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THE CRIMINAL CHARGE SHEETS

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Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

The Criminal Charge Sheets:

1. ALDF-CDC Enterprise Conspires to Defraud USA in Dearborn-Vaccines Scam

Details on the Lyme Cabal and the RICO charges; How the Lyme "case definition" was falsified at the Dearborn "Consensus Conference" to facilitate the enterprise.

2. The Lyme Disease Patents

Lyme disease patents owned by the Dearborn scammers, CDC officers, Yale in association with Corixa, Mayo Clinic and Imugen. Leaving OspA and B out of the Dearborn standard was intended to facilitate a monopoly on post-LYMERix approval on blood testing for all vector-borne disease.

3. Lyme Disease Biomarkers

Lyme Disease Biomarkers, as compared to scientifically invalid psychiatric check lists. These biomarkers were identified by the very people who later said Lyme was not even a disease, and who are the same people who own the vaccine patents and falsified the testing at Dearborn.

4. The Primers Shell Game

The very people who own all the patents and falsified the testing for Lyme in order to falsify the outcomes of those bogus products, use the wrong DNA to not-find Lyme or other spirochetes in humans, while using the correct DNA to patent borrelia-specific DNA.

5. Occam's Razor

The very people who own all the patents and falsified the testing for Lyme in order to falsify the those bogus products, use the wrong DNA to not-find Lyme or other spirochetes in humans, while using the correct DNA to patent borrelia-specific DNA.

6. The Common Mechanisms of Fungal-Viral Damage in C FIDS, Vaccines-Autism, and "Chronic Lyme"/ New Great Imitator, per the CDC, NIH and IDSA

This paper reveals the CDC's own data on how immunosuppression and fungal contamination explain the failed childhood vaccines, giving children the very viruses that the vaccines are intended to prevent (with resultant encephalitis); plus, how this mechanism parallels Lyme, CFS/ME, et al, AKA Post Sepsis Syndrome.

7. Assaulting Czech Children

The State of Connecticut and Yale Assaulted Czech Children with a known fake vaccine (OspA or LYMERix) just to see how serious would be the adverse events:

8) Gulf War Veterans' Abuse

Simon Wessely and the abuse of Gulf War veterans, Justina Pelletier and 21st century witch trials; with scientifically valid evidence for real illness, a vast majority of post-sepsis and vaccine injured persons are slandered and libeled with invalid psychiatric terminology:



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CHARGE SHEET 1: THE LYME ENTERPRISE

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

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Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 1: The Lyme Enterprise

1.) The “Enterprise” (a [RICO](#) term) is the “American Lyme Disease Foundation.” Here we will refer to them as “the Cabal.”

The testing for Lyme disease was falsified to pass off fake vaccines and test kits for the “Enterprise ”

Note: You are going to see a lot of redundancy in these charge sheets. This is necessary because the crime is multi-dimensional. We have to show what certain people did, how they did it, and why they did it. There are CDC staff patent owners who publish slander and libel, there are slanderers and libelers who also publish scientifically valid biomarkers of nerve and brain degradation in Lyme victims. There are people who play the Primers Shell Game but who also published that the OspA vaccines cause immunosuppression and also slander and libel their victims. There are people who assaulted Czech children with a known fake Lyme vaccine that would do no good for Europeans, since there is none of the American (Yale’s) LYMERix kind of OspA in Europe, who also slander and libel.

In 1995 Yale’s Robert Schoen and Mayo Clinic’s David Persing together worked on and published a method for the detection of “Lyme disease” with a strain of *Borrelia* that had dropped the OspA-B plasmid (PubMed, or PMID # [8968914](#)) that Persing also patented (USPTO # [6,045,804](#)). In that patent, Persing states that you can’t tell the difference between late, “multi-system” Lyme and LYMERix injury (they both are essentially the same as post-septic shock). In the same patent, they state that this testing method would be useful especially after LYMERix or an OspA vaccine was on the market because it does not have the OspA-B plasmid, and therefore it would not matter if the OspA or B antibodies were present and come from a vaccinated person. One could just ignore those “primary, immunodominant antigens.” If the tested *Borrelia* strain does not have those antigens and they show up in the Western Blot of a patient, one can discount those antibodies and see if there are other bands present, which would mean the person had been bitten by a tick and got Lyme. This was the reason Steere committed research fraud in Europe to assure OspA and B would be left out of the U. S. Center for Disease Control and Prevention’s (CDC’s) diagnostic criteria for Lyme (which we call “Dearborn”).

The associated companies involved in this RICO-within-the-RICO, the ones licensed to use this Post-LYMERix criminal method (*Borrelia* without the OspA-B plasmid), were Persing’s new adjuvant company, Corixa, Yale’s L2 Diagnostics, and Imugen, in Norwood, MA. In other words, Steere falsified the testing in Europe to assure that this RICO cabal would be the only companies in North America (yes, they mentioned Canada, too) to be able to receive blood for Vector Borne Diseases (VBDs) testing, and thereby have access to all the new VBDs to **ALSO** patent. This whole Dearborn scam was about an intended monopoly on testing and DNA products, test kits and vaccines. Everything, the whole scam, depended on Yale’s LYMERix vaccine being on the market. Obviously there will be a lot of overlap in these chapters or charge sheets citing the citations and patents.

Etc. We hope the redundancy will also help with learning about and understanding these crimes.

And, before we go any further, you want to meet the World's New Best Friend, OspA (or LYMERix, or Pam3Cys), because this molecule given what it is/does, not only explains the Autism (brain damage is the more correct term)-from-Vaccines-Pandemic, but why the U.S. Government staff employees trash, stalk, harass and deny all access to care [Deprivation of Rights Under Color of Law], people with Chronic Fatigue Syndrome, Lyme, Myalgic Encephalomyelitis, Fibromyalgia, Gulf War Illness and so on with the "syndromes."

Biochemistry: Defoort *et al.*

Proc. Natl. Acad. Sci. USA 89 (1992) 3881

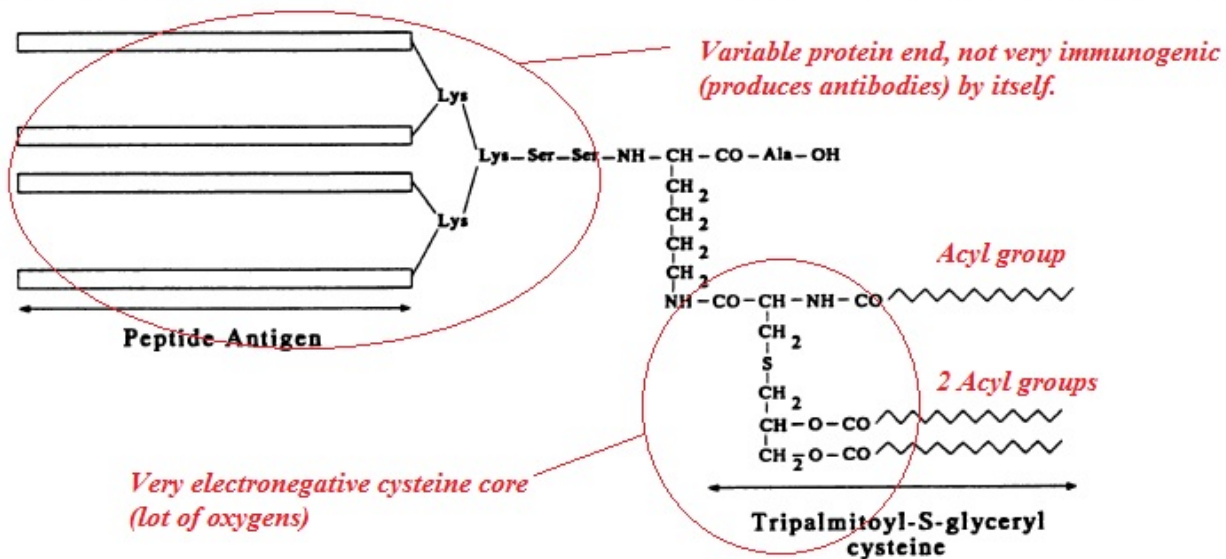


FIG. 1. Schematic representation of MAP-P3C.

Image from a hypothetical HIV vaccine with Pam3Cys or Tri-Palmitoyl Cysteine attached:

[Int J Pept Protein Res.](#) 1992 Sep-Oct;40(3-4):214-21.

A rational design of synthetic peptide vaccine with a built-in adjuvant. A modular approach for unambiguity.

Defoort JP1, Nardelli B, Huang W, Tam JP.

<https://www.ncbi.nlm.nih.gov/pubmed/1478779>

You can discover on your own that Pam3Cys is managed by TLRs 2 and 1, but we will show many references here that prove this. Something that is triacylated and managed by TLRs 2 and 1 could never have been and was never a "vaccine." It was the opposite, a fungal endotoxin more toxic than lipopolysaccharide (LPS), a TLR4 agonist.

Chronology:

Originally, Lyme borrelia were perceived by the U. S. Centers for Disease Control and Prevention (CDC) to be just another group of Relapsing Fever organisms. Borreliae (the whole genus) undergo constant antigenic variation, making vaccines and valid testing impossible except for detection via an anti-flagellar antibody method. Chapter 5, the DNA Primers Shell Game, explains more about the genetics.

At some point, it was decided by CDC officers that they should commercialize Lyme and other emerging, tick-borne diseases by patenting vaccines and test kits based on recombinant antigens, anyway. No one knows who gave the CDC the authority to do this, but this decision coincided with the establishment of the fake non-profit, the **American Lyme Disease Foundation** (ALDF.com), Valhalla, NY, in 1990, by Edward McSweegan, Durland Fish, Gary Wormser, and John J. Connolly, the then-president of **New York Medical College** (NYMC) in association with **Kaiser-Permanente** (KP). KP is still at NYMC writing MD-training modules. The CDC is often found in collaboration with KP; we knew this even before their fake “Morgellon’s investigation.”

The ALDF.com is a Government-Defrauding, Racketeering, and “Deprivation of Rights under Color or Law” organization, where the wealthy “sponsors” were apparently given some inside information regarding the companies that would be manufacturing the bogus recombinant vaccines and test kits. Those companies appeared to have been given some assurance against the prosecution of the testing scam necessary to pass off these bogus recombinant products. The Cabal, via changing the diagnostic standard, claimed Lyme was not just another Relapsing Fever organism, but some entirely different disease. Yet, spirochetes were for the last 100+ years known to be permanent brain and lymph node infections, and that rodent brains used to be the formal storage media (Barbour, 1986) before the CDC learned how to freeze-dry spirochetes in 1964:

[Microbiol Rev.](#) 1986 Dec;50(4):381-400.

Biology of Borrelia species.

[Barbour AG, Hayes SF.](#)

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC373079/pdf/microrev00055-0033.pdf>

And:

[J Bacteriol.](#) 1964 Sep;88:811.

RECOVERY OF TREPONEMA AND BORRELIA AFTER LYOPHILIZATION.

[HANSON AW, CANNEFAX GR.](#)

<https://www.ncbi.nlm.nih.gov/pubmed/14208528>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC277387/pdf/jbacter00438-0287.pdf>

Everyone will recall the **Tuskegee “Bad Blood”** experiment was precisely about the dementia experienced by Caucasians as opposed to people with an African background, while the “Enterprise” says Lyme borreliosis (borrelia are more virulent than treponemes) cause only autoimmune arthritis in a knee.:

[Sex Transm Dis.](#) 2014 Jul;41(7):440-6. doi: 10.1097/OLQ.000000000000149.

Toll-like receptor polymorphisms are associated with increased neurosyphilis risk.

Marra CM1, Sahi SK, Tantaló LC, Ho EL, Dunaway SB, Jones T, Hawn TR.

"Clinicians in the early 20th century posited that race influenced susceptibility to neurosyphilis, citing a decreased risk in African Americans compared to Caucasians (7). Subsequent work suggested a genetic basis for such differences, with an increased risk of syphilitic dementia, but not other forms of neurosyphilis, in patients with certain HLA types (8) that differed in African Americans compared to Caucasians (9). While more recent reports suggest that there may be genetic contributions to syphilis susceptibility (10-13), to the best of our knowledge there have been no recent investigations of genetic susceptibility to neurosyphilis."

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4414322/>

More background or old news about borrelia:

Br Med J. 1911 Apr 1;1(2622):752.

***THE INFECTIVE GRANULE IN CERTAIN PROTOZOAL INFECTIONS,
AS ILLUSTRATED BY THE SPIROCHAETOSIS OF SUDANESE FOWLS.***

Balfour A.

"AT the first meeting of the Tropical Medicine Section of the British Medical Association in London last year I advanced the view that, in all probability, what might be called the "infective granule" would yet be found to play an important part in certain protozoal infections, and more especially in spirochaetosis and trypanosomiasis. I based this belief on the work of Leishman as regards the changes undergone by Spirochaeta duttonii in Ornithodoros mioubata, and on the allied changes which I had found to occur in the Sudan fowl spirochaete when ingested by Argas persicus. I have been continuing the work on fowl spirochaetosis and have recently arrived at some most interesting and significant results, which may yet have considerable bearing on the view we must take of the pathology of this and other spirochaetal diseases, and possibly also on their treatment.

"The full account of these later researches will be presented in the fourth report of these laboratories, which is now in the press, and is due to appear in the autumn of the present year; here I wish merely to place on record a few of the more salient features of the work.

"It will perhaps be remembered that one found intracorporeal forms in this fowl spirochaetosis, and that, following Sambon, one had come to the conclusion that these endoglobular bodies represented a stage in the life cycle of the spirochaete—constituted, in short, its stage of schizogony in the fowl. Sambon, however, who expressed this view from the study of a few slides I gave him, did not indicate how this red cell invasion occurred. For a long time I believed the spirochaetes themselves entered the red cells and broke up, or coiled up, within them to form these remarkable bodies. As the parasites can and do enter and leave the erythroblasts of the fowl, there was good ground for this supposition. Now, however, I know better.

"By the use of the dark-field method, and more especially by practising liver puncture on chicks at the crisis or on chicks which have been given a sufficiently large dose of salvarsan, I have found that in the liver in particular, also in the spleen and lung, the spirochaetes undergo an astonishing change. They discharge from their periplastic sheaths spherical granules, and it is

apparently these granules which enter the red cells, develop in them and complete a cycle of schizogony. The appearance is very remarkable. If a well-infected chick be given a dose of salvarsan, the peripheral blood is soon cleared, or nearly cleared, of spirochaetes. If then a drop of liver juice be examined by the dark-field method, it will be found swarming with spirochaetes and with highly refractile granules. The source of the latter is soon apparent, for attention will be directed to spirochaetes which are not moving in the usual way, but are' in a state of violent contortion, or are, so to speak, shaking themselves to and fro. Indeed, I cannot give a more apt comparison than by likening their movements to those of dogs which have been in water and are shaking themselves vigorously to dry their coats. The object of the spirochaetes, however, is to rid themselves of the bright, spherical granules which can be seen within them, and which may or may not be aggregations of the so-called chromatin core. These are forced along the periplastic sheath and suddenly discharged, so that they become free in the medium and dance hither and thither as tiny, solid, spherical, brilliantly white particles. In process of time the spirochaete loses its activity, becomes difficult to see, and eventually all that is left of it is the limp and lifeless sheath drifting aimlessly in the fluid and liable to be caught up and swept away by some still vigorous parasite. Such a sheath may still retain one or two of the granules which it has been unable to discharge.

“As may be imagined, the process is most fascinating to watch, and my observations have been confirmed by Captain Fry and Mr. Buchanan, of these laboratories, and by Captain O'Farrell, R.A.M.C. I may also say that the first-named had previously seen a shedding-off of granules by trypanosomes in the peripheral blood of experimental animals, a phenomenon which he is now - studying.

“It is these spirochaete granules in the liver, spleen, and lung, and possibly also in other internal organs, which, I believe, invade the red cells. I think I have seen the penetration occur, but require to make further observations in order to be certain as to the mode of entry. Such a chain of events fully explains all the puzzling features which this intracorpuseular infection has hitherto presented, and, moreover, brings it into line with the infective granules found in the ticks, for these very closely resemble those seen in liver juice films both when examined by the dark-field method and when stained by the Levaditi process. There are various other pVints, more especially as regards the peculiar staining reactions of these granules, into which I need not enter beyond saying that the fact that, when free, they do not appear to take on the Romanowsky stain may explain why they have not previously been noticed. The work is also not yet complete, as it is necessary to find out if the spirochaetes ingested by ticks behave in a similar manner and thereby produce the granules of Leishman.

“I see that Jowett in South Africa has recently discovered what appears to be an identical form of fowl spirochaetosis, and I trust he will employ the dark-field method and endeavour by liver puncture and the use of salvarsan, for the purpose of creating an artificial crisis, to follow out the curious cycle I have indicated.

“From these observations and others which will be fully detailed at a later date I have come to the conclusion that this fowl spirochaete must be classed as a specific entity, and I am proposing for it the name *S.pirochaeta granulo8a penetrans*, which, though lengthy, suitably indicates its more important peculiarities. At the same time it is quite possible-nay, even probable-that other pathogenic spirochaetes behave in a similar manner. I have found these granules to be resistant forms, and their presence in countless num-bers in the tissues might explain part of the mechanism of relapse and the

difficulty of curing completely some of the more chronic spirochaetal infections, as, for example, syphilis and yaws.

“In conclusion, I must thank Professor Ehrlich for most kindly placing at my disposal an ample supply of his new and valuable remedy.”

<https://www.ncbi.nlm.nih.gov/pubmed/20765548>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2333723/pdf/brmedj07824-0022.pdf>

Recall now, if you aren't recalling already, the remarks of Willy Burgdorfer in the “Under Our Skin” movie interview that you can't see borrelia in the blood, confirming the observations of 1911, above:

”Dr Willy Burgdorfer granted an interview (which was supervised by staff from the Rocky Mountain Laboratory, National Institutes of Health, NIH). Excerpts from that interview, concerning the circumstances of his discovery of the spirochetal agent of Lyme borreliosis:

“Excerpt from an Interview with Dr Willy Burgdorfer;

“It was a ‘What in the hell? What's in that smear?’ And then my work [on relapsing fever] as a Swiss student came back. [I said to myself], ‘Willy, these are spirochetes!’ The slide showed long slender forms, a little bit curved, and they were only in the mid-part of the tick. Nowhere else. There were so many people who said, ‘That is impossible Willie. You can't get spirochetes out of hard-bodied ticks.’ [But from my work on] relapsing fever ticks from Africa, I knew what a spirochete looked like. The Belgian Congo and Kenya are hotspots for relapsing fever. Even Livingston [the African explorer and Scottish missionary] was exposed, and he called it ‘tick fever.’

“And [we] can't even make a [blood] smear with *Borrelia burgdorferi* and see the organism. **It's there. But you don't see it. You cannot find this spirochete. Why not? After all, I have a sick person here. He is trembling all over. His synovial fluid is full of spirochetes. But when it comes to blood, it's not there.** So there is something associated with this organism that makes it different.” “

”Andy Wilson: ‘Why is *Borrelia burgdorferi* so hard to find in the body and culture outside the body?’”

“Dr. Burgdorfer: ‘*Borrelia burgdorferi* in the tissues of a patient is extremely difficult to demonstrate, because, first of all, you don't like somebody to take samples out of your brain [to look] for spirochetes. The same with other tissues. Every system in your body can be infected with spirochete. But to prove that is extremely difficult. It demands surgical work, which is very expensive

Andy Wilson: Are you a believer in the idea of persistent Lyme infections?

Dr. Burgdorfer: I am a believer in persistent infections because people suffering with Lyme disease, ten or fifteen or twenty years later, get sick [again]. Because it appears that this organism has the ability to be sequestered in tissues and [it] is possible that it could reappear, bringing back the clinical manifestations it caused in the first place. These are controversial issues for microbiologists, as well as the physicians who are asked to treat patients.’”

<https://web.archive.org/web/20120814003829/http://www.underourskin.com/news/lyme-discoverer-willy-burgdorfer-breaks-silence-heated-controversy?page=1>

So, spirochetes are there, you can't kill them, you can't always see them, and they tend to hide out in the brain and lymph nodes. See more on this in the Primers Shell Game chapter.

The American Lyme Disease Foundation, or ALDF.com enterprise of intended Vector Borne Disease (VBDs) vaccine and test kit DNA profiteers (henceforth, "the Cabal") changed the disease's name to "Lyme disease" from "Lyme borreliosis." And yes, the participants in the scam literally referred to themselves as an "enterprise" (Arthur Weinstein, 1998). They conspired to make Lyme relapsing fever even more undetectable. Theirs was a 50-year roll out plan for DNA patented vaccines and test kits due to the emerging tropic infections from global pollution.

Their first commercialized attempt at a recombinant DNA product scam, with the toxic, fungal-ish (managed by Toll Like Receptors 2 and 1; TLR2/1) lipoprotein Outer Surface Protein A (OspA) was to vaccinate ~5000 people and send them out in the world to see if they got Lyme disease. They then would test the people who became ill with a test that only detects 15% of the cases (the "Dearborn" "case definition").

Their plan: make Lyme only 15% detectable so that the Cabal would be guaranteed to have an at least 85% "effective" vaccine. If they **maliciously discredited** the people who became ill as a result of the "vaccine" itself (septic shock) or vaccine failure (Lyme), then the vaccine would be "safe," too. We call both the crime of falsifying the testing and the resultant – and current – bogus testing criteria, "Dearborn." This slander and libel are "Deprivation of Rights via Color of Law" criminal charges because the Cabal includes CDC officers Alan Barbour and Barbara Johnson. You'll read more about that event soon, here.

What was eventually discoverable with this scam was that the vaccine choice, OspA (Pam3Cys or a triacylated lipoprotein), was a fungal antigen, a TLR2/1-agonist, and as such caused immunosuppression in humans. It never could have been a vaccine. Shed fungal antigens like OspA were the very things responsible for the New Great Imitator outcomes.

In dogs, Gary Wormser saw the same immunosuppression result with an OspA vaccine:

[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](#).

"OspA interferes with the response of lymphocytes to proliferative stimuli including a blocking of cell cycle phase progression."

<http://www.ncbi.nlm.nih.gov/pubmed/10865170>

The short version - and even the technical version -, is that OspA or a triacyl lipopeptide or Pam3Cys

gums up the immunity-works. This 2000 report by Gary Wormser proves he knew Dearborn and OspA were false. Or “fraud.”

“Changed!?!?” Yes, They Changed the Diagnostic Standard for Lyme disease.

[Who said “Changed?!?” Senator Blumenthal’s 3 staff lawyers when I met with them in person in July 2003 and showed them that the case definition changed at Dearborn, which no longer defined Lyme as a relapsing fever organism, and which added the ELISA as a screen-out test for Chronic Neurologic Lyme. That was when they referred me to Kevin J. O’Connor, the U.S. Attorney in Connecticut at the time, because this was a federal case that crossed state lines.]

The following article by Allen Steere is the foundation of the CDC’s original, fairly accurate and correct, 1990, “Lyme disease” “case definition” blood test (serology). It was later thrown out and replaced at a farce of a serology consensus conference put on by the CDC in 1994 in Dearborn, MI.

[J Clin Invest.](#) 1986 Oct;78(4):934-9.

Antigens of Borrelia burgdorferi recognized during Lyme disease. Appearance of a new immunoglobulin M response and expansion of the immunoglobulin G response late in the illness.
[Craft JE, Fischer DK, Shimamoto GT, Steere AC.](#)

“... Using immunoblots, we identified proteins of Borrelia burgdorferi bound by IgM and IgG antibodies during Lyme disease. In 12 patients with early disease alone, both the IgM and IgG responses were restricted primarily to a 41-kD antigen. This limited response disappeared within several months. In contrast, among six patients with prolonged illness, the IgM response to the 41-kD protein sometimes persisted for months to years, and late in the illness during arthritis, a new IgM response sometimes developed to a 34-kD component of the organism. The IgG response in these patients appeared in a characteristic sequential pattern over months to years to as many as 11 spirochetal antigens. **The appearance of a new IgM response and the expansion of the IgG response late in the illness, and the lack of such responses in patients with early disease alone, suggest that B. burgdorferi remains alive throughout the illness.**”

<http://www.ncbi.nlm.nih.gov/pubmed/3531237>

1990, CDC published this case definition based on the above:

<ftp://ftp.cdc.gov/pub/Publications/mmwr/rr/rr3913.pdf>

“Laboratory criteria for diagnosis

”• Isolation of Borrelia burgdorferi from clinical specimen, or

“• Demonstration of diagnostic levels of IgM and IgG antibodies to the spirochete in serum or CSF, or

”• Significant change in IgM and IgG antibody response to B. burgdorferi in paired acute – and convalescent-phase serum samples.”

That means Lyme disease should be perceived as a relapsing fever organism, undergoing antigenic variation. Victims are able to produce new, IgM bands if the organism is still alive and not killed by

antibiotics. This is a well-known fact in immunology. New IgM bands mean the infection is ongoing.

Steere also wrote in the 1986 report that became the basis of this 1990 case definition that all you need is band 41 to diagnose Lyme; just rule out syphilis. That is important to remember: You only need band 41, or the anti-flagellar antibody and the triad of symptoms to diagnose Lyme with common sense rule-outs. The U.S. patent #5,618,533 of Yale's is for a specific recombinant fragment of *Borrelia burgdorferi* flagellin. It is an improvement on the band 41-only antibody test, and is an actual FDA-validation according to the FDA's criteria for the validation of an analytical method (as shown in the Primers Shell Game criminal charge sheet).

Before a diagnosis of Lyme, and of course in all illnesses, it is recommended to rule out blood cancers. The symptoms of Chronic Lymphocytic Leukemia are identical to chronic Lyme or Multiple Sclerosis (MS), not to mention the fact that Lyme and LYMERix both are known to cause cancer, MS, Lupus, and possibly Rheumatoid Arthritis (RA) via the reactivation of latent herpes viruses. Mycoplasma are also known to be associated with the production of cancer and RA. Chronic, late, neurologic Lyme victims are tolerized to these fungal type-, TLR2/1-agonist bearing diseases. The truth about the "New Great Imitator" is that it is these other, secondary, opportunistic herpes viruses and other bacterial/fungal infections are responsible for that variety show of outcomes. It's similar to AIDS. It is mechanistically a form of Post-Sepsis Syndrome ("overwhelming the immune system").

This is 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 **1994**, current, diagnostic criteria are very different from the 1990 criteria and basically refer to only the late, HLA-linked, arthritis, hypersensitivity response. It was developed via research fraud committed by Allen Steere in Europe in 1992. OspA and B (bands 31 and 34) are notably absent. Instead of only, now, having "the appearance of new IgM bands," which mean the infection or spirochetes was ongoing or the spirochetes were still alive, we are now required to have the late, autoimmune Lyme arthritis presentation in order to have a "case" of Lyme.

As an aside, we can assume that the reason the Cabal did not want anyone treated for Lyme is because late in the disease, it's really about fungal antigen tolerance and cross tolerance, reactivated herpes viruses, or is NIH's incurable Post-Sepsis Syndrome. This outcome is paralleled in many other conditions such as the failed Tuberculosis vaccines, Malaria and Epstein-Barr resulting in Burkitt's lymphoma, etc. You'll read more about that in later chapters.

Most recently (March 2015) the IDSA had this to say, confirming our supposition:

PRACTICE MANAGEMENT

Infectious Diseases Society of America 2014 Practice Guidelines To Diagnose, Manage Skin, Soft Tissue Infections

The Hospitalist. 2015 March;2015(3)

Author(s): Norihiro Yogo, MD, Carla C. Saveli, MD

"Likewise, the use of broad spectrum gram-negative coverage is not recommended in most common, uncomplicated SSTIs and should be reserved for special populations, such as those with immune compromise."

<http://www.the-hospitalist.org/article/infectious-diseases-society-of-america-2014-practice-guidelines-to-diagnose-manage-skin-soft-tissue-infections/>

Treatment of "Lyme" would allegedly compromise the treatment of severe sepsis infections by creating an environment where those secondary infections acquire antibiotic resistance genes from Lyme victims being treated with the tougher antibiotics. The truth, however, is that most infectious disease pathogens pick up resistance genes in **swine lagoons**. Go ahead and look that up in the National Library of Medicine. That should be well known by normal people. "Normal people" excludes this Cabal and the Infectious Diseases Society of America (IDSA).

How Lyme or Borreliosis causes disease we learned from the OspA vaccine or LYMERix fiasco. The fungal OspA vaccines caused the same "multi-system," "protean," post-sepsis syndrome, chronic active infections/disease, as per Ben Luft, Dave Persing, other scientists, and the vaccine victims themselves as reported to the FDA through the VAERS. (You'll see those links and quotes, here in these chapters.) This is what we Lyme activists witnessed in the first year LYMERix was on the market, early 1999. We said, "HOW are these people saying they 'have Lyme again?' The vaccine wasn't whole spirochetes!" Later, we learned that the fact that the OspA vaccine was giving victims the same "multi-system" disease, was already known to both the Cabal and the U.S. Food and Drug Administrations' (FDA) committee members.

Follow: First, Lyme was a plain old regular Relapsing Fever organism and the "New Great Imitator!" because it caused ALS, Lupus, MS, Cancer, RA, stroke, etc.

Later, at the same time the crooks had a vaccine candidate in early phase trials, it became nothing and a non-disease (psychiatric and hysteria and other libel and slander, Barbour and Fish, 1993, etc.). We were then about to get "a vaccine for a disease that causes no illness."

This is still the current position of Yale, CDC, IDSA, and the ALDF/EUCALB (EUCALB is the European counterpart of the criminal RICO organization, the ALDF.com): **"The Dearborn event was not real and not a crime scene, Lyme patients are not sick, and OspA was a vaccine."** Every time the Cabal makes a public claim about "Lyme disease" based on the falsified Dearborn definition, that

resets the clock on the Statute of Limitations. Amusingly, IDSA and the Cabal are happy to say what diseases *are-not*, but they never say what diseases *are*. MD-America does not even notice that the CDC and the Cabal appear to be insane, even after the FDA ordered LYMERix off the market in February 2002 via ultimatum,... after Senator Richard Blumenthal (a former USDOJ prosecutor) sued them for Anti-Trust, ... after Edward McSweegan became America's infamous NIH employee as America's one and only "Man With No Work" (Google that), and even after Senators Markey, Blumenthal, et al, ordered the FDA to assure Lyme testing was valid according to the FDA's own criteria. It's really whacked that we have to be writing this for you, in 2017 - a hundred years since we knew what Relapsing Fever was about, un-killable, goes to the lymph nodes and brain... blah, blah, white people get dementia which was why the CDC performed 2 war crime bioweapons experiments on American Blacks and Native Americans... It's completely crazy that 99.9999% of Americans and all the "MDs" in America do not know what spirochetes are and do.

The Not-Thinking and Not-Wondering may be an even more infamous characteristic of Americans than our bioweapons-, and other war crimes.

Continuing the Chronology of Events in Redefining Lyme as a Non-Disease to Pass Off a Bogus Vaccine:

1986, Edward McSweegan, in a fake whistleblower letter to Senator Barry Goldwater, discredited the U.S. Navy to divert their vector borne diseases funding to his ALDF.com cabal. See the Navy's furious response in the link below. McSweegan thinks there can be a vaccine for Relapsing Fever, confirming the parapsychical theory that arrogance is the seed corn or germinal element in true, genuine stupidity and/or the development of a criminal mind:

http://www.actionlyme.org/GOLDWATER_LETTER.htm

1988, Raymond Dattwyler, JJ Halperin, et al, & immune-suppressing, seronegative Lyme; supernatant (lipid layer) of borrelia mash causes NK cell anergy or a blunted immune response. Later, Dattwyler tells the FDA Vaccine committee that the seronegative patients are the sickest. Now we know why; Lyme and LYMERix are the Great Detonators of the latent herpes viruses and expanded or cross tolerance to other antigens than TLR2/1-agonist bearing kinds; in short, they're double-fatigued and neurologically damaged:

[N Engl J Med](#). 1988 Dec 1;319(22):1441-6.

Seronegative Lyme disease. Dissociation of specific T- and B-lymphocyte responses to *Borrelia burgdorferi*.

[Dattwyler RJ](#), [Volkman DJ](#), [Luft BJ](#), [Halperin JJ](#), [Thomas J](#), [Golightly MG](#).

"We conclude that the presence of chronic Lyme disease cannot be excluded by the absence of antibodies against *B. burgdorferi* and that a specific T-cell blastogenic response to *B. burgdorferi* is evidence of infection in seronegative patients with clinical indications of chronic Lyme disease."

"The disorder in these seronegative patients reflected a dissociation between T-cell and B-cell immune

responses, in which the cell-mediated arm of the immune response was intact yet the humoral portion of the immune response to *B. burgdorferi* appeared to be blunted. This diminished antibody response is in contrast to the T-cell anergy commonly observed in several chronic infections (e.g., infection with *Mycobacterium leprae* or *M. marinum*, filiarasis, and some chronic fungal infections (29-33))."

<http://www.ncbi.nlm.nih.gov/pubmed/3054554> http://www.actionlyme.org/dattwyler1988_1.pdf

And:

[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](#).

"Effect of *B burgdorferi* Culture on Normal PBL

"...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 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> http://www.actionlyme.org/golightly_Datt1988.pdf

Perhaps the difference between the "diminished antibody response is in contrast to the T-cell anergy commonly observed in several chronic infections (e.g., infection with *Mycobacterium leprae* or *M. marinum*, filiarasis, and some chronic fungal infections (29-33))" and the B cell incompetence in Borreliosis speaks to the fact that *Borrelia* like to go directly to the lymph nodes as well as the brain. The lymph nodes are where B cells mature or become specialized. This will be discussed later (Baumgarth, et al).

1990, CDC: "Diagnose Lyme as if it was Relapsing Fever" as previously mentioned.

<ftp://ftp.cdc.gov/pub/Publications/mmwr/rr/rr3913.pdf>

1990, Allen Steere reports that "chronic, neurologic Lyme won't test positive," uses Dattwyler and Volkman's Seronegative Lyme T Cell Assay

N Engl J Med. 1990 Nov 22;323(21):1438-44.

Chronic neurologic manifestations of Lyme disease.

[Logigian EL1](#), [Kaplan RF](#), [Steere AC](#).

"METHODS

"Neurological Evaluation...

"If the patient was seronegative according to these methods, the serum was further tested by immunoblotting (25) and peripheral blood mononuclear cells were tested for reactivity with borrelial antigens by proliferative assay. (26)" <https://www.ncbi.nlm.nih.gov/pubmed/2172819>

<http://www.nejm.org/doi/pdf/10.1056/NEJM199011223232102>

And what was reference number 26?

[N Engl J Med](#). 1988 Dec 1;319(22):1441-6.

Seronegative Lyme disease. Dissociation of specific T- and B-lymphocyte responses to *Borrelia burgdorferi*.

[Dattwyler RJ](#), [Volkman DJ](#), [Luft BJ](#), [Halperin JJ](#), [Thomas J](#), [Golightly MG](#).

<http://www.ncbi.nlm.nih.gov/pubmed/3054554>

1990, ALDF.com founded-- a self-proclaimed “entrepreneurial” quartet, includes Edward McSweegan, Durland Fish, Gary Wormser and John J. Connolly. This evidence is in the office of U. S. Department of Justice (USDOJ or DOJ) in New Haven, CT, USA on Church Street. It is a quote by Arthur Weinstein in a publication the DOJ has been given (we no longer have the link, just this:

http://www.actionlyme.org/CONNOLLY_FISH_WEINSTEIN.htm).

1992, CDC officer Allen Steere falsifies testing in Europe:

The PubMed links to those 2 reports – no full text available, that is why I got them out of the Yale Medical Library in 2002 and scanned them in are:

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

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

[Dressler F](#)1, [Ackermann R](#), [Steere AC](#).

<http://www.ncbi.nlm.nih.gov/pubmed/8106763> <http://www.actionlyme.org/dressler1994.pdf>

[J Infect Dis](#). 1993 Feb;167(2):392-400.

Western blotting in the serodiagnosis of Lyme disease.

[Dressler F](#)1, [Whalen JA](#), [Reinhardt BN](#), [Steere AC](#).

<http://www.ncbi.nlm.nih.gov/pubmed/8380611> , which is in full text in the Dearborn booklet, downloadable here: http://www.actionlyme.org/DEARBORN_PDF.pdf

Of those two reports of Steere’s lab shenanigans in Europe, only the second one was made a part of CDC’s Dearborn booklet. The first one—the one left out of the Dearborn booklet—is where you can see how he falsified the testing for his later monopoly on post-LYMERix-approval for North America, with Corixa, Yale’s L2 Diagnostics and Imugen. These 3 entities were officially listed on the Securities and Exchange Commission (SEC) as “partners” in sharing licensing of the RICO Monopoly patent with the strain of *Borrelia* that had dropped an OspA-B plasmid under US Patent 6,045,804.

Steere, in Europe, used “high-passage” borreliae strains that drop plasmids, and recombinant OspA and B without the lipids attached, helping leave OspA and B out of the diagnostic standard (see the Dearborn criteria above, there is no OspA or B, bands 31 and 34). The lipid parts of the

lipoprotein are known to be immune-stimulatory, or to produce antibodies, so they obviously are necessary to come up with a legitimate criteria.

Steere knew before the Dearborn event that people without the arthritis HLAs were mostly seronegative against the fungal Osps:

[Infect Immun.](#) 1993 Jul;61(7):2774-9.

Association of treatment-resistant chronic Lyme arthritis with HLA-DR4 and antibody reactivity to OspA and OspB of Borrelia burgdorferi.

[Kalish RA1](#), [Leong JM](#), [Steere AC](#).

OspB fusion proteins in single serum samples from 128 patients with various manifestations of Lyme disease and from 36 normal control subjects (Table 1). None of the 43 patients with early manifestations of Lyme disease (erythema migrans or meningitis) and none of the 36 normal control subjects showed reactivity with the Osp proteins. Compared with these individuals, 57 of the 80 patients (71%) with Lyme arthritis showed reactivity to OspA or OspB ($P < 0.00001$). Of the 57 patients, 49 showed reactivity with both OspA and OspB, 7 showed reactivity with only OspA, and 1 showed reactivity with only OspB. Compared with the 80 patients with arthritis, only 1 of the 5 patients who had chronic neuroborreliosis and who never had arthritis showed weak reactivity with the Osp proteins ($P = 0.03$).

<https://www.ncbi.nlm.nih.gov/pubmed/7685738>

The Dearborn case definition says you need 5 of 10 IgG bands (arthritis only), after the non-HLA-linked-, ***non-arthritis cases are screened out in the first step, the ELISA.*** That is, Chronic Neurologic Lyme cases, which are immunosuppression outcomes—like AIDS, where the opportunistics do most of the damage and are what keep you ill -, ***were left out at the first step, the ELISA, because this outcome was caused by injections of the fungal toxin OspA (the Lyme vaccines), too.***

The Disease (fungal-toxic immunosuppression or post-sepsis syndrome)...***is the Cryme*** (saying OspA was a “vaccine” when it caused the same toxic, post-sepsis syndrome). The Dearborn case definition was not a consensus. The average accuracy was 15%, as is shown in this booklet covering the Dearborn conference: http://media.wix.com/ugd/47b066_90b3f15f334346c1aede0bf8e98522ba.pdf

The other, twin report by Steere in Europe left out of the Dearborn booklet:

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

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

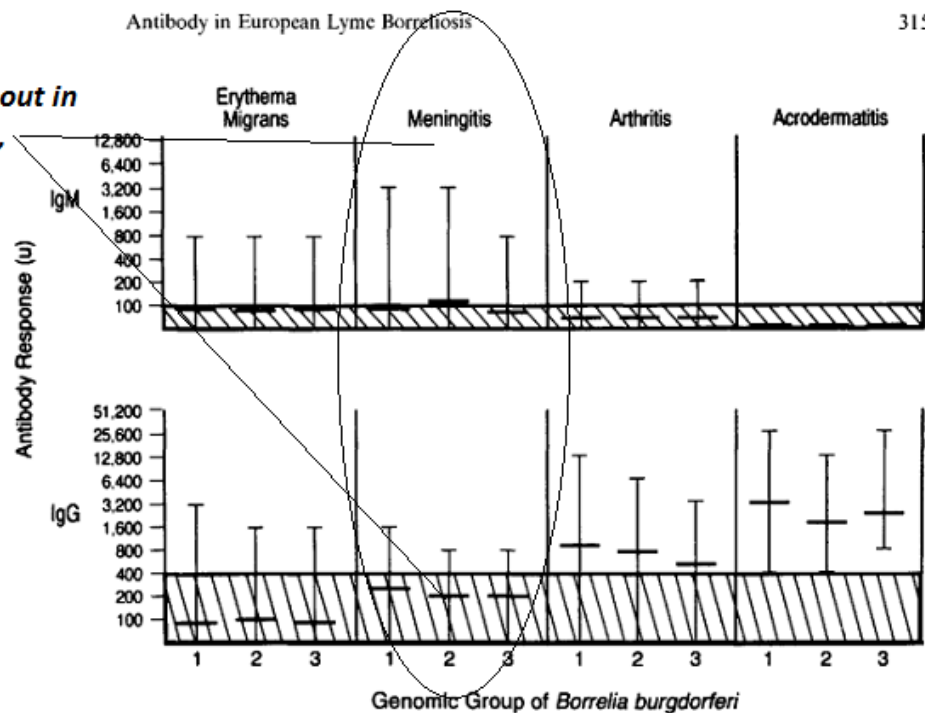
Dressler F1, Ackermann R, Steere AC.

JID 1994;169 (February)

315

NeuroLyme is screened out in the first step, the ELISA, via research fraud.

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>

What's scientific fraud about *this "Antibody Responses in Europe"* report?

1) Allen Steere in the **IgM** ELISA arbitrarily raised the "noise" cutoff of 3 standard deviations to 5 std dev (3 is normally done) such that most Neurologic cases would be missed (arthritis cases produce lower IgM, for some reason).

2) Steere in the IgG-falsification step, averaged the concentration of IgGs from the meningitis or neurologic Lyme (lower, like 1:400 dilution), with acrodermatitis (autoimmune, very high antibody concentration of about 1:1600 to 1:3200) and arthritis (1:800 dilution).

This fraud deliberately excluded the sickest patients in the first step of the Dearborn "2-tiered" testing criteria for Lyme. Note that it is strange that Steere felt he had to develop this new Dearborn panel in Europe, presumably where American Justice would have a hard time verifying the data.

