4.) The Occam's Razor, or, "the NIH admits Lyme and Chronic Fatigue Syndrome are really Post Sepsis Syndrome," which means "global immunosuppression and ongoing infections of all kinds."

The Shocking But Obvious (introduction):

J Leukoc Biol. 2013 Aug;94(2):291-300. doi: 10.1189/jlb.0812401. Epub 2013 May 21.
IRAK4 kinase activity is not required for induction of endotoxin tolerance but contributes to TLR2-mediated tolerance.
Xiong Y1, Pennini M, Vogel SN, Medvedev AE.

"Development of endotoxin tolerance following the initial "cytokine storm" phase of sepsis is thought to protect the host from an overexuberant immune response and tissue damage but at the same time, may render the host immunocompromised and more susceptible to secondary infection [18,–20]. ...
"Reprogramming [21] of TLR4 signaling in endotoxin-tolerant monocytes and macrophages does not occur as a result of decreased TLR4 expression but involves altered recruitment, tyrosine phosphorylation, and K63-linked polyubiquitination of proximal receptor-adapter-kinase complexes [22,–27] and induction of negative regulators IRAK-M, SHIP1, and A20 [24, 25, 28]. Although a few studies have sought to dissociate kinase and adapter functions of IRAK4 in IL-1R/TLR signaling, albeit with conflicting results [13,–16, 29,–31], it is unclear how IRAK4 kinase activity affects induction of TLR2 and TLR4 homo- and heterotolerance. To address these questions, we used **IRAK4KDKI mice to determine the impact of kinase deficiency of IRAK4 on the induction of TLR tolerance. Our data showed comparable induction of endotoxin tolerance in WT or IRAK4KDKI PMs and BMDMs, as judged by attenuated MAPK phosphorylation, inhibited expression of proinflammatory cytokines and chemokines, and up-regulation of negative TLR regulators, A20 and IRAK-M. Notably, IRAK4 kinase activity was found to be a prerequisite for conferring inhibition of LPS-inducible JNK and p38 MAPK activation following prior exposure to Pam3Cys.** These results represent the first systematic analyses of the role of IRAK4 kinase activity in TLR homo- and heterotolerance and pave the way for improved understanding of how IRAK4 kinase dysregulation may underlie immunocompromised states in late sepsis."
https://www.ncbi.nlm.nih.gov/pubmed/23695305 *http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3714565/*

J Immunol. 2012 Feb 1;188(3):1019-26. doi: 10.4049/jimmunol.1102181. Epub 2012 Jan 6.
TLR2 signaling depletes IRAK1 and inhibits induction of type I IFN by TLR7/9.
Liu YC1, Simmons DP, Li X, Abbott DW, Boom WH, Harding CV.

Pathogens may signal through multiple TLRs with synergistic or antagonistic effects on the induction of cytokines, including type I IFN (IFN-I). IFN-I is typically induced by TLR9, but not TLR2. Moreover, we previously reported that TLR2 signaling by Mycobacterium tuberculosis or other TLR2 agonists inhibited TLR9 induction of IFN-I and IFN-I-dependent MHC-I Ag cross processing. The current studies revealed that lipopeptide-induced TLR2 signaling inhibited induction of first-wave IFN-α and IFN-β mRNA by TLR9, whereas induction of second-wave IFN-I mRNA was not inhibited. TLR2 also inhibited induction of IFN-I by TLR7, another MyD88-dependent IFN-I-inducing receptor, but did not inhibit IFN-I induction by TLR3 or TLR4 (both Toll/IL-1R domain-containing adapter-inducing IFN-β dependent, MyD88 independent). The inhibitory effect of TLR2 was not dependent on new protein synthesis or

intercellular signaling. IL-1R-associated kinase 1 (IRAK1) was depleted rapidly (within 10 min) by TLR2 agonist, but not until later (e.g., 2 h) by TLR9 agonist. Because IRAK1 is required for TLR7/9-induced IFN-I production, **we propose that TLR2 signaling induces rapid depletion of IRAK1, which impairs IFN-I induction by TLR7/9. This novel mechanism, whereby TLR2 inhibits IFN-I induction by TLR7/9, may shape immune responses to microbes that express ligands for both TLR2 and TLR7/TLR9, or responses to bacteria/virus coinfection.**
https://www.ncbi.nlm.nih.gov/pubmed/22227568

Let's just cut right to the quick and show (above) that OspA causes endotoxin tolerance (and cross tolerance to LPS or TLR4-agonists, as well as TLR7/9 agonists, or vial infections) or post-septic shock (host rendered incompetent to "secondary infections") as you've just seen. And there are plenty of other examples in the literature that show Pam3Cys or OspA is a fungal toxin, TLR2/1 agonist. As usual, we recommend you use those links and "See Related," or "Cited By" on PubMed.

We even in December 2016 saw Allen Steere and Gary Wormser saying so. And that was *the third time* Gary Wormser reported that OspA caused immunosuppression rather than "was a vaccine":

New, December 2016:
Lyme Cabal members Gary Wormser and Allen Steere - and even the "CDC officer" criminal Paul Mead - finally admit Late Lyme and LYMErix diseases are immunosuppression outcomes.
'Say the "TLR2/1 agonism" (immunosuppression) is probably the "more important" driver of the disease outcome.

Nat Rev Dis Primers. 2016 Dec 15;2:16090. doi: 10.1038/nrdp.2016.90.
Lyme borreliosis.
Steere AC1,2, Strle F3, Wormser GP4, Hu LT5, Branda JA6, Hovius JW7, Li X8, Mead PS9.

in one TLR or adaptor does not diminish inflammation during infection in animals, and might even result in increased inflammation, as observed in mice deficient in the TLR components TLR2, MYD88, TIR domain-containing adapter molecule 1 (TRIF) or CD14 (REFS 66,67,73,76). This finding suggests that there is redundancy in the ability of the innate immune system to recognize *B. burgdorferi* and/or that these components can activate pathways that produce anti-inflammatory cytokines, such as IL-10. During later stages of infection — namely, stage 2 (in humans known as early disseminated infection that is manifested by inflammation at multiple sites) and stage 3 (in humans known as late infection, typically involving arthritis in the United States) — the anti-inflammatory effects might be the more important function of TLR signalling[79,80]. In

https://www.ncbi.nlm.nih.gov/pubmed/27976670

”This finding suggests that there is redundancy in the ability of the innate immune system to recognize B. burgdorferi and/or that these components can activate pathways that produce **anti-inflammatory cytokines, … - the anti-inflammatory effects might be the more important function of TLR signaling.”**
http://www.actionlyme.org/Steere_Wormser_Admit_Immunosuppression.2016_Dec_15.pdf

And that is what we are here to show you ☺ OspA never could have been a vaccine because it was a fungal toxin that is handled by TLRs 2 and 1. It’s triacylated and therefore much more toxic than even lipopolysaccharide (LPS, a TLR4 agonist).

Mario Philipp at Tulane has long been the scientist who says Borrelia and OspA cause immunosuppression via IL-10, so look at all of his reports:
https://www.ncbi.nlm.nih.gov/pubmed/?term=Philipp+and+borrelia

Ref 79, above, (Mario Philipp):

Infect Immun. 2006 Oct;74(10):5780-9.
Interleukin-10 anti-inflammatory response to Borrelia burgdorferi, the agent of Lyme disease: a possible role for suppressors of cytokine signaling 1 and 3.
Dennis VA1, Jefferson A, Singh SR, Ganapamo F, Philipp MT.
https://www.ncbi.nlm.nih.gov/pubmed/16988256

The Other Two Times Gary Wormser reported that OspA caused immunosuppresson:

Arthritis Rheum. 2012 May;64(5):1311-5. doi: 10.1002/art.34386.
The Toll of a TLR1 polymorphism in lyme disease: a tale of mice and men.
Sellati TJ, Sahay B, Wormser GP.

Association of clinical manifestations of Lyme disease with host immune response to infection

Although certain aspects of the pathogenesis of Lyme disease remain ill-defined, it is generally accepted that clinical manifestations result primarily, perhaps entirely, from the host's immune response to the spirochetes in infected tissue (5–7). Coordinate signaling through pattern-recognition receptors, such as CD14 and Toll-like receptor 2 (TLR-2), expressed on professional phagocytes (e.g., macrophages and neutrophils) and other innate immune cells orchestrate both the initiation and the resolution of inflammatory responses to *B burgdorferi*.

During natural infection, initiation of the host response begins with CD14 recognition of triacylated borrelial lipoproteins and subsequent activation of TLR-2 in partnership with TLR-1 (5–7). Such bacterial recognition activates the NF-κB, phosphatidylinositol 3-kinase/Akt, and p38 MAPK pathways. The ensuing signaling cascades initiate inflammation-associated gene activities responsible for host defense, as well as negative regulatory pathways intended to mitigate the severity and duration of the inflammatory response to spirochetes. The latter goal is achieved, in part, through the action of p38 MAPK–induced suppressors of cytokine signaling (SOCS), which down-regulate inflammation by targeting various points in the NF-κB pathway (7).

https://www.ncbi.nlm.nih.gov/pubmed/22246662

"…negative regulatory pathways intended to mitigate the severity and duration of the inflammation" means exactly post-septic shock response with long term immunosuppression afterwards.

And of course, Wormser's ever infamous inverse-of-an-OspA-dog-vaccine attempt in 2000:

FEMS Immunol Med Microbiol. 2000 Jul;28(3):193-6.
Modulation of lymphocyte proliferative responses by a canine Lyme disease vaccine of recombinant outer surface protein A (OspA).
Chiao JW1, Villalon P, Schwartz I, Wormser GP.

"The modulation of human lymphocyte proliferative responses was demonstrated with a recombinant outer surface protein A (OspA) vaccine preparation for the prevention of Borrelia burgdorferi infection. After exposure to either the unaltered vaccine preparation or OspA prepared in saline, normal lymphocyte responses to the mitogens concanavalin A, phytohemagglutinin-M or pokeweed mitogen, or the antigen BCG were consistently reduced. Whole cell extracts of B. burgdorferi also modulated immune responses but required a much greater quantity of protein than needed for the OspA preparation. The magnitude of modulation was directly dependent on the quantity of OspA. **OspA interferes with the response of lymphocytes to proliferative stimuli including a blocking of cell cycle phase progression**. Future

studies designed to delete the particular region or component of the OspA molecule responsible for this effect may lead to improved vaccine preparations."
https://www.ncbi.nlm.nih.gov/pubmed/10865170

> You know what they say about scholars and true academics: Sometimes they're really no good at practical applications because their heads all wrapped up in theory. But these clowns are neither. We just thought we'd mention it because it's a thing.

So, why is this important? Because the falsified case definition, Dearborn, was designed around passing off a bogus vaccine such as to claim "Lyme disease" was only an HLA-linked hypersensitivity response, limited to an arthritis in a joint so they could sell OspA as a vaccine,… which would then be the cornerstone of their intended monopoly ("enterprise") on testing and vaccines for VBDs. Everything the Cabal does revolves around **maintaining the PRETENSE that Dearborn was real and not a crime scene**, including issuing "guidelines" and other reports about Lyme based on the Dearborn definition.

Falsifying the case definition at Dearborn happened to pass off the bogus OspA vaccines. The triacyl Osps are fungal toxins, and the Cabal knew there would problems with OspA vaccines causing the same "multi-system disease" (Persing and Schoen) that we know of as Late Chronic Neurologic Lyme (really, post-sepsis syndrome) by 1993 from the early Phase I and Phase II trials (Barbour and Fish). Yet here we find 3 times Gary Wormser published that OspA or the Osps, being triacyl lipoproteins, cause instead, immunosuppression. If the vaccine is a lie, surely the testing designed around it was. We know that anyway from what the contributors to the Dearborn conference said about its accuracy, which was an average of 15%, or that the Dearborn Two-Tiered Testing criteria missed 85% of the cases. In order to prosecute, you have to show the perpetrators knew all of this was a lie. OspA was never a vaccine, and the Dearborn case definition is not real. And as you have already seen, Allen Steere knew that people with neurologic, chronic Lyme don't make antibodies against the Osps (published that in 1993, same year he falsified the testing), so *that* majority, the majority of people in the world who don't have the genetic background for a pre-disposition to Rheumatoid Arthritis (don't have the RA HLAs), wont test positive, especially not to OspA, their first vaccine choice.

They were ***conforming*** a disease around an intended vaccines-and-test-kits enterprise, the ALDF.com, but most VBDs are bearer of fungal antigens and most humans can't handle those. Fungal antigens are seen by most human immune systems as toxins. Being straight up evil, the Cabal decided to ram this "Dearborn and OspA" thing through anyway, by trashing their victims. It almost worked. But in the end it seriously backfired because now the USA has an integrity problem in addition to being behind the game with all these Great Imitator disease outcomes like cancer.

This Occam's Razor report contains many proofs that the Health and Human Services (HHS.gov) knows what Lyme and Chronic Fatigue Syndrome are. We chose the term "Occam's Razor" for this section of the Cryme-ology due to all the decades-long chatter in the self-help groups that Chronic Fatigue Syndrome was due to some mysterious, unknown virus.

Eight 8 million people in the USA have Fibromyalgia (says the NIH) and 4 million have Chronic Fatigue Syndrome (CFIDS, says the NIH) and for decades the Lyme Cabal said non-Dearborn Lyme was CFIDS and Fibro, ... and if OspA caused the same immunosuppression/AIDS-like outcome as Chronic Lyme as the Cabal members themselves claimed, ... and if the commonest thing reactivated in immunosupression is Epstein-Barr and its brothers, Cytomegalovirus, HHV-6, as well as Coxsackie (Foot-and-Mouth Disease), etc., … and if the NIH had a "Lyme and MS group, " at the National Institute of Neuroological Disorders and Stroke (NINDS) and who now say Lyme and LYMErix activated EBV (or whatever we're thinking is the combination of herpes viruses that are responsible for MS) via immunosuppression,.... and who say that OspA vaccination alone causes the exact same disease as Chronic Lyme, Chronic Fatigue Syndrome and Fibromyalgia, … and if the NIH now says post-sepsis syndrome is characterized by reactivated EBV and CMV, etc., ... then Chronic Lyme, Chronic Fatigue, and Fibromyalgia are probably not due to some mysterious, unknown virus.

They know what it is. It's something common, and not extraordinary. It's something that happens in other cases of immunosuppression such as with Humira and Stelara, transplant victims who receive immune suppressing drugs, it happens when people have Malaria plus latent Epstein-Barr (Burkitt's Lymphoma) it happens in experiments with other fungal antigen vaccines or vaccine-ish experiments such as Tuberculosis or with the fungal lipoproteins from Brucella, and it happens when perhaps children are injected with a live attenuated virus vaccine that is contaminated with mycoplasma or some other fungal antigens. It happens when astronauts and medical school students are stressed from strange hours and their environment, this reactivation of latent herpes viruses. It is a well-known thing, and that is why it is ignored. But when this immunosuppression from fungal antigens occurs via tick bites or ridiculous choices for vaccines, such as the triacyl lipoprotein "vaccine," LYMErix or OspA, these same government employees trash and harass their victims - which is a Deprivation of Rights via Color of Law charge -, because then we are barred from access to real healthcare, being labeled "psychiatric."

"Medicine" defaulted. No "doctors" were involved in this discovery. They have left a power and authority vacuum. Therefore, we, SASH, are taking over medical science and science reporting, because Simple Things are Big Data. As you have seen, we present a new style of science reporting where the references are built into the text rather than footnoted so you can follow the crimes and fraud exactly.

"Science," "doctors" and the "journals" all defaulted. There are 30 million people in the United States alone with Fibromyalgia, Lyme, chronic fatigue syndrome, etc., …and all we get is the various explanations that involve VooDoo witch magic with the self-backfiring incantations ("somatoform"), etcetera etcetera nonsense.

The recently-former Director of the National Institute of Mental Health, Thomas Insel, said the following about psychiatry (it is not valid, it's just a religion or a belief system):

"The goal of this new manual, as with all previous editions, is to provide a common language for describing psychopathology. While DSM has been described as a "Bible" for the field, it is, at best, a dictionary, creating a set of labels and defining each. The strength of each of the editions of DSM has been "reliability" – each edition has ensured that clinicians use the same terms in the same ways. **The weakness is its lack of validity.** Unlike our definitions of ischemic heart disease, lymphoma, or AIDS, the DSM diagnoses are based on a consensus about clusters of clinical symptoms, not any objective laboratory measure. In the rest of medicine, this would be equivalent to creating diagnostic systems based on the nature of chest pain or the quality of fever. Indeed, symptom-based diagnosis, once common in other areas of medicine, has been largely replaced in the past half century as we have understood that

symptoms alone rarely indicate the best choice of treatment. .. Patients with mental disorders deserve better..."
http://www.nimh.nih.gov/about/director/2013/transforming-diagnosis.shtml

Richard Horton, editor of Lancet:

"The case against science is straightforward: much of the scientific literature, perhaps half, may simply be untrue," Dr. Horton commented in The Lancet.
http://newswire.net/newsroom/news/00088806-world-s-top-scientists-agree-most-researches-findings-are-fraud.html

We take what the editor of the Lancet and Thomas Insel say to be true. Medical science today is just too much malarkey and mentally incompetent. Consider the New Great Imitator. That's a lot of diseases under one umbrella. Multiple Sclerosis, Fibromyalgia, Lupus, **Chronic Fatigue Syndrome**, Dementia, Rheumatoid Arthritis, CANCER, Stroke (BTW, LYMErix also caused strokes and "vascular events" also), ALS, … and ***"after ~~30~~ 40 years we have nothing,"*** – Willy Burgdorfer in the "Under Our Skin," movie.

