



Canadian Lyme Disease Foundation

2495 Reece Rd., Westbank, B.C. V4T 1N1

www.canlyme.org

Ph. 250-768-0978

CLD Fdn.

Fax 250-768-0946

AUTISM-A TYPE OF LYME DISEASE

Medical Hypothesis

Kathy Blanco

December 15, 2004

Copywrite 2004

Autism is growing at epidemic rates. (1) It is reported to be prevalent in 1-150 children, some states 1-80 children. Autism is a complex neurological disorder with seemingly provable and many etiologies such as maternal viruses and bacteria (2), immediate cord clamping and birth drugs (3), metabolic insufficiency during development in utero or infancy (ie, thyroid t3 and t4) (4), purine disorders, adenosine deaminase deficiency (5), mitochondrial disorders (6), iron fragile metabolism (7), metallothionein deficiency (8), oxidative stress, childhood vaccinations which act as triggers to events above through viral persistence in gut and brain (9), and heavy metal poisoning from additives in vaccines (10), and lastly chronic disseminated Lyme neuroborreliosis disease. Lyme disease is transmitted through a tick bite. But it can also be transmitted through semen, breast milk and gestational fluids. This means that a fetus can be infected by its mother. *B. burgdorferi* has been proven by PCR analysis to establish a persistent infection in the mammalian host (Straubinger, R. Persistence of *B. burgdorferi* in experimentally infected dogs after antibiotic treatment. *J.Clin.Microbiol.* 1997 Jan; 35(1): 111-116). Lyme disease is the fastest growing vector-borne disease in the nation. In 2001, the Center for Disease Control recorded 18,000 new cases, but some experts estimate that the actual number is closer to 200,000. This is four times the number of HIV cases per year. Autism grows as exponentially as that, and an NIH bulletin explained of late it is seen in 1/166 children. Amongst those statistics, is the further frightening 1-6 children now have a developmental, psychiatric problems in the US.

Lyme disease results in underconnectivity of brain areas, defects of the fusiform gyrus and loss of Purkinje cells in the cerebellum. Recently reports of white matter disease are prevalent in children and adults with autism paralleling patterns in Lyme disease. (11) Late in the progression of this disease neurological, cognitive, and psychiatric symptoms predominate, overlapping symptoms of autism, such as food avoidance, facial recognition problems, sleep disorders, ocular symptoms such as light sensitivity, speech and language loss or word retrieval problems, noise sensitivity, bed wetting,

aggression, panic attacks, headaches, movement disorders, sore throats and poor swallowing, hypofusion-poor blood flow particularly to temporal lobes, swollen tongues and lymphs, chronic fatigue symptoms, hyperactivity, gut problems (ie, diarrhea, constipation, IBD symptoms, Celiac symptoms), liver dysfunction, thyroid problems (t3 and t4 conversions), iron disorders, heavy metal toxicity, tics, food sensitivities, depression, serotonin uptake problems, autoimmune brain antibodies, problems with sequencing, problems with sensory perceptions, seizures, cardiac conduction abnormalities, illiod hyperplasia, heavy metal toxicity, co infections of HHV6 and Mycoplasma (12), various aches and pains masked as lyme arthritis particularly in the neck area, and zinc copper iron imbalances.

The basic hierarchy is pre-frontal cortex, para limbic association areas, limbic structures, and brain stems - hypothalamus. Lyme encephalopathy can result in dysfunction of the modulation centers, inhibitory pathways, and stimulatory pathways. (13) Autopsies, animal studies, and brain imaging tests have contributed to this understanding. The presenting symptoms of NPLD are sometimes emotional in nature, and include obsessive-compulsive disorder, depression, and aggression, panic disorder, and other phobic disorders. These self same observations are also seen in autism as reports from parents of these symptoms are typical.

