Society for the Advancement of Scientific Hermeneutics

Interpreting the Evolution of Linguistics from Hippocrates to Hypocrises

Lyme Disease Biomarkers

CDC/ALDF's Valid Biomarkers in "Lyme Disease," not used in Klempner/IDSA's Lyme disease "re-treatment" study, and "guidelines."

Here we reveal the valid biomarkers of illness in "Lyme Disease" discovered by the CDC's ALDF.com (American Lyme Disease Foundation and later IDSociety.org) cabal, yet they were not used for the assessments or outcomes of Mark Klempner's Lyme disease "re-treatment" study or IDSociety.org's "Guidelines on the Diagnosis and Treatment of Lyme disease."

Through this contrast we demonstrate criminal acts of those responsible for the Lyme disease scam. The scam was essentially the CDC falsifying the testing and "case definition" at a conference in Dearborn, MI (1994) in order to falsely qualify their OspA and other patent outcomes. The purpose of the Dearborn stunt was to falsely claim that "Lyme disease is only an HLA-linked hypersensitivity or allergy response," knowing otherwise. Criminal charges will include "fraud with malice" because of the slander and libel against their victims. If "fraud on the government" is performed by government employees "with malice" or intent to cause harm, they do not have immunity from criminal charges. Those chargeable in the criminal Lyme disease scam include CDC officers Allen Steere, Alan Barbour, Barbara Johnson and Mark Klempner; NIH employee Edward McSweegan; NYMC and Yale's Durland Fish; and their associates with the ALDF.com.

These perpetrators claimed that vector borne diseases were a "rich vein of gold from which to mine…" patent royalties (Alan Barbour). There are more quotes revealing clear malicious intent towards their victims in the 2003 complaint to the UN about these crimes here, in a formal complaint to the UN (which they answered by saying they needed volumes of complaints): http://www.actionlyme.org/UN_PETITION.htm

This science of fungal antigen-induced immunosuppression exposes 1) the mechanisms that produce the Autism pandemic, 2) the nature of Bioweapons (stealth, no antibodies), and that 3) the CDC and the NIH are embarrassed that they allowed this bunch of clowns to run a "OspA/Pam3Cys is a vaccine" scam. But spirochetes are not typical bacteria. They are their own phylum and shed fungal antigens. They might as well be called myco-chetes.

1. Mechanisms that produce the autism pandemic parallel chronic Lyme disease

The link between Lyme disease (the real name is Relapsing Fever) and Autism is the fungal antigen OspA (Pam3Cys). OspA and antigens like it are shed all the time in borreliosis or Relapsing Fever (RF) in a process called blebbing. This blebbing or shedding of fungal, lipopeptide surface antigens has something to do with RF's immune evasion. But they cause immunosuppression, the reactivation of latent herpesviruses, and also tolerance-spreading from TLR2/1-agonist tolerance to viral (Harding, http://www.ncbi.nlm.nih.gov/pubmed/20660347) and to other bacterial type tolerance, such as LPS/TLR4-agonists (Redmond, http://www.ncbi.nlm.nih.gov/pubmed/16461741).

Thimerosal is put in vaccines to prevent fungi. It has been known at least since the 1950s that you *can't inject fungi together with viruses into a mammal* as this causes the viruses to become activated and lethal (Mice infected with mycoplasma plus a hepatitis virus: http://www.ncbi.nlm.nih.gov/pubmed/13109101).

Conversely, pediatricians give children with cold viruses antibiotics to prevent secondary ear infections because they knew one infection tends to invite another. The CDC's influenza mortality data does not directly mention that the vast majority of deaths were due to the secondary pneumonia infections. In the 1918 Spanish Flu pandemic, it was again the secondary, mycobacterial infections that killed most people. In these examples, you've see the very real dynamic of fungal-viral synergy working in both directions: fungal infections assist viral, and viral invections invite bacterial.

