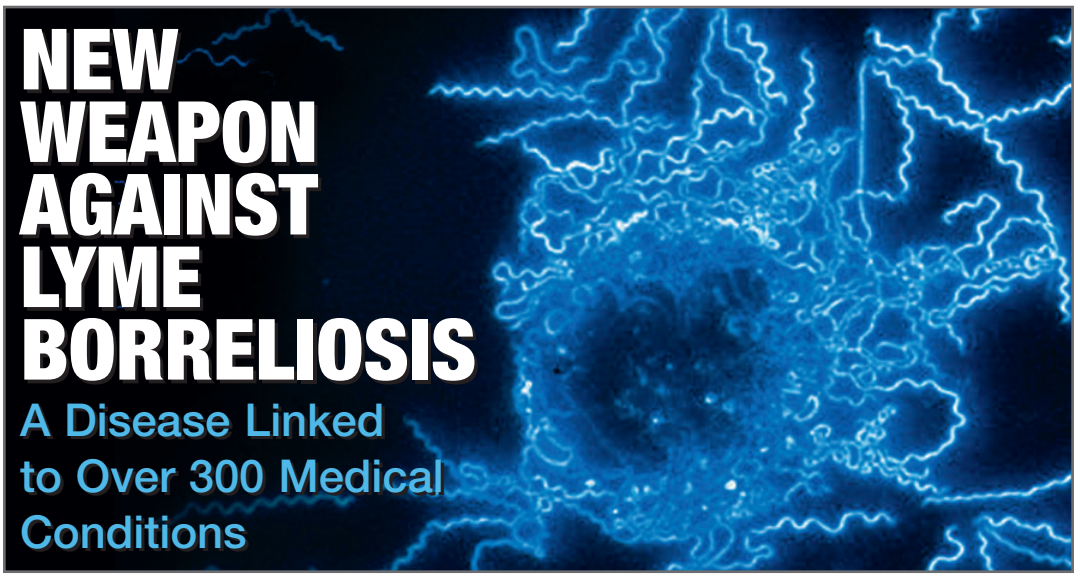
NUTRANEWS

New Thinking, New Discoveries in Nutraceutical Research — *October 2003*

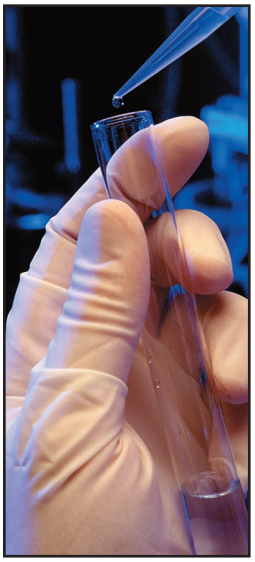
PILOT STUDY REVEALS...

NEW WEAPON AGAINST LYME BORRELIOSIS

A Disease Linked to Over 300 Medical Conditions

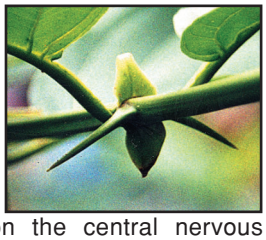


An 8-month pilot study was recently conducted with 28 patients suffering from Advanced Chronic Lyme borreliosis. All the patients tested positive for Lyme borreliosis utilizing the Western Blot blood test for Borrelia burgdorferi, the bacteria that causes Lyme disease. The control group was treated with antibiotics, and at the end of the study, from 14 patients in the group, 3 improved slightly, 3 got worse and the rest remained with no change in their clinical condition. The experimental group was treated with Samento (Pentacyclic Alkaloid Chemotype Uncaria tomentosa). At the end of the study 85% of the patients in this group tested negative for Borrelia burgdorferi, and all the patients experienced a dramatic improvement in their clinical condition. A full report will be available soon.



SAMENTO®
Medical Breakthrough

Samento, also known as TOA-Free Cat's Claw, is a rare chemotype of a medicinal plant commonly known as Cat's Claw, botanical name Uncaria tomentosa. Unlike traditional Cat's Claw products, this chemotype does not contain a group of chemical antagonists called tetracyclic oxindole alkaloids (TOAs) that act upon the central nervous system and can greatly inhibit the positive effect of the pentacyclic oxindole alkaloids (POAs). Samento contains a standardized amount of (POAs) that primarily affect the immune cells responsible for non-specific and cellular immunity, and demonstrate powerful immune system modulating properties. According to research conducted in Austria, traditional Cat's Claw products may contain as much as 80% TOAs, and as little as 1% TOAs can cause a 30% reduction in immune system modulating properties that POAs provide.



ECUADOR: Samento® Approved as Medicine

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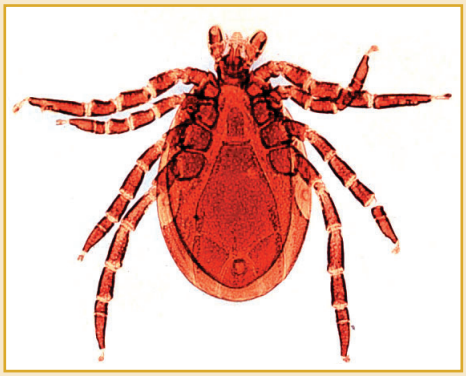
CONTRIBUTORS

William Lee Cowden, M.D.
Luis Romero M.D., Ph.D.
Joan Vandergriff, N.D.
Hamid Moayad, D.O.
Svetlana Ivanova, M.D., Ph.D.
Jo Anne Whitaker, M.D.

BIONATUS
LABORATORIOS DEL ECUADOR S.A.
9 de Octubre 424 y Chile.
Edificio Gran Pasaje 1er. Piso Ofic. 109
Guayaquil, Ecuador

tel: 593-4-2562155
fax: 593-4-2561495
E-mail: bionatus@porta.net
Web: www.samento.com.ec

NEW FINDINGS ON LYME BORRELIOSIS



History of the Disease

Lyme disease was first recognized in the United States in 1975, following a mysterious outbreak of juvenile rheumatoid arthritis near the community of Lyme, Connecticut. The rural location of the Lyme outbreak and the onset of illness during summer and early fall suggested that the transmission of the disease was by an arthropod vector.

In 1982, the etiologic agent of Lyme disease was discovered by Willy Burgdorfer. Burgdorfer isolated spirochetes belonging to the genus Borrelia from the mid-guts of Ixodes ticks. He showed that these spirochetes reacted with immune serum from patients that had been diagnosed with Lyme disease. Consequently, the lyme spirochete resembling the syphilis spirochete was given the name Borrelia burgdorferi.