Someone assigned Allen Steere to it. No one knows why. Perhaps someone at the CDC detected he was unhappy with medicine as a profession – after all, it was one he chose to avoid the VietNam draft – and that he also had a severe case of myopia. At the 1998 FDA meeting on LYMErix, Dattwyler said of Steere's Bad Knees Disease:

DR. DATTWYLER: "I see a lot of patients, and I must say that treatment resistance lyme arthritis in our center is low, it is very rare. **We see maybe one case a year**. And, you know, that is using very strict criteria, saying that the person had, you know, **CDC criteria for seropositivity**, good history, and **usually is monoarticulate knee arthritis."**
http://www.fda.gov/ohrms/dockets/ac/01/transcripts/3680t2.rtf

Dattwyler sees about uno cases a year. There aren't very many Dearborn, CDC, 2-tiered positive cases of "Lyme disease." There never were. It was never about arthritis. Neither the disease nor the OspA vaccine trial results were ever really about arthritis. What "Lyme disease" is really about (and LYMErix too), is much, much worse.

FORTY years down the drain. No one is getting better thanks to too much research fraud, and downright stupidity (definition: willful ignorance) and a quack squad of various tooty-frooty "treatment" flavors. We can't believe the science. We can't believe how science is reported. *We can't believe* the FDA never looked at the Dearborn case definition (they told us so). This is the reason Senator Richard Blumenthal and company had to have the Office of Budget and Management ***ORDER*** the FDA to assure the Lyme testing is valid according to the FDA's rules on the validation of an analytical method.
http://www.fda.gov/downloads/MedicalDevices/ProductsandMedicalProcedures/InVitroDiagnostics/ucm407409.pdf

The first criterion in a validation is ACCURACY, or, *"does your method detect 100% of the samples where the analyte in question is known to be present?* (and then give the range of what % of the known analyte your method found, but the first premise is that is should detect SOME of the analyte if it is

known to be there)." If it can't be 100% for anti-flagellar antibody cases (it is about 95% present in known, erythema migrans sampled, DNA for Borrelia, cases), it should be close. And for this reason, to increase SENSITIVITY (or "how low in concentration of the analyte in question can your method detect"), this problem was addressed in 1992 by the late Lou Magnarelli and the people who own the patent for the only FDA-valid test for Lyme – and who are also the owners of the LYMErix patent, Yale's Fikrig and Flavell:

J Clin Microbiol. 1992 Dec;30(12):3158-62.
Comparison of whole-cell antibodies and an antigenic flagellar epitope of Borrelia burgdorferi in serologic tests for diagnosis of Lyme borreliosis.
Magnarelli LA1, Fikrig E, Berland R, Anderson JF, Flavell RA.
"A recombinant protein (p41-G) of an antigenic region of flagellin was used in a standard and amplified enzyme-linked immunosorbent assay (ELISA) to detect antibodies to Borrelia burgdorferi, the causative agent of Lyme borreliosis. Comparable sensitivities (88 to 94%) were noted when sera from 17 persons who had erythema migrans and antibodies to whole-cell B. burgdorferi were tested against the p41-G antigen. In tests of a second study group of 36 persons who had erythema migrans but no detectable antibodies to whole-cell B. burgdorferi, 3 (8%) were positive when the p41-G antigen was used. Assay specificity likewise increased when the p41-G fragment was included in an ELISA with human sera containing treponemal antibodies. Recombinant flagellar proteins of B. burgdorferi, such as the p41-G antigen, can be used in an ELISA and may help confirm Lyme borreliosis during early stages of infection and improve specificity. http://www.ncbi.nlm.nih.gov/pubmed/1280650

Fikrig, Magnarelli and Flavell basically said, "Here we have made the common anti-flagellar antibody (found in most Lyme patients – and the ONLY specific biomarker for *Borrelia burgdorferi*, thus meeting 2 FDA validations requirements, accuracy and specificity) not only SPECIFIC (FDA validation criterion – does not detect anything else besides Borrelia burgdorferi flagellin) but even more useful by adding in or spiking it into an ELISA made of borrelia sonicate, *BECAUSE* … some people don't even make a lot of anti-flagellar antibodies, the one most people make if they make any at all. And if one wanted to go nano tech, the thing to do is put these sorts of fragments of Borrelia(s)-specific flagellins on nanotubes and look for these specific antibodies in human blood, since antigen itself is less likely to be there. Borreliae like to live in the brain and lymph nodes."

That was 1991 and 1992. Fikrig and Flavell own (patented) that test (US # 5,618,533). They own the LYMErix patent,… and they own this method, the only FDA-valid test to assess it. But they very clearly did not use a valid test to assess LYMErix. We know why.

In the late 1980s and early 1990s many researchers were looking for ways to use the anti-flagellar antibody as the primary means of diagnosis. Detecting anti-flagellar antibodies was common idea at the time. It's a valid approach because most people with Lyme are known to at least have the flagellar antibody. Here is the report from 1991 that goes with the Yale flagellin method patent (5,618,533):

Infect Immun. 1991 Oct;59(10):3531-5.
Molecular characterization of the humoral response to the 41-kilodalton flagellar antigen of Borrelia burgdorferi, the Lyme disease agent.
Berland R1, Fikrig E, Rahn D, Hardin J, Flavell RA.
"The earliest humoral response in patients infected with Borrelia burgdorferi, the agent of Lyme disease, is directed against the spirochete's 41-kDa flagellar antigen. In order to map the epitopes recognized on this antigen, 11 overlapping fragments spanning the flagellin gene were cloned by polymerase chain reaction and inserted into an Escherichia coli expression vector which directed their expression as fusion proteins containing glutathione S-transferase at the N terminus and a flagellin fragment at the C terminus.

Affinity-purified fusion proteins were assayed for reactivity on Western blots (immunoblots) with sera from patients with late-stage Lyme disease. **The same immunodominant domain was bound by sera from 17 of 18 patients. This domain (comprising amino acids 197 to 241) does not share significant homology with other bacterial flagellins** and therefore may be useful in serological testing for Lyme disease."
https://www.ncbi.nlm.nih.gov/pubmed/1894359 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC258917/

Yale did not use their valid test, above, to assess for the efficacy of LYMErix because it was known by 1993 that Lyme was causing a disease like chronic neurologic Lyme, and therefore was not "safe" or efficacious.

The NIH's Lyme-And-MS Division of NINDS found and reported in 2006 that exposure to the fungal antigens exported by Borrelia like OspA (blebbing) can turn off the function of the TLR5 receptor that handles flagellin as well as tolerizes to other fungal antigens (or TLR2/1 agonists) as you have previously seen and we'll reference again. Because TLR2/1 agonism seems to cause cross-tolerance to other TLRs-agonists, this could be the reason some Lyme victims are totally seronegative (no antibodies against Lyme at all).

IMPORTANTLY, these two, Martin and Marques at the NIH's NINDS' Lyme and MS Division (note, there was a Lyme and MS Division, and not a Lyme and RA Division), **were specifically tasked to discover what on Borrelia caused cross reacting antibodies or T cells against myelin** (the definition of MS). What they found was nothing. They found that Lyme and OspA caused MS via immunosuppression in the body (humoral) with chronic brain inflammation, and hypothesized that this could be due to the reactivation of EBV and others in the brain (NYTimes, Jane Brody article, below).

Here are the 2 reports by this MS-Lyme group that say OspA is responsible for causing nearly complete immunosuppression, in the end:

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.
"The spirochete Borrelia burgdorferi is the agent of Lyme disease, which causes central nervous system manifestations in up to 20% of patients. We investigated the response of human brain microglial cells, glial progenitors, neurons, astrocytes, as well as peripheral blood monocytes to stimulation with B. burgdorferi. We used oligoarrays to detect changes in the expression of genes important for shaping adaptive and innate immune responses. 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."
https://www.ncbi.nlm.nih.gov/pubmed/16783164

And

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.

"Toll-like receptors (TLRs) trigger innate immune responses via the recognition of conserved pathogen-associated molecular patterns. Lipoproteins from Borrelia burgdorferi, the agent of Lyme disease, activate inflammatory cells through TLR2 and TLR1. We show that stimulation of human monocytes with B. burgdorferi lysate, lipidated outer surface protein A, and triacylated lipopeptide Pam3CysSerLys4 results in the up-regulation of both TLR2 and TLR1 but the down-regulation of TLR5, the receptor for bacterial flagellin, and that this effect is mediated via TLR2. TLR4 stimulation had no effect on TLR2, TLR1, and TLR5 expression. 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.
https://www.ncbi.nlm.nih.gov/pubmed/16479520

Recall from the DNA Shell Game and Biomarkers charge sheets that the bogus Klempner long term non-retreatment study where 2/3rds of his victims had never had IV ceftriaxione before - the standard of care at the time -, and which was assessed with the non-scientifically valid FIQ or Fibromyalgia Impact Questionaire ("questionaires" or "check lists" mean psychiatry is the dominant assessment criteria for a real medical illness), when the IDSA/CDC Lyme crooks were the authors of all the scientifically valid physiological biomarkers of brain and CNS destruction, was based on the inclusion/exclusion criteria of the fraudulent Dearborn case definition, rendering the entire study invalid. Yet, this Klempner report is the basis of the IDSA 2001 and 2006 "guidelines" on the non-diagnosis and non-treatment of Lyme disease. Therefore once the fraud of the Dearborn event is prosecuted, out go all of IDSA's "guidelines."

The Silly IDSA "Guidelines" = brainscramble and nonsense (one can cross-apply, probably all the rest of IDSA's "Guidelines" on other diseases are brainscramble and silly nonsense, too):

Clin Infect Dis. 2006 Nov 1;43(9):1089-134. Epub 2006 Oct 2.
The Clinical Assessment, Treatment, and Prevention of Lyme Disease, Human Granulocytic Anaplasmosis, and Babesiosis: Clinical Practice Guidelines by the Infectious Diseases Society of America
Gary P. Wormser, Raymond J. Dattwyler, Eugene D. Shapiro, John J. Halperin, Allen C. Steere , Mark S. Klempner, Peter J. Krause, Johan S. Bakken, Franc Strle, Gerold Stanek, Linda Bockenstedt, Durland Fish, J. Stephen Dumler, Robert B. Nadelman
https://www.ncbi.nlm.nih.gov/pubmed/17029130 http://cid.oxfordjournals.org/content/43/9/1089.long

Go ahead and read ^^^ that for all the ridiculousness and false statements they make and in which they repeatedly quote their own previous research fraud. This is called a circle jerk in the common vernacular you're welcome but we all know it.

Mark Klempner, himself, found ceftriaxone did not kill all the spirochetes even when there weren't human cells to hide inside:

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."**
http://www.ncbi.nlm.nih.gov/pubmed/1634816

Spirochetal diseases are not curable, and spirochetal infections are un-eradicable. But the disease, the illness, is caused by the immune damage by spirochetes invading the lymph nodes, destroying the B cell germinal centers (Baumgarth and Barthold), as well as the shed fungal antigens on the blebs render the immune system totally inert. This is like AIDS, or an acquired immune deficiency. It's called post-sepsis syndrome.

As you will see later in this report (G., below) Mark Klempner and Gary Wormser re-state that there are 2 kinds of Lyme: the HLA-linked hypersensitivity "one case a year" bad-knee only, and everyone else, the 85% left out of the Dearborn case definition - the definition that includes the Triad of Fatigue, Musculoskeletal signs, and Neurocognitive deficits -, all well known long term outcomes of Sepsis.

Since the Dearborn "case definition" only describes and refers to the HLA-linked, arthritis associated "monoarticular arthritis and no other illness signs," the "guidelines" only apply to people with that genetic background. **The guidelines are actually a form of racial discrimination**. Only the people with the HLAs for arthritis are allowed to have a "disease." The rest of us are slandered and libeled (see "Deprivation of Rights under Color of Law").

Again, the current, 1994, CDC falsified, Dearborn case definition:
http://www.cdc.gov/mmwr/preview/mmwrhtml/00038469.htm

"It was recommended that an IgM immunoblot be considered positive if two of the following three bands are present: 24 kDa (OspC)*, 39 kDa (BmpA), and 41 kDa (Fla) (1).
"It was further recommended that an IgG immunoblot be considered positive if five of the following 10 bands are present: 18 kDa, 21 kDa (OspC)*, 28 kDa, 30 kDa, 39 kDa (BmpA), 41 kDa (Fla), 45 kDa, 58 kDa (not GroEL), 66 kDa, and 93 kDa (2)."

This ridiculous research-fraud diagnostic standard, Dearborn, requires a person to first have a positive ELISA, which is a screening test that only allows the late Lyme arthritis, autoimmune, HLA-hypersensitivity cases to be detected.

This is research fraud - How Steere falsified the testing in Europe, excluding all but late Lyme arthritis:

J Infect Dis. 1994 Feb;169(2):313-8.

Antibody responses to the three genomic groups of Borrelia burgdorferi in European Lyme borreliosis.

Dressler F1, Ackermann R, Steere AC.

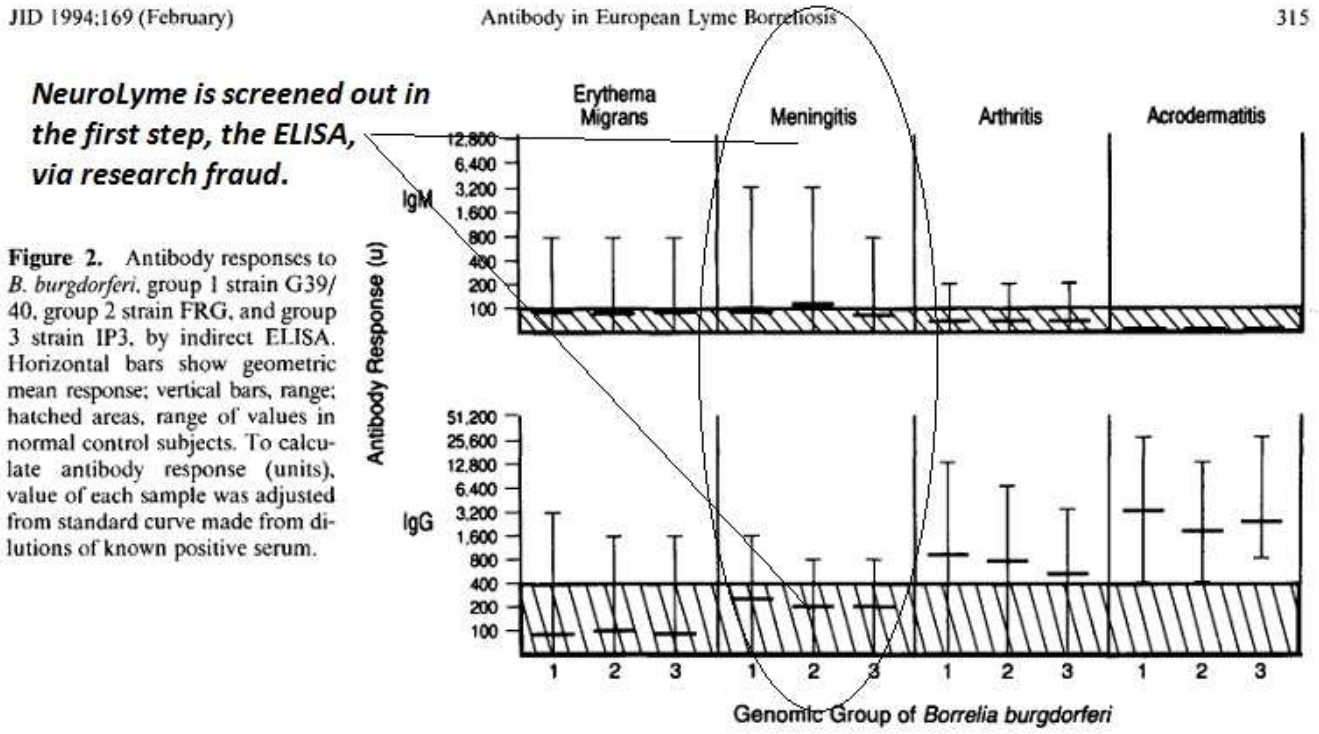


Figure 2. Antibody responses to *B. burgdorferi*, group 1 strain G39/40, group 2 strain FRG, and group 3 strain IP3, by indirect ELISA. Horizontal bars show geometric mean response; vertical bars, range; hatched areas, range of values in normal control subjects. To calculate antibody response (units), value of each sample was adjusted from standard curve made from dilutions of known positive serum.

https://www.ncbi.nlm.nih.gov/pubmed/8106763

Additionally, again: **Steere used high passage strains** (which drop plasmids, and which is why spirochetes become less virulent over time, if they are not pharmed back into multiple-pathogen-infected ticks periodically) with recombinant OspA and B without the lipids attached. If the lipids are not attached, this is barely an antigen and not likely to produce antibodies. Hence, OspA and B, bands 31 and 34 were excluded from the Dearborn case definition. This was what K. Dickson told the FDA when she blew the whistle: Although you may have 5 bands, if one or more of them are OspA and B, you don't have a "case" of Lyme, even though supposedly OspA and B are so specific they made vaccines out of them: http://www.fda.gov/ohrms/dockets/ac/01/slides/3680s2_11.pdf

OspA is specific enough to prevent Lyme, they say, but not specific enough to diagnose?