2. The nature of bioweapons – stealth or no HLA- or hypersensitivity-response

The second reason the CDC does not want anyone to know about the mechanisms of illness from spirochetes constantly shedding/blebbing outer surface fungal lipoproteins and with antigenic variation ("multi-clonal populations overwhelm the immune system," Barbour's US Patent 6,719,983 and related), "even if infected with just one spirochete" (http://www.ncbi.nlm.nih.gov/pubmed/14861181, Barbour, et al, referenced that "single spirochete" report), is that the description of a bioweapon happens to match Alan Barbour's "multiclonal populations... overwhelm the immune system." **A bioweapon will have no antibodies that identify the original detonator infection.**

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However, others are leaking this information. And Russia knows the NYMC-associated Russians were HLA-datapharming (meaning they were looking at local populations' HLAs) all over the world. Bioweapons are not designed against a population who will make strong, robust, healthy antibodies. Stealth bioweapons target populations where there is no association to HLA groups that will produce many antibodies and potentially identify the original infections. See "Ethnic Bioweapons" in Wikipedia where the Russian Duma banned the export of their populations' DNA to America in 2007 for this reason.

3. NIH and CDC are embarrassed that they allowed these scientifically incompetent people to run "Lyme Disease."

The fungal OspA non-vaccines caused the same systemic, "multi-system" (Persing and Schoen), "protean" (Luft) disease as "Chronic Lyme," and the NIH and CDC are terrified of everyone knowing how badly that has screwed up all U.S. medical science for decades. The crooked USA "government" currently stands behind the IDSA's spin on short-term-treatment-only because they know Late Neurologic Chronic Lyme is really about reactivated latent herpesviruses and systemic fungal and bacterial diseases. It's AIDS-like.

If the USDA.gov and CDC wanted to hide an accidental release of the modified-for-the-hard-bodied-Ixodes-tick African Bird Borreliosis *anserina* (called burgdorferi now), they certainly picked the wrong bumbling, obtuse, low-life gang to try to pull it off. Deploying vicious, foul-mouthed, stalking, slandering, libeling cowards who used criminal "anonymous internet harassment" and all their other transparent and stupid lab stunts such as what Steere did to falsify the Dearborn case definition and this moronic "Klempner study," was the wrong way to play it. Western society is just not familiar with such vicious, aggressive sledgehammer "treatment" of very sick people from a self-alleged "medical society." Their aggressive behavior towards very sick people is classic "defensive behavior" (means aggressive behavior, believe it or not, but that's psychiatry) and betrays their guilt.

Post Sepsis Syndrome

Despite all this, the new news is that the NIH has endorsed the description of all the similar chronic fatiguing illnesses – CFIDS, ME, Fibromyalgia, Lyme and possibly Gulf War Illness - by Washington University St Louis (wustl.edu) in summer of 2014, **shown below**. We'll just agree with them and call these diseases post-sepsis syndrome (PSS). PSS implies ongoing, active infections, and not just the post-septic shock's well-known organ, tissue and immune system damage. They, wustl and the NIH, refer to the herpesviruses, especially Epstein-Barr in PSS.

Notice that that PSS description is in parallel with what happens when a child is immunosuppressed naturally or is immunosuppressed because she/he has a concurrent active bacterial infection, and is vaccinated anyway. Or, in the cases where the vaccine vial has been contaminated with mycoplasma [which is "myco" (which is fungal)], which is like OspA, and causes immunosuppression and the lack of antibody production. The child will get the viruses instead of the protection, as reported by the CDC themselves. Congenital Rubella causes Autism and that was the reason they decided to vaccinate against it in the first place. Measles is also a neurotropic virus. We call the general dynamic Fungal-Viral Synergy.

The "IDSA Guidelines" are intended to give the appearance that the Lyme cabal believes the Dearborn case definition is real. But most of the cabal members were present for the Dearborn stunt. For example, Gary Wormser's contribution was that the Steere's research-fraud criteria was only 15% accurate in IgG (detects 9/59 cases), or misses 85%.

In 1997 Mark Klempner received a $4.7 million grant to perform research fraud and then declare that more treatment does not help Lyme victims. The IDSociety.org's "Guidelines" on the diagnosis and treatment of Lyme disease are based on this bogus Klempner report.

Two Controlled Trials of Antibiotic Treatment in Patients with Persistent Symptoms and a History of Lyme Disease
http://content.nejm.org/cgi/reprint/345/2/85.pdf

There were numerous fraudulent events in that Klempner study design and in the results-reporting.

- Klempner used the falsified Dearborn case definition as the inclusion/exclusion criteria. Dearborn was not FDA-valid, was invented via research fraud by Allen Steere in Europe in 1992, and was not even a consensus at that 1994 Dearborn consensus conference.

