Lyme Disease and Autism A New Paradigm

by Kathy Blanco with Geoffrey P. Radoff, MD, MD (H)

Commentary on the Proceedings of the LIA Foundation Think Tank San Diego, 2007

In January 2007, this article's co-author, Dr. Geoffrey Radoff, was invited to a physicians' think tank, sponsored by the Lyme Induced Autism (LIA) Foundation. The purpose of the event was to explore the connection between Lyme and autism and discuss the best approaches to identify and treat autistic children infected with the spirochete *Borrelia burgdorferi*. Dr. Radoff has been to many medical conferences and has personally contacted physicians extremely knowledgeable about the autistic spectrum. At the think tank conference, he found that everyone was keenly interested in the possibility of Borrelia involvement in the disease and wanted to expand their treatment options. Event organizers and attendees hope that the information put together in San Diego will be helpful to health care providers and that Borrelia will be considered in the diagnosis and treatment plan.

According to Dr. Radoff, those experienced in the autism spectrum know that there are many autisms, not a single autism. There has been no gene identified that causes autism. There are immunologic weaknesses, chronic infections, vitamin and mineral deficiencies, and biochemical defects that contribute to the disease. There are environmental toxins contributing to the dysfunction of the immune system and subsequent increases in certain diseases.

Autism is increasing in California, a state that keeps very good statistics. At the University of California at San Diego, David Kirby, author of *Evidence of Harm*,[1] made this statistic available. An excellent discussion and debate about the widespread implications of mercury and vaccinations on autism is available on the Autism Research Institute (ARI) website (www.autismresearchinstitute.com). Other states are finding increasing numbers of autism diagnoses, including the states on the West and East coasts, all are known to have endemic Lyme Borrelia. In the state of Oregon, it is estimated that one in 98 children are affected, the worst in the country. And, even as this article is going to press, a new study released by the Centers for Disease Control (February 7, 2007) reported that autism is now thought to affect one in 150 American children.

Many characteristics of Lyme disease and autism are shared by the neurological system. These include white matter and gray matter involvement, antibodies to myelin basic protein, hypo perfusion (blood flow restriction in the brain), thyroid antibodies, paralysis of the gut (many presentations, megacolon, encopresis, diarrhea, constipation, pain), hyperacusis (sound sensitivity), light sensitivity, touch sensitivity, sleep problems involving the melatonin cascade, including serotonin uptake, violent behavior outbursts, seizures, rapid mood swings, obsessive compulsive disorder, visual spatial involvement, slow processing and word retrieval, cognition loss, memory impairment, brain fog, dyslexia and word-finding problems, stuttering, bladder dyscontrol, and depression.[2] Other known signs are low muscle tone at birth and higher incidences of birth defects. Included in this category are the known *in utero* infections that can cause autism, including syphilis, which is in the Borrelia family. It has also been surmised that seasonal births and autism have connections, which would confirm the infectious agent responsible for autism, encompassing all infections during pregnancy, including a Lyme bacterial load in mother or father and other viral co-infections.

According to Dr. Radoff, patients infected with *Borrelia burgdorferi* have many other infections much like a complex of infections known to occur in AIDS, Babesia, Mycoplasma Pneumonia, HSV I (one) and VI (six), cytomegalic virus, bartonella, Chlamydia P, EBV, and Ehrlichia. It could be said that autism is a Borreliosis complex or that autism has a complex of infections.[3] This is well-proven when you test children for these bacterial and viral co-infections. These children are greatly immunosuppressed and have numerous metabolic dysfunctions, including low glutathione, known to

be caused by Borrelia infections and increased Reactive Oxygen Species (ROS).

How these infections cause autism is conjecture. However, no human studies have proven that Borrelia cannot pass through the placenta or through breast milk, although veterinarian models have illustrated this connection. We know that the neurological system can be damaged by an autoimmune reaction, which deranges the biochemistry of the body. Physical and long-lasting damage to the brain/body systems/organs is possible in the Borreliosis complex if not addressed. There are other plausible theories relating to biochemistry and immunology. These can be measured on lab tests, such as chronic low CD 57, Low IgG subclass four, low C4B complement proteins (tag and lyse viruses, bacteria and fungi), HLA-DR4 (immune haplotype that sees typically chronic Lyme), and are very close to Common Variable Immune Deficiencies.[4] Neurological Borrelia complex would lower CD 57 natural killer cells, especially the neurological presentation of Lyme, excluding the musculoskeletal presentations. This is illustrated by the fact that neurological Borrelia complex is a very specific marker that generally does not cross over another disease state, even AIDS. Typically, children with autism have low CD 57. There are also signs of lowered NKT cells in autism, a known borrelial stratagem for survival

Other factors can be found on radiological exams via MRI, Neurospect, and other skilled highly powered radiological exams. One was reported in a radiological journal in the seventies, which stated that children with large sulci widths often have infantile autism.[5] An interesting note is that this condition is sometimes the result of an encephalitis course and an initial event of immune-regulated inflammation resulting from a major infection in the brain, which could not be mounted up against or handled correctly, sometimes to the sub-clinical level. This infection enhances the dropping of signals, if you will, to other parts of the brain, making the brain unable to communicate its needs from one hemisphere to the other. Known as mirror neuron theory, this is a recognized problem in autism.

