



Canadian Lyme Disease Foundation

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August 26th, 2007

The Honorable Jon S. Corzine Chairman
Health and Human Services Committee
National Governors Association
444 North Capitol Street, NW, Suite 267
Washington, DC 20001

Dear Governor Corzine,

This is further to the Infectious Disease Society of America (IDSA) president, Dr. Henry Masur's letter to you dated August 7th, 2007. [see <http://www.idsociety.org/newsArticle.aspx?id=4262>]

We do not normally involve ourselves outside of Canada on the political front, but the issue of Lyme disease is borderless so please indulge us for our imposition.

The IDSA guidelines have done more harm to people throughout North America than any other medical guidelines we are aware of. Because the infectious disease community in Canada are so closely tied with the IDSA, this Canadian group adopted the guidelines (Canada has done no research of it's own) and these guidelines have now become entrenched in our closed socialized system. This is one massive flaw in our medical system, it allows outside lobby groups such as the IDSA who for the most part represent business interests, to easily dominate our medical care. We've become too comfortable in avoiding the cost of doing research here in Canada by simply adopting research of others, no matter how poorly executed the research is.

We attach a copy of our letter to the IDSA medical journal in response to their published guidelines.

Until proper post mortem, and multiple live tissue studies per victim, employing all testing technology available is done on victims of those several diseases linked to *Borrelia burgdorferi*, the bacteria that causes Lyme disease, very little can be said about present testing accuracy, prevalence, what symptoms can be attributed to chronic Lyme disease, and what treatment methods are effective. This makes the entire IDSA document premature and not worthy of a health care guideline.

We are attaching copies of research abstracts showing just a fraction of the research that has been done relative to other diseases and Lyme. We are also attaching research showing transmission of this disease via a mother's placenta to her unborn child.

If the IDSA is that confident in their recommended testing protocol, how possibly could Lyme bacteria be showing up in the numbers it is in these other diseases (Alzheimer's, Multiple Sclerosis, Colitis, Crohn's disease and many many others).

Where along the line is it being missed?

Only now that victims' associations are becoming strong enough to fund the necessary research that government has been reluctant to fund in both our countries for over two decades will the evidence flow forth to even begin to look at creating guidelines.

2)

It is this reason that laws must be put in place, to tell insurers it is not okay to deny longer term treatment for Lyme disease, and to protect those doctors who are doing their very best to keep their patients' quality of life manageable. Premature guidelines are being pushed upon doctors who know they will only cause harm to their patients.

The IDSA has stated concerns about the dangers of antibiotics, and this is a good example of how poorly they support their position. Antibiotic harm pales in comparison to the heavy narcotics, anti-psychotics and other medications used to treat the many individual symptoms Lyme disease is responsible for. Some Lyme victims have to wean themselves off these dangerous drugs before they can start antibiotic therapy. Antibiotic resistance is a non-issue so long as we pump antibiotics into our food farm industry by the ton as we do now.

The IDSA boasts about the 400 references they cite in their guidelines as though it somehow gives evidence as to their expertise. The majority of these references are the author's themselves and their cohorts citing each other's work after carefully pruning out any contradictory research from the in excess of 20,000 research papers on Lyme disease from around the world.

Doctors who dare look at the entire global databank of research are quickly put in place by their medical professional licensing bodies. Professional witch-hunts occur using tactics that would not be allowed in a public court setting.

What never ceases to amaze us is how few people actually run the medical professional organizations who then also author guidelines, set testing criteria, and then police their members to enforce members do only what they tell them they can do.

We all need to take a hard look at how often the leaders of one organization are also in the leader group of another, and who then also sit on policing panels.

Why are the tens of thousands of good medical minds who do not own patents or represent medical insurance or other for profit interests not making the decisions?

Have a look at who sat on the Association of State and Territorial Public Health Laboratory Directors panel in 1994 that set the harmful two-tier testing protocol and then look at who authored or provided the research papers for the 2006 IDSA Lyme guidelines.

