CHARGE SHEET 3: BIOMARKERS

Lobbying for a hearing for referral to the USDOJ for a prosecution of the Lyme disease crimes.

Biomarkers of Real Illness Discovered and Described by Yale and IDSA.

The entire Lyme scam, as you know by now, was performed by CDC officers (Allen Steere, Alan Barbour, Barbara Johnson, Mark Klempner) with the assistance of self-alleged smart people like Edward McSweegan and Durland Fish. The latter 2 see themselves as Double-Oh Secret Bioweaponeers. The CDC officers admire themselves as clever business people, beating everyone else to the patent office. They claimed that vector borne diseases were a "rich vein of gold" from which to mine patent royalties.

However, in addition to attempting to profiteer off the calamity, the Lyme scam was performed for 2 reasons. The first reason has to do with the Autism pandemic. The association between Lyme and Autism is OspA, not necessarily spirochetes. OspA causes imunosuppression and the reactivation of latent herpes viruses and also tolerance-spreading from TLR2/1-agonist tolerance to viral and bacterial tolerance (other TLR-agonists, like TLR4 and TLR7 and TLR9 - See Medvedev and Harding). Now it appears that the NIH has endorsed the description by Washington University St Louis the summer of 2014 and we are calling this post-sepsis. It implies ongoing active infections, and not just post-septic shock damage.

They (wustl.edu and the NIH) refer to the herpes viruses, especially Epstein-Barr. This is in parallel with what happens when a child is immunosuppressed, has a concurrent active bacterial infection and is vaccinated anyway, or the vaccine vial has been contaminated with mycoplasma, which is myco, which is fungal, which is like OspA: causes immunosuppression and the lack of antibody production. The child will get the virus instead of the protection. Congenital Rubella causes Autism—that was the reason they decided to vaccinate against it in the first place. The Occam's Razor and SASH policy paper on Autism Vaccines and ME/CFS contain more on this.

The second reason the CDC does not want anyone to know about the mechanisms of illness from spirochetes constantly shedding outer surface proteins in a process called blebbing-plus-antigenic variation ("multi-clonal populations overwhelm the immune system," (Barbour), "even if infected with just one spirochete" (Barbour, et al), is that the description of a bioweapon happens to match Alan Barbour's "multiclonal populations... overwhelm the immune system." And have no antibodies that identify the original detonator infection. See the Primers Shell Game report for that data.

However, others are leaking this information. And Russia knows the NYMC associated Russians were HLA-datapharming (this means they were looking at HLAs all over the world); one does not design a bioweapon against a population that will make strong, robust, healthy antibodies. No. You go for the reverse—populations where there is ***NO*** association to HLA groups that will produce many antibodies and identify the original infections. See Ethnic Bioweapons in Wikipedia where the Russian Duma kicked all Americans out in 2007 for this reason.

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On Biomarkers, let's look at the present view (due to Mark Klempner's "Re-treatment study" scam) and the work backwards.

In 1997 Mark Klempner took a 4.7 million dollar grant to perform research fraud and then declare that more treatment does not help Lyme victims. Here is that report:

N Engl J Med. 2001 Jul 12;345(2):85-92.

Two controlled trials of antibiotic treatment in patients with persistent symptoms and a history of Lyme disease.

Klempner MS1, Hu LT, Evans J, Schmid CH, Johnson GM, Trevino RP, Norton D, Levy L, Wall D, McCall J, Kosinski M, Weinstein A.

"BACKGROUND:
"It is controversial whether prolonged antibiotic treatment is effective for patients in whom symptoms persist after the recommended antibiotic treatment for acute Lyme disease.
METHODS:
We conducted two randomized trials: one in 78 patients who were seropositive for IgG antibodies to Borrelia burgdorferi at the time of enrollment and the other in 51 patients who were seronegative. The patients received either intravenous ceftriaxone, 2 g daily for 30 days, followed by oral doxycycline, 200 mg daily for 60 days, or matching intravenous and oral placebos. Each patient had well-documented, previously treated Lyme disease but had persistent musculoskeletal pain, neurocognitive symptoms, or dysesthesia, often associated with fatigue. The primary outcome measures were improvement on the physical- and mental-health-component summary scales of the Medical Outcomes Study 36-item Short-Form General Health Survey (SF-36)--a scale measuring the health-related quality of life--on day 180 of the study.
RESULTS:
After a planned interim analysis, the data and safety monitoring board recommended that the studies be discontinued because data from the first 107 patients indicated that it was highly unlikely that a significant difference in treatment efficacy between the groups would be observed with the planned full enrollment of 260 patients. Base-line assessments documented severe impairment in the patients' health-related quality of life. In intention-to-treat analyses, there were no significant differences in the outcomes with prolonged antibiotic treatment as compared with placebo. Among the seropositive patients who were treated with antibiotics, there was improvement in the score on the physical-component summary scale of the SF-36, the mental-component summary scale, or both in 37 percent, no change in 29 percent, and worsening in 34 percent; among seropositive patients receiving placebo, there was improvement in 40 percent, no change in 26 percent, and worsening in 34 percent (P=0.96 for the comparison between treatment groups). The results were similar for the seronegative patients.
CONCLUSIONS:
There is considerable impairment of health-related quality of life among patients with persistent symptoms despite previous antibiotic treatment for acute Lyme disease. However, in these two trials, treatment with intravenous and oral antibiotics for 90 days did not improve symptoms more than placebo."