Prevalent on 6 Continents

Lyme disease, known as Lyme Borreliosis in much of the world, is prevalent on 6 continents and recognized as an epidemic in many countries. Samento has been available to the public in Bulgaria, where a high incidence of Lyme disease exists, since January 2001. Within 2 months it became the most widely sold natural medicine in that country. Dr. Atanas Tzonkov, director of Bulgaria's largest private medical clinic, has treated thousands of patients with Samento. He reports that it has been used successfully to treat over 100 conditions. A possible theory is that most of these conditions were actually misdiagnosed Lyme disease or Lyme disease was a component of the illnesses that the patient was suffering from.

Methods of Transmission

W.T. Harvey, MD, MS, MPH and Patricia Salvato, MD of Diversified Medical Practices in Houston, Texas recently published the article- *Lyme disease: Ancient Engine of an Unrecognized Borreliosis Pandemic.* They were puzzled by the high number of patients testing positive for Lyme disease. Many of these patients presented with "established" criteria for Lyme disease, but others did not. The fact that southeastern Texas is a 'non-endemic' region, and that many of the patients had no history of erythema migrans rash, led the doctors to question established methods for Lyme disease consideration. Careful reflection of published research leads them to conclude the following. First, the arthropod is not the exclusive vector of Lyme disease. In addition to ticks, Borrelia burgdorferi may be carried and transmitted by fleas, mosquitos, and mites. Second, Lyme disease is not exclusively vector-borne. Compelling evidence supports horizontal (sexual) and vertical (congenital) human to human transfer.

"Compelling evidence supports... human to human transfer."

Other front-line physicians are arriving at the same conclusions. "Of the more than 5,000 children I've treated, 240 have been born with the disease," says Charles Ray Jones, MD. Dr. Jones, who is the world's leading pediatric specialist on Lyme Disease, says that about 90% of his practice is comprised of patients with the disease. He also states, "Twelve children who've been breast-fed have subsequently developed Lyme".

University of Wisconsin researchers state that dairy cattle and other food animals can be infected with B. burgdorferi and hence some raw foods of animal origin might be contaminated with the pathogen. Recent findings indicate that the pathogen may be transmitted orally to laboratory animals, without an arthropod vector. Thus, the possibility exists that Lyme disease can be a food infection.

Citing limitations of laboratory tests for the detection of antibodies to Borrelia, a study was conducted in 1995 at the University of

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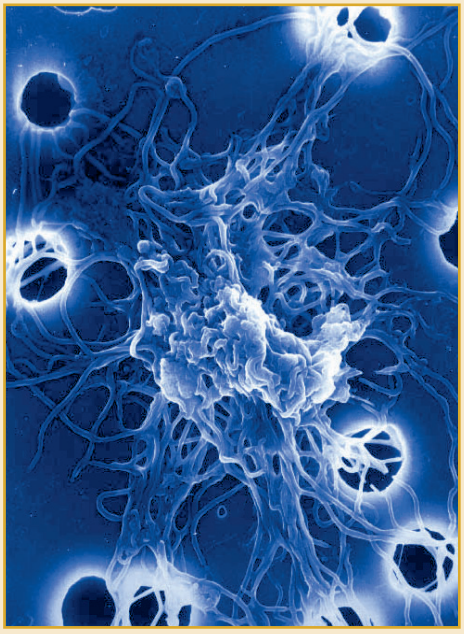
Vienna (Austria) for the detection of Borrelia. Utilizing polymerase chain reaction testing for DNA, Borrelia was found to be present in both the urine and breast milk of patients previously diagnosed with Lyme disease. A study conducted at the Sacramento (California) Medical Foundation Blood Center in 1989 states that there is evidence that the transmission of Borrelia is possible by blood transfusion. Furthermore, in 1990, a study by the Centers for Disease Control (CDC) in Atlanta, Georgia stated that the data demonstrates that Borrelia burgdorferi can survive the blood processing procedures normally applied to transfused blood in the USA.



Dormancy and Activation

It is believed that years can pass before symptoms appear in a patient that has been infected with Borrelia. In 1998, a study conducted in Switzerland demonstrated that only 12.5% of the patients that tested positive for Borrelia developed clinical symptoms confirming that Borrelia burgdorferi infection is often asymptomatic. A report from Germany outlines the case of a 12 year old boy that developed Lyme Arthritis 5 years after being bit by a tick. The case indicates that the latency period between tick bite and onset of Lyme Arthritis may last up to 5 years.

All asymptomatic carriers of Borrelia are at risk of developing Lyme disease at some point. Stress, an increasing health concern for physicians worldwide, may have been the trigger that activated Lyme disease in a patient in Sweden. The case is reported of a 26 year old woman with latent Lyme borreliosis that was concurrently activated with a herpes simplex virus type 1 infection. Immune suppression by stress may have caused activation of both infections.

Number of Cases

Lyme disease is the fastest-growing epidemic in the world. The Center for Disease Control (CDC) in Atlanta, Georgia, U.S.A. affirms that “there is considerable underreporting” of Lyme disease, maintaining that the actual infection rate may be 1.8 million, 10 times higher than the 180,000 cases currently reported. Nick Harris, Ph.D., Director of the International Lyme and Associated Diseases Society (ILADS), states “Lyme is grossly under-reported. In the U.S., we probably have about 200,000 cases per year.” Dan Kinderleher, MD an expert on Lyme disease, stated on the Today Show on June 10, 2002 that the number of cases may be 100 times higher (18 million in the United States alone) than reported by the CDC.

Jo Anne Whitaker, MD has developed a “Rapid Identification of Borrelia burgdorferi” and has over 3200 positive specimens for Borrelia burgdorferi from forty-six (46) states, including Alaska and Hawaii. In addition, Dr. Whitaker has had positive specimens from Australia, Canada, Canary Islands, Brazil, Denmark, England, France, Germany, Ireland, Netherlands, Scotland, Spain, Sweden and Switzerland.