So, while Mark Klempner said at one time that Lyme was incurable due to intracellular spirochetes, now, in the "guidelines" he says there is no such thing as neurologic Lyme. The reason these criminals do not want anyone diagnosed with Lyme disease, in whatever form, is because antibiotics don't cure it. It is an AIDS-like disease, with reactivated viral infections, and most accurately called Post Sepsis syndrome or Endotoxin Tolerance,... with the multiple herpes virus reactivation, fungal antigen tolerance and B cell

changes that are like mutations or pre-cancerous.

> *The FIRST and MAIN REASON*, for this Lyme-fraud-in-perpetuity, is that the LYMErix or OspA vaccines caused the same ***Post-Sepsis Syndrome, or Endoxin Tolerance or AIDS-like disease*** - with the Chronic Fatigue Syndrome (Yale and Steere) or/and Fibromyalgia (Steere) being predominant features -, being a worse fungal toxin for humans than lipopolysaccharide or LPS (TLR4 agonist) and they lied about this to the FDA and to the public and in the journals.
> The *second reason* is that the mechanisms of illness in Lyme and CFIDS betray the mechanisms of the ***Autism pandemic***.

There are other examples of research fraud in this report perpetrated by CDC officers, particularly Suzanne Vernon, as you will see. A "stealth disabler" would have the same definition: no antibodies, or makers of classic "inflammation," or allergy or hypersensitivity or "autommunity" (they all basically mean the same thing). If you wanted to create a biowewapon against a certain racial population, you would look to see if there are low- to no- genetic HLA links to a hypersensitivity response in that population.

This scam is GAME OVER at this point; all that remains are the prosecutions.

You will see many times in this report, that OspA never could have been a vaccine – which is the entire point, really, of this report. It was the complete opposite. It was a fungal toxin that caused generalized immunosuppression. You will see that spirochetes and Epstein Barr hang out together in the lymph nodes. You will see that OspA, spirochetes shedding OspA, and Epstein-Barr inhibit apoptosis. That seems to be the first step in all dysimmunity outcomes: Inhibition of Apoptosis of an infected cell.

The Cabal has done nothing besides attack their victims since the early 1990s in order to maintain the *PRETENSE* - a false position (that being that Dearborn was real, that "Steere in Europe" falsifying the testing was not research fraud, that OspA "vaccines" were not research fraud) - that the Dearborn case definition of "Lyme is just a bad knee with no other illness signs," was ***real and not a crime scene*** because they do not want to go to jail.

As an aside, maybe we should say this now: If the treatment fits the model the science presents, Rituximab, with its 2/3rds' cure rate in Chronic Fatigue Syndrome, it must be a pretty close model.

You've heard of an Occam's Razor by now. It's the principle that the simplest explanation is usually the correct one. 'If you hear hoofbeats, think 'horses,' not 'zebras.'" "If it quacks like a duck…etc." Don't overthink this stuff. It's all there. You just have to look at the big picture.

I. Start with the most compelling data; Yale/CDC Lyme perps did a 180 on everything (Much of this you have already seen).

1) The CDC, IDSA and Yale claim that only the HLA linked arthritis cases were allowed to be called "Lyme disease." This is the Dearborn, 1994 but current "case definition." The 2005 Klempner and

Wormser HLA report re-stated that the case definition was HLA-linked and the victims had no other illness signs but arthritis. So, that's the only "case" of Lyme one is allowed to have. It means you may have arthritis, only; an HLA-linked hypersensitivity response with lots of antibodies, and no fatigue or meningitis or anything else. The other symptom set people, the non-HLA linked people, well, that's a mystery, right? Must be psychiatric.

2) But this definition came after the same people claimed Lyme caused everything (MS, Lupus, ALS, dementia, stroke, Chronic Fatigue Syndrome, Fibromyalgia, etc.), particularly that chronic neurologic Lyme was incurable in half the cases (Dattwyler, Luft, Sigal, Steere, in 1989 IDSA Review special supplement on Lyme and Spirochetal diseases), and that spirochetal diseases were incurable, even with ceftriaxone, even when there were no human cells for the spirochetes to hide in (Klempner, 1992).

3) At that time, in the 1989 IDSA Reviews, a one Paul Duray, pathologist for Yale, the US Army, the National Cancer Institute, etc, found that the immune cells in the spinal fluid of chronic neurologic looked like immature-, and mutated, or neoplastic or EBV-transformed. Look those words up, "EBV-transformed" or "EBV-immortalized" cells is a known thing and very relevant to the OspA crime. CAUTION: If the reader is not familiar with what "EBV transformed" means, please study the topic and do not make assumptions based on no background. Ever.

4) The OspA vaccine victims were acquiring the same "multi-system," (Dave Persing), "protean" (Ben Luft) disease manifestations that the Cabal threw out of the case definition at Dearborn. The Chronic Fatigue Syndrome, the Fibromyalgia, the chronic systemic disease with dementia signs and neurological signs, etc,. co-definitions or known (Great Imitator) were outcomes thrown out of the vaccine trial results and described as "Unconfirmed Lyme." Those cases were not counted as vaccine failures.

5) Never lose track of this statement by U.S. Department of Energy and likely a physician at SUNY-StonyBrook:

"It's the perfect stealth bacteria, says one frustrated physician. He's talking about Borrelia burgdorferi, the bacterium that causes Lyme disease. This illness, which is often mistaken for diseases ranging from multiple sclerosis to Lupus, can inflict excruciating headaches and muscle pain, affect the brain and nervous system, attack major organs, and inflame joints..."-- Energy Science News, pnl.gov

MS and Lupus are not "solely a monoarticular arthritis with a high antibody concentration against Borrelia with no other symptoms" – the current CDC, Dearborn "case definition." Says them.

So, we get a variety show of autoimmune diseases out of Borreliosis, plus all the slander and libel waste basket cases, don't we? It only makes sense if you know what OspA is/does.

II. So what exactly is OspA? People say, "I did not get the vaccine so this does not concern me."

Oh, yes, you got the vaccine. Everyone with Lyme got LYMErix. Here is what LYMErix is, and how this vaccine-was-the-disease works (you've seen this previously):

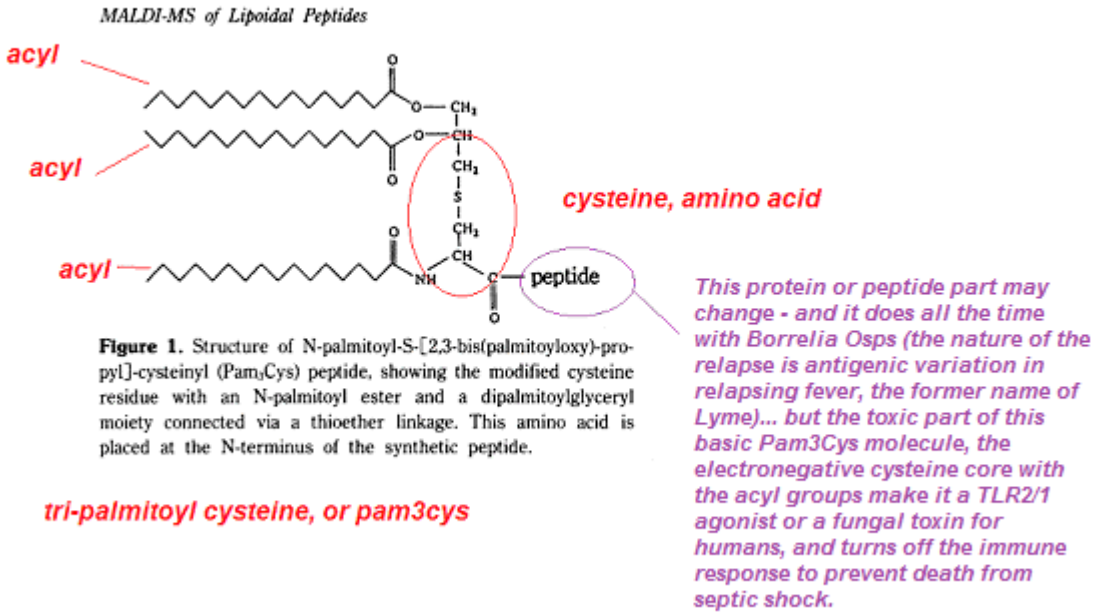


Figure 1. Structure of N-palmitoyl-S-[2,3-bis(palmitoyloxy)-propyl]-cysteinyl (Pam_3Cys) peptide, showing the modified cysteine residue with an N-palmitoyl ester and a dipalmitoylglyceryl moiety connected via a thioether linkage. This amino acid is placed at the N-terminus of the synthetic peptide.

Graphic Source:
Bull. Korean Chem. Soc. 1996, Vol. 17 Number 11
Characterization of Extremely Hydrophobic Immunostimulatory Lipoidal Peptides by Matrix-Assisted Laser Desorption ionization Mass Spectrometry
Jung-Suk Jang, Sung-Taek Lee, et al, Korea
http://newjournal.kcsnet.or.kr/main/j_search/j_download.htm?code=B961118

It's Pam3Cys or a triacylated lipoprotein, the degree of acylation is equated with its toxicity. So what is acylation? It's the zig-zaggy lines that mean Carbon-Carbon-Carbon, yes, hydrocarbons, like margarine or octane. Exactly, the name just refers to the number of carbons in each carboxyl or acyl group. Palmitic (the Pam in Pam3Cys) has X number of carbons, gasoline, 8, linoleic acids, like 14. Look up what are alkanes then add a COOH group and you have one of these fatty acids.

Something highly acylated like this (3 or more fatty acids hanging off) are managed by Toll-like Receptor (TLR) 2 and TLR1, together. Therefore a "TLR2/1-agonist" is another term that generally refers to lipoproteins like those from Borrelia, mycoplasma, mycobacteria, and others like Brucella. (But they can manage other compounds.)

This thing, Pam3Cys and fungal lipid molecules like it, is shed with the blebs. In other words, like this:

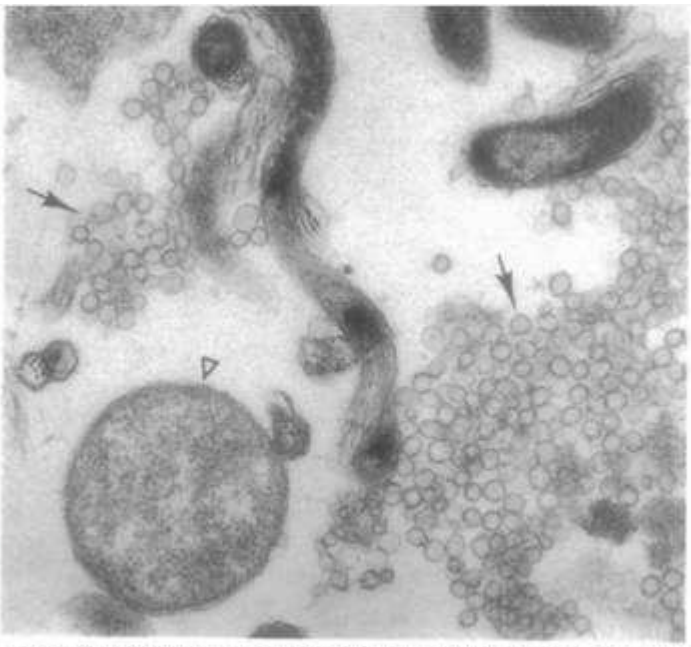


FIG. 2. Electron photomicrograph of a thin section of *B. hermsii* cells exposed to 3.1 nmol of benzylpenicillin per ml for 10 h. Large ovoid and spherical structures are illustrated; an open triangle points to one example. Arrows indicate small membranous blebs. Bar = 1 μm.

http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pubmed&pubmedid=7103461

The likes of OspA is on these blebs. They go to the brain, inflame it, get eaten up by immune cells - which renders them incompetent-, they go to the kidneys (LUAT), etc. You will find this to be so in an NIH-owned patent (5,217,872) and elsewhere.

So, the fungal antigens are on the shed blebs and they go everywhere and they render the immune cells incompetent, resulting in an AIDS like disease. Everyone who has Lyme disease also has LYMErix disease.

The NIH patent explaining how Lyme causes LYMErix-disease ("stealth bomber"):

"The invention relates to novel antigens associated with Borrelia burgdorferi which are **exported (or shed) in vivo** and whose detection is a means of diagnosing Lyme disease. The antigens are extracellular membrane vesicles and other **bioproducts including the major extracellular protein antigen**. Another object of the invention is to provide antibodies, monoclonal and/or polyclonal, labeled and/or unlabeled, that are raised against the antigens. A further object of the invention is to provide a method of diagnosing Lyme disease by detecting the antigens in a biological sample taken from a host using the antibodies in conventional immunoassay formats. Another object of the invention is to provide kits, for the diagnosis of Lyme disease, comprising the antibodies and ancillary reagents. The advantage of the antibodies used in the invention is that they react with the antigens from geographically diverse strains of Borrelia burgdorferi, but do not react with antigens from related Borrelia spirochetes."
http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetahtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5,217,872.PN.&OS=PN/5,217,872&RS=PN/5,217,872

The shed blebs (or exosomes or vesicles) have LYMErix on them (delayed fuse or "time bomb"):

J Proteome Res. 2011 Oct 7;10(10):4556-66. doi: 10.1021/pr200395b. Epub 2011 Sep 13.

Characterization of multiprotein complexes of the Borrelia burgdorferi outer membrane vesicles.
Yang X1, Promnares K, Qin J, He M, Shroder DY, Kariu T, Wang Y, Pal U.

"Although we uncovered the existence of at least 10 distinct OM complexes harboring several unique subunits, **the complexome is dominated by the frequent occurrence of a limited diversity of membrane proteins, most notably P13, outer surface protein (Osp) A, -B, -C, and -D and Lp6.6."**
http://www.ncbi.nlm.nih.gov/pubmed/21875077

The thing you should be doing, now that establishment medicine and all the universities have basically defaulted on the BigPicture (20-30 million people disabled from the incompetent witch phenomenon, somatoformia), is follow up on these reports in PubMed, and "See Related" articles, and "See Cited by" articles and do your own research. Don't be afraid to take your time to develop the vocabulary; use multiple sources for basic biology and chemistry facts. By using multiple sources, you'll capture some sources that use a language set you already have. Then you can cross over and back to other sources until the picture is clearer for you. And VERIFY, VERIFY, VERIFY. Don't be afraid. There are no experts.

III. THE EVIDENCE. And now some of the Alphabet, A-to-the-Double-Alphabet, which all point to, well, LYMErix was never a vaccine and caused the same immunosuppression disease as Chronic Lyme. What are the common opportunistics we see emerge in *ALL* immunosuppression cases?

A. Here next we see **Brucella** and its TLR2/1 agonist antigens do the same thing: turning off the immune response and causing immunosuppression or producing no antibodies. MHC II or HLA molecules deliver antigens to the surface of the immune cell against which antibodies will be made. If, along comes a TLR2/1 agonist, in time, this function stops. No more antigen is presented, no more antibodies will be made. There are multiple explanations for this mechanism but we have a "THE LIST" at the end of this report with researchers who present information on mechanisms of TLR2-agonist related immunosuppression.

Use your search feature to look for "MHC" elsewhere in this report.

PLoS One. 2012; 7(11): e50214.
Published online 2012 Nov 26. doi: 10.1371/journal.pone.0050214
PMCID: PMC3506553
Outer Membrane Vesicles from* Brucella abortus *Promote Bacterial Internalization by Human Monocytes and Modulate Their Innate Immune Response
Cora N. Pollak, M. Victoria Delpino, Carlos A. Fossati, and Pablo C. Baldi*

"Previous studies have shown that smooth and rough strains of *Brucella* spontaneously release OMVs that contain **outer membrane proteins**, LPS and other bacterial components [20], [21]. While these OMVs were initially characterized by chemical and immunochemical methods, a proteomic analysis performed more recently [21] revealed that such vesicles contain several factors known or presumed to be related to the virulence of the bacterium, including the outer membrane proteins Omp16, Omp19, Omp25 and Omp31. It has been shown that Omp16 and Omp19 are lipoproteins that **modulate MHC II expression** in monocytes[22]. On the other hand, Omp25 has been linked to the ability of *Brucella* to modulate TNF-α secretion in human macrophages [23]. Therefore, it can be speculated that OMVs

from *Brucella* may mediate the transfer of virulence factors to the host cell to generate immunomodulation or other effects that may **favor the survival of the pathogen within cells**. To our knowledge, the interaction of *Brucella* OMVs with mammalian cells and the potential immunological consequences of such interaction have not been studied. The evaluation of these phenomena was the goal of the present study."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3506553/

B. **Tuberculosis, Harding & Radolf**. Now, here, let us for once and for all, pay attention to the fungal antigen specialist, Clifford Harding; "several studies have shown fungal antigens like LYMErix (TLR2-agonists) decrease antibody production or cause seronegativity"

Nat Rev Microbiol. 2010 Apr;8(4):296-307. doi: 10.1038/nrmicro2321.
Regulation of antigen presentation by Mycobacterium tuberculosis: a role for Toll-like receptors.
Harding CV1, Boom WH.