Note this same report reveals an intended a later monopoly on testing for Lyme once the bogus OspA vaccines were on the market (you never test for a disease with the same antigens that are the vaccine

antigens since you would not know if the antibodies are from the actual infection or from the vaccine antigens):

Antigen preparations. Supernatants from sonicated lysates of whole spirochetes were prepared as described [20]. The group 1 strain of *B. burgdorferi*, G39/40, used in this study and in the previous study of US patients, was isolated from an *Ixodes dammini* tick in Guilford, Connecticut [21]. The group 2 strain, FRG, was isolated from *Ixodes ricinus* near Cologne [22]. The group 3 strain, IP3, was isolated from *Ixodes persulcatus* near Leningrad [23]. All 3 strains used in this study were high-passage isolates, which were classified by Richard Marconi (Rocky Mountain Laboratory, Hamilton, MT) using 16S ribosomal RNA sequence determination as described [11, 24]. The recombinant preparations of OspA and OspB used in this study were purified maltose-binding protein-Osp fusion proteins derived from group 1 strain B31 [25]. These fusion proteins contained the full-length OspA or OspB sequence without the lipid moiety or the signal sequence.

The above graphic from the same report shows:

3) Steere used high passage strains which lose plasmids and therefore potential antigens (meaning if you *have* those antibodies, they wont be detected).

4) Steere used strain B31 which essentially does not have the European kinds of OspAs, and he assured no one would have antibodies against OspA and B in this Dearborn antibody panel for Western Blotting by leaving off the Pam3 or the tri-acyl or the lipid groups of these triacyl lipoproteins which cause antibodies. The protein ends by themselves are **not** immunogenic (meaning they do not cause antibodies to be produced).

The following is the text (not in the abstract) of what is in the report on exactly how Steere defrauded the U.S. Government and people:

[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](#).

“The group 1 strain of *B. burgdorferi*, G39/40, used in this study and in the previous study of US patients was isolated from an *Ixodes dammini* tick in Guilford, Connecticut [21]. The group 2 strain, FRG [Federal Republic of Germany], was isolated from *Ixodes ricinus* near Cologne [22]. The group 3 strain, IP3, was isolated from *Ixodes persulcatus* near Leningrad [23]. All three strains used in this study were high passage isolates, which were classified by Richard Marconi (Rocky Mountain

Laboratory, Hamilton, MT) using 16S ribosomal RNA sequence determination as described [11, 24]. The recombinant preparations of OspA and OspB used in this study were purified maltose-binding protein-Osp fusion proteins derived from group 1 strain B31 [25]. **The fusion proteins contained the full-length OspA or OspB sequence without the lipid moiety or the signal sequence...**

<http://www.ncbi.nlm.nih.gov/pubmed/8106763>

He left the OspA (band 31) and OspB (band 34) out, deliberately.

The following is what it says in the Persing/Schoen/Steere or Imugen RICO Monopoly patent, that shows the intended monopoly - which required that OspA and B be missing from the diagnostic panel and from the spirochetes used to test the human population after the population was vaccinated with OspA:

Method for detecting B. burgdorferi infection

"...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."

and

"The present invention provides a method useful to detect a B. burgdorferi infection in a subject. The method provided by the invention is particularly useful to discriminate B. burgdorferi infection from OspA vaccination, although it is sufficiently sensitive and specific to use in any general Lyme disease screening or diagnostic application. Thus, the method of the invention is particularly appropriate for large scale screening or diagnostic applications where only part of the subject population has been vaccinated or where the vaccination status of the population is unknown. "

<http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnethtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804>

The monopoly on post-LYMERix-FDA-approval testing for all vector borne diseases in America and Canada was their stated intention (entrepreneurial or enterprise = RICO). Once LYMERix was on the market, a strain of borreliae that did not have the vaccine antigens in it would have to be used for testing for "Lyme." Vaccine efficacy is never assessed with the very same antigen as the vaccine antigen. Otherwise, it would not be known if the victim has the actual infection in question, or that the antibody that shows up came from the vaccine. This Lyme/Vector-Borne Diseases monopoly depended on LYMERix being on the market. That way, Corixa, L2 Diagnostics and Imugen would be the only labs in the country licensed to use this RICO strain. They would have access to all the human blood to pharm all sorts of DNA data to patent from humans as well as any new and emerging infectious diseases. That was the monopoly: LYMERix and the bogus testing criteria together with Persing's RICO patent had the intention of gathering all the blood, and all the potential infections in that blood, and what mean meant even more vaccine patents in the future would go to this Cabal. The three, Corixa, Imugen and Yale's L2 Diagnostics, listed themselves as "partners" in a Securities and Exchange Commission announcement and advertised that this test would be available for the vaccinated population.

The Cabal falsified the “case definition” to leave out neurologic Lyme cases, and they left OspA and B out for a later monopoly on testing and future patents. And there, you just read that that intention is clearly stated in a patent and method developed by Schoen and Persing in 1995 (US patent 6,045,804), next:

[J Clin Microbiol](#). 1997 Jan;35(1):233-8.

Borrelia burgdorferi enzyme-linked immunosorbent assay for discrimination of OspA vaccination from spirochete infection.

Zhang YQ1, Mathiesen D, Kolbert CP, Anderson J, Schoen RT, Fikrig E, Persing DH.
<http://www.ncbi.nlm.nih.gov/pubmed/8968914>

Whose name do you see there, with Persing’s? Right, Yale’s Robert Schoen’s. Therefore he knew that there was a problem with LYMERix and that an opportunity was presented by patenting this bug with the no-OspA/B plasmid in it: a monopoly on all future vaccines and test kits for vector borne diseases (VBDs).

1992, CDC staff, Barbara Johnson and Joe Piesman, own patents with SmithKline that show 2 kinds of Lyme, HLA-linked and non-HLA-linked antigens:

COMPOSITIONS USEFUL IN DIAGNOSIS AND PROPHYLAXIS OF LYME DISEASE

"Summary of the Invention

"In one aspect, the invention provides isolated *B. burgdorferi* antigens which are regulated and differentiated by growth of the *B. burgdorferi* in a tick vector. Novel antigens of the invention are listed below in Table I.

"Certain of these antigens are characterized as being *B. burgdorferi* B31 strain specific and major histocompatibility complex (MHC) nonrestricted. Certain other of these antigens are characterized as being MHC-restricted."

<http://worldwide.espacenet.com/publicationDetails/biblio?>

[DB=worldwide.espacenet.com&II=0&ND=3&adjacent=true&locale=en_EP&FT=D&date=19931209&CC=WO&NR=9324145A1&KC=A1](http://worldwide.espacenet.com&II=0&ND=3&adjacent=true&locale=en_EP&FT=D&date=19931209&CC=WO&NR=9324145A1&KC=A1)

Why is the CDC talking about “MHC-restricted” vs. “MHC non-restricted?”

What we know that to mean is that classic “autoimmune” diseases tend to be MHC-(or HLA-) restricted, or the antigens, due to intermolecular forces, either bind in the HLA groove too strongly, the HLA-antigen complex is released as yet another free, new antigen, or the antigen does NOT bind tightly enough and the antigen falls out of the HLA groove to re-stimulate.

This “autoimmune” -only is the new definition Steere claimed in these 1992 reports and at the CDC’s 1994 Dearborn conference. He falsely claimed Lyme disease is only the HLA- or MHC-arthritis-restricted and threw out the other, meningitis cases.

Yet, here, in their 1992 patents with SmithKline, the CDC mentions the other outcome—the no- or fewer- antibody result. Therefore, they recognize the two kinds of Lyme: the 15% of the population with the Rheumatoid Arthritis genetic background or HLA-restricted or arthritis cases,... and the 85% with seronegative, neurologic, long term, New Great Imitator Lyme.

The 85% of the chronic disease sufferers most likely suffer from the opportunistics (NIH's "Post-Sepsis Syndrome") from the immunosuppression that is caused by shed Borrelial TLR2/1-agonist antigens. Regardless, the falsified tests result in more early Lyme cases going undiagnosed and therefore progressing to permanent disability and early death.

1993, Barbour and Fish slam Neurologic Lyme victims in:

[Science](#). 1993 Jun 11;260(5114):1610-6.

The biological and social phenomenon of Lyme disease.

[Barbour AG1](#), [Fish D.](#)

<https://www.ncbi.nlm.nih.gov/pubmed/8503006>

Barbour and Fish admit in this report that Phase I and Phase II trials of OspA vaccines are underway. Therefore, as is shown in the Persing RICO Monopoly patent (US 6,045,804), they already knew the OspA vaccines were causing a disease indistinguishable from vaccine failure, or CHRONIC LYME:

Here would be a good place to show what data was received by the USDOJ in New Haven, CT, on this fraud and RICO scam, because the difference between neurologic Lyme and arthritis Lyme is so clear:

Compare the blots from the two kinds of Lyme in this (above) July 2003 RICO complaint. On the left with the faint antibody bands is neurological Lyme (the sickest, according to Ray Dattwyler), and on the right are the HLA-linked outcomes of arthritis and acrodermatitis:

http://www.actionlyme.org/USDOJ_COMPLAINT_RICO.htm

Hence, the Cabal left out the neurological outcomes in their Dearborn scam. The whole point of the redefinition of Lyme at Dearborn was to narrow it to just the HLA-linked, arthritis, supposedly autoimmune, hypersensitivity cases. This is how and why they get away with perjury. When the IDSA/ Yale Lyme Cabal say "Lyme Disease," they mean exclusively "HLA-linked arthritis AND NO OTHER SYMPTOMS." No lawyer was or is aware of this semantics scam.

Jump to 2005; Here Klempner and Wormser re-revealed that "Lyme disease" is *just one thing*: a bad knee and no other illness signs. However, as shown above, there are two distinct outcomes of Lyme borreliosis. The controversial one (neurologic-, chronic fatigue- Lyme) really does not have a name right now. Therefore, "Lyme disease" is defined as ONLY a bad knee. It's a legal definition. It's also criminal one, based on fraud and no consensus, but here is what it is again (2005):

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 chronic Lyme disease.

Klempner MS1, Wormser GH, Wade K, Trevino RP, Tang J, Kaslow RA, Schmid C.

“... There appear to be at least 2 distinct syndromes after antibiotic treatment. [They have no data on un-treated people, obviously, since they could not ethically conduct such a study-KMD] 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 generally negative.... [See the *DNA/RNA Shell Game* report, this is not true <http://www.actionlyme.org/PRIMERSHELLGAME.htm> ; it's a shell game; they use DNA that they know won't be there in that sequence due to antigenic variation to claim “No Lyme here.”-- KMD]

“...Patients generally feel well aside from their arthritis symptoms.”

<https://www.ncbi.nlm.nih.gov/pubmed/16107953> <http://jid.oxfordjournals.org/content/192/6/1010.full>

Let's restate what Wormser and Klempner are trying to say in that 2005 report:

“The people with the falsified Dearborn case definition of ‘only an HLA-linked arthritis in a knee’ have only an HLA-linked arthritis in a knee and no other symptoms.”

If you falsify the case definition and say “ONLY the HLA-linked hypersensitivity response of bad knee can be a ‘case’ of ‘Lyme disease,’” you can then, 11 years later say, “Oh, how amazing for us to find only the HLA-linked case definition of arthritis-only is an HLA-linked arthritis-only, and is only a bad knee.”

These people are crazy, yes, if that is what you were thinking.

Also, the CDC recently reacted to the Senators' (Blumenthal, Markey, et al) letter to the Office of Policy and Management, where the Senators are forcing the FDA to do their jobs and assure that the testing for Lyme is validated according to their own FDA rules. (See the Primers Shell Game for more on that.) The CDC is trying to say that the Dearborn method was FDA validated, when it was not:

”Washington – Senator Edward J. Markey (D-Mass.) was joined by Senators Richard Blumenthal (D-Conn.), Elizabeth Warren (D-Mass.), Sherrod Brown (D-Ohio), and Dick Durbin (D-Ill.) in calling on the Obama administration to release draft guidance to ensure appropriate oversight of laboratory developed diagnostic tests (LDTs), which are used to help diagnose specific forms of cancer and other diseases and are not approved by the Food and Drug Administration (FDA). Laboratories initially manufactured LDTs that could be used for low-risk diagnostics or for rare diseases, but with new

technology, they have become a staple of clinical decision-making and are being used to diagnose high-risk but relatively common diseases such as ovarian cancer. Recently, the Centers for Disease Control and Prevention (CDC) reviewed a frequently utilized LDT to detect Lyme disease and found “serious concerns” about false-positive results and misdiagnosis. The CDC recommended that the diagnosis of Lyme disease should instead be left to tests approved by the FDA. ...”

<http://politicalnews.me/?id=29174&keys=DIAGNOSES-CONDITIONS-MEDICAL-OBAMACARE>

Here are the FDA’s rules for the validation of an analytical method:

For the Purpose of Notification to Congress Only

requirements under the FD&C Act. Namely, CLIA requirements address the laboratory’s testing process (i.e., the ability to perform laboratory testing in an accurate and reliable manner). Under CLIA, accreditors do not evaluate test validation prior to marketing nor do they assess the clinical validity of a LDT (i.e., the accuracy with which the test identifies, measures, or predicts the presence or absence of a clinical condition or predisposition in a patient). Under the FD&C Act, the FDA assures both the analytical validity (e.g., analytical specificity and sensitivity, accuracy and precision) and clinical validity of diagnostic tests through its premarket clearance or approval process. In addition to premarket review, FDA requirements provide other controls to ensure appropriate design, manufacture, and safety and effectiveness of the device. As a result, while CLIA oversight is important, it alone does not ensure that LDTs are properly designed, consistently manufactured, and are safe and effective for patients.

...which were met by Yale’s 1991 Flagellin Method Patent US # 5,618,533 and this report:

[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 R I](#), [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.” <http://www.ncbi.nlm.nih.gov/pubmed/1894359>

As you can see, the FDA has not changed their rules on how to validate a method:

“Under the FD&C Act, the FDA assures both the analytical validity (e.g., analytical specificity and sensitivity, accuracy and precision) and clinical validity through its premarket clearance and approval process.”

<http://www.fda.gov/downloads/MedicalDevices/ProductsandMedicalProcedures/InVitroDiagnostics/ucm407409.pdf>

Also, *Borrelia burgdorferi* is closest genetically to *B. anserina*, an African bird borreliosis, so it is not surprising that Lyme is found all across the United States, being carried by birds:

Many California bird species host the Lyme disease bacterium, study finds:

<http://www.latimes.com/science/sciencenow/la-sci-sn-california-birds-lyme-disease-20150225-story.html>

See more on the the phylogeny or the genetics that shows Lyme is closest to *B. anserina* (from Africa) in the DNA Shell Game document. Therefore there cannot be any “disease calculator” for Lyme as there fraudulently had been in the past, in an attempt to limit diagnoses. Just as all kinds of *Borreliae* are everywhere, so is this specific one, *burgdorferi*.

Returning to the Chronology of the Crime

1994, June; FDA LYMERix Meeting (note that June precedes October--when the Dearborn stunt took place-- so the FDA never approved of the Dearborn method, not to mention it was research fraud, and not a consensus):http://www.actionlyme.org/1994_FDA_MEETING_LYMERIX.htm

Transcript of June 1994 FDA Meeting Minutes on Lyme and potential vaccines:

Dr. O'BRIEN: "I was concerned about your last slide where you said there was a poor correlation between serologic response and clinical disease. And as I heard you to say, some people who mount better immune responses get worse disease. Did I hear you say that?"

DR. DATTWYLER: "No, no, I said the reverse. The better responses tended to have better response. And I should clarify where this is from. This is from antibiotic trials. These are treatment trials of erythema migrans, in which individuals given an antibiotic regimen which was not optimal – we did not know that it was not optimal at the time – the ones that failed to mount a vigorous response tended to do worse, clinically. So, there was an inverse correlation between the degree of serologic response and the outcome."

"So, individuals with a poor immune response tend to have worse disease."

We know why, now, that “individuals with a poor antibody response have worse disease.” *Borrelial* fungal antigens cause immunosuppression and a classic post-sepsis-like result with chronic active

EBV, HHV-6, et al. And we know this is not just from antibiotic treatment as Dattwyler said at this FDA meeting—that the diminished responses are due to the organism or its supernatants, like OspA, and that that is typical for fungal infections:

[N Engl J Med](#). 1988 Dec 1;319(22):1441-6.

Seronegative Lyme disease. Dissociation of specific T- and B-lymphocyte responses to *Borrelia burgdorferi*.

[Dattwyler RJ](#), [Volkman DJ](#), [Luft BJ](#), [Halperin JJ](#), [Thomas J](#), [Golightly MG](#).

“We conclude that the presence of chronic Lyme disease cannot be excluded by the absence of antibodies against *B. burgdorferi* and that a specific T-cell blastogenic response to *B. burgdorferi* is evidence of infection in seronegative patients with clinical indications of chronic Lyme disease.”

“The disorder in these seronegative patients reflected a dissociation between T-cell and B-cell immune responses, in which the cell-mediated arm of the immune response was intact yet the humoral portion of the immune response to *B. burgdorferi* appeared to be blunted. This diminished antibody response is in contrast to the T-cell anergy commonly observed in several chronic infections (e.g., infection with *Mycobacterium leprae* or *M. marinum*, filariasis, and some chronic fungal infections (29-33).”

<http://www.ncbi.nlm.nih.gov/pubmed/3054554> http://www.actionlyme.org/dattwyler1988_1.pdf

And (1988):

[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](#).

"Effect of *B burgdorferi* Culture on Normal PBL

"...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 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>

The diminution of antibody response might be instead due to the fungal antigens shed by *Borrelia* and not antibiotics since this phenomenon is seen in parallel in other human fungal-exposure immunology. See those other scientific examples, including from the CDC on the failed Autism vaccines and the failed Tuberculosis vaccines, here: http://www.actionlyme.org/SASH_POLICY_PAPER_MECFS.htm

1994, CDC's invitation to participate in the Dearborn event. Labs were invited; they said the Steere proposal was only, on average, 15% accurate; CDC then blew off these labs' recommendations:

<http://www.actionlyme.org/DEARBORNINVITATION.pdf>

1994, October; CDC's Dearborn Booklet .pdf

http://www.actionlyme.org/DEARBORN_PDF.pdf

1994 - Dearborn, Who Said What (also summarized for the FDA at their Jan 2001 hearing on adverse events to LYMERix): http://www.actionlyme.org/DEARBORN_WHO_SAID_WHAT.htm

1) Gary Wormser at New York Medical College reports that Steere's Dearborn proposal method detected 9/59 of IgG cases or is 15% accurate, missing 85% of the cases:

J Clin Microbiol. 1993 Dec;31(12):3090-5.

Serodiagnosis in early Lyme disease.

Aguero-Rosenfeld ME1, Nowakowski J, McKenna DF, Carbonaro CA, Wormser GP.

Overall, 51 of 59 (86%) convalescent phase serum specimens were reactive by IB [Dearborn criteria Immunoblot-SASH], 35 of which were interpreted as positive; 26 based on IgM criteria, 8 based on both IgM and IgG, and 1 based on IgG criteria..."

% of
positive
in IgG
criteria of
Dearborn
9/59

Convalescent-phase sera were available from 33 patients whose sera were either negative ($n = 22$) or indeterminate ($n = 11$) by IB in the acute phase of the disease. Twenty-one patients seroconverted (64%); of the 22 whose sera were initially negative, 6 (27%) became indeterminate and 9 (41%) became positive by IB; in addition, 6 of 11 (55%) serum specimens that were indeterminate by IB in the acute phase became positive in the convalescent phase. Overall, seroconversion was observed in 24 of 31 patients (77%) by ELISA or IB. Sera from all 21 patients that were positive by ELISA in the acute phase remained positive in the convalescent phase. Convalescent-phase sera were available from 26 patients whose sera were positive by IB during the acute phase; of these serum specimens, 20 (77%) remained positive and the other 6 became indeterminate (all 6 serum specimens tested positive by ELISA). Overall, 51 of 59 (86%) convalescent-phase serum specimens were reactive by IB, 35 of which were interpreted as positive: 26 based on IgM criteria, 8 based on both IgG and IgM criteria, and 1 based on IgG criteria. As for the acute-phase sera, the most frequent immunoreactive antigens during the convalescent phase were 41 and 25 kDa. The 41-kDa band was found in 88 and 78% of IgG- and IgM-reactive blots, respectively, and the 25-kDa band was found in 41 and 63% of the IgG- and IgM-reactive blots, respectively. Reactivity to the 39-kDa band was found in 37 and 33% of IgG- and IgM-reactive blots, respectively.

<https://www.ncbi.nlm.nih.gov/pubmed/8308100>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC266355/pdf/jcm00024-0026.pdf>

That is, according to Gary Wormser, 9 out of 59 cases were positive to Dearborn later in the disease; Gary Wormser assessing Steere's Dearborn panel proposal in this report, says it only detects 15% of the cases in IgG.

Others at Dearborn said...

- 2) Igenex —Steere’s IgG panel detected 8% of the cases
- 3) Imugen —Steere’s IgG panel detected 14% of the cases
- 4) Wisconsin —Steere’s method was 15% accurate
- 5) UCONN —Larry Zemel was referring to Lyme as comparable to only juvenile rheumatoid arthritis when of course it isn’t. Recommended adding band 50 for children’s blots.
- 6) Roche— 28% were positive for 5 of 10 Steere IgG bands.
- 7) Wadsworth— had some different scoring system. Did not report on accuracy of Steere's method
- 8) Ontario Ministry of Health—did not give an assessment of the Steere proposal (page 86)
- 9) Lutheran Hospital— 22% were accurate by Steere’s IgG
- 10) MarDx Labs— recommended adding bands 31 and 34, but were given CDC positive arthritis positive blood to falsely qualify their test strips. Their Western Blot test strips were used in both OspA vaccine trials. MarDx was later sold to an Irish company, Trinity Biotech, Dublin; all the data they had about this crime was taken out of the country.
- 11) CDC Atlanta— talked about mice, not humans. The mouse criteria was 2 out of three from OspC, 16 kD, 17.9 kD, for the mice.

We got this standard anyway, even though none of the invited participants agreed—not by a long shot.

See the Primers Shell Game for an explanation of how VALID testing is performed according to the FDA rules, and how Yale knows all about how to validate a method for Lyme (Bb-specific flagellar antigen) and patented it (US 5,618,533). This is all obvious criminal fraud. Yale owned a valid test for Lyme but did not use it to qualify their other patented product, rOspA, LYMERix.

Who was involved with approving the bogus Dearborn method at Dearborn when all the invited labs said it was only 15% accurate (and FDA criterion for validation)?

None other than the CDC vaccine patent owners and all the scammers you see here: http://www.actionlyme.org/Dearborn_Who_Approved.htm

*Members of the CDC/ASTPHLD Work Group:

Alan G. Barbour, MD, Univ of TX Hlth Sciences Ctr
 Eric Blank, DrPH, ASTPHLD
 Raymond J. Dattwyler, MD, SUNY at Stony Brook
 Sharon Hanson, PhD, Food and Drug Administration

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Russell C. Johnson, PhD, University of Minnesota
 Frank W. Lambert, DrPH, ASTPHLD
 Robert Martin, DrPH, ASTPHLD
 Raymond W. Ryan, Univ of CT Hlth Ctr
 Edward McSweegan, PhD, National Institutes of Health
 Thomas Schwan, PhD, Rocky Mountain Labs, NIH
 Richard Steece, PhD, ASTPHLD
 Allen Steere, MD, Tufts/New England Medical Center
 Ralph J. Temperli, DrPh, ASTPHLD
 Arthur Weinstein, MD, New York Medical College
 The CDC Lyme Disease Group

“Alan Barbour,” “Edward McSweegan,” “Allen Steere,” “Arthur Weinstein,” “The CDC Lyme Disease Group” (Barbara Johnson), etc. (The same people involved in the OspA vaccines scam were involved in falsifying the testing and who were the original members and “advisors” of the ALDF.com.)

A view of the Dearborn event by a participant. It’s an independent paper about it; Igenex’s Nick Harris’ report published in the Lyme Disease Foundation’s journal:

http://www.actionlyme.org/HARRIS_IGENEX_DEARBORN.pdf

Evidence Lyme Cabal knew LYMERix produced the same "multisystem disease" as "Chronic Lyme"

1) Ben Luft said it at the 1998 FDA meeting:

<http://www.fda.gov/ohrms/dockets/ac/98/transcript/3422t1.rtf>

BEN LUFT: "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. ..."

2) Dave Persing said it in his RICO patent:

Method for detecting B. burgdorferi infection

"...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=%2Fnethtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804>

3) Fish and Barbour trashed Lyme disease victims with their "Social Aspects" report in 1993, paving the way to slander and libel their future LYMERix victims. They reveal that the OspA vaccine trials are underway in that report. This shows intent to cause harm:

[Science](#). 1993 Jun 11;260(5114):1610-6.

The biological and social phenomenon of Lyme disease.

[Barbour AG1](#), [Fish D.](#)

<https://www.ncbi.nlm.nih.gov/pubmed/8503006> <http://actionlyme.org/BarbourFishpdf.pdf>

4) Dave Persing (who worked on this with Robert Schoen, as shown above) and his company Corixa wanted to sell vaccine adjuvants, but they had to drop OspA as a candidate adjuvant because, as Persing said in another patent (applied for May, 2001, while LYMERix was still on the market, harming people; he never said anything to the FDA about it):

Prophylactic and therapeutic treatment of infectious and other diseases with mono- and disaccharide-based compounds

"Accordingly, the methods of the invention provide a powerful and selective approach for modulating the innate immune response pathways in animals without giving rise to the toxicities often associated with the native bacterial components that normally stimulate those pathways."

<http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=/nethtml/PTO/srchnum.htm&r=1&f=G&l=50&s1=6,800,613.PN.&OS=PN/6,800,613&RS=PN/6,800,613>

In this complaint to the UN Human Rights Commission and the foreign embassies:

http://www.actionlyme.org/EMBASSIES_CORIXA_TLR_13_JULY_06.htm

it shows that Corixa got an 11 million dollar "biodefense contract" from the NIH and the adjuvants they are allegedly producing are TLR4 agonists, not TLR2/1 agonists like LYMERix, because Persing et al know OspA as an adjuvant is "too toxic in the native form" and "...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," which means they know OspA is too toxic and causes a chronic illness identical to chronic Lyme.

5) In 1998 Yale's Robert Schoen wrote the following article in the ALDF's book, *Lyme Disease, ACP Key Diseases Series*, published in 1998 to coincide with the release of LYMERix onto the market. Once again. Schoen is paving the way, instructing other "doctors" to view LYMERix-injured people and Chronic Lyme victims (which are essentially the same disease, Post-Sepsis Syndrome) through the same victim-blaming lens.

The article is called *Clinical Vignettes, Case 13, A Vaccine Recipient who Develops Arthralgia and Fatigue, page 238-9*, and is about what to do with a person who has had the Yale dangerous rOspA non-vaccine. He says not to test these LYMERix victims and he minimizes their symptoms, knowing that late, neurologic chronic Lyme symptoms are identical to what Schoen says are "nonspecific" (fatigue, meningitis, etc.; post-sepsis syndrome).

Schoen says the exact reverse in the Persing-Schoen-Corixa-RICO patent (US. Pat. No. 6,045,804 and associated journal report, <http://www.ncbi.nlm.nih.gov/pubmed/8968914>): "multisystem complaints characteristic of late Lyme," where the two developed the assay together but only Persing is listed on the patent.

WRITES SCHOEN (you can tell this is BS because it does not make any real sense):

“QUESTION

“Is this patient's presentation compatible with Lyme disease?

“COMMENT

This patient presents with nonspecific symptoms, including arthralgias and fatigue. Although he lives in an area endemic for Lyme disease, these findings by themselves do not point to Lyme disease.

“The risks of a false-positive serologic test result in this patient will be significant because the prevalence of Lyme disease in such individuals is low. More importantly, this patient has already received a Lyme disease vaccine. Because of this, he will have antibodies against the 31-kd OspA *Borrelia burgdorferi* protein. These antibodies will be directed by the Lyme ELISA and will generate a positive test result.

“In the absence of specific clinical features suggesting a diagnosis of Lyme disease, the best course of action may be not to do serologic testing for Lyme disease at all. If such testing is to be done in a person who has received the Lyme disease vaccine, it will need to be sent to a laboratory where the Western blot analysis can be done that omits the 31-kd response.”

"CONCLUSION:

"In Lyme disease recipients (sic), Western blot analysis is indicated to distinguish Lyme disease from seroconversion caused by vaccination."

Schoen (above) probably means “In Lyme disease vaccine recipients, Western blot analysis is indicated to distinguish disease from seroconversion by vaccination.”

This does not make a whole lot of sense because Schoen first said not to test them, just blow these people off, essentially, because their symptoms were vague (means, “not a red, swollen knee”). But then Schoen went on to say that if you MUST test them, use the Persing-Schoen RICO patent method with the OspA-B plasmid missing, making it very clear that the reason OspA and B were left out of the Dearborn standard was to satisfy this subsequent racketeering condition or monopoly on testing, once LYMERix was on the market. That is why I call this the RICO patent:

<http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnethtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804>

This transcript of Schoen’s “Clinical Vignettes” above is in that textbook with the libel and false statements including the [Munchausen’s accusations](#):

“Lyme Disease, ACP Key Diseases Series, by Rahn and Evans”

ISBN-10: 0-943126-58-4 , ISBN-13: 9780943126586

Publisher: American College of Physicians Year: January 15, 1998

http://www.amazon.com/Lyme-Disease-Key-Diseases-Series/dp/0943126584/ref=sr_1_fkmr0_2?ie=UTF8&qid=1341914626&sr=8-2-fkmr0&keywords=lyme+disease+rhan+and+evans

See more at <http://www.actionlyme.org/>

[SCHOEN_INSTRUCTING_DOCS_TO_BLOW_OFF_LYMERIX_INJUREES.htm](#)

From start to finish, from when the ALDF.com was established in 1990... to Steere going to Europe in 1992 to falsify the case definition antibody panel and adding the ridiculous ELISA “screening test” (for arthritis only) for a fungal-like disease ... to the CDC falsifying the testing for Lyme at Dearborn in 1994 ... to lying to the FDA and the journals about their outcomes of the 2 vaccine trials in 1998, to fake “Guidelines” based on the bogus Klempner non-retreatment non-study in 2001.... the point of this scam was to create a condition where only *they*—the CDC staff and the ALDF.com—would be able to capitalize on vector-borne diseases vaccines and test kits.

They intended to get all the grants, all the royalties, and to define the diseases based on their fake products.

Most importantly, they wanted this post-LYMERix monopoly on human blood testing because they could pharm from that not only human DNA and disease susceptibilities, but new vector borne disease DNA to patent. It was all about the money. It was all about cornering the market on this new genre of potential diseases resulting from global pollution.

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Falsifying the Vaccine Trial Results, Part 2 of the Cryme – the Unreadable Western Blots.

The 1998 Vaccines Reports (ImuLyme and LYMERix):

LYMERix results (76% "safe and effective"):

[N Engl J Med](#). 1998 Jul 23;339(4):209-15.

Vaccination against Lyme disease with recombinant *Borrelia burgdorferi* outer-surface lipoprotein A with adjuvant. Lyme Disease Vaccine Study Group.

[Steere AC](#)1, [Sikand VK](#), [Meurice F](#), [Parenti DL](#), [Fikrig E](#), [Schoen RT](#), [Nowakowski J](#), [Schmid CH](#), [Laukamp S](#), [Buscarino C](#), [Krause DS](#).

<https://www.ncbi.nlm.nih.gov/pubmed/9673298> <http://content.nejm.org/cgi/content/abstract/339/4/209>

ImmuLyme results (92% "safe and effective"):

[N Engl J Med](#). 1998 Jul 23;339(4):216-22.

A vaccine consisting of recombinant *Borrelia burgdorferi* outer-surface protein A to prevent Lyme disease. Recombinant Outer-Surface Protein A Lyme Disease Vaccine Study Consortium.

[Sigal LH](#)1, [Zahradnik JM](#), [Lavin P](#), [Patella SJ](#), [Bryant G](#), [Haselby R](#), [Hilton E](#), [Kunkel M](#), [Adler-Klein D](#), [Doherty T](#), [Evans J](#), [Molloy PJ](#), [Seidner AL](#), [Sabetta JR](#), [Simon HJ](#), [Klempner MS](#), [Mays J](#), [Marks D](#), [Malawista SE](#).

<https://www.ncbi.nlm.nih.gov/pubmed/9673299> <http://content.nejm.org/cgi/content/abstract/339/4/216>

From the LYMERix trial, "categories of outcomes:"

<http://content.nejm.org/cgi/content-nw/full/339/4/209/T1>

YET, here are the Cabal claiming "**we can't read our OspA vaccine results**" reports, which means they lied in their OspA vaccine safety and efficacy reports, since they both claimed to be using the Dearborn method and MarDx's Western Blot test strips:

[Clin Infect Dis](#). 2000 Jul;31(1):42-7. Epub 2000 Jul 17.

Detection of multiple reactive protein species by immunoblotting after recombinant outer surface protein A lyme disease vaccination.

[Molloy PJ](#)1, [Berardi VP](#), [Persing DH](#), [Sigal LH](#).

“...The manufacturer of the only currently FDA-approved (and released) recombinant OspA Lyme disease vaccine has suggested that vaccination does not interfere with serological evaluation of Lyme disease in vaccine recipients—a statement that is not supported by the data presented here.”

<https://www.ncbi.nlm.nih.gov/pubmed/10913394> <https://academic.oup.com/cid/article-lookup/doi/10.1086/313920>

Yale’s Robert Schoen and Mayo’s/Corixa’s David Persing, with John Anderson, 1995-6; the RICO within the RICO report which shows the intended monopoly on post-LYMERix testing for vector borne diseases (for Yale, Imugen and Corixa, officially “partners” listed on the SEC):

J Clin Microbiol. 1997 Jan;35(1):233-8.

Borrelia burgdorferi enzyme-linked immunosorbent assay for discrimination of OspA vaccination from spirochete infection.

Zhang YQ1, Mathiesen D, Kolbert CP, Anderson J, Schoen RT, Fikrig E, Persing DH.

<https://www.ncbi.nlm.nih.gov/pubmed/8968914> <http://jcm.asm.org/cgi/reprint/35/1/233?view=long&pmid=8968914>

Same: Schoen and Persing in their 1995-6 RICO method patent:

<http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnethtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804>

In this patent, they state:

“... 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.** Vaccine failures have been occasionally noted in animal models (E. Fikrig et al., *Science*, 250, 553-6 (1990)), and infection with antigenically variant strains of *B. burgdorferi*, which are being increasingly documented in the U.S., might still occur.”

They state that they can't tell the difference between Lyme and LYMERix disease, they're both multi-system diseases (post-sepsis).

Yale's Robert Schoen, as you've seen previously in “Clinical Vignettes” above, in the 1998 Munchausen's Book, instructed MDs to blow off LYMERix-systemically-injured people (“but send the post-vaccination blood to the Yale L2 Diagnostics RICO lab if you must bother to be a physician”). They used the bogus Dearborn method, reported that they had “safe and effective vaccines,” did not report that their Western Blots were unreadable. Which means they had **NO** vaccines, and also reported in their patents that the vaccines caused a disease identical to “multisystem” Lyme. Each vaccine trial report and summary was 3 false claims. Not safe, and not effective, and the Dearborn case definition was false and not even a consensus.

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In the fall of 1998, the LYMERix vaccine was approved by the FDA, anyway (the FDA panel being loaded with people like Allen Steere, Robert Schoen, and Vijay Sikand—the very people who ran the OspA trials). It came onto the market in late 1998 “despite numerous provisos.”

More than 1,000 systemic adverse events were reported through the VAERS from September 1999 to November 2000, whereupon the FDA granted a public hearing, January 31,

2001: <http://www.fda.gov/ohrms/dockets/ac/01/slides/3680s2.htm>

Whereupon, the whistle was blown on Dearborn and how LYMERix actually caused immunosuppression (the FDA did not scan in the last 19 pages of this booklet, which were 19 pages out of the Dearborn booklet, proving no one agreed with Steere's proposal for an antibody panel for a "case definition"): http://www.fda.gov/ohrms/dockets/ac/01/slides/3680s2_11.pdf

Several months later, in the fall of 2001, Karen Forschner of the Hartford, CT based Lyme Disease Foundation (Lyme.org) delivered to the FDA—in person, a patent owned by Brigitte Huber at Tufts University, where it was declared that OspA was technically a “toxin,” right in the abstract (US Patent 6,689,384). The FDA then gave SmithKline and Yale, the assignee of the LYMERix patent, an ultimatum that said essentially this: “Either you remove LYMERix voluntarily or we will order it off the market.” SmithKline chose to avoid the embarrassment and pulled their own non-vaccine off the market.

We’re still stuck with this bogus Dearborn case definition, despite numerous attempts at lawsuits against IDSA, SmithKline, and filing complaints to the U. S. Department of Justice. It is still very dangerous for the public to be unaware that the average person, or 85% of us—who are the “seronegative patients are the sickest,” have no chance of testing positive to this criminal CDC- Dearborn standard, because the actual disease is one of immunosuppression, or is an Acquired Immune Deficiency, or is similar to AIDS with all the opportunistic viral infections and lymphocyte mutations that can’t be treated with antibiotics, alone.

It was said at the time LYMERix was still on the market that this vaccine, via its claimed mechanism of disinfecting ticks with human antibodies (yes, if you can believe it), that LYMERix would turn humans into walking canisters of tick disinfectant, when in fact, LYMERix turned people into walking “cesspools of disease.” The same is true for Chronic Lyme. Chronic Lyme victims’ immune systems are “overwhelmed”- a term used by CDC officer Alan Barbour, when describing what antigenic variation in spirochetes does to humans (US Patent 6,719,983). This is a term you want to remember in case you hear it again: “overwhelmed” immune system means: “turned off.” “Turned off” is the complete opposite of an “inflammatory” or “autoimmune disease.”



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CHARGE SHEET 2: THE PATENTS

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

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Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 2: The Patents

The “Lyme Disease” Patents – the research fraud and racketeering complaint data for the DOJ to Prosecute.

Again, a little background about this Dearborn/OspA-scam, since it is the central concept or essence of the crimes: “Dearborn” refers to the 1994 CDC conference (took place in Dearborn, MI) where the testing for Lyme was falsified, or changed from that which represented the Lyme spirochete as just another relapsing fever organism...to something else entirely contrived (not even empirically perceived) and false. That event is discussed in the main “Cryme” video and the other ones in the YouTube series called “Lyme Crymes.”

For years, no one in the Cabal would admit that rOspA (recombinant outer surface protein A of the *Borrelia burgdorferi* organism or the Lyme vaccines that came and went) was Pam3Cys, because they couldn't. If they said “OspA is Pam3Cys,” everyone would know from officialdom that it was never a vaccine and the ALDF.com's (now IDSA's) whole house of cards would collapse. rOspA is a fungal antigen that causes immunosuppression – the opposite of a “vaccine.” If OspA was not a vaccine, then the CDC's 1994 “Dearborn” 2-tiered testing schema was a lie.

The falsified Dearborn case definition was the lie invented to pass off bogus OspA vaccines. You can tell for sure because they left OspA and B out (A and B are encoded on the same plasmid so you can't leave out one without the other) of the diagnostic standard. One never tests for vaccine efficacy with the same antigen that is the vaccine.

For example, if someone made a recombinant measles vaccine based on a DNA sequence that coded for say, “XYZ” surface antigen, they would not use recombinant “XYZ” surface antigen in the testing schema to see if a person had measles because they would not know if the antibody band was from the organism or the vaccine.

It was known at least since the late 1980s that people with late neurologic Lyme disease ceased to produce antibodies. However, the Dearborn case definition (Steere, that is) rejected those classes of disease—early and late meningitis or chronic neurologic cases—and said instead that only the blatantly, highly immunoreactive class of Lyme victims, those with the HLA-linked or arthritis-linked or hypersensitivity-linked cases, or those who produced abundant IgG antibodies could be called a “case” of “Lyme Disease.” This falsification of the testing was as much a semantics game as it was straight up research fraud from these DNA patentees. This Dearborn event left the sickest people with no disease diagnosis.

If someone intended a monopoly on a new diseases set or an entirely new class of diseases, such as what African vector borne diseases arriving in North America were discovered to be, what would they do? They'd make sure they got all of it: vaccines, test kits, grants, funding, all future blood testing for all future potentially patentable goodies in that blood, publicity, their names on plaques and statues (like Alan Barbour), awards like an “Astute Clinician Award,”... or, they could be knighted like Simon Wessely, a psychiatrist who helps by calling all the victims of the Lyme scam and Gulf War Illness “crazy” and “terrorists,” or like some US States naming, like, “Allen Steere Day” after them...

The Dearborn stunt is to the present day, the lie these criminals are trying to defend by issuing fake “guidelines” based on the fraudulent, 2001, Klempner non-retreatment report,

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

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

[Klempner MS](#)¹, [Hu LT](#), [Evans J](#), [Schmid CH](#), [Johnson GM](#), [Trevino RP](#), [Norton D](#), [Levy L](#), [Wall D](#), [McCall J](#), [Kosinski M](#), [Weinstein A](#).

<http://www.ncbi.nlm.nih.gov/pubmed/11450676>

And with this cabal’s chronic hystriionics over the development of other tests for Lyme, etc., because that, Dearborn, will be the most serious of criminal charges – the Fraud, Negligence, and Denial of Rights via Color of Law charges. All the ALDF.com and DNA patent-owners, here, have slandered and libeled against Lyme victims, making this not simply a negligence charge. All of their derogatory slander and label and trash-talking Lyme and LYMERix victims show “intent to cause harm.”

Barbour, Alan G., CDC officer and participant in the Dearborn conference, owns 30+ patents including the ImuLyme OspA patent. The ImuLyme vaccine trial report authors (Sigal, et al) and Barbour assert that one must be sure lipids are attached to all Osps or else they will not be immunogenic, yet Steere’s Dearborn panel was developed from high passage G39/40 and FRG with recombinant OspA and B with no lipids attached, leaving OspA and B out of the case definition panel.

Barbour’s ImuLyme patent (Berstrom, Magnarelli) European Patent # (5092/88, DK)

<http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetahml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5523089.PN.&OS=PN/5523089&RS=PN/5523089>

Yale’s LYMERix OspA patent (5,747,294):

<http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetahml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5747294.PN.&OS=PN/5747294&RS=PN/5747294>

ImuLyme trial result (falsified; used Dearborn, blots were unreadable):

[N Engl J Med.](#) 1998 Jul 23;339(4):216-22.