All involved with late state Lyme disease agree there is a large amount of inaccurate information on this subject. This disagreement exists at every level - journals, scientific meetings, clinical practice, and media outlets. The same can be said of autism, in which denials of it's etiologies are profusely displayed as only genetic, and never any environmental iatrogenic factors involved. Profusely are denials of the existance that autism is based on the bodies response to a foreign material either of neurotoxicity, bacteria or virus, such as lyme, such as vaccinal viruses, such as autoimmune processes that become overabundant. Reports are clear, that parents of autistic children seem to have higher incidences of autoimmune conditions. This is telling us something. It is my belief, this is a sign that the parents themselves have lyme disease-and are giving this infection to their children. Lyme disease is prevelant in all fifty states of the union, and know no geographical areas. However, it is interesting to note, that in states that have the less lyme, there is more availability of selenium in the soils, which is a immune neuro protectant against viruses and bacteria. Parents of autistic children routinely show low levels of selenium in blood work, as well as metallothionein deficiency, a glutathione dependent oxidant. Parents also have higher sedimentation rates (ESR) and thrombaphilia and fibrin deposits showing inflammation as well as thyroid TRH dysfunction. (14) They also often have skewed hormonal balances. Lyme is capable of changing these aspects on blood work.

Interestingly, susceptibility genes for autism parallel the susceptibility genes for arthritis and other autoimmune disorders, such as HLA-B27, and HLA-DR4. Complement immune deficiencies are common in both diseases, including C4B and C3A. The frequency of autoimmune disorders was significantly higher in families of the PDD probands compared with families of both the autoimmune and healthy control probands. Autoimmunity was highest among the parents of PDD probands compared with parents of the healthy control subjects. Hypothyroidism/Hashimoto's thyroiditis and rheumatic fever were significantly more common in families with PDD probands than in the healthy control families. Interestingly, reports of Hashimoto's and hypothyroidism are prevalent in Lyme disease. (15) Interestingly, reports of mycoplasma are consistent with parents having a mycoplasma infection, and giving that to their children. (16) Mycoplasma infections are alleviated by the self same medications for Lyme disease. Many parents report that Lyme and mycoplasma are prevalent in their children with autism. Familial association studies have also reported an increased risk of several systemic autoimmune diseases among relatives of patients with a systemic autoimmune disease. This association may reflect a common etiologic pathway with shared genetic or environmental influences among these diseases. Environmentally, there is an increase in levels of heavy metals, toxicity, neurotoxins, mold, and most of all but not least, the prevalence of the Lyme bacterium. Prolactin may also have important immune-modulating influences affecting the risk of autoimmune disease. The prolactin gene is located close to the MHC region of chromosome 6, hotspots for Lyme AND autism. (17) Another protein eaten by Lyme is melatonin, which may account for levels of serotonin, prolactin and the ability to detox heavy metals. In autism, melatonin levels are dysregulated, causing sleep disorders, and antibodies are found against serotonin.

Recently there have been reports of short term antibiotic use to kill Clostridium and or AGBN's. Vancomycin was recently used, and reports are that when on the antibiotic for thirty days, symptoms of autism decreased. The Jarish Herxheimer reaction is seen when antibiotics are having a therapeutic effect as well, as evidenced when the child is on the antibiotics. This often scares off the weary parent of an autistic child, stopping the antibiotic for fear it is contributing to their autism symptoms when in reality it was killing Lyme with mild to moderate reactions. Unfortunately, in these studies, the children regressed back to autism symptoms as soon as they were removed after thirty days. If the bacteria is not completely eliminated, the symptoms will return. This tells us, I believe, that Lyme is involved in their autism. This reasoning has made others think of why these children respond so favorably while on the antibiotic. It could be that it is going after the Lyme bacterium (however not it's stages due to it's short length), but also calming down the cytokine and chemokine activity in the immune system, and having some kind of effect on viruses which depend on cytokines and chemokines. Typically those viruses are of the herpes CMV class. Reports of HHV6 and autism are abundant. Many children have high herpes titres, sometimes to that of 4-6 times of normal,

often correlating with autism symptoms or seizures. Although the American Academy of Pediatrics recommends a three week course of antibiotics, Dr. Jones-a pediatric lyme specialist, has found that the bacteria that causes Lyme has become increasingly hardy and even when the disease is caught early, it often needs to be treated with an eight to twelve week course of antibiotics or beyond.