"Several studies have demonstrated that ***M. tuberculosis***-infected macrophages have decreased MHC class II molecule expression and decreased antigen presentation, reducing CD4+ T cell recognition of infected macrophages**20**,**22**–**24**,**30**,**32**–**38**. Comparison of the T cell responses to model antigens presented by ***M. tuberculosis***-infected macrophages and to antigens presented by uninfected macrophages showed that ***M. tuberculosis*** reduced antigen presentation by macrophages 12–18 hours or more after infection**32**,**35**.
"Recent studies have provided insights into the molecular mechanisms involved in the inhibition of MHC class II antigen presentation by *M. tuberculosis*. Viable *M. tuberculosis* is not required for inhibition of macrophage MHC class II expression and antigen presentation, which can be achieved by exposure of macrophages to *M. tuberculosis* lysate22,30,33–35,39. Biochemical fractionation was used to identify *M. tuberculosis* components that inhibited MHC class II molecule expression, and several *M. tuberculosis*lipoproteins, including LpqH32, LprG40 and LprA41, were found to be key inhibitors. These lipoproteins, as well as PhoS1 (also known as PstS1), are agonists of TLR2 (REFS 23,32,40–43) (TABLE 1), and their inhibition of MHC class II molecule expression and antigen presentation is dependent on TLR2 and its adaptor, myeloid differentiation primary-response protein 88 (MYD88)19,23,32,40. Furthermore, MHC class II inhibition that is mediated by viable *M. tuberculosis* is itself also largely dependent on TLR2 (REFS23,32) and, to an even greater degree, on MYD88 (REF. 23), although some MHC class II inhibition might be due to non-lipoprotein components of *M. tuberculosis* and could be MYD88 independent18,19,21.
"Thus, prolonged TLR2 signalling induced by *M. tuberculosis* lipoproteins (and, potentially, by other TLR2 agonists expressed by *M. tuberculosis*18) results in inhibition of MHC class II molecule expression and antigen presentation by *M. tuberculosis*-infected macrophages."
https://www.ncbi.nlm.nih.gov/pubmed/20234378
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3037727/

Clifford Harding and Justin Radolf on the downregulation of MHC or HLA molecules, resulting in immunosuppression or lack of antibodies from exposure to the likes of OspA covered blebs. More:

J Immunol. 2001 Jul 15;167(2):910-8.
Toll-like receptor 2-dependent inhibition of macrophage class II MHC expression and antigen processing by 19-kDa lipoprotein of Mycobacterium tuberculosis.
Noss EH1, Pai RK, Sellati TJ, Radolf JD, Belisle J, Golenbock DT, Boom WH, Harding CV.

"Mycobacterium tuberculosis (MTB) induces vigorous immune responses, yet persists inside macrophages, evading host immunity. MTB bacilli or lysate was found to inhibit macrophage expression of class II MHC (MHC-II) molecules and MHC-II Ag processing. This report characterizes and identifies a specific component of MTB that mediates these inhibitory effects. The inhibitor was extracted from MTB lysate with Triton X-114, isolated by gel electroelution, and identified with Abs to be MTB 19-kDa lipoprotein. Electroelution- or immunoaffinity-purified MTB 19-kDa lipoprotein inhibited MHC-II expression and processing of both soluble Ags and Ag 85B from intact MTB bacilli. Inhibition of MHC-II Ag processing by either MTB bacilli or purified MTB 19-kDa lipoprotein was dependent on Toll-like receptor (TLR) 2 and independent of TLR 4. Synthetic analogs of lipopeptides from Treponema pallidum also inhibited Ag processing. **Despite the ability of MTB 19-kDa lipoprotein to activate microbicidal and innate immune functions early in infection, TLR 2-dependent inhibition of MHC-II expression and Ag processing by MTB 19-kDa lipoprotein during later phases of macrophage infection may prevent presentation of MTB Ags and decrease recognition by T cells**. This mechanism may allow intracellular MTB to evade immune surveillance and maintain chronic infection."

http://www.jimmunol.org/cgi/content/full/167/2/910

Sounds like post-sepsis syndrome via fungal antigen tolerance to me. In the beginning of this report, you al;so saw Harding talk about how exposure to TLR2 agonists like fungal lipopeptides also cause cross tolerance to the TLRs that handle viruses, TLRs 7 and 9 and proposed that exposure to too much fungal OspA might render you incompetent to the likes of the common, latent herpes viruses.

Fungal antigens cause immunosuppression and ***not antibodies*** against Borrelia, particularly not OspA. Not TLR2/1 agonists. Not Pam3Cys. No. It does not result in antibodies. Period. You have this outcome if you have "chronic Lyme." OspA never could have been a vaccine. Clearly Yale falsified their LYMErix vaccine results. And Dearborn, with the requirement for high antibody production, is research fraud. Lyme Osps, Brucella Omps, and Mycobacteria Lprs…. and *several studies* say so.

Whoever does not know what LYMErix disease is does not know what Lyme disease is. This includes International Lyme and Associated Diseases Society (ILADS.org(and all of the Lyme non-profits. One has to know what the antigen is, in order to know what it does. This is basic science.

C. **Norman Latov** on how OspA vaccination caused the same disease as chronic Lyme:

J Peripher Nerv Syst. 2004 Sep;9(3):165-7.
Neuropathy and cognitive impairment following vaccination with the OspA protein of Borrelia burgdorferi.
Latov N1, Wu AT, Chin RL, Sander HW, Alaedini A, Brannagan TH 3rd.
"Neurological syndromes that follow vaccination or infection are often attributed to autoimmune mechanisms. We report six patients who developed neuropathy or cognitive impairment, within several days to 2 months, following vaccination with the OspA antigen of Borrelia burgdorferi. Two of the patients developed cognitive impairment, one chronic inflammatory demyelinating polyneuropathy (CIDP), one multifocal motor neuropathy, one both cognitive impairment and CIDP, and one cognitive impairment and sensory axonal neuropathy. The patients with cognitive impairment had T2 hyperintense white matter lesions on magnetic resonance imaging. **The similarity between the neurological sequelae observed in the OspA-vaccinated patients and those with chronic Lyme disease suggests a possible**

role for immune mechanisms in some of the manifestations of chronic Lyme disease that are resistant to antibiotic treatment." http://www.ncbi.nlm.nih.gov/pubmed/15363064

D. **Donald H. Marks** on how LYMErix caused the same disease as chronic Lyme:

Int J Risk Saf Med. 2011;23(2):89-96. doi: 10.3233/JRS-2011-0527.
Neurological complications of vaccination with outer surface protein A (OspA).
Marks DH1.

"**A wide range of neurological complications have been reported via the medical literature and the VAERS system after vaccination with recombinant outer surface protein A (OspA) of Borrelia**. To explore this issue, 24 patients reporting neurological adverse events (AE) after vaccination with Lymerix, out of a group of 94 patients reporting adverse events after Lymerix vaccination, were examined for causation. Five reports of cerebral ischemia, two transient Ischemic attacks, five demyelinating events, two optic neuritis, two reports of transverse myelitis, and one non-specific demyelinating condition are evaluated in this paper. Caution is raised on not actively looking for neurologic AE, and for not considering causation when the incidence rate is too low to raise a calculable difference to natural occurrence."
http://www.ncbi.nlm.nih.gov/pubmed/21673416

It's not "Autoimmune." It's Subimmune. This Subimmunity represents the entire class of the DSM VooDoo Somatoformia – as well as cancer. Cancer is in the Subimmune class, at the other end of the immunity spectrum from Autoimminity. This fact or condition completely flips the entire medical paradigm where you have to have a biomarker that is above-, or more-than- the normal range. Lyme is not an inflammatory disease. There are always negative correlations to biomarkers of autoimmunity or illness or infection except when using sophisticated DNA techniques using spinal fluid, in particular. Henceforth, Autoimmunity will be an obsolete word that connotes the previous Medical Establishment where BigPharma is going to "block" something with their drugs. They are dinosaurs. You can't block a mechanism that is already permanently blocked and you can't unblock it.

It could be that a person has an HLA-linked outcome to one of the secondary infections like Epstein-Barr or HHV-6, reactivated by the AIDS-like Lyme and LYMErix. Those people would for instance have the official, hypersensitivity outcomes of MS or Lupus or whatever. But they are not also called Incompetent Incantation-ators and they are not mistreated by the entire universe (family, friends, Social Security, "doctors," everyone, including ILADS and the non profits).

E. **Ben Luft** at the 1998 FDA Vaccine Meeting on LYMErix:

"The point that I wanted to make in regard to the study is that there is very heavy dependence on serologic confirmation. And when we start thinking about the adverse events, **it was stated originally when we got the overview of the disease that the disease is really quite protean. And actually the adverse events are very similar to what the disease manifestations are.** And if you start to, as I think Dr. Hall was eluding to -- if you start to kind of say well how often do you actually become seropositive, you can start to have a different take on when someone has an adverse event or whether it is disease specific or infection specific versus vaccine specific. And I think that that is an important issue that we

have to deal with. ..." http://www.fda.gov/ohrms/dockets/ac/98/transcpt/3422t1.rtf

F. **Dave Persing** who together with Yale's Robert Schoen developed this test in 1994 or 1995 says this about the similarities between Lyme and LYMErix disease:

"Additional uncertainty may arise if the vaccines are not completely protective; **vaccinated patients with multisystem complaints characteristic of later presentations of Lyme disease may be difficult to distinguish from patients with vaccine failure...**"

http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=/netahtml/PTO/srchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804

Oh, you mean LYMErix causes the same disease as late Chronic Neurologic Lyme (causes post-sepsis syndrome)?

G. **Wormser and Klempner** say there are 2 disease outcomes, Sepsis, and Bad Knees (and you saw the other 3 reports by Wormser and even Steere and CDC's Paul Mead in the beginning of this report, stating that the immunosuppression might be the more important driver of late, Chronic Lyme (post-sepsis).

J Infect Dis. 2005 Sep 15;192(6):1010-3. Epub 2005 Aug 4.
A case-control study to examine HLA haplotype associations in patients with posttreatment chronicLyme disease.
Klempner MS1, Wormser GH, Wade K, Trevino RP, Tang J, Kaslow RA, Schmid C.

"Lyme disease is caused by infection with the tickborne bacterium *Borrelia burgdorferi*. Antibiotic treatment is highly effective for the acute symptoms of Lyme disease and is also effective **for late septic manifestations** . … There appear to be at least 2 distinct syndromes in patients with persistent symptoms after antibiotic treatment. One syndrome has localized symptoms that are similar to pretreatment symptoms. Patients with this syndrome often have recurrent episodes of arthritis/synovitis. Results of synovial fluid cultures and polymerase chain reaction (PCR) for *B. burgdorferi* are negative [2]. **Patients generally feel well aside from their arthritis symptoms**.
"Specific HLA haplogroups (i.e., HLA-DR4 and HLA-DR2) have been associated with the failure to respond to antibiotics in this group of patients, and their arthritis may be due to molecular mimicry between a dominant epitope of outer surface protein A (OspA) of *B. burgdorferi* and lymphocyte function–associated antigen–1 (LFA-1) [3]. A much more common syndrome of persistent symptoms is a systemic illness that is characterized by profound fatigue, myalgias, polyarthralgias without arthritis, paresthesias, and mood and memory disturbances. This syndrome has been variously referred to as "chronic Lyme disease," "post–Lyme disease syndrome," and "posttreatment chronic Lyme disease" (PTCLD). The cause of the persistent systemic symptoms in these patients is unknown. However, we have reported elsewhere that the impact that PTCLD has on health-related quality of life was highly significant and that treatment with placebo or 90 days of additional antibiotics did not differentially affect patients' health-related quality of life [4]. We also did not find evidence of persistent infection with ***B. burgdorferi*** or exposure to other tickborne infectious agents that could explain the persistent systemic symptoms."
http://jid.oxfordjournals.org/content/192/6/1010.full

So, there are 2 distinct diseases: Arthritis ("one case a year" - Dattwyler), and the other thing – the chronic neurologic. The first thing, where people do not feel sick, is a Dearborn "case" of Lyme. But, these criminals claim, those chronic neurologic cases are not sick from *B. burgdorferi.*
No, it's much much worse. The OspA, Pam3Cys, LYMErix and ImmuLyme vaccines caused it, too. And then there's those irksome "Epstein-Barr like mutated B cells in the spinal fluid of chronic neurologic Lyme victims…"

H. **The 3 Tuberculosis vaccine attempts** that all failed the same way LYMErix failed, by making people sicker and more susceptible to disease:

Clin Exp Immunol. 2000 May;120(2):274-9.
The 19-kD antigen and protective immunity in a murine model of tuberculosis.
Yeremeev VV1, Lyadova IV, Nikonenko BV, Apt AS, Abou-Zeid C, Inwald J, Young DB.
"The 19-kD antigen is a cell wall-associated lipoprotein present in Mycobacterium tuberculosis and in bacille Calmette-Guérin (BCG) vaccine strains. Expression of the 19-kD antigen as a recombinant protein in two saprophytic mycobacteria-M. vaccae and M. smegmatis-resulted in abrogation of their ability to confer protection against M. tuberculosis in a murine challenge model, and in their ability to prime a DTH response to cross-reactive mycobacterial antigens. Induction of an immune response to the 19-kD antigen by an alternative approach of DNA vaccination had no effect on subsequent M. tuberculosis challenge. These results are consistent with a model in which the presence of the 19-kD protein has a **detrimental effect on the efficacy of vaccination with live mycobacteria**. Targeted inactivation of genes encoding selected antigens represents a potential route towards development of improved vaccine candidates."
http://www.ncbi.nlm.nih.gov/pubmed/10792376

Infect Immun. 2001 Mar;69(3):1433-9.
Mycobacterium tuberculosis 19-kilodalton lipoprotein inhibits Mycobacterium smegmatis-induced cytokine production by human macrophages in vitro.
Post FA1, Manca C, Neyrolles O, Ryffel B, Young DB, Kaplan G.

"Vaccination of mice with Mycobacterium vaccae or M. smegmatis induces some protection against M. tuberculosis challenge. The 19-kDa lipoprotein of M. tuberculosis, expressed in M. vaccae or M. smegmatis (M. smeg19kDa), abrogates this protective immunity. To investigate the mechanism of this suppression of immunity, human monocyte-derived macrophages (MDM) were infected with M. smeg19kDa. Infection resulted in reduced production of tumor necrosis factor alpha (TNF-alpha) ($P < 0.01$), interleukin-12 (IL-12) ($P < 0.05$), IL-6 ($P < 0.05$), and IL-10 ($P < 0.05$), compared to infection with M. smegmatis vector (M. smegV). Infection with M. smeg19kDa and with M. smegV had no differential effect on expression of costimulatory molecules on MDM, nor did it affect the proliferation of presensitized T cells cocultured with infected MDM. When MDM were infected with M. smegmatis expressing mutated forms of the 19-kDa lipoprotein, including non-O-glycosylated (M. smeg19NOG), nonsecreted (M. smeg19NS), and nonacylated (M. smeg19NA) variants, the reduced production of TNF-alpha or IL-12 was not observed. When the purified 19-kDa lipoprotein was added directly to cultures of infected monocytes, there was little effect on either induction of cytokine production or its inhibition. Thus, the immunosuppressive effect is dependent on glycosylated and acylated 19-kDa lipoprotein present in the phagosome containing the mycobacterium. These results suggest that the diminished protection against challenge with M. tuberculosis seen in mice vaccinated with M. smegmatis expressing the 19-kDa lipoprotein is the result of reduced TNF-alpha and IL-12 production, possibly

leading to reduced induction of T-cell activation."
http://www.ncbi.nlm.nih.gov/pubmed/11179309

Infect Immun. 2003 Jun;71(6):3146-54.
The Mycobacterium tuberculosis recombinant 27-kilodalton lipoprotein induces a strong Th1-typeimmune response deleterious to protection.
Hovav AH1, Mullerad J, Davidovitch L, Fishman Y, Bigi F, Cataldi A, Bercovier H.

"Th1 immune response is essential in the protection against mycobacterial intracellular pathogens. Lipoproteins trigger both humoral and cellular immune responses and may be candidate protective antigens. We studied in BALB/c mice the immunogenicity and the protection offered by the recombinant 27-kDa Mycobacterium tuberculosis lipoprotein and the corresponding DNA vaccine. Immunization with the 27-kDa antigen resulted in high titers of immunoglobulin G1 (IgG1) and IgG2a with a typical Th1 profile and a strong delayed hypersensitivity response. A strong proliferation response was observed in splenocytes, and significant nitric oxide production and gamma interferon secretion but not interleukin 10 secretion were measured. Based on these criteria, the 27-kDa antigen induced a typical Th1-type immune response thought to be necessary for protection. Surprisingly, in 27-kDa-vaccinated mice (protein or DNA vaccines) challenged by M. tuberculosis H37Rv or BCG strains, there was a significant increase in the numbers of CFU in the spleen compared to that for control groups. Furthermore, the protection provided by BCG or other mycobacterial antigens was completely abolished once the 27-kDa antigen was added to the vaccine preparations. This study indicates that **the 27-kDa antigen has an adverse effect on the protection afforded by recognized vaccines.** We are currently studying how the 27-kDa antigen modulates the mouse immune response."
https://www.ncbi.nlm.nih.gov/pubmed/12761093
http://www.pubmedcentral.nih.gov/articlerender.fcgi?tool=pubmed&pubmedid=12761093

"A deleterious effect on immunity," "an adverse effect on the protection," "the immunosuppressive effect," "diminished protection," "reduced T cell activation,…"

THEY DON'T WORK; lipoproteins are the *opposite* of vaccines.