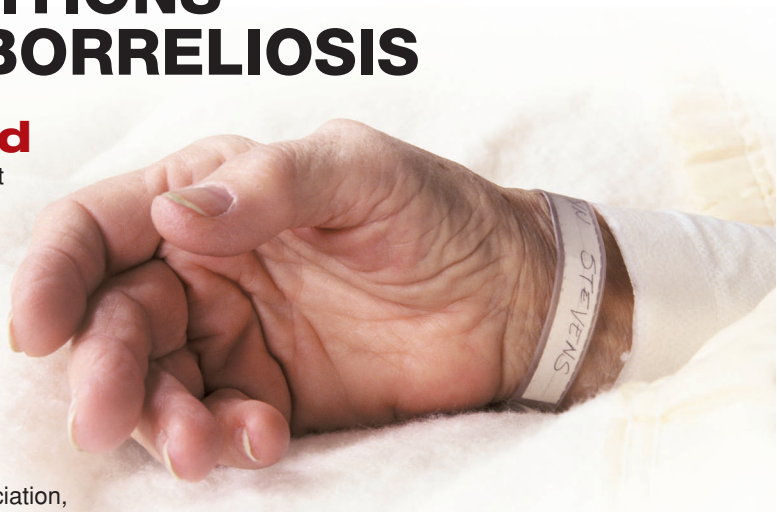
Considering vector, congenital and sexual transfer, Dr. Harvey and Dr. Salvato estimate that 15.5% of the global population, nearly 1 billion people, could be infected with Borrelia.

Lee Cowden, MD states that there are very few symptoms where one should not consider Lyme, especially given that a quarter of the U.S. population may be affected. It is estimated that Lyme disease may be a contributing factor in more than 50% of chronically ill people.

The Sierra Integrative Medicine Clinic in Reno, Nevada, states that “Authorities estimate that up to 90 percent of the population could be carrying the Lyme spirochete and that Lyme is a factor in over 50 percent of chronic illnesses.”

300 MEDICAL CONDITIONS RELATED TO LYME BORRELIOSIS

Frequently Misdiagnosed



Katrina Tang, M.D., HMD, founder and Director of Research at the Sierra Integrative Medicine Clinic in Reno, Nevada, states that Lyme disease eludes many doctors because of its ability to mimic many other diseases. According to an informal study conducted by the American Lyme disease Alliance (ALDA), most patients diagnosed with Chronic Fatigue Syndrome (CFS) are actually suffering from Lyme disease. In a study of 31 patients diagnosed with CFS, 28 patients, or 90.3%, were found to be ill as a result of Lyme.

Dr. Paul Fink, past president of the American Psychiatric Association, has acknowledged that Lyme disease can contribute to every psychiatric disorder in the Diagnostic Symptoms Manual IV (DSM-IV). This manual is used to diagnose psychiatric conditions such as attention deficit disorder (ADD), antisocial personality, panic attacks, anorexia nervosa, autism and Aspergers syndrome (a form of autism) to name a few.

List of Conditions

Lyme Borreliosis causes, mimics, is manifested as, is misdiagnosed as or is a contributing factor to many conditions. The following list of over 300 conditions was compiled by means of a non exhaustive search of published scientific literature and includes:

Abdominal pseudo-eventration,
Acrodermatitis chronica atrophicans (ACA)
Acute Acral Ischemia
Acute conduction disorders
Acute coronary syndrome
Acute exogenous psychosis
Acute meningitis
Acute myelo-meningo-radiculitis
Acute peripheral facial palsy
Acute perimyocarditis
Acute pyogenic arthritis
Acute reversible diffuse conduction system disease
acute transitory auriculoventricular block
Acute transverse myelitis
Acute urinary retention
Acquired Immune Deficiency Syndrome (AIDS)
Algodystrophy
Allergic conditions
Allergic conjunctivitis
Alopecia
Alzheimer's Disease
Amyotrophic lateral sclerosis (ALS - Lou Gehrig's Disease)
Amyotrophy
Anamnesis
Anetoderma
Anorexia nervosa
Antepartum fever
Anxiety
Arrhythmia
Arthralgia
Arthritis
Asymmetrical hearing loss
Atraumatic spontaneous hemarthrosis
Atrioventricular block
Attention Deficit Disorder (ADD)
Attention Deficit Hyperactivity Disorder (ADHD)
Bannwarth's Syndrome
Behcet's disease
Bell's Palsy
Benign cutaneous lymphocytoma
Benign lymphocytic infiltration (Jessner-Kanof)
Bilateral carpal tunnel syndrome
Bilateral facial nerve palsy
Bilateral follicular conjunctivitis
Bilateral keratitis
Bilateral papilloedema
Biphasic meningoencephalitis
Bipolar Disorder
Brain Tumor
Brown recluse spider bite
Brown-Sequard syndrome
Cardiac Disease
Cardiomegaly
Cardiomyopathy
Carditis
Carpal tunnel syndrome
Catatonic syndrome
Cauda equina syndrome
Central vestibular syndrome
Cerebellitis
Cerebral atrophy
Cerebro-vascular disease
Cervical facet syndrome
Cheilitis granulomatosa
Chiasmal optic neuritis
Chorea
Choriocapillaritis
Chronic encephalomyelitis
Chronic Fatigue Syndrome
Chronic muscle weakness
Chronic urticaria
Cerebellar ataxia
Cogan's syndrome
Collagenosis
Complete flaccid paraplegia
Complex Regional Pain Syndrome (CRPS)
Concomitant neuroretinitis
Conduction disorder
Conus medullaris syndrome
Coronary aneurysm
Cortical blindness
Coxitis
Cranial Neuritis
Cranial polyneuritis
Craniopharyngioma
Cutaneous B-cell lymphoma
Dementia
Demyelinating disorders
Depression
Dermatomyositis
Diaphragmatic paralysis
Diffuse fasciitis
Dilated cardiomyopathy
Diplopia
Discopathy
Disseminated choroiditis
Dorsal epiduritis
Encephalitis
Encephalomyelitis
Encephalopathy
Endogenous paranoid-hallucinatory syndrome
Eosinophilia
Eosinophilic fasciitis (Shulman syndrome)
Epilepsy
Epileptic crises
Episcleritis
Epstein Barr
Erythema chronicum migrans
Exanthema (local and generalized)
Extrapyramidal disorders

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List of Conditions (continued)