Why do this group of researchers control every aspect of the disease? It doesn't equate, other than for control. Why is it that so few people control so much in this field (health care) where even a hint of bias or conflict should be alarming (because life and death or somewhere horribly in between are at stake)? Who are they and what is their interest? Why are they allowed to dominate every aspect of a disease for so many years?

How did rules get put in place so this small group, relatively speaking, are able to operate almost with impunity and no public oversight? You will need to find out how many of these of people are also those who sit as reviewers to select which research gets the funding nod from federal and state taxpayer dollars through the many funding bodies. Only like-minded researchers get funded. We have the same problem in Canada.

How has this domination affected the quality and direction of research? Could it be that the medical financial crisis in North America is in part fabricated because of missed research opportunities? Why is so little common sense basic 'find the root cause' research not funded anymore? Post mortem and live tissue study are the foundation of medicine.

These are the same hard questions we are asking our government now.

We refer to the present system as one of 'farming sickness'. In some cases doctors are used as pawns in the farming process requiring legislation to offer them protection from a very powerful, well funded, and carefully positioned group of sickness farmers spread across several continents.

3)

In order for there to truly be experts in a particular disease, they must have studied all aspects of the illness. With there being such an apparent avoidance of 'no strings attached' research into how many of the millions of people (1.5 million in Canada alone) who are left with diagnoses of diseases with no known cause, may actually have Lyme disease, there are no true experts.

Until the IDSA can present verifiable research showing that Lyme bacteria plays no role in other disease processes that other research has indicated Lyme in fact does play a role in, how can they refer to themselves as experts? The IDSA, in regards to Lyme disease, are experts at creating algorithms backed by their own narrowly focused research creating data that no one else has validated except from within their own network of well-connected, well-funded cohorts. It should be no surprise that they want you ask them to provide 'experts'.

Please put legislation in place to help patients get the treatment those of us who suffer know very well works regardless of what a contrived document says. Protect the doctors, who treat their very sick patients, from witch-hunts.

After the protections and treatments are in place for the sick, and the doctors who treat them, go ahead and hold hearings and commissions to look into the entire matter of Lyme as the IDSA suggests. That process will go on for years if history is any indicator and those who have created the mess will have retired before any change comes about, which is their likely goal. Victims' lives are being destroyed now, so the legislation must be put in place now. If you do this, Canada will no doubt follow so therefore we have a very real and selfish interest in what unfolds.

Instead of spending millions on hearings as the IDSA suggests funding multiple Lyme disease research facilities to do the necessary post-mortem and live tissue study of all those diseases linked to Lyme (and the coinfections) may be a better use of money, especially if Lyme disease victims associations and 'no-strings attached' researchers are given a role in research and oversight. Again, that is a selfish wish but one that would be good for all.

We've been telephoned and emailed by many Americans asking if we have such a facility where they could donate their bodies or organs, or those of their loved ones. We do not yet but we are in the process and it will have the necessary oversight.

Rather than enclosing sensational media reports as did the IDSA, we enclose research abstracts indicating Lyme disease is linked to other serious, life altering diagnoses that are collectively bankrupting our various governments' health care coffers.

Thank-you for your time and we apologize for our uninvited input but this is such a serious matter for so many people now and in the future, we thought we must point out that the IDSA guidelines' harm is not limited to the United States.

Yours truly,

Jim M. Wilson AIIC
President, Canadian Lyme Disease Foundation

cc. Matt Salo, HHS Committee Director, NGA
Kathleen Nolan, Health Division Director, NGA Center for Best Practices

Letter to the Editor-Clinical Infectious Diseases

Dec. 15th, 2006 (published April, 2007)

Re: Infectious Disease Society of America; Lyme Disease Clinical Practice Guidelines

From: J.M.Wilson, President, Canadian Lyme Disease Foundation, 250-768-0978,
jimwilson@telus.net www.canlyme.com

The Canadian Lyme Disease Foundation has several concerns with the guidelines. I shall elaborate.