In an examination of the older autistic, the Harvard Brain Tissue Program looked at the brains of deceased adult autistics and found an unusually high amount of brain atrophy and cerebellum shrinkage, pons atrophy, and superior and inferior olive atrophy. This discovery was explained as the result of the subjects having undergone years of incorrect or nonexistent neurological stimulation. We believes it is more than that. These two valuable observations were found in co-author Kathy Blanco's autistic son at ages nine and 25, respectively, on MRI imaging; he has neuroborreliosis complex.

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Below, we've excerpted an alarming report by Mark Grier.[6] Lyme is perfectly capable of doing major damage to the brain, as the Grier article states, in sort of a delayed fashion, if you will. And you will note that the author states that the infection lies at the blood brain barrier (BBB) and is the gatekeeper to let in other infections. This is curious in autism, in that, for such children with Borrelia infection, other infections would be allowed into the brain, including vaccine viruses, a major hot button in autism causes. It is possible that the two are conjoined in damage and that the long-term effects of congenital Borrelia complex could hamper the body's ability to mount a significant, timely, and typical response to vaccine viruses. This would explain the higher incidences of reactions to vaccinations in children with autism. In his article, Mark Grier states the following:

> Other neurogenic strains of Borrelia that cause Relapsing Fevers in Africa can be deadly within mere weeks of entering the brain. While Lyme disease may be a bit more subtle upon penetrating the brain, its silent and insidious invasion may be the reason that brain involvement can and is often be overlooked by physicians for months or even years in neurological Lyme patients.
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> Just as the spirochete that causes syphilis can remain active within the brain for decades in tertiary Syphilis, there is equal cause for concern that the Lyme bacteria can also become a sequestered unwelcome interloper to the central nervous system. How does the Lyme bacteria enter the brain when the blood brain barrier fights against foreign invasion?
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> Once the Lyme spirochete enters the peripheral blood circulation through an infected tick bite, the bacteria fights to escape the confines of the blood vessel walls. The spirochete's motility allows it to swim in the blood stream until it lodges a wiggling tip into a endothelial cell junction, where it bores between endothelial cells lining the capillaries and causes a specific inflammation and irritation that causes the endothelial cells to release digestive proteins that create holes within the capillary bed.
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> In 1989, experiments were done using umbilical cord vein to show that *Borrelia burgdorferi* attaches tip first to endothelial cells and microscopic examination found holes near the areas of attachment. If these vessels were within the heart or brain, it would be clear that there is nothing to stop Borrelia and other blood stream components from entering those sites. In the case of the brain, allowing bacteria and white blood cells access to the brain is setting up the brain for a series of events to occur which we can call neurocascade events. This is where one event will trigger another, which in turn will trigger another and so on until a small

nonsymptomatic event becomes noticeable.

To see if *Borrelia burgdorferi* truly breaks down the blood brain barrier, several animal experiments were done. Since blood albumin protein should not be in the cerebral spinal fluid (CSF), researchers tagged normal albumin with radioactive Iodine. In mice, hamsters, and dogs without infection the radioactive Iodine never penetrated into the CSF of the normal control animals. But when the animals were infected with Lyme disease, within mere hours the blood brain barrier became permeable and radioactive iodine was found within the CSF of infected animals usually for about two weeks following initial infection. This window of permeability certainly gives ample time for the Lyme bacteria to establish itself within the brain. Detailed collection of CSF from recently bitten Lyme patients reveals that sub clinical infections of the CNS occurs often before the infection in the body is even detected.

Before we talk about the neurotoxic effects of *Borrelia burgdorferi* within the brain, let's first look at some other neurocascade events that occur in acute brain trauma and repeated brain trauma such as in sports accidents like soccer, football and boxing. Repeated concussions in football players and boxers can cause a slow onset of a syndrome sometimes called Pugilistic Dementia, or Sports-Related Encephalopathy. Common symptoms begin months to years after injury occurs and usually includes: headaches, muscle twitches, tics, sensitivity to bright lights and loud noises, inability to retrieve words, loss of time, depression, suicidal thoughts. Later these symptoms can progress to fatigue, lethargy, loss of interest, severe depression, Parkinson-like tremors, loss of motor control, and overall slowness, and finally dementia.