Facial diplegia
Fascicular tachycardia
Fatal adult respiratory distress syndrome
Fetal death
Fever
Fibromyalgia
Fibrositis
Focal nodular myositis
Frontotemporal atrophy
Generalised motor neuron disease
Geniculate neuralgia
Giant cell arteritis
Gonarthritis
Granuloma annulare
Guillain-Barré Syndrome
HLA-B27 negative sacroiliitis
Headaches (severe)
Hearing loss
Heart block
Hemiparesis
Hemophagocytic syndrome
Hepatic disorders
Hepatitis
Herniated discs
Holmes-Adie syndrome
Horner's syndrome
Human necrotizing splenitis
Hydrocephalus
Hyperacusis
Hyperbilirubinemia
Hypothyroidism
Idiopathic atrophoderma of Pasini and Pierini (IAPP)
Idiopathic facial paralysis
Infarction pain
Impaired Brainstem response
Infantile sclero-atrophic lichen
Infectious Mononucleosis
Infiltrating lymphadenosis benigna cutis
Inflammatory cerebrospinal fluid syndrome
Influenza
Internuclear ophthalmoplegia
Interstitial granulomatous dermatitis
Intracerebral haemorrhage
Intracranial aneurysm
Intracranial hypertension
Intracranial mass lesions
Intrauterine growth retardation
Iritis
Irritable Bowel Syndrome
Isolated acute myocarditis
Isolated lymphadenopathy
Isolated neuritis of the sciatic nerve
Isolated oculomotor nerve paralysis
Isolated posterior cord syndrome
Jaundice
Juvenile Rheumatoid Arthritis
Keratitis
Keratoconus
Left sided sudden hemiparesis
Lichen sclerosus
Livedo racemosa
Lofgren's syndrome
Lupus
Lymphadenosis benigna cutis
Lymphocytoma cutis
Lymphoma
Lumboradicular syndrome
Melkersson-Rosenthal syndrome
Memory impairment
Meningeal lymphoma
Meningitis
Meningoencephalomyelitis,
Meningoencephalomyeloradiculoneuritis
Meningoradiculitis
Migraines
Mono-arthritis
Monolateral chorioretinitis
Morgagni-Adams-Stokes syndrome (MAS)
Morning glory syndrome
Morphea
Motor neuron syndrome
Multiple mononeuropathy
Multiple Sclerosis
Myelopathy
Myofascial pain syndrome
Myositis
Neonatal respiratory distress
Neuromyotonia
Nodular panniculitis
Normal-pressure hydrocephalus (NPH)
Oculomotor paralysis
Oligoarthritis
Opsoclonus-myoclonus syndrome
Nodular fasciitis
Non-Hodgkin's lymphoma
Obsessive-compulsive disorder
Optic atrophy
Optic disk edema
Organic mood syndrome
Optic nerve lesion
Otoneurological Disorders
Panuveitis
Papillitis
Paralysis of abdominal muscles
Paraneoplastic polyneuropathy
Paranoia
Parkinsonism
Parotitis
Pars plana vitrectopy
Parsonage and Turner syndrome
Peripheral facial palsy
Peripheral neuropathy
Peripheral vascular disorder
Pericarditis
Perimyocarditis
Persistent atrioventricular block
Pigment epitheliitis
Polymyalgia rheumatica
Polyneuritis cranialis
Polyneuropathy
Polysymptomatic autoimmune disorder
Porphyrinuria
Posterior scleritis
Primary lymphoma of the nervous system
Presenile dementia
Progressive cerebral infarction
Progressive facial hemiatrophy (Parry-Romberg syndrome)
Progressive stroke
Progressive supranuclear paralysis
Prolonged pyrexia
Propriospinal myoclonus
Pseudo tumor Cerebrae
Pseudolymphoma
Pseudoneoplastic weight loss
Psychosomatic disorders
Radiculoneuritis
Ramsay Hunt syndrome (pleocytosis)
Raynaud's syndrome
Recurrent paralysis
Reflex sympathetic dystrophy
Reiter's Syndrome
Respiratory failure
Restless legs syndrome
Retinal pigment epithelium detachment
Retinal vasculitis
Reversible dementia
Rheumatic Fever
Rheumatoid Arthritis
Rhombencephalitis
Sacro-iliitis infection
SAPHO syndrome
Sarcoidosis
Schizophrenia
Schoenlein-Henoch purpura
Scleroderma
Secondary syphilis
Seizure Disorders
Sensorineural Hearing Loss
Septal panniculitis
Septic arthritis
Seventh nerve paralysis
Sick sinus syndrome
Spontaneous brain hemorrhage
Stevens-Johnson syndrome
Stiff-man syndrome
Still's disease
Stroke
Subacute Bacterial Endocarditis
Subacute multiple-site osteomyelitis
Subacute organic psychosyndrome
Subacute multiple-site osteomyelitis
Subacute presenile dementia
Subarachnoid hemorrhage
Sudden deafness
Sudden hemiparesis
Sudden infant death syndrome (SIDS)
Sudeck's atrophy
Synovitis
Syphilis
Symmetric Polyarthritis
Temporal arteritis
Temporomandibular joint syndrome
Thrombocytopenic purpura
Thyroiditis
Tourette's syndrome
Transient Ischemic Attack
Transient left ventricular dysfunction
Trigeminal Neuralgia
Unilateral interstitial keratitis
Unilateral papillitis
Urticaria
Uveitis
Vasculitic neuropathy
Vasculitic mononeuritis multiplex
Vasculitis
Ventricular asystole
Vertigo
Vestibular neuronitis
Vitreous clouding

UNDERSTANDING THE MECHANISM OF SAMENTO

Toxins and the Immune System

A great deal of global research exists on microbial toxins and the evaluation of their clinical and molecular toxicology on cells. This includes both tissue direct effects and effects on the blood stream (toxinemia). In particular, Borrelia burgdorferi (Lyme Borreliosis) toxicant production and its direct effect on cells, tissues and organs, is a highly relevant topic in terms of both a) the mechanism of action and b) showing targets for proposed and potential therapies.

There are reported cases of patients with diseases today known to be Lyme Borreliosis mimics, who have received Samento and have shown remarkable clinical and physical improvement within a period of as little as 24 to 72 hours. These are individuals who have been suffering for years and have been treated with conventional and CAM therapies. The rapid response to this treatment may be assumed to be toxicants blockage – inhibitions more than immune system response or spirochete bactericidal effects in a very short period of time.

Since 1819, when James Parkinson described Parkinson Disease (PD) by stating "No pathologic finding was conclusive to brain specific lesions as the true clue for the origin and evolution of PD", we have more questions than answers about the etiology of PD and other diseases such as Multiple Sclerosis, Alzheimer and many others. This leads to the reality of NOT having good and effective treatments with no side effects, and more importantly, treatments that control, stop, or reverse these diseases.