- Two-thirds of Klempner's "re-treatment" victims never had IV ceftriaxone before, yet he claimed he was retreating with the standard of care at the time, which was 30 days of ceftriaxone. Two-thirds of those patents were not "re-treated," so there is no data here to report.

Klempner also did not report which DNA primers he used to detect "NO LYME" in the spinal fluid of his victims (see the DNA & RNA Primers Shell Game). It turns out Klempner used the OspA gene, which undergoes antigenic variation and is not likely to be found with OspA primers from spirochetes fresh out of a tick. And in fact, whenever Mark Klempner did find such OspA-gene-positive-DNA in the spinal fluid of his potential victims, he rejected them from the study. Not only did Klempner *say* in his write up of the report protocol that if they were positive for Bb DNA in the spinal fluid, they would be rejected from the study—this actually happened. We know of at least one person who had Bb DNA in her spinal fluid that Klempner rejected from the study, yet Klempner did not report this. He said publicly at the 2001 Rhode Island Diseases of Summer Conference at South County Hospital that there were not any cases of

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DNA-positive Lyme to be found among his study candidates. (We have him on audiotape.)

In 2005 Klempner wrote 2 important reports; one with a man named Kaplan at UConn and another with Gary Wormser. In the report with Wormser, they revealed that there were 2 kinds of Lyme: The Dearborn, HLA-linked arthritis in a knee kind, and the other, the 85%, the neurological, seronegative kind. Once again we heard Lyme arthritis cases—cases where the patients are not actually sick—are the only ones allowed to have a disease. That is, the only people who test positive to the false Dearborn case definition have a genetic, HLA-linked arthritis or hypersensitivity; the "C6 Peptide Test" is the same—it only detects Lyme arthritis:
A case-control study to examine HLA haplotype associations in patients with posttreatment chronic Lyme disease.
"Patients generally feel well aside from their arthritis symptoms."
http://www.ncbi.nlm.nih.gov/pubmed/16107953

In the report with Kaplan, Klempner reported that these people had no neurological compromise and therefore their symptoms were psychiatric:

"Cognitive function in post-treatment Lyme disease: do additional antibiotics help?"
"CONCLUSION:
"Patients with post-treatment chronic Lyme disease who have symptoms but show no evidence of persisting Borrelia infection do not show objective evidence of cognitive impairment. Additional antibiotic therapy was not more beneficial than administering placebo." http://www.ncbi.nlm.nih.gov/pubmed/12821733

Everyone knows that's false. Mark Klempner himself reported extensively about cognitive impairment and biomarkers of central nervous system degradation. Klempner, in addition to finding that Lyme was not curable with IV ceftriaxone—that is, it does not kill all the spirochetes, even without cells to hide within—he found that the majority (79%) of Lyme victims have a unique sign or biomarker of a nerve and brain degrading enzyme called matrix-metalloproteinase-130.

Here are those 2 reports:

Matrix metalloproteinases in the cerebrospinal fluid of patients with Lyme neuroborreliosis.
"Neurologic manifestations of Lyme disease include meningitis, encephalopathy, and cranial and peripheral neuropathy….The 130-kDa MMP was found without the 92-kDa MMP9 in the CSF of 11 (79%) of 14 patients with neuroborreliosis and only 7 (6%) of 118 control patients (P < .001). This pattern of CSF gelatinase activity may be a useful marker for neuroborreliosis. http://www.ncbi.nlm.nih.gov/pubmed/9466528
FULL TEXT: http://www.actionlyme.org/Retro_Klempnerization.htm

and

Fibroblasts protect the Lyme disease spirochete, Borrelia burgdorferi, from ceftriaxone in vitro.
"The Lyme disease spirochete, Borrelia burgdorferi, can be recovered long after initial infection, even from antibiotic-treated patients, indicating that it resists eradication by host defense mechanisms and antibiotics…. The ability of the organism to survive in the presence of fibroblasts was not related to its infectivity. Fibroblasts protected B. burgdorferi for at least 14 days of exposure to ceftriaxone. Mouse keratinocytes, HEp-2 cells, and Vero cells but not Caco-2 cells showed the same protective effect. Thus, several eukaryotic cell types provide the Lyme disease spirochete with a protective environment contributing to its long-term survival."
http://www.ncbi.nlm.nih.gov/pubmed/1634816
FULL TEXT: http://actionlyme.org/Mark_Klempner_Fibroblasts.htm