A vaccine consisting of recombinant Borrelia burgdorferi outer-surface protein A to prevent Lyme disease. Recombinant Outer-Surface Protein A Lyme Disease Vaccine Study Consortium.

[Sigal LH](#)¹, [Zahradnik JM](#), [Lavin P](#), [Patella SJ](#), [Bryant G](#), [Haselby R](#), [Hilton E](#), [Kunkel M](#), [Adler-Klein D](#), [Doherty T](#), [Evans J](#), [Molloy PJ](#), [Seidner AL](#), [Sabetta JR](#), [Simon HJ](#), [Klempner MS](#), [Mays J](#), [Marks D](#), [Malawista SE](#).

<http://www.ncbi.nlm.nih.gov/pubmed/9673299>

LYMERix trial result (falsified; used Dearborn, blots were unreadable):

[N Engl J Med.](#) 1998 Jul 23;339(4):209-15.

Vaccination against Lyme disease with recombinant Borrelia burgdorferi outer-surface lipoprotein A with adjuvant. Lyme Disease Vaccine Study Group.

Steere AC1, Sikand VK, Meurice F, Parenti DL, Fikrig E, Schoen RT, Nowakowski J, Schmid CH, Laukamp S, Buscarino C, Krause DS.
<http://www.ncbi.nlm.nih.gov/pubmed/9673298>

Barbour says OspA will not work as a vaccine due to antigenic variation, 1992:

J Exp Med. 1992 Sep 1;176(3):799-809.

Antibody-resistant mutants of Borrelia burgdorferi: in vitro selection and characterization.

Sădziene A1, Rosa PA, Thompson PA, Hogan DM, Barbour AG.

<http://www.ncbi.nlm.nih.gov/pubmed/1339462>

Fikrig and Flavell say OspA will not work as a vaccine due to “selection pressure” or antigenic variation, 1995:

Infect Immun. 1995 May;63(5):1658-62.

Selection of variant Borrelia burgdorferi isolates from mice immunized with outer surface protein A or B.

Fikrig E1, Tao H, Barthold SW, Flavell RA.

<http://www.ncbi.nlm.nih.gov/pubmed/7729870>

1992-1994. Steere Falsifies Test in Europe: uses “high passage strains” and “OspA and B without the lipid attached” to leave OspA and B out of the standard for his later monopoly on vector borne diseases testing. Only Corixa, Imugen and Yale were to be licensed to use the RICO strain patent by Dave Persing (US Patent # 6,045,804)

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.

“The group 1 strain of B. burgdorferi, G39/40, used in this study and in the previous study of US patients was isolated from an Ixodes damini tick in Guilford, Connecticut [21]. The group 2 strain, FRG [Federal Republic of Germany], was isolated from Ixodes ricinus near Cologne [22]. The group 3 strain, IP3, was isolated from Ixodes persulcatus near Leningrad [23]. All three strains used in this study were high passage isolates, which were classified by Richard Marconi (Rocky Mountain Laboratory, Hamilton, MT) using 16S ribosomal RNA sequence determination as described [11, 24]. The recombinant preparations of OspA and OspB used in this study were purified maltose-binding protein-Osp fusion proteins derived from group 1 strain B31 [25]. The fusion proteins contained the full-length OspA or OspB sequence without the lipid moiety or the signal sequence -”

<http://www.ncbi.nlm.nih.gov/pubmed/8106763>

Full Text @ http://www.actionlyme.org/STEERE_IN_EUROPE.htm

Dearborn: http://www.actionlyme.org/DEARBORN_PDF.pdf

See in the Dearborn booklet: none of the labs agreed with this Dearborn proposal in that Dearborn pdf. (Except MarDx Labs, which was given arthritis-positive blood to qualify their Western Blot strips prior to Dearborn; MarDx was then given both vaccine trial contracts; MarDx was then sold to a company in Ireland, taking all the fraud data with them; Trinity Biotech was the name of the company that bought MarDx).

The CDC sent labs an invitation to participate, but then CDC blew them all off:

<http://www.actionlyme.org/DEARBORNINVITATION.pdf>

Barbour also patented Masters' disease or STARI while the crooks played the DNA/RNA shell game to pull the wool over Edwin Masters' eyes and to say "There is no 'Lyme' in Missouri or the south." To patent a unique species, you have to patent the flagellin gene. The same should be true for diagnostics – using unique recombinant flagella from all *Borreliae*.

Telford Phylogeny with Masters' *Amblyomma* *Borrelia*:

J Clin Microbiol. 2001 Feb;39(2):494-7.

Lone star tick-infecting borreliae are most closely related to the agent of bovine borreliosis.

Rich SM1, Armstrong PM, Smith RD, Telford SR 3rd.

<http://www.ncbi.nlm.nih.gov/pubmed/11158095>

Barbour's Patent for Masters' disease or something close to it [either *lonestari* or *barbouri*; the phylogenetic data says they are the same in 16S RNA and only slightly different in the flagellin gene (96% homology), so both evolved from *theileri*, which is cow relapsing fever; one species is now called *barbouri* and the other is called *lonestari*, but really they are Masters disease, since he was the one who for years claimed there was a Lyme-like illness in the south, associated with a Lyme-like rash and tick bite]:

<http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetahml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5,932,220.PN.&OS=PN/5,932,220&RS=PN/5,932,220>

Johnson, Barbara J., CDC officer, Ft. Collins, CO., owner of 5 patents in Europe with SmithKline, talks about differences in HLAs of mice, referring to their tendency to produce HLA-linked hypersensitivity responses or not, meaning she is aware that the same applies to humans. CDC's BJ Johnson oversaw this Dearborn-Falsification-of-Lyme-Testing stunt (Oct 1994). She said to not use high passage strains, yet high passage G39/40 and FRG (Federal Republic of Germany) were what Steere used to develop the Dearborn panel along with recombinant OspA and B with no lipids attached. This was done, again, to assure OspA and B were not included in the Dearborn panel, ... to facilitate what Steere, Corixa, L2 Diagnostics and Imugen intended to do after LYMERix was on the market, ... which was to monopolize the Lyme or tick-borne diseases testing market where "the vaccination status was unknown." RICO patent 6,045,804]

Johnson's Patents (5 in all):

http://worldwide.espacenet.com/publicationDetails/biblio?DB=worldwide.espacenet.com&II=0&ND=3&adjacent=true&locale=en_EP&FT=D&date=19931209&CC=WO&NR=9324145A1&KC=A1

Dearborn Booklet http://www.actionlyme.org/DEARBORN_PDF.pdf

Fikrig and Flavell – own both the only scientifically valid method to detect Lyme and also own the LYMERix OspA patent. ***Their FDA-valid flagellin method was not used to assess the outcome of LYMERix because they knew not only did LYMERix not work because Lyme is a Relapsing Fever organism and undergoes antigenic variation (OspA itself, Fikrig and Flavell said, undergoes antigenic variation or “selection pressure” and would be no good as a vaccine), but Pam3Cys or TLR2/1 agonists (OspA is Pam3Cys) are fungal and cause immunosuppression in most people – especially people without Steere's alleged HLA-linked hypersensitivity responses.***

OspA patent

<http://patft1.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=5747294.PN.&OS=PN/5747294&RS=PN/5747294>

Fikrig and Flavell's (Yale's) Valid (per FDA) flagellin method patent 5,618,533:

<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=5,618,533.PN.&OS=PN/5,618,533&RS=PN/5,618,533>

The PubMed report that goes with the Yale FDA-validated flagellin method (detects 94.4% of all cases, including earliest and late neurologic), 1991:

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.

<http://www.ncbi.nlm.nih.gov/pubmed/1894359>

Fikrig and Flavell say OspA will not work due to antigenic variation:

Infect Immun. 1995 May;63(5):1658-62.

Selection of variant *Borrelia burgdorferi* isolates from mice immunized with outer surface protein A or B.

Fikrig E1, Tao H, Barthold SW, Flavell RA.

<http://www.ncbi.nlm.nih.gov/pubmed/7729870>

Padula and OspC – says *Borrelia burgdorferi* strain B31 has little to no OspC in it, meaning whoever Western Blots with this strain will be leaving OspA, B and C out of the standard. If you have those bands, you will be told you do not have Lyme, yet they are the “primary, immunodominant antigens,” which was why they got the assignments A, B, C, etc. SmithKline used this strain, B31, to WB LYMERix victims and claimed to be using the Dearborn method to detect Lyme or vaccine failure.

Borrelia burgdorferi enzyme-linked immunosorbent assay for discrimination of OspA vaccination from spirochete infection.

Zhang YQ1, Mathiesen D, Kolbert CP, Anderson J, Schoen RT, Fikrig E, Persing DH.
<http://www.ncbi.nlm.nih.gov/pubmed/8968914>

Dattwyler, Raymond J. - owns a patent that describes OspA as Pam3Cys. Therefore it could not have been a blood-stream-injected “vaccine,” because it is a human TLR2/1 agonist. This Dattwyler patent is for an inhalation form of OspA/Pam3Cys. Lung immunity is different from injecting fungal antigens directly into the blood stream:

(US20090324638) LIVE BACTERIAL VACCINE

"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 sulfhydryl 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 through 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>

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The Mayo Clinic advertised the RICO within the RICO – a patent of which they were the assignee and would have gotten royalties (6,045,804); CT AG and now Senator Richard Blumenthal’s staff were interested to know if this RICO cabal including Yale, Imugen and Corixa ever advertised their intended monopoly on post-LYMERix blood testing for North America “where the vaccination status was unknown.” This is one example. Yale also advertised this new test:

Can be found at:

<https://groups.google.com/forum/#!original/sci.med.diseases.lyme/D6v-QHQdMbc/WupHjKwFilJ>

http://www.mayo.edu/comm/mcr/news/news_361.html

“Mayo Clinic Rochester News

Tuesday, August 4, 1998

“New Tests Set Standard for Diagnosing Lyme Disease

ROCHESTER, MINN. — Mayo Medical Laboratories and IMUGEN Inc. announced today the newest and most accurate test series available for diagnosing Lyme disease. The tests also are the only reliable means of diagnosing Lyme disease in people who have been vaccinated against Lyme disease.

“Mayo Medical Laboratories, the laboratory for Mayo Clinic, and IMUGEN Inc. of Norwood, Mass., are jointly offering the new proprietary tests through local hospitals and clinics. Availability of the new tests coincides with the anticipated release of new Lyme disease vaccines, such as the widely-publicized LYMERix and ImuLyme.

“In research trials, all other Lyme tests have been shown to produce false-positive results in people vaccinated against Lyme disease. Moreover, the downstream costs of medical care delivered on the basis of just one false-positive Lyme test can be as much as \$15,000.

“According to Dr. David Persing, a Mayo Clinic molecular biologist involved in the discovery of the new test components, physicians now have a new and more reliable means of diagnosing patients who present with symptoms of Lyme disease.

"These tests should help reduce the human and financial costs associated with the number of undiagnosed, misdiagnosed, untreated or improperly treated patients," Dr. Persing added.

“Scientists at IMUGEN, recognized nationally as the leading reference laboratory for tick-borne diseases, are responsible for developing the highly accurate immunologic methods to utilize Dr.Persing’s discovery.

"Diagnosing Lyme disease has been highly problematic for a long time," said Victor Berardi, chief executive officer of IMUGEN, whose laboratories have performed more than a half-million Lyme disease tests. "Our new tests will greatly help physicians in distinguishing patients who are actually infected from those who aren't. Furthermore, the accuracy of these tests will not be affected by Lyme vaccine. In any case, the tests will help physicians render more appropriate and cost effective care.”

“Lyme disease is a tick-borne illness that if left undiagnosed or untreated can severely damage the human heart and nervous system. Nationally more than 16,000 cases of Lyme disease were reported to the Centers for Disease Control and Prevention (CDC) in 1996. The majority of cases were reported in New England and the Northeast. The CDC reports that the overall number of Lyme disease cases could climb to 25,710 by the year 2000.

“In a study of 10,936 people in states with a high incidence of Lyme disease, one new vaccine proved 79 percent effective at preventing Lyme disease infections after complete dosage. Given the potential popularity of the vaccine, and the recent epidemic of Lyme disease in the Northeast, the new tests offered by Mayo Medical Laboratories and IMUGEN will be of considerable value.

“The new Lyme disease tests detect multiple classes of antibody isotypes, enabling them to discriminate between the vaccine and a true Lyme infection. Existing Lyme disease tests, however, have shown to produce false-positive results in patients vaccinated for Lyme disease.

“IMUGEN Inc. of Norwood, Mass., is a pioneer in the research, development and testing of tick-borne diseases, including Lyme disease, babesiosis and ehrlichiosis. For the past decade, IMUGEN has provided clinics and hospitals in the Northeast with

high-quality serologic testing from its facilities in Norwood, Mass., and Southhampton Hospital in Southhampton, N.Y. For more information, call 781-255-0770.

“Mayo Medical Laboratories is the laboratory for Mayo Clinic and provides lab services to community-based healthcare organizations throughout the nation and world. Mayo Medical Laboratories draws from the expertise of Mayo Clinic’s 1,600 physicians and scientists who provide specialized consultation on test selection, utilization and interpretation.

“For information, call 800-533-1710.

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“Contact: “Tom Huyck “507-284-0003 (days)

“507-284-2511 (evenings)”



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CHARGE SHEET 3: THE BIOMARKERS

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

© 2017, February, Society for the Advancement of Scientific Hermeneutics, (\$A\$H).

Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 3: The Biomarkers

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

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

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

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

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

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

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

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

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

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

[Klempner MS](#)¹, [Hu LT](#), [Evans J](#), [Schmid CH](#), [Johnson GM](#), [Trevino RP](#), [Norton D](#), [Levy L](#), [Wall D](#), [McCall J](#), [Kosinski M](#), [Weinstein A](#).

“BACKGROUND:

“It is controversial whether prolonged antibiotic treatment is effective for patients in whom symptoms persist after the recommended antibiotic treatment for acute Lyme disease.

METHODS:

We conducted two randomized trials: one in 78 patients who were seropositive for IgG antibodies to *Borrelia burgdorferi* at the time of enrollment and the other in 51 patients who were seronegative. The patients received either intravenous ceftriaxone, 2 g daily for 30 days, followed by oral doxycycline, 200 mg daily for 60 days, or matching intravenous and oral placebos. Each patient had well-documented, previously treated Lyme disease but had persistent musculoskeletal pain, neurocognitive symptoms, or dysesthesia, often associated with fatigue. The primary outcome measures were improvement on the physical- and mental-health-component summary scales of the Medical Outcomes Study 36-item Short-Form General Health Survey (SF-36)--a scale measuring the health-related quality of life--on day 180 of the study.

RESULTS:

After a planned interim analysis, the data and safety monitoring board recommended that the studies be discontinued because data from the first 107 patients indicated that it was highly unlikely that a significant difference in treatment efficacy between the groups would be observed with the planned full enrollment of 260 patients. Base-line assessments documented severe impairment in the patients' health-related quality of life. In intention-to-treat analyses, there were no significant differences in the outcomes with prolonged antibiotic treatment as compared with placebo. Among the seropositive patients who were treated with antibiotics, there was improvement in the score on the physical-component summary scale of the SF-36, the mental-component summary scale, or both in 37 percent, no change in 29 percent, and worsening in 34 percent; among seropositive patients receiving placebo, there was improvement in 40 percent, no change in 26 percent, and worsening in 34 percent (P=0.96 for the comparison between treatment groups). The results were similar for the seronegative patients.

CONCLUSIONS:

There is considerable impairment of health-related quality of life among patients with persistent symptoms despite previous antibiotic treatment for acute Lyme disease. However, in these two trials, treatment with intravenous and oral antibiotics for 90 days did not improve symptoms more than placebo.”

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

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

new Primers Shell Game report), when in fact, whenever he did find such people, he rejected them from the study. Not only did he say this in the write up of the report protocol—if they were positive for Bb DNA in the spinal fluid, they would be rejected from the study—this actually happened. We know of at least one person who had Bb DNA in her spinal fluid that Klemmpner rejected from the study, yet he did not report this.

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

[Neurology](#). 2003 Jun 24;60(12):1916-22.

Cognitive function in post-treatment Lyme disease: do additional antibiotics help?

[Kaplan RF1](#), [Trevino RP](#), [Johnson GM](#), [Levy L](#), [Dornbush R](#), [Hu LT](#), [Evans J](#), [Weinstein A](#), [Schmid CH](#), [Klemmpner MS](#).

"CONCLUSION:

"Patients with post-treatment chronic Lyme disease who have symptoms but show no evidence of persisting Borrelia infection do not show objective evidence of cognitive impairment. Additional antibiotic therapy was not more beneficial than administering placebo."

<https://www.ncbi.nlm.nih.gov/pubmed/12821733>

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

Here are those 2 reports:

[J Infect Dis](#). 1998 Feb;177(2):401-8.

Matrix metalloproteinases in the cerebrospinal fluid of patients with Lyme neuroborreliosis.

[Perides G1](#), [Charness ME](#), [Tanner LM](#), [Péter O](#), [Satz N](#), [Steere AC](#), [Klemmpner MS](#).

"Neurologic manifestations of Lyme disease include meningitis, encephalopathy, and cranial and peripheral neuropathy. There are no sensitive markers for neuroborreliosis, and diagnosis is often based on clinical presentation and cerebrospinal fluid (CSF) abnormalities, including intrathecal antibody production. Matrix metalloproteinase (MMP) activity in CSF was compared in patients with neuroborreliosis, patients with diverse neurologic disorders, and healthy controls. The CSF of 17 of 18 healthy subjects and 33 of 37 patients with neurologic symptoms and normal CSF and imaging studies contained only MMP2. The CSF of several patients with neurologic disorders contained MMP2, MMP9, and gelatinolytic activity at 130 and 250 kDa. The 130-kDa MMP was found without the 92-kDa MMP9 in the CSF of 11 (79%) of 14 patients with neuroborreliosis and only 7 (6%) of 118 control patients (P < .001). This pattern of CSF gelatinase activity may be a useful marker for neuroborreliosis."

<https://www.ncbi.nlm.nih.gov/pubmed/9466528>

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

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

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

[Georgilis K I](#), [Peacocke M](#), [Klempner MS](#).

"The Lyme disease spirochete, *Borrelia burgdorferi*, can be recovered long after initial infection, even from antibiotic-treated patients, indicating that it resists eradication by host defense mechanisms and antibiotics. Since *B. burgdorferi* first infects skin, the possible protective effect of skin fibroblasts from an antibiotic commonly used to treat Lyme disease, ceftriaxone, was examined. Human foreskin fibroblasts protected *B. burgdorferi* from the lethal action of a 2-day exposure to ceftriaxone at 1 microgram/mL, 10-20 x MBC. In the absence of fibroblasts, organisms did not survive. Spirochetes were not protected from ceftriaxone by glutaraldehyde-fixed fibroblasts or fibroblast lysate, suggesting that a living cell was required. The ability of the organism to survive in the presence of fibroblasts was not related to its infectivity. Fibroblasts protected *B. burgdorferi* for at least 14 days of exposure to ceftriaxone. Mouse keratinocytes, HEp-2 cells, and Vero cells but not Caco-2 cells showed the same protective effect. Thus, several eukaryotic cell types provide the Lyme disease spirochete with a protective environment contributing to its long-term survival."

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

REPEAT: Mark Klempner also wrote in 1998 that anti-OspA antibodies might be the cause of anti-myelin antibodies or probably contributed to the MS form of Lyme. I think he may have meant OspC, since that was my reading of Roland Martin's 1988 "Lyme causes Multiple Sclerosis" report, but regardless, MS is not a personality or anxiety disorder:

http://actionlyme.org/KFORSCHNER_DISCOVERS_LYME_TOXIN.htm

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

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

A) MMP-130 - Klempner as shown above.

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

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

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

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

[Ann Intern Med.](#) 2000 Apr 18;132(8):661-8.

The Lyme disease vaccine: conception, development, and implementation.

[Thanassi WT1](#), [Schoen RT](#).

"Other peripheral neuropathies and Lyme meningitis are also seen at this stage. In late-stage disease, the central nervous system may be involved. A new diagnostic test measuring glial fibrillary acidic protein in cerebrospinal fluid may prove to be a useful tool for measuring such involvement (20)."

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

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

[Cell Mol Neurobiol.](#) 2001 Oct;21(5):477-95.

H9724, a monoclonal antibody to *Borrelia burgdorferi*'s flagellin, binds to heat shock protein 60 (HSP60) within live neuroblastoma cells: a potential role for HSP60 in peptide hormone signaling and in an autoimmune pathogenesis of the neuropathy of Lyme disease.

[Sigal LH1](#), [Williams S](#), [Soltys B](#), [Gupta R](#).

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

role in peptide-hormone-receptor function and demonstrates the modulatory potential of a monoclonal antibody on living cells."

<http://www.ncbi.nlm.nih.gov/pubmed/11860186>

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

D) QEEG or electroencephalograms (Sigal, primary [Munchausen's](#) accuser)

[Clin Electroencephalogr.](#) 1995 Jul;26(3):137-45.

QEEG and evoked potentials in central nervous system Lyme disease.

[Chabot RJ](#)1, [Sigal LH](#).

"Quantitative EEG, flash visual evoked potentials, auditory evoked potentials to common and rare tones, and median nerve somatosensory evoked potentials were obtained from 12 patients with active CNS Lyme disease and from 11 patients previously treated for active CNS Lyme disease. Abnormal QEEG and/or EPs were found in 75% of the active Lyme disease patients and in 54% of the post CNS Lyme disease patients. Three different types of neurophysiological abnormality were observed in these patients including QEEG slowing, possible signs of cortical hyperexcitability, and focal patterns indicating disturbed interhemispheric relationships. In patients tested before and after treatment QEEG and EP normalization was associated with clinical improvement."

<https://www.ncbi.nlm.nih.gov/pubmed/7554300>

E) SPECT or brain perfusion scanning (Steere)

[Neurology.](#) 1997 Dec;49(6):1661-70.

Reversible cerebral hypoperfusion in Lyme encephalopathy.

[Logigian EL](#)1, [Johnson KA](#), [Kijewski MF](#), [Kaplan RF](#), [Becker JA](#), [Jones KJ](#), [Garada BM](#), [Holman BL](#), [Steere AC](#).

"Lyme encephalopathy (LE) presents with subtle neuropsychiatric symptoms months to years after onset of infection with *Borrelia burgdorferi*. Brain magnetic resonance images are usually normal. We asked whether quantitative single photon emission computed tomography (SPECT) is a useful method to diagnose LE, to measure the response to antibiotic therapy, and to determine its neuroanatomic basis. In 13 patients with objective evidence of LE, SPECT demonstrated reduced cerebral perfusion (mean perfusion defect index [PDI] = 255), particularly in frontal subcortical and cortical regions. Six months after treatment with 1 month of intravenous ceftriaxone, perfusion significantly improved in all 13 patients (mean PDI = 188). In nine patients with neuropsychiatric symptoms following Lyme disease, but without objective abnormalities (e.g., possible LE), perfusion was similar to that of the treated LE group (mean PDI = 198); six possible LE patients (67%) had already received ceftriaxone prior to our evaluation. Perfusion was significantly lower in patients with LE and possible LE than in 26 normal subjects (mean PDI = 136), but 4 normal subjects (15%) had low perfusion in the LE range.

We conclude that LE patients have hypoperfusion of frontal subcortical and cortical structures that is partially reversed after ceftriaxone therapy. However, SPECT cannot be used alone to diagnose LE or determine the presence of active CNS infection."

<http://www.ncbi.nlm.nih.gov/pubmed/9409364>

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

F) Antiphospholipid antibodies (Steere and Yale claiming Lyme caused Lupus - probably more likely to be due to the reactivated EBV, but we will look more closely later)

"Reactivity of neuroborreliosis patients (Lyme disease) to cardiolipin and gangliosides."

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

<https://www.ncbi.nlm.nih.gov/pubmed/8410057>

FULL TEXT:

http://www.actionlyme.org/STEERE_AND_LUPUS_LYME.htm

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

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

[Neurology](#). 1992 Jan;42(1):43-50.

Neuroactive kynurenes in Lyme borreliosis.

[Halperin JJ1](#), [Heyes MP](#).

"In patients with encephalopathy, serum QUIN was elevated with corresponding increments in CSF QUIN. Lymphokine concentrations were not consistently elevated. We conclude that CSF QUIN is significantly elevated in *B burgdorferi* infection--dramatically in patients with CNS inflammation, less

in encephalopathy. The presence of this known agonist of NMDA synaptic function--a receptor involved in learning, memory, and synaptic plasticity--may contribute to the neurologic and cognitive deficits seen in many Lyme disease patients...."

<http://www.ncbi.nlm.nih.gov/pubmed/1531156>

H) Lyme Is associated with ALS (Halperin, Dattwyler):

Arch Neurol. 1990 May;47(5):586-94.

Immunologic reactivity against *Borrelia burgdorferi* in patients with motor neuron disease.

[Halperin JJ1](#), [Kaplan GP](#), [Brazinsky S](#), [Tsai TF](#), [Cheng T](#), [Ironside A](#), [Wu P](#), [Delfiner J](#), [Golightly M](#), [Brown RH](#), et al.

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

<https://www.ncbi.nlm.nih.gov/pubmed/2334308>

FULL TEXT:

<http://www.actionlyme.org/ALSLYME47.htm>

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

I) NO in the brain (Steere):

J Infect Dis. 1994 May;169(5):1014-22.

Borrelia burgdorferi and Escherichia coli lipopolysaccharides induce nitric oxide and interleukin-6 production in cultured rat brain cells.

[Tatro JB1](#), [Romero LI](#), [Beasley D](#), [Steere AC](#), [Reichlin S](#).

<http://www.ncbi.nlm.nih.gov/pubmed/7513330>

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

Nitric Oxide is a free-radical, neurotoxin.

J) Anti-ganglioside antibodies (Benach)

Infect Immun. 1995 Oct;63(10):4130-7.

Experimental immunization with *Borrelia burgdorferi* induces development of antibodies to gangliosides.

Garcia-Monco JC1, Seidman RJ, Benach JL.

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

<https://www.ncbi.nlm.nih.gov/pubmed/7558329>

<http://iai.asm.org/content/63/10/4130.full.pdf+html?view=long&pmid=7558329>

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

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

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

Rev Infect Dis. 1989 Sep-Oct;11 Suppl 6:S1487-93.

Clinical pathologic correlations of Lyme disease.

Duray PH1.

"Immature B cells can also be seen in the spinal fluid. These cells can appear quite atypical- not unlike those of transformed or neoplastic lymphocytes."

<http://www.ncbi.nlm.nih.gov/pubmed/2814170>

Full Text: http://www.actionlyme.org/IDSA_CLINIPATH_DURAY.htm

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

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1992:

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

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

Paul Duray, NCI, NIH, Ft. Detrick, at the 1992 Cold Spring Harbor Crooks' Conference, published in Steve Schutzer's *Lyme Disease: Molecular and Immunologic Approaches*. – book: Publisher, Cold Spring Harbor Press, 1992, ISBN: 087693770, 978087969377

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2006:

The NIH (NINDS' MS-Lyme Group) group that discovered that *** OspA *** was the cause of the MS/New Great Imitator outcome of Lyme reporting in the *New York Times* in the summer of 2013 by saying that Epstein-Barr might be the real culprit. (OspA causes immunosuppression and the reactivation of latent herpes viruses.)

The following journal article says these OspA like antigens constantly shed by *Borrelia* cause immunosuppression in the humoral immune system, but apparently a chronic inflammatory state in the central nervous system:

[J Neuropathol Exp Neurol](#). 2006 Jun;65(6):540-8.

***Borrelia burgdorferi* Induces TLR1 and TLR2 in human microglia and peripheral blood monocytes but differentially regulates HLA-class II expression.**

[Cassiani-Ingoni R1](#), [Cabral ES](#), [Lünemann JD](#), [Garza Z](#), [Magnus T](#), [Gelderblom H](#), [Munson PJ](#), [Marques A](#), [Martin R](#).

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

<http://www.ncbi.nlm.nih.gov/pubmed/16783164>

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

This next report by the same people (NINDS' Martin and Marques) means you might not even have anti-flagellar antibodies (flagellin is a TLR5-agonist) after being exposed to shed fungal OspA like antigens (TLR2/1-agonists):

[J Infect Dis](#). 2006 Mar 15;193(6):849-59. Epub 2006 Feb 8.

***Borrelia burgdorferi* lipoprotein-mediated TLR2 stimulation causes the down-regulation of TLR5 in human monocytes.**

[Cabral ES1](#), [Gelderblom H](#), [Hornung RL](#), [Munson PJ](#), [Martin R](#), [Marques AR](#).

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

<http://www.ncbi.nlm.nih.gov/pubmed/16479520>

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

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

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

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

"Complicating the picture is the fact that some people with PTLDS symptoms apparently never had Lyme disease in the first place, Dr. Marques said in an interview. There are other infectious organisms — **Epstein-Barr virus, for example** — **that can produce similar symptoms and may be the real culprits.**"

<http://well.blogs.nytimes.com/2013/07/08/when-lyme-disease-lasts-and-lasts/>

!!!

Ya think? What causes everything? MS, Lupus, Cancer, etc... ??

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2014:

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

"Dormant viruses re-emerge in patients with lingering sepsis, signaling immune suppression"

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

"These viruses generally don't lead to significant illness in people who are healthy but can cause problems in patients who are immune-suppressed. "

<http://news.wustl.edu/news/Pages/27015.aspx>

FULL JOURNAL REPORT, snippet...

[PLoS One](#). 2014 Jun 11;9(2):e98819. doi: 10.1371/journal.pone.0098819. eCollection 2014.

Reactivation of multiple viruses in patients with sepsis.

[Walton AH1](#), [Muenzer JT2](#), [Rasche D1](#), [Boomer JS3](#), [Sato B4](#), [Brownstein BH1](#), [Pachot A5](#), [Brooks TL3](#), [Deych E3](#), [Shannon WD3](#), [Green JM3](#), [Storch GA2](#), [Hotchkiss RS1](#).

"Sepsis is the host's non-resolving inflammatory response to infection that leads to organ dysfunction [1], [2]. A current controversial hypothesis postulates that if sepsis pursues a protracted course, it progresses from an initial primarily hyper-inflammatory phase to a predominantly immunosuppressive state [3]–[7]. Experimental therapeutic approaches in sepsis have almost exclusively focused on blocking early inflammation or host-pathogen interaction and failed [8]–[10]. Recently, immunoadjuvant 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-adjvant therapies might improve sepsis survival if administered during the later immunosuppressive phase, these agents might worsen outcome if given during the early hyper-inflammatory phase [4], [14]. Thus, a means to distinguish these two contrasting phases of sepsis is needed not only to verify the hypothesis that sepsis progresses to an immunosuppressive state but also to guide use of potential agents which boost immunity.

“Latent viruses such as cytomegalovirus are normally held in abeyance by cellular and immune surveillance mechanisms which if impaired, for example by immunosuppressive medications, often result in viral reactivation, replication, and virally-mediated tissue injury [15]–[20]. Sepsis impairs innate and adaptive immunity by multiple mechanisms including apoptosis-induced depletion of immune effector cells and induction of T-cell exhaustion thereby possibly predisposing to viral reactivation and dissemination [21]–[23]. ...”

<https://www.ncbi.nlm.nih.gov/pubmed/24919177>

<http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0098819>

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

“Surviving Sepsis: Detection and Treatment Advances”

By Carolyn Beans for the National Institutes of Health | August 18, 2014 08:43am ET

"Preventing Secondary Infections

"Some people who survive sepsis can develop secondary infections days or even months later. A research team that included Richard Hotchkiss, Jonathan Green and Gregory Storch of Washington University School of Medicine in St. Louis suspected that this is because sepsis might cause lasting damage to the immune system. To test this hypothesis, the scientists compared viral activation in people with sepsis, other critically ill people and healthy individuals. The researchers looked for viruses like Epstein-Barr and herpes simplex that are often dormant in healthy people but can reactivate in those with suppressed immune systems. [Sepsis Has Long-Term Impact for Older Adults, Study Finds]" <http://www.livescience.com/47387-sepsis-diagnosis-treatment-research-nigms.html>

Sepsis Has Long-Term Impact for Older Adults, Study Finds

By Rachael Rettner, Senior Writer | October 26, 2010

“About 60 percent of sepsis patients experienced worsening cognitive or physical function, or both, after their infection, the researchers say... Nearly 17 percent showed signs of moderate to severe cognitive impairment, compared with about 6 percent before the sepsis infection. Patients hospitalized for something other than sepsis did not show an increase in cognitive problems.”

<http://www.livescience.com/8831-sepsis-long-term-impact-older-adults-study-finds.html>

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



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healthcare justice

CHARGE SHEET 4: THE PRIMERS SHELL GAME

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

© 2017, February, Society for the Advancement of Scientific Hermeneutics, (\$A\$H).

Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 4: Primers Shell Game

The Primers Shell Game – using the *right* DNA and RNA to identify spirochetes to patent, but using the *wrong* DNA/rDNA (the DNA known to *not* be present) when assessing for spirochetes in humans.

Updates on Mechanisms of Illness, Baumgarth, Chiu/Aucott, Duray, Rockefeller University (Lyme and LYMERix are incurable, spirochetes target lymph nodes, fungal antigens cause B cell immortalization), **page 1**

Background, “Clinical Violence” or “Deprivation of Rights via Color of Law abuses”, **page 14**

I. Phage-vectored plasmids are variable DNA (like OspA); not to be used for human disease, **page 17**

II. Borrelia Acquiring Sticky OspA, and OspA Sticking to Itself (falsified vaccines reporting, blot smudging, Korean Chemists on OspA being sticky and clumping) – **page 19**

III. Lyme spirochetes did not evolve naturally and are closest to an African bird borreliosis, **page 21**

IV. Brain Permanence, Tropism and the Single Spirochete Infection with *resultant MULTIPLE VARIANTS* **page 26**

V. SIDESTEPPING - Alert on “Biofilms” **page 32**

VI. On using the correct DNA to look for spirochetes in humans by using recombinant Borrelia-specific flagellin DNA product to detect those specific antibodies **page 33**

VII. The FDA being forced to assure Lyme testing is valid according to FDA’s own rules by the Senators (summer, 2014 1), **page 33**

VIII. SIDE-STEPPING - CDC’s Other Research Fraud: A) Lying about the viability of the cyst or spheroplast form of spirochetes and B) lying about mycoplasma not being involved in Chronic Fatigue Syndrome **page 35**

IX. The CDC Cabal Play the DNA and RNA Shell Game: *Alan Barbour, Durland Fish, Gary Wormser, Mark Klempner, Robert Schoen, and Allen Steere* **page 36**

X. The Guidelines – Who signed on to this perverted science and is therefore responsible for endorsing this fraud? **Page 57**

2017 Update:

This charge sheet is a revision of the 2014 Criminal Charges Sheet called the “DNA or Primers Shell Game.” Since that time we learned from Nicole Baumgarth, et al, at UC Davis, and Chiu/Aucott at UCSF, that spirochetes go right to the lymph nodes and destroy the B cell maturation or germinal centers,... and that around half of all tick bite sepsis victims have long term changes to their immune systems (despite claiming that it doesn’t happen) and don’t recover:

[PLoS Pathog.](https://doi.org/10.1371/journal.ppat.1004976) 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 RA](#)¹, [Hastey CJ](#)¹, [Olsen KJ](#)², [Baumgarth N](#)³.

“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>

And

[MBio.](https://doi.org/10.1128/mBio.00100-16) 2016 Feb 12;7(1):e00100-16. doi: 10.1128/mBio.00100-16.

Longitudinal Transcriptome Analysis Reveals a Sustained Differential Gene Expression Signature in Patients Treated for Acute Lyme Disease.

[Bouquet J](#)¹, [Soloski MJ](#)², [Swei A](#)³, [Cheadle C](#)², [Federman S](#)¹, [Billaud JN](#)⁴, [Rebman AW](#)², [Kabre B](#)¹, [Halpert R](#)⁴, [Boorgula M](#)², [Aucott JN](#)⁵, [Chiu CY](#)⁶.

“Lyme disease is a tick-borne illness caused by the bacterium *Borrelia burgdorferi*, and approximately 10 to 20% of patients report persistent symptoms lasting months to years despite appropriate treatment with antibiotics. To gain insights into the molecular basis of acute Lyme disease and the ensuing development

of post-treatment symptoms, we conducted a longitudinal transcriptome study of 29 Lyme disease patients (and 13 matched controls) enrolled at the time of diagnosis and followed for up to 6 months. The differential gene expression signature of Lyme disease following the acute phase of infection persisted for at least 3 weeks and had fewer than 44% differentially expressed genes (DEGs) in common with other infectious or noninfectious syndromes. Early Lyme disease prior to antibiotic therapy was characterized by marked upregulation of Toll-like receptor signaling but lack of activation of the inflammatory T-cell apoptotic and B-cell developmental pathways seen in other acute infectious syndromes. Six months after completion of therapy, Lyme disease patients were found to have 31 to 60% of their pathways in common with three different immune-mediated chronic diseases. No differential gene expression signature was observed between Lyme disease patients with resolved illness to those with persistent symptoms at 6 months post-treatment. The identification of a sustained differential gene expression signature in Lyme disease suggests that a panel of selected human host-based biomarkers may address the need for sensitive clinical diagnostics during the "window period" of infection prior to the appearance of a detectable antibody response and may also inform the development of new therapeutic targets.

<https://www.ncbi.nlm.nih.gov/pubmed/26873097>

The devil is in the details about this Chiu/Aucott article:

"Importantly, Lyme disease patients did not show any changes in the calcium-dependent T-cell apoptosis pathway, in contrast to the marked downregulation observed in other bacterial and viral diseases (Fig. 4B). **In addition, an absence of significant DEGs linked to B-cell development in Lyme disease relative to other infections was observed. These findings suggest that Lyme disease may be associated with a smaller proportion of B and T cells in peripheral blood than other diseases. Interestingly, suppression of long-lived humoral responses has been observed in a mouse model of Borrelia infection (31).** The absence of DEGs corresponding to B-cell maturation may also potentially explain why prior infection with *B. burgdorferi* is associated with a serological response yet does not appear to confer immunity to reinfection. Certain alleles of HLA genes have been previously reported to be associated with serological responses to Lyme disease infection (32). **Here we found that upregulation of certain HLA genes (HLA-DQA1, HLA-DQB1, HLA-DRB5) is associated with seronegativity in Lyme disease and may thus constitute potential diagnostic biomarkers for seronegative patients.**

"Following the acute phase of infection, recent treatment trials among patients with EM have estimated that approximately 10 to 20% of patients treated for Lyme disease experience lingering symptoms that may progress to PTLDS, although the incidence can be as high as 50% (4). The pathogenetic mechanisms of PTLDS remain unknown, but autoantigens and/or central nervous system sensitization have been postulated to play a role (10, 33–35). In our study, the relatively large proportion of posttreatment Lyme disease patients with persistent symptoms of fatigue, widespread musculoskeletal pain, and/or cognitive dysfunction (13 [46.4%] of 28) can be potentially accounted for by more stringent enrollment criteria at the time of presentation (**requiring the presence of EM and concurrent influenza-like symptoms rather than EM alone**). This may have resulted in the selection of patients with more severe disease and thus with an increased likelihood of persistent symptoms (36). Of note, according to the

proposed formal case definition for PTLDS, which requires a functional decline in patients in addition to lingering symptoms, only 4 (14.3%) of our 28 patients met all of the criteria, within the range of the 10 to 20% frequency reported in the literature (4).

”Notably, Lyme disease at 6 months post-treatment (V5) had **60** and 31% of their predicted pathways overall in common with **SLE** and **RA**, respectively. Circulating immune complexes have been identified as features common to all three conditions (37, 38). Symptoms of fatigue and cognitive impairment occur in a variety of chronic syndromes, including SLE, CFS, and PTLDS. Although some pathways were common to Lyme disease at V5 and CFS, melatonin signaling, prominent in CFS, was not predicted to be involved in Lyme disease (Fig. 4D). As melatonin is a hormone that regulates the circadian rhythms of the sleep-wake cycle and thus is strongly linked to fatigue, the absence of increased melatonin signaling suggests that the fatigue in Lyme disease patients with persistent symptoms is related to a different mechanism. Overall, our results, showing only 18% of the DEGs and 34% of the pathways common to CFS and Lyme disease, are consistent with a proteomic study of cerebrospinal fluid that clearly discriminates between the two conditions (39).

In a news article about this report, this was said:

“Early Lyme disease prior to antibiotic therapy was characterized by marked upregulation of Toll-like receptor signaling but ***lack of activation of the inflammatory T-cell apoptotic and B-cell developmental pathways seen in other acute infectious syndromes,***” wrote the study’s authors. “Six months after completion of therapy, Lyme disease patients were found to have 31 to 60% of their pathways in common with three different immune-mediated chronic diseases. No differential gene expression signature was observed between Lyme disease patients with resolved illness to those with persistent symptoms at six months post-treatment.”

“Six months after treatment, 15 of the 29 patients in the study had fully recovered, while 13 had persistent symptoms, and one had dropped out.”

<http://www.genengnews.com/gen-news-highlights/lyme-disease-may-be-diagnosable-via-transcriptome-signature/81252365/>

The first thing Chiu, et al, do, in the actual journal report, is discount the fairly extensive evidence that Lyme, yes is associated with B cell changes and suppression of long lived immunity (Duray, Baumgarth, Dattwyler, even Steere in the early days; see below). Secondly, he shows again the association to the MS and Lupus HLAs that Klempner talked about, caught on tape in the summer of 2001 at South County Hospital, Rhode Island, and that means, essentially, “yes, probably EBV and/or the other herpesviruses are reactivated.” These HLA-DQs are associated with MS and Lupus (active EBV, et al). Thirdly, he falsely states that the sicker patients have fatigue with EM, whereas in most cases, no one notices the EM because they are not sick at the time it shows up.

The title of the Chiu/Aucott journal article basically says there are changes to the “transcriptome” (which likely does not apply to the condition we are talking about, immune blunting), and then says “not really” in the details. In the news article they claim that half the tick bite victims remain sick regardless of treatment. In the journal article they say 60% of the tick bite victims have the Lupus or reactivated EBV pathways in common, and then they go off pooh-pah-ing Chronic Fatigue victims as having a sleep disorder in the typically sinister way they discount people with post tick bite sepsis.