An interesting comparison between Lyme encephalitis and Pugilistic dementia besides sharing similar symptoms is that both patients can have global-cerebral-atrophy years after their initial trauma. In other words, the brain shrinks, and appears to have lost the ability to properly repair damage that accumulates over many years. These abnormal MRIs of the brains of boxers and Lyme patients may be caused by different mechanisms, but the end result can appear similar in both the symptoms and pathology of the two conditions.

Dr. Radoff believes the journal article that best explains why we need to change our old-fashioned view of Lyme is "Lyme Disease: An Ancient Engine of an Unrecognized Pandemic."[7] In that article, Dr. Harvey skillfully steers away from the established sequence of the tick bite-only etiology of Lyme. Dr. Harvey states that when he tested chronically ill patients with multi-system presentation of Borrelia, he obtained unexpected results because his part of Texas was not an endemic region of Lyme disease. About one-third of his initial tests were positive using CDC Western blot criteria or serum/urine PCR. No patient had the typical skin rash, erythema migrans, and most had been ill for many years with similarly ill family members. Dr. Harvey's cases did not match the Centers for Disease Control (CDC) case definition or the epidemiological evidence for late Lyme disease. Dr. Harvey decided to re-think the CDC position.

This conclusion is most curious to a parent of an autistic child, since it implies that it's quite possible that parents do no know they or their children have this infection, and therefore the imprisoned thought that Lyme is only caused by a tick bite with a self-limiting rash cured by antibiotics within thirty days cannot be relied upon. Let's get the "tick bite" out of our heads, once and for all. We are finding that multiple family members with different presentations of Borreliosis complex are found within the family. For example, members of Kathy Blanco's own family have connective tissue disorders, heart valve involvement, neurological neuroborreliosis involvement, hormonal involvement, gastrointestinal Lyme, and mitochondrial involvement of Borrelia – again, all within one family.

"Lyme disease" is the label given to human illness first recognized in Old Lyme, Connecticut in 1975. The initial cased resulted from zoonosis (tick biting the patient and infecting the host with *Borrelia bugdorferi*). As the knowledge of Lyme disease expanded, the zoonosis-only conception of Lyme needed to be changed. The exclusive focus on zoonosis resulted in a major portion of resources being concentrated on reservoir and vector prevalence.[8] The CDC defines Lyme disease exclusively as a zoonosis illness. Congenital and gestational transfer cases have been disregarded for reasons not evident to Dr. Harvey or the authors of this article.

"I agree with Dr. Harvey," says Dr. Radoff. "There are two forms of *B. burgdorferi* infection: "Lyme disease" and epidemic Borreliosis (disease spread directly between humans). Dr. Harvey proposes a worldwide, non-Lyme pool of *B. burgdorferi* humans with a clinical presentation of extraordinary variability, global geographic distribution, and far greater prevalence. Transfer is intra-human (congenital and almost certainly sexual) and is silent or unrecognized."

Curiously, many mothers of autistic children are hampered by infections that could cause autism in their children. It is quite possible that the prevalence of autoimmune disorders found in families with autism is an infection that has existed chronically in the body for years, if not decades, much like syphilis.[5] Sweeten et al. has proposed a higher propensity to autoimmune disorders in mothers of autistic children, such as Hashimotos Thyroiditis (a known Babesia resultant infection), rheumatioid arthritis (viral and bacterial), rheumatic fevers (infection based viral or bacterial), and other less well-known condition such as vitiligo, depression, fibromyalgia, Chronic Fatigue Syndrome (CFS), Attention Deficit Disorder (ADD) (known to also be caused by autoimmune processes), etc.[5]

"Testing is not as good as we would like," says Dr. Radoff. "There

are specialty labs that treat for Borrelia with ways different than the traditional Western Blot. I suggest you use one of these labs and not the commercial laboratories except perhaps for a screening. If it is positive, one need not go on. A treatment protocol is an article unto itself. I leave that search to the reader. I end on an observation relayed to me by more than one mother: 'When my child was on amoxicillin for ten days, he wasn't autistic.' Could it be that they were treating Borrelia?"