Erythema migrans rash

Throughout the document, the erythema migrans rash (EM) is referred to 108 times and is claimed to be the predominant diagnostic feature of LD. Headaches, fatigue, cognitive dysfunction, neuropsychiatric issues, myalgias, tremors, tics and parasthesias are given little or no attention, yet they can present in all stages of the illness; the EM rash, on the other hand, normally does not, and has been overemphasized as a predominant indicator of the disease [1]. As reported by Dr. S. Banerjee in 1995, for example, only 18% of confirmed cases reported a rash [2]. The research studies cited in the guidelines in support of percentages relative to rash incidence were not designed specifically to measure incidence of the EM rash therefore minimal value can be given to the data.

Symptomatic Chronic LD and Late Stage LD

The following quote from the guidelines is evidence of poor scholarship; “There is no convincing biologic evidence for the existence of symptomatic chronic *B. burgdorferi* infection among patients after receipt of recommended treatment regimens for LD.”. Strong evidence exists to the contrary in both animal and human model studies, and has for years [3].

Late stage LD is very poorly defined. Arthritic and neurologic manifestations are discussed, but are not well linked to the various symptoms that coexist with them. It is this array of variable symptoms, many times in the absence of arthritis or classic neurological manifestations, that collectively are so disabling in terms of quality of life for the patient and costly for governments in terms of disability payments. The guideline refers to many of these symptoms as “aches and pains of daily living” yet no research has been presented that has had the specific design of determining all the symptoms of late LD. Symptoms such as overwhelming fatigue, pain, muscle dysfunction, cognitive dysfunction, psychiatric issues, breathing restrictions, eyesight and hearing problems, bowel dysfunction and other manifestations that can be objectively measured, if proper measuring tools are employed, are common to late LD. Labelling these as “aches and pains of daily living” or as a “post-lyme disease syndrome” is a travesty at worst and premature at best. Many symptoms have been discounted. In the guidelines, entire classes of potential therapies are discounted because of this poor recognition of symptoms.

Seronegative Lyme Disease

In Grignolo, et al, 50% of PCR positive results of serum and cerebrospinal fluid samples corresponded to patients who were true positives at clinical examination but negatives at serologic tests. Over 60% of positive urine samples belonged to patients who had negative serologic results from analysis of serum [4]. In many other studies seronegative LD was proven [5].

In light of the above, and of the large global databank of LD research, until a definitive diagnostic test is found few conclusions can be drawn about late stage LD. The often referred to 'two-tiered testing' is not accurate enough to identify LD reliably [5,6]. In addition, until a comprehensive series of post-mortem studies of many similarly presenting diagnoses (e.g. Multiple Sclerosis, Alzheimer's, Chronic Fatigue Syndrome, etc.) is conducted to look for evidence of spirochaetal infection, few conclusions can be drawn about what objective symptoms are to be attributed to late LD.

The IDSA guidelines are far too exclusive, and thus inadequate, as a health care guide for physicians. The document represents a small cohort of authors largely supporting each others work. The guidelines do not meet the stated objective of the guidelines document itself.

The IDSA guidelines should be withdrawn and replaced with a document written in collaboration with victims groups and treating physicians.

1. Stricker RB, Laitin A, and Burrascano JJ. 2006. LD: The Quest for Magic Bullets. *Chemotherapy*. 52: 53-59.
2. Gill R., Banerjee S. and Banerjee M. 1995. LD Cases Acquired in British Columbia 1992-1994 canlyme.com/vanc1995.html
3. <http://www.lymeinfo.net/medical/LDPersist.pdf>
4. Grignolo MC, Buffrini L, Monteforte P, Rovetta G. 2001 Reliability of a polymerase chain reaction (PCR) technique in the diagnosis of Lyme borreliosis. *Minerva Med.* 2001 92: 29-33.
5. <http://www.lymeinfo.net/medical/LDSeronegativity.pdf>
6. Coulter P, Lema C, Flayhart D, Linhardt AS, Aucott JN, Auwaeter PG, Dumler JS. 2005. Two-Year Evaluation of *Borrelia burgdorferi* Culture and Supplemental Tests for Definitive Diagnosis of Lyme Disease. *J. Clin. Microbiol.* 43: 5080-5084.