What can everyone take away from this article? These clowns discount what’s real and try to associate “Chronic Lyme” with the mental disorder (“nervousness,” let’s say) of Chronic Fatigue Syndrome, when they are both the same disease (post-sepsis) but acquired through different immune assaults, probably. He also tries to maintain the false view that “if you don’t have an autoimmune disease, you must have a mental illness.”

If you understand the real mechanisms of this illness, whereby the uptake of fungal antigens (TLR2/1 agonists or triacyl lipopeptides), causes “tolerance” or “lack of antigen processing or inhibition of antigen processing,” and even B cell immortalization, we don’t really think anyone should be concerned with “gene expression changes.” It is enough to know about fungal antigen tolerance and cross tolerance or what happens in post-septic shock from a tick bite.

OspA-ish antigens causing immune cell immortalization or inhibition of apoptosis:

[Cell Microbiol.](#) 2007 Jan;9(1):142-53. Epub 2006 Aug 2.

“The inhibitory effect of Mycoplasma fermentans on tumour necrosis factor (TNF)-alpha-induced apoptosis resides in the membrane lipoproteins.”

[Gerlic M1](#), [Horowitz J](#), [Farkash S](#), [Horowitz S](#).

“Mycoplasma have been shown to be involved in the alteration of several eukaryotic cell functions, such as cytokine production, gene expression and more. We have previously reported that infection of human myelomonocytic U937 cell line with live Mycoplasma fermentans (*M. fermentans*) **inhibited tumour necrosis factor (TNF-alpha)-induced apoptosis**. Mycoplasmal membrane lipoproteins are considered to be the most potent initiators of inflammatory reactions in mycoplasmal infections. The aim of this study was to clarify whether the inhibitory effect on TNFalpha-induced apoptosis is exerted by *M. fermentans* lipoproteins (LPMf). A significant reduction in TNFalpha-induced apoptosis was demonstrated by stimulation of U937 cells with *M. fermentans* total proteins, LPMf or MALP-2 (*M. fermentans* synthetic lipopeptide), but not with *M. fermentans* hydrophilic protein preparation (AqMf). To investigate the mechanism of *M. fermentans* antiapoptotic effect, the reduction of mitochondrial transmembrane potential ($\Delta\psi_m$) was measured. *M. fermentans* total proteins LPMf and MALP-2, but not AqMf, inhibited the reduction of $\Delta\psi_m$. In addition, *M. fermentans* total proteins LPMf and MALP-2, but not AqMf, downregulated the formation of active caspase-8. NF-kappaB was transactivated in cells treated with *M. fermentans* lipoproteins, and was essential for host cell survival, but not for the inhibition of TNFalpha-induced apoptosis by LPMf. Our results suggest that the inhibitory effect exerted by *M. fermentans* on TNFalpha-induced apoptosis in U937 cells is due to the membrane lipoproteins of these bacteria.

<https://www.ncbi.nlm.nih.gov/pubmed/16889623>

OspA-ish antigens causing immune cell immortalization or inhibition of apoptosis:

Curr Genomics. 2009 Aug;10(5):306-17. doi: 10.2174/138920209788920967.

Anti-apoptotic genes in the survival of monocytic cells during infection.

Busca A1, Saxena M, Kryworuchko M, Kumar A.

“Macrophages are cells of the immune system that protect organisms against invading pathogens by fulfilling critical roles in innate and adaptive immunity and inflammation. They originate from circulating monocytes and show a high degree of heterogeneity, which reflects the specialization of function given by different anatomical locations. Differentiation of monocytes towards a macrophage phenotype is also accompanied by an increase of resistance against various apoptotic stimuli, a required characteristic that allows macrophages to accomplish their function in a stressful environment. Apoptosis, a form of programmed cell death, is a tightly regulated process, needed to maintain homeostasis by balancing proliferation with cellular demise. Caspases, a family of cysteine proteases that are highly conserved in multicellular organisms, function as central regulators of apoptosis. FLIP (FLICE-inhibitory protein), anti-apoptotic members of the Bcl2 family and inhibitors of apoptosis (IAP) are the main three groups of anti-apoptotic genes that counteract caspase activation through both the extrinsic and intrinsic apoptotic pathways. **Modulation of the apoptotic machinery during viral and bacterial infections, as well as in various malignancies, is a well established mechanism that promotes the survival of affected cells.** The involvement of anti-apoptotic genes in the survival of monocytes/macrophages, either physiological or pathological, will be described in this review. How viral and bacterial infections that target cells of the monocytic lineage affect the expression of anti-apoptotic genes is important in understanding the pathological mechanisms that lead to manifested disease. The latest therapeutic approaches that target anti-apoptotic genes will also be discussed.

M.tb also exploits TLRs to induce anti-apoptotic genes that enhance cell survival and promote bacterial persistence [109]. Exploiting TLRs is not a mechanism unique to M.tb. As mentioned earlier, we have shown that TLR3, TLR4 and TLR9, when stimulated by their ligands PolyI:C, LPS and CpG DNA, respectively, protected monocytic cells from HIV-Vpr induced apoptosis by induction of NFκB and anti-apoptotic cIAP genes (unpublished data). ***Stimulation of TLR2, found in abundance at sites of M.tb infection, by components of M.tb cell wall, has been shown to protect human macrophages against apoptosis.*** THP1-derived macrophages when stimulated with 19kDa mycobacterial lipoprotein or mannosylated LAM were shown to induce resistance to apoptosis via activation of NFκB and subsequent induction of anti apoptotic cFLIP which inhibits death receptor-mediated apoptosis [25, 109].”

<http://www.ncbi.nlm.nih.gov/pubmed/20119528>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2729995/?tool=pubmed>

OspA-ish antigens causing immune cell immortalization or inhibition of apoptosis:

Eur J Immunol. 1997 Sep;27(9):2450-6.

Mycobacterium bovis Bacillus Calmette Guérin infection prevents apoptosis of resting human monocytes.

[Kremer L1](#), [Estaquier J](#), [Brandt E](#), [Ameisen JC](#), [Locht C](#).

“Apoptosis plays an essential role in the development and homeostasis of multicellular organisms. Some infectious agents interfere with this programmed cell death to their own benefit. Here, we show that infection of resting human monocytes with *Mycobacterium bovis* Bacillus Calmette Guérin (BCG) increases monocyte viability by preventing them from undergoing apoptosis. ***Heat-killed BCG also prevented apoptosis, indicating that replication of BCG is not required to prevent cell death.*** Analysis of BCG-infected monocytes revealed an up-regulation of the A1 mRNA, whereas the bcl-2 mRNA was not up-regulated. Interestingly, preinfection with BCG renders the cells resistant to interleukin (IL)-10-induced apoptosis which may be one of the mechanisms mycobacteria use to modulate immune responses. ***BCG infection was also accompanied by an impairment of the capacity of monocytes to secrete IL-10 and by an induction of the capacity to secrete tumor necrosis factor-alpha, two cytokines known to induce and prevent human monocyte apoptosis, respectively.*** Since it has been reported that apoptosis is involved in killing of intracellular mycobacteria, the ***prevention of apoptosis may represent a strategy for mycobacterial survival in the infected host.***”

<https://www.ncbi.nlm.nih.gov/pubmed/9341792>

Gary Wormser saying—while LYMERix was still on the market—that how sick you become, depends on how much OspA you got stuck with, either by ticks/spirochetes or syringe. You can’t make this up:

“The magnitude of modulation [immunosuppression – KMD] 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.” – Gary Wormser:

[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.

“We have previously demonstrated that proteins of *B. burgdorferi* are capable of modulating human cellular immune responses [7]. **Suppression of in vitro mitogen- or antigen-mediated proliferative responses of lymphocytes and reduced production of interleukin-2 (IL-2) from lymphocytes were demonstrated using protein extracts of *B. burgdorferi*.** These early studies were confirmed by a report of de Souza et al. [8], who observed that *B. burgdorferi* infection in mice resulted in impaired T and B cell proliferation to mitogens and reduced IL-2 and IL-4 production. The nature of the *B. burgdorferi* proteins responsible for suppression of cellular immunity has not been defined. In this study we examined the modulating activity of a recombinant outer surface protein A (OspA) vaccine preparation on cellular immune responses.”

<http://femsim.oxfordjournals.org/content/28/3/193.long>

Notice: “OspA blunts the immune response mechanism,” says Gary Wormser (above), 2000, while LYMERix was still on the market. You can see that the intention of the falsified Dearborn case definition and all the shenanigans by this Cabal regarding what Lyme is and does, revolves around the notion that Lyme *Borrelia* only cause an inflammatory disease (HLA-linked autoimmune), and that OspA would tolerize against that, as it presumably does in animal OspA vaccines. The reason for that baloney is because actually OspA is a fungal toxin that causes global immunosuppression in most humans. Ray Dattwyler said at the FDA meeting in 1998 on the Lyme vaccines that he only sees about one such “case” a year of this arthritis-only outcome.

1989, and 1992, Paul Duray on immortalized B cells in the spinal fluid of Lyme victims:

“The immature B cells can also be seen in the spinal fluid. These cells appear quite atypical – not unlike those of transformed of neoplastic lymphocytes.”

Duray in IDSA’s journal:

[Rev Infect Dis.](#) 1989 Sep-Oct;11 Suppl 6:S1487-93.

“Clinical pathologic correlations of Lyme disease.

“The multisystem effects caused by *Borrelia burgdorferi* in Lyme disease are multiple, varied, and unpredictable. In some patients, the full extent of the infection consists of a stage I acute systemic viral-like illness. Stage II primarily involves the cardiovascular system (myocarditis) and/or the central nervous system (CNS) (meningoencephalitis, polyradiculitis). More inflammatory cells are found in the heart and nervous system structures during this intermediate stage than are found in any tissues involved during stage I. Stage III is characterized by peripheral neuropathy and CNS disorders such as dementia or transverse myelitis and arthritis and synovitis of large joints such as the knee. Chronic Lyme disease is also associated with multiple and seemingly unrelated cutaneous manifestations such as acrodermatitis chronica atrophicans, sclerodermoid-like reactions, lichen sclerosus et atrophicus, subcuticular fibrous nodules, eosinophilic fasciitis-like lesions of the extremities, and, possibly, granuloma annulare. With care, spirochetes can be recovered or demonstrated by silver staining in most of the above lesions.

Spirochetes have yet to be seen in the tissues of autonomic ganglia or peripheral nerves.”

<https://www.ncbi.nlm.nih.gov/pubmed/2814170>

In the text of that article:

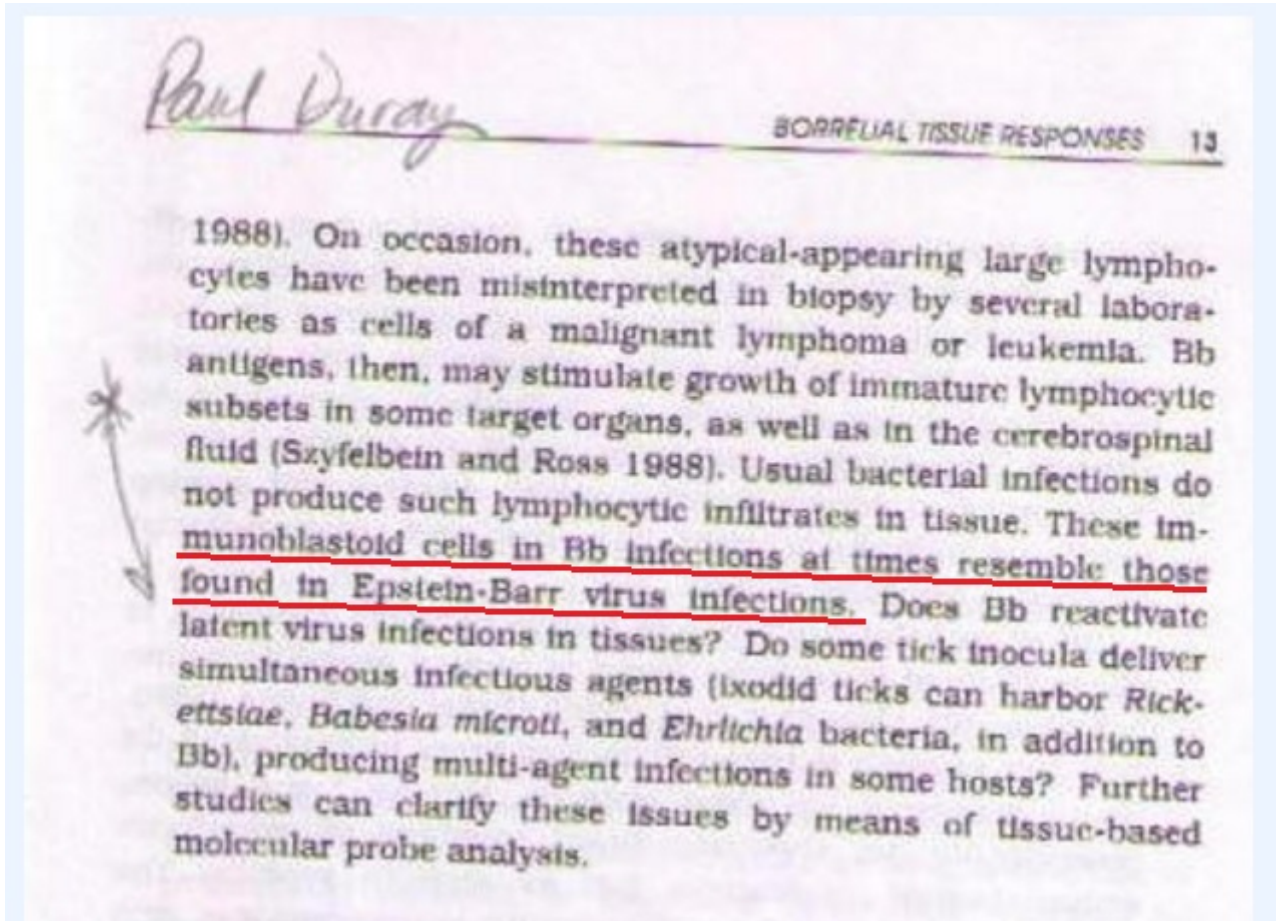
is usually no pleocytosis until the onset of stage II disease. Frank signs of meningeal irritation herald stage II illness, reflected by an increase in CSF lymphocytes and plasma cells and moderate increases in total protein in CSF [9, 16, 17]. Immature B cells can also be seen in the spinal fluid. These cells can appear quite atypical – not unlike those of transformed or neoplastic lymphocytes. Although it is known that spirochetes can be isolated from the spinal fluid, they are not recovered in all cases.

Duray in IDSA's journal, 1989.

The following is from the Steven Schutzer book, *Lyme Disease, Molecular and Immunological Approaches*:

<https://www.amazon.com/Lyme-Disease-Immunologic-Approaches-Communications/dp/0879693770>

Paul Duray says in that book, “**These immunoblastoid cells resemble those found in Epstein-Barr infections. Does Bb reactivate latent virus infections in tissues?**”



So, we know OspA inhibits apoptosis and we know Epstein-Barr virus inhibits apoptosis and is responsible for the Great Imitator outcomes of Lyme and Syphilis. And we know spirochetes go after lymph nodes and cause the “B cell maturation centers” to fail. And we know even from “Seronegative Lyme-Patients-are-the-Sickest” Dattwyler who in 1998 showed that Borrelial supernatant was responsible for the NK and other immune cell senescence:

[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](#).

"Effect of *B burgdorferi* Culture on Normal PBL

"..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)."

<https://www.ncbi.nlm.nih.gov/pubmed/3056196>

AKA: "TOLERANCE" or a septic shock-like result.

1922: Ancient history on how spirochetes target the lymph nodes

J Exp Med. 1922 Jan 1;35(1):39-62.

A STUDY OF THE RELATION OF TREPONEMA PALLIDUM TO LYMPHOID TISSUES IN EXPERIMENTAL SYPHILIS.

[Pearce L1](#), [Brown WH](#).

A widespread dissemination of *Treponema pallidum* from a local focus of inoculation in the rabbit constantly occurs by way of the lymphatics. Spirochetes were regularly recovered from the satellite lymph nodes by animal inoculation after scrotal inoculation; they were present as early as 2 days, when no specific primary reaction was detected, and at later periods of from 5 to 61 days after inoculation. Other superficial nodes at remote sites such as the popliteals and with no syphilitic lesions in the drainage area have also been shown to harbor active organisms. Although spirochetes were found in relatively few of the lymph node emulsions, the orchitis resulting from their injection was of a rapidly progressive type with an incubation period but slightly longer than that produced by a testicular or skin nodule emulsion rich in spirochetes. It has further been shown that a syphilitic infection is sufficiently established in the rabbit body within 48 hours after scrotal inoculation so that the primary lesion is no longer essential for its maintenance. Active treponemata survive in the popliteal lymph nodes for long periods of time and have been regularly recovered from them in cases of true latency. **The lymph nodes, therefore, function as reservoirs of the organisms. The ability to recover the spirochetes from lymphoid tissue through successive generations is seen in the serial passage of lymph node emulsion to testicle during an 18 months period.** The persistence of spirochetes in lymphoid tissue irrespective of the presence or absence of syphilitic lesions is a characteristic and fundamental feature of syphilis of the rabbit. The existence of infection, therefore, may be demonstrated at any time by the recovery of spirochetes from the popliteal lymph nodes by animal inoculation. This fact is of great practical importance in the therapy of the infection and may be profitably utilized in determining the ultimate effect of a therapeutic agent. These experiments demonstrate that the disease is not confined to the site of local inoculation but that lymphogenous dissemination of treponemata regularly takes place, and that during the course of this process organisms become localized in the lymph nodes and exist there indefinitely irrespective of the occurrence of manifestations of disease. The intimate relation of *Treponema pallidum* to lymphoid tissue is an essential concept of syphilis of the rabbit, and from this point of view, the infection is primarily one of lymphoid tissue.

<https://www.ncbi.nlm.nih.gov/pubmed/19868586>

J Exp Med. 1925 Jun 30;42(1):33-42.

STUDIES IN EXPERIMENTAL SYPHILIS : IV. THE SURVIVAL OF TREPONEMA PALLIDUM IN THE INTERNAL ORGANS OF TREATED AND UNTREATED RABBITS.

[Chesney AM1](#), [Kemp JE](#); [Assistance of Allan K. Poole, M.D.](#)

“Simultaneous transfers to the testes of normal rabbits of circulating blood, heart muscle, liver, brain, spleen and bone marrow (mixed), inoculated testicle, and popliteal lymph nodes from a series of untreated syphilitic rabbits, demonstrated the persistence of the original infection uniformly in the lymph nodes and less regularly in the liver, mixed spleen and bone marrow, and testis originally inoculated. In one instance the circulating blood was found to be infectious. Transfer of similar tissues from syphilitic rabbits treated with arsphenamine late in the course of the disease failed to disclose syphilitic infection of any of these tissues. In one animal, in which keratitis developed both before and after treatment, the blood, internal organs, and lymph nodes were found to be non-infectious in spite of the fact that the cornea was shown to be the site of a syphilitic inflammation. Transfer of lymph nodes or internal organs of treated syphilitic rabbits is probably the best method of evaluating an antisiphilitic agent, but it must be supplemented by careful observation of treated animals over an appreciable interval of time following treatment. The results of this study support the idea that failure to reinoculate a treated syphilitic animal does not necessarily mean the existence of the first infection but might be interpreted as indicating the presence of an acquired resistance which persists in rabbits in which no trace of the first infection can be demonstrated.”

<https://www.ncbi.nlm.nih.gov/pubmed/19869032>

Spirochetes have long been known to hang out in lymph nodes, cause antibody-negative disease, be incurable and produce a variety show of outcomes. It wasn't until we found out what OspA was exactly were we able to see that that alone, injected, could cause the same “these look like Epstein-Barr transformed lymphocytes” and “oh, spirochetes and Epstein-Barr both hang out in the lymph nodes” outcome, solving the 500 year old mystery of why ancient dinosaur phyla like Spirochaeta are responsible for dementia in white human males with “MD” after their names, where those demented “MD” males don't deliberately infect non-Caucasians with these organisms to find out why spirochetal infections in African humans do not cause the dementia they do in Caucasians (Tuskegee “Bad Blood”).

[Sex Transm Dis](#). 2014 Jul;41(7):440-6. doi: 10.1097/OLQ.000000000000149.

Toll-like receptor polymorphisms are associated with increased neurosyphilis risk.

[Marra CM1](#), [Sahi SK](#), [Tantalo LC](#), [Ho EL](#), [Dunaway SB](#), [Jones T](#), [Hawn TR](#).

"Clinicians in the early 20th century posited that race influenced susceptibility to neurosyphilis, citing a decreased risk in African Americans compared to Caucasians (7). Subsequent work suggested a genetic basis for such differences, with an increased risk of syphilitic dementia, but not other forms of neurosyphilis, in patients with certain HLA types (8) that differed in African Americans compared to Caucasians (9). While more recent reports suggest that there may be genetic contributions to syphilis susceptibility (10-13), to the best of our knowledge there have been no recent investigations of genetic susceptibility to neurosyphilis."

<https://www.ncbi.nlm.nih.gov/pubmed/24922103> <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4414322/>

Meanwhile, importantly, everyone will recall that the Cabal (published in IDSA's journal) themselves dubbed Lyme a New Great Imitator because it causes Lupus, MS, [ALS](#), stroke, cancer and so forth. Yale even had a “Lyme and Lupus Clinic,” now called “L2 (for Lyme and Lupus) Diagnostics.”

[Rev Infect Dis.](#) 1989 Sep-Oct;11 Suppl 6:S1482-6.

Neurologic manifestations of Lyme disease, the new "great imitator".

<https://www.ncbi.nlm.nih.gov/pubmed/2682960>

(Note that "Reviews of Infectious Diseases" was IDSA's own journal.)

Reviews of Infectious Diseases

Volume 11 • Supplement 6 • September–October 1989

And yet now Yale says Lupus is mostly like caused by Epstein-Barr, so what is happening? Right, Lyme activates Epstein-Barr via immunosuppression because not only do they both live in the lymph nodes (spirochetes and EBV), but injecting people with OspA alone causes this same immunosuppression and multi-system disease outcome:

This is Yale:

[J Immunol.](#) 2004 Jan 15;172(2):1287-94.

Defective control of latent Epstein-Barr virus infection in systemic lupus erythematosus.

[Kang H, 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. In the current report we assessed immune responsiveness to EBV in patients with SLE and healthy controls, determining virus-specific T cell responses and EBV viral loads using whole blood recall assays, HLA-A2 tetramers, and real-time quantitative PCR. 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. By contrast, the frequency of EBV-specific CD69+ CD8+ T cells producing IFN-gamma in patients with SLE appeared lower than that in healthy controls, although this difference was not statistically significant. These findings suggest a role for CD4+ T cells in controlling, and a possible defect in CD8+ T cells in regulating, increased viral loads in lupus. These ideas were supported by correlations between viral loads and EBV-specific T cell responses in lupus patients. EBV viral loads were inversely correlated with the frequency of EBV-specific CD69+ CD4+ T cells producing IFN-gamma and were positively correlated with the frequencies of CD69+ CD8+ T cells producing IFN-gamma and with EBV-specific, HLA-A2 tetramer-positive CD8+ T cells. **These results demonstrate that patients with SLE have defective**

control of latent EBV infection that probably stems from altered T cell responses against EBV.”

<https://www.ncbi.nlm.nih.gov/pubmed/14707107>

Amazingly stupid people injected the very thing that causes complete ruination of the immune system, from organisms known for almost 100 years to target and survive in lymph nodes (clue, “immune system”), and said that thing was a “vaccine” against “a disease that does not exist but is just hypochondria, drug-seeking, and drama queen-itis.” You can’t make this up.

Some Normal People recently objecting to the idea that Borreliae is more than one genus, which is what the criminal CDC cabal would like to (falsely) claim; look how many signers!!

Int J Syst Evol Microbiol. 2016 Dec 8. doi: 10.1099/ijsem.0.001717. [Epub ahead of print]

There is inadequate evidence to support the division of the genus Borrelia.

[Margos G1](#), [Marosevic D2](#), [Cutler S3](#), [Derdakova M4](#), [Diuk-Wasser M5](#), [Emler S6](#), [Fish D7](#), [Gray J8](#), [Hunfeld KP9](#), [Jaulhac B10](#), [Kahl O11](#), [Kovalev S12](#), [Kraiczky P13](#), [Lane RS14](#), [Lienhard R15](#), [Lindgren PE16](#), [Ogden N17](#), [Ornstein K18](#), [Rupprecht T19](#), [Schwartz I20](#), [Sing A21](#), [Straubinger RK22](#), [Strle F23](#), [Voordouw M24](#), [Rizzoli A25](#), [Stevenson B26](#), [Fingerle V27](#).

“As for the clinical symptoms caused by Borrelia species, the symptomology that differentiates RF spirochaetes from the LB group of spirochaetes has been blurred by recent case descriptions. For example, a patient with clinical symptoms resembling those of Lyme neuroborreliosis was diagnosed as being infected with the RF group species *B. miyamotoi* (Boden et al., 2016). Interestingly, infection with the recently described genospecies of the *B. burgdorferi* s.l. complex, *B. mayonii*, produced high spirochaetal blood densities, akin to that seen following infection with species of the RF group (Pritt et al., 2016).

”Thus, splitting the genus does not provide any assistance as far as clinical evaluation is concerned. It does not help end user communities including those in clinical medical practice, public health or those studying the ecology of the bacteria. Collectively, in view of the inadequate genetic evidence supporting the genus split and the biological features shared between RF and LB group spirochaetes, at present we strongly oppose the proposed division of the genus Borrelia. This division complicates an already complicated situation which will serve only to lead to further confusion among scientists, clinicians, public health authorities and the general public. Taken together, we believe that such a change is inadvisable based on currently available biological and clinical evidence, and therefor respectfully request that it be repealed.”

<https://www.ncbi.nlm.nih.gov/pubmed/27930271>

http://ijs.microbiologyresearch.org/deliver/fulltext/ijsem/ijsem_pap.001717.zip/ijsem.0.001717.pdf?itemId=/content/journal/ijsem/10.1099/ijsem.0.001717.v1&mimeType=pdf&isFastTrackArticle=true

And here is the Cabal’s answer:

Int J Syst Evol Microbiol. 2017 Jan 27. doi: 10.1099/ijsem.0.001815. [Epub ahead of print]

Division of the genus Borrelia into two genera (corresponding to Lyme disease and relapsing fever groups) reflects their genetic and phenotypic distinctiveness and will lead to a better understanding of

these two groups of microbes (Margos et al. (2016) There is inadequate evidence to support the division of the genus Borrelia. Int. J. Syst. Evol. Microbiol. doi: 10.1099/ijsem.0.001717).
[Barbour AG](#)¹, [Adeolu M](#)², [Gupta RS](#)³.

“This rebuttal Letter responds to a Letter in the IJSEM by Margos et al. challenging division of the genus *Borrelia* into two genera. We discuss here point-by-point the issues raised by Margos et al. and show that much of their criticism is unfounded and in several cases based on misreading of the presented results. We summarize here the extensive evidence based on genomic, genetic and phenotypic properties showing that the members of the family Borreliaceae (containing mainly the genus *Borrelia*) comprises two distinct and cohesive groups of microbes, differing in diseases they cause and other phenotypes. Prior to the proposed division, *Borrelia* spp. causing Lyme disease (LD) were already functionally treated as a distinct group, referred to as “*B. burgdorferi sensu lato*” to distinguish them from the other cluster of *Borrelia* spp. which includes all known species causing relapsing fever (RF). With the more explicit division of Borreliaceae species into two genus level groups, which are distinguishable from each other based on numerous unique genetic and molecular characteristics, the attention can now be focused on the biological significance of different molecular characteristics differentiating the two groups. The clear distinction of the LD and the RF groups of microbes based on numerous highly reliable markers, which are expected to be present even in uncharacterized members of these two groups, should aid in the improved diagnosis as well treatment of both these diseases, which is hindered by the conflation of a common name for agents causing two different types of diseases.”

<https://www.ncbi.nlm.nih.gov/pubmed/28141502>

What would be the advantage of claiming the Lyme *Borrelia* are a separate genus?

Right: “To maintain the lie that Dearborn was real and that “Lyme” is only a bad knee. This response was another attempt to manufacture a Get Out of Jail Free card. It’s all about maintaining the PRETENSE (a legal word meaning FRAUD) that Dearborn was real, and not a crime scene.

Lyme *Borrelia* do not only cause “bad knees.” They’re spirochetes and do what spirochetes do, which is shed fungal antigens and go right to the lymph nodes where they ruin the immune system. They do not Get Out Of Jail Free or Pass Go, Right to The Knees and Collect 200 Dollars. They do not participate in semantics games. They are found in Alzheimer’s brains. They’re not nanobots, and they’re not even regular bacteria, with lipopolysaccharide as a main membrane component (LPS). When they show up in a courtroom, such as a “[Railroad Case](#),” they go back to being a regular Great Imitator, instead of a bad knee. When they show up at the FDA, Glaxo-SmithKline, *themselves*, make fun of Allen Steere and say, basically, “Steere’s is a crazy idea; if spirochetal OspA antibodies cross-attack a fragment of the HLA molecule, they would be found everywhere in the body and not just in the knees...” “ http://www.fda.gov/ohrms/dockets/ac/01/slides/3680s2_02_lobet.pdf

As much as delusional persons would like to *will* Spirochetes into being subservient little thugs in their RICO club, well, let’s say you really can’t train them. They don’t even *live in colonies in vivo*, which is a bogus claim of another wrong-headed organization, ILADS.org...

The following, Primer Shell Game charge sheet is more formal training in Spirochete biology and taxonomy.

BACKGROUND: The essence of these criminal charge sheets is that the Cabal makes false claims based on research fraud, and our job (apparently), is to show *point-by-point, crime-by-crime*, research fraud and false claims that result in tremendous human (and even animal) harm, and billions in lost research-dollar-lives in related diseases such as cancer, MS, RA, and Lupus, not to mention the harm to USA's scientific reputation. "MDs" apparently have no responsibility to know what they're talking about. There is no accountability system for them in the United States. USA's medical schools do not require a science background.

These are the research-fraud "Guidelines," the signers of which will be prosecuted among others (CDC):

[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.

[Wormser GP1](#), [Dattwyler RJ](#), [Shapiro ED](#), [Halperin JJ](#), [Steere AC](#), [Klempner MS](#), [Krause PJ](#), [Bakken JS](#), [Strle F](#), [Stanek G](#), [Bockenstedt L](#), [Fish D](#), [Dumler JS](#), [Nadelman RB](#).

<http://www.ncbi.nlm.nih.gov/pubmed/17029130>

<http://www.cid.oxfordjournals.org/cgi/pmidlookup?view=long&pmid=17029130>

The Cabal will probably attempt to say the data we present in these criminal charge sheets for the USDOJ is taken out of context, but you can go to all the PubMed links in all these SASH/TruthCures criminal charge sheets and find how many *other scientists* referenced their work when this gang was telling the truth. This CDC/ALDF organized crime gang that hijacked IDSociety.org certainly could not have been mistaken on *EVERYTHING*, either, if that is what they will try to claim.

START by understanding the DNA Shell Game, by finding out what DNA and RNA primers are:

http://en.wikipedia.org/wiki/Primer_%28molecular_biology%29 and

http://en.wikipedia.org/wiki/16S_ribosomal_RNA

Primers are like a starting DNA or RNA sequence to look for a match in your sample. If you start with the wrong primer probes, you won't find what are looking for. When looking for spirochetes in humans, particularly when trying to claim "NO LYME," either in EM rashes in Missouri, or after "treatment," the

Cabal either uses the wrong primers (they prefer to use OspA primers in particular, when trying to *not* find Lyme), or using inadequate primers such that only one or 2 species are probed for in humans, when there are probably a hundred *formal* different types of borrelia.

It would be therefore reasonable to either sequence the DNA and not rely on probes, or use several different probes for the commonest borrelia in the region, be they *hermsii*, and subdivisions thereof from the other relapsing fever, or several from the new, *burgdorferi* clade including some of the newer ones that have evolved from it. Recently, we learned of a new Mass Spec--ToF-PCR method endorsed by the CDC and Infectious Diseases Society of America to detect central nervous system (CNS) infections:

"Unmet diagnostic needs in infectious disease"

"...A number of new diagnostic technologies for ID are rapidly emerging: e.g., broad-range PCR, next-generation sequencing, and matrix-assisted laser desorption/ionization time of flight mass spectrometry.***

http://ein.idsociety.org/media/publications/papers/2014/Blaschke_DMID_14_Unmet_Diagnostic_Needs.pdf

And

[J Clin Microbiol](#). 2014 Jan;52(1):212-7. doi: 10.1128/JCM.02270-13. Epub 2013 Nov 6.

Virological diagnosis of central nervous system infections by use of PCR coupled with mass spectrometry analysis of cerebrospinal fluid samples.

[Lêvêque N1](#), [Legoff J](#), [Mengelle C](#), [Mercier-Delarue S](#), [N'guyen Y](#), [Renois F](#), [Tissier F](#), [Simon F](#), [Izopet J](#), [Andréoletti L](#).

"Viruses are the leading cause of central nervous system (CNS) infections, ahead of bacteria, parasites, and fungal agents. A rapid and comprehensive virologic diagnostic testing method is needed to improve the therapeutic management of hospitalized pediatric or adult patients. In this study, we assessed the clinical performance of PCR amplification coupled with electrospray ionization-time of flight mass spectrometry analysis (PCR-MS) for the diagnosis of viral CNS infections. Three hundred twenty-seven cerebrospinal fluid (CSF) samples prospectively tested by routine PCR assays between 2004 and 2012 in two university hospital centers (Toulouse and Reims, France) were retrospectively analyzed by PCR-MS analysis using primers targeted to adenovirus, human herpesviruses 1 to 8 (HHV-1 to -8), polyomaviruses BK and JC, parvovirus B19, and enteroviruses (EV). PCR-MS detected single or multiple virus infections in 190 (83%) of the 229 samples that tested positive by routine PCR analysis and in 10 (10.2%) of the 98 samples that tested negative. The PCR-MS results correlated well with herpes simplex virus 1 (HSV-1), varicella-zoster virus (VZV), and EV detection by routine PCR assays (kappa values [95% confidence intervals], 0.80 [0.69 to 0.92], 0.85 [0.71 to 0.98], and 0.84 [0.78 to 0.90], respectively), whereas a weak correlation was observed with Epstein-Barr virus (EBV) (0.34 [0.10 to 0.58]). Twenty-six coinfections and 16 instances of uncommon neurotropic viruses (HHV-7 [n = 13], parvovirus B19 [n = 2], and adenovirus [n = 1]) were identified by the PCR-MS analysis, whereas only 4 coinfections had been prospectively evidenced using routine PCR assays (P < 0.01). In conclusion, our results demonstrated that PCR-MS analysis is a valuable tool to identify common neurotropic viruses in CSF (with, however, limitations that were identified regarding EBV and EV detection) and may be of major interest in better understanding the clinical impact of multiple or neglected viral neurological infections."

<http://www.ncbi.nlm.nih.gov/pubmed/24197874>

We should be clear about this Primers Shell Game aspect of the criminal behavior of the Cabal: The Cabal deliberately uses the wrong DNA to assess for the presence of spirochetes patients, yet use the correct DNA and RNA analyses when looking for spirochetes to patent. This bait-and-switch game could be called **clinical violence** or medical violence because the victims are left not only sick, but declared mentally ill, are slandered against, or libeled against, are denied income and disability benefits, as well as suffer social ostracism. How different is this abuse than that suffered by the tortured African American community all these centuries? It's a **“Deprivation of Rights via Color of Law” criminal charge**, where the Govt employees deny you your rights. In this case, it is the CDC staff involved in these crimes who can be charged with “Color of Law” (Alan Barbour, Barbara Johnson, etc.).

These victim-patients are deprived of their humanity, as well as functionality. They're tossed aside, sick, demoralized, ostracized, and despised, yet they suffer a complex of several exhausting, neurologic diseases at the same time. While the CDC now claims that Lyme is 10 times underreported - meaning the new annual cases number around 300,000 rather than 30,000 because the falsified case definition misses 85% of the cases as shown in the other criminal charge sheets -, they of course never mention **it is only 15% reportable due to the fraud of Dearborn**. That is a lot of human cost and disability for which Uncle Sam will have to pay. Somehow a gang of low-lives was put in charge so they could potentially capitalize on this new vaccines and test kits racket, the emerging, global pollution-related vector-borne-diseases. The ALDF's was a 50 year to roll-out plan for every new type of disease: rickettsia, babesia, borrelia, any new viruses they find, etc. Their model was to in each instance, invent a vaccine, and *then* the falsify the serological description of the disease. Whoever did not meet their Vaccine First disease definition was to be trashed. It's the same violence seen in any mob-related activity. **“You do it our way or we'll break your legs, we'll kill you or ruin your family, but you will be taken out. Silenced.”**

To continue your background training in the Primers Shell Game, go to the National Library of Medicine and search for Borrelia in the Taxonomy database. Click on the word Borrelia until you come to the genetics page and find that flagellin – and not plasmid DNA (which is varied, added to- and subtracted from via bacteriophages, as well as variable within each plasmid) - is the species distinguisher.

<http://www.ncbi.nlm.nih.gov/Taxonomy/Browser/wwwtax.cgi?mode=Info&id=138&lvl=3&lin=f&keep=1&srchmode=1&unlock>

Use Google Images to discover the basic structure of a spirochete; see the internal flagellar bundle that facilitates movement by expanding and contracting like a muscle; the organism borrows. There may be no help from “physicians” in this campaign, but that really doesn't even mean anything any more. The victims themselves have carried this campaign all these 20+ years and in the end, we'll probably welcome robot-doctor kiosks in the malls and at Walmart, perhaps with a nurse standing by to take blood and write the orders for the radioimaging. ‘No need to overpay a middle-man for their incompetence. You'll at the end of this campaign be convinced no one needs a man with perverted, unscientific ideas about disease and medicine getting in the way of the machines. “Doctors” had their shot. They chose Kool-Aid and the

age-old cliquish, clannish default position of looking down their noses and blaming their victims. “Chose,” people, and that’s a spiritually dangerous thing from a bunch of First Do No Harm, oath-takers. It was dangerous also, because BS is never not a boomeranger. We saw that loud and clear with the 911 stunt and then the subsequent quintuple financial and military superpower of Iran, Russia, Brazil, China, and South Africa (BRICS), not to mention ISIS and losing Syria and the Middle East oil wars.

I. Phage-vectored plasmids are variable DNA, not to be used for probes in human disease

PHYLOGENY means how the organism evolved and how it is genetically related to other organisms, for example, such as dogs evolving from wolves and being related to bears. *B. burgdorferi* is genetically closest to *B. anserina*, an African Bird Borreliosis. Borreliae undergo constant variation in their plasmid DNA, and the plasmid DNA is bacteriophage-vectored and changes all the time, also. The plasmid content is variable inside the spirochetes, and variable phage-vectored DNA for the plasmids come from other organisms to an important extent.

The genus, Borreliae, is the name for the relapsing fever organisms, and the nature of the relapse is antigenic variation. Therefore you cannot use any DNA from borrelia’s plasmids – which is where the variable surface antigens are ordered manufactured and remanufactured – to assess for the presence of spirochetes. No researchers outside the United States *EVER* use plasmid DNA to assess for spirochetes. They only use species-specific genes like 5-, 16- and 23-S RNA or flagellin. When CDC officers like Alan Barbour or Yale staff patent borrelia species, they patent the specific flagellin that differentiates that particular bug from the other borrelia.

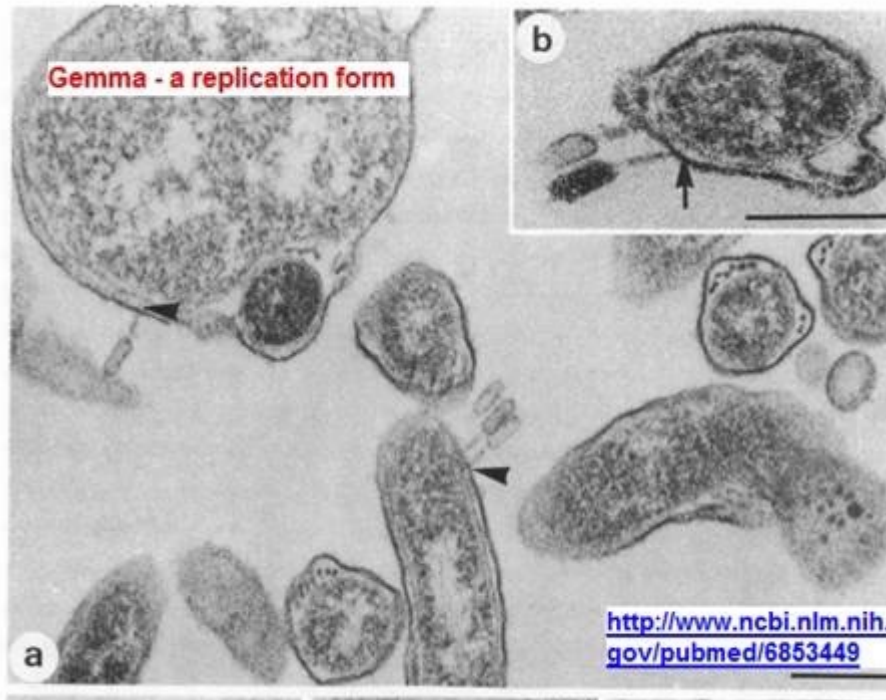
Plasmid content changes all the time *within* individual spirochetes and this is known as **antigenic variation**. CDC officer Alan Barbour is an expert on how this plasmid content changes and produces the well-known antigenic variation in spirochetes. Oscar Felsenfeld once said there was no point in differentiating Borreliae species since they were so variable and changing to constantly due to this phage-vectored-, variable plasmid content. *Just call them all Borreliae, the genus*, is what Felsenfeld recommended. It’s best if you see this with your own eyes:

CDC’s Barbour and NIH’s Burgdorfer on bacteriophages transferring plasmids (the arrows point to the phages or viruses of bacteria):

[J Bacteriol.](#) 1983 Jun;154(3):1436-9.

Bacteriophage in the Ixodes dammini spirochete, etiological agent of Lyme disease.

[Hayes SF](#), [Burgdorfer W](#), [Barbour AG](#).