Mark Klempner also wrote in 1998 that OspA was the cause of anti-myelin antibodies or probably contributed to the MS form of Lyme. (He may have meant OspC, since that was my reading of Roland Martin's 1988 "Lyme causes Multiple Sclerosis" report, but regardless, MS is not a personality or anxiety disorder):
Is it thee or me?--autoimmunity in Lyme disease.
http://www.ncbi.nlm.nih.gov/pubmed/10581067
http://actionlyme.org/KFORSCHNER_DISCOVERS_LYME_TOXIN.htm

According to Mark Klempner, Lyme is incurable, causes nerve and brain degrading enzymes as a marker of this terrible disease, and antibodies against OspA cause anti-myelin antibodies or causes MS. But later he performed the research fraud reports where Lyme is nothing but psychiatrically induced imaginings of disability and cognitive dysfunction.

The other biomarkers

discovered by the same persons who libel us with the likes of Munchausen's and Munchausen's-by-Proxy accusations?

A) MMP-130 - Klempner as shown above.

B) ROBERT SCHOEN and GFAp, or glial-fibrillary acidic protein. GFAp is found in the CNS as a biomarker of glial cell degradation in late chronic neurologic Lyme victims:

The Lyme Disease Vaccine: Conception, Development, and Implementation
"Other peripheral neuropathies and Lyme meningitis are also seen at this stage. In late-stage disease, the central nervous system may be involved. A new diagnostic test measuring glial fibrillary acidic protein in cerebrospinal fluid may prove to be a useful tool for measuring such involvement (20)."
http://annals.org/article.aspx?articleid=713400

C) SIGAL and BARBOUR and Anti-heat-shock antibodies (anti-flagellar antibodies)

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H9724, a monoclonal antibody to Borrelia burgdorferi's flagellin, binds to heat shock protein 60 (HSP60) within live neuroblastoma cells: a potential role for HSP60 in peptide hormone signaling and in an autoimmune pathogenesis of the neuropathy of Lyme disease.
"Although Borrelia burgdorferi, the causative agent of Lyme disease, is found at the site of many disease manifestations, local infection may not explain all its features. B. burgdorferi's flagellin cross-reacts with a component of human peripheral nerve axon, previously identified as heat shock protein 60 (HSP60). The cross-reacting epitopes are bound by a monoclonal antibody to B. burgdorferi's flagellin, H9724. Addition of H9724 to neuroblastoma cell cultures blocks in vitro spontaneous and peptide growth-factor-stimulated neuritogenesis. Withdrawal of H9724 allows return to normal growth and differentiation. Using electron microscopy, immunoprecipitation and immunoblotting, and FACS analysis we sought to identify the site of binding of H9724, with the starting hypotheses that the binding was intracellular and not identical to the binding site of II-13, a monoclonal anti-HSP60 antibody. The current studies show that H9724 binds to an intracellular target in cultured cells with negligible, if any, surface binding. We previously showed that sera from patients with neurological manifestations of Lyme disease bound to human axons in a pattern identical to H9724's binding; these same sera also bind to an intracellular neuroblastoma cell target. II-13 binds to a different HSP60 epitope than H9724: II-13 does not modify cellular function in vitro. As predicted, II-13 bound to mitochondria, in a pattern of cellular binding very different from H9724, which bound in a scattered cytoplasmic, nonorganelle-related pattern. H9724's effect is the first evidence that HSP60 may play a role in peptide-hormone-receptor function and demonstrates the modulatory potential of a monoclonal antibody on living cells."
http://www.ncbi.nlm.nih.gov/pubmed/11860186

So they're saying antibodies against flagellin causes some pathology, while at the same time saying band 41 means nothing and you have a non-disease. It happens to be for the very reason - says Barbour - that antibodies against flagellin cause cross-reactive antibodies against human heat shock protein-60 that there is no flagellin vaccine. So, because the anti-flagellar antibody causes harm and damage, the crooks say if you HAVE that antibody, it means you're psychiatric and don't have a real disease.