Alzheimers Research - evidence of borrelial infection in an unknown percentage of Alzheimers brains. Research in this area has been grossly under-funded.

Neurobiol Aging. 2006 Feb;27(2):228-36

Beta-amyloid deposition and Alzheimer's type changes induced by *Borrelia* spirochetes.

[Miklossy J](#), [Kis A](#), [Radenovic A](#), [Miller L](#), [Forro L](#), [Martins R](#), [Reiss K](#), [Darbinian N](#), [Darekar P](#), [Mihaly L](#), [Khalili K](#).

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The pathological hallmarks of Alzheimer's disease (AD) consist of beta-amyloid plaques and neurofibrillary tangles in affected brain areas. The processes, which drive this host reaction are unknown. To determine whether an analogous host reaction to that occurring in AD could be induced by infectious agents, we exposed mammalian glial and neuronal cells in vitro to *Borrelia burgdorferi* spirochetes and to the inflammatory bacterial lipopolysaccharide (LPS). Morphological changes analogous to the amyloid deposits of AD brain were observed following 2-8 weeks of exposure to the spirochetes. Increased levels of beta-amyloid precursor protein (A β PP) and hyperphosphorylated tau were also detected by Western blots of extracts of cultured cells that had been treated with spirochetes or LPS. These observations indicate that, by exposure to bacteria or to their toxic products, host responses similar in nature to those observed in AD may be induced.

[J Alzheimers Dis](#). 2004 Dec;6(6):639-49; discussion 673-81

***Borrelia burgdorferi* persists in the brain in chronic lyme neuroborreliosis and may be associated with Alzheimer disease.**

[Miklossy J](#), [Khalili K](#), [Gern L](#), [Ericson RL](#), [Darekar P](#), [Bolle L](#), [Hurlimann J](#), [Paster BJ](#).

University Institute of Pathology, Division of Neuropathology, University Medical School (CHUV), 1011, Lausanne, Switzerland. judmik@telus.net

The cause, or causes, of the vast majority of Alzheimer's disease cases are unknown. A number of contributing factors have been postulated, including infection. It has long been known that the spirochete *Treponema pallidum*, which is the infective agent for syphilis, can in its late stages cause dementia, chronic inflammation, cortical atrophy and amyloid deposition. Spirochetes of unidentified types and strains have previously been observed in the blood, CSF and brain of 14 AD patients tested and absent in 13 controls. In three of these AD cases spirochetes were grown in a medium selective for *Borrelia burgdorferi*. In the present study, the phylogenetic analysis of these spirochetes was made. Positive identification of the agent as *Borrelia burgdorferi* sensu stricto was based on genetic and molecular analyses. *Borrelia* antigens and genes were co-localized

with beta-amyloid deposits in these AD cases. The data indicate that *Borrelia burgdorferi* may persist in the brain and be associated with amyloid plaques in AD. They suggest that these spirochetes, perhaps in an analogous fashion to *Treponema pallidum*, may contribute to dementia, cortical atrophy and amyloid deposition. Further in vitro and in vivo studies may bring more insight into the potential role of spirochetes in AD.

PMID: 15665404 [PubMed - indexed for MEDLINE]

Med Hy 2007;68(5):1059-64. Epub 2006 Nov 17

Alzheimer's disease Braak Stage progressions: reexamined and redefined as *Borrelia* infection transmission through neural circuits.

MacDonald AB.