<http://www.ncbi.nlm.nih.gov/pubmed/6853449>
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC217620/pdf/jbacter00247-0414.pdf>

Plasmids change all the time, are bacteriophage-vectored and responsible for intra-Kingdom gene transfer. The antigens *produced* by the plasmid change all the time. So, there is only one species-determinant, flagellin. See also Casjens on this topic in the PubMed literature.

Spirochetes from human brains were shown to undergo antigenic variation (Pachner, below), but we can assume they're all weakened over time from dropping plasmids. Spirochetes do all their damage early in the disease by shedding these varying, fungal antigens, as CDC officer Alan Barbour says in (the probably mis-titled) the next article, and causing what the NIH prefers to call Post-Sepsis Syndrome:

“Researchers Finding Rewarding Careers As Software Entrepreneurs”

"It's using some sort of stealth-bomber-type mechanism," he says. Or, using another diversionary tactic called blebbing, the spirochete can pinch off bits of its membrane in order to release its surface proteins.

Explains Barbour: "It's like a bacterial Star Wars defense program," in which released surface proteins might intercept incoming host antibodies, keeping the spirochete safe from immunological attack."

<http://www.the-scientist.com/?articles.view/articleNo/17985/title/Researchers-Finding-Rewarding-Careers-As-Software-Entrepreneurs/>

They, the shed fungal antigens like OspA, turn off the immune response. It's the secondary infections, the reactivated latent infections (herpes) or the opportunistics that mainly cause the majority of disease signs. A better and more acceptable description of Lyme is that it is AIDS-like or Post Sepsis Syndrome.

Says CDC officer Alan Barbour about antigenic variation even from a single spirochete (and Section IV, below, page 25-26):

VMP-like sequences of pathogenic Borrelia

”2.1 Methods of Treatment

”An important aspect of the invention is the recognition that *Borrelia* VMP-like sequences recombine at the vls site, with the result that antigenic variation is virtually limitless. **Multiclonal populations therefore can exist in an infected patient so that immunological defenses are severely tested if not totally overwhelmed.** Thus there is now the opportunity to develop more effective combinations of immunogens for protection against *Borrelia* infections or as preventive inoculations such as in the form of cocktails of multiple antigenic variants based on a base series of combinatorial VMP-like antigens. “

<http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetacgi%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6,719,983.PN.&OS=PN/6,719,983&RS=PN/6,719,983>

The Vmps are little different from the Osps. They call Osps from the non-Lyme relapsing fever organisms, VMPs or variable major proteins. There is no data on whether or not the VMPs are triacyl lipopeptides; we just know spirochetes and *Mycoplasma*/*Mycobacteria* (and *Brucella*) are lumped together as producers of these TLR2/1-agonists. The take home point is that Osps/Vmps undergo constant variation such as to adapt to new hosts and tissues, within themselves and among the genus, *Borrelia*. **They can’t be used to assess human cases of Lyme. Non-variable DNA/RNA should be used.** See more at:

[J Bacteriol.](#) 2006 Jun; 188(12): 4522–4530.

Crystal Structure of Neurotropism-Associated Variable Surface Protein 1 (Vsp1) of Borrelia turicatae†

Catherine L. Lawson,^{1,*} Brian H. Yung,¹ Alan G. Barbour,² and Wolfram R. Zückert^{2,3,*}

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1482977/>

II. *Borrelia* Acquiring Sticky OspA, and OspA Sticking to Itself (falsified vaccines reporting, blot smudging, Korean Chemists on OspA being sticky and clumping)

We’ve wondered how Lyme spirochetes “took” to hard-bodied, *Ixodes* ticks, as they were originally found in the guts of soft-bodied *Ornithodoros* ticks. OspA or Pam3ys is a ligand for chitinous or collagenous tissue. OspA/Pam3Cys also binds plasminogen and maintains the plasminogen as biologically active even when OspA is as a free molecule (Philipp, Tulane). *Mycoplasma*, *Brucella* and Lyme spirochetes all cause arthritis, so one may wonder if these molecules just stick to joint tissue? And do they, as bearers of biologically active plasminogen, aid the spirochetes in penetrating the hard bodies of hard bodied ticks?

We know these Pam3Cys molecules tick to each other and to intracellular components, gumming up the immunity works as seen in other charge sheets for the U. S. Justice Department. We also suspect that the fact that OspA sticks to itself is a probable reason the LYMERix vaccines had unreadable Western Blots as well as is the reason for the large number of strokes, cancer, and other “vascular events” resulting from LYMERix or OspA vaccination. Next, Yale’s Robert T. Schoen on LYMERix damage, the Phase IV data (strokes, cancer, “vascular events”):

[Clin Ther.](#) 2003 Jan;25(1):210-24.

An open-label, nonrandomized, single-center, prospective extension, clinical trial of booster dose schedules to assess the safety profile and immunogenicity of recombinant outer-surface protein A (OspA) Lyme disease vaccine.

Schoen RT1, Deshefy-Longhi T, Van-Hoecke C, Buscarino C, Fikrig E. or ([OspA_4.htm](#))
<https://www.ncbi.nlm.nih.gov/pubmed/12637121>

Korean Chemistry Journal on the Structure of OspA/Pam3Cys

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

”We are currently using mass spectral techniques to characterize the amino acid sequence of the Pam3Cys peptides found in the envelope glycoproteins of HIV-1 and the Simian Immunodeficiency Virus (SIV) (17). Conventional FAB-MS analysis using standard matrices such as glycerol and nitrobenzyl alcohol is not particularly effective for these molecules, largely due to their tendency to aggregate.”

Introduction

Synthetic peptides are prepared to contain an N-palmitoyl moiety at the N-terminal residue of the peptide which is a modified cysteine, containing a S-[2,3-bis(acyloxy)-(2-R,S)-propyl] moiety. When this residue is placed at the N-terminus of various synthetic peptides, it has been found to be potent immunoadjuvant which enhances both IgM and IgG antibody responses to the attached peptide.¹⁻⁶ Synthetic analogues of these compounds include those bearing palmitoyl groups (Pam₃Cys) as shown in Figure 1. These synthetic peptides have significant advantages, since the addition of other adjuvants are not required, and most importantly, the epitope can be specifically defined.

It is critical, however, that these peptides should be structurally characterized prior to their use in immunological studies.⁷⁻⁸ This is most important, since the synthesis involves several steps where the peptide is exposed to conditions that can provide amino acid side chain modification and/or deacylation. And, the lipoidal nature of the peptides make

them extremely difficult to be purified and analyzed. Reverse phase HPLC can lead to irreversible adsorption to the bonded phase. Although we have found that the N-methyl-2-pyrrolidone (NMP) is useful for solubilization and isocratic elution for some of these lipopeptides, it is not successful in all cases for HPLC purification.⁹ The peak broadening resulting from the inherent self-aggregation of these compounds may, in even favorable cases, obscure contaminating peaks. Thus, amino acid analysis is not adequate to fully characterize these peptides prior to their use in immunological studies. Further, because the N-terminal is blocked, traditional Edman sequencing cannot be employed to determine the proper sequence of synthetic peptide.

We are currently using several mass spectral techniques to characterize the amino acid sequences of the Pam₃Cys peptides found in the envelop glycoproteins of HIV-1 and the Simian Immunodeficiency Virus (SIV).¹⁷ Conventional FAB-MS analysis using standard matrices, such as glycerol and nitrobenzyl alcohol, is not particularly effective for these molecules, largely due to their tendency to aggregate. Here,

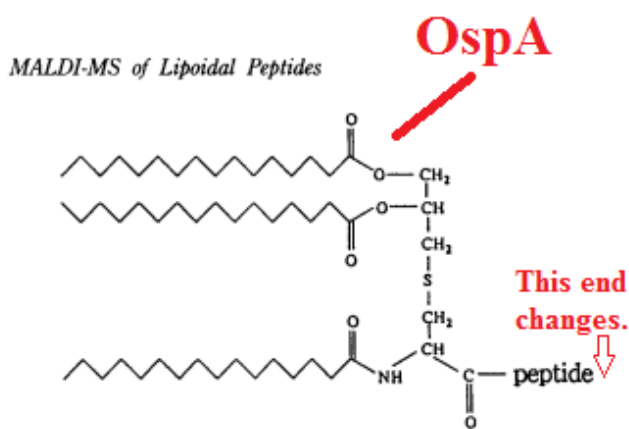


Figure 1. Structure of N-palmitoyl-S-[2,3-bis(palmitoyloxy)-propyl]-cysteinyl (Pam₃Cys) peptide, showing the modified cysteine residue with an N-palmitoyl ester and a dipalmitoylglycerol moiety connected via a thioether linkage. This amino acid is placed at the N-terminus of the synthetic peptide.

Bull. Korean Chem. Soc. 1996, Vol. 17, No. 11 1037

Ltd., Akishima, Japan) in positive mode. The resolution was set at either 1,500 or 3,000. 3-Nitrobenzyl alcohol (NBA) was used as the matrix, but detectable signals were obtained only when the lipoidal peptides were solubilized in N-methyl-2-pyrrolidone before MS analysis. Samples were prepared for FAB-MS analysis by mixing 1 μ l of the peptide solution (1-2 nmol) with 1 μ l of NBA on the probe tip. The accelerating voltage was 10 kV and the JEOL Cs⁺ ion gun was operated at 25 keV. Five scans were acquired and averaged between m/z 100-3,500. The instrument was calibrated by using (CsI) nCs⁺ cluster ions. Mass spectra were acquired with a JEOL DA 7,000 data system running on a HP 9,000 computer.

Results and Discussion

Preliminary results show that fast atom bombardment-mass spectrometry (FAB-MS) could be a useful technique

http://newjournal.kcsnet.or.kr/main/j_search/j_download.htm?code=B961118

As shown by the Korean chemists, OspA sticks to itself. We suspect that while OspA molecules are in vaccine vial they are not completely micellized. It would seem this could be responsible for the strokes, cancer, and other vascular events described by Schoen and Steere in their Phase IV trial results and also the totally unreadable Western Blots in both OspA vaccine trials, ImuLyme and LYMERix as shown in this next report by Persing (Mayo and Corixa), Molloy (Imugen), and Sigal:

[Clin Infect Dis](#). 2000 Jul;31(1):42-7. Epub 2000 Jul 17.

Detection of multiple reactive protein species by immunoblotting after recombinant outer surface protein A Lyme disease vaccination.

[Molloy PJ](#), [Berardi VP](#), [Persing DH](#), [Sigal LH](#).

”... The manufacturer of the only currently FDA-approved (and released) recombinant OspA Lyme disease vaccine has suggested that vaccination does not interfere with serological evaluation of Lyme disease in vaccine recipients—a statement that is not supported by the data presented here.”

<http://www.ncbi.nlm.nih.gov/pubmed/10913394>

Let’s hypothesize that OspA molecules in a vaccine vial was probably never 100% micellized and was probably injected into people in clumps. The unreadable, smudged Western Blots of the LYMERix and ImuLyme victims make this appear to be the case. The Cabal did not report to the FDA that they could not read their Western Blots. Instead they falsely claimed they had 76% and 92% safe and effective OspA vaccines based on the falsified Dearborn Western Blot criteria without mentioning to the FDA and the public that the blots in the trials were unreadable. This is clearly in itself a **FRAUD AGAINST THE GOVERNMENT** charge.

<https://www.law.cornell.edu/uscode/text/18/1031>

We don’t know for sure if this particular ligand for plasminogen and chitinous tissue, OspA, was, say added or spliced in or “evolved” such that Lyme spirochetes were allegedly, suddenly found in New England ticks, *Ixodes*. But we can look at the other circumstantial evidence.

III. Lyme spirochetes are closest to an African bird borreliosis and evolutionarily “contrary to its arthropod vector,” Plum Island

You can believe the CDC’s theory that Lyme spirochetes/West Nile blew/flew from Africa to the northeastern United States on seabirds during hurricanes, or, you can consider the circumstantial scientific evidence against the backdrop of CDC’s other lies. For the sake of believing this hurricane BS from your own eyes, see the following report:

[Emerg Infect Dis](#). 2000 Jul-Aug;6(4):319-28.

Migratory birds and spread of West Nile virus in the Western Hemisphere.

[Rappole JH](#), [Derrickson SR](#), [Hubálek Z](#).

“Displacement of West African Birds to the New World by Tropical Storms

”A very few birds, particularly seabirds, are carried by tropical storms across the Atlantic each summer from their normal environs on or near the coast of West Africa (39). A number of such storms form each

summer and fall near the Cape Verde Islands off the western coast of Africa, travel across the Atlantic, and occasionally reach land along the East Coast of North America, depositing birds that were carried thousands of kilometers from their homes. Species known to have been infected by West Nile virus and whose habitat and distribution indicate that they might be affected by such displacement include the Gray Heron (*Ardea cinerea*), the Little Egret (*Egretta garzetta*), the Cattle Egret (*Bubulcus ibis*), the Black-headed Gull (*Larus ridibundus*), and the Yellow-legged Gull (*Larus cachinnans*) (Table 1). The same objections apply to this scenario for the introduction of the virus to the New World as for normal migration, i.e., low numbers and the likelihood that a storm transported bird would be infected with the West African rather than the Middle Eastern form of the virus.”

http://wwwnc.cdc.gov/eid/pdfs/vol6no4_pdf-version.pdf

The following is a key report from the NIH's NLM's Taxonomy (Fukunaga, et al) database showing *burgdorferi* is closest to *anserina*, an African bird borreliosis. They just happen to do this kind of African-Diseases-With-North-American-Vectors-kind of "Research" on Plum Island, as you will see.

[Int J Syst Bacteriol.](#) 1996 Oct;46(4):898-905.

Phylogenetic analysis of Borrelia species based on flagellin gene sequences and its application for molecular typing of Lyme disease borreliae.

[Fukunaga M](#), [Okada K](#), [Nakao M](#), [Konishi T](#), [Sato Y](#).

anserina is genetically closest to burgdorferi in flagellin gene -KMD

of *Borrelia* strains

<i>B. andersonii</i> 19857	<i>B. andersonii</i> 21038	<i>B. andersonii</i> 21123	<i>Borrelia</i> sp. strain Spain	<i>B. hispanica</i>	<i>B. duttonii</i> 406K	<i>B. crocidurae</i>	<i>B. crocidurae</i> ORI	<i>B. miyamotoi</i> HT31	<i>B. miyamotoi</i> FR64b	<i>B. lonestari</i> Texas	<i>B. hermsii</i> HSI	<i>B. hermsii</i>	<i>B. turicatae</i>	<i>B. parkeri</i>	<i>B. anserina</i>	<i>B. coriaceae</i> Co53
96.2	96.2	96.3	82.2	80.1	82.2	82.3	81.0	81.2	81.2	80.8	83.1	83.4	83.5	83.0	84.2	82.3
95.8	95.8	95.9	82.1	80.9	82.0	82.1	80.8	81.1	81.1	80.5	83.0	83.2	83.4	82.8	84.1	82.2
96.1	96.1	96.2	82.3	80.2	82.2	82.3	81.0	81.3	81.3	80.8	83.2	83.5	83.6	83.0	84.3	82.4
93.4	93.3	93.5	82.6	79.8	82.2	82.3	81.0	81.2	81.2	80.3	83.4	83.6	83.6	83.0	84.9	82.8
93.2	93.0	93.3	82.1	79.3	81.7	81.8	80.5	80.9	80.9	80.2	83.0	83.2	83.5	82.9	85.2	83.1
93.2	93.0	93.3	82.2	79.6	81.5	81.6	80.3	80.6	80.6	80.3	82.3	82.5	83.4	82.8	84.4	82.5
93.4	93.0	93.3	82.7	80.2	82.1	82.2	80.9	81.2	81.2	81.1	83.1	83.4	84.0	83.6	85.5	83.0
93.4	93.0	93.3	82.7	80.2	82.1	82.2	80.9	81.3	81.3	81.1	83.1	83.4	83.7	83.2	85.6	83.1
93.4	93.0	93.3	82.4	79.8	81.8	81.9	80.6	81.0	81.0	80.7	83.0	83.2	83.6	83.1	85.5	83.1
94.4	94.3	94.5	83.1	80.8	82.9	83.0	81.7	81.6	81.6	81.3	83.7	83.9	84.2	83.8	84.9	83.3
94.4	94.3	94.5	83.1	80.8	82.9	83.0	81.7	81.6	81.6	81.3	83.7	83.9	84.2	83.8	84.9	83.1
92.2	92.0	92.2	82.0	80.5	82.5	82.2	81.2	80.7	80.7	80.9	82.5	82.7	82.7	82.0	84.0	82.0

← goes closer and closer to 100% at burgdorferi flagellin gene. hermsii and duttonii are less like burgdorferi than anserina is, as you can see.

<http://ijs.sgmjournals.org/content/46/4/898.long>

1995 -- Next, New York Medical College (NYMC) and Marconi at Medical College of Virginia at Virginia Commonwealth University, Richmond, VA, say *anserina* is an “out-group” when comparing *burgdorferi* or the Lyme group from other borrelia. It is not some random out-group. It is the origin of *burgdorferi* as you will see when we talk more about 1) Plum Island as the original outbreak area, where 2) UPenn says this vector-pathogen match-up was evolutionarily unlikely, and 3) where they just happen to do that kind of African-diseases-with-North-American Vectors kind of research on Plum, not to mention, 4) all the CDC’s lies and attempts to have us believe “Lyme disease” is not even a spirochetal disease, but autoimmune arthritis (Dearborn).

[J Clin Microbiol.](#) 1995 Sep;33(9):2427-34.

Identification of novel insertion elements, restriction fragment length polymorphism patterns, and discontinuous 23S rRNA in Lyme disease spirochetes: phylogenetic analyses of rRNA genes and their intergenic spacers in *Borrelia japonica* sp. nov. and genomic group 21038 (*Borrelia andersonii* sp. nov.) isolates.

[Marconi RT1](#), [Liveris D](#), [Schwartz I](#).

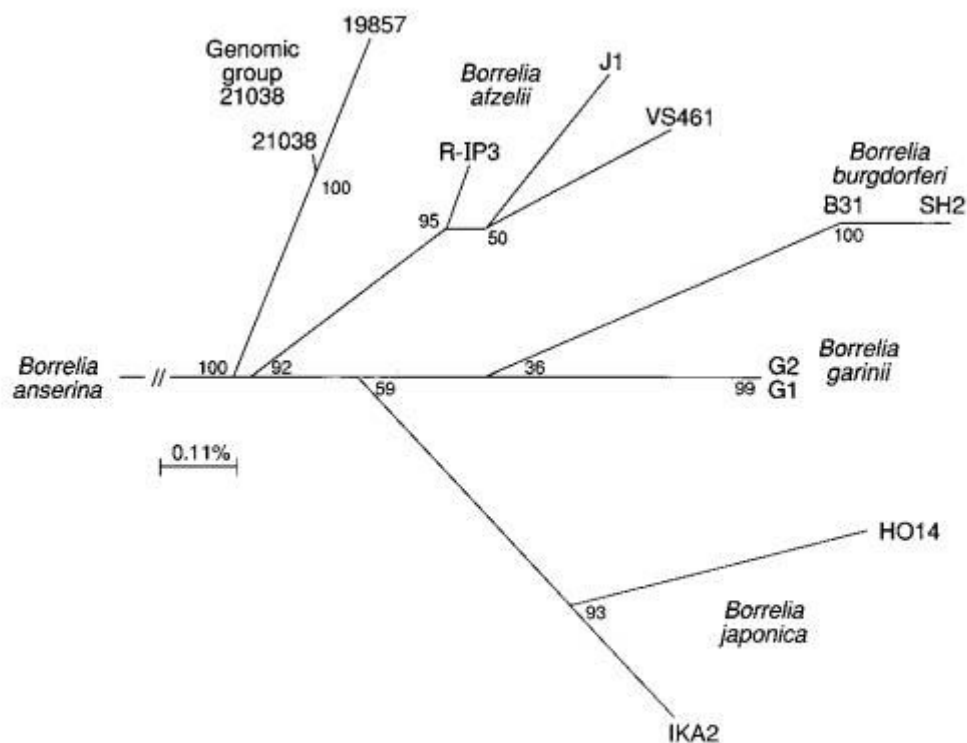


FIG. 7. Phylogenetic tree of 16S rRNA derived from LDS isolates. The phylogenetic tree was constructed as described in the text. Numbers at the branch nodes indicate the results of bootstrap analysis. The 16S rRNA sequence from *Borrelia anserina* served as an outgroup.

<https://www.ncbi.nlm.nih.gov/pubmed/7494041>

UPenn on Lyme spirochetes being evolutionarily unlikely:

Evolution. 2010 Sep;64(9):2653-63. doi: 10.1111/j.1558-5646.2010.01001.x.

Uncoordinated phylogeography of Borrelia burgdorferi and its tick vector, Ixodes scapularis.

Humphrey PT1, Caporale DA, Brisson D.

”Despite the intimate association of *B. burgdorferi* and *I. scapularis*, the population structure, evolutionary history, and historical biogeography of the pathogen **are all contrary to its arthropod vector.**”

<http://www.ncbi.nlm.nih.gov/pubmed/20394659>

More on evolution and expansion north and west from eastern Long Island of the anserina-come-burgdorferi-Plum-Island phenomenon; SUNY-SB on Lyme/Plum Island as the original outbreak area (Ed Bosler):

[Zentralbl Bakteriell Mikrobiol Hyg A](#). 1986 Dec;263(1-2):65-71.

Evolution of a focus of Lyme disease.

[Schulze TL](#), [Shisler JK](#), [Bosler EM](#), [Lakat MF](#), [Parkin WE](#).

<http://www.ncbi.nlm.nih.gov/pubmed/3577493>

1998-- Yale's Durland Fish performing vector-pathogen studies on Plum Island (Borrelia are also found in these pig ticks in Africa):

African swine fever virus infection in the argasid host, Ornithodoros porcinus porcinus.

[J Virol](#). 1998 Mar;72(3):1711-24.

[Kleiboeker SB](#)1, [Burrage TG](#), [Scoles GA](#), [Fish D](#), [Rock DL](#).

1Plum Island Animal Disease Center, Agricultural Research Service, U.S. Department of Agriculture, Greenport, New York 11944, USA.

“The pathogenesis of African swine fever virus (ASFV) infection in *Ornithodoros porcinus porcinus* was examined in nymphal ticks infected with the ASFV isolate Chiredzi/83/1. At times postinfection (p.i.) ranging from 6 h to 290 days, ticks or dissected tick tissues were titrated for virus and examined ultrastructurally for evidence of virus replication. The ASFV infection rate in ticks was 100% in these experiments, and virus infection was not associated with a significant increase in tick mortality. Initial ASFV replication occurred in phagocytic digestive cells of the midgut epithelium. Subsequent infection and replication of ASFV in undifferentiated midgut cells was observed at 15 days p.i. Generalization of virus infection from midgut to other tick tissues required 2 to 3 weeks and most likely involved virus movement across the basal lamina of the midgut into the hemocoel. Secondary sites of virus replication included hemocytes (type I and II), connective tissue, coxal gland, salivary gland, and reproductive tissue. Virus replication was not observed in the nervous tissue of the synganglion, Malpighian tubules, and muscle. Persistent infection, characterized by active virus replication, was observed for all involved tick tissues. After 91 days p.i., viral titers in salivary gland and reproductive tissue were consistently the highest detected. Successful tick-to-pig transmission of ASFV at 48 days p.i. correlated with high viral titers in salivary and coxal gland tissue and their secretions. A similar pattern of virus infection and persistence in *O. porcinus porcinus* was observed for three additional ASFV tick isolates in their associated ticks...

“African swine fever (ASF) is a highly lethal disease of domestic pigs for which animal slaughter and area quarantine are the only methods of disease control. ...

<http://www.ncbi.nlm.nih.gov/pubmed/9499019>

Note that the end-point, here, slaughtering your infected livestock, is a Plum Island-, or as we call it, Von Traub Island-, goal. We should mention there is at least one “Plum Island” strain of *Mycoplasma*:

[J Hyg \(Lond\)](#). 1983 Jun;90(3):441-9.

Immunogenic variation among the so-called LC strains of Mycoplasma mycoides subspecies mycoides.

[Smith GR](#), [Oliphant JC](#).

“Much evidence of immunogenic heterogeneity among the LC strains of *Mycoplasma mycoides* ssp. *mycoides* emerged from cross-immunization and -hyper-immunization experiments in mice in which

three LC strains (Vom/**Plum** Island, 74/2488, and Mankefår 2833) were used for challenge purposes. All heterologous LC-strain vaccines cross-immunized against the three challenge strains, but protection was usually only 'partial', i.e. significantly less than that given by homologous vaccine. Cross-hyperimmunization with all heterologous LC but not SC strains produced protection against challenge with Vom/**Plum** Island that was virtually 'complete', i.e. similar to that produced by homologous vaccine. Challenge with 74/2488 gave generally similar results; but against Mankefår 2833 six heterologous LC vaccines gave complete protection and six did not. Vaccines prepared from the Smith (1423) strain of *M. mycoides* ssp. *capri* gave some protection against Vom/Plum Island but none against 74/2488 or Mankefår 2833. The cross-immunizing ability of three further *M. mycoides* ssp. *capri* strains appeared to resemble that of Smith (1423). In a cross-hyperimmunization experiment, vaccines prepared from SC strains of *M. mycoides* ssp. *mycoides* varied greatly in their ability to protect against challenge with strains 74/2488 and Mankefår 2833”

<http://www.ncbi.nlm.nih.gov/pubmed/6190898>

"*Mycoplasma mycoides mycoides*" = "Fungal-plasma fungal, fungal," nice. Triple fungal mycoplasma on Plum Island. That's adorable. 😊

So, challenging various vectors (bugs) with diseases from Africa is what Plum Island does. Naturally, an odd one could have escaped, one way or another – an African bird borreliosis -, genetically unlikely, and Plum Island was the original outbreak area. That's all the real data we'll ever have because we'll never have the lab notebooks from Plum Island.

If we were prosecuting a murder trial, this all would probably fly circumstantial evidence case as "beyond a reasonable doubt," especially considering all the other lies about Lyme disease, like the hurricane fairy tale, IDSA's "Guidelines on the Diagnosis and Treatment of Lyme disease," the Dearborn "case definition," and most of all the very idea that everyone should get a vaccine against an imaginary disease. No one has ever met or heard from a person who can come up with a sound reason there would be a vaccine against a disease that does not exist and needs no treatment.

IV. Brain Permanence, Tropism and the Single Spirochete Infection with resultant MULTIPLE VARIANTS

J Bacteriol. 1951 Aug;62(2):215-9.

Relapse phenomena in rats infected with single spirochetes (Borrelia recurrentis var. turicatae).
SCHUHARDT VT, WILKERSON M.

“Antigenic variation by the spirochete is generally believed to be responsible for the relapse phenomena in spirochetal relapsing fever. Schuhardt (1942) has reviewed the literature prior to 1942 on this subject, and little if any evidence has been presented subsequently to alter or extend this concept. Among the

unanswered questions in spirochetal relapse phenomena are: (a) the antigenic variation capacity of a single spirochete, and (b) the capacity of an antigenic variety to recur in a series of relapses in a given animal. Although Cunningham, Theodore, and Fraser (1934) believe that antigenic varieties do not recur, other workers are not convinced that this possibility has been ruled out. Consequently we undertook a study of single spirochete infections in white rats in an effort to answer these two and possibly other questions related to the relapse phenomenon in spirochetal relapsing fever.”

<https://www.ncbi.nlm.nih.gov/pubmed/14861181>

<http://jb.asm.org/cgi/reprint/62/2/215?view=long&pmid=14861181>

Oscar Felsenfeld, CDC officer Alan Barbour, Russell Johnson (ALDF member), and Diego Cadavid talking about/referencing this Single Spirochete Phenomena:

http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed&cmd=link&linkname=pubmed_pubmed_citedin&uid=14861181

Oral spirochetes infecting Alzheimer’s brains and traveling along inside nerves (this is not the only report that says this, you’ll find it in syphilis reports too; from the older published data and from the Cabal on the incurability of relapsing fever; an independent study on spirochetes in the brain from dentists and they say:

[Oral Microbiol Immunol.](#) 2002 Apr;17(2):113-8.

Molecular and immunological evidence of oral Treponema in the human brain and their association with Alzheimer's disease.

[Riviere GR1](#), [Riviere KH](#), [Smith KS](#).

“The purpose of this investigation was to use molecular and immunological techniques to determine whether oral Treponema infected the human brain. Pieces of frontal lobe cortex from 34 subjects were analyzed with species-specific PCR and monoclonal antibodies. PCR detected Treponema in 14/16 Alzheimer's disease (AD) and 4/18 non-AD donors ($P < 0.001$), and AD specimens had more Treponema species than controls ($P < 0.001$). PCR also detected Treponema in trigeminal ganglia from three AD and two control donors. Cortex from 15/16 AD subjects and 6/18 controls contained Treponema pectinovorum and/or Treponema socranskii species-specific antigens ($P < 0.01$). T. pectinovorum and/or T. socranskii antigens were also found in trigeminal ganglia and pons from four embalmed cadavers, and 2/4 cadavers also had Treponema in the hippocampus. **These findings suggest that oral Treponema may infect the brain via branches of the trigeminal nerve.**”

<http://www.ncbi.nlm.nih.gov/pubmed/11929559>

1975 -- Jay Sanford, Uniformed Services University School of Medicine, Bethesda, Maryland, page 391, in the book, The Biology of Parasitic Spirochetes, 1976 edited by ALDF.com’s Russell C. Johnson

”**The Biology of parasitic spirochetes**” / edited by **Russell C. Johnson**

Bib ID 2160125, New York ; Academic Press, 1976, ISBN 012387050X

”The ability of the borrelia, especially tick-borne strains to persist in the brain and in the eye after treatment with arsenic or with penicillin or **even after apparent cure is well known** (1). The persistence

of treponemes after treatment of syphilis is a major area which currently requires additional study (3,5,10,11).”

http://www.actionlyme.org/Biology_of_Parasitic_Spirochetes1976.htm

See more at: The History of Relapsing Fever: <http://www.actionlyme.org/RICOCHRON.htm>

There was never any issue with persistence or neurotropism or even lymph node tropism of *Borreliae* despite the CDC’s attempts to defraud and have everyone believe Lyme is not a spirochetal disease. They do play a shell game, though, so as not to find borrelia in humans – and especially, meaning *ALL* *Borreliae* (see the Taxonomy database), not *just burgdorferi*.

Says CDC officer Alan Barbour in 1986:

[Microbiol Rev.](#) 1986 Dec;50(4):381-400.

Biology of Borrelia species.

[Barbour AG, Hayes SF.](#)

“When relapsing fever borreliae are no longer detectable in the blood, they may still be found in organs (120). Although borreliae can usually be recovered from such organs as the spleen, liver, kidneys, and eyes of infected animals (37, 120), the organ usually with the most persistent infections is the brain. Humans with relapsing fever have had borreliae recovered from the cerebrospinal fluid (72). *Borreliae* can be recovered from the brains of animals that are immune to challenge with that strain (119, 127, 148, 178). Detection or isolation of borreliae from brains of animals that had been infected several months and up to 3 years previously has been reported (12, 181, 197, 223). Before the advent of modern ultracold freezers, strains were kept in the brains of rodents and passed once or twice a year (92).”

<https://www.ncbi.nlm.nih.gov/pubmed/3540570>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC373079/pdf/microrev00055-0033.pdf>

Rodent brains use to be the storage media says Barbour, above. And borrelia are often absent from blood even with valid DNA methods like flagellin DNA or species specific 16S genes, because, as Alan Barbour says, they are in the organs, especially the brain and lymph nodes. Obviously a culture method from blood can’t be used for the same reason – they’re not always in the blood.

CDC officer Alan Barbour also says in the same report:

“A strain of *B. duttonii* that had been passed many times in mice was found to have lost virulence for humans (212). When using borreliae for pyrotherapy of neurosyphilis, the authors of this report recommended that no more than 30 to 40 passages in mice be made before inoculation of the strain back into humans (212).”

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC373079/pdf/microrev00055-0033.pdf>

It is fair to say this CDC officer, Alan Barbour, was not too confident in antibiotics if he suggested giving people a fever from a weakened (high passage) relapsing fever organism as a way to cure syphilis. Barbour shows us above that he is aware that one should not use high-passage strains – which Steere did

to develop the Dearborn method -, since the point of high passages is to weaken the strain and have the organisms drop plasmids. We assume the reason Steere falsified the testing for the CDC's Dearborn case definition panel (leaving OspA and B out; OspA and B are encoded on the same plasmid, so you can't drop one without dropping the other), using high-passage strains, was that he and his co-conspirators intended to **develop a test for Lyme that would be okay to use in a population "where the vaccination status was unknown."** **The Schoen-Persing-Steere RICO method patent, US 6,045,804, uses a strain of Borrelia that had dropped the OspA-B plasmid.** It's possible to do that with repeat passages; you can get the bugs to drop plasmids and "virulence determinants" in this way.

We will see from this report CDC officer Allen Steere played the shell game while he falsified the case definition strains, identifying borrelia using the correct primers when he developed that bogus Dearborn method in 1992. Later Steere used mainly the wrong DNA (OspA and in one instance 1 primer probe of 16S RNA) to assess human treatment results. Despite using the wrong primers, Steere found DNA persisted in spinal-fluid, and synovial-fluid of patients to the tune of at least a third of the patients.

1990 – Pachner, on human brain strains changing plasmid DNA code in mice:

Neurology. 1990 Oct;40(10):1535-40.

Borrelia burgdorferi infection of the brain: characterization of the organism and response to antibiotics and immune sera in the mouse model.

Pachner AR1, Itano A.

“To learn more about the neurologic involvement in Lyme disease, we inoculated inbred mice with the causative agent of Lyme disease, *Borrelia burgdorferi*. We cultured brains and other organs, and measured anti-B burgdorferi antibody titers. We further studied a brain isolate for its plasmid DNA content and its response in vitro to immune sera and antibiotics. One strain of B burgdorferi, N40, was consistently infective for mice, and resulted in chronic infection of the bladder and spleen. SJL mice developed fewer culture-positive organs and had lower antibody titers than Balb/c and C57Bl/6 mice. Organism was cultured from the brain early in the course of infection, and this isolate, named N40Br, was further studied in vitro. **The plasmid content of N40Br was different from that of the infecting strain, implying either a highly selective process during infection or DNA rearrangement in the organism in vivo.** N40Br was very sensitive to antibiotics, but only after prolonged incubation. Immune sera from both mice and humans infected with B burgdorferi were unable to completely kill the organism by complement-mediated cytotoxicity. These data demonstrate that B burgdorferi infects the brain of experimental animals, and is resistant to immune sera in vitro but sensitive to prolonged treatment with antibiotics.”

<http://www.ncbi.nlm.nih.gov/pubmed/2215944>

After a time the plasmid content was different from the original strain, says Pachner. That would be because Lyme is just another relapsing fever borreliosis. Antibiotics merely cause the organisms to convert into a spheroplast form, but that is a topic for another DOJ criminal charge sheet. The cyst or spheroplast form is not an “end-stage,” as some claim. It is a replication form. Previously we said, “The thing to do about Lyme is to catch it early before the shed Osp or fungal antigen-related immunosuppression invites (cross-tolerance) other pathogens or reactivates old, dormant ones like the

herpes viruses, do most of the damage.” But apparently this damage is done right away, according to Baumgarth and Chiu.

Where else to we find these fungal, OspA-like antigens?

[J Biol Chem](#). 1999 Nov 19;274(47):33419-25.

Toll-like receptor 2 functions as a pattern recognition receptor for diverse bacterial products.

[Lien E1](#), [Sellati TJ](#), [Yoshimura A](#), [Flo TH](#), [Rawadi G](#), [Finberg RW](#), [Carroll JD](#), [Espevik T](#), [Ingalls RR](#), [Radolf JD](#), [Golenbock DT](#).

”Toll-like receptors (TLRs) 2 and 4 are signal transducers for lipopolysaccharide, the major proinflammatory constituent in the outer membrane of Gram-negative bacteria. We observed that membrane lipoproteins/lipopeptides from *Borrelia burgdorferi*, *Treponema pallidum*, and *Mycoplasma fermentans* activated cells heterologously expressing TLR2 but not those expressing TLR1 or TLR4. These TLR2-expressing cells were also stimulated by living motile *B. burgdorferi*, suggesting that TLR2 recognition of lipoproteins is relevant to natural *Borrelia* infection. Importantly, a TLR2 antibody inhibited bacterial lipoprotein/lipopeptide-induced tumor necrosis factor release from human peripheral blood mononuclear cells, and TLR2-null Chinese hamster macrophages were insensitive to lipoprotein/lipopeptide challenge. The data suggest a role for the native protein in cellular activation by these ligands. In addition, TLR2-dependent responses were seen using whole *Mycobacterium avium* and *Staphylococcus aureus*, demonstrating that this receptor can function as a signal transducer for a wide spectrum of bacterial products. We conclude that diverse pathogens activate cells through TLR2 and propose that this molecule is a central pattern recognition receptor in host immune responses to microbial invasion.”

<http://www.ncbi.nlm.nih.gov/pubmed/10559223> <http://www.jbc.org/content/274/47/33419.long>

What you should do when reading this general science material, particularly like the 1999 one above and see related and see cited by on PubMed.

These triacyl-lipopeptides are only *initially* inflammatory. After a time, this same researcher, Radolf, wrote that these fungal lipoproteins cause immunosuppression and a lack of antibody production:

[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.ncbi.nlm.nih.gov/pubmed/11441098>

Spirochetes create multiple variants and all the individual spirochetes do their own thing, varying their surface antigens on their own, shedding these fungal antigens in a process called blebbing, ruining a person's immune system. And a ruined immune system *is the DAMAGE* and *is the ILLNESS* and *is the specific goal of a bioweapon*:

Types of Biological Agents

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

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

⁶¹ *Ibid.*

⁶² *Ibid.*, p. 9, section 1502.

AND

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

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

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

Do you see the disease now? It's fungal (shed borrelial antigens are TLR2/1-agonists or fungal); it is about "overwhelming the immune system" (which is another way to say, "post-sepsis syndrome"); it is about not producing identifiable antibodies; your bioweapon should be like a Trojan Horse, setting off other latent infections; your immune system is now turned off ("overwhelmed" means "turned off"); you don't have "biofilms" at least of borrelia; Lyme was the "perfect stealth disabler." See more in the Occam's Razor chapter.

V. SIDESTEPPING - Alert on "Biofilms"

Use "Borrelia Staining" or "Borrelia Silver Staining" as search terms in PubMed to discover that Borrelia *in vivo* do not cluster at all, much less under a "biofilm." Here is one. Look closely for the "clustered spirochetes hiding under a biofilm" (there is no such thing):

[J Med Microbiol.](#) 1987 May;23(3):261-7.

Demonstration of spirochaetes in patients with Lyme disease with a modified silver stain.

[De Koning J, Bosma RB, Hoogkamp-Korstanje JA.](#)

<https://www.ncbi.nlm.nih.gov/pubmed/2438410> <http://jmm.sgmjournals.org/content/23/3/261.long>

Here is another one by Paul Duray [same guy who revealed that congenital Lyme brain damage kills babies and who revealed that Lyme- and LYMERix- diseases cause a leukemia-like illness and that the cells in the CSF of Lyme patients "look like Epstein-Barr transformed (mutated, pre-cancerous) cells]:

[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>

Additionally, some biofilms are covered in TLR2/1 agonists so the body does not even see them at all any more, if they are there in this post-sepsis disease called Chronic Lyme, with the multiple reactivated herpes viruses, etc., and the expansion of tolerance to other toll-like-receptor-managed antigen types. The biofilms could be covering other organisms, but spirochetes are all independent operators and the illness and the damage is mainly from the secondary, "Post Sepsis Syndrome," infections.

REVIEW: Biofilms covering spirochetes are NOT responsible for the persistent symptoms in Chronic Lyme Disease. Spirochetes, while permanent, and while they have been shown to be draped in lymphocyte membrane, or have a "mucopeptide layer" (or while were always known to be covered in a slime layer), are *not* the main cause of the disease or the reason antibiotics fail.

Yes, spirochetal diseases are incurable. No, the disease is not about spirochetes, since they shed fungal antigens and ruin the immune system, inviting in other opportunistics or reactivating old ones. We learned this from LYMERix disease where the vaccine gave people the same systemic disease we know of as Chronic Lyme or Chronic Fatigue Syndrome. It is a NO-IMMUNE disease, post-sepsis.

VI. On using the correct DNA to look for spirochetes in humans by using recombinant *Borrelia*-specific flagellin DNA product to detect those specific antibodies

Says Yale:

[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."

<http://www.ncbi.nlm.nih.gov/pubmed/1894359>

Yale says that their method (same method patented as US patent 5, 618, 533) detects, early, late, neurological, and every other possible kind of Lyme outcome *and that it detects 94.4% of the cases*, which means it is the closest possible method we could possibly have to detect Lyme ("should be 100% of the cases," says the FDA, verbatim), *and this method was made SPECIFIC, which means it does not detect any other flagellins from non-Borreliae organisms.*

When the FDA says "sensitivity," they really mean "LIMIT OF DETECTION" and refer to the METHOD and not the "CASES." "Accuracy" addresses cases. Yale, as you can see, took care of all that in 1991 and went ahead and patented it. They did not, however, use this method to qualify LYMERix, their other patent, which is the essence of this False Claims Act case.

The only way to detect a spirochetal disease is to use recombinant specific flagellin antibody test from most of the Borreliae species that we know to be at least in the United States. THAT is what is "VALID," and the FDA and NIH agree.

VII. The FDA being forced to assure Lyme testing is valid according to their own rules by the Senators (summer, 2014):

Here we have to talk about the FDA and what their rules are for the "Validation of an Analytical Method." As you can see there is Accuracy (should detect 100% of the instances when the analyte in question is present), Specificity (only detects one thing), Linearity, Ruggedness, Precision (refers to instrumentation), Limit of Detection (this would be something like, "How low in concentration of the analyte in question can your method detect?").