Lyme has many names. Among its "symptoms" are ALL of the "ideopathic" (unknown-cause) diseases that plague americans, too many to list here - it affects the body by making sugar turn to lactic acid, which in turn can scar the kidneys over time, irritate the bladder, damage the liver, and upset the pancreas. Lactic acid causes muscle soreness and sensitive intestines/cramping problems (nervous stomach). Interestingly, it is reported that many autistic children have skewed lactic and pyruvate ratios. This creates a mitochondrial disorder, oxidative stress in these children. Lyme also eats proteins, many types. It eats myelin protein, which is known to be going missing in MS patients. The localities for MS hot spots exactly match the pattern of lyme disease, and I believe it is the same disease with a different name. Myelin is what nerves are made of - and damage to myelin, without a meat and potatoes diet to replace protein constantly, causes brain troubles (alzheimers, parkinsons, alateral myotrophic scleroses, myocarditis), numbness of the bladder and/or muscle spasms in the elderly mistaken as "incontinence", nerve troubles, stuttering, etc. This correlates nicely with what we are seeing in autism. Typically there are myelination antibodies in children with autism, even Neural Axon Filament Protein antibodies, and various other antibodies against brain tissue. Many children with autism lose their ability to sleep through the night without bedwetting. Stuttering is often seen in children with autism. It is also reported that when these children harbor heavy metals, that viruses and bacteria seem to live in areas of that damage. Since these children lack the oxidant metallothionein, they cannot detox the EPA over standard of safe of thimerosal that were in childhood vaccine series, or other environmental factors including their mothers in utero exposure of amalgams. Unfortunately, they still are according to HAPI, a NPO who just tested mercury free vaccines (ARI newsletter 2004). As they work in consortium, there is a theory that the heavy metals bind to these bacteria and viruses, and when they are eliminated, heavy metals start to chelate out of the body. This is evidenced by EDTA chelation which has the ability to kill nanobacteria. Observations and biomarkers of damage are seen soon after vaccinations, but what is equally intriguing, is that lyme may be awoken after an immune lowering event such as vaccinations.

Recent reports show that children with autism are harboring lyme disease. The cry for chromosomal faults are numerous, and often paid and backed to be equally the only way you get autism. What researchers know, is that no money is funded or researched or goes into the immune lowering/autoimmunity events that create autism, and is simply put down. The cogent finding of lyme bacterium being a major risk factor for autism needs to be explored. This is superimposed on the

already sustained belief, that autism is an autoimmune disorder. Many of the imbalances of autism could be explained as the body's inability to detox and to work on this bacteria. Lyme could also be a trigger or a circumstance that initiate or worsening of the autistic condition. This includes milk allergies, strep infections, mercury, dietary intolerances of wheat and milks, and inability to deal with toxic inhibitors. Interestingly, many of the imbalances and deficiencies can be caused by lyme bacteria.

Many of the meds used for autism seem to also slow down the lyme bacterium, such as digestive enzymes, anti fungals, of course, antibiotics, anti parasticals, addressing food allergies, increasing zinc, antiinflammatories and even secretin (secretin is tied to insulin, which lyme attacks). Lately, the use of Methylcobalamine B-12 with folinic acid would also reduce lyme symptoms. Since there is already evidence of abnormal gut bacteria and exaggerated production of cytokines which result in irreversable cellular damage, it behooves parents and clinicians and researchers to look into this paralell and seek state of the art laboratory connections to lyme and autism. Three labs are recommended; STONYBROOK, BOWEN and IGENEX. This may result in the first of its kind treatment for autism via lyme therapy.

References

- (1) Changes in the Population of Persons with Autism and PDD, Dept Develop Services, California, Health and Human Services March 1 1999
- (2) The Journal of Neuroscience, January 1, 2003, 23(1):297-302
Maternal Influenza Infection Causes Marked Behavioral and Pharmacological Changes in the Offspring
- (3) IMFAR-IMMEDIATE CORD CLAMPING and AUTISM, Dr Morley
www.cordclamping.com
- (4) Abramson J, Stagnaro-Green A. Thyroid antibodies and fetal loss: an evolving story. *Thyroid* 2001;11(1):57-63.
- (5) Adenosine Deaminase - Neurogenetics. 2001 Mar;3(2):111-3. Related Articles, Links
Autism: evidence of association with adenosine deaminase genetic polymorphism. Bottini N, De Luca D, Saccucci P, Fiumara A, Elia M, Porfirio MC, Lucarelli P, Curatolo P. Department of Internal Medicine, Tor Vergata University of Rome, Rome, Italy
- (6) *J Child Neurol.* 2000 Jun;15(6):357-61. Related Articles, Links Autism associated with the mitochondrial DNA G8363A transfer RNA(Lys) mutation. Graf WD, Marin-Garcia J, Gao HG, Pizzo S, Naviaux RK, Markusic D, Barshop BA, Courchesne E, Haas RH. Department of Pediatrics, University of Washington, Seattle, USA.
- (7) *Arch Dis Child* 1997;76:264-267 (March) Fragile X, iron, and neurodevelopmental screening in 8 year old children with mild to moderate learning difficulties N Corrigan,a M Stewart,a M Scott,b F Feece a North and West Belfast Community Paediatric Unit, Belfast, Northern Ireland, b Queens University Belfast Department of Epidemiology and Public Health, Belfast,