https://www.ncbi.nlm.nih.gov/pubmed/11450676 http://content.nejm.org/cgi/reprint/345/2/85.pdf

He reported his "results" in the July 13, 2001 NEJM. There were numerous aspects of fraud committed in the protocol including using the falsified Dearborn case definition, and that 2/3 of his victims never had ceftriaxone before, yet he claimed he was re-treating with the standard of care at the time, which was 30 days of ceftriaxone. So, those patients, the 2/3ds, were not "re-treated." He also did not report which primers he used to detect NO LYME in the spinal fluid of his victims (this is written up in the new Primers Shell Game report), when in fact, whenever he did find such people, he rejected them

from the study. Not only did he say this in the write up of the report protocol—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 he did not report this.

In 2005 Klempner wrote 2 important reports; one with a man named Kaplan at UConn and another with Gary Wormser. The one with Wormser we already talked about. It was the one where he revealed there were 2 kinds of Lyme: The Dearborn, HLA-linked arthritis in a knee kind,... and the other, the 85%, the neurological, seronegative kind, which we learned about in the new report called "The Lyme Vaccine Scam": The patients with arthritis feel fine except for their arthritis signs. The report with Kaplan, Klempner reported that these people had no neurological compromise and therefore their symptoms were psychiatric:

Neurology. 2003 Jun 24;60(12):1916-22.
Cognitive function in post-treatment Lyme disease: do additional antibiotics help?
Kaplan RF1, Trevino RP, Johnson GM, Levy L, Dornbush R, Hu LT, Evans J, Weinstein A, Schmid CH, Klempner MS.
"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."
https://www.ncbi.nlm.nih.gov/pubmed/12821733

Everyone knows that's false. Cognitive impairment and biomarkers of the central nervous system degradation, even Mark Klempner wrote about and reported extensively. 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), 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:

J Infect Dis. 1998 Feb;177(2):401-8.
Matrix metalloproteinases in the cerebrospinal fluid of patients with Lyme neuroborreliosis.
Perides G1, Charness ME, Tanner LM, Péter O, Satz N, Steere AC, Klempner MS.
"Neurologic manifestations of Lyme disease include meningitis, encephalopathy, and cranial and peripheral neuropathy. There are no sensitive markers for neuroborreliosis, and diagnosis is often based on clinical presentation and cerebrospinal fluid (CSF) abnormalities, including intrathecal antibody production. Matrix metalloproteinase (MMP) activity in CSF was compared in patients with neuroborreliosis, patients with diverse neurologic disorders, and healthy controls. The CSF of 17 of 18 healthy subjects and 33 of 37 patients with neurologic symptoms and normal CSF and imaging studies contained only MMP2. The CSF of several patients with neurologic disorders contained MMP2, MMP9, and gelatinolytic activity at 130 and 250 kDa. 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."
https://www.ncbi.nlm.nih.gov/pubmed/9466528

FULL TEXT: http://www.actionlyme.org/Retro_Klempnerization.htm and

J Infect Dis. 1992 Aug;166(2):440-4.

Fibroblasts protect the Lyme disease spirochete, Borrelia burgdorferi, from ceftriaxone in vitro.

Georgilis K1, Peacocke M, Klempner MS.