I. **Raymond Dattwyler**, 1988 Not surprisingly, that data on the failed Tuberculosis (Tb) fungal vaccines is all quite reminiscent of what Ray Dattwyler said about Borrelial supernatant - the stuff that floats on the top, the oil of the oil and vinegar, yeah, the oil, the lipids, the lipoproteins, the Osps…

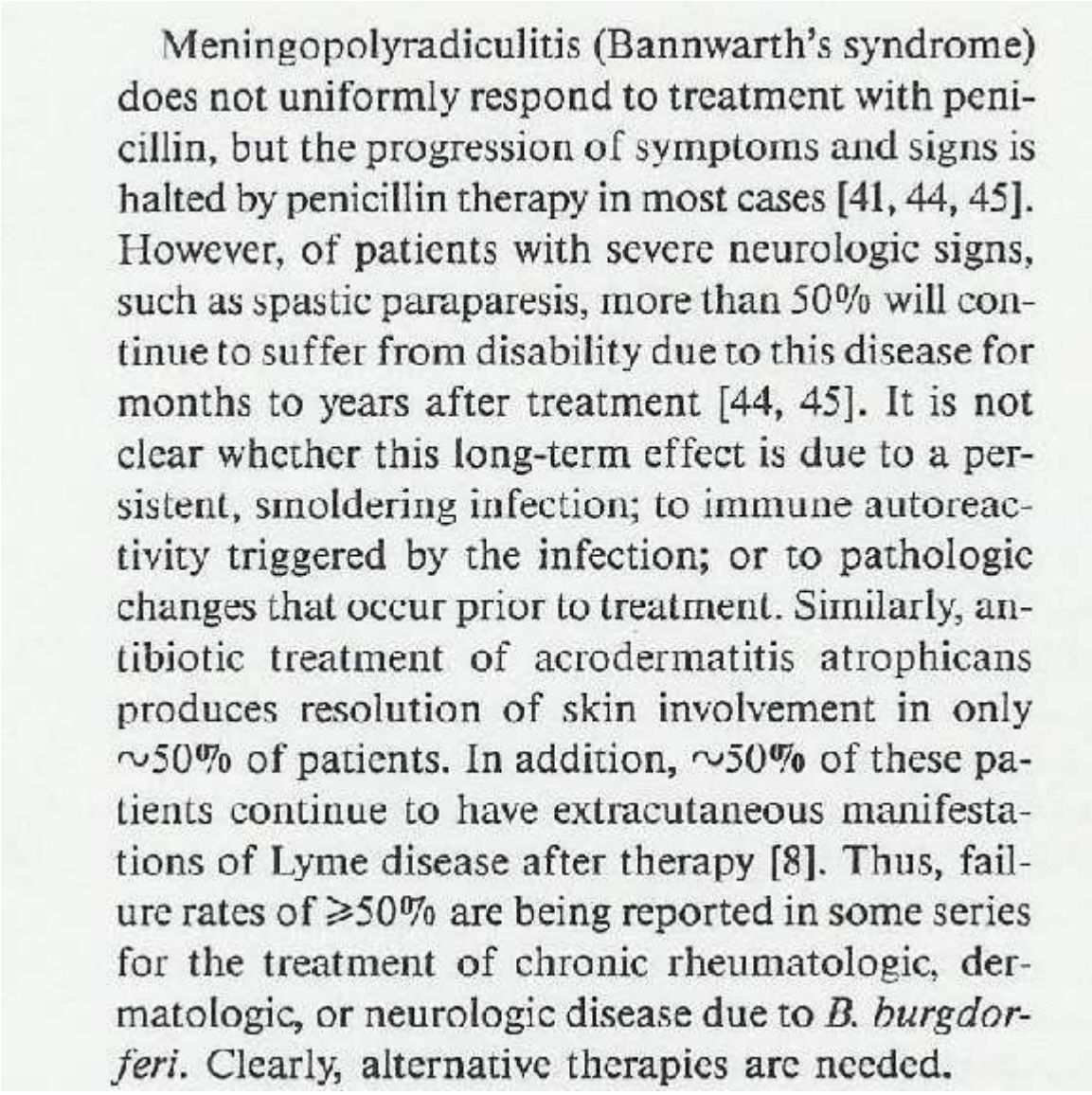
Ann N Y Acad Sci. 1988;539:103-11.
Modulation of natural killer cell activity by Borrelia burgdorferi.
Golightly M1, Thomas J, Volkman D, Dattwyler R.
"..when lymphocytes are cultured in the presence of growing Bb there is a marked inhibition ($p < .0005$) of NK activity on days 3, 5, and 7 when compared to lymphocytes cultured in BSKII media in the absence of spirochetes. This effect is not due to a selective depletion or or toxicity to endogenous NK since viability studies and monoclonal antibodies demonstrate no significant changes after culture with the organism.
"The inhibition is directly attributable to the organism or its supernatants (data not shown)."
http://www.ncbi.nlm.nih.gov/pubmed/3056196

Borrelial lipids cause immunosuppression, is what he is saying.

And here is what else Luft and Dattwyler said in IDSA's journal in 1989 about how treatment fails, and how this may be due to **"pathological changes that occur prior to treatment**" confirmed by Baumgarth

when she showed infection with Borrelia damaged B cell germinal centers and rendered victims unable to deal with a common viral infection like influenza

Rheum Dis Clin North Am. 1989 Nov;15(4):747-55.
Treatment of Lyme borreliosis.
Luft BJ1, Dattwyler RJ. https://www.ncbi.nlm.nih.gov/pubmed/2555849



Meningopolyradiculitis (Bannwarth's syndrome) does not uniformly respond to treatment with penicillin, but the progression of symptoms and signs is halted by penicillin therapy in most cases [41, 44, 45]. However, of patients with severe neurologic signs, such as spastic paraparesis, more than 50% will continue to suffer from disability due to this disease for months to years after treatment [44, 45]. It is not clear whether this long-term effect is due to a persistent, smoldering infection; to immune autoreactivity triggered by the infection; or to pathologic changes that occur prior to treatment. Similarly, antibiotic treatment of acrodermatitis atrophicans produces resolution of skin involvement in only ~50% of patients. In addition, ~50% of these patients continue to have extracutaneous manifestations of Lyme disease after therapy [8]. Thus, failure rates of ≥50% are being reported in some series for the treatment of chronic rheumatologic, dermatologic, or neurologic disease due to *B. burgdorferi*. Clearly, alternative therapies are needed.

J. **Nicole Baumgarth and Stephen Bartold**

PLoS Pathog. 2015 Jul 2;11(7):e1004976. doi: 10.1371/journal.ppat.1004976. eCollection 2015.
Suppression of Long-Lived Humoral Immunity Following Borrelia burgdorferi Infection.
Elsner RA1, Hastey CJ1, Olsen KJ2, Baumgarth N3.

"Lyme Disease caused by infection with Borrelia burgdorferi is an emerging infectious disease and already by far the most common vector-borne disease in the U.S. Similar to many other infections, infection with B. burgdorferi results in strong antibody response induction, which can be used clinically as a diagnostic measure of prior exposure. However, clinical studies have shown a sometimes-precipitous

decline of such antibodies shortly following antibiotic treatment, revealing a potential deficit in the host's ability to induce and/or maintain long-term protective antibodies. This is further supported by reports of frequent repeat infections with B. burgdorferi in endemic areas. The mechanisms underlying such a lack of long-term humoral immunity, however, remain unknown. We show here that B. burgdorferi infected mice show a similar rapid disappearance of Borrelia-specific antibodies after infection and subsequent antibiotic treatment. This failure was associated with development of only short-lived germinal centers, micro-anatomical locations from which long-lived immunity originates. These showed structural abnormalities and failed to induce memory B cells and long-lived plasma cells for months after the infection, rendering the mice susceptible to reinfection with the same strain of B. burgdorferi. The inability to induce long-lived immune responses was not due to the particular nature of the immunogenic antigens of B. burgdorferi, as antibodies to both T-dependent and T-independent Borrelia antigens lacked longevity and B cell memory induction. **Furthermore, influenza immunization administered at the time of Borrelia infection also failed to induce robust antibody responses, dramatically reducing the protective antiviral capacity of the humoral response.** Collectively, these studies show that B. burgdorferi-infection results in targeted and temporary immunosuppression of the host and bring new insight into the mechanisms underlying the failure to develop long-term immunity to this emerging disease threat."
https://www.ncbi.nlm.nih.gov/pubmed/26136236

More:
https://www.ncbi.nlm.nih.gov/pubmed/?term=baumgarth+and+borrelia

https://www.ncbi.nlm.nih.gov/pubmed/?term=bartold+SW+and+borrelia

So Lyme infection renders you unable to handle viral infections. This seems to have to do with damaged B cell maturation centers. We wonder if what Baumgarth found has anything to do with Duray's findings that we have EBV-transformed lymphocytes in our spinal fluid, and whether all the biomarkers of central nervous system disease associated with Lyme (discovered by the Cabal) has more to do with these secondary opportunistics…. ??

One thing is for sure, it does not help to superimaginate that 20-30 million people are incompetent witches who chronically issue backfiring incantations and are sticking themselves with their Voodoo pins meant for other people. <Sigh>

K. **Gary Wormser on OspA-as-a-non-vaccine**, which you've already seen: Lipoproteins BLUNT immunity

FEMS Immunol Med Microbiol. 2000 Jul;28(3):193-6.
Modulation of lymphocyte proliferative responses by a canine Lyme disease vaccine of recombinant outer surface protein A (OspA).
Chiao JW1, Villalon P, Schwartz I, Wormser GP.
"… After exposure to either the unaltered vaccine preparation or OspA prepared in saline, normal lymphocyte responses to the mitogens concanavalin A, phytohemagglutinin-M or pokeweed mitogen, or the antigen BCG were consistently reduced. Whole cell extracts of B. burgdorferi also modulated immune responses but required a much greater quantity of protein than needed for theOspA preparation. The magnitude of modulation was directly dependent on the quantity of OspA. OspA interferes with the response of lymphocytes to proliferative stimuli including a blocking of cell cycle phase progression.

Future studies designed to delete the particular region or component of theOspA molecule responsible for this effect may lead to improved vaccine preparations."
http://www.ncbi.nlm.nih.gov/pubmed/10865170

Once more (you've just seen 5+ examples), lipoproteins and lipoprotein vaccines suppress immunity, even in animals, which are known to have more broad natural immunity than humans (making animals diseases very good sources of human disease bioweapons). Three Tb vaccines based on lipoproteins, Dattwyler, et al, claiming Borrelia oily lipoproteins blunt immunity, and Gary Wormser himself said lipoproteins do that mysterious thing… "blocking of cell cycle phase progression." Later we will learn OspA inhibits apoptosis, which is the same thing EBV does. That is what "EBV-immortalized" means. The infected cell does not kill itself, or undergo apoptosis as a way of keeping the pathogens from reproducing themselves.

SIDESTEPPING -- BCL2 Class molecules and OspA inhibit apoptosis; No "biofilms" *in vivo*

BCL2 class molecules do the same thing, they inhibit apoptosis or they block the auto-kill or apoptosis kinases (enzymes). BCL means B Cell Lymphoma (clue). If you have too many copies of a BCL2 class gene, as is the case with "nerve overgrowth syndromes" such as Neurofibromatosis or/and Autism (the Einstein, Telsa, Newton, Grandin kind), their over expression leads to inhibition of apoptosis. This is thought to be the case with the genetic, large-brain kind of Autism; a "lack of normal synaptic pruning." A BCL2 class gene happens to co-confer with other copies in the case of reversed duplication, as shown here:

J Med Genet. 2002 Mar;39(3):170-7.
Organisation of the pericentromeric region of chromosome 15: at least four partial gene copies are amplified in patients with a proximal duplication of 15q.
Fantes JA1, Mewborn SK, Lese CM, Hedrick J, Brown RL, Dyomin V, Chaganti RS, Christian SL, Ledbetter DH.

"We identified a fourth pseudogene, BCL8, which maps to the pericentromeric region and is coamplified along with the NF1 sequences. Interphase FISH ordering experiments show that IgH D lies closest to the centromere, while BCL8A is the most distal locus in this pseudogene array;"
http://www.ncbi.nlm.nih.gov/pubmed/11897815 (And see related, as always.)

People should investigate independently, anyway, to see if there is a genetic link between Autism and Neurofibromatosis Type 1. There is, and it is quite well-known. Therefore, if it is well-known that NF1 and Autism co-occur at a very high rate, ***there must be a genetic form of Autism*** as well as is the brain damage kind from vaccines, which should be called "Brain Damage from Vaccine Viruses" and not Autism: https://www.ncbi.nlm.nih.gov/pubmed/?term=Neurofibromatosis+and+Autism
Once again, the unfortunate thing about not requiring people with MDs after their names to have a science background, is that they have a hard time putting scientific facts together. They don't know about a requirement for Scientific Validity. They don't have backgrounds in Genetics, Taxonomy and Evolution, or even basic Biology. They don't know the basic Chemistry of asking, ***"WHAT IS IT? So I can know what it does…?"***

> 'Sad, really. Pathetic. What we are talking about here is a complete failure in Medicine and Mental Health "Medicine" if you could call mental health, "medicine." How many people know

that the genetic kind of Autism co-confers with NF1, … and that the inhibition of apoptosis is programmed in in Autism and NF1,… and also that the inhibition of apoptosis of immune cells is also ***acquired*** by exposure to fungal Osps and the like,… and that the mechanism of inhibiting apoptosis is also hijacked by Epstein-Barr? That's practically everything you could know about ***all*** disease, and sitting right in Yale's lap. 'Almost literally. And they threw it all away and chose instead, debauchery and sleaze.

OspA-like lipoproteins act like extra BCL2 molecules, inhibiting apoptosis. They gum up the immunity works. They stick to even the membranes of mitochondria, depolarizing it. They stick to red blood cell membranes, also depolarizing them. This is shown in numerous examples of the literature with mycoplasmal and mycobacterial lipoproteins, as well as Brucella lipoproteins. One can use PubMed or the National Library of Medicine Anyone can find out OspA is the basic Pam3Cys molecule. It occurs naturally and is synthetic (Braun lipoprotein). Epstein-Barr has the ability to use human BCL2. The first step in dysimmunity, one could claim, is the inhibition of apoptosis.
https://www.ncbi.nlm.nih.gov/pubmed/?term=EBV+and+BCL2 (You'll see more reports on this later.)

Fungal lipoproteins, of the TLR2/1 type, highly lipidated, with 3 or more acyl (fatty acid, like palmitic acid or linoleic acid, etc) groups, gum up immunity. They inhibit apoptosis. In particular, OspA is sticky and even sticks to itself. This may be the reason spirochetes appear to cluster *in vitro*. However they don't cluster or grow in colonies in humans; biofilms are not the reason antibiotic treatment fails. This data summary and explanation of the science abundantly shows spirochetes and "biofilms" are not what makes Chronic Lyme chronic. Especially not if the vaccine caused the same chronic neurologic disease.

Paul Duray
J Clin Microbiol. 1991 Apr;29(4):764-72.
Morphology of Borrelia burgdorferi: structural patterns of cultured borreliae in relation to staining methods.
Aberer E1, Duray PH.

"The microscopic recognition of Borrelia burgdorferi in biologic fluids and tissues is difficult and challenging because of low numbers of organisms occurring as single isolated spirochetes, the **apparent lack of colony formation in tissues**, and differing lengths and structural morphologies."
http://www.ncbi.nlm.nih.gov/pubmed/1716264

Anyone who has a science background, which apparently dis-includes anyone with an 'MD" after their names has for 15 years been able to discover what exactly OspA was and why it caused systemic disease and why it failed.

L. Adriana Marques formerly of NINDS' MS-Lyme group and who now works for NIAID, and specializes only in Lyme and the MS and herpesviruses (clue):

"When Lyme Disease Lasts and Lasts,"* by Jane Brody in the *New York Times.

"'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/

And, as you have previously seen, Marques and Martin have stated that it is OspA or borrelial triacyl lipoproteins responsible for all the trouble. The vaccine caused the disease. So what is the vaccine? A fungal endotoxin.
https://www.ncbi.nlm.nih.gov/pubmed/?term=marques+and+martin+and+tlr2

M. Carolyn Beans, NIH:

"Surviving Sepsis: Detection and Treatment Advances" by Carolyn Beans for the National Institutes of Health, August 18, 2014
"…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…**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**."
http://www.livescience.com/47387-sepsis-diagnosis-treatment-research-nigms.html

You're shaking your head, right?