Current molecular and clinical toxicology have permitted the introduction of the term "Bio-toxins induced illness", the most important in this category being Lyme Borreliosis, which is a rapidly-spreading worldwide epidemic.

From the molecular toxicological point of view, as stated by Dr. C. Shoemaker, MD and H. Kenneth Hudnell, PhD, "Borrelia burgdorferi produces a large suite of Bio-toxins that have tissue (cells) affinity, mainly NEUROTOXINS with high molecular tropism for lipid structures, i.e., central nervous system (CNS), peripheral nerves, muscles, Joints (Synovial fluid composition and joint cartilage), lungs, and many others. Bd's Bio-toxins are more cellular than toxinemics (blood stream)". If this is true, the origin and evolution of, and complications from, chronic degenerative diseases such as PD in young adults is much more understandable. In many cases, autopsies performed on individuals in their early 30's have not demonstrated the "degenerative process" of basal brain ganglia associated with their diagnosed brain altering diseases.

These deaths seem to have been caused by the introduction of BIO-TOXINS that have altered a specific site (i.e.: neurotransmitters – pre- and post synapses membrane, altered dopamine, serotonin, GABA, acetyl-choline molecules, blocking surface membrane receptors of different kinds, altering molecular normal action of enzymes, co-enzymes and hormones). All of these and many more are widely demonstrated to be the route of action of different Bio-toxins.

Finally, in explaining the lack of energy and fatigue that is almost invariably present in Lyme Borreliosis and in the list of more than 300 illnesses reported to be "related" to Bb's bio-toxins, one molecular toxicology fact has been correlated. This is that the calcium channels' normal functioning may be altered by Bb's neurotoxicants. Therefore those neurotoxins will act on cell membrane surfaces and receptors, and within the inner cell membrane sub-molecular components, and in the cytosol. There are published reports attesting toxicant effects on cell granules and even at RNA and DNA expressions level.

In conclusion, Samento may have three "modulating" and direct actions on individuals suffering from Lyme Borreliosis and related illnesses:

- The proven Immune System modulator effect
- The proven broad spectrum anti-microbial effect
- The modulating "blocking" effects on the adverse Bio-Neurotoxins molecular actions.

Nonetheless, further research is indispensable in this matter.

How Samento May Eliminate the Pathogen

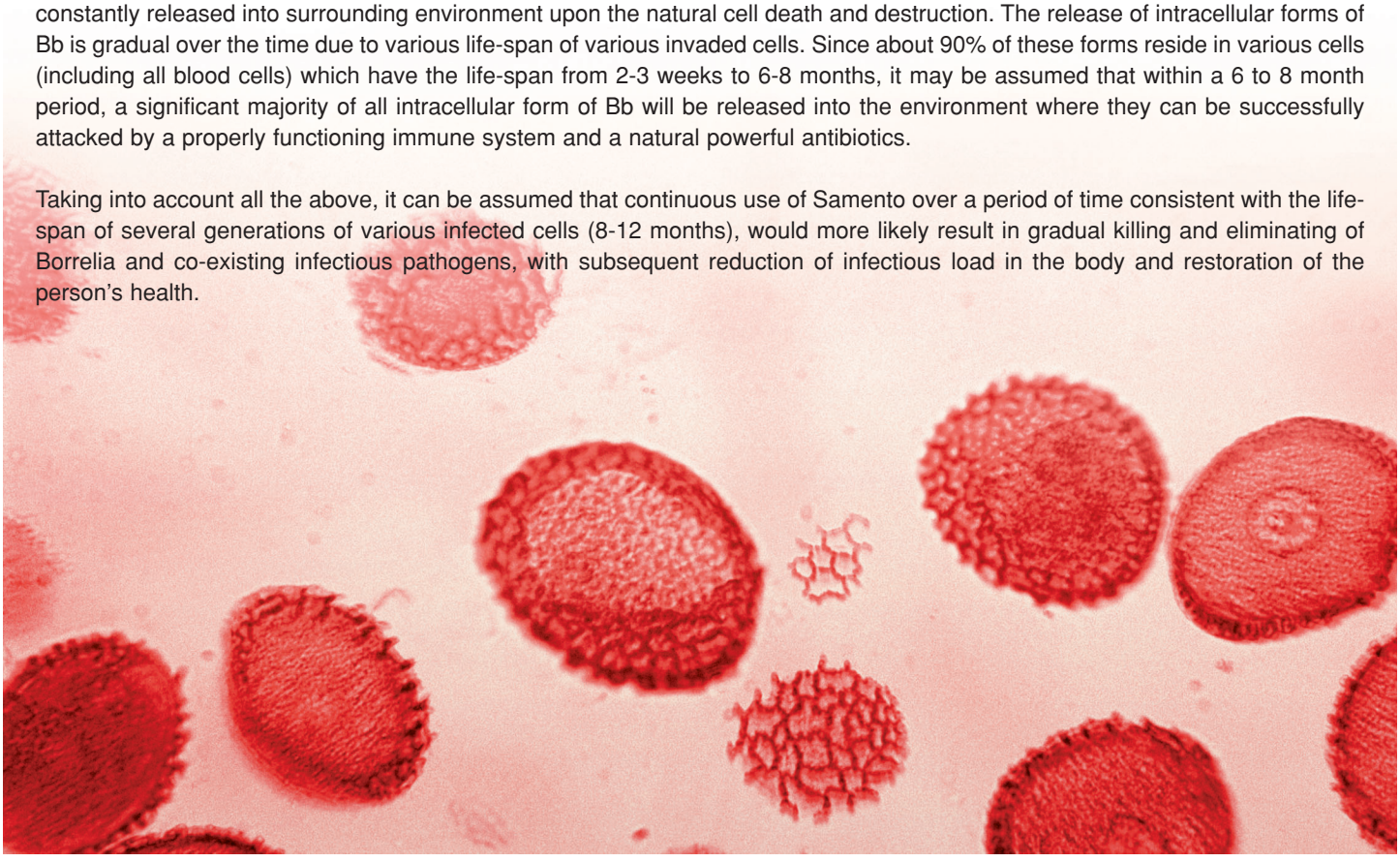
The latest research on Borrelia burgdorferi (Bb) shows that it exists in at least three different forms: the spirochete, the spheroplast (also known as L-form), and the cyst form. During the course of infection, Bb can shift among these three forms, converting from the spirochete form to the others when presented with an unfavorable environment (antibiotics, changes in pH of body fluids in chronic inflammation, etc.), and reverting back to the spirochete form to grow and reproduce upon being released from naturally aging and dying infected cells. It is during the growth period after re-conversion to the spirochete form, as well as in adult spirochete form, that Bb is most vulnerable and susceptible to antibiotics and natural elimination by the body's immune system.