This is from the new announcement July 31, 2014 regarding the FDA now about to ENFORCE their validation rules:

<http://www.fda.gov/downloads/MedicalDevices/ProductsandMedicalProcedures/InVitroDiagnostics/ucm407409.pdf>

For the Purpose of Notification to Congress Only

requirements under the FD&C Act. Namely, CLIA requirements address the laboratory's testing process (i.e., the ability to perform laboratory testing in an accurate and reliable manner). Under CLIA, accreditors do not evaluate test validation prior to marketing nor do they assess the clinical validity of a LDT (i.e., the accuracy with which the test identifies, measures, or predicts the presence or absence of a clinical condition or predisposition in a patient). Under the FD&C Act, the FDA assures both the analytical validity (e.g., analytical specificity and sensitivity, accuracy and precision) and clinical validity of diagnostic tests through its premarket clearance or approval process. In addition to premarket review, FDA requirements provide other controls to ensure appropriate design, manufacture, and safety and effectiveness of the device. As a result, while CLIA oversight is important, it alone does not ensure that LDTs are properly designed, consistently manufactured, and are safe and effective for patients.

The FDA says: "Under the FD&C Act, the FDA assures both the analytical validity (e.g., analytical specificity and sensitivity, accuracy, and precision) and clinical validity of diagnostic tests through its premarket clearance or approval process."

"Sensitivity" MEANS "Limit of Detection." The closest thing to Sensitivity in the FDA (real) requirements is "Limit of Detection." Keep that in mind because the Cabal misuses that word all the time.

FDA Rules on the Validation of an Analytical Method:

<http://www.fda.gov/downloads/drugs/guidancecomplianceregulatoryinformation/guidances/ucm368107.pdf>

Specificity (only detects one thing)

Accuracy (Should detect 100% of the instances where the analyte is present, and the concentration should be close to 100% of that known to be spiked in, and never should detect "none" as is the case with Lyme Western Blotting and the Lyme ELISA, especially)

Limit of Detection (means "What is the lowest concentration of the analyte in question does your method detect?")

Precision (system has integrity in performance)

Ruggedness (anyone can run the test with their own equipment and get the same results)

Linearity (concentration range of analyte for which the test is valid in and out of matrix or "inert ingredients")

Your test should primarily detect *all* the cases in question, - or be 100% ACCURATE - and that means, in the case of Lyme, the only analyte for which we can test is flagellin or anti-flagellar antibodies. Anti-flagellar antibodies can be found in probably 95% of Lyme cases. So, Yale went ahead and made that Specific (also described in US patent 5,618,533) in 1991, as shown previously, above.

[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](#).

<http://www.ncbi.nlm.nih.gov/pubmed/1894359>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC258917/pdf/iai00046-0199.pdf>

For the other *Borrelia* in North America and Europe, at least, such a recombinant-specific-multiple-flagellin method should be developed and the NIH agrees with this (May 2012, phone conversation). There is no other way to detect most cases of Borreliosis. All the other antigens are plasmid-encoded and variable. *Borrelia* spirochetes are not always in the blood, so there is no point to using a blood DNA method. Flagellin is the only reliable antibody and it can be made specific. There is no “end point” to treatment, since late Lyme is more about the other opportunistics. But early Lyme, all agree that the flagellar antibody test is the only test that captures the majority of cases and meets the FDA criteria for “ACCURACY.”

Lyme and LYMERix cause immunosuppression and an AIDS-like disease or an acquired immune deficiency, or as the NIH describes it, post-sepsis with the all kinds of still-active herpes and other infections. It should be said that Lyme and LYMERix diseases are far worse than just spirochetes. Apparently that has always been the case. **The Great Imitator, Syphilis was probably really the Great Detonator of the latent herpes and other infections.** Syphilis was probably the original AIDS, via OspA-like or fungal-antigen-like immunosuppression and the reactivation of mostly Epstein-Barr.

VIII. SIDE-STEPPING - CDC's Other Research Fraud: A) Lying about the viability of the cyst or spheroplast form of spirochetes and B) lying about mycoplasma not being involved in Chronic Fatigue Syndrome

CDC and IDSA claimed the cyst form was not viable, and that *Borrelia* DNA-positive human samples were “just dead DNA” (never happens, the body cleans up such debris). Yet here is the CDC in 1964 explaining how to dessicate and weaponize your *Borrelia* (freeze-drying – and good for at least a year, they say):

[J Bacteriol.](#) 1964 Sep;88:811.

RECOVERY OF TREPONEMA AND BORRELIA AFTER LYOPHILIZATION.

[HANSON AW](#), [CANNEFAX GR](#).

<https://www.ncbi.nlm.nih.gov/pubmed/14208528> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC277387/pdf/jbacter00438-0287.pdf>

Next we see the CDC is throwing out the blood cells (throwing out whole cells of any kind), including red blood cells and immune cells or white cells to which mycoplasma adhere, while alleging to look for

mycoplasma in Chronic Fatigue Syndrome. Mycoplasma or epERTHROzoa are called epERYTHROzoa because they attach to red blood cells. Such epERYTHROzoa are famous for changing the erythrocyte membrane potential and the ability of oxygen to cross the red blood cell membrane, causing tremendous fatigue even in animals.

CDC did this to allegedly show Mycoplasma were not involved in Chronic Fatigue Syndrome:

[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](#).

"Plasma, the liquid portion of peripheral blood that is devoid of cells, is known to contain remnants of numerous physiological and disease processes. We used plasma DNA to detect and characterize bacterial 16S rDNA sequences in a group of individuals with CFS and a group of non-fatigued controls (Vernon et al., 2002). Whilst a variety of bacterial sequences were detected in both fatigued and non-fatigued groups, no Mycoplasma sp. 16S rDNA sequences were found."

<https://www.ncbi.nlm.nih.gov/pubmed/14532349> <http://jmm.sgmjournals.org/cgi/pmidlookup?view=long&pmid=14532349>

That is important. The CDC does not want anyone to know fungal antigens and/or fungal antigen tolerance cause(s) extreme fatigue. They must be important bioweapons, a problem with the pediatric vaccines causing Autism, or both.

Next up the specific DNA Shell Game played by members of the Cabal. You will see it is almost entirely CDC officers committing this fraud. The data you have seen so far reveals 1) how to test for all Borrelioses, 2) how we got this particularly evolutionarily unlikely bird borreliosis in New England "on hurricanes?" and 3) catching the CDC staff committing research fraud in other arenas.

IX. The CDC Cabal Play the DNA and RNA Shell Game (we learned what is proper detection DNA: flagellin, and other non-variable specific RNA)

Alan Barbour playing the DNA/RNA shell game.

You will want to look at **The Patents** Criminal Charge sheet for this multi-Crymes Disease to see that CDC officer and former head of the NIH's Rocky Mountain Bioweapons Montana Lab (you're familiar with Montana, the place where there are tons of relapsing fever borrelia but no "Lyme?"), **Alan Barbour**, reported that, basically, "antigenic variation in one spirochete, times all the spirochetes you

have, leaves the immune system ‘overwhelmed’ with ‘an infinite number of new antigens.’” This is a characteristic or attribute of bioweapons, well described by the US Army when speaking to Congress as shown previously in this document.

With all this malarkey about “Lyme disease” as opposed to relapsing fever, and how the pediatric Autism vaccines fail and give children the very brain infections they’re meant to prevent (same mechanisms; immunosuppression either via fungal exposure or some other exposure, or genetic immune insufficiency, plus live, attenuated viruses that become un-attenuated), you get the impression that the CDC was never mentally or morally competent to maintaining theirs and the USDA’s fallacies. We’ve long called the CDC the Centers for Disease Confabulation.

Alan Barbour states below that OspA undergoes true antigenic variation and basically that, therefore you cannot use this as a vaccine (while he owns the patent for the ImmuLyme OspA non-vaccine). If it undergoes “true antigenic variation,” it certainly cannot be used for DNA diagnostics as Klemperer allegedly did in his “*BREAKING NEWS!!!*” bogus “re-treatment” “study” that is now the data used by IDSA for their “Guidelines on Lyme” from 2001 and 2006. Klemperer said he looked for DNA of *Borrelia* in the spinal fluid of his victims – and used OspA primers (ones that will CHANGE and therefore not be there).

[J Exp Med.](#) 1992 Sep 1;176(3):799-809.

Antibody-resistant mutants of Borrelia burgdorferi: in vitro selection and characterization.

[Sādziene A1](#), [Rosa PA](#), [Thompson PA](#), [Hogan DM](#), [Barbour AG](#).

Notwithstanding infrequent application for bacterial studies presently, there were compelling reasons to use in vitro antibody selection with *Borrelia burgdorferi*, the agent of Lyme disease. First, we had shown for the related species, *B. hermsii*, that an antiserum specific for one serotype could select for new serotypes in an isogenic population undergoing in vitro growth (20). The ability of polyclonal antisera and mAbs to agglutinate (21) and inhibit the growth of *B. burgdorferi* (21a) indicated that this was also possible with the Lyme disease agent. Second, previous studies had shown antigenic differences in outer membrane proteins, OspA and OspB, between strains (21–26) and also true antigenic variation of these proteins within a strain (25, 27–30). If borrelias did “escape”

medicine • Volume 176 September 1992 799–809

Says Barbour above: “Second, previous studies had shown antigenic differences in outer membrane proteins, OspA and OspB, between strains (21-26) and also true antigenic variation of these proteins within a strain (25, 27-30).”

<http://www.ncbi.nlm.nih.gov/pubmed/1339462> , <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2119346/>

None of the OspA vaccines ever prevented Lyme or spirochetes in any animal. OspA vaccination may have prevented arthritis by tolerance, but no animal study showed prevention of spirochetes.

Remember, “mutants” is code language. They’re all mutants. Antigenic variation or “selection pressure” is

the nature of the relapse in relapsing fever. To call them mutants is silly and redundant, and not the least bit correct as you've seen in Barbour's patents and in the older data such as the Single Spirochete outcome.

Here are Fikrig and Flavell, Yale employees and inventors of the LYMERix patent saying the same thing: Due to antigenic variations and antibodies forcing the bugs to change surface antigens, OspA or variable DNA can never be a vaccine against Lyme or Relapsing Fever:

[Infect Immun.](#) 1995 May;63(5):1658-62.

Selection of variant *Borrelia burgdorferi* isolates from mice immunized with outer surface protein A or B.

[Fikrig E1](#), [Tao H](#), [Barthold SW](#), [Flavell RA](#).

"...B. burgdorferi organisms expressing wild-type OspA (data not shown), showing that immunization against a clonal population of spirochetes is also dependent upon the challenge dose. Therefore, we postulate that during tick-borne infection, a population of antigenically heterogeneous spirochetes may be transmitted to the host (27) and that the spirochetes that persist in the immune host during the evolution of infection and the development of chronic disease are more likely to be partially resistant to borreliacidal immune responses.

"This report describes the ability of OspA and OspB antibodies to cause the in vivo selection of B. burgdorferi organisms with subtle genetic alterations that result in the expression of OspA or OspB which do not bind to, or weakly bind with, antibodies that are protective in nature. These data suggest a potential reason for the lack of complete efficacy of an Osp-based Lyme disease vaccine. Over extended periods of time, the administration of an OspA- or OspB-based vaccine to hosts that are involved in the natural life cycle of the spirochete may result in the expansion of variant B. burgdorferi isolates within ticks at a higher frequency than would normally be found in the general population. If this selection pressure was to be maintained, the number of variant spirochetes could rise to a significant level, such that the efficacy of a monovalent OspA- or OspB-based vaccine could be impaired in the future."

<http://www.ncbi.nlm.nih.gov/pubmed/7729870> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC173206/>

Alan Barbour's patent saying antigenic variation and "overwhelms the immune system":

Patent filed in 2002 -

VMP-like sequences of pathogenic *Borrelia*

"2.1 Methods of Treatment

"... An important aspect of the invention is the recognition that *Borrelia* VMP-like sequences recombine at the vls site, with the result **that antigenic variation is virtually limitless. Multiclonal populations therefore can exist in an infected patient so that immunological defenses are severely tested if not totally overwhelmed.**"

<http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetacgi%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6,719,983.PN.&OS=PN/6,719,983&RS=PN/6,719,983>

So, you can't use the OspA gene for a vaccine, for post-treatment or late Lyme DNA diagnostics, or for "Lyme case" detection in antibodies. The only thing you can do or say about OspA is that it apparently helped the normally, formerly non-borreliae-bearing *Ixodes* (hard bodied) ticks acquire a ligand (OspA-B plasmid) with which to attach to and invade the hard bodies of hard bodied (*Ixodes*) ticks. Lyme spirochetes were **probably** adapted on Plum Island to local vectors. Genetically, the Lyme spirochete is closest to *anserina*, an African bird borreliosis, making it potentially likely to spread around fast.

1995 – Barbour's patent for Lyme in Missouri, using 16S RNA sequencing and flagellin primer probes that many of the Lyme Cabal members did not use when assessing human treatment outcomes.

Barbour says this is in Lone Star ticks in Missouri:

Diagnostic tests for a new spirochete, Borrelia lonestari sp. nov.

"Bites from *Amblyomma americanum*, a hard tick, have been associated with a Lyme disease-like illness in the southeastern and south-central United States. Present in 2% of ticks collected in four states were uncultivable spirochetes. Through use of the polymerase chain reaction, partial sequences of the flagellin and 16s rRNA genes of microorganisms from Texas and New Jersey were obtained. The sequences showed that the spirochete was a *Borrelia* sp. but distinct from other known members of this genus, including *B. burgdorferi*, the agent of Lyme disease. Species-specific differences in the sequences of the flagellin protein, the flagellin gene and the 16s rRNA gene between the new *Borrelia* species and previously known species provide compositions and methods for assay for determining the presence of this new spirochete, or for providing evidence of past or present infection by this spirochete in animal reservoirs and humans...

"...SUMMARY OF THE INVENTION

"The present invention provides compositions, methods, and kits for the detection of a new spirochete that is associated with a Lyme disease-like illness. **The compositions are based on a *Borrelia lonestari* sp. nov.-specific allotype or combination of allotypes of the flagellin protein, or a *Borrelia lonestari* sp. nov.-specific allele or combination of alleles of the flagellin or 16s rRNA genes of the new spirochete.** The allotypes and alleles provided by the present invention have been determined by nucleic acid sequencing of portions of the flagellin and rRNA genes from this new spirochete. Detection of a species-specific amino acid or nucleotide as defined herein, or a species-specific combination of amino acids or nucleotides as defined herein, in a subject sample is indicative of infection with *Borrelia lonestari* sp. nov."

<http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetachtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5%2C932%2C220.PN.&OS=PN%2F5%2C932%2C220&RS=PN%2F5%2C932%2C220>

Barbour sequenced the RNA and DNA, obviously and did not use someone else's primers. Using primer probes from *Borreliae* not expected to be there (*burgdorferi* flagellin and another specific *lonestari* gene) rather than sequencing. Therefore, *Borreliae* cannot be ruled out as Wormser did when assaying EM rashes in Missouri. This patent of Barbour's also shows Barbour knows what are the species-distinguishers: NOT THE OSPS, VMPS or the PLASMID DNA. He then can't sign on to any claims

about Lyme that mimic the CDC's and IDSA's current fraudulent positions without expecting to be indicted. Barbour does claim however, that *the species identifier is... FLAGELLIN*.

Gary Wormser playing the DNA/RNA shell game.

Next we are going to look at Gary Wormser who is in 1992 **using the correct primers**; this proves he knows exactly how to identify *Borrelia* species. Later, in order to “prove” there is no Lyme in Missouri, he does not apply this same technique.

[J Clin Microbiol](#). 1992 Dec;30(12):3082-8.

Diagnosis of early Lyme disease by polymerase chain reaction amplification and culture of skin biopsies from erythema migrans lesions.

[Schwartz I1](#), [Wormser GP](#), [Schwartz JJ](#), [Cooper D](#), [Weissensee P](#), [Gazumyan A](#), [Zimmermann E](#), [Goldberg NS](#), [Bittker S](#), [Campbell GL](#), [Pavia CS](#).

”rRNA-based PCR detection assay for *B. burgdorferi*.

”The organization of the rRNA genes of *B. burgdorferi* and the sequences of the corresponding rRNAs have been determined (32). Figure 1 presents a schematic diagram of the rRNA operon and the positions of the primers and probes employed for PCR amplification and detection. The 23S rRNA sequence was compared for homology to other rRNA sequences in the GenBank data base. On the basis of these comparisons, a region near the 5' end of the 23S RNA sequence (nucleotides 689 through 948) was chosen as a likely target for amplification. The equivalent regions of the 23S rRNA genes in the related species *Borrelia hermsii* and *B. anserina* and several isolates of *B. burgdorferi* were also sequenced (Fig. 2). PCR primers (designated JS1 and JS2) were designed to contain perfect homology to the *B. burgdorferi* sequence but maximum mismatch at their 3' ends with the related *Borrelia* species (Fig. 2). The sensitivity of the PCR assay was determined with serially diluted, titered *B. burgdorferi* samples. Fewer than 10 spirochetes in a total sample could be detected efficiently (Fig. 3). The sensitivity and specificity of the assay were also investigated by performing PCR amplification with 10 different isolates of *B. burgdorferi*, *B. hermsii*, *B. anserina*, and *Borrelia turicatae*. Samples containing 50 spirochetes were subjected to PCR amplification, and one-fifth of the amplified product (equal to 10 spirochetes) was detected by hybridization with a radiolabeled probe (FS1) corresponding to a portion of the amplified sequence. All isolates of *B. burgdorferi* were detected by the procedure with essentially equal efficiency (Fig. 4). These included isolates from North America (isolates 24430, 24352, HK, B31, 297), Europe (20004, GI, 20047), and Russia (IP90, IP3). Furthermore, only *B. burgdorferi* was detected by this method; samples containing the other closely related *Borrelia* species produced no amplified product.

”To provide a second primer pair that could be employed for specific detection of *B. burgdorferi*, we took advantage of the unusual and unique tandem duplication of the 23S rRNA gene (Fig. 1). This feature was observed in all *B. burgdorferi* isolates tested and, furthermore, was not found in other *Borrelia* species (32). Thus, a PCR primer pair with the forward primer targeted to a sequence at the 3' end of the first copy of 23S RNA gene and a reverse primer complementary to a sequence near the 5' end of the second 23S RNA gene copy should have absolute specificity for *B. burgdorferi*. The locations of this primer pair (designated IS1 and IS2, respectively) relative to the rRNA operon are presented in Fig. 1. The sensitivity and specificity of this primer pair were tested in a manner similar to that described above for the JS1-JS2

primer pair. The IS1-IS2 amplicon set displayed a degree of specificity and sensitivity similar to that of JS1-JS2 (Fig. 5).

<http://www.ncbi.nlm.nih.gov/pubmed/1452688> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC270592/pdf/jcm00036-0064.pdf>

In 2005, in Missouri, Wormser did not use these same methods. We'll see that report shortly.

In this next report, **Gary Wormser** in 1999, when using the correct primers (16S) finds some people are infected with more than one species of *Borrelia burgdorferi* (yet, ignoring the other *Borrelia*) and that you can't really use Barbour-Kelly-Stoener culture media, the only one anyone sells in the USA. It shows Wormser knows what DNA to use when looking for spirochetes, yet he is a signer of the IDSA "Guidelines" which are based on Klempner's bogus "re-treatment" "study," which is based on the falsified Dearborn case definition, and which is based on the bogus OspA gene to determine "no Lyme" after "treatment."

[J Clin Microbiol](#). 1999 Mar;37(3):565-9.

1999 - Genetic diversity of *Borrelia burgdorferi* in lyme disease patients as determined by culture versus direct PCR with clinical specimens.

[Liveris D1](#), [Varde S](#), [Iyer R](#), [Koenig S](#), [Bittker S](#), [Cooper D](#), [McKenna D](#), [Nowakowski J](#), [Nadelman RB](#), [Wormser GP](#), [Schwartz I](#).

"... The data confirmed the presence of the three major RFLP types previously described (17). Of 183 skin isolates, 46 (25.1%) were type 1, 70 (38.3%) were type 2, and 55 (30.1%) were type 3; **the remaining 6.6% (12 of 183) were mixed cultures composed of at least two genotypically distinct isolates.**"

<http://www.ncbi.nlm.nih.gov/pubmed/9986813>

1995-6—Alan Barbour does proper sequencing for the analysis of the spirochetes in the Lone Star tick (compare to what Wormser does, following this report):

[J Infect Dis](#). 1996 Feb;173(2):403-9.

Identification of an uncultivable *Borrelia* species in the hard tick *Amblyomma americanum*: possible agent of a Lyme disease-like illness.

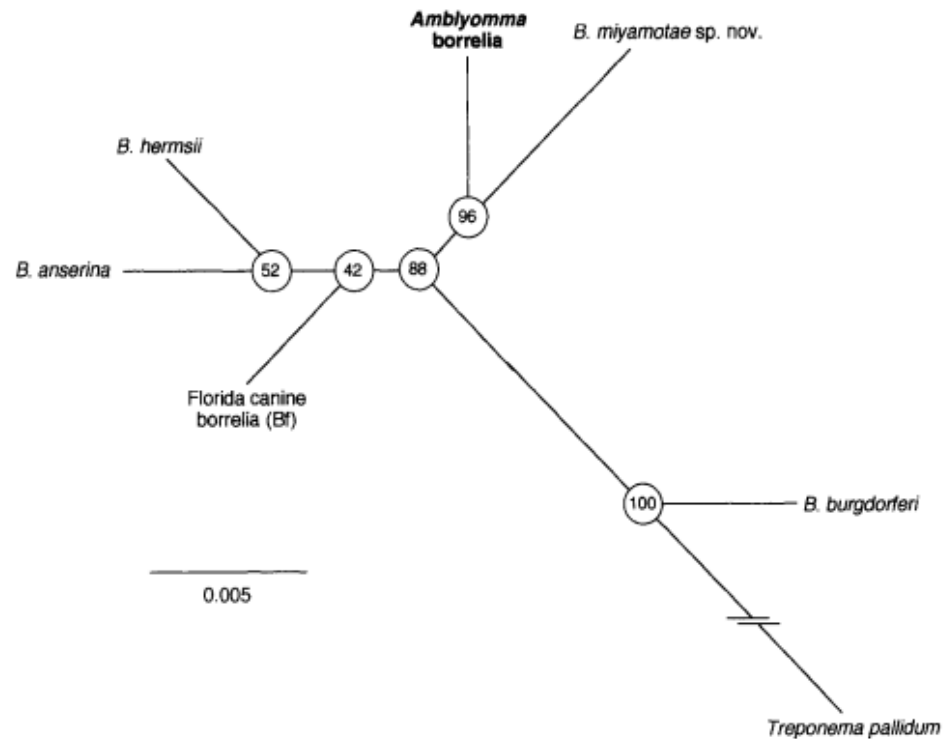
[Barbour AG1](#), [Maupin GO](#), [Teltow GJ](#), [Carter CJ](#), [Piesman J](#).

"...The deduced amino acid sequences for flagellin proteins of the 2 microorganisms found in *A. americanum* were identical over 213 residues; the nucleotide differences between strains were synonymous. Figure 3 shows the alignment of part of the deduced flagellin sequences of the spirochetes found in *A. americanum* in Texas and New Jersey with the comparable variable regions of the flagellin proteins of 8 *Borrelia* species and *Treponema pallidum*, the spirochete that causes syphilis. The amino acid positions are numbered according to the full length *B. burgdorferi* flagellin protein. The flagellin proteins of microorganisms found in *A. americanum* differed from other borrelial flagellins at several

positions and, uniquely among the *Borrelia* species, lacked most of a proline-alanine-rich region beginning around residue 220. The spirochetes found in *A. americanum* resembled *B. turicatae*, *B. hermsii*, *B. parkeri*, *B. crocidurae*, and *B. anserina* in being without the QAA at residues 204-206 of the Lyme disease agents *B. burgdorferi*, *B. garinii*, and *B. afzelii*...

“Analysis of 16S rRNA genes. Further phylogenetic classification was provided by comparison of 16S rRNA gene sequences (figures 4 and 5). The sequence of the spirochete found in *A. americanum* from Texas had the following identities with selected other spirochete 16S rRNA genes: *T.pallidum*, 79.6%; *B. burgdorferi*, 96.0%; *B. anserina*, 97.5%; *B. hermsii*, 97.8%; *B. miyamotoe* sp. nov., 98.3%; and the "Florida canine borrelia," 98.4%. By distance matrix and parsimony analyses of the aligned sequences (figure 4), the spirochete found in *A. americanum* clustered with a group containing the relapsing fever species *B. hermsii*, *B. anserina*, the unnamed organism recovered from the blood of 2 dogs in Florida [25], and *B. miyamotoe* sp. nov. (accession no. 045192).”

Figure 4. Unrooted distance matrix phylogenetic tree of *Borrelia* species with *Treponema pallidum* as outgroup. 16S rRNA sequences corresponding to base positions 36–1371 of *Borrelia burgdorferi* 16S rRNA gene were aligned and analyzed with PHYLIP program package. Exhibited tree in New Hampshire standard format is: (((Florida canine borrelia: 100, (*Borrelia anserina*: 100, *Borrelia hermsii*, 100): 52): 42, (borrelia from *A. americanum*: 100, *Borrelia miyamotoe* sp. nov.: 100): 96): 88, *T. pallidum*: 100, *B. burgdorferi*: 100). Circled numbers indicate number of times (in 100) that particular node was supported by bootstrap analysis. Approximate evolutionary distances are measured along line segments; bar represents distance by Jukes-Cantor criteria of 0.005. Similar tree (not shown) was obtained by parsimony analysis of 100 bootstrapped datum sets: ((((((borrelia from *A. americanum*: 100, *B. miyamotoe*: 100): 94, *B. hermsii*: 100): 34, Florida canine borrelia: 100): 25, *B. anserina* 100): 81, *B. burgdorferi*: 100): 100, *T. pallidum*: 100).



5). Among the 6 sequences represented in figure 5, there were 49 aligned positions at which only 1 of the 6 species differed; 27 (53%) of these differences were in *B. burgdorferi*. The following tree was produced with 100 bootstrapped data sets of these positions: (((borrelia from *A. americanum*: 100, *B. miyamotoe*: 100): 94.4, (Florida canine borrelia: 100, (*B. hermsii*: 100, *B. anserina*: 100): 38): 64): 100, *B. burgdorferi*: 100).

Again, the borrelia from *A. americanum* clustered with the non-Lyme disease *Borrelia* species; it was most closely related to *B. miyamotoe* sp. nov. and the Florida canine borrelia.

Discussion

In this study, ~2% of *A. americanum* ticks from Missouri, New Jersey, New York, North Carolina, and Texas contained

”Figure 4. Unrooted distance matrix phylogenetic tree of *Borrelia* species with *Treponema pallidum* as outgroup. 16S rRNA sequences corresponding to base positions 36–1371 of *Borrelia burgdorferi* 16S rRNA gene were aligned and analyzed with PHYLIP program package. Exhibited tree in New Hampshire standard format is: «(Florida canine borrelia: 100, (*Borrelia anserina*: 100, *Borrelia hermsii*, 100): 52): 42, (borrelia from *A. americanum*: 100, *Borrelia miyamotoe* sp. nov.: 100): 96): 88, *T. pallidum*: 100, *B. burgdorferi*: 100). Circled numbers indicate number of times (in 100) that particular node was supported by bootstrap analysis. Approximate evolutionary distances are measured along line segments; bar represents distance by Jukes-Cantor criteria of 0.005. Similar tree (not shown) was obtained by parsimony analysis of 100 bootstrapped datum sets: «««borrelia from *A. americanum*: 100, *B. miyamotoe*: 1(0): 94, *B. hermsii*: 1(0): 34, Florida canine borrelia: 100): 25, *B. anserina* 100): 81, *B. burgdorferi*: 100): 100, *T.*

pallidum: 100).”

<http://www.ncbi.nlm.nih.gov/pubmed/8568302> jid.oxfordjournals.org/content/173/2/403.full.pdf

Barbour actually sequenced for flagellin and 16S RNA and found all kinds of spirochetes in this way.

For the Missouri Lyme Masters’ Disease spirochete, Barbour said...

Again, the borrelia from *A. americanum* clustered with the non-Lyme disease *Borrelia* species; it was most closely related to *B. miyamotoe* sp. nov. and the Florida canine borrelia.

... while Wormser tried to say “No Lyme In Missouri.” (By the way, no one cares if they have *burgdorferi* or *antarcticii* or *siberii* or freakin *jupiterii*. They just want to know if the science shows they’re sick.)

Here is Wormser trying to fool Edwin Masters, using the wrong DNA and RNA so he can say, “There is no Lyme in Missouri”

[Clin Infect Dis](#). 2005 Feb 1;40(3):423-8. Epub 2005 Jan 10.

Microbiologic evaluation of patients from Missouri with erythema migrans.

Wormser GP1, Masters E, Liveris D, Nowakowski J, Nadelman RB, Holmgren D, Bittker S, Cooper D, Wang G, Schwartz I.

“PCR amplifications were performed in a 50- μ L reaction mixture containing 10 mmol/L Tris-HCl (pH 8.3); 1.5 mmol/L MgCl₂; 50 mmol/L KCl, 0.1% (w/v) gelatin; 100 μ mol/L each of dATP, dGTP, dCTP, and TTP; 1.25 units Taq polymerase; and 20 pmol of each primer. **Detection of borrelial DNA in patient specimens and ticks was accomplished by the nested PCR amplification of flaB using primers FlaLL, FlaLS, FlaRL, and FlaRS as described by Barbour et al [11]. PCR of 16S rDNA was performed with broad-range eubacterial primers 8FPL and 1492RPL [26], which yields a product of ~1.5 kbp.** In cases in which no detectable product was obtained, second-round heminested PCR was performed with 8FPL and a reverse primer (519R: 5'-TTACCGCGGCTGCTGGC-3') targeted at residues 535–518 (numbering corresponds to residues in the 16S RNA sequence of Escherichia coli) in 16S rDNA; this resulted in a fragment of 500 bp. Some specimens were also tested by PCR targeted at ospA (forward primer, 5'-CTGCAGCTTGGAATTCAGGCACTTC-3'; reverse primer, 5'-GTTTTGTAAATTTCAACTGCTGACCCCTC-3') and/or recA [27].”

<http://www.ncbi.nlm.nih.gov/pubmed/15668867> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2773674/>

Wormser did not use the correct Borreliae-specific (for non-*burgdorferi* or from any other relapsing fever groups) flagellin genes, or 16S rDNA specific to Borreliae species, nor did he actually try to sequence any of these Borreliae as Barbour did (and Telford, below). Wormser would have known to use the correct method to detect spirochetes in Lone Star ticks, since he referenced Barbour’s work (ref 11

was: ***Identification of an uncultivable Borrelia species in the hard tick Amblyomma americanum: possible agent of a Lyme disease-like illness.*** (shown above). Wormser knows how to do this kind of DNA analysis and that there are all sorts of Borrelia in Lone Star ticks – and ones that cause human disease.

The enzyme Wormser talks about, GlpQ (next reference here, by the NIH) is specific to *B. lonestari*, but that does not mean there are no disease-causing Borreliae in Lone Star ticks or Missouri.

[J Bacteriol.](#) 2003 Feb;185(4):1346-56.

Glycerol-3-phosphate acquisition in spirochetes: distribution and biological activity of glycerophosphodiester phosphodiesterase (GlpQ) among Borrelia species.

[Schwan TG1](#), [Battisti JM](#), [Porcella SF](#), [Raffel SJ](#), [Schrumpf ME](#), [Fischer ER](#), [Carroll JA](#), [Stewart PE](#), [Rosa P](#), [Somerville GA](#).
<http://www.ncbi.nlm.nih.gov/pubmed/12562805> , <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC142843/>

Wormser’s whole point was to say there are no Borrelia causing disease or “Lyme” in Missouri. The very last statement he makes in that report is: “Although it is unknown whether this rash illness has an infectious etiology, it is important to emphasize that this study does not indicate the absence of a therapeutic role for antibiotic treatment.” (AKA, CYA, in the common vernacular.) **It could have been some other Borrelia, just not burgdorferi or lonestari. Check the Taxonomy database for all the Borrelia in the USA alone.**

Additionally, these were Wormser’s actual results:

Table 1**Test results for specimens obtained from patients with erythema migrans (EM) or EM-like lesions**

Laboratory test	Missouri patients with EM-like lesions (n = 30)	New York State patients with EM (n = 143)	P ^a
Skin PCR/culture			
Positive <i>flaB</i> PCR	5/31 (16) ^b	70/139 (50)	<.001
Positive eubacterial 16S RNA PCR	0/20 (0) ^b	Not done	
Positive <i>gfpQ</i> PCR	0/22 (0) ^b	Not done	
Positive <i>Borrelia burgdorferi</i> culture	0/19 (0)	89/142 (63)	<.001
Serum serologic analysis			
Acute phase, seropositive ^c	0/25 (0)	81/143 (57)	<.001
Convalescent phase, seropositive (1–3 months)	0/22 (0)	99/135 (73)	<.001
Seroconversion ^d	0/22 (0)	25/52 (48)	<.001
Acute or convalescent phase, seropositive	0/25 (0)	107/143 (75)	<.001
Overall ^e	5/31 (16) ^b	130/143 (90.9)	<.001

NOTE. Data are no. of specimens with positive test results/no. that were evaluable (%).

^aBy 2-tailed Fisher's exact test.

^bTwo biopsies of separate EM-like lesions were performed for 1 patient.

^cBy ELISA.

^dNegative-to-positive or equivocal-to-positive results ≤3 months after baseline.

^ePositive *B. burgdorferi* culture, positive PCR result, or seropositive.

**5 were positive
for a Bb fla gene.**

Here is Wormser's abstract:

[Clin Infect Dis](#). 2005 Feb 1;40(3):423-8. Epub 2005 Jan 10.

Microbiologic evaluation of patients from Missouri with erythema migrans.

Wormser GP1, Masters E, Liveris D, Nowakowski J, Nadelman RB, Holmgren D, Bittker S, Cooper D, Wang G, Schwartz I.

"*Borrelia lonestari* infects *Amblyomma americanum*, the tick species that is the most common cause of tick bites in southeast and south-central United States, and this spirochete has been detected in an erythema migrans (EM)-like skin rash in 1 patient. Therefore, *B. lonestari* is considered to be a leading candidate for the etiologic agent of EM in this region.

"Skin biopsy specimens obtained from patients from the Cape Girardeau area of Missouri who had EM-like lesions were cultured in Barbour-Stoenner-Kelly medium and evaluated by polymerase chain reaction

(PCR) targeting multiple genes. Serum specimens were tested by enzyme-linked immunosorbent assay for antibodies against sonicated whole-cell *Borrelia burgdorferi*. Results were compared with those obtained over the same period for patients from New York State who had EM.

“*B. lonestari* was not detected by PCR in any of 31 skin biopsy specimens collected from 30 Missouri patients. None of 19 cultures of Missouri skin samples that were suitable for evaluation were positive for *B. burgdorferi*, compared with 89 (63%) of 142 cultures of samples collected from New York State patients ($P < .001$). None of the 25 evaluable Missouri patients were seropositive for antibodies against *B. burgdorferi*, compared with 107 (75%) of 143 New York State patients ($P < .001$).

“Neither *B. lonestari* nor *B. burgdorferi* is likely to be the cause of EM-like skin lesions in patients from the Cape Girardeau area of Missouri. The etiology of this condition remains unknown.

<https://www.ncbi.nlm.nih.gov/pubmed/15668867>

So, just to reiterate Wormser’s Take-Home Message here: ”There is no Lyme in Missouri except for those 5 out of ~30, and we were playing around with the DNA primers. (Shhh: It’s closer to *B. miyamotoe* and Florida Canine Lyme disease *Borrelia*. Also, Shhhh, the Barbour culture medium is specific to *burgdorferi* spirochetes and does not grow all spirochetes.) The EM-Lyme illness in Missouri could have been from some other Relapsing Fever or Ehrlichial pathogen but we are pretending there is no such thing as an illness from a tick bite. Lyme is imaginary so get the placebo vaccine. We used no *hermsii* or other common spirochetes known to be in the west and south of the United States. The end game is to not find illness and to say that *there is no illness from a tick bite*, while Barbour patented the *lonestari* bug right under Masters’ nose for later use as a vaccine against this disease that does not exist and no one has, so get the vaccine....especially in Missouri.”

It is, yes. This is a dizzying kaleidoscope of lies, is what you’re thinking now. The whole point of it all, and the reason this scam continues, is because the Cabal does not want anyone to know they falsified the case definition at Dearborn. They LIED to everyone and said, “No, ‘Lyme Disease’ is a self-limiting autoimmune monoarticulate joint disease. It does not cause any illness.” (It’s “pretense.”)

Well, why do modern spirochetes not cause any illness? Because OspA injections had the same outcome. These criminal dummy wannabee scientists are mortified, really, over being discovered to be not only that stupid, but crooked. Think about it. They never *were* “the smartest guys in the room,” but they wanted to be seen as an authority on something, didn’t they? It’s obvious. Why else would they e-stalk us and e-trash us for going on 3 decades now, as way to allegedly be “scientific?”

Mark Klempner, playing the DNA/RNA shell game.

You have previously seen that the OspA gene undergoes antigenic variation and is not found in all *Borreliae*. You can't use this DNA for anything, especially not vaccines or detection. We move on to the Klempner “study” which unfortunately resulted in the 2001 and 2006 IDSA “Guidelines” and where he references which DNA he used to assess "NO LYME IN LYME VICTIMS." Klempner doesn’t actually

say what DNA he uses (only by reference) to determine “No Lyme” in Lyme victims, and the peer reviewers at the New England Journal of Medicine (NEJM) never noticed he did not list his primers:

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

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

[Klempner MS](#), [Hu LT](#), [Evans J](#), [Schmid CH](#), [Johnson GM](#), [Trevino RP](#), [Norton D](#), [Levy L](#), [Wall D](#), [McCall J](#), [Kosinski M](#), [Weinstein A](#).

<https://www.ncbi.nlm.nih.gov/pubmed/11450676> <http://www.nejm.org/doi/full/10.1056/NEJM200107123450202#t=articleMethods>

Laboratory Studies

Western blotting for IgG antibodies against *B. burgdorferi* antigens was performed with the IgG MarBlot (MarDx Diagnostics, Carlsbad, Calif.) according to the manufacturer's instructions.⁶ The intrathecal production of antibodies against *B. burgdorferi* was measured as previously described.²⁰ Base-line specimens of cerebrospinal fluid and plasma specimens obtained at base line and on days 3, 5, 21, and 45 were tested by PCR for the presence of *B. burgdorferi* DNA, as previously described.²¹ All samples of cerebrospinal fluid were cultured in Barbour–Stoenner–Kelly II medium to detect *B. burgdorferi* and were monitored by dark-field microscopy for six weeks.²² Some blood samples were cultured for *B. burgdorferi* in hypertonic medium.²³

“Laboratory Studies

“Western blotting for IgG antibodies against *B. burgdorferi* antigens was performed with the IgG MarBlot (MarDx Diagnostics, Carlsbad, Calif.) according to the manufacturer's instructions.⁶ The intrathecal production of antibodies against *B. burgdorferi* was measured as previously described.²⁰ Base-line specimens of cerebrospinal fluid and plasma specimens obtained at base line and on days 3, 5, 21, and 45 were tested by PCR for the presence of *B. burgdorferi* DNA, as previously described.²¹ All samples of cerebrospinal fluid were cultured in Barbour–Stoenner–Kelly II medium to detect *B. burgdorferi* and were monitored by dark-field microscopy for six weeks.²² Some blood samples were cultured for *B. burgdorferi* in hypertonic medium.²³”

So, what was that mysterious REFERENCE 21 above ^^ DNA that Klempner failed to report and the so-called peer-reviewers did not notice?

Steere’s

[J Infect Dis.](#) 1996 Sep;174(3):623-7.

Detection of Borrelia burgdorferi DNA by polymerase chain reaction in cerebrospinal fluid in Lyme neuroborreliosis.

[Nocton JJ](#), [Bloom BJ](#), [Rutledge BJ](#), [Persing DH](#), [Logigian EL](#), [Schmid CH](#), [Steere AC](#).

<https://www.ncbi.nlm.nih.gov/pubmed/8769624> <http://jid.oxfordjournals.org/content/174/3/623.full.pdf>

WHICH SAYS:

PCR assay. CSF samples from case and control patients were processed simultaneously in a blinded manner, as described [11]. Briefly, DNA was isolated from 100 μ L of CSF, and the DNA extract was resuspended in 30 μ L of ultrapure water. A 5- μ L aliquot from this suspension was amplified with primer-probe set 1, which targets base pairs 788–943 at the 3' end of the 50-kb plasmid of *B. burgdorferi* that encodes OspA [11]. This primer-probe set detects most strains of *B. burgdorferi* from New England. A second 5- μ L aliquot was amplified with primer-probe set 2, which targets base pairs 149–343 at the 5' end of the *ospA* gene [11]. This set detects all North American and European isolates tested to date, with the exception of rare natural isolates that lack the 50-kb plasmid. Amplification consisted of 45 cycles of denatur-

ONLY an OspA gene and later added an OspB gene (read the whole report). And where Steere found many positive patients, Klemmner says he found none (2001 RI “Diseases of Summer” conference at South County Hospital, audiotaped).

The Cabal – including Klemmner in his 2001 bogus non-retreatment report that is now the basis of the IDSA “Guidelines” - say if the OspA gene is not there, there is no Lyme, right? This, despite the fact that 1) Lyme is a relapsing fever borrelia and OspA is a variable plasmid gene and therefore not likely to be in the same form or produced by the exact same genetic code as one produced inside a tick, late in the disease in humans; 2) they've used and sequenced for 16S or flagellin DNA (non-variable, although species-specific and for which there are more copies) in the past, particularly to patent and therefore own species; 3) and referenced the NIH recommendation for using these 16 and 23S probes.

Durland Fish, using *the correct* primers to look for new species of Borreliae to patent in 2001, yet is a signer of the IDSA “Guidelines” once again, based only on an OspA gene and Dearborn as “Lyme” or the chronic illness caused by a tick bite.

[Vector Borne Zoonotic Dis.](#) 2001 Spring;1(1):21-34.

A relapsing fever group spirochete transmitted by Ixodes scapularis ticks.

[Scoles GA1](#), [Papero M](#), [Beati L](#), [Fish D](#).