The NIH supports the Hotchkiss, Washington University report on Sepsis and Post-Sepsis outcomes (see next below). The Cabal claims that what happens after early Lyme is called "Post-Lyme Syndrome," and that that is psychiatric. But actually you saw Klempner call it a Septic event ("G.," above), particularly as regards for the Central Nervous System (CNS). People should be aware that these criminals are the authors of all the scientifically valid signs or **BIOMARKERS of CNS degradation** (see the other charge sheets). That is why the psychiatric slander, libel and downright genetic discrimination ("No arthritis HLA's? You must be crazy") is a criminal charge, Deprivation of Rights under Color of Law. The biomarkers will probably not be found in the blood, except for reduced cytokines, perhaps.

N. **Hotchkiss** Washington University, Saint Louis, MO (wustl.edu):

wustl.edu discovers that sepsis is like Chronic 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

O. **Paul Auwaerter**, specializes in only Lyme and herpesviruses:
http://www.hopkinsmedicine.org/profiles/results/directory/profile/0000525/paul-auwaerter

P. **Brigitte Huber of Tufts**, former partner with Allen Steere in "Lyme is Only a Bad Knee Theatre," who now specializes in ONLY herpesviruses (actually, claiming EBV could be reactivating a human endogenous retrovirus or a HERV):
http://www.ncbi.nlm.nih.gov/pubmed/?term=huber+bt+and+epstein-barr

Q. If pathologist **Colonel Paul H. Duray** (NCI, Yale, US Army Ft. Detrick) were still alive, you could ask him why he said, "these look like Epstein-Barr transformed cells" in the spinal fluid of chronic neurologic Lyme victims in 1989, in IDSA's journal.

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

Or why Duray said it again, in 1992: "In Chronic Lyme victims' cerebrospinal fluid, I see what look like Epstein-Barr transformed lymphocytes."

"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 suibsets 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 ALDF.com Conference**, published in Steve Schutzer's Lyme Disease: Molecular and Immunologic Approaches, book, as previously referenced.

R. **Patricia K. Coyle**, **SUNY-SB**. Coyle once was the author of several reports and even methods to detect borrelia antigens in the central nervous system because of the absence of antibodies…. now only specializes in Multiple Sclerosis???

http://www.ncbi.nlm.nih.gov/pubmed/?term=Coyle%20PK%5BAuthor%5D&cauthor=true&cauthor_uid=25406727

S. **Roland Martin and Adrianna** Marques at the NINDS MS and Lyme Division. Martin quit and went home to Germany once he found out LYMErix was responsible for the immunosuppression-come-New Great Imitator (in other words, that LYMErix or OspAish antigens were responsible for the MS outcome of Lyme:

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.
"…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

That's Auwaerter (Johns Hopkins), Huber (Tufts), Coyle (SUNY-SB), Duray (NIH, NC) Martin and Marques (NIH, NINDS), all either talking about Lyme and EBV-transformed cells, Lyme and EBV as the real culprit, specializing only in Lyme and EBV, or in Lyme and MS. Think about it.

T. **Anthony FAUCI** on immunosuppression and common opportunistics:

NIAID director Anthony Fauci says this in his **patent for IL-2 as an immune booster**. He lists fungi and stuff like *common opportunistics*, you know like…

"FIELD OF THE INVENTION
"The present invention pertains to a method for activating the immune system of a patient by intermittently administering interleukin-2 (IL-2) to that patient. Such administration of IL-2 can optionally be combined with other therapies, such as anti-retroviral, anti-bacterial or anti-fungal therapies, suitable for treatment of the patient's condition. This invention also relates to an approach to gene therapy that entails administering IL-2 to a patient so as to facilitate in situ lymphocyte transduction by a retroviral vector also administered to the patient.

"BACKGROUND OF THE INVENTION
"....Illustrative of specific disease states in treatment of which the present invention can be applied are HIV infection and other diseases characterized by a decrease of T-cell immunity, for example, **mycobacterial infections like tuberculosis and fungal infections such as cryptococcal disease. This method also can be used in the treatment of secondary infections that occur in patients with suppressed immune systems, such as the opportunistic infections that occur in AIDS patients. ..."**
"...Opportunistic infections may also be treated using the present invention. For example, AIDS related opportunistic infections are described in Mills et. al. (1990) Scientific American 263:51-57, which is hereby incorporated by reference in its entirety. Mills show that **common opportunistic infections are caused by, for example, Cytomegalovirus, Pneumocystis carnii, Candida albicans, Varicella-Zoster virus, Epstein-Barr virus, Toxoplasma gondii, Mycobacterium avium, Cryptococcus neoformans**. It is envisioned that IL-2 may be administered along with other compounds used to treat infectious diseases or other diseases. Examples of other agents include antifungal, antiviral, or antibacterial drugs. Additionally, IL-2 may be administered in combination with other efficacious cytokines. For example, combination therapy may include IL-2 with GM-CSF, G-CSF, M-CSF, IL-3, IL-12, IL-15, a-, b-, or g-interferons."
http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=/netahtml/PTO/srchnum.htm&r=1&f=G&l=50&s1=5,696,079.PN.&OS=PN/5,696,079&RS=PN/5,696,079

"Diseases of immunosuppression like fungal diseases." "Opportunistic infections like the herpesviruses and other fungal infections which now have a free ride due to TLR2-agonist tolerance and cross tolerance."

Yes. I think so. Common opportunistics, yeah, probably especially since Epstein-Barr and the other herpesviruses because those can be chronic and cause a chronic fatiguing disease – says the CDC -, not to mention is associated with MS and Lupus. Great Imitators…

U. **CDC's Suzanne Vernon** explaining how Epstein-Barr contributes to fatigue; How she committed research fraud to try to say fungal antigens are not involved in fatigue; How we know fungal antigens adhere to erythrocyte membranes causes hypoxic fatigue, stick to internal cell components, depolarizing membranes, etc.

BMC Infect Dis. 2006 Jan 31;6:15.
Preliminary evidence of mitochondrial dysfunction associated with post-infective fatigue after acute infection with Epstein Barr virus.
Vernon SD1, Whistler T, Cameron B, Hickie IB, Reeves WC, Lloyd A.

"Those who developed post-infective fatigue had gene expression profiles indicative of an altered host response during acute mononucleosis compared to those who recovered uneventfully. Several genes including ISG20 (interferon stimulated gene), **DNAJB2 (DnaJ [Hsp40] homolog and CD99), CDK8 (cyclin-dependent kinase 8), E2F2 (E2F transcription factor 2), CDK8 (cyclin-dependent kinase 8), and ACTN2 (actinin, alpha 2), known to be regulated during EBV infection, were differentially expressed in post-infective fatigue cases. Several of the differentially expressed genes affect mitochondrial functions including fatty acid metabolism and the cell cycle."**
"CONCLUSION: These preliminary data provide insights into alterations in gene transcripts associated with the varied clinical outcomes from acute infectious mononucleosis."
http://www.ncbi.nlm.nih.gov/pubmed/16448567

Now, go back to the Primers Shell Game and look at "gene expression" by Chiu and Aucott. Right. If these people were any more full of crap, they'd rival a Pacific Ocean-sized swine lagoon.

But, here, Vernon commits research fraud by throwing out the mycoplasma before she even starts to allegedly look for mycoplasmal DNA:

J Med Microbiol. 2003 Nov;52(Pt 11):1027-8.
Absence of Mycoplasma species DNA in chronic fatigue syndrome.
Vernon SD, Shukla SK, Reeves WC.

"Blood was collected in sodium citrate Vacutainer tubes (Beckton Dickinson) and shipped by overnight courier to the Centers for Disease Control (CDC), where plasma was collected by separation on lymphocyte separation medium (LSM; ICN Biomedicals). Plasma (1 ml) was concentrated to approximately 250 µl in a Centricon centrifugal filter unit YM-100 (Millipore). Cell-free plasma DNA was extracted by using a QIAamp DNA Mini kit (Qiagen) according to the manufacturer's instructions and quantified by using a DyNA Quant 200 fluorometer (Amersham Biosciences)."

https://www.ncbi.nlm.nih.gov/pubmed/14532349 http://jmm.sgmjournals.org/content/52/11/1027.long

CDC's Suzanne Vernon committed research fraud by centrifuging out the very cells to which mycoplasma adhere and then said, ***"How Amazing, there is no mycoplasma here!!"***

The other spooks, the NSA and FBI, if they don't have a culprit to define their existence, they invent one. They go to mosques, pick out a dummy and say, "Hey wouldn't it be fun to make bombs?" Then they

even go ahead and provide the dummy with a dummy bomb. But the CDC can't find any diseases. 'Unless the newspapers report some accidental escapes and releases. Then everyone helps them. ☺

V. **Mycoplasma adhere to erythrocytes** interfering with membrane potential and therefore the potential for oxygen to cross the erythrocyte membrane (causing fatigue):

Berl Munch Tierarztl Wochenschr. 1992 Nov 1;105(11):380-3.
[The effect of Eperythrozoon suis infection on the osmotic fragility of erythrocytes].
[Article in German]
Heinritzi K1, Plank G.

"Osmotic fragility of erythrocytes was tested in weaned pigs experimentally infected with Eperythrozoon (E.) suis. Acute eperythrozoonosis of splenectomized pigs led to an increase of osmotic fragility. It is supposed that E. suis infection causes a structural change in erythrocyte membrane. Possible mechanisms of this cell membrane injury are discussed."

http://www.ncbi.nlm.nih.gov/pubmed/1471973

Cell Death Differ. 2004 Nov;11(11):1204-12.
Mycoplasma fermentans inhibits tumor necrosis factor alpha-induced apoptosis in the human myelomonocytic U937 cell line.
Gerlic M1, Horowitz J, Horowitz S.

"Loss of mitochondrial inner transmembrane potential induced by TNFa is reduced in U937 cells infected with M. fermentans…
"In many apoptosis scenarios, including TNF-mediated apoptosis, the mitochondrial inner transmembrane potential ($_m$) collapses.[19, 20] To investigate whether the antiapoptotic effect of M. fermentans in TNF-induced apoptosis is upstream or downstream of the mitochondria, we measured the loss in Delta-Sigma$_m$, induced by TNF (20 ng/ml), in infected and noninfected cells. At 24 h post infection, the cultures were stimulated with TNF (20 ng/ml) for 2 h, and each culture was stained with 3,3'-dihexyloxacarbocyanine iodide ($DiOC_6$ (3)) and analyzed by FACS (a typical experiment is shown in Figure 6a).
http://www.ncbi.nlm.nih.gov/pubmed/15286682
http://www.nature.com/cdd/journal/v11/n11/full/4401482a.html

That's also cute, though, right? CDC throws out the stuff that causes fatigue by inhibiting the Energy Producing subcellular mitochondrial function – the cell's "powerhouse" – when allegedly looking for it. And these organisms also adhere to erythrocyte membranes, also inhibiting oxygen from transferring across it.

IF, Epstein-Barr alone were responsible for Chronic Fatigue Syndrome, then one can see their idiot point of view that "stress" causes the reactivation of Epstein-Barr ("somatiformical" = reactivating EBV) and that de-stressing solves the problem. Maybe that is the case with the somatoformical medical students and astronauts (one of the last criminal charge sheets in this series) and the like, who are so well known to have stress-reactivated Epstein-Barr or mono. But here we see something much more sinister at work. The CDC does not want anyone to know how tolerance to fungi causes irreversible fatigue and how that

tolerance spreads to other infections ("common, now, opportunistics").

We think the reason for this CDC tardation has to do with childhood vaccines being contaminated with fungal antigens, which is the reason for Thimerosal in the first place. We think the reason for this fraud on the part of the CDC is that they do not want us to be aware of the common mechanisms at work in vaccines-virus-acquired Autism (brain damage is the more correct term). We think the 20-30 million alleged witches and warlocks (somatoformers) in the country are the price the CDC pays to continue to brain damage around 1:60 (?) children for life. It's a great bargain for the CDC. They even say "it is a calculated risk," this vaccines enforcement and the brain-damaged-for-life outcome. CDC does the calculating. You know who all the *real* Scary People are.

People should follow up on these reports; here is good/typical one from 2008:

Trends Microbiol. 2008 Apr;16(4):173-80. doi: 10.1016/j.tim.2008.02.001. Epub 2008 Mar 18.
Staying alive: bacterial inhibition of apoptosis during infection.
Faherty CS1, Maurelli AT.

"The ability of bacterial pathogens to inhibit apoptosis in eukaryotic cells during infection is an emerging theme in the study of bacterial pathogenesis. Prevention of apoptosis provides a survival advantage because it enables the bacteria to replicate inside host cells. Bacterial pathogens have evolved several ways to prevent apoptosis by protecting the mitochondria and preventing cytochrome c release, by activating cell survival pathways, or by preventing caspase activation. This review summarizes the most recent work on bacterial anti-apoptotic strategies and suggests new research that is necessary to advance the field."
http://www.ncbi.nlm.nih.gov/pubmed/18353648

W. **Medvedev.** Tolerance and Cross Tolerance

One of the most important mechanisms of synergy between fungal antigens and viruses – and we have mentioned this many times in our reports and criminal charge sheets against the Cabal -, has to do with tolerance and cross tolerance and we have explained what this means in the past. Tolerance means your body no longer sees the invading pathogen's components are a threat and stops responding to them immunologically. Cross-tolerance is when an infection with one pathogen or antigen type, renders the immune system incompetent to other types. "Endotoxin Tolerance" is a known thing, known for decades. Endotoxin is considered mainly to be LPS or lipopolysaccharide (feel free to Google the structure or the image) which are TLR4 agonists. TLR4 agonists are not as toxic as the fungal TLR2/1 agonists of say spirochetes, mycoplasma, Brucella, or mycobacteria. You have seen some of this with Clifford Harding and others have proposed other observed the mechanics or function of other intracellular compounds ("in the mileux") being inhibited, even by Gary Wormser, et al.

http://www.ncbi.nlm.nih.gov/pubmed/?term=medvedev+ae+and+tolerance

J Innate Immun. 2016;8(2):171-84. doi: 10.1159/000440838. Epub 2015 Oct 13.
Endotoxin Tolerance Inhibits Lyn and c-Src Phosphorylation and Association with Toll-Like Receptor 4 but Increases Expression and Activity of Protein Phosphatases.
Xiong Y1, Murphy M, Manavalan TT, Pattabiraman G, Qiu F, Chang HH, Ho IC, Medvedev AE.

"Endotoxin tolerance protects the host by limiting excessive 'cytokine storm' during sepsis, but compromises the ability to counteract infections in septic shock survivors. It reprograms Toll-like receptor (TLR) 4 responses by attenuating the expression of proinflammatory cytokines without suppressing anti-inflammatory and antimicrobial mediators, but the mechanisms of reprogramming remain unclear. In this study, we demonstrate that the induction of endotoxin tolerance in human monocytes, THP-1 and MonoMac-6 cells inhibited lipopolysaccharide (LPS)-mediated phosphorylation of Lyn, c-Src and their recruitment to TLR4, but increased total protein phosphatase (PP) activity and the expression of protein tyrosine phosphatase (PTP) 1B, PP2A, PTP nonreceptor type (PTPN) 22 and mitogen-activated protein kinase phosphatase (MKP)-1. Chemical PP inhibitors, okadaic acid, dephostatin and cantharidic acid markedly decreased or completely abolished LPS tolerance, indicating the importance of phosphatases in endotoxin tolerization. Overexpression of PTPN22 decreased LPS-mediated nuclear factor (NF)-x03BA;B activation, p38 phosphorylation and CXCL8 gene expression, while PTPN22 ablation upregulated LPS-induced p65 NF-x03BA;B and p38 phosphorylation and the expression of TNF-α and pro-IL-1β mRNA, indicating PTPN22 as an inhibitor of TLR4 signaling. Thus, LPS tolerance interferes with TLR4 signaling by inhibiting Lyn and c-Src phosphorylation and their recruitment to TLR4, while increasing the phosphatase activity and expression of PP2A, PTPN22, PTP1B and MKP1.
http://www.ncbi.nlm.nih.gov/pubmed/26457672

A person who knows how to use the National Library of Medicine can follow up on all this. The NIH endorses it, as you have seen. And it's pretty ridiculous that IDSA thinks they can maintain the ruse that OspA was a vaccine and Lyme is only about a bad knee with these hundreds of reports that say the complete opposite is true.

X. **Multiple Sclerosis and EBV**? Let's look:
http://www.ncbi.nlm.nih.gov/pubmed/?term=multiple+sclerosis+and+virus+and+DNA

Some say yes, some say no, some say Cytomegalovirus, some say HHV-6, some say an EBV reactivated HERV, some say it's more than one herpes, and some say Viola! what do you know, immunosuppression from malaria seems to be associated with EBV-associated Burkitt's Lymphoma. Synergy. Another kind of another in-parallelism to our model. One infection invites the other, such as when in the old days it was known a cold virus could result in a dual bacterial infection and they gave children antibiotics to prevent a secondary ear infection. Or when in 1918 we had Spanish Flumonia, wherein one infection invited the other. Regardless, it seems to be unsettled as to which common virus or which two or which three, but it does seem to be a consensus that the herpesviruses are associated with MS.