D) LENNY SIGAL and QEEG or electroencephalograms (Sigal = Munchausen's accuser)

QEEG and evoked potentials in central nervous system Lyme disease.
"Quantitative EEG, flash visual evoked potentials, auditory evoked potentials to common and rare tones, and median nerve somatosensory evoked potentials were obtained from 12 patients with active CNS Lyme disease and from 11 patients previously treated for active CNS Lyme disease. Abnormal QEEG and/or EPs were found in 75% of the active Lyme disease patients and in 54% of the post CNS Lyme disease patients. Three different types of neurophysiological abnormality were observed in these patients includingQEEG slowing, possible signs of cortical hyperexcitability, and focal patterns indicating disturbed interhemispheric relationships. In patients tested before and after treatment QEEG and EP normalization was associated with clinical improvement."
http://www.ncbi.nlm.nih.gov/pubmed/7554300
http://www.actionlyme.org/MUNCHAUSENS.htm
in http://www.amazon.com/Lyme-Disease-Key-Diseases-Series/dp/0943126584

E) ALLEN STEERE and Brain SPECT or Hypoperfusion

Reversible cerebral hypoperfusion in Lyme encephalopathy.
"Lyme encephalopathy (LE) presents with subtle neuropsychiatric symptoms months to years after onset of infection with Borrelia burgdorferi. Brain magnetic resonance images are usually normal. We asked whether quantitative single photon emission computed tomography (SPECT) is a useful method to diagnose LE, to measure the response to antibiotic therapy, and to determine its neuroanatomic basis. In 13 patients with objective evidence of LE, SPECT demonstrated reduced cerebral perfusion (mean perfusion defect index [PDI] = 255), particularly in frontal subcortical and cortical regions. Six months after treatment with 1 month of intravenous ceftriaxone, perfusion significantly improved in all 13 patients (mean PDI = 188). In nine patients with neuropsychiatric symptoms following Lyme disease, but without objective abnormalities (e.g., possible LE), perfusion was similar to that of the treated LE group (mean PDI = 198); six possible LE patients (67%) had already received ceftriaxone prior to our evaluation. Perfusion was significantly lower in patients with LE and possible LE than in 26 normal subjects (mean PDI = 136), but 4 normal subjects (15%) had low perfusion in the LE range. We conclude that LE patients have hypoperfusion of frontal subcortical and cortical structures that is partially reversed after ceftriaxone therapy. However, SPECT cannot be used alone to diagnose LE or determine the presence of active CNS infection."
http://www.ncbi.nlm.nih.gov/pubmed/9409364

F) STEERE and YALE on Lyme Causing Lupus: Antiphospholipid antibodies (probably more likely to be due to the reactivated EBV, but we will look more closely later)

Reactivity of neuroborreliosis patients (Lyme disease) to cardiolipin and gangliosides.
"A subset of patients (50%) with neuroborreliosis (Lyme disease) showed IgG reactivity to cardiolipin in solid phase ELISA. In addition, a subset of patients with neuroborreliosis (29%) and syphilis (59%) had IgM reactivity to gangliosides with a Gal(beta 1-3) GalNac terminal sequence (GM1, GD1b, and asialo GM1). Anti-ganglioside IgM antibodies were significantly more frequent in these two groups of patients compared to patients with cutaneous and articular Lyme disease, primary antiphospholipid syndrome, systemic lupus erythematosus and normal controls. Correlative evidence and adsorption experiments indicated that antibodies to cardiolipin had separate specificities from those directed against the gangliosides. IgM antibodies to Gal(beta 1-3) GalNac gangliosides appeared to have similar specificities since these were positively correlated and inhibitable by cross adsorption assays. Given the clinical associations of patients with neuroborreliosis and syphilis with IgM reactivity to gangliosides sharing the Gal(beta 1-3) GalNac terminus,

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we suggest that these antibodies could represent a response to injury in neurological disease or a cross reactive event caused by spirochetes."
http://www.ncbi.nlm.nih.gov/pubmed/8410057
FULL TEXT: http://www.actionlyme.org/STEERE_AND_LUPUS_LYME.htm

G) JJ HALPERIN and Quin or quinolinic acid found in the central nervous system, which is a product of the immune response against a bacterial infection (JJ Halperin)