"A 1,347-bp portion of 16S rDNA was amplified from a pool of infected nymphs, sequenced, and compared with the homologous fragment from 26 other species of Borrelia. The analysis showed 4.6% pairwise difference from *B. burgdorferi*, with the closest relative being Borrelia miyamotoi (99.3% similarity) reported from *Ixodes persulcatus* in Japan. Phylogenetic analysis showed the unknown Borrelia to cluster with relapsing fever group spirochetes rather than with Lyme disease spirochetes. A 764-bp fragment of the flagellin gene was also compared with the homologous fragment from 24 other Borrelia species. **The flagellin sequence of *B. burgdorferi* was 19.5% different from the unknown Borrelia and showed 98.6% similarity with *B. miyamotoi*.**"

<http://www.ncbi.nlm.nih.gov/pubmed/12653133>

What that means is the probably-Plum-Island-and-unlikely-hurricane-borrelia, *B. burgdorferi*, migrated to

Japan and back to the United States again, “mutating” to adapt to a Japanese *Ixodes* tick. Yet, a year later, we see Durland Fish using the WRONG DNA (OspA gene again), to assess treated mice, to determine if there is any *Borrelia*, coming to the conclusion that there is pretty much no *Borrelia*:

Detection of Attenuated, Noninfectious Spirochetes in Borreliaburgdorferi–Infected Mice after Antibiotic Treatment

"PCR of DNA. DNA was isolated from individual ethanol-fixed nymphs or pooled larvae by means of the Isoquick DNA isolation kit (ORCA Research) and was resuspended in 20 µL of double-distilled H₂O. Primers used for amplification were as follows: *** ospA *** (GenBank accession no. M57248, product amplicon coordinates 80–781): forward, 5'-AAAACAGCGTTTCAGTAGATTTGCCTGGTG-3', and reverse, 5'-CAACTGCTGACCCCTCTAATTTGGTGCC-3'; BBE21.1 (GenBank accession no. AE000785, product amplicon coordinates 14663–14921): forward, 5'-AGAATTATGTCGGTGGCGTTGT-3', and reverse, 5'-ATTAAAGCCGCCTTTTCCTTGGT-3'; and p37-47 (GenBank accession no. AE000794, product amplicon coordinates 1309–1457): forward, 5'-TTCTGATGGCACTGAGCAAACCA-3', and reverse, 5'-AACCCCTTACACTTTCTTCGATTGCGCT-3'. The primer set for p37–47 has 100% homology to sequences in both *B. burgdorferi* strains B31 and N40, and the gene has been localized to lp28-1 in both strains [26, 27]. The primer set for BBE21.1 amplifies a unique region in lp25 of *B. burgdorferi* strain B31 downstream of BBE21 (amplicon coordinates 13403–14530) [28]. BBE21 is located on a similar-size plasmid within *B. burgdorferi* strain N40 [29]. We have been able to amplify by PCR the region corresponding to GenBank accession number AE000785, product amplicon coordinates 14195–14921, indicating that BBE21 and BBE21.1 reside on the same plasmid in N40 (authors' unpublished data)" <http://jid.oxfordjournals.org/content/186/10/1430.long>

Those are plasmids, those "lp" things. Plasmids are from where the variable surface protein antigens vary themselves. So, that is a classical Durland Fish type “bogus article.” See: http://www.actionlyme.org/TICK_BITE_CONSPIRACY.htm where Durland admits that he writes “bogus articles,” not that you’re not already convinced. Anyway, previously he used the correct DNA, 16rDNA and flagellin, but then in a post-treated case or cases, he used a gene not likely to be there, OspA.

It probably is true that the spirochetes become attenuated as we have seen with Jay Sanford stating that spirochetes “persist in the brain and eye even after apparent cure” (or they do all their damage early), and Alan Barbour recommending infecting syphilis patients with old, wimpy, high-passage *Borrelia* spirochetes to raise a fever. Spirochetal diseases are all un-eradicable, as shown above. And surely it is true that older spirochete populations in the same host lose plasmids [NEVER use plasmid DNA to assess Lyme in humans], but the end game, and the point of all this crime, is that the Cabal is trying to say that their chronically ill victims are not sick, just crazy. In the end it was OspA itself, a fungal antigen causing the reactivation of the herpesviruses, Post-Sepsis Syndrome, and humoral immunosuppression with chronic inflammation in the brain due to all the neurotropic herpes viruses and *Mycoplasma*, etc., that blew up their intended RICO “enterprise” scam (ALDF.com). It was a very dumb choice for a vaccine.

All of what you see in these SASH criminal charge sheets is evolving criminal fraud in an attempt to hide

all their previous lies. The most serious offense, falsifying the case definition and rendering the 85% without the arthritis HLAs - the million or so per year - permanently disabled, is the one offense the Cabal so vigorously tries to mask by issuing “Guidelines.” The “Guidelines,” based on Klempner, which is based on Dearborn, is a way of Offense being the Best Defense. It’s Pretense, or FRAUD. The Cabal would have the world believe that *they* all believe the case definition was real and valid. We know for sure they know it was not valid acceptable based on the non-consensus at Dearborn alone.

To put such debased individuals in charge of humans and vector-borne diseases? That’s the United States; everything happens exclusively for profit. Greed is our nature. It is synonymous with Exceptional! It’s ALL ABOUT ME!! And it’s ALL ABOUT MONEY!! Hence, the new and totally novel in human history, the BS de-scrambler Society for the Advancement of Scientific Hermeneutics. Now we have Scientific Hermeneutics because this BS has become like a religion or belief system, a DOCTRINE, if you will, that to date, no “doctor” ever unscrambled on behalf of humanity.

Sam Telford's 2001 report saying “Southern Lyme” is closest to *theileri* or bovine relapsing fever (the former “Tick Fever” that the cowboy/farmer wars were all about):

[J Clin Microbiol](#). 2001 Feb;39(2):494-7.

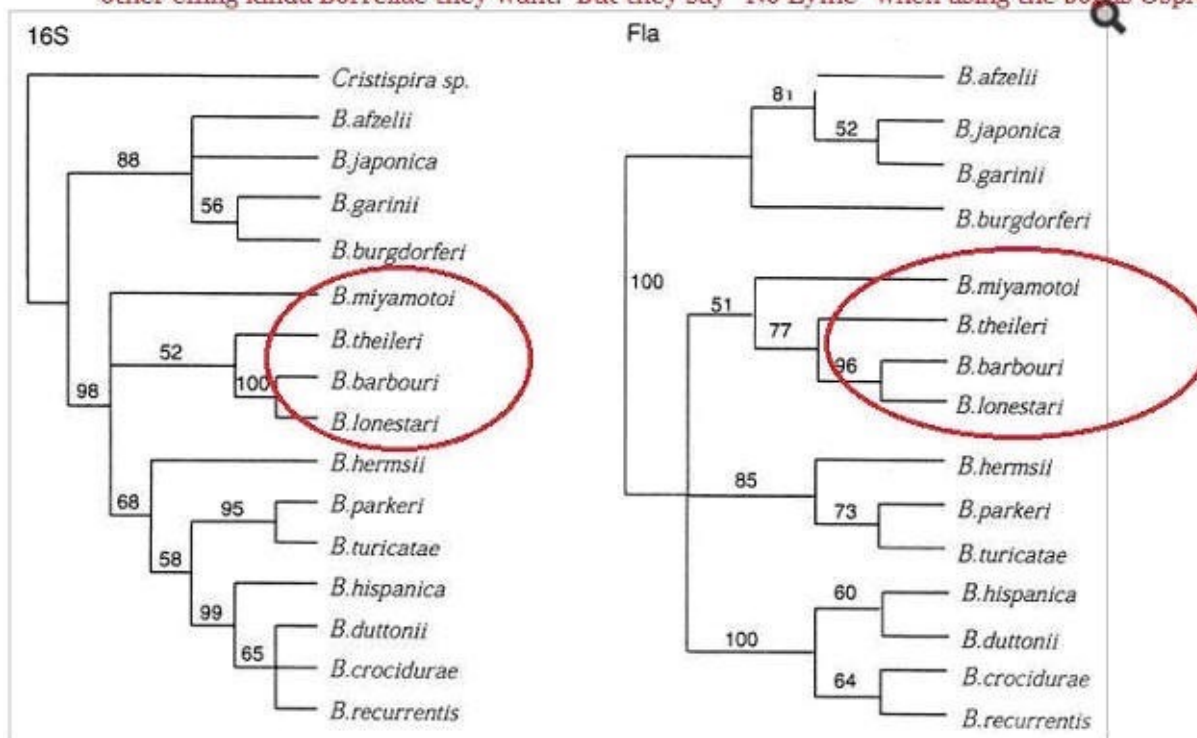
Lone star tick-infecting borreliae are most closely related to the agent of bovine borreliosis.

[Rich SM1](#), [Armstrong PM](#), [Smith RD](#), [Telford SR 3rd](#).

“Although *Borrelia theileri*, the agent of bovine borreliosis, was described at the turn of the century (in 1903), its relationship with borreliae causing Lyme disease or relapsing fever remains undescribed. We tested the previously published hypothesis that spirochetes infecting Lone Star ticks (*Amblyomma americanum*) may comprise *B. theileri* by analyzing the 16S ribosomal DNAs (rDNAs) and flagellin genes of these spirochetes. 9, the *Amblyomma* agent, and *B. miyamotoi* formed a natural group or clade distinct from but most closely related to that of the relapsing fever spirochetes. *B. theileri* and the *Amblyomma* agent were 97 and 98% similar at the nucleotide level within the analyzed portions of the 16S rDNA and the flagellin gene respectively, suggesting a recent divergence. The agent of bovine borreliosis might be explored as a surrogate antigen for the as-yet-uncultivable *Amblyomma* agent in studies designed to explore the etiology of a Lyme disease-like infection associated with Lone Star ticks.”

<http://www.ncbi.nlm.nih.gov/pubmed/11158095>

FIG. 1 When using the correct DNA or RNA primers, crooks find Masters' Disease or Southern Lyme or any other effing kinda *Borreliae* they want. But they say "No Lyme" when using the bogus *OspA* gene.



Bootstrap consensus (1,000 times) of neighbor-joining trees of *Borrelia* spirochetes using both 16S rDNA (left) and flagellin (right) loci. *Cristispira* sp. was used as an outgroup. Distances were derived from the Tamura-Nei algorithm (27a). Numbers indicate the bootstrap s

You can see that the Cabal had already sequenced the 3 similar strains of flagellar and genus specific 16S RNA spacer genes that are derived from a cow or bovine relapsing fever (*theileri*, *barbouri*, and *lonestari*). But there is no "Lyme" in Missouri. You can see it is a shell game.

TO THIS DATE – from 1995 to 2015 - still, no one is using any other of this proper DNA or RNA or SEQUENCING rather than using bogus primer probes they know will not be found in humans to detect human illness. *They all know* the only way to detect Lyme/Relapsing Fever is with specific recombinant flagellin from all the *Borreliae*, similar to Yale's Lyme specific flagellin patented method, US 5,618,533.

1995- Yale's Robert Schoen and Erol Fikrig say, essentially, "OspA is bogus due to antigenic variation"

[J Immunol.](#) 1995 Dec 15;155(12):5700-4.

An ospA frame shift, identified from DNA in Lyme arthritis synovial fluid, results in an outer surface protein A that does not bind protective antibodies.

[Fikrig E1](#), [Liu B](#), [Fu LL](#), [Das S](#), [Smallwood JI](#), [Flavell RA](#), [Persing DH](#), [Schoen RT](#), [Barthold SW](#), [Malawista SE](#).

“Passive immunization with murine or human Abs to outer surface protein A (OspA) can protect mice against *Borrelia burgdorferi*, but OspA Abs elicited during natural infection in mice or humans are unable to clear the spirochete from the infected host. To examine Ab binding by OspA during the course of human infection, we amplified the operon encoding full-length ospA and ospB from synovial fluids of a patient with chronic Lyme arthritis, the first such recoveries from human material, at four separate time points over 4.5 mo, and expressed OspA in *Escherichia coli*. OspA mAbs that passively protected mice from infection did not bind one of the expressed OspAs, because of a deletion in ospA that resulted in a frame shift and premature stop codon near the carboxyl terminus. However, expressed OspA from a later synovial fluid sample did not contain this deletion. Thus, although altered forms of OspA, which potentially can influence host immune effectiveness, do occur in the human host, they cannot be the only factors responsible for microbial persistence.”

<http://www.ncbi.nlm.nih.gov/pubmed/7499856>

Oh, you mean Lyme is a Relapsing Fever organism, so you can't use the OspA gene for human treatment outcomes assessment or vaccines, huh Mr. Schoen, or to detect “Lyme” in EM rashes in Missouri?

Telford, Fish, Schoen, Steere, Persing, Barbour, etc., were able to find any kind of *Borrelia* anywhere America – and they are everywhere, North, South, Central, West -, sequencing for species-specific non-variable, non-plasmid DNA.

Yale's Robert Schoen (who says Lyme is not a real disease, says, “I call it Lyme paranoia,” and needs no treatment) using 23S RNA primers to assure his RICO monopoly strain (and later patent with Dave Persing, US Patent 6,045,804) is *burgdorferi*. On page 235 of the .pdf, Schoen says:

[J Clin Microbiol](#). 1997 Jan;35(1):233-8.

***Borrelia burgdorferi* enzyme-linked immunosorbent assay for discrimination of OspA vaccination from spirochete infection.**

[Zhang YQ1](#), [Mathiesen D](#), [Kolbert CP](#), [Anderson J](#), [Schoen RT](#), [Fikrig E](#), [Persing DH](#).

“...Subsequent evaluation of this isolate in our laboratory showed that this strain was nonreactive with an OspA-based PCR assay designed to detect all North American and European isolates of *B. burgdorferi* but that it contained **23S ribosomal DNA** sequences indistinguishable from those of most North American strains of *B. burgdorferi* sensu stricto such as strains B31 and N40 (22). Genomic macrorestriction analysis of this isolate by PFGE is shown in Fig. 1. By PFGE, the isolate is related to *B. burgdorferi* N40, relatives of which are widely distributed in the northeastern United States, the Upper Midwest, and California (22). These isolates are also closely related to type strain B31, in contrast to isolates from moderate-climate regions of the southeastern and southwestern United States, which are often related to strain 25015 (19, 22). However, in contrast to strain N40, strain 49736 apparently lacked the ca. 53-kb linear plasmid species presumed to encode OspA and B. To verify this observation, we hybridized Southern blots of the MluI digest with a probe specific for the OspA gene. In contrast to strains N40 and B31, which were strongly OspA probe positive, no detectable signal was observed in the digest derived

from strain 49736 (not shown). This observation was consistent with the absence of the 53-kb plasmid species. Similar results were obtained from N40-like isolates 46047, 48510, and B31-like isolates 46794 and 50772 (1).”

<http://www.ncbi.nlm.nih.gov/pubmed/8968914> <http://jcm.asm.org/content/35/1/233.full.pdf>

FIG. 1. PFGE analysis of *Mlu*I-digested genomic DNA from *B. burgdorferi* B31, N40, and 49736. The unmarked lane contains a mixture of lambda DNA *Hind*III fragments, lambda DNA, and lambda concatemers (Sigma) used as a molecular size marker. Southern blotting of this gel followed by hybridization with an OspA probe (OspA6s-3a) also showed that isolate 49736 lacked OspA (data not shown).

profile by protein gel electrophoresis (1). Subsequent evaluation of this isolate in our laboratory showed that this strain was nonreactive with an OspA-based PCR assay designed to detect all North American and European isolates of *B. burgdorferi* but that it contained 23S ribosomal DNA sequences indistinguishable from those of most North American strains of *B. burgdorferi* sensu stricto such as strains B31 and N40 (22). Genomic macrorestriction analysis of this isolate by PFGE is shown in Fig. 1. By PFGE, the isolate is related to *B. burgdorferi* N40, relatives of which are widely distributed in the northeastern United States, the Upper Midwest, and California (22). These isolates are also closely related to type strain B31, in contrast to isolates from moderate-climate regions of the southeastern and southwestern United States, which are often related to strain 25015 (19, 22). However, in contrast to strain N40, strain 49736 apparently lacked the ca. 53-kb linear plasmid species presumed to encode OspA and B. To verify this observation,

RNA/DNA Shell Game

Here Yale's Robert Schoen (who says Lyme is not a real disease and needs no treatment) using 23S RNA primers to assure his RICO monopoly strain (and later patent with Dave Persing) is related to *burgdorferi*,...

and also reveals there is "Lyme" in the Southern and Western states in 1996.

PubMed ID # 8968914
US Pat No. 6,045,804

and also reveals there is "Lyme" in the Southern and Western states in 1996:

Says Schoen, “These isolates are closely related to type strain B31, in contrast to isolates from moderate-climate regions of the southeastern and southwestern United States which are often related to strain 25015 (19,22).”

And what are those references, 19 and 22?

REF 19 - [Res Microbiol.](#) 1995 Jun;146(5):415-24.

Two geographically distinct isolates of Borrelia burgdorferi from the United States share a common unique ancestor.

[Kolbert CP1](#), [Podzorski DS](#), [Mathiesen DA](#), [Wortman AT](#), [Gazumyan A](#), [Schwartz I](#), [Persing DH](#).

“The genetic diversity of *Borrelia burgdorferi* isolates from several geographic regions was evaluated by nucleotide sequence analysis of the genes encoding **23S ribosomal RNA** and outer surface protein A.

Comparison of nucleotide sequences spanning 738 bp of the 23S ribosomal DNA from two unusual isolates, DN127 (Del Norte County, California) and 25015 (Millbrook, New York), to homologous sequences from other *B. burgdorferi* isolates from the United States and Russia identified several nucleotide sequence polymorphisms that are unique to these two isolates. Sequence analysis of a 615 nucleotide segment of the gene encoding outer surface protein A also revealed greater similarity of strains DN127 and 25015 (94.1%) compared to other US and Eurasian isolates. These data were further corroborated by genomic macrorestriction analysis, in which DN127 and 25015 demonstrated unique restriction digestion patterns. Our findings suggest that substantial genetic diversity of *B. burgdorferi*, rivaling that of European strains, exists among isolates from the United States. Strains DN127 and 25015 are unique among all *B. burgdorferi* isolates tested to date, and though isolated from opposite longitudinal extremes of the North American continent, are closely related.”

<http://www.ncbi.nlm.nih.gov/pubmed/8525058>

In other words, this funny-, like, accidental- Ixodes-come-Plum Island borrelia had already reached the American West, not to mention Europe, by 1995. Are we to believe Missouri has an invisible anti-bird and anti-rodent barrier?

REF 22- 1997 – **Persing and Telford**, again (and you’ll just have to look at the full text pdf of this article because you’re not going to believe how many different kinds of Borrelia are found in just about every state):

J Infect Dis. 1997 Jan;175(1):98-107.

Genetic heterogeneity of Borrelia burgdorferi in the United States.

[Mathiesen DA1](#), [Oliver JH Jr](#), [Kolbert CP](#), [Tullson ED](#), [Johnson BJ](#), [Campbell GL](#), [Mitchell PD](#), [Reed KD](#), [Telford SR 3rd](#), [Anderson JF](#), [Lane RS](#), [Persing DH](#).

"To examine in detail *Borrelia burgdorferi* strain diversity in the United States, 186 isolates from human, tick, and rodent sources were analyzed from multiple distinct geographic regions of the United States and abroad. Strains were characterized by genomic macrorestriction analysis and *ospA* and **23S rDNA gene** sequencing followed by phylogenetic analysis. Results indicate that spirochetal isolates from the United States fall into two major divisions and nine or more subdivisions; human isolates fell into five of these subdivisions. Greater genetic diversity was observed among *B. burgdorferi* isolates from moderate climatic regions, consistent with increased tick vector and reservoir diversity. All of the *Borrelia* isolates were reactive by *ospA* polymerase chain reaction except for *Borrelia hermsii* controls and several tick isolates from the Northeast, which were shown to lack the 49-kb plasmid encoding outer surface protein A (*OspA*). The data suggest that US *B. burgdorferi* isolates demonstrate substantial genetic heterogeneity, with regional differences in spirochete populations.

<http://www.ncbi.nlm.nih.gov/pubmed/8985202>

<http://jid.oxfordjournals.org/content/175/1/98.long>

Citing Authors:

<http://jid.oxfordjournals.org/cgi/crossref-forward-links/175/1/98>

This is all in comparison to what IDSA says about Lyme and particularly what Klempner did with his research-fraud re-treatment study published in 2001, which is now the basis of the IDSA “Guidelines.” Klempner allegedly looked for the *OspA* gene in people, so he could declare that no one had Lyme after

“re-treatment”: <http://www.ohioactionlyme.org/wp-content/uploads/2015/02/Biomarkers1.pdf>

Allen Steere playing the DNA-RNA Shell Game; from 1992 when he falsified the Dearborn case definition (ref Marconi and the NIH re 16S probes), his 2 DNA analyses of post-treatment of humans where he found treatment failed in at least a third of the cases, and in the spinal fluid analysis where he used only an OspA probe, dropping the 16S probe he used in bad knees. Steere signs the “Guidelines” anyway and denies that treatment fails.

Allen Steere in 1992 when he falsified the Dearborn case definition, see his reference to Marconi and assessments of strains with 16S RNA; notice references 11, 24...

1992-1994 -- [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](#).

“The group 1 strain of *B. burgdorferi*, G39/40, used in this study and in the previous study of US patients was isolated from an *Ixodes damini* tick in Guilford, Connecticut [21]. The group 2 strain, FRG [*Federal Republic of Germany*], was isolated from *Ixodes ricinus* near Cologne [22]. The group 3 strain, IP3, was isolated from *Ixodes persulcatus* near Leningrad [23]. All three strains used in this study were high passage isolates, which were classified by Richard Marconi (Rocky Mountain Laboratory, Hamilton, MT) using **16S ribosomal RNA** sequence determination as described [11, 24]. The recombinant preparations of OspA and OspB used in this study were purified maltose-binding protein-Osp fusion proteins derived from group 1 strain B31 [25]. The fusion proteins contained the full-length OspA or OspB sequence without the lipid moiety or the signal sequence -”

bite, and clinical manifestations of the infection.
Antigen preparations. Supernatants from sonicated lysates of whole spirochetes were prepared as described [20]. The group 1 strain of *B. burgdorferi*, G39/40, used in this study and in the previous study of US patients, was isolated from an *Ixodes damini* tick in Guilford, Connecticut [21]. The group 2 strain, FRG, was isolated from *Ixodes ricinus* near Cologne [22]. The group 3 strain, IP3, was isolated from *Ixodes persulcatus* near Leningrad [23]. All 3 strains used in this study were high-passage isolates, which were classified by Richard Marconi (Rocky Mountain Laboratory, Hamilton, MT) using 16S ribosomal RNA sequence determination as described [11, 24]. The recombinant preparations of OspA and OspB used in this study were purified maltose-binding protein-Osp fusion proteins derived from group 1 strain B31 [25]. These fusion proteins contained the full-length OspA or OspB sequence without the lipid moiety or the signal sequence.

<http://www.ncbi.nlm.nih.gov/pubmed/8106763>

And what are those references, 11 and 24? See the full text at:

<http://www.actionlyme.org/dressler1994.pdf>

10. Adam T, Cassman G. Analysis of *Borrelia burgdorferi* isolates from various sources. *Infect Immun* 1991;59:2579-85.
11. Marconi RT, Lubke L, Hauglum W, Garon CF. Species-specific identification of and distinction between *Borrelia burgdorferi* genomic groups using 16S rRNA-directed oligonucleotide probes. *J Clin Microbiol* 1992;30:628-32.
24. Marconi RT, Garon CF. Development of polymerase chain reaction primer sets for diagnosis of Lyme disease and for species-specific identification of Lyme disease isolates by 16S rRNA signature nucleotide analysis. *J Clin Microbiol* 1992;30:2830-4.
25. Kalish RA, Leong JM, Steere AC. Association of treatment resistant chronic Lyme arthritis with HLA-DR4 and antibody reactivity to

Steere's Dearborn Reference 11: 1992, Marconi and Garon, NIH Bioweapons Lab, Montana--

[J Clin Microbiol](#). 1992 Mar;30(3):628-32.

Species-specific identification of and distinction between *Borrelia burgdorferi* genomic groups by using 16S rRNA-directed oligonucleotide probes.

[Marconi RT](#), [Lubke L](#), [Hauglum W](#), [Garon CF](#).

“Examination of a number of previously published aligned *Borrelia* 16S rRNA sequences revealed the presence of regions which could serve as oligonucleotide probe targets for both species-specific identification of *Borrelia burgdorferi* and distinction between genomic groups. Total cellular RNA isolated from *Borrelia* cultures was used in slot blot analysis. Radiolabeled oligonucleotides designed to hybridize to specific 16S rRNA targets were used as probes. These probes allowed for both species-specific identification and genomic group typing of *B. burgdorferi*...

“... Using *Borrelia* 16S rRNA sequences, we constructed probes that serve to distinguish *B. burgdorferi* from other *Borrelia* species and to distinguish between the genomic groups of *B. burgdorferi*. Other groups have developed *B. burgdorferi* species-specific probes by using polymerase chain reaction amplification (13, 15, 19, 22). We chose rRNA as the target molecule since it is present in large quantities within a cell, so rRNA targets can be considered to be naturally highly amplified. In addition, rRNA molecules are highly conserved and presumably are subject to a very low mutation frequency. The specificity of the probes was demonstrated through the use of slot blots with total cellular RNA as the target. This approach allows the reliable identification and genomic typing of *B. burgdorferi* from cultures, typically within 36 h.

<http://www.ncbi.nlm.nih.gov/pubmed/1372620>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC265123/pdf/jcm00027-0106.pdf>

Steere's Dearborn Reference 24: 1992, Marconi and Garon, NIH:

[J Clin Microbiol](#). 1992 Nov;30(11):2830-4.

Development of polymerase chain reaction primer sets for diagnosis of Lyme disease and for species-specific identification of Lyme disease isolates by 16S rRNA signature nucleotide analysis.

[Marconi RT1](#), [Garon CF](#).

“We have determined and compared partial **16S rRNA** sequences from 23 Lyme disease spirochete isolates and aligned these with 8 sequences previously presented. The 16S rRNA signature nucleotide compositions were defined for each isolate and compared with the genomic species signature nucleotide sets previously established. To identify positions truly indicative of species classification which could serve as targets for polymerase chain reaction species-specific identification primers, 16S rRNA-based phylogenetic analyses were conducted. On the basis of the identified signature nucleotides, we designed polymerase chain reaction primer sets which (i) amplify all spirochete species associated with Lyme disease and (ii) differentiate between these species. The primer sets were tested on 38 *Borrelia* isolates associated with Lyme disease and were found to be sensitive and specific. All Lyme disease isolates tested were amplification positive. These primers allow for the rapid species identification of Lyme disease isolates.”

<http://www.ncbi.nlm.nih.gov/pubmed/1280643> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC270537/>

Steere’s Treatment DNA/RNA Shell Game, synovial (knees) and spinal fluid.

In the first report, knees, Steere finds treatment fails. He is using 3 OspA probes and one **16S rDNA** probe. He finds about 1/3 of the patients were positive with these probes after treatment and concludes longer than 30 days is necessary, as the longer the treatment, the lower the frequency of DNA-positive cases. After he makes these claims, he never again says treatment fails, only that everyone is cured and there are no positive cases after treatment by signing the “Guidelines”:

[N Engl J Med](#). 1994 Jan 27;330(4):229-34.

Detection of *Borrelia burgdorferi* DNA by polymerase chain reaction in synovial fluid from patients with Lyme arthritis.

[Nocton JJ1](#), [Dressler F](#), [Rutledge BJ](#), [Rys PN](#), [Persing DH](#), [Steere AC](#).

”BACKGROUND:

”*Borrelia burgdorferi* is difficult to detect in synovial fluid, which limits our understanding of the pathogenesis of Lyme arthritis, particularly when arthritis persists despite antibiotic therapy.

”METHODS:

”Using the polymerase chain reaction (PCR), we attempted to detect *B. burgdorferi* DNA in joint-fluid samples obtained over a 17-year period. The samples were tested in two separate laboratories with four sets of primers and probes, three of which target plasmid DNA that encodes outer-surface protein A (OspA).

”RESULTS:

”*B. burgdorferi* DNA was detected in 75 of 88 patients with Lyme arthritis (85 percent) and in none of 64 control patients. Each of the three OspA primer-probe sets was sensitive, and the results were moderately concordant in the two laboratories ($\kappa = 0.54$ to 0.73). Of 73 patients with Lyme arthritis that was untreated or treated with only short courses of oral antibiotics, 70 (96 percent) had positive PCR results. In contrast, of 19 patients who received either parenteral antibiotics or long courses of oral antibiotics ($> = 1$ month), only 7 (37 percent) had positive tests ($P < 0.001$). None of these seven patients had

received more than two months of oral antibiotic treatment or more than three weeks of intravenous antibiotic treatment. Of 10 patients with chronic arthritis (continuous joint inflammation for one year or more) despite multiple courses of antibiotics, 7 had consistently negative tests in samples obtained three months to two years after treatment.

“CONCLUSIONS:

“PCR testing can detect *B. burgdorferi* DNA in synovial fluid. This test may be able to show whether Lyme arthritis that persists after antibiotic treatment is due to persistence of the spirochete.

“...In 7 of the 19 patients, *B. burgdorferi* DNA was detected in samples obtained 1 day to 17 months after the completion of antibiotic therapy. Three of these patients were treated with both oral and intravenous antibiotics, two received three weekly doses of intramuscular penicillin G benzathine, and two were given only oral antibiotics. The median duration of their oral treatment was 37 days (range, 20 to 58), and the median duration of intravenous therapy was 14 days (range, 14 to 20). In the remaining 12 patients, samples obtained one day to four years after antibiotic treatment were all negative. Seven of these patients were treated with intravenous antibiotics, two received intramuscular penicillin, and three were given only oral antibiotics. Their median duration of oral treatment was 48 days (range, 21 to 120), and the median duration of intravenous therapy was 30 days (range, 7 to 44). Although the patients with negative PCR results tended to have been treated longer than those with positive PCR results, the differences were not statistically significant. Of 10 patients who had chronic Lyme arthritis despite multiple courses of antibiotic therapy, 7 had negative test results in all post-treatment samples.

“Altogether, of 73 patients with Lyme arthritis who were untreated or treated with short courses of oral antibiotics before testing, 70 (96 percent) had positive PCR results. In contrast, of 19 patients who received either parenteral antibiotics or long courses of oral antibiotics, **only 7 (37 percent) had positive test results after treatment (P<0.001)**. In the 29 patients for whom serial samples were available, all pretreatment samples were positive. Once post-treatment samples became negative, all subsequent samples remained negative.”

<http://www.ncbi.nlm.nih.gov/pubmed/8272083> <http://www.nejm.org/doi/full/10.1056/NEJM199401273300401>

And

[J Infect Dis.](#) 1996 Sep;174(3):623-7.

Detection of Borrelia burgdorferi DNA by polymerase chain reaction in cerebrospinal fluid in Lyme neuroborreliosis.

[Nocton JJ1](#), [Bloom BJ](#), [Rutledge BJ](#), [Persing DH](#), [Logigian EL](#), [Schmid CH](#), [Steere AC](#).

“A polymerase chain reaction (PCR) assay that detects *Borrelia burgdorferi* DNA in cerebrospinal fluid (CSF) was evaluated as a diagnostic test for acute or chronic Lyme neuroborreliosis. In one laboratory, 102 samples were tested blindly, and 40 samples were retested in a second laboratory. In the first laboratory, *B. burgdorferi* DNA was detected in CSF samples in 6 (38%) of 16 patients with acute neuroborreliosis, 11 (25%) of 44 with chronic neuroborreliosis, and none of 42 samples from patients with other illnesses. There was a significant correlation between PCR results and the duration of previous intravenous antibiotic therapy. The overall frequency of positive results was similar in the second laboratory, but concordance between the laboratories and among primer-probe sets was limited because many samples were positive with only one primer-probe set. Thus, PCR testing can sometimes detect *B.*

burgdorferi DNA in CSF in patients with acute or chronic neuroborreliosis, but with current methods, the sensitivity of the test is limited.

“...Previous studies using PCR to detect *B. burgdorferi* DNA in cerebrospinal fluid (CSF) have been done primarily in small numbers of patients with early, acute neuroborreliosis [5-10]. In these studies, which have used several different probes and techniques, the PCR test had sensitivities of 24%-100%. We previously reported that a PCR assay targeting outer surface protein A (OspA) DNA is highly sensitive and specific for the detection of *B. burgdorferi* DNA in joint fluid of patients with Lyme arthritis [11]. We report here on the evaluation of this assay as a diagnostic test for the detection of spirochetal DNA in CSF in a large number of patients with **acute or chronic Lyme neuroborreliosis.**”

<http://www.ncbi.nlm.nih.gov/pubmed/8769624> <http://jid.oxfordjournals.org/content/174/3/623.long>

From this report:

PCR assay. CSF samples from case and control patients were processed simultaneously in a blinded manner, as described [11]. Briefly, DNA was isolated from 100 μ L of CSF, and the DNA extract was resuspended in 30 μ L of ultrapure water. A 5- μ L aliquot from this suspension was amplified with primer-probe set 1, which targets base pairs 788–943 at the 3' end of the 50-kb plasmid of *B. burgdorferi* that encodes *OspA* [11]. This primer-probe set detects most strains of *B. burgdorferi* from New England. A second 5- μ L aliquot was amplified with primer-probe set 2, which targets base pairs 149–343 at the 5' end of the *ospA* gene [11]. This set detects all North American and European isolates tested to date, with the exception of rare natural isolates that lack the 50-kb plasmid. Amplification consisted of 45 cycles of denaturation at 94°C for 45 s, annealing at 50°C for 45 s, and extension at 72°C for 1 min. The cycles were preceded by a 4-min phase at 94°C and followed by a final 7-min phase at 72°C. Amplified products were resolved by 4% agarose gel electrophoresis and blotted onto nylon membranes, which were hybridized with the appropriate ³²P–5' end–labeled internal oligonucleotide probe [11] and exposed to film for 4–72 h.

You can see that Steere does not use the 16S RNA probe in this Neuroborreliosis assessment, whereas he had before in knees and found more than a third of the patients were positive after treatment. The *OspA* gene was seen more frequently in those arthritis knees patients probably due to the exosomes or blebs, which contain DNA (Dorward, 1990, PMID: 16348232). And as shown by Pachner, above, using a human neuroinvasive strain (N40 was taken from the spinal fluid of a human patient), the plasmids change. It is quite evident that Steere does not want to find Neuroborreliosis. He referenced Garon and Marconi's work and recommendations for using the 16S probe to assure the Dearborn strains were *burgdorferi*. Steere used DNA he knew would not likely be found in human brains to falsely show that people are not infected with spirochetes, the CDC's goal from the beginning. Not every strain of *Borrelia* has *OspA*, and one should use DNA that detects the GENUS, or at least more of the common species, flagellin or the spacer DNAs.

The CDC deployed Allen Steere in the first place – a rheumatologist -- to manage a vector-borne, neurologic disease? This never made any sense unless the CDC wanted to spin it from the beginning,

once they found out, “Oops (Plum Island).” Nevertheless, here in these two reports, despite the shell game, Steere found treatment fails at least a third of the time in both knees and spinal fluid. Yet, he never mentioned this again and signed the “IDSA Guidelines” that state that spirochetes do not persist after 2 weeks to 30 days.

Surely by the late 1980s, this Cabal knew antibiotic treatment failed because spirochetes cause damage via activating Epstein-Barr and the like, and that no spirochetal infection has ever been “curable,” or eradicable. **All of their nonsense is about the OspA vaccines causing the same immunosuppression-and-not-arthritis disease and that they performed the Dearborn stunt to hide this fact.**

X. The Guidelines – Who signed on to this perverted science and are therefore responsible for endorsing this fraud?

The IDSA “Guidelines” are based on the Dearborn-Falsified case definition, Klempner’s 2001, bogus “re-treatment” “study” where Klempner neglected to mention to the NEJM – who did not catch this flaw – that he used OspA primers to detect “No Lyme” (yet found some and rejected them from the study, but did not report this), knowing OspA changes, knowing not all borrelia are bearers of OspA, and knowing that Lyme was incurable since he had published that it was in the past?

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.

Wormser GP, Dattwyler RJ, Shapiro ED, Halperin JJ, Steere AC, Klempner MS, Krause PJ, Bakken JS, Strle F, Stanek G, Bockenstedt L, Fish D, Dumler JS, Nadelman RB.

<http://www.ncbi.nlm.nih.gov/pubmed/17029130>

Search the references for “Klempner” in the above report. You will see the basis of the “Guidelines” is Klempner’s research-fraud article on the non-retreatment of 2/3rds of his victims, using the falsified case definition and a bogus psychiatric check-list instead of the Cabal’s own valid biomarkers.

Notice that the authors say:

”Another study similarly was unsuccessful in recovering *B. burgdorferi* from the blood of 12 patients with chronic post-Lyme disease symptoms, using both conventional and hypertonic media (M.S.K., unpublished data) [288]. The latter study also cultured 128 CSF specimens for *B. burgdorferi* and evaluated blood specimens and CSF specimens by PCR. None of the 843 specimens tested in total was either culture or PCR positive [288, 289]. Therefore, the most plausible explanation for the positive results using the novel blood culture method reported by a single group of investigators [303] is that the microscopic findings were not, in fact, due to *B. burgdorferi*.

“In another study, *B. burgdorferi* DNA was detected by PCR in urine samples of 74.2% of 97 United States patients who were diagnosed as having “chronic Lyme disease” and who were previously treated with antibiotics for extended periods of time [306]. Few additional details were provided by the authors as to the characteristics of the patient population. ****Because the authors did not sequence the amplicons to confirm their identity, the results should be regarded as questionable in the absence of confirmation by other investigators.****”

Klempner in his bogus non-retreatment article (Ref 288) used bogus OspA (which were not listed, one had to dig and find he used OspA) primers, *and this entire criminal gang is guilty of the same thing – not sequencing for Borrelia DNA or using proper primers* (Wormser in Missouri, for instance). Wormser just used bogus probes of DNA not necessarily expected to be there (*burgdorferi* Fla and a specific *lonestari* enzyme, knowing there were plenty of other borrelia in ticks in Missouri, as shown above by Telford, Schoen, and Barbour).

This proves Wormser, et al, know they should have done the same thing. **All their own bogus articles have to be retracted in addition to the Cabal’s prosecution.**

And the 2001 “Guidelines” signers:

<http://www.guideline.gov/content.aspx?id=9537>

Guideline Title

Infectious Diseases Society of America practice guidelines for clinical assessment, treatment and prevention of Lyme disease, human granulocytic anaplasmosis, and babesiosis.

Bibliographic Source(s)

Wormser GP, Dattwyler RJ, Shapiro ED, Halperin JJ, Steere AC, Klempner MS, Krause PJ, Bakken JS, Strle F, Stanek G, Bockenstedt L, Fish D, Dumler JS, Nadelman RB. The clinical assessment, treatment, and prevention of lyme disease, human granulocytic anaplasmosis, and babesiosis: clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis. 2006 Nov 1;43(9):1089-134. PubMed

Guideline Status

This is the current release of the guideline.

This guideline updates a previous version: Wormser GP, Nadelman RB, Dattwyler RJ, Dennis DT, Shapiro ED, Steere AC, Rush TJ, Rahn DW, Coyle PK, Persing DH, Fish D, Luft BJ. Practice guidelines for the treatment of Lyme disease. Clin Infect Dis 2000 Jul;31(Suppl 1):1-14.

The guideline was reaffirmed for currency by the developer in 2010.

What else can you say? The Cabal and the CDC do not want anyone to discover a Borreliosis infection much less treat it. If you understand that the disease is far more devastating than a common infection with

a common bacteria – it's functionally and physiologically like Post Sepsis Syndrome -, there really is no treatment for it at this time. But there is the threat that these walking cesspools of disease called humans with Lyme or Chronic Fatigue Syndrome might just be the cauldron from which emerges the next pandemic. There are a million such new cases of Tick Bite Sepsis (Disability) per year in the USA alone.

It's going to be pretty bad Karma to abuse and neglect (Deprivation of Rights Under Color of Law) the very sick, one way or another. Either by natural disaster or the other nations will decide to boycott American businesses and especially ban the CDC staff from traveling to their countries. Are they not criminal terrorists? Who else but the NAZIs and the Japanese during World War II repeatedly and blatantly experimented on races and or with bioweapons?

Right. The CDC.

Tuskegee.

Guatemala.



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CHARGE SHEET 5: OCCAM'S RAZOR

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

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Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 5: Occam's Razor

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 AC](#)1,2, [Strle F](#)3, [Wormser GP](#)4, [Hu LT](#)5, [Branda JA](#)6, [Hovius JW](#)7, [Li X](#)8, [Mead PS](#)9.

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 immunosuppression:

[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- κ B, 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- κ B 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 head's 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 be 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 that 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), won't 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 bearers 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 immunosuppression 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 Neurological 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 are 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"), [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 [sic] 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 monoarticular 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 it 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 LA](#), [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 a 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 LYMERix 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 TLR

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 2/3 of his victims had never had IV ceftriaxone before—the standard of care at the time—and which was assessed with the non-scientifically valid FIQ or Fibromyalgia Impact Questionnaire ("questionnaires" 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.

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 rendering 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.

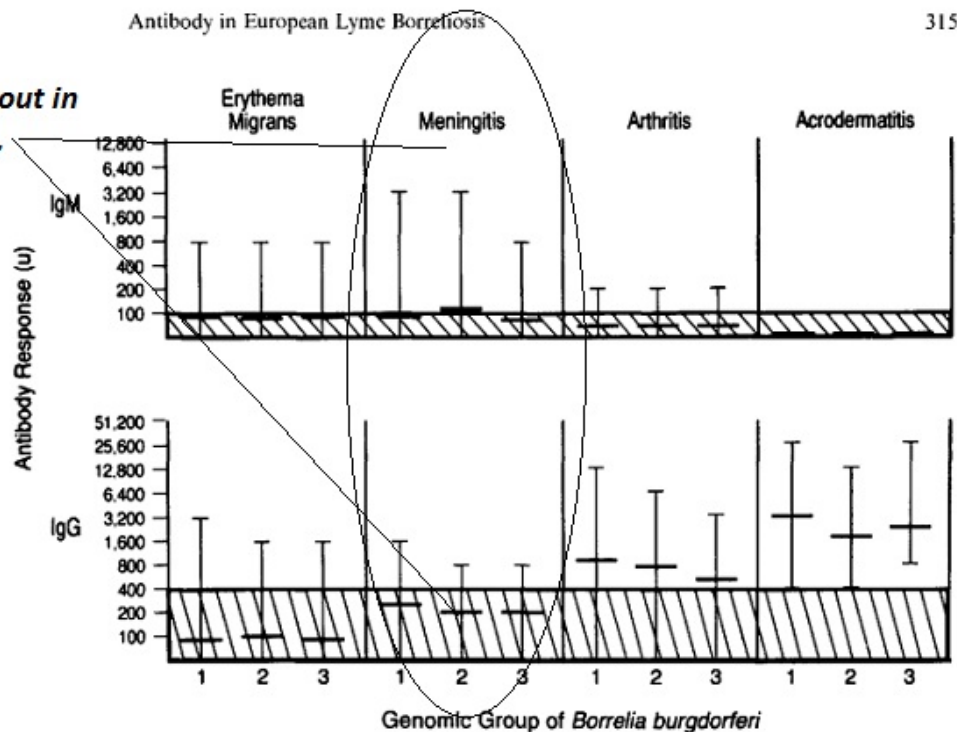
JID 1994;169 (February)

Antibody in European Lyme Borreliosis

315

NeuroLyme is screened out in the first step, the ELISA, via research fraud.

