energy in the motor neurone found in amyotrophic lateral sclerosis or is due to failure of proper calcium channel function is not clear.

Favorable Therapeutic Results With TOA-Free Cat's Claw In Lyme Disease

A pilot study treated 28 patients with Advanced Chronic Lyme Disease with TOA-free Uncaria tomentosa (cat's claw). Conventional cat's claw contains TOA alkaloids that interfere with the desired immune modulation. The 14 person control group was given antibiotic therapy. At the study's termination 85 % of those receiving the cat's claw preparation no longer had positive blood tests for Bb. All 28 persons had experienced a dramatic improvement in their clinical condition. No significant changes were seen in the control group.

Currently it is believed that nearly all adults are infected with stealth organisms (Borrelia burgdorferi, yeast, fungi, mycoplasma, anerobic bacteria,) and have picked up toxic metals (mercury, lead, cadmium, aluminum, fluoride, aluminum etc.) both of which lead to detrimental effects on health. Samento may be of great value in eliminating some of these infectious (certainly Bb) and has also proven very effective in cancer therapy.

The Prima Una de Gato can be obtained from Allergy Research Group 800-545-9960, Nutramedix (product name Samento) 800-730-3130 or 561-745-2917, Farmacopia at 800-896-1484. and from Natural Health Team 800-416-2806. Dr. Whitaker's lab can be reached by Internet at www.bowen.org or by calling 727-937-9077 to arrange blood Bb testing. Improving nutrition, detoxifying and improving mental health all contribute to good results in treating Lyme Disease.

Removal of mercury amalgams and treatment of heavy metals may be needed.

There is convincing evidence that the Lyme Disease epidemic may have originated from the bio-warfare laboratory in Plum Island off the coast of Lyme, Connecticut. This, however, would require a lengthy discussion not relevant to this article.

Much of this information about LD was obtained from Lyme disease: Nutraceutical Breakthrough Using TOA-Free Cat's Claw published in Focus by Allergy Research Group (October 2003) and from the November and December 2003 issues of Dr. Robert Rowen's Second Opinion.

Footnotes:

1. Rowen Robert If you have ANY chronic debilitating disease, you could be the victim of a Monster Epidemic! Second Opinion Vol X111 No. 11 November 2003
2. Scott, D.W., Crusador P.O. Box 618205, Orlando, Fl. 32861-8205 October-November 2002 pg.26-32 Also see Scott, D.W. and Scott, W.L.C. Amyotrophic Lateral Sclerosis: The Probable Cause; A possible Cure 233 Government St., Suite 6 E, Victoria, B.C. Canada V* T 4P4 TOLL FREE 1-888-232-4444 ISBN 1-55395-214-6
3. Rottem, Pfend, Hayflick Sterol Requirements Of T-strain Mycoplasmas Journal Of Bacteriology 1971
4. Daniel Daniel H., Nagler, Goritz, Muller, Otto, Pfrieger. CNS Synaptogenesis Promoted By Glia-Derived Cholesterol. Science Nov. 9, 20015. Romero, Louis M.D. Ph.D Neurotoxins Focus Allergy Research Group Newsletter pg. 10 Oct. 2003
6. Shoemaker, C. M.D., Hudnall, Kenneth, Ph.D. Focus ,Allergy Research Group Newsletter pg. 10 Oct 2003
7. Scott, Donald W. Lou Gehrig's Disease is Not a Mystery Anymore Crusader pg. 31 Oct-November 2002

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