*"*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. Since B. burgdorferi first infects skin, the possible protective effect of skin fibroblasts from an antibiotic commonly used to treat Lyme disease, ceftriaxone, was examined. Human foreskin fibroblasts protected B. burgdorferi from the lethal action of a 2-day exposure to ceftriaxone at 1 microgram/mL, 10-20 x MBC. In the absence of fibroblasts, organisms did not survive. Spirochetes were not protected from ceftriaxone by glutaraldehyde-fixed fibroblasts or fibroblast lysate, suggesting that a living cell was required. 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."

https://www.ncbi.nlm.nih.gov/pubmed/1634816 http://actionlyme.org/Mark_Klempner_Fibroblasts.htm

REPEAT: Mark Klempner also wrote in 1998 that anti-OspA antibodies might be the cause of anti-myelin antibodies or probably contributed to the MS form of Lyme. I think 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:
http://actionlyme.org/KFORSCHNER_DISCOVERS_LYME_TOXIN.htm

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So, obviously that guy Klempner is lying about everything. Lyme is incurable and causes nerve and brain degrading enzymes as a marker of this terrible disease... that is not a disease and people are inventing their symptoms?

Next, what are the other biomarkers discovered by the Same-Crooks-Who-Now-Call-Us-Psychiatric or Poisoners-of-Our-Children (Munchausen's, yes, straight up Munchausen's accusations; this was meant for what happened after the fake vaccines were on the market—they intended to blame the parents for poisoning their children should they become sick from the OspA vaccines)??

A) MMP-130 - Klempner as shown above.

B) GFAp, or glial-fibrillary acidic protein - ROBERT SCHOEN, - and this one you are really going to love perhaps even more than Klempner, as you will later see, re what Schoen says to the press about us - found in the CNS as a biomarker of glial cell degradation. Now what is a glial cell?

To surround neurons and hold them in place
To supply nutrients and oxygen to neurons
To insulate one neuron from another
To destroy pathogens and remove dead neurons.

From http://en.wikipedia.org/wiki/Neuroglia

When trying to push the Yale LYMErix vaccine, Schoen mentions this biomarker, when trying to show how devastating Lyme is, and that you'd better get that vaccine (2000, while LYMErix was still on the market), mentioning the destruction of these cells, the sign of which is GFAp in the spinal fluid:

Ann Intern Med. 2000 Apr 18;132(8):661-8.
The Lyme disease vaccine: conception, development, and implementation.
Thanassi WT1, Schoen RT.

"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)."
https://www.ncbi.nlm.nih.gov/pubmed/10766685 http://annals.org/article.aspx?articleid=713400

C) Anti-heat-shock antibodies (Sigal and Barbour, re anti-flagellar antibodies crossreacting):

Cell Mol Neurobiol. 2001 Oct;21(5):477-95.
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.
Sigal LH1, Williams S, Soltys B, Gupta R.

"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, if means you're psychiatric and don't have a real disease :)

D) QEEG or electroencephalograms (**Sigal**, primary Munchausen's accuser)

Clin Electroencephalogr. 1995 Jul;26(3):137-45.
QEEG and evoked potentials in central nervous system Lyme disease.
Chabot RJ1, Sigal LH.

"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 including QEEG 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."
https://www.ncbi.nlm.nih.gov/pubmed/7554300

E) SPECT or brain perfusion scanning (Steere)

Neurology. 1997 Dec;49(6):1661-70.
Reversible cerebral hypoperfusion in Lyme encephalopathy.
Logigian EL1, Johnson KA, Kijewski MF, Kaplan RF, Becker JA, Jones KJ, Garada BM, Holman BL, Steere AC.

"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

Keep in mind that Allen Steere's official position is that Lyme only causes a bad knee and no other symptoms.

F) Antiphospholipid antibodies (Steere and Yale claiming Lyme caused Lupus - 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, we suggest that these antibodies could represent a response to injury in neurological disease or a cross reactive event caused by spirochetes."
https://www.ncbi.nlm.nih.gov/pubmed/8410057
FULL TEXT:
http://www.actionlyme.org/STEERE_AND_LUPUS_LYME.htm

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

Now all of these fellows, remember, say Lyme disease does not cause a disease at all, but is a mental illness similar to a somatoform illness, the definition of which is that you can have valid, scientifically detectable outlier markers of a real illness, but that you don't have a real illness. Somatoform means "magic" or done with your brain like a paranormal event. That is the technical definition; Magic.