You've seen NINDS basically settle on EBV, maybe HHV-6, too. It's something though, and like Chronic Fatigue Syndrome and Fibromyalgia and Lupus and all the other autoimmune and non-immune outcomes, they ALL start with a viral-like illness, people claim. There are 2 outcomes. Autoimmune and non-immune. The latter are not recognized, but Anthony Fauci, head if NIAID (National Institute of Allergy and Infectious Diseases) mentions it in his patent.

And you can notice also that there is no Opposite of NIAID, or no National Institute of Immunosuppression and Infectious Diseases or NIIID. No, can't have that. People would say, "Oh, so Cancer, Brain Damage from vaccine viruses, and the Wastebasket Diagnoses AKA Somatoformers, they all belong to NIIID, right? The 'failure of the immune system' classes of diseases as you call them?"

Y. What is **Bell's Palsy** caused by?
http://www.ncbi.nlm.nih.gov/pubmed/?term=bell%27s+palsy+and+Epstein-Barr

Some say EBV, some say Varicella, some say Simplex… Maybe it's not spirochetes, maybe it is spirochetes, maybe it is a herpesvirus, maybe it is a combination of herpesviruses, maybe it is herpesviruses and spirochetes. But given that more than one kind of spirochete is associated with Alzheimer's, and given that immunosuppression diseases are reactivation of COMMON VIRUSES, well, maybe that is the reason ILADS can't cure anyone. They don't know what they're doing and willfully do not look at the big picture.

Lyme spirochetes and EBV live in B cells and lymph nodes (use PubMed). And it just so happens, Rituximab, a bad-B-cell depleter works for Chronic Fatigue Syndrome (67% and 64% cure rate); adds much credibility to the idea that these _____ [insert waste basket /psych diagnosis word] diseases are about post sepsis immunosuppression and reactivated herpesviruses:

Z. **Rituximab**

PLoS One. 2011;6(10):e26358. doi: 10.1371/journal.pone.0026358. Epub 2011 Oct 19.
Benefit from B-lymphocyte depletion using the anti-CD20 antibody rituximab in chronic fatigue syndrome. A double-blind and placebo-controlled study.
Fluge Ø1, Bruland O, Risa K, Storstein A, Kristoffersen EK, Sapkota D, Næss H, Dahl O, Nyland H, Mella O.
http://www.ncbi.nlm.nih.gov/pubmed/22039471

PLoS One. 2015 Jul 1;10(7):e0129898. doi: 10.1371/journal.pone.0129898. eCollection 2015.
B-Lymphocyte Depletion in Myalgic Encephalopathy/ Chronic Fatigue Syndrome. An Open-Label Phase II Study with Rituximab Maintenance Treatment.
Fluge Ø1, Risa K1, Lunde S1, Alme K1, Rekeland IG1, Sapkota D2, Kristoffersen EK3, Sørland K1, Bruland O4, Dahl O5, Mella O5.
http://www.ncbi.nlm.nih.gov/pubmed/26132314

Could be about bad B cells, since the treatment fits the model. Ya think?

Remember now, Lyme causes Chronic Fatigue Syndrome and Fibromyalgia, says the Cabal. And 12 million people in the United States alone have those… things. So it can't be anything too mysterious if it also causes Lupus and MS and the Uncle Sam of Tardmerica ignores it.

AA. **The Yale "Lupus and Lyme Clinic"**

The NIH used to have an MS-Lyme section of the NINDS, and Yale used to have a "Lyme and Lupus Clinic" before that became the criminal entity "L2 Diagnostics," led by none other than Robert Schoen of "we can't tell LYMErix apart from multisystem late Lyme" infamy.

Steere (formerly at Yale) on Lyme and Lupus:

J Neurol Sci. 1993 Jul;117(1-2):206-14.
Reactivity of neuroborreliosis patients (Lyme disease) to cardiolipin and gangliosides.
García Moncó JC1, Wheeler CM, Benach JL, Furie RA, Lukehart SA, Stanek G, Steere AC.

"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."
http://www.ncbi.nlm.nih.gov/pubmed/8410057

Now the Yale Lyme and Lupus gang say this about Lupus (and EBV):

J Immunol. 2004 Jan 15;172(2):1287-94.
Defective control of latent Epstein-Barr virus infection in systemic lupus erythematosus.
Kang I1, Quan T, Nolasco H, Park SH, Hong MS, Crouch J, Pamer EG, Howe JG, Craft J.

"EBV infection is more common in patients with systemic lupus erythematosus (SLE) than in control subjects, suggesting that this virus plays an etiologic role in disease and/or that patients with lupus have impaired EBV-specific immune responses…Patients with SLE had an approximately 40-fold increase in EBV viral loads compared with controls, a finding not explained by disease activity or immunosuppressive medications. The frequency of EBV-specific CD69+ CD4+ T cells producing IFN-gamma was higher in patients with SLE than in controls…These results demonstrate that patients with SLE have defective control of latent EBV infection that probably stems from altered T cell responses against EBV."
http://www.ncbi.nlm.nih.gov/pubmed/14707107

BB. **China.** Remember Pam3Cys is the basic molecule of LYMErix or OspA and others shed by Borrelia:

PLoS One. 2014 Jan 28;9(1):e87528. doi: 10.1371/journal.pone.0087528. eCollection 2014.
A20 is critical for the induction of Pam3CSK4-tolerance in monocytic THP-1 cells.
Hu J1, Wang G2, Liu X1, Zhou L3, Jiang M1, Yang L4.

"A20 functions to terminate Toll-like receptor (TLR)-induced immune response, and play important roles in the induction of lipopolysacchride (LPS)-tolerance. However, the molecular mechanism for Pam3CSK4-tolerance is uncertain. Here we report that TLR1/2 ligand Pam3CSK4 induced tolerance in monocytic THP-1 cells. The pre-treatment of THP-1 cells with Pam3CSK4 down-regulated the induction of pro-inflammatory cytokines induced by Pam3CSK4 re-stimulation. Pam3CSK4 pre-

treatment also down-regulated the signaling transduction of JNK, p38 and NF-κB induced by Pam3CSK4 re-stimulation. The activation of TLR1/2 induced a rapid and robust up-regulation of A20, suggesting that A20 may contribute to the induction of Pam3CSK4-tolerance. This hypothesis was proved by the observation that the over-expression of A20 by gene transfer down-regulated Pam3CSK4-induced inflammatory responses, and the down-regulation of A20 by RNA interference inhibited the induction of tolerance. Moreover, LPS induced a significant up-regulation of A20, which contributed to the induction of cross-tolerance between LPS and Pam3CSK4. A20 was also induced by the treatment of THP-1 cells with TNF-α and IL-1β. The pre-treatment with TNF-α and IL-1β partly down-regulated Pam3CSK4-induced activation of MAPKs. Furthermore, pharmacologic inhibition of GSK3 signaling down-regulated Pam3CSK4-induced A20 expression, up-regulated Pam3CSK4-induced inflammatory responses, and partly reversed Pam3CSK4 pre-treatment-induced tolerance, suggesting that GSK3 is involved in TLR1/2-induced tolerance by up-regulation of A20 expression. Taken together, these results indicated that A20 is a critical regulator for TLR1/2-induced pro-inflammatory responses."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3905037/

TLR2/1-induced tolerance or LYMErix or Lyme tolerance is a *thing*, like Endotoxin Tolerance, only worse, since so far it is not reversible. In other words, IDSA and the CDC have no idea what they are talking about, and this concerns every major disease, if not every disease.

CC. **Poland**, 2013

Pol J Microbiol. 2013;62(3):237-42.
The influence of toll-like receptor stimulation on expression of EBV lytic genes.
Siennicka J1, Trzcińska A2, Cześcik A2, Dunal-Szczepaniak M2, Lagosz B2.

"Epstein-Barr virus (EBV) establishes latency in the resting memory B-cell compartment. It has been recently suggested that maintenance of chronic infection is dependent on periodic reactivation. Although the stimuli for EBV reactivation in vivo during natural infections are largely unknown, there is evidence indicating that heterologous infections could trigger herpesviruses reactivation. The purpose of this work was to identify the influence of Toll-like receptors stimulation on EBV replication in EBV latently infected Burkitt lymphoma cells (P3HR-1, Raji and Namalwa). The cells were stimulated with Pam3CSK4 (synthetic triacylated lipoprotein), PolyI:C (synthetic analog of dsRNA), LPS (lipopolysaccharide from E.coli), measles virus (MeV) and PMA (phorbol myristate acetate). Non-stimulated cells (NS) served as control. EBV expression was investigated at mRNA level for three viral lytic genes: BZLF1 (immediate early, ZEBRA), BALF2 (early, EA) and BcLF1 (late, VCA). Additionally, the effect of stimulation on NF-kBp65 and inflammatory cytokines (IL-lb, IL-6, IL-8, IL-10, IL-12p70, and TNF) was investigated. Stimulation of TLRs led to limited changes in EBV expression manifesting as increase of ZEBRA at mRNA level in cells treated with PolyI:C and Pam3CSK4. Stimulation with PolyI:C, Pam3CSK4 and LPS also lead to considerable increase of NF-kBp65, while increased levels of inflammatory cytokines were observed for IL-8, TNF and IL-6 in cells treated with PMA and MeV. **In conclusion, the results of our experiments support the suggestion that TLRs stimulation with microbial ligands influences EBV virus replication."**
http://www.ncbi.nlm.nih.gov/pubmed/24459828

DD. **Seronegative reactivated Epstein-Barr, and Clifford Harding again on how Pam3cys-ish molecules down-regulate the management of the TLRs that handle viruses**

Here are 4 examples from the literature of how Epstein-Barr also can be seronegative via the same mechanism of downregulation of antigen-presenting molecules or downregulation of HLA molecules (shows antigen so that B cells can make antibodies) or the MHC or "Major Histocompatibility Class" of cell components (all the same thing):

J Immunol. 2009 Feb 15;182(4):1799-809. doi: 10.4049/jimmunol.0802686.
Down-regulation of MHC class II expression through inhibition of CIITA transcription by lytic transactivator Zta during Epstein-Barr virus reactivation.
Li D1, Qian L, Chen C, Shi M, Yu M, Hu M, Song L, Shen B, Guo N.

The presentation of peptides to T cells by MHC class II molecules is of critical importance in specific recognition to a pathogen by the immune system. The level of MHC class II directly influences T lymphocyte activation. The aim of this study was to identify the possible mechanisms of the down-regulation of MHC class II expression by Zta during EBV lytic cycle. The data in the present study demonstrated that ectopic expression of Zta can strongly inhibit the constitutive expression of MHC class II and CIITA in Raji cells. The negative effect of Zta on the CIITA promoter activity was also observed. Scrutiny of the DNA sequence of CIITA promoter III revealed the presence of two Zta-response element (ZRE) motifs that have complete homology to ZREs in the DR and left-hand side duplicated sequence promoters of EBV. By chromatin immunoprecipitation assays, the binding of Zta to the ZRE(221) in the CIITA promoter was verified. Site-directed mutagenesis of three conserved nucleotides of the ZRE(221) substantially disrupted Zta-mediated inhibition of the CIITA promoter activity. Oligonucleotide pull-down assay showed that mutation of the ZRE(221) dramatically abolished Zta binding. Analysis of the Zta mutant lacking DNA binding domain revealed that the DNA-binding activity of Zta is required for the trans repression of CIITA. The expression of HLA-DRalpha and CIITA was restored by Zta gene silencing. The data indicate that Zta may act as an inhibitor of the MHC class II pathway, suppressing CIITA transcription and thus interfering with the expression of MHC class II molecules.
http://www.ncbi.nlm.nih.gov/pubmed/19201831

How many "doctors" know you can't rely on antibody testing to know if EBV has been reactivated? Right, I never met one or heard of one either.

Herpesviridae. 2011 Jan 5;2(1):1. doi: 10.1186/2042-4280-2-1.
Innate immune modulation in EBV infection.
Ning S1.

"Dysregulation of EBV-specific immune responses is also characteristic of EBV-associated autoimmune diseases such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). CTL response to EBV infection has been well documented since the discovery of EBV [11]. However, significant progresses in characterizing individual viral proteins involved in evasion of the T cell-mediated adaptive immune response have only been made in the last decade [12-16]. For example**, the functional homologue of human IL10,** BCRF1, elicits CD8+ T cell responses, and can be processed and presented to CD8+ CTLs through a TAP-independent pathway [17]."

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063194/?tool=pubmed

'A functional homolog of IL-10, the immune-suppressing cytokine. Awesome.

J Virol. 2002 Aug;76(16):8179-88.
The lytic cycle of Epstein-Barr virus is associated with decreased expression of cell surface major histocompatibility complex class I and class II molecules.
Keating S1, Prince S, Jones M, Rowe M.

Human herpesviruses utilize an impressive range of strategies to evade the immune system during their lytic replicative cycle, including reducing the expression of cell surface major histocompatibility complex (MHC) and immunostimulatory molecules required for recognition and lysis by virus-specific cytotoxic T cells. Study of possible immune evasion strategies by Epstein-Barr virus (EBV) in lytically infected cells has been hampered by the lack of an appropriate permissive culture model. Using two-color immunofluorescence staining of cell surface antigens and EBV-encoded lytic cycle antigens, we examined EBV-transformed B-cell lines in which a small subpopulation of cells had spontaneously entered the lytic cycle. Cells in the lytic cycle showed a four- to fivefold decrease in cell surface expression of MHC class I molecules relative to that in latently infected cells. Expression of MHC class II molecules, CD40, and CD54 was reduced by 40 to 50% on cells in the lytic cycle, while no decrease was observed in cell surface expression of CD19, CD80, and CD86. Downregulation of MHC class I expression was found to be an early-lytic-cycle event, since it was observed when progress through late lytic cycle was blocked by treatment with acyclovir. The immediate-early transactivator of the EBV lytic cycle, BZLF1, did not directly affect expression of MHC class I molecules. However, BZLF1 completely inhibited the upregulation of MHC class I expression mediated by the EBV cell-transforming protein, LMP1. This novel function of BZLF1 elucidates the paradox of how MHC class I expression can be downregulated when LMP1, which upregulates MHC class I expression in latent infection, remains expressed in the lytic cycle.
http://www.ncbi.nlm.nih.gov/pubmed/12134023

Remember, Chiu and Aucott says there is no change to immune genes expression. There is just the down-regulation of all mechanisms related to immune competence in the Post-Sepsis outcome of Lyme and LYMErix disease. Tardmerica may be stupid, but it's not boring.

Semin Cancer Biol. 2008 Dec;18(6):397-408. doi: 10.1016/j.semcancer.2008.10.008. Epub 2008 Oct 25.
Epstein-Barr virus evasion of CD8(+) and CD4(+) T cell immunity via concerted actions of multiple gene products.
Ressing ME1, Horst D, Griffin BD, Tellam J, Zuo J, Khanna R, Rowe M, Wiertz EJ.

"Evidence is accumulating that this paradoxical situation is the result of actions of multiple viral gene products, inhibiting discrete stages of the MHC class I and class II antigen presentation pathways. Immediately after initiation of the lytic cycle, BNLF2a prevents peptide-loading of MHC class I molecules through inhibition of the Transporter associated with Antigen Processing, TAP. This will reduce presentation of viral antigens by the large ER-resident pool of MHC class I molecules. Synthesis of new MHC class I molecules is blocked by BGLF5. Viral-IL10 causes a reduction in mRNA levels of TAP1 and bli/LMP2, a subunit of the immunoproteasome. MHC class I molecules present at the cell

surface are downregulated by BILF1. **Also the antigen presenting capacity of MHC class II molecules is severely compromised by multiple EBV lytic gene products, including gp42/gH/gL, BGLF5, and vIL-10.** In this review, we discuss how concerted actions of these EBV lytic proteins result in highly effective interference with CD8(+) and CD4(+) T cell surveillance, thereby providing the virus with a window for undisturbed generation of viral progeny."
http://www.ncbi.nlm.nih.gov/pubmed/18977445

Therefore, *never use antibody testing* to show an association between an illness and an infectious disease.

Clifford Harding says the chronic agonism of TLR2/1 by these lipoproteins also inhibit TLR7/9 function (manages the viruses like EBV); people want to know how Lyme and LYMErix activate EBV, besides that being about what happens commonly, in all general immunosuppression such as Humira and Stelara and post-transplant patients who acquired EBV-induced lymphoma, which we will get to:

J Immunol. 2012 Feb 1;188(3):1019-26. doi: 10.4049/jimmunol.1102181. Epub 2012 Jan 6.
TLR2 signaling depletes IRAK1 and inhibits induction of type I IFN by TLR7/9.
Liu YC1, Simmons DP, Li X, Abbott DW, Boom WH, Harding CV.