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Brain structure in health is a dynamic energized equation incorporating chemistry, neuronal structure, and circuitry components. The chemistry "piece" is represented by multiple neurotransmitters such as Acetylcholine, Serotonin, and Dopamine. The neuronal structure "piece" incorporates synapses and their connections. And finally circuits of neurons establish "architectural blueprints" of anatomic wiring diagrams of the higher order of brain neuron organizations. In Alzheimer's disease, there are progressive losses in all of these components. Brain structure crumbles. The deterioration in Alzheimer's is ordered, reproducible, and stepwise. Drs. Braak and Braak have described stages in the Alzheimer disease continuum. "Progressions" through Braak Stages benchmark "Regressions" in Cognitive function. Under the microscope, the Stages of Braak commence in brain regions near to the hippocampus, and over time, like a tsunami wave of destruction, overturn healthy brain regions, with neurofibrillary tangle damaged neurons "marching" through the temporal lobe, neocortex and occipital cortex. In effect the destruction ascends from the limbic regions to progressively destroy the higher brain centers. Rabies infection also "begins low and finishes high" in its wave of destruction of brain tissue. Herpes Zoster infections offer the paradigm of clinical latency of infection inside of nerves before the "marching commences". Varicella Zoster virus enters neurons in the pediatric years. Dormant virus remains inside the neurons for 50-80 years, tissue damage late in life (shingles) demonstrates the "march of the infection" down neural pathways (dermatomes) as linear areas of painful blisters loaded with virus from a childhood infection. Amalgamation of Zoster with Rabies models produces a hybrid model to explain all of the Braak Stages of Alzheimer's disease under a new paradigm, namely "Alzheimer's neuroborreliosis" in which latent *Borrelia* infections ascend neural circuits through the hippocampus to the higher brain centers, creating a trail of neurofibrillary tangle injured neurons in neural circuits of cholinergic neurons by transsynaptic transmission of infection from nerve to nerve.

Multiple Sclerosis – How many diagnoses of MS may in fact be Lyme? Not all, for sure, but how many...no one knows to date because this area of research is grossly under-funded.

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<http://infection.thelancet.com/>

Commentary

Bacterial infection as a cause of multiple sclerosis

Multiple sclerosis is an inflammatory demyelinating disease in which the immune system of genetically susceptible individuals is inexplicably activated to attack the central nervous system.

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Multiple sclerosis is an inflammatory demyelinating disease in which the immune system of genetically susceptible individuals is inexplicably activated to attack the central nervous system. Epidemiological studies strongly suggest that environmental factors are involved on a background of genetic susceptibility. (1) The possible involvement of infectious pathogens, most often viruses, has been much studied. (2,3)

Multiple sclerosis has a unique geographic distribution--temperate zones have a low prevalence and more northerly areas have a prevalence more than ten times that in warmer climates. (4) Sanitation, climate, ultraviolet radiation, hours of sunshine, socioeconomic status, and other environmental factors have been examined with little success. (1) Much early research used case-control designs with potential recall bias. (5) More recently, seroepidemiological research has suggested the involvement of infectious pathogens in multiple sclerosis: specific antibody responses in cerebrospinal fluid and blood, isolation of the pathogen from tissue of patients with multiple sclerosis, or in-situ or ex-vivo pathogen detection. The results have rarely been harmonious. Laboratory markers cannot be easily studied at the population level because infection by some agents (eg, with human herpesvirus 6 or *Chlamydia pneumoniae*) does not result in identifiable clinical disease or

infection occurs in childhood and is not reliably reported by study subjects.

The convergence of epidemiology and seroepidemiology of research, however, is seen with Epstein-Barr virus. (6,7) Data from the Nurses' Health study, (8) for example, show a moderately increased risk of multiple sclerosis in nurses with a history of infectious mononucleosis (odds ratio 2.1, 95% CI 1.5-2.9). Taking only those nurses whose report of infectious mononucleosis was confirmed by a positive heterophil-antibody-test, the risk remained (2.3, 1.6-3.5). Although there was no association found between multiple sclerosis and reports of other common viral diseases before disease onset, there was an association with mumps after 15 years of age and with late age at measles infection. Whether Epstein-Barr virus is a necessary cause requiring additional triggers to produce disease or merely a marker for a true cause is unresolved. (9)