Neuroactive kynurenines in Lyme borreliosis.
"In patients with encephalopathy, serum QUIN was elevated with corresponding increments in CSF QUIN. Lymphokine concentrations were not consistently elevated. We conclude that CSF QUIN is significantly elevated in B burgdorferi infection--dramatically in patients with CNS inflammation, less in encephalopathy. The presence of this known agonist of NMDA synaptic function--a receptor involved in learning, memory, and synaptic plasticity--may contribute to the neurologic and cognitive deficits seen in many Lyme disease patients...."
http://www.ncbi.nlm.nih.gov/pubmed/1531156

H) HALPERIN, DATTWYLER, "Lyme Is associated with ALS":

Immunologic reactivity against Borrelia burgdorferi in patients with motor neuron disease.
"Of 19 unselected patients with the diagnosis of amyotrophic lateral sclerosis (ALS) living in Suffolk County, New York (an area of high Lyme disease prevalence), 9 had serologic evidence of exposure to Borrelia burgdorferi; 4 of 38 matched controls were seropositive. Eight of 9 seropositive patients were male (8 of 12 male patients vs 2 of 24 controls). Rates of seropositivity were lower among patients with ALS from nonendemic areas. All patients had typical ALS; none had typical Lyme disease. Cerebrospinal fluid was examined in 24 ALS patients—3 (all with severe bulbar involvement) appeared to have intrathecal synthesis of anti-B burgdorferi antibody. Following therapy with antibiotics, 3 patients with predominantly lower motor neuron abnormalities appeared to improve, 3 with severe bulbar dysfunction deteriorated rapidly, and all others appeared unaffected. There appears to be a statistically significant association between ALS and immunoreactivity to B burgdorferi, at least among men living in hyperendemic areas."
http://www.ncbi.nlm.nih.gov/pubmed/2334308
FULL TEXT: http://www.actionlyme.org/ALSLYME47.htm

I) STEERE and NITRIC OXIDE in the brain (by Allen Steere):

Borrelia burgdorferi and Escherichia coli lipopolysaccharides induce nitric oxide and interleukin-6 production in cultured rat brain cells.
http://www.ncbi.nlm.nih.gov/pubmed/7513330

J) BENACH and Anti-ganglioside antibodies
Experimental immunization with Borrelia burgdorferi induces development of antibodies to gangliosides.
"Patients with neuroborreliosis produce antibodies, mostly of the immunoglobulin M (IgM) class, to gangliosides, particularly to those with Gal(beta 1-3)GalNac terminal sequences. Lewis rats were immunized with a nonpathogenic strain of Borrelia burgdorferi and with a chloroform-methanol extract (nonprotein) of this organism (CM) to determine whether antibodies to B. burgdorferi also recognized gangliosides. Rats were also immunized with asialo-GM1 to determine whether the elicited antibodies recognized antigens in B. burgdorferi. Rats immunized with B. burgdorferi produced low levels of IgM antibodies that cross-reacted with asialo-GM1 and GM1. Rats immunized with CM had marked IgM reactivity to asialo-GM1 and GM1. Immunization with asialo-GM1 resulted in antibodies that cross-reacted with B. burgdorferi antigens. Although antibodies to B. burgdorferi were of both the IgM and IgG classes, those to CM and to asialo-GM1 and GM1 were predominantly in the IgM fraction. Reactivity of the IgM antibodies decreased after adsorption with the heterologous and the homologous antigens, indicating bidirectional cross-reactivity between CM, asialo-GM1, and GM1 and that immunization with one produces antibodies to the other. There was no in vivo deposition of Ig in peripheral nerves, nor was there nerve pathology as a result of immunizations, but IgM antibodies to asialo-GM1 and CM recognized homologous antigens in the nodes of Ranvier of peripheral nerves from nonimmunized rats. This immunization model suggests that antibodies to gangliosides in Lyme disease have a microbial origin and are potentially relevant in pathogenesis."
http://iai.asm.org/content/63/10/4130.full.pdf+html?view=long&pmid=7558329

K) 1989, PAUL DURAY in IDSA's journal with the most important biomarker of all,.....