Northern Ireland, c Belfast Education and Library Board, Belfast, Northern Ireland

- And

Life Sci. 2004 Oct 8;75(21):2539-49. Related Articles, Links

Oxidative stress in autism: increased lipid peroxidation and reduced serum levels of ceruloplasmin and transferrin--the antioxidant proteins. Chauhan A, Chauhan V, Brown WT, Cohen I. NYS Institute for Basic Research in Developmental Disabilities, 1050 Forest Hill Road, Staten Island, New York 10314, USA.

(8) - Metallothionein Deficiency - see www.hriptc.org Walsh, W, Monograph: Metallothionein and Autism". {Pfeiffer Treatment Center, Illinois, Oct 2001, Over 100 articles referenced.

(9) - Wakefield AJ, A Anthoney et al "Enterocolitis in Children with Developmental Disorders" Am.J Gastroenterology 95 No 9 (2000) p 2285-2295

(10) A case control study of mercury burden in Autistic children in autistic spectrum disorders (Journal of American Physicians and Surgeons, Vol 8, No 3, 3 Fall 2003) J Bradstreet, MD, D Geier, J Kartzinel, Md, J Adams PhD, M Geier, MD, PhD.

(11) Cure Autism Now Foundation, White Matter Disease and Autism, see website

(12) HHV-6 and Autism - Clin Immunol Immunopathol. 1998 Oct;89(1):105-8

Related Articles, Links Serological association of measles virus and human herpesvirus-6 with brain autoantibodies in autism.

Singh VK, Lin SX, Yang VC. College of Pharmacy, University of Michigan, Ann Arbor, Michigan, 48109-1065, USA.

(13) Activation of the fusiform gyrus when individuals with autism spectrum disorder view faces, N Hadjikhani, RM Joseph, J Synder, CF Chabris, J Clark, S Steel, L McGrath, M Vangel, I Aharon, E Feczko, GJ Harris, and H Tager-Flusberg, Neuroimage, Vol 22, No 3 July 2004,

(14) J Neuroimmunol. 2004 Jul;152(1-2):176-82. Related Articles, Links

Autoantibody repertoires to brain tissue in autism nuclear families. Silva SC, Correia C, Fesel C, Barreto M, Coutinho AM, Marques C, Miguel TS, Ataide A, Bento C, Borges L, Oliveira G, Vicente AM.

Instituto Gulbenkian de Ciencia, Rua da Quinta Grande 6, 2781-196 Oeiras, Portugal.

(15) Pediatrics. 2003 Nov;112(5):e420. Related Articles, Links

Increased prevalence of familial autoimmunity in probands with pervasive developmental disorders.

Sweeten TL, Bowyer SL, Posey DJ, Halberstadt GM, McDougle CJ. Department of Psychiatry, Indiana University School of Medicine, and James Whitcomb Riley Hospital for Children Indianapolis 46202-4800, USA.

(16) Chronic Mycoplasmal Infections in Autism Patients

Garth L. Nicolson,¹ PhD, Marwan Y. Nasralla,² PhD, Paul Berns,¹ MD and Jeorg Haier,³ MD, PhD

¹ The Institute for Molecular Medicine, Huntington Beach, California, USA,,

² International Molecular Diagnostics, Inc., Huntington Beach, California, USA,

3 Department of Internal Medicine, and 3Department of Surgery,
Wilhelm-University, Munster, Germany. Correspondence: Prof. Garth L. Nicolson,
Office of the President, The Institute for Molecular Medicine, 15162 Triton
Lane, Huntington Beach, California 92649. Tel: 714-903-2900; Fax: 714-379-2082;
Website: www.immed.org

(17) Am J Med Genet. 1999 Nov 5;87(1):17-22. Related Articles, Links

Subtle overlapping deletions in the terminal region of chromosome 6q24.2-q26:
three cases studied using FISH. Sukumar S, Wang S, Hoang K, Vanchiere CM,
England K, Fick R, Pagon B, Reddy KS.

Cytogenetics Department, Quest Diagnostics Inc., San Juan Capistrano, California