Neurology. 1992 Jan;42(1):43-50.
Neuroactive kynurenines in Lyme borreliosis.
Halperin JJ1, Heyes MP.
"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) Lyme Is associated with ALS (Halperin, Dattwyler):

Arch Neurol. 1990 May;47(5):586-94.
Immunologic reactivity against Borrelia burgdorferi in patients with motor neuron disease.
Halperin JJ1, Kaplan GP, Brazinsky S, Tsai TF, Cheng T, Ironside A, Wu P, Delfiner J, Golightly M, Brown RH, et al.
"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 24ALS 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."
https://www.ncbi.nlm.nih.gov/pubmed/2334308
FULL TEXT:
http://www.actionlyme.org/ALSLYME47.htm

Keep in mind that if it is not Borrelia causing all these signs, it would be due to all the secondary opportunistics that take over in post-sepsis syndrome. Meanwhile, the Cabal says all sorts of slanderous and libelous things about neurologic Lyme victims, which is a criminal charge.

I) NO in the brain (Steere):

J Infect Dis. 1994 May;169(5):1014-22.
Borrelia burgdorferi and Escherichia coli lipopolysaccharides induce nitric oxide and interleukin-6 production in cultured rat brain cells.
Tatro JB1, Romero LI, Beasley D, Steere AC, Reichlin S.
http://www.ncbi.nlm.nih.gov/pubmed/7513330

"Lyme Disease" refers to "only the bad knee or arthritis," so brains are not knees unless Steere has finally made that fantastic discovery for which he's always longed.

Nitric Oxide is a free-radical, neurotoxin.

J) Anti-ganglioside antibodies (Benach)

Infect Immun. 1995 Oct;63(10):4130-7.
Experimental immunization with Borrelia burgdorferi induces development of antibodies to ganglio sides.
Garcia-Monco JC1, Seidman RJ, Benach JL.

"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."
https://www.ncbi.nlm.nih.gov/pubmed/7558329
http://iai.asm.org/content/63/10/4130.full.pdf+html?view=long&pmid=7558329

So, follow. "Experimental infection with Borrelia by immunization causes cross reacting antibodies to nerve and brain, but Lyme is just a bad knee." (Benach is not part of the cabal, but one could look to see who cited that report, and do that routinely with these reports.)

K) And Last but Not Least, Paul Duray in IDSA's own journal with the most important biomarker of all !!!! (How can they deny this?)...

1989 (this is in IDSA's own journal): NCI and US Army Ft Detrick Pathologist Paul Duray on the CSF cells looking like "Epstein-Barr-like transformed cells" in IDSA's 1989 Reviews Supplement on Spirochetal Diseases:Rev Infect Dis. 1989 Sep-Oct;11 Suppl 6:S1487-93.

Rev Infect Dis. 1989 Sep-Oct;11 Suppl 6:S1487-93.
Clinical pathologic correlations of Lyme disease.
Duray PH1.

"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

Epstein-Barr like transformed or neoplastic (pre-cancerous) B cells found in the spinal fluid of persons who just have bad knees and no other symptoms. Hmmm.

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1992:

Duray again in 1992, in Steve Schutzer's review of the 1992 Cold Spring Harbor Conference on Lyme:

"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 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 Crooks' Conference, published in Steve Schutzer's ***Lyme Disease: Molecular and Immunologic Approaches***. – book:
Publisher, Cold Spring Harbor Press, 1992, ISBN: 087693770, 978087969377

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2006:

The NIH (NINDS' 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 by saying that Epstein-Barr might be the real culprit. (OspA causes immunosuppression and the reactivation of latent herpes viruses.)

The following journal 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:

J Neuropathol Exp Neurol. 2006 Jun;65(6):540-8.
Borrelia burgdorferi Induces TLR1 and TLR2 in human microglia and peripheral blood monocytes but differentially regulates HLA-class II expression.
Cassiani-Ingoni R1, Cabral ES, Lünemann JD, Garza Z, Magnus T, Gelderblom H, Munson PJ, Marques A, Martin R.

"We found that stimulation with B. burgdorferi lysate increased the expression of Toll-like receptors (TLRs) 1 and 2 in all cell types except neurons. However, despite similarities in global gene profiles of monocytes and microglia, only microglial cells responded to the stimulation with a robust increase in HLA-DR, HLA-DQ, and also coexpressed CD11-c, a dendritic cell marker. In contrast, a large number of HLA-related molecules were repressed at both the RNA and the protein levels in stimulated monocytes, whereas secretion of IL-10 and TNF-alpha was strongly induced. These results show that signaling through TLR1/2 in response to B. burgdorferi can elicit opposite immunoregulatory effects in

blood and in brain immune cells, which could play a role in the different susceptibility of these compartments to infection."
http://www.ncbi.nlm.nih.gov/pubmed/16783164

> So, OspA and spirochetes cause humoral immunosuppression (no antibodies and a result like post-sepsis syndrome), with chronic brain inflammation, says the NIH. And since they are talking about TLR2/1 ligands, that means the triacyl Osps, like OspA. You can't make this up.