Clinical pathologic correlations of Lyme disease.
"Immature B cells can also be seen in the spinal fluid. These cells can appear quite atypical- not unlike those of transformed or neoplastic lymphocytes." -- http://www.ncbi.nlm.nih.gov/pubmed/2814170
Full Text: http://www.actionlyme.org/IDSA_CLINIPATH_DURAY.htm

1992, Duray again in 1992, in Steve Schutzer's review of the 1992 Cold Spring Harbor Conference on Lyme:
Lyme Disease: Molecular and Immunologic Approaches **(book)**

"On occasion, these atypical-appearing large lymphocytes have been misinterpreted in biopsy by several laboratories as cells of a malignant lymphoma or leukemia. Bb antigens, then, may stimulate growth of immature lymphocytic subsets in some target organs, as well as in the cerebrospinal fluid (Szyfelbein and Ross 1988). Usual bacterial infections do not produce such lymphocytic infiltrates in tissue. ****These immunoblastoid cells in Bb infections at times resemble those found in Epstein-Barr virus infections.**** Does Bb reactivate latent virus infections in tissues? Do some tick inocula harbor simultaneous infectious agents (ixodid ticks can harbor Rickettsiae, Babesia microti, and Ehrlichia bacteria, in addition to Bb), producing multi-agent infections

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in some hosts? Further studies can clarify these issues by mans of tissue-based molecular probe analysis." -

Paul Duray, NCI, NIH, Ft. Detrick, at the 1992 Cold Spring Harbor ALDF.com conference, published in Steve Schutzer's Lyme Disease: Molecular and Immunologic Approaches (book)

2006, The NIH (NINDS's MS-Lyme Group) group that discovered that *** OspA *** was the cause of the MS/New Great Imitator outcome of Lyme reporting in the New York Times in the summer of 2013 (Martin and Marques, 2006); this article says these OspA like antigens constantly shed by Borreliae cause immunosuppression in the humoral immune system, but apparently a chronic inflammatory state in the central nervous system:

Borrelia burgdorferi Induces TLR1 and TLR2 in human microglia and peripheral blood monocytes but differentially regulates HLA-class II expression.
http://www.ncbi.nlm.nih.gov/pubmed/16783164

And this report means you might not even have anti-flagellar antibodies (flagellin is a TLR5-agonist) after being exposed to shed fungal OspA like antigens (TLR2/1-agonists):

Borrelia burgdorferi lipoprotein-mediated TLR2 stimulation causes the down-regulation of TLR5 in human monocytes.
http://www.ncbi.nlm.nih.gov/pubmed/16479520

2013, Same NIH MS-Lyme Group as above, Martin and Marques:

When Lyme Disease Lasts and Lasts **– Jane Brody, NYTimes**
"Complicating the picture is the fact that some people with PTLDS symptoms apparently never had Lyme disease in the first place, Dr. Marques said in an interview. There are other infectious organisms — Epstein-Barr virus, for example — that can produce similar symptoms and may be the real culprits."
http://well.blogs.nytimes.com/2013/07/08/when-lyme-disease-lasts-and-lasts/

2014, Wustl.edu discovers that sepsis is like Lyme, in that the survivors of it are likely to have survived via the immunosuppression (TLR2-agonist tolerance/Endotoxin tolerance), but the result is the reactivation of latent viruses:

Dormant viruses re-emerge in patients with lingering sepsis, signaling immune suppression
"Patients with lingering sepsis had markedly higher levels of viruses detectable in the blood, compared with the healthy controls and critically ill patients without sepsis. Among the sepsis patients, for example, the researchers found that 53 percent had Epstein-Barr virus, 24 percent had cytomegalovirus, 14 percent had herpes-simplex virus, and 10 percent had human herpes simplex virus-7.

"These viruses generally don't lead to significant illness in people who are healthy but can cause problems in patients who are immune-suppressed. "
http://news.wustl.edu/news/Pages/27015.aspx

FULL JOURNAL REPORT, snippet…

Reactivation of Multiple Viruses in Patients with Sepsis
"Sepsis is the host's non-resolving inflammatory response to infection that leads to organ dysfunction [1], [2]. A current controversial hypothesis postulates that if sepsis pursues a protracted course, it progresses from an initial primarily hyper-inflammatory phase to a predominantly immunosuppressive state [3]–[7]. … However, several issues have limited this approach including lack of consensus that immunosuppression is a clinically important phenomenon [5], [6], [13]… Latent viruses such as cytomegalovirus are normally held in abeyance by cellular and immune surveillance mechanisms which if impaired, for example by immunosuppressive medications, often result in viral reactivation, replication, and virally-mediated tissue injury [15]–[20]. Sepsis impairs innate and adaptive immunity by multiple mechanisms including apoptosis-induced depletion of immune effector cells and induction of T-cell exhaustion thereby possibly predisposing to viral reactivation and dissemination [21]–[23]. …" http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0098819