The severity of Lyme presentation is directly related to the spirochete load: low load results in mild or even asymptomatic infections. With increased spirochete load from subsequent repeated infections and/or reactivated dormant infections, the severity of the disease increases. Higher loads also impair key cells of the immune system and modify the immune response, thus making the immune system unable to fight the pathogen. The negative effects on the immune system increase the longer the spirochetes are present. To prevail in the effort to fight Lyme disease, it is necessary to not only restore the immune system to normal functioning, but to boost it as well. Even a normal functioning immune system is unable to attack and eliminate Bb in all its forms.

The results of research on Samento (TOA-free Chemotype Cat's Claw) demonstrate its powerful immune system modulating and stimulating properties, along with pronounced anti-inflammatory, antioxidant, and anti-infectious effects. The diverse spectrum of the biological activities of Samento is due to its biologically active compounds. The pentacyclic oxindole alkaloids (POAs) contained in this Chemotype are generally accepted as the principal immunomodulating and immunostimulating agents. POAs are actively involved in the repair of many elements and functional mechanisms of both the innate and acquired immunity damaged by the Borrelia and other co-infections, assisting in restoration of structural and functional integrity of the immune system, enhancing its ability to eliminate the pathogens by natural way. In addition, this Chemotype contains quinovic acid glycosides – compounds with strong natural antibiotic properties (the latest generations of conventional synthetic antibiotics "Quinolones" are based on quinovic acid glycosides), which further enhance the medicinal effect of Samento in fighting the infection.

Considering the life-time of intracellular forms of Bb equivalent to the life-span of the cells invaded by these forms, they are constantly released into surrounding environment upon the natural cell death and destruction. The release of intracellular forms of Bb is gradual over the time due to various life-span of various invaded cells. Since about 90% of these forms reside in various cells (including all blood cells) which have the life-span from 2-3 weeks to 6-8 months, it may be assumed that within a 6 to 8 month period, a significant majority of all intracellular form of Bb will be released into the environment where they can be successfully attacked by a properly functioning immune system and a natural powerful antibiotics.

Taking into account all the above, it can be assumed that continuous use of Samento over a period of time consistent with the life-span of several generations of various infected cells (8-12 months), would more likely result in gradual killing and eliminating of Borrelia and co-existing infectious pathogens, with subsequent reduction of infectious load in the body and restoration of the person's health.



NEW TEST FOR IDENTIFYING THE MORPHING MENACE

Q-RIBb© *"Quantitative-Rapid Identification of Borrelia Burgdorferi"*

by Dr. Jo Anne Whitaker

Jo Anne Whitaker, M.D., a prominent international medical researcher suffering from Lyme disease and her associates have developed a new method to provide physicians with an accurate quick diagnosis of Lyme disease. Dr. Whitaker has authored over 70 scholarly publications and has accumulated numerous awards and citations throughout her career.

She has had extensive fellowship programs in pediatrics, hematology, oncology, nutrition and psychiatry. She taught in seven different medical schools and retired as a full professor of pediatrics. She spent 9 years in Southeast Asia, starting a new medical school and nutritional laboratory in Thailand and a post-graduate training program in Vietnam during the war. After returning from Vietnam, she was director of the Florida Mental Health Center in Tampa. She helped start and develop the first hospice in Florida and initiated the Little Kids Program for Abused Children at the Chi Chi Rodriguez Children's Program. Because of her personal healing experience and subsequent commitment to the Bowen Technique, she has become a Master Bowen Practitioner and teacher. She established Bowen Research and Training Institute, Inc., a not for profit corporation in 1996 to provide a research and training center for Bowen therapy.

She conducted the first clinical study to identify pathogenic E. coli by using the florescent antibody test (FAT) on infant stool specimens at a children's hospital in Detroit in 1956. She adapted the methodology to identify beta hemolytic strep disease, diphtheria and pertussis. Also using the FAT she was instrumental in developing an anti nuclear antibody test for lupus; a method for blood and parasitic antigens and tumor markers. Now some 40 years later Dr. Whitaker has found this technique to be applicable in identifying the causative agent of Lyme disease.

LYME DISEASE

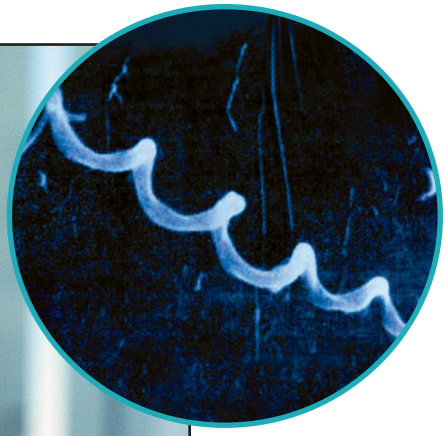
Lyme disease is called the "New Great Imitator" because, like syphilis, it attacks multiple organ systems and mimics many diseases. Both diseases are caused by a spirochete. Lyme disease is caused by Borrelia burgdorferi (Bb), an elongated spiral shaped bacterium that infects humans and animals. Bb, previously thought to be transmitted only by the deer tick (Ixodes dammini) is now recognized to be transmitted by fleas, mosquitoes and mites. There is more compelling evidence to support sexual and congenital transfer and even more recently it has been identified as a food infection.

If ignored, the early symptoms may disappear but more serious problems can develop months to years later. The later symptoms of Lyme disease can be quite severe and chronic. Muscle pain and arthritis, usually of the large joints is common. Neurological symptoms include cognitive impairment, memory loss, depression, numbness, tingling, and burning sensations in the extremities, Bell's palsy, severe pain and fatigue. Involvement of all systems such as cardiac, ophthalmic, respiratory and gastrointestinal problems can develop. Miscarriage, premature births, stillbirths, birth defects and transplacental infection of the fetus have been reported. Symptoms are often intermittent lasting from a few days to several months and sometimes years. Chronic Lyme disease, because of its diverse symptoms, mimics many other diseases and can be difficult to diagnose.