This next report by the same people (NINDS' Martin and Marques) 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):

J Infect Dis. 2006 Mar 15;193(6):849-59. Epub 2006 Feb 8.
Borrelia burgdorferi lipoprotein-mediated TLR2 stimulation causes the down-regulation of TLR5 in human monocytes.
Cabral ES1, Gelderblom H, Hornung RL, Munson PJ, Martin R, Marques AR.

"Human monocytes stimulated with TLR5 ligands (including p37 or flaA, the minor protein from B. burgdorferi flagella) up-regulated TLR5. In addition, TLR2 stimulation rendered cells hyporesponsive to a TLR5 agonist. These results indicate that diverse stimuli can cause differential TLR expression, and we hypothesize that these changes may be useful for either the pathogen and/or the host."
http://www.ncbi.nlm.nih.gov/pubmed/16479520

You'll see a lot of these same reports again in the Occam's Razor report, since that is what happened: We saw these outcomes in parallel in other instances of fungal vaccine attempts and other immunosuppression outcomes, and in the end… All Roads Lead to Epstein-Barr, et al. That's a Razor.

One has to remark about how amazing it is that the State of Connecticut and Yale university wanted to throw away all this discovery related to how OspA caused an immunosuppression disease and New Great Imitator outcomes too. They would have been 20 years ahead of the curve in bioscience and discovery. Instead they chose to terrorize their victims in every way imaginable.

2013 – The same NIH MS-Lyme Group as above, Martin and Marques:

"When Lyme Disease Lasts and Lasts" – Jane Brody, *NYTimes.com*

"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/

!!!
Ya think? What causes everything? MS, Lupus, Cancer, etc… ??

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2014:

Washington University at St. Louis (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…

PLoS One. 2014 Jun 11;9(2):e98819. doi: 10.1371/journal.pone.0098819. eCollection 2014.
Reactivation of multiple viruses in patients with sepsis.
Walton AH1, Muenzer JT2, Rasche D1, Boomer JS3, Sato B4, Brownstein BH1, Pachot A5, Brooks TL3, Deych E3, Shannon WD3, Green JM3, Storch GA2, Hotchkiss RS1.

"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]. Experimental therapeutic approaches in sepsis have almost exclusively focused on blocking early inflammation or host-pathogen interaction and failed [8]–[10]. Recently, immuno-adjuvant therapies that boost host immunity, e.g., GM-CSF and interferon-γ, have been successful in small clinical trials thereby supporting the concept that reversing immunosuppression in sepsis is a plausible strategy to improve outcome [11], [12]. However, several issues have limited this approach including lack of consensus that immunosuppression is a clinically important phenomenon [5], [6], [13]. Also, difficulty in identifying patients with impaired immunity as well as determining optimal timing for administration pose significant challenges to pursuing this approach [14]. While immuno-adjuvant therapies might improve sepsis survival if administered during the later immunosuppressive phase, these agents might worsen outcome if given during the early hyper-inflammatory phase [4], [14]. Thus, a means to distinguish these two contrasting phases of sepsis is needed not only to verify the hypothesis that sepsis progresses to an immunosuppressive state but also to guide use of potential agents which boost immunity.
"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]. …"
https://www.ncbi.nlm.nih.gov/pubmed/24919177
http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0098819

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

"*Surviving Sepsis: Detection and Treatment Advances"*
By Carolyn Beans for the National Institutes of Health | August 18, 2014 08:43am ET
"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]" http://www.livescience.com/47387-sepsis-diagnosis-treatment-research-nigms.html

Sepsis Has Long-Term Impact for Older Adults, Study Finds
By Rachael Rettner, Senior Writer | October 26, 2010
"About 60 percent of sepsis patients experienced worsening cognitive or physical function, or both, after their infection, the researchers say… Nearly 17 percent showed signs of moderate to severe cognitive impairment, compared with about 6 percent before the sepsis infection. Patients hospitalized for something other than sepsis did not show an increase in cognitive problems."
http://www.livescience.com/8831-sepsis-long-term-impact-older-adults-study-finds.html

One is allowed to wonder how IDSA gets off saying Lyme has no illness signs other than an autoimmune bad knee,… or else is a somatoform disorder.