2014, Here the NIH agrees that post-sepsis, like wustl above describes, matches their own observations of what happens as a result of Chronic Lyme (EBV reactivated; ie, that being generally accepted as the main driver of MS and Lupus):

NEW, by the NIH:
Surviving Sepsis: Detection and Treatment Advances

By Carolyn Beans for the National Institutes of Health | August 18, 2014 08:43am ET
http://www.livescience.com/47387-sepsis-diagnosis-treatment-research-nigms.html

Preventing Secondary Infections
"Some people who survive sepsis can develop secondary infections days or even months later. A research team that included Richard Hotchkiss, Jonathan Green and Gregory Storch of Washington University School of Medicine in St. Louis suspected that this is because sepsis might cause lasting damage to the immune system. To test this hypothesis, the scientists compared viral activation in people with sepsis, other critically ill people and healthy individuals. The researchers looked for viruses like Epstein-Barr and herpes simplex that are often dormant in healthy people but can reactivate in those with suppressed immune systems. [Sepsis Has Long-Term Impact for Older Adults, Study Finds]"

In the end, one wonders how the CDC and IDSA get off saying Lyme has no illness signs or is a somatoform disorder. As long as people don't know what OspA is, they'll get away with this charade.

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On USA's Bioweapons from the Congressional Record, 103rd Congress:

Types of Biological Agents

Different antipersonnel agents require varying periods of time before they take effect, and the periods of time for which they will incapacitate a person also vary. Most of the diseases having antipersonnel employment potential are found among a group of diseases that are naturally transmitted between animals and man. Mankind is highly vulnerable to them since he has little contact with animals in today's urban society. The micro-organisms of possible use in warfare are found in four naturally occurring groups - the fungi, bacteria, rickettsiae, and viruses.[62]

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[60] Nuclear and Chemical Operations, MCI 7711B, Marine Corps Institute, Command and Staff College's nonresident program (Marine Barracks, Washington, D.C., 1983), p. 8, section 1501.

[61] Ibid.

[62] Ibid, p. 9, section 1502.

agent. An aerosol or mist of biological agent is borne in the air. These agents can silently and effectively attack man, animals, plants, and in some cases, materiel. Agents can be tailored for a specific type of target.[60]

Methods of using antipersonnel agents undoubtedly vary so that no uniform pattern of employment or operation is evident. It is likely that agents will be used in combinations so that the disease symptoms will confuse diagnosis and interfere with proper treatment. It is also probable that biological agents would be used in heavy concentrations to insure a high percentage of infection in the target area. The use of such concentrations could result in the breakdown of individual immunity because the large number of micro-organisms entering the body could overwhelm the natural body defenses.[61]

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Lyme Disease Biomarkers, continued

"Methods of using antipersonnel agents undoubtedly vary so that no uniform pattern of employment or operation is evident [make sure it does not produce antibodies, is the short version- KMD]. It is likely that agents will be used in combinations so that disease symptoms will confuse diagnosis and interfere with proper treatment. It is also probable that biological agents would be used in heavy concentrations to insure [SIC] a high percentage of infection [or just use the OspA vaccine- KMD] in the target area. The use of such concentrations [or the multiple infections it causes, due to the immunosuppression like HIV, Lyme, or LYMErix as acquired immune deficiencies - KMD] could result in the breakdown of individual immunity because the large number of micro-organisms entering the body could overwhelm the natural body defenses [or just infect or inject people with an immune suppressor like OspA from a tick or a syringe, and the reverse will happen: people will acquire multiple infections because their immunity is trashed by OspA- KMD].

It is extremely important that people actually read that. It matches the “single spirochete producing multiple variants” and “these multiple variants each undergoing limitless antigenic variation…,” “could overwhelm the immune system,” claims, especially if they are of the OspA or fungal type.

Basically these crazy people associated with the CDC and ALDF.com wanted to inject people with the very thing that causes the New Great Imitator outcomes. It was like a Tuskegee “Bad Blood” experiment on steroids.

150219,KMD,SASH