Infection with *Borrelia burgdorferi*, the spirochaete responsible for Lyme disease, can involve the central nervous system and the later stages of the disease may mimic the clinical symptoms of multiple sclerosis. (10) Seroepidemiological studies of *B burgdorferi* and multiple sclerosis have produced conflicting results. Chmielewska-Badora and colleagues (11) reported that ten of 26 (38%) patients with multiple sclerosis were seropositive for *B. burgdorferi* compared with 149 of 743 (20%) patients with other neurological disorders ($p=0.042$). Yet others reported negative findings. (12,13) More recently, O Brorson and colleagues (14) studied the presence of the infectious agent, or at least its cystic structure, in the cerebrospinal fluid of ten patients with multiple sclerosis, in five controls who had lower back pain, and in one patient infected with *B burgdorferi*. Cystic structures were found in eight of the ten with multiple sclerosis with use of immunofluorescence before culture and in all the multiple sclerosis patients by transmission electron microscopy and acridine-orange staining. No cystic structures were found in the controls with any method. The investigators also reported a positive reaction to antispirochaetal antiserum, a similarity between the cystic structures with known cystic forms of spirochaetes, and the similarity between the cysts found in the multiple sclerosis patients and the patient with *B burgdorferi* infection. These results led the team to suggest that the multiple sclerosis patients were infected with a spirochaete, most likely *B burgdorferi*. Whether this infection really was *B burgdorferi* and whether it occurred before or after the onset of multiple sclerosis cannot be determined from this study and indeed, given current methodology, it is difficult to imagine how this could be determined.

Whether infection with *B burgdorferi* is a cause of multiple sclerosis or whether it is merely a result of heightened susceptibility of multiple sclerosis patients to infection due to damage to the blood-brain barrier remains one of the enigmas of multiple sclerosis research. Indeed, this caveat applies to all infectious pathogens that have been associated with multiple

sclerosis. Current thinking on how infections could trigger the autoimmune/immunopathological manifestations of multiple sclerosis target the following mechanisms: molecular mimicry between the pathogen and myelin antigens, determinant spreading after injury to the central nervous system by the pathogen, and bystander inflammation caused by central nervous system infection. (3) It needs to be explained how a ubiquitous infection, such as that with Epstein-Barr virus, could be involved in the pathogenesis of multiple sclerosis. Moreover, several pathogens could be associated with multiple sclerosis and their presence in the central nervous system may not be a necessary requirement for disease initiation or perpetuation.

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(3) Talbot PJ, Arnold D, Antel JP. Virus-induced autoimmune reactions in the CNS. *Curr Top Microbiol Immunol* 2001; 253: 247-71.

(4) Rosati G. The prevalence of multiple sclerosis in the world: an update. *Neurol Sci* 2001; 22: 117-39.

(5) Wolfson C, Granieri E, Lauer K. Case-control studies in multiple sclerosis. *Neurology* 1997; 49 (suppl 2): S5-S14.

(6) Ascherio A, Munch M. Epstein-Barr virus and multiple sclerosis. *Epidemiology* 2000; 11: 220-24.

(7) Marrie R, Wolfson C. Multiple sclerosis and Epstein-Barr virus. *Can J Infect Dis* 2002; 13: 111-18.

(8) Hernan MA, Zhang SM, Lipworth L, Olek MJ, Ascherio A. Multiple sclerosis and age at infection with common viruses. *Epidemiology* 2001; 12: 301-06.

(9) Wolfson C. Multiple sclerosis and antecedent infections. *Epidemiology* 2001; 12: 298-99.

(10) Karussis D, Weiner HL, Abramsky O. Multiple sclerosis vs Lyme disease: a case presentation to a discussant and a review of the literature. *Mult Scler* 1999; 5: 395-402.

(11) Chmielewska-Badora J, Cisak E, Dutkiewicz J. Lyme borreliosis and multiple sclerosis: any connection? A seroepidemic study. *Ann Agric Environ Med* 2000; 7: 141-43.

(12) Coyle PK. *Borrelia burgdorferi* antibodies in multiple sclerosis patients. *Neurology* 1989; 39: 760-61.

(13) Schmutzhard E, Pohl P, Stanek G. *Borrelia burgdorferi* antibodies in patients with relapsing/remitting form and chronic progressive form of multiple sclerosis. *J Neurol Neurosurg Psychiatry* 1988; 51: 1215-18.