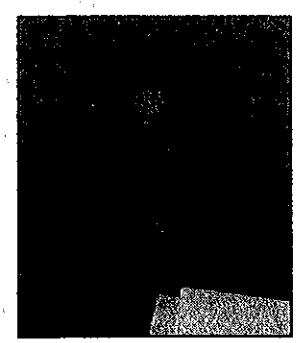
On the LIA Foundation list-serv (lyme-autism@yahoogroups.com), a high resultant infection rate of Borrelia in autism has been reported in whole families, and, concurrently, therapies that are effective against this pathogen are relieving symptoms of autism and, in some cases, children are recovering from autism, due to the pathogen's multi-systemic nature of infection, especially its affinity to the brain/gut. This is not an easy course. Children with Borrelia and autism have many detox pathway problems and other infections in the mix, if not harsh clinical presentations of neuroborrelisosis, including seizures, considered the late stage of Borrelia. Herxing is hard, sometimes unbearable. However, the earlier the pathogen is caught, the better chance a newly diagnosed autistic child will recover from this devastating illness. This is why this an important new paradigm is so important, because, if left to its devices, Borreliosis complex can ruin lives and families throughout lifetimes and generations. Borrelia is known to be one of the sneakiest, hardest, and quickest-to-survive organisms known to man. It can evade the immune system in 100 different ways and change its outer surface proteins to hide and sequester away from most of our sophisticated antibiotic regimes.

Apropos of Dr. Radoff's comments, we have seen cases of children on long-term antibiotics and related therapies who are coming out of autism, even for short periods of times. For instance, in one case, we saw a child who had a surgery with IV antibiotics in use and, for five days, was not acting autistic! Other reports exist, such as that reported in the DAN! conference (2003) with the use of Vancomycin, a known toxic pharmaceutical antibiotic against Borrelia. The children in that case were placed on the regime to effectively kill aerobic growth bacteria; Borrelia is one of that bacteria group's family members. Within three months, children were responding favorably and losing autism symptoms. Unfortunately, after the study, when removed from treatment, the child regressed back to the original state and often worsened. This possibly occurs because the drug was not used long enough to kill all the populations and life cycles of Borrelia that are known to repopulate for at least a year into other forms such as cysts and L forms – with the mutated version being worse than the original. Such would be the case with other antibiotics as well; this goes against IDSA guidelines which recommends a 30-day treatment as the best way to treat Lyme. We are talking about a complex infection that requires chronic and symptom-dependent therapy continuation, until all markers of infection are either not present or are silenced deeply.

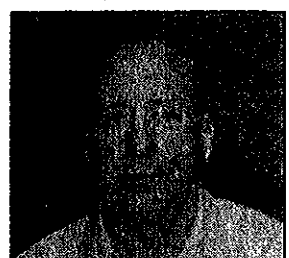
A consensus group such as LIA Foundation would help this population understand what needs to be done in these children and conceptualize the diagnosis and treatment of a very interesting infection, heretofore unrecognized in autistic children and families. Borrelia is not always just an acute infection; it can be a chronic, non-self-limiting disease and, unfortunately, a disease that mimics 300 disease states and many, if not all, the psychiatric diagnoses in our latest diagnostic manuals.

Notes

1. Kirby D. *Evidence of Harm: Mercury in Vaccines and the Autism Epidemic: A Medical Controversy.* New York: St. Martin's Press; 2005.
2. Hajek, et al. ILADS meeting 2005 consensus of psychiatric signs of Borrelia complex. *Am J Psychiatry.* 2002; 159;297-301.
3. Nicholson G. Gan R, Nicholson N, Haeir J. Evidence for mycoplasma ssp; chylamidia P and human herpes 6 virus, co infections in the blood of patients with autistic spectrum disorders. The Institute for Molecular Medicine, Huntington Beach, California; Department of Surgery, University Hospital, Munster, Germany.
4. Stricker RB, Burrascano J, Winger E. Long-term decrease in CD57 lymphocyte subset in a patient with chronic Lyme. *Ann Agric Environ Med.* 2002;9(1):111-3.
5. Sweeten TL, et al. Increased prevalence of familial autoimmunity in probands with pervasive developmental disorders. *Pediatrics.* November 2003; 112 (5):e420.
6. Grier M. The complexities of Lyme disease. Available at: http://www.canlyme.com/tom.html. Accessed February 10, 2007.
7. Harvey W'I', Salvato P. Lyme disease: An ancient engine of an unrecognized pandemic. *Medical Hypothesis.* 2003.
8. Barbour A.G., Fish D. The biological and social phenomenon of Lyme disease. *Science.* 1993.



Kathy Blanco is the mother of four children, two of whom are autistic. She has been featured in *Treating Autism and Recovering Autism* (Rimland B, Edelson S. Autism Research Institute; 2003); and her articles have been included in *The Autism Experience: Stories of Hope and Love* (Simmons K, Hoke M. Booksurge Publishing; 2003). She was the Health Chairman of Voices of Safety (VOSI) [wwww.voicesofsafety.com] and is co-founder of the Lyme Induced Autism (LIA) Foundation, which seeks to find the infections in autism, especially the Borrelia complex.



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LIA Foundation is a 501c3 non-profit public charity focusing on the link between Borrelia, multiple-infections and Autism Spectrum Disorders. To make a donation or for more information, please visit www.liafoundation.org.