"Pathogens may signal through multiple TLRs with synergistic or antagonistic effects on the induction of cytokines, including type I IFN (IFN-I). IFN-I is typically induced by TLR9, but not TLR2. Moreover, we previously reported that TLR2 signaling by Mycobacterium tuberculosis or other TLR2 agonists inhibited TLR9 induction of IFN-I and IFN-I-dependent MHC-I Ag cross processing. The current studies revealed that lipopeptide-induced TLR2 signaling inhibited induction of first-wave IFN-α and IFN-β mRNA by TLR9, whereas induction of second-wave IFN-I mRNA was not inhibited. TLR2 also inhibited induction of IFN-I by TLR7, another MyD88-dependent IFN-I-inducing receptor, but did not inhibit IFN-I induction by TLR3 or TLR4 (both Toll/IL-1R domain-containing adapter-inducing IFN-β dependent, MyD88 independent). The inhibitory effect of TLR2 was not dependent on new protein synthesis or intercellular signaling. IL-1R-associated kinase 1 (IRAK1) was depleted rapidly (within 10 min) by TLR2 agonist, but not until later (e.g., 2 h) by TLR9 agonist. Because IRAK1 is required for TLR7/9-induced IFN-I production, we propose **that TLR2 signaling induces rapid depletion of IRAK1, which impairs IFN-I induction by TLR7/9. This novel mechanism, whereby TLR2 inhibits IFN-I induction by TLR7/9, may shape immune responses to microbes that express ligands for both TLR2 and TLR7/TLR9, or responses to bacteria/virus coinfection."**
http://www.ncbi.nlm.nih.gov/pubmed/22227568

OspA and Borrelia render you unable to manage viral infections by the viral-managing TLRs.

"Won-der-ful" as the rich people in Fairfield Country, Corrupticut like to say.

EE. The **Stelera and Humira** and other mab (monoclonal antibody) commercials

We've all seen them. They warn particularly against fungal infections, against taking immune suppressing drugs like steroids, and that there is a risk of Lymphoma. Well, what causes Lymphoma?

http://www.ncbi.nlm.nih.gov/pubmed/?term=immunosuppression+and+lymphoma+and+epstein-barr

Over 600 articles. Could be a thing. A thing like, you know the Chronic Fatigue Syndrome patients who ended up with cancer, and who were then treated with Rituximab and to-everyone's-surprise-except-us…

FF. **Raymond Dattwyler's Flumonia (almost) patent**. Dattwyler is so convinced LYMErix causes immunosuppression he proposes to use it in combination with a virus for an inhalation form something we proposed years ago, given the pandemic flu of 1918 killed people due to the secondary infection, the mycobacteria, or flumonia. It only killed healthy people, remember, people with strong immune responses. Therefore, he thinks it could be an inoculum or a tolerizer against the serious septic shock event (but in the lungs) that results in death or near death for the post-sepsis survivors of Lyme and LYMErix:

"A lipidation/processing reaction has been described for the intact OspA gene of B. burgdorferi. The primary translation product of the full-length B. burgdorferi OspA gene contains a hydrophobic N-terminal sequence, of 16 amino acids, which is a substrate for the attachment of a diacyl glyceryl to the sulflhydryl side chain of the adjacent cysteine (Cys) residue (at position 17). Following this attachment, cleavage by signal peptidase II and the attachment of a third fatty acid to the N-terminus occurs. The completed lipid moiety, a tripalmitoyl-S-glycerylcysteine modification, is termed Pam3Cys (or is sometimes referred to herein as Pam(3)Cys or Pam3Cys). It has been suggested that the lipid modification allows membrane localization of proteins, with polypeptide portions exposed as immune targets. In addition to serving as targets for the immune response, Pam3Cys-modified proteins, such as OspA, have been reported to act as potent inflammatory stimulants though the toll-like 2 receptor mechanism (TLR2).
http://patentscope.wipo.int/search/en/detail.jsf?docId=US42934470&recNum=9&maxRec=30&office&prevFilter&sortOption=Pub+Date+Desc&queryString=tripalmitoyl+cysteine+or+Pam3Cys+and+Epstein-Barr&tab=NationalBiblio

Dattwyler says OspA is Pam3Cys and is a TLR2 agonist. So far, he is the only one who has openly admitted LYMErix never could have been an injectable vaccine. Or even admitted said what it was (Pam3Cys). HHS.gov claims to not know. Yale says they do not know what OspA is (it was their vaccine, LYMErix), the CDC said they do not know what OspA is, Paul Auwaerter said he does not know what OspA is, NIH Director Francis Collins did not know, NIAID director Anthony Fauci did not know, and IDSA refused to reply to our emails or phone calls.

"Here, take this here vaccine. We don't know what it, OspA, is. And just about no one has this disease it prevents. And when they do, like Wormser and Klemper said, the people only have arthritis and no other symptoms.
"Thanks and have a nice day,
"--- HHS.gov, IDSA, The Entire U.S. and Western Medical Establishment, et al."

GG. It being **empirically observed** that Lyme helps activate EBV:

Folia Biol (Praha). 2003;49(1):40-8.
Interaction of Borrelia burgdorferi sensu lato with Epstein-Barr virus in lymphoblastoid cells.

Hulínská D1, Roubalová K, Schramlová J.

”Since the possibility of interruption of latent EBV infection has been suggested by the induction of the lytic virus cycle with chemical substances, other viruses, and by immunosuppression, we hypothesized that the same effect might happen in B. burgdorferi sensu lato infection as happens in Lyme disease patients with positive serology for both agents. We have observed EBV replication in lymphoblastoid cells after superinfection with B. garinii and B. afzelii strains after 1 and 4 h of their interaction. We found that viral and borrelial antigens persisted in the lymphoblasts for 3 and 4 days. Morphological and functional transformation of both agents facilitate their transfer to daughter cells. Association with lymphoblasts and internalization of B. garinii by tube phagocytosis increased replication of viruses more successfully than B. afzelii and chemical inductors. Demonstration of such findings must be interpreted cautiously, but **may prove a mixed borrelial and viral cause of severe neurological disease**.”
http://www.ncbi.nlm.nih.gov/pubmed/12630667

HH. It being **empirically observed** that the Cabal has observed that Lyme causes immunosuppression:

Borrelia burgdorferi-induced tolerance as a model of persistence via immunosuppression
http://www.ncbi.nlm.nih.gov/pubmed/12819085

Here they are citing
it: http://www.ncbi.nlm.nih.gov/pubmed?linkname=pubmed_pubmed_citedin&from_uid=12819085
(Auwaerter, Fish, Krause, Radolf)

II. Mario Philipp (Tulane) has for years said OspA was associated with the production of the immunosuppressive cytokine, IL-10 (this was mentioned to the FDA by Dickson, Jan 31, 2001)

http://www.ncbi.nlm.nih.gov/pubmed/?term=Philipp+and+OspA+and+il-10

Infect Immun. 2006 Oct;74(10):5780-9.
Interleukin-10 anti-inflammatory response to Borrelia burgdorferi, the agent of Lyme disease: a possible role for suppressors of cytokine signaling 1 and 3.
Dennis VA1, Jefferson A, Singh SR, Ganapamo F, Philipp MT.

”It has been established that interleukin-10 (IL-10) inhibits inflammatory cytokines produced by macrophages in response to Borrelia burgdorferi or its lipoproteins. The mechanism by which **IL-10** exerts this anti-inflammatory effect is still unknown. Recent findings indicate that suppressors of cytokine signaling (SOCS) proteins are induced by cytokines and Toll-like receptor (TLR)-mediated stimuli, and in turn they can down-regulate cytokine and TLR signaling in macrophages. Because it is known that SOCS are induced by IL-10 and that B. burgdorferi and its lipoproteins most likely interact via TLR2 or the heterodimers TLR2/1 and/or TLR2/6, we hypothesized that SOCS are induced by IL-10 and B. burgdorferi and its lipoproteins in macrophages and that SOCS may mediate the inhibition by IL-10 of concomitantly elicited cytokines. We report here that mouse J774 macrophages incubated with IL-10 and added B. burgdorferi spirochetes (freeze-thawed, live, or sonicated) or lipidated outer surface protein A (L-OspA) augmented their SOCS1/SOCS3 mRNA and protein expression, with SOCS3

being more abundant**. Pam(3)Cys, a synthetic lipopeptide, also induced SOCS1/SOCS3 expression under these conditions, but unlipidated OspA was ineffective. Neither endogenous IL-10 nor the translation inhibitor cycloheximide blocked SOCS1/SOCS3 induction by B. burgdorferi and its lipoproteins, indicating that the expression of other genes is not required. This temporally correlated with the IL-10-mediated inhibition of the inflammatory cytokines IL-1beta, IL-6, IL-12p40, IL-18, and tumor necrosis factor alpha.** Our data are evidence to suggest that expression of SOCS is part of the mechanism of IL-10-mediated inhibition of inflammatory cytokines elicited by B. burgdorferi and its lipoproteins."
http://www.ncbi.nlm.nih.gov/pubmed/16988256

Pam3Cys Or OspA never could have been a vaccine. That is what he is (still) saying. Who else says it? Not anybody we know who works for the Gubbamint, officially. Nobody who works for IDSA. No one at Yale. No one in ILADS. Not in a hat, not with a bat, not on TV, not in a snarkblog, not in a regular blog, not in any institute. Not in a house with a mouse or boat with a goat. Dr. Seuss was not writing children's books. Dr-Seuss-Oh-the-Places-You'll-Go!!! (People as footsy and brainy as they!)

Epstein-Barr is known to have a human homolog of IL-10 and down-regulates the MHC or antigen-presenting cells and may be antibody-negative or seronegative in active disease. These could be 2 more reasons EBV contributes to so many cancers – in the Subimmune Class of diseases -, as well as its well-known association to Autoimmune diseases. Fungal infections contribute to all Great Imitator Autoimmune and Great Imitator No-immune diseases like cancer, also.

JJ. Lymphoma and leukemia in **TRANSPLANT recipients** (from reactivated EBV, et al, from the immmunosuppression drugs they must take)

https://www.ncbi.nlm.nih.gov/pubmed/?term=organ+transplant+and+epstein-barr+and+(leukemia+or+lymphoma)

A mere 818 reports to date (February, 2017)

KK. **Coxsackie in Chronic Fatigue Muscles and Ticks**, also very cute.

Br Med Bull. 1991 Oct;47(4):852-71.
Persistent virus infection of muscle in postviral fatigue syndrome.
Cunningham L1, Bowles NE, Archard LC.

"Nucleic acid was extracted from muscle biopsy samples from a series of highly selected patients suffering from chronic muscle fatiguability following a viral infection (Postviral Fatigue Syndrome: PVFS). Samples were examined for the presence of enteroviral RNA sequences or Epstein-Barr (EBV) virus DNA sequences by molecular hybridisation as these two agents have been implicated by retrospective serology in the aetiology of PVFS. We found enteroviral RNA in 24% of biopsy samples and EBV DNA in a further 9% of biopsy samples: no biopsy was positive for both enteroviral RNA and EBV DNA. In addition, in the case of enteroviruses we found that the persisting virus is defective in control of RNA replication as both strands of enteroviral RNA are present in similar amounts: this is

unlike the asymmetric synthesis of genomic RNA seen in a productive, cytolytic enterovirus infection. The implications of these data in relation to mechanisms of viral persistence and muscle dysfunction are discussed."
https://www.ncbi.nlm.nih.gov/pubmed/1665379

"Diseases caused by enterovirus infection (Cocksackie, Foot and Mouth Disease)

Poliomyelitisprimarily via the fecal-oral route
Polio-like syndromefound in children who tested positive for enterovirus 68.[23][24]
Nonspecificfebrile illness is the most common presentation of enterovirus infection. Other than fever, symptoms include muscle pain, sore throat, gastrointestinal distress/abdominal discomfort, and headache. In newborns the picture may be that of sepsis however, and can be severe and life-threatening.
Enteroviruses are by far the most common causes ofaseptic meningitis in children. In the United States, enteroviruses are responsible for 30,000 to 50,000 meningitis hospitalizations per year as a result of 30 million to 50 million infections.[2]
Bornholm diseaseor epidemic pleurodynia is characterized by severe paroxysmal pain in the chest and abdomen, along with fever, and sometimes nausea, headache, and emesis.
Pericarditisand/or myocarditis are typically caused by enteroviruses; symptoms consist of fever with dyspnea and chest pain. Arrhythmias, heart failure, and myocardial infarction have also been reported.
Acute hemorrhagic conjunctivitiscan be caused by enteroviruses.
Herpanginais caused by Coxsackie A virus, and causes a vesicular rash in the oral cavity and on the pharynx, along with high fever, sore throat, malaise, and often dysphagia, loss of appetite, back pain, and headache. It is also self-limiting, with symptoms typically ending in 3–4 days.
Hand, foot and mouth diseaseis a childhood illness most commonly caused by infection by Coxsackie A virus or EV71.
Encephalitisis rare manifestation of enterovirus infection; when it occurs, the most frequent enterovirus found to be causing it is echovirus 9.
A 2007 study suggested that acute respiratory or gastrointestinal infections associated with enterovirus may be a factor inchronic fatigue syndrome.[25]
Diabetes mellitus type 1It has been proposed that type 1 diabetes is a virus-triggered autoimmune response in which the immune system attacks virus-infected cells along with the insulin-producing beta cells in the pancreas.[26] A team working at University of Tampere, Finland has identified a type of enterovirus that has a possible link to type 1 diabetes (which is an autoimmune disease).[27][28]

From https://en.wikipedia.org/wiki/Enterovirus

More on enteroviruses, possibly in ticks:
https://www.ncbi.nlm.nih.gov/pubmed/?term=enterovirus+and+ticks

Are people getting foot and mouth disease from Plum Island-escaped ticks, too (Plum Island has always experimented with Hoof and Mouth disease)? Imagine how sick people are with Lyme, if they have all these combined devastating illnesses? Yet, we're all trashed aren't we? Are we trashed because this is crime or are we trashed because we represent a bioweapons experiment (escaped ticks) gone horribly wrong? Why is the CDC lying about all this? For CDC personnel/staff vaccines incomes reasons? Has this scam gone on so long the CDC and NIH finds no way of backing away from all their lies? Is the

HHS.gov mortified at the prospect at having been discovered to 200% incompetent to their mission?

J Clin Microbiol. 2005 Jul;43(7):3471-3.
Possible tick-borne human enterovirus resulting in aseptic meningitis.
Freundt EC1, Beatty DC, Stegall-Faulk T, Wright SM.

"Enterovirus-specific genetic sequences were isolated from two **Amblyomma americanum** tick pools. Identical genetic sequences were later obtained from cerebrospinal fluid of a patient with aseptic meningitis and a recent history of tick attachment. These observations suggest the possibility of an emerging tick-borne human enterovirus associated with aseptic meningitis."
https://www.ncbi.nlm.nih.gov/pubmed/16000481

But everyone who says tick bites cause chronic disease is called crazy, including Edwin Masters.

But get the cabal's vaccines.

And, '"Oh, the poor ALDF.com racketeers, they're victims of anti-vaxxers (which was not even a thing at the time LYMErix was yanked, per the FDA)."

One minute it's our fault for getting rid of LYMErix, and the next minute no one has any kind of real disease anyway. This is them. The experts. At making everyone's head spin.

MM. Lymphomas initiating with exposure to fungal antigens:

J Exp Med. 2013 Jan 14;210(1):59-70. doi: 10.1084/jem.20121801. Epub 2013 Jan 7.
A mutated B cell chronic lymphocytic leukemia subset that recognizes and responds to fungi.
Hoogeboom R1, van Kessel KP, Hochstenbach F, Wormhoudt TA, Reinten RJ, Wagner K, Kater AP, Guikema JE, Bende RJ, van Noesel CJ.

B cell chronic lymphocytic leukemia (CLL), the most common leukemia in adults, is a clonal expansion of CD5(+)CD19(+) B lymphocytes. Two types of CLLs are being distinguished as carrying either unmutated or somatically mutated immunoglobulins (Igs), which are associated with unfavorable and favorable prognoses, respectively. More than 30% of CLLs can be grouped based on their expression of stereotypic B cell receptors (BCRs), strongly suggesting that distinctive antigens are involved in the development of CLL. Unmutated CLLs, carrying Ig heavy chain variable (IGHV) genes in germline configuration, express low-affinity, poly-, and self-reactive BCRs. However, the antigenic specificity of CLLs with mutated IGHV-genes (M-CLL) remained elusive. In this study, we describe a new subset of M-CLL, expressing stereotypic BCRs highly specific for β-(1,6)-glucan, a major antigenic determinant of yeasts and filamentous fungi. β-(1,6)-glucan binding depended on both the stereotypic Ig heavy and light chains, as well as on a distinct amino acid in the IGHV-CDR3. Reversion of IGHV mutations to germline configuration reduced the affinity for β-(1,6)-glucan, indicating that these BCRs are indeed affinity-selected for their cognate antigen. Moreover, CLL cells expressing these stereotypic receptors proliferate in response to β-(1,6)-glucan. This study establishes a class of common pathogens as functional ligands for a subset of somatically mutated human B cell lymphomas.
https://www.ncbi.nlm.nih.gov/pubmed/23296468

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http://www.ncbi.nlm.gov/pubmed/23296468

Cute. You can see how dangerous it is to have stupid criminals at the CDC, Yale, NYMC, NIH and elsewhere be in charge of something called a “GREAT” “Imitator” and for medical schools not to require a science pre-med Bachelors degree.

Chronic Lyme can't be about spirochetes and biofilms and co-infections (Oh, My!) if LYMErix vaccination caused the exact same systemic and neurologic disease as Lyme. The following are scientists who know what they are talking about regarding Lyme/spirochetes and OspA as immunosuppressive.

Notice that none of the Lyme "non-profits" tell you what Lyme and Lyme cryme are all about. They do not want anything to change. They are happy about all the people who die from Lyme disease as long as their "CEOs" make several hundred thousand dollars a year for doing nothing but being blowhard self-promoters.

Sick.