(14) Brorson O, Brorson S-H, Henriksen T-H, Skogen PR, Schoyen R. Association between multiple sclerosis and cystic structures in cerebrospinal fluid. *Infection* 2001; 29: 315-19.

* Christina Wolfson, Pierre Talbot

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Crohn's Disease and Colitis ... Clear evidence of borrelia infection but in what percentage of cases? Under-funding of this research will leave the numbers an unknown.

Dr. Martin Freid recently presented evidence of infection with the Lyme germ in patients with Crohn's disease, an inflammation of the bowel which affects some 50,000 Canadians, and whose precise cause has eluded medical researchers.

Dr. Gordon Greenberg, Professor of Medicine at the University of Toronto and head of the Division of Gastroenterology at Mount Sinai, says it's not clear exactly what causes either Crohn's disease, or another inflammatory bowel disease called ulcerative colitis. He notes, "There has always been the concept that a single infectious etiology might be the cause of Crohn's or ulcerative colitis, but to date no single bacterium or virus has been linked with either disease. What is clear, however, is that bacterial flora within the gut, at least in a secondary way, perpetuate the inflammatory process in Crohn's."

Greenberg cites several lines of evidence, including studies from his own center, on the effect of specific antibiotics, which he's found to be particularly effective in helping to control the inflammation of Crohn's disease. His initial data suggest improvement or remission in up to 63% of Crohn's patients treated with antibiotics. "More and more the concept is emerging that bacteria do play an important role, and that selected antibiotics are quite helpful in the management of patients with Crohn's disease," Greenberg notes.

Dr. Freid recently saw an 8-year-old girl with blood in her stool, a typical symptom of ulcerative colitis. He prescribed medicine to calm the inflammation, but he also sent a

tissue biopsy off for analysis. Surprisingly, it revealed an active Lyme infection. He put the girl on antibiotics for a month, and she made a complete recovery. "That's not the nature of ulcerative colitis, which would come back. But an infection would go away if treated properly. I thought it was fascinating."

Neither Dr. Fried nor Dr. Greenberg is sure just what's going on in their patients, but the evidence certainly points to a role for bacteria. This uncertainty over causation extends as well to mysterious problems like chronic fatigue and fibromyalgia. Some think the Lyme bug may be to blame for a lot of cases, others suspect another organism called a mycoplasma - it's going to be a while until we know for sure.

Mother passes Lyme to fetus ...

[Ann Intern Med.](#) 1985 Jul;103(1):67-8.

Lyme cultured from kidney, spleen and liver of newborn fetus who did not survive.

Full text can be seen at http://www.canlyme.com/Schlesinger_1985.pdf

Arthritis Rheum 1987; Volume 30, Number 4, 3(Suppl):S50.

Death 8-Day Old Californian Baby Boy

Culture positive seronegative transplacental Lyme borreliosis infant mortality.

Lavoie PE;Lattner BP;Duray PH; Barbour AG; Johnson HC.

"Transplacental infection by *Borrelia burgdorferi* (Bb), the agent of Lyme Borreliosis (LB), has recently been documented (L.E. Markowitz, et al; P.A. Schlesinger, et al). Fetal infection confirmed by culture has been reported by A.B. MacDonald (in press) from a highly endemic region (Long Island, NY).

We report a culture positive neonatal death occurring in California, a low endemic region. The boy was born by C-section because of fetal distress. He initially appeared normal. He was readmitted at age 8 days with profound lethargy leading to unresponsiveness. Marked peripheral cyanosis, systemic hypertension, metabolic acidosis, myocardial dysfunction, & abdominal aortic thrombosis were found. Death ensued. Bb was grown from a frontal cerebral cortex inoculation. The spirochete appeared similar to the original Long Island tick isolate. Silver stain of brain & heart was confirmatory of tissue infection.

The infant was the second born to a California native. The 20 m/o sibling was well. The mother had been having migratory arthralgias and malaise since experiencing horse fly & mosquito bites while camping on the Maine coast in 1971. The family was seronegative for LB by ELISA at Yale. Cardiolipin antibodies were also not found."