TREATMENT

Successful management and treatment of Lyme disease rests on early and accurate diagnosis. Timely treatment increases chances of recovery and may lessen the severity of any later symptoms. The most effective treatment will depend on the stage of the disease. Treatment for later stages is more difficult often requiring extended and repeated courses of antibiotic therapy and a wholistic approach to therapy. The diagnostic tests now being used for Lyme disease are neither sensitive nor specific and consequently results are not reliable.

It is well known that the serologic blood test for Lyme is insensitive, inaccurate and misses over 40 percent of cases. It is important to understand the nature of the Bb organism. Bb can change its shape from a spiral to a filament, cyst, granule, hooked rod or elbow. These variants are called L-forms, a name given by the Lister Institute where they were first studied. These L-forms are



Borrelia burgdorferi spirochete

also called cell-wall deficient (CWD) bacteria taking the non-spiral shape when they have lost much of the cell wall. In this form they do not produce an antibody response, as they have no cell wall for the individual's immune system to respond to. Classic L-forms are active metabolism centers for the production of CWD pleomorphic organisms (Bb). In this form they are able to hide within most tissues in the body, thus protecting them from any host response adverse to their well-being. CWD organisms can revert to typical morphology and may revert into adult forms of other genera, depending on the milieu. For this reason most of the diagnostic tests, i.e. ELISA and Western Blot, which depend on the production of antibodies, are inadequate. Much like the hepatitis model, antigen is present early after initial infection. Later there is an antibody response in about 70% of patients. Tests that look for antibody response will not support an early diagnosis, nor reliably confirm presence of the disease.

BOWEN RESEARCH & TRAINING

After learning about the Bowen Technique and experiencing how this simple gentle therapy relieved so many of my symptoms, I established Bowen Research & Training Institute primarily to research how the Bowen Technique affects the body. Bowen Therapy is a gentle non-invasive body therapy that seems to bring the autonomic nervous system into balance. I was investigating the effect of the Bowen Technique on the autonomic nervous system (ANS) in patients diagnosed with Fibromyalgia, it was noted that soon after the Bowen therapy, some patients developed flu-like symptoms. Dr. Lida Mattman, who has been culturing cell wall deficient (CWD) organisms from blood for 40 years was contacted to culture specimens from 25 individuals diagnosed with Fibromyalgia Syndrome. She found every sample positive for CWD Borrelia burgdorferi, the causative organism of Lyme disease.

Following this finding 103 seriously ill subjects with a variety of diagnoses were tested and found to be positive for Bb based on Mattman's Gold Standard Culture method. The conditions included: Fibromyalgia, Osteoarthritis, Mixed Connective Tissue Diseases, Polymyalgia Rheumatica, Ankylosing Spondylitis, Lupus Erythematosus, Palindromic Rheumatism, Chronic Fatigue Syndrome, Multiple Sclerosis, and Amyotrophic Lateral Sclerosis. I was shocked as I was one of that group (my diagnosis at the time was Polymyalgia Rheumatica).

As I tried to come to grips with this finding—that I might have Lyme disease, I thought about my childhood. When did this all start? I grew up in Polk County, Florida and as a kid I spent a lot of time in the woods and had numerous tick bites. I was never diagnosed with any particular malady in my childhood and I never had an EM rash. As a young adult I had bouts of multiple muscle and joint aches and pains but was able to function. I finished high school in 3 years and went on to college at USF and later to medical school at Wake Forrest in Winston Salem, NC. I was very athletic in my youth and won the Florida State Amateur Golf Championship in 1948 and again in 1952. I loved golf but because of my profession never had much time to play.

I recall having periodic muscle and joint aches and pains throughout my life receiving a variety of diagnoses, —Rheumatoid arthritis, Lupus, and Polymyositis Rheumatica. Because of my strong constitution I continued to live a productive life.

For the past six to seven years I have had severe muscle and joint pain and, in retrospect, these symptoms started around the

time I saw continuous changes in my number 18 left lower molar. This tooth was extracted January 2000 and contents tested positive for cell wall deficient (CWD) Borrelia burgdorferi (Bb) by the RIBb test and Mattman culture.

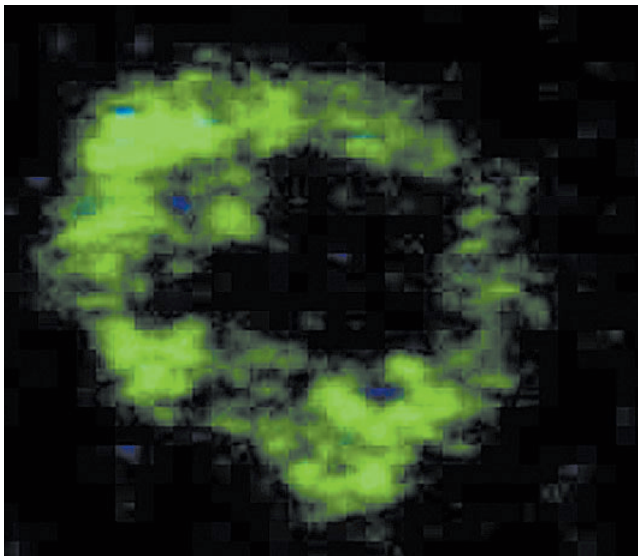
During the development of the RIBb test my blood was examined and was positive! I did not believe it. To my surprise the usual negative antibody tests were also all positive including a Lyme Urine Antigen Test (LUAT), which was exceptionally high (over 400). There was no doubt about it. I had Lyme disease and have probably had it since a little girl when I had many tick bites on hunting outings. At that time I was aware that my symptoms were becoming more intense. I had many neurological symptoms—brain fog, short-term memory loss and stiff neck; night sweats, alternating feeling hot and then feeling cold I had extreme hypersensitivity to light, sound and odors. I started wearing dark glasses even inside. I had very little energy, I was easily fatigued, and often had a sore throat. It was very difficult for me to work, I could not last more than and hour or two and even then I was "not worth much". I began to search for more information on this nonspecific disease and found that I was not atypical. My case was most likely chronic. Finally I was convinced that I did indeed have Lyme disease, so what was I to do about it. It was difficult to find a physician locally to treat my condition. I contacted several known specialists in Lyme disease and one advised me to go on long-term doxycycline, which I did. I also discovered a host of alternative therapies, which I have tried. I have experienced an improvement in many of the symptoms from regular Bowen Therapy treatments. I have tried numerous herbal and nutritional supplements; many have helped and some have not over the long term.

Since discovering that I did indeed have Lyme disease, I have been more or less on continuous antibiotic therapy.

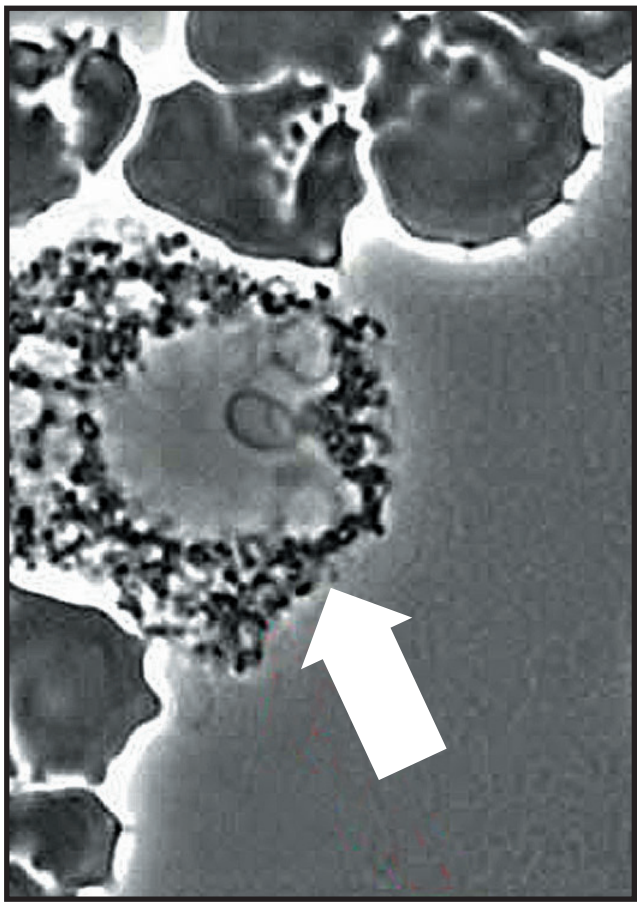
DEVELOPING A NEW TEST

After finding that there were few accurate tests for Borrelia burgdorferi (Bb), my colleague Eleanor Fort, a medical laboratory technologist, with a long history of research involvement in pediatric hematology/oncology and I, at Bowen Research and Training, developed a Rapid identification profile (RIBb©) for this organism. The method uses a fluorescent antibody technique on whole blood and is noteworthy for sensitivity and for the brief time required to complete the test, less than 60 minutes. The accuracy of this method was tested in two other laboratories with identical results. In addition we look at a concentrated suspension of red and white blood cells (rather than a routine blood smear) to identify the co-infections associated with Lyme disease (Ehrlichia in the white blood cell and the parasite Babesia, in the red blood cell. Occasionally we see all three infections in the same individual—Bb, Ehrlichia, and Babesia. All of these patients have definite abnormal peripheral red blood cell morphology. This is noteworthy, as all require different treatment.

The RIBb test has been further refined. We are currently doing Quantative Rapid Identification of Borrelia Burgdorferi Q-RIBb©. This process provides a quantitative titration (serial dilution) method of detecting the antigen in a fluid sample of a subject. The test is considered positive for Lyme disease upon detection of brightly fluorescent antigen-antibody complexes. Antibiotics do not affect the test so it is effective whether or not the person being tested is on antibiotics. When observed in phase contrast, the L-forms can be described morphologically. A preliminary report of the findings is provided within 24 hours of receiving the specimen and the final report includes digital



Specific Fluorescence of a Cell Wall deficient Borrelia burgdorferi bacteria.



Phase contrast image of the same cell showing Borrelia burgdorferi.

photographs of the findings. This test is useful in evaluating treatment by comparing pre and post serial dilution results.

We have now tested over 3500 specimens, with 500 of these very sick children, from a wide geographical distribution as previously described, and all are positive for cell wall deficient Lyme disease. The primary question is "why are there no negatives?" Does everyone have it? While the majority of our specimens come from individuals who have been diagnosed clinically, we have tested individuals who we "thought" were asymptomatic but were positive for the Bb. An interesting finding is that in 1995, Mattman found forty-three of forty-seven (43/47) patients with chronic diseases to be positive for Lyme disease, while twenty-three of twenty-two (22/23) control cultures were negative. Since 1999, all blood cultures have been positive with Bb, there were no negatives. We believe this indicates the magnitude of the problem. The CDC is now reporting that Lyme disease is more widespread than earlier thought. We believe the problem is not only endemic but may also be reaching epidemic proportions. Early diagnosis is mandatory so that treatment can begin immediately to provide opportunity for cure and prevent chronic Lyme disease.

EXAMPLES OF MISDIAGNOSIS

The following stories of 4 individuals with diagnosis of ALS illustrate how important early diagnosis is.

THE FIRST is an individual with a 10-year diagnosis of ALS from whom we received a spinal fluid and blood specimen. The spinal fluid was highly positive for Bb, as was the blood. We reported the findings within a 24-hour period of receiving the specimens only to learn that the individual had died.

THE SECOND individual also had a long history of problems identified as ALS. His RIBb test was positive and he was not able to get any physician to treat him for Lyme disease. His health deteriorated and he was admitted to a hospital and was on life support. When his wife was told of his impending death she obtained a court order to have him treated with antibiotic therapy for Lyme disease. He recovered enough to get off life support and was subsequently discharged. He gained weight (32 pounds) and lived eight more months and then died of a heart attack.

THE THIRD individual is a 25-year-old golfer on a golf team. He became very ill and was unable to play golf. He was diagnosed with ALS. A family friend knew about our test and sent a blood specimen to be tested for Lyme disease. The results were positive. He was started on appropriate antibiotic therapy and was soon able to resume his golf career. He is now a professional golfer. Having an early diagnosis seems to have made the difference for this young man in living a productive active life.

THE FOURTH is a young college student who began having cognitive difficulties and had to drop out of school. He learned about our laboratory and was tested and found to be positive for Lyme disease. After four months on antibiotic he was able to resume his normal active life and is on the deans list and writing classical music.

These examples may shed some light on the importance of early diagnosis and appropriate treatment for Lyme disease. Left untreated the out-come of Lyme disease can result in a chronic debilitating condition and possible death. Are you sure you don't have Lyme disease? Use RIBb for life.

Dr. Jo Anne Whitaker is President and Director of Research at Bowen Research & Training Institute, Inc. 38541 US Highway 19 North, Palm Harbor, Florida, She can be reached at 727-937-9077 Email JoAnne@bowen.org Web: www.bowen.org

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