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 Endotoxin 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 "autoimmunity" (they all basically mean the same thing). If you wanted to create a biowepson 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 2/3cure 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 Klemmner 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 (Klemmner, 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 Lyme victims looked 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:

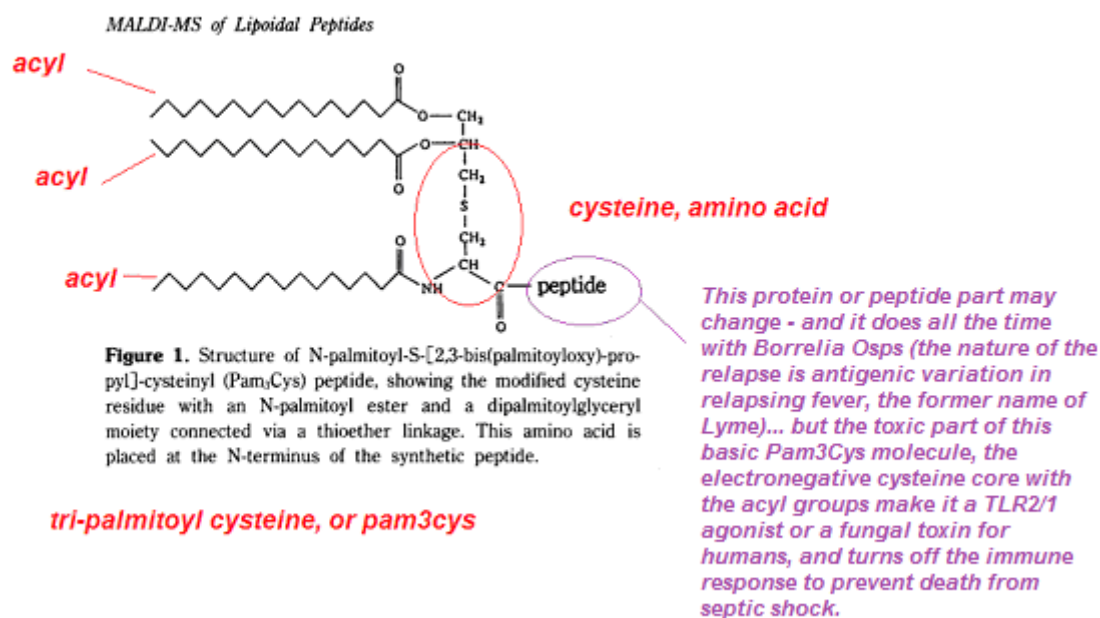
"It's the perfect stealth bacteria, says one frustrated physician. He's talking about *Borrelia burgdorferi*, the bacterium that causes Lyme disease. This illness, which is often mistaken for diseases ranging from multiple sclerosis to Lupus, can inflict excruciating headaches and muscle pain, affect the brain and nervous system, attack major organs, and inflame joints..."-- [Energy Science News, pn1.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):



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

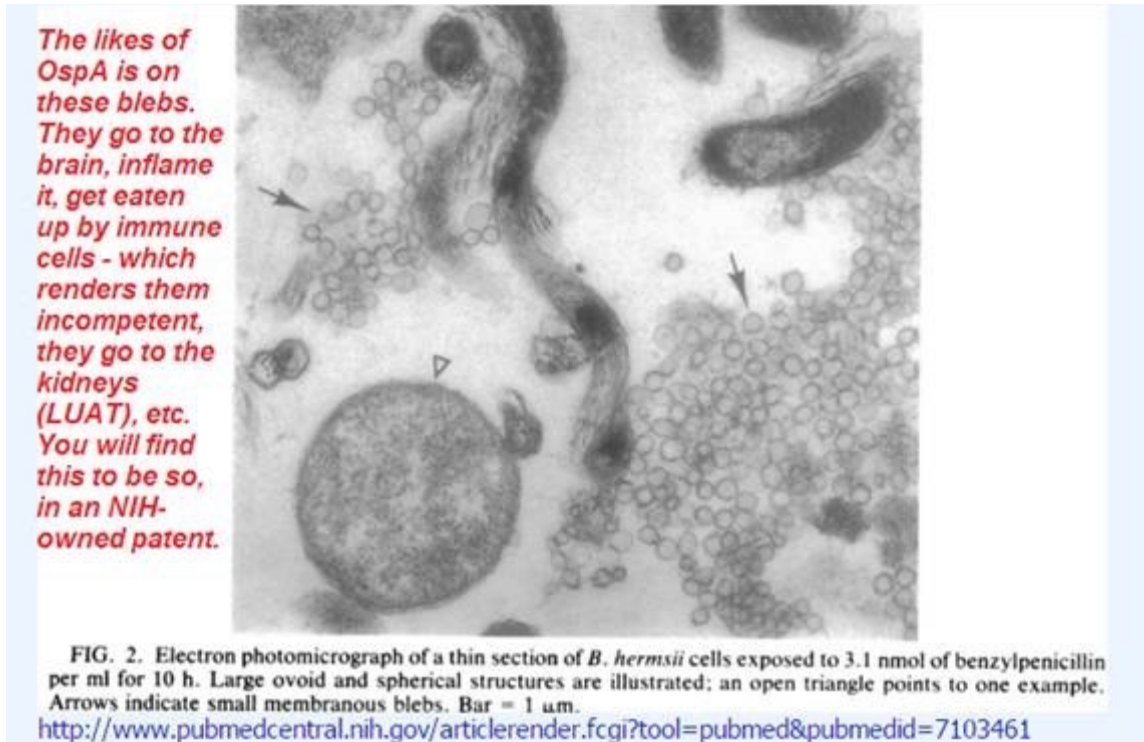
http://newjournal.kcsnet.or.kr/main/j_search/j_download.htm?code=B961118

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:



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=%2Fmetahtml%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](https://doi.org/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 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* lysate^{22,30,33–35,39}. 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 (REFS^{23,32}) 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 independent^{18,19,21}.

"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 also 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 Omgs, 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 NI](#), [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 Autoimmunity. 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 [sic] 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/transcript/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 chronic Lyme 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-type immune 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 $\geq 50\%$ 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 superimagine 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 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.”

<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 animal 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 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 make 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-cludes 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.

Returning to the alphabet:

L. Adriana Marques, formerly of NINDS' MS-Lyme group and who now works for NIAID, and specializes only in Lyme and 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 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](https://doi.org/10.1371/journal.pone.0098819). 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 subsets in some target organs, as well as in the cerebrospinal fluid (Szyfelbein and Ross 1988). Usual bacterial infections do not produce such lymphocytic infiltrates in tissue. These immunoblastoid cells in Bb infections at times resemble those found in Epstein-Barr virus infections. Does Bb reactivate latent virus infections in tissues? Do some tick inocula harbor simultaneous infectious agents (ixodid ticks can harbor Rickettsiae, Babesia microti, and Ehrlichia bacteria, in addition to Bb), producing multi-agent infections in some hosts? Further studies can clarify these issues by means 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 carinii, 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 adhered 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 TNF α is reduced in U937 cells infected with *M. fermentans*...

”In many apoptosis scenarios, including TNF-mediated apoptosis, the mitochondrial inner transmembrane potential (ψ_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 ψ_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'-dihexyloxycarbocyanine iodide (DiOC₆ (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 brainscramble 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 1:47 (?) 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)- κ B activation, p38 phosphorylation and CXCL8 gene expression, while PTPN22 ablation upregulated LPS-induced p65 NF- κ B and p38 phosphorylation and the expression of TNF- α and pro-IL-1 β 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 Voila! what do you know, immunosuppression from malaria seems to be associated with EBV-associated Burkitt's Lymphoma. Synergy. Another kind of in-parallelism to our model. One infection invites the other, such as when in the old days it was known that 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 Flu, 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 of 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 NIID. 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 NIID, 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 United States of Med for Morons 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 (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- κ B 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-1 β . The pre-treatment with TNF- α and IL-1 β 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](#), [Cześćcik 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-1b, 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 say 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.

[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 **Stelara 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 he 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 sulfhydryl 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 through 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 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 Klempner 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](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 immunosuppression drugs they must take)

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

Poliomyelitis primarily via the fecal-oral route

Polio-like syndrome found in children who tested positive for enterovirus 68. [23][24]

Nonspecific febrile 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 of aseptic 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 disease or epidemic pleurodynia is characterized by severe paroxysmal pain in the chest and abdomen, along with fever, and sometimes nausea, headache, and emesis.

Pericarditis and/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 conjunctivitis can 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 disease is a childhood illness most commonly caused by infection by Coxsackie A virus or EV71.

Encephalitis is a 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 in chronic fatigue syndrome. [25]

Diabetes mellitus type 1 It 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-income reasons? Has this scam gone on so long the CDC and NIH find no way of backing away from all their lies? Is the HHS.gov mortified at the prospect of having been discovered to be 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.

<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.

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.



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healthcare justice

CHARGE SHEET 6: COMMON MECHANISMS

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

© 2017, February, Society for the Advancement of Scientific Hermeneutics, (\$A\$H).

Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 6: Common Mechanisms

Criminal Charge Sheet #6) Common Mechanisms in ME/CFS and the Brain Damage we call Autism (and Chronic Lyme disease or post-sepsis syndrome)

- 1.) **Thimerosal** is used to prevent immune suppressing fungal antigens like LYMERix because such a condition activates viruses. Such TLR2 agonists are bad for children’s developing brains. Suzanne Vernon’s research fraud on mycoplasma. (Pg 2)
- 2.) Borna virus and other live viruses are accepted to be the models of the brain damage we call Autism (Plotkin). (Pg 3)
- 3.) It's well known that **measles causes immunosuppression**, **Auwaerter** says (confirming the idea of synergy or dual or multiple infections causing immunosuppression or being the result of immunosuppression or vaccine contamination with especially fungal antigens), and Auwaerter says measles, etc. may take months to manifest. (Pg 7)
- 4.) Adverse events related to reactivated brain damaging viruses are not recorded in the safety and efficacy calculations. Children are NEVER followed in these **MMR qualifications** for more than 3 weeks. A **book** on vaccine safety shows they are **officially throwing out data** on vaccinating children and when the vaccines revert back to wild type (which happens easily since viruses mutate for a living); says they are looking at children **up to 5 years later** (which does not happen in the official “safety and efficacy” studies) and finding the vaccine strain as a cause of illness. (Pg 10)
- 5.) CDC says vaccines fail by giving the victims the actual viruses; don’t vaccinate immunosuppressed kids. (Pg 16)
- 6.) Pharma and others says vaccines fail by giving people the live reactivated brain damaging viruses. (Pg 17)
- 7.) CDC and others say people can get animal vaccine diseases, particularly if the animal or the human are immunosuppressed (but no one is allowed to talk about immunosuppression, are they?) (Pg 18)
- 8.) **Cortisol** as a mechanism of virus reactivation – CDC. (Pg 20)
- 9.) IDSA actually publishes that vaccines are not safe and not properly vetted. (Pg 21)
- 10.) Offit and Shapiro reveal the prevailing lies, slander, libel, verbal violence are about hiding the mechanisms of immunosuppression. (Pg 22)
- 11.) Hepatitis B and the vaccine HbsAg cause immunosuppression. (Pg 24)
- 12) Chicken Pox vaccine reactivated via immunosuppression, contrary to “exposure to wild type” claims. (Pg 25)

- 13.) Growth of cancer rate in children follows hypervaccination schedule. (Pg 26)
- 14.) “Over-vaccination” and the danger of producing a pandemic. (Pg 28)
- 15.) Rubella and “low responders” having the actual “viremia,” spreading the virus. (Pg 29)
- 16.) Synergism, ME/CFIDS and Burkitt’s Lymphoma in Africa, hmmm. (Pg 31)
- 17.) Seronegative Epstein-Barr. (Pg 42)
- 18.) “Our Best Frenemy” means “Be careful using OspA and other fungal antigens because they inhibit apoptosis and cause immunosuppression.” (Pg 46)
- 19.) What about Diagnostics? Thank IDSA for their recommendations :D (Pg 47)

This report is actually just a continuation of the Occam’s Razor Criminal Charges Sheet: the mechanisms of post-sepsis syndrome, fungal exposures, how fungal antigens cause immunosuppression, how there are no antibody markers for the diseases set we are talking about, and how Chronic Fatigue/ME and Fibromyalgia are essentially the same as Post Sepsis syndrome, with or without a tick bite. In Lyme, spirochetes are not what is causing the disease except for the initial immunosuppression event. It is the secondary opportunistic, like the fatigue-causing reactivated herpes viruses, the TLR2/1 agonist-bearing, fatigue-causing mycoplasma, and the like. However, there are a few independent data sets regarding Chronic Fatigue Syndrome that are worth reviewing.

But let’s start with the very first three things everyone should know:

1) 2012, Dec, *New York Times*; Doctors admit Thimerosal is put in vaccines to prevent fungi:

Vaccine Rule Is Said to Hurt Health Efforts

"But a proposal that the ban include thimerosal, which has been used since the 1930s to prevent bacterial and **fungal contamination** in multidose vials of vaccines, has drawn strong criticism from pediatricians.... They say that the ethyl-mercury compound is critical for vaccine use in the developing world, where multidose vials are a mainstay...Banning it would require switching to single-dose vials for vaccines, which would cost far more and require new networks of cold storage facilities and additional capacity for waste disposal, the authors of the articles said."

http://www.nytimes.com/2012/12/17/health/experts-say-thimerosal-ban-would-imperil-global-health-efforts.html?_r=2&

2) **Borna virus is a model of the “neurodevelopmental brain damage” we call Autism.** That is, a live virus infection which no doubt is responsible for the inflammation, SSPE, SIDS (warned about in the MMR monograph), is what does the damage; the active viruses destroy neurons, etc.

[Proc Natl Acad Sci U S A](#). 1999 Oct 12;96(21):12102-7.

An infection-based model of neurodevelopmental damage.

[Hornig M1](#), [Weissenböck H](#), [Horscroft N](#), [Lipkin WI](#).

“Perinatal exposure to infectious agents and toxins is linked to the pathogenesis of neuropsychiatric disorders, but the mechanisms by which environmental triggers interact with developing immune and neural elements to create neurodevelopmental disturbances are poorly understood. We describe a model for investigating disorders of central nervous system development based on neonatal rat infection with Borna disease virus, a neurotropic noncytolytic RNA virus. Infection results in abnormal righting reflexes, hyperactivity, inhibition of open-field exploration, and stereotypic behaviors. Architecture is markedly disrupted in hippocampus and cerebellum, with reduction in granule and Purkinje cell numbers. Neurons are lost predominantly by apoptosis, as supported by increased mRNA levels for pro-apoptotic products (Fas, caspase-1), decreased mRNA levels for the anti-apoptotic bcl-x, and in situ labeling of fragmented DNA. Although inflammatory infiltrates are observed transiently in frontal cortex, glial activation (microgliosis > astrocytosis) is prominent throughout the brain and persists for several weeks in concert with increased levels of proinflammatory cytokine mRNAs (interleukins 1alpha, 1beta, and 6 and tumor necrosis factor alpha) and progressive hippocampal and cerebellar damage. The resemblance of these functional and neuropathologic abnormalities to human neurodevelopmental disorders suggests the utility of this model for defining cellular, biochemical, histologic, and functional outcomes of interactions of environmental influences with the developing central nervous system.”

<https://www.ncbi.nlm.nih.gov/pubmed/10518583> <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC18419/>

More:

<https://www.ncbi.nlm.nih.gov/pubmed/?term=borna+virus+and+autism>

The rubella vaccines were invented in the first place because rubella was known to cause “congenital Autism.”

Stanley Plotkin article, next, and others show that rubella causes immunosuppression, infected infants shed the live viruses and give them to other people,.... while the CDC et al deny this, and say the people are not getting the viruses from the vaccinated person, but some other wild type strain (even though they are they same strain). And also, people taking immunosuppressing drugs are told not to be near someone “recently vaccinated.”

Plotkin, 1975:

[Am J Dis Child](#). 1975 Apr;129(4):444-9.

Routes of fetal infection and mechanisms of fetal damage.

[Plotkin SA](#).

“... Once the rubella virus infects the fetus, a chronic, nonlytic infection is established. This was first demonstrated in vitro.²⁷ Infection of strains of human fibroblasts, once established, persists for weeks or months in stationary cultures. When the cell cultures are placed in fresh vessels under conditions that allow uninfected control cells to divide, mitotic inhibition is observed. Rubella virus carrier cultures derived from congenitally infected infants exhibit decreased cell division rate, and are not susceptible to cure with antibody. They also show resistance to superinfection not mediated by Interferon.²⁸ Crucial evidence was added when the number of cells in fetal organs was measured. There was a 50% decrease in rubella-infected fetuses compared to controls.²⁹ The possibility that this inhibition of cell division is mediated by a soluble protein was suggested.³⁰

“Four additional mechanisms of fetal damage by rubella virus remain to be considered. First, it seems certain from histologic examination of the brain and the organ of Corti that much rubella damage is vascular in origin. Damage to endothelial cells leads to thrombosis of small blood vessels and surrounding tissue necrosis.³¹

“Second, some cells, particularly those in the lens of the eye, are probably killed by rubella virus.

”Third, study of rubella carrier cell cultures from aborted fetuses shows increased incidence of chromatid breaks. Specific chromosomal anomalies in fetuses with rubella syndrome have been reported or suggested,³² but the evidence that chromosomal abnormalities are a cause of rubella anomalies is not compelling.

“Fourth, there are many parts of the rubella syndrome that are the direct result of persistent infection. Among these are the encephalomeningitis, which often continues during the first year of life³³; the cataracts, which may grow worse after birth and in which the virus survives for years³⁴; the postnatal hepatitis; the thrombocytopenia, which is partly due to megakaryocyte destruction and which eventually resolves after birth; the pneumonias, which occur in the early months of postnatal life; the myocarditis, which may be present at birth³⁵; and the osseous lesions.

The relationship between the persistent virus carrier state and function of the immunologic system is difficult to resolve, as the facts are somewhat confusing. **It is clear that (1) lymphocytes of normal individuals can be infected in vitro and show decreased phytohemagglutinin (PHA) response after infection³⁶; (2) lymphocytes from infants with rubella syndrome often carry virus for long periods after birth³⁷; (3) infants with congenital rubella syndrome usually have high titers of rubella antibody,³⁴ particularly of the IgM type; (4) humoral antibody responses to antigens such as diphtheria toxoid, tetanus toxoid, blood group antigens, and types 1 and 3 poliovirus are decreased in infants with rubella syndrome when they are excreting virus, but not after they stop.**

“Just recently, absence of cell-mediated immunity to rubella was demonstrated in nine of 12 infants with rubella syndrome.³⁸ One can formulate an explanation for viral persistence in the following way: 1. Antibody-forming cells (B lymphocytes) are only partly damaged in infants with rubella syndrome. 2. Thymus lymphocytes are themselves infected with the virus, do not go into mitosis, and therefore have reduced competence to destroy infected cell clones. The occasional

defective PHA response, the relative immune defects, and the slow conversion from IgM to IgG antibody would be explained by damage to lymphocytes. 3. Antibody to rubella, secreted by uninfected lymphocytes, is stimulated in utero without the development of tolerance. When uninfected clones of lymphocytes become available, they attach to and destroy infected cells, releasing virus that is then neutralized by the secretions of lymphocytes.

“It is difficult, however, to reconcile the absence of cell-mediated immunity to rubella in those infants with rubella syndrome who no longer excrete the virus. Since the Interferon response remains intact in infants with rubella syndrome, persistence cannot be explained by failure of this mechanism. Patterns of acquisition of cytomegalovirus (CMV) antibodies vary from early seropositivity in many developing countries, to slow seroconversion with a high percentage of susceptible child-bearing women in urban centers of industrialized countries. ...

”... **Fetal brain damage may also result from selective lysis of dividing cells. The best example of this is provided by the H-1 picornavirus, which destroys the cerebellar granular cells in immature cats or hamsters.**⁴⁸ Other mechanisms that have been shown to operate in animals, but not yet in man, are alteration of neural tube closure by influenza virus, or cavitation of the brain through cell destruction in bluetongue disease of sheep. Recent support for the supposition that influenza A2 virus is teratogenic was provided by the development of hydrocephalus in monkeys inoculated intracerebrally with this virus during the fetal state.⁴⁹ **Thus, some viruses may be capable of destroying certain brain cells during fetal development, leaving behind morphological derangement without inflammation.”**

<https://www.ncbi.nlm.nih.gov/pubmed/165711>

Picornavirus, he says, is a model of the brain damage we call Autism. The vaccines are the live, attenuated viruses. We just wanted to make sure everyone knew that live viruses are the model for the brain damage, and that the rubella vaccine was invented specifically to prevent Autism. It seems most people don't know this.

The CDC claims something to the effect that “the increasing Autism rate could be due to something in the environment,” not mentioning which environment. The child's body with viruses from vaccines, congenital CMV, congenital other herpes, the contaminated vaccine vial - contaminated with fungal antigens? No one ever assesses the immune status of the child prior to MMR vaccination, and no one is instructed as to how this testing should be done. The MMR monograph merely claims immunosuppressed children should not be vaccinated.

Thimerosal is put in vaccines to prevent LYMERix, or the immune suppressing fungal endotoxin, OspA and here next we see fungi or fungal antigens injected into babies could be bad, but there is no way to assess the status of the MMR batch or individual vials for mishandling or contamination:

Brain Behav Immun. 2015 Aug;48:301-12. doi: 10.1016/j.bbi.2015.04.020. Epub 2015 May 27.

Postnatal TLR2 activation impairs learning and memory in adulthood.

Madar R1, Rotter A1, Waldman Ben-Asher H2, Mughal MR3, Arumugam TV4, Wood WH 3rd3, Becker KG3, Mattson MP5, Okun E6.

"Neuroinflammation in the central nervous system is detrimental for learning and memory, as evident from epidemiological studies linking developmental defects and maternal exposure to harmful pathogens. Postnatal infections can also induce neuroinflammatory responses with long-term consequences. These inflammatory responses can lead to motor deficits and/or behavioral disabilities. Toll like receptors (TLRs) are a family of innate immune receptors best known as sensors of microbial-associated molecular patterns, and are the first responders to infection. TLR2 forms heterodimers with either TLR1 or TLR6, is activated in **response to gram-positive bacterial infections, and is expressed in the brain during embryonic development. We hypothesized that early postnatal TLR2-mediated neuroinflammation would adversely affect cognitive behavior in the adult. Our data indicate that postnatal TLR2 activation affects learning and memory in adult mice in a heterodimer-dependent manner. TLR2/6 activation improved motor function and fear learning, while TLR2/1 activation impaired spatial learning and enhanced fear learning.** Moreover, developmental TLR2 deficiency significantly impairs spatial learning and enhances fear learning, stressing the involvement of the TLR2 pathway in learning and memory. Analysis of the transcriptional effects of TLR2 activation reveals both common and unique transcriptional programs following heterodimer-specific TLR2 activation. These results imply that adult cognitive behavior could be influenced in part, by activation or alterations in the TLR2 pathway at birth."

<http://www.ncbi.nlm.nih.gov/pubmed/26021559>

Research Fraud by CDC officer Suzanne Vernon – trying to make it appear that mycoplasma or global immunosuppression is not a factor in Chronic Fatigue Syndrome/Myalgic Encephalomyelitis:

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>

Vernon committed research fraud by centrifuging out the very cells to which mycoplasma adhere and then said, **“How Amazing! There is no mycoplasma here!!”** This is important because it shows that the CDC does not want to admit to the mechanisms of immunosuppression especially via fungi (no

antibodies, increased susceptibility to reactivating latent viruses) because that betrays the source of the Autism pandemic. The same thing is happening when you inject a child with a live attenuated vaccine that is contaminated with fungal antigens.... And against which Thimerosal was added to vaccines as a “preservative.”

“Preservative” means “prevent the likes of fungal mycoplasmal antigens or LYMERix growing in vaccine vials with mercury.”

3) Johns Hopkins’ Paul Auwaerter says vaccines fail by becoming reactivated - by reverting to the virulent, active form; that measles itself causes immunosuppression (confirming the synergy with multiple infections); and that the virus symptoms occur “months later” (whereas none of the MMR qualifications followed these children for any of these outcomes much less more than a few weeks):

3.A) J Virol. 1999 Oct;73(10):8791-7.

Altered virulence of vaccine strains of measles virus after prolonged replication in human tissue.

Valsamakis A1, Auwaerter PG, Rima BK, Kaneshima H, Griffin DE.

“...Our data suggest that the adverse outcomes associated with immunization of patients suffering from congenital and acquired immunodeficiency syndromes are due to the emergence of an MV strain with increased virulence in a host unable to mount a sufficient immune response to clear the originally inoculated vaccine virus. This situation is mimicked in the SCID-hu mouse. Sequence analyses of pMor-1 H and M and other isolates derived from immunodeficient patients demonstrate that these human tissue-passaged vaccine isolates are highly related to parent vaccine strains ([1](#), [15](#)).

“...However, fatal infections have been documented in immunodeficient children vaccinated with these strains ([1](#), [12](#), [14](#), [15](#)). **The symptoms of infection occur many months after immunization, and the viruses isolated are similar to the original LA [live, attenuated] vaccine ([1](#), [15](#)), suggesting that in the absence of an effective host immune response, persistent infection with the vaccine strain can lead to fatal disease. Viruses isolated from these children could potentially represent virulent revertants of the original LA vaccine.**”

<https://www.ncbi.nlm.nih.gov/pubmed/10482633>

Fatal disease or disabling, like, with brain damage (“Autism”), ya mean, right, Paul?

3.B.) Paul Auwaerter Also Says (about how measles causes immunosuppression and that the fatal brain infections can come from the vaccines)...

“Increased virulence of **vaccine strains isolated from immunocompromised infants with fatal infections** was not evident.” (Just regular virulence is good enough.)

[J Infect Dis](#). 1999 Oct;180(4):950-8.

Measles virus infection in rhesus macaques: altered immune responses and comparison of the virulence of six different virus strains.

[Auwaerter PG1](#), [Rota PA](#), [Elkins WR](#), [Adams RJ](#), [DeLozier T](#), [Shi Y](#), [Bellini WJ](#), [Murphy BR](#), [Griffin DE](#).

“Measles remains a major cause of childhood mortality, with questions about virus virulence and pathogenesis still requiring answers. Rhesus macaques were infected with 5 different culture-adapted strains of measles virus, including 2 from patients with **progressive vaccine-induced disease**, and a sixth nonculture-adapted strain, Bilthoven. All caused infection detectable by reverse transcriptase-polymerase chain reaction and induction of antibody. Chicago-1 and Bilthoven induced viremias detectable by leukocyte cocultivation. Bilthoven induced Koplik's spots, conjunctivitis, and rash. Lymphopenia and depressed interleukin (IL)-2 production were followed by monocytosis and eosinophilia. **All monkeys, including 41 involved in a primate facility outbreak, showed suppressed responses to phytohemagglutinin.** As the rash resolved production of IL-2, IL-1beta, tumor necrosis factor-alpha, IL-6, and IL-5 mRNA increased. Monkeys are useful for studies of measles immunopathogenesis, but virus strains must be carefully chosen. Increased virulence of vaccine strains isolated from immunocompromised infants with fatal infections was not evident.

“Measles is an important human disease that causes the death of ~1,000,000 children each year. Most of these deaths are due to secondary infections [1]. This increase in susceptibility to other pathogens is associated with a well-documented measles-induced immunosuppression [2]. This suppression of immune responses is incompletely understood and is probably multi-factorial: it is likely that different mechanisms are of primary importance in early and late phases of infection. Human studies of necessity focus on the time of the appearance of the rash and thereafter, because that is when measles is recognized clinically. Studies in primates offer the opportunity to look at all phases of infection.

“Many of the deaths associated with secondary infection could be prevented by more widespread application of measles immunization. The vaccine against measles is a live attenuated virus with an impressive record of efficacy and safety, although suppression of immune responses is often detectable after immunization [3]. ...”

<https://www.ncbi.nlm.nih.gov/pubmed/10479117>

Notice he says:

The latest thing Auwaerter said this: **“This suppression of immune responses is incompletely understood and is probably multi-factorial: it is likely that different mechanisms are of primary importance in early and late phases of infection. Human studies of necessity focus on the time of the appearance of the rash and thereafter, because that is when measles is recognized clinically.”**

So, inject 3 or more live, allegedly attenuated viruses into infants with immature immune systems, several of which are known to cause immunosuppression, and we know what happens in immunosuppression – reactivated viruses.

Auwaerter is also suggesting there is the non-spots form of the disease, or a non-inflammatory form of the disease. The Lyme criminals say “you cannot have a disease without inflammation,” or that “there is no disease other than autoimmune,” when we know the opposite is the most damaging outcome: live viruses and infections without immunity. See the quotes by Eugene Shapiro and Paul Offit, below where they reveal this “policy.” A woman was vaccinated with immunosuppressing Hep B and then developed MS, yet Offit claims, how “ironic,” basically, “since she had no immune response to the vaccine” (implying therefore, she could not possibly have a disease). It’s not ironic, it’s a **fact** and a phenomenon they’re trying to hide with snarkasm, harassment of their victims and research fraud (CDC’s Vernon, Lyme criminals, and here, in the MMRs, where the CDC and BigPharma throw out vaccine failure or injury data just as they did with LYMERix, claiming those injury/failure cases were “Unconfirmed Lyme”).

Next: How convenient for Auwaerter to pretend to know nothing about fungal diseases/antigens or spirochetes that shed them, **and the fact that they cause immunosuppression**. His office literally returned a phone call to us saying, “No, Auwaerter does not know what OspA is.”

[Medscape Infectious Diseases](#) > [Auwaerter on Infectious Diseases](#)

COMMENTARY

Candida auris: Time to Prick Up Your Ears?

Paul G. Auwaerter, MD

“Hello. This is Paul Auwaerter, speaking for Medscape Infectious Diseases and from Johns Hopkins University School of Medicine.

“*Candida* and antifungal resistance, to **me as a nonmycologist**, seems relatively static...”

<http://www.medscape.com/viewarticle/872986>

Remember from the Occam’s Razor, what was unique about Paul Auwaerter was that he claimed on his webpage to have expertise in 2 areas: Lyme and EBV. Curious enough that he excludes this work he did on why the MMR vaccines fail. Auwaerter insists the Cabal is right, and that Lyme is only an autoimmune bad knee and that the post-sepsis Lyme outcome is due to some frail emotional status. Yet here we find him in 1999 reporting on how you should not vaccinate immunosuppressed people with live, attenuated viruses because those viruses could become reactivated (and clearly they did—they were the same “type”). So, while we have claimed that the reason the Cabal and the CDC do not want to admit to immunosuppression/post-sepsis outcomes as the actual diseases of Lyme, CFIDS, Fibro, etc., here we finally have the first proof that our theory was correct. The lies about Lyme and ME/CFS/Fibro have to do with how the pediatric vaccines fail and give these children the very brain damaging viruses that they claim to prevent—immunosuppression/sepsis.

Auwaerter also reveals two other aspects of these simultaneous scandals: the vaccine brain damaged children are not followed officially, ever, for more than a few weeks in the “safety and efficacy” studies. Secondly, it is very likely the only “adverse events” signs the pediatricians are allowed to report are the “autoimmune” ones, like rashes. We proposed there is a data set somewhere (hidden) of children known to have been vaccinated while immunosuppressed with each live viral vaccine type, which were excluded from the “safety and efficacy” results because the CDC and BigPharma would say, “Oh, well those children should not have been vaccinated in the first place, so we can’t count them.” And indeed at the end of February, 2017, we found that proof (below).

No Autism groups are asking for or showing the correct data. Most of them are on the Thimerosal-Go-Round—nowhere.

Thimerosal was put in vaccines to prevent LYMERix, really. Exactly. And no vaccine against spirochetal diseases ever prevented spirochetes. The Cabal does not even technically make this claim. They just say OspA or LYMERix creates antibodies, which is false. *The previous lawsuit against SmithKline-Beecham (GSK) was about not warning or pre-notifying people with the HLAs for autoimmune arthritis not to get the LYMERix vaccine. But the reality about the vast majority of the adverse events to LYMERix was that they were NON-HLA-LINKED immunosuppression outcomes. We’re making the same claim with this paper: No children are assessed for existing co-infection or immunosuppression status prior to vaccination. Pediatricians are not informed as to how to assess immune status.*

4) The MMR Monograph warns against babies actually getting the live viruses or potentially pregnant women (clue), but essentially sloughs off (like a snake) responsibility/liability on the injecting pediatrician:

http://www.merck.com/product/usa/pi_circulars/m/mmr_ii/mmr_ii_pi.pdf

“CONTRAINDICATIONS Hypersensitivity to any component of the vaccine, including gelatin. {40} Do not give M-M-R II to pregnant females; the possible effects of the vaccine on fetal development are unknown at this time. If vaccination of postpubertal females is undertaken, pregnancy should be avoided for three months following vaccination (see INDICATIONS AND USAGE, Non-Pregnant

“Adolescent and Adult Females and PRECAUTIONS, Pregnancy).

Anaphylactic or anaphylactoid reactions to neomycin (each dose of reconstituted vaccine contains approximately 25 mcg of neomycin). 4 Febrile respiratory illness or other active febrile infection.

“However, the ACIP has recommended that all vaccines can be administered to persons with minor illnesses such as diarrhea, mild upper respiratory infection with or without low-grade fever, or other low-grade febrile illness. {41} Patients receiving immunosuppressive therapy. This contraindication does not apply to patients who are receiving corticosteroids as replacement therapy, e.g., for Addison's disease.

“Individuals with blood dyscrasias, leukemia, lymphomas of any type, or other malignant neoplasms affecting the bone marrow or lymphatic systems. **Primary and acquired immunodeficiency states, including patients who are immunosuppressed in association with AIDS or other clinical manifestations of infection with human immunodeficiency viruses;**{41-43} **cellular immune deficiencies; and hypogammaglobulinemic and dysgammaglobulinemic states.**

“**Measles inclusion body encephalitis**{44} (MIBE), **pneumonitis**{45} **and death as a direct consequence of disseminated measles vaccine virus infection have been reported in immunocompromised individuals inadvertently vaccinated with measles-containing vaccine. Individuals with a family history of congenital or hereditary immunodeficiency, until the immune competence of the potential vaccine recipient is demonstrated.**

What they are saying is, don't vaccinate someone who is immunosuppressed, but whose pediatrician ever pre-screens for immune incompetence prior to vaccination? We've heard of infants with cold viruses going to the pediatrician, being vaccinated, and then being carried out never the same again. You see clearly they write in the MMR “Contraindications” the same warnings we are proving to you – don't vaccinate someone who is immunosuppressed and be sure the vaccine vials are not contaminated with fungal mycoplasma and the like, but how does anyone know what are the states of the vaccine vial or the children?

IDSA believes (below) that there is a problem, here, especially regarding the AGE of the vaccinee and they CLAIM basically, that “this has killed some babies” - whose parents were probably blamed; let's remember Roy Meadows, the original Munch-meister and SIDS deaths -, and that the vaccine schedule “*suits the manufacturers and not their victims.*”

From a book on Adverse Events – it indeed shows that they threw out cases where the child should not have been vaccinated from the safety and efficacy data (*kids were immunosuppressed, presently infected with a cold or EBV, etc.*) – just as we guessed. See those references they threw out:

Adverse Effects of Vaccines: Evidence and Causality.

[Show details](#)

Committee to Review Adverse Effects of Vaccines; Institute of Medicine; Stratton K, Ford A, Rusch E, et al., editors. Washington (DC): [National Academies Press \(US\)](#); 2011 Aug 25.

“The committee identified 18 publications reporting encephalitis or meningoencephalitis after the administration of vaccines containing measles, mumps, and rubella alone or in combination. [Mustafa et al. \(1993\)](#) described one case of encephalitis developing after administration of a MMR vaccine; however, wild-type measles virus was demonstrated in the patient. Fourteen publications did not provide evidence beyond temporality ([Ehregut and Zastrow, 1989](#); [Fescharek et al., 1990](#); [Forster and Urbanek, 1982](#); [Jagdis et al., 1975](#); [Jorch et al., 1984](#); [Kumar et al., 1982](#); [Landrigan and Witte,](#)

[1973](#); [Pollock and Morris, 1983](#); [Ross and Yeager, 1977](#); [Schneck, 1968](#); [Schuil et al., 1998](#); [Shuper, 2011](#); [Wiersbitzky et al., 1992b, 1993a](#)). In addition, five publications reported concomitant infections that could contribute to the development of symptoms ([Ehregut and Zastrow, 1989](#); [Forster and Urbanek, 1982](#); [Jorch et al., 1984](#); [Wiersbitzky et al., 1992b, 1993a](#)). **These publications did not contribute to the weight of mechanistic evidence.”**

“Described below are three publications reporting clinical, diagnostic, or experimental evidence that contributed to the weight of mechanistic evidence.

“[Bakshi et al. \(1996\)](#) described a 16-month-old boy presenting with a focal seizure on the right side and left hemipareses and a left gaze preference 5 months after receiving a measles, mumps, and rubella vaccine and 3 days after undergoing bone marrow transplantation. The patient was administered the vaccine prior to being diagnosed with sickle cell trait and a severe combined immunodeficiency. Serum and CSF were negative for bacteria and fungi. Mumps virus was demonstrated in the urine, serum, and CSF. The patient was diagnosed with meningoencephalitis and died 2 months after the onset of symptoms. Pathological examination of the leptomeninges showed chronic and focally prominent meningitis.

“[Lacroix et al. \(1995\)](#) describe a 5-year-old acquired immune deficiency syndrome (AIDS) patient presenting with fever, generalized seizures, and the inability to stand or walk approximately 2 years after vaccination against measles. The patient died months after presenting with neurological symptoms. Retrospective serum analysis showed measles antibody prior to vaccination. Viral cultures of brain samples were negative for measles virus. Frozen sections of basal ganglia, frontal cortex, and white matter were stained with antibodies against measles virus indicating the presence of measles virus in the brain.

[Valmari et al. \(1987\)](#) described a 7-year-old girl presenting with vomiting, headache, twitching of upper extremities, followed by coma lasting for several hours 54 days after receiving a measles, mumps, and rubella vaccine containing the Moraten measles strain and 5.5 years after receiving a measles vaccine containing the Schwarz measles strain. On the day the measles, mumps, and rubella vaccine was administered the patient complained of back pains leading to a diagnosis of acute lymphoblastic leukemia 23 days after vaccination. The patient presented with the symptoms described above 1 day after the fourth methotrexate treatment. Treatment with acyclovir was started and the patient seemed to improve. Measles virus was demonstrated in the CSF. The patient experienced a recrudescence of the neurological symptoms 58 days postvaccination and fever, photophobia, conjunctival inflammation, and a maculopapular rash 63 days postvaccination. Measles virus was demonstrated in the CSF again.

Weight of Mechanistic Evidence

“Encephalitis is considered a complication of infection with wild-type measles, mumps, and rubella viruses ([Gershon, 2010a,b](#); [Litman and Baum, 2010](#)). Encephalitis develops in 1:1,000 to 1:2,000 patients infected with measles virus ([Gershon, 2010a](#)). In addition many patients upon recovering suffer from neurologic sequelae ([Gershon, 2010a](#)). Encephalitis develops in 1:400 to 1:6,000 patients infected with mumps virus ([Litman and Baum, 2010](#)). In patients developing early-onset encephalitis upon infection with mumps virus, the damage to the neurons is by direct viral invasion ([Litman and Baum, 2010](#)). In patients infected with rubella virus, encephalitis develops in 1:5,000 patients ([Gershon, 2010b](#)). The committee considers the effects of natural infection one type of mechanistic evidence.

“The three publications described above, when considered together, did not present evidence sufficient for the committee to conclude the vaccine may be a contributing cause of encephalitis after administration of a measles or MMR vaccine. The patients described in the cases above had demonstrated immunodeficiencies. The publications presented evidence of the detection of viral antigens on frozen sections or the isolation of mumps or measles virus from the patients. However, the authors did not identify the virus as vaccine strain.

“The latency between vaccination and the development of encephalitis in the publications described above ranged from **5 months to 2 years**, suggesting persistent viral infection as the mechanism. Direct viral infection and viral reactivation may contribute to encephalitis; however, the publications did not provide evidence linking these mechanisms to MMR vaccine.

“The committee assesses the mechanistic evidence regarding an association between MMR vaccine and encephalitis as weak based on knowledge about the natural infection and three cases.

<https://www.ncbi.nlm.nih.gov/books/NBK190025/>

As you have just seen, it is precisely as we proposed. The kids being damaged from vaccines were immunosuppressed (or got a contaminated vaccine); there is a warning in the MMR about not vaccinating immunosuppressed children; **they are throwing out the vaccine failure cases by claiming the reversion to wild type can't be distinguished from natural infection**; and no doctor is given a tool for assessing immune status prior to vaccination.

These pediatric vaccines are One Size Fits All and the CDC says these “adverse events” are “a calculated risk.” Right now the CDC calculates that the risk of 1:68 kids becoming brain damaged for life is a good risk 😊 The CDC/BigPharma make the claim that vaccines are safe – excluding the phrase “for children who are not already sick or immunosuppressed” - and they BLATANTLY claim that this is not a contributing mechanism. We have shown the mechanism of immunosuppression-reactivates-viruses in parallel with LYMERix and Lyme, and CFIDS/ME post-sepsis syndrome.

Remember from Paul Auwaerter (and there are other reports on “reversion to wild type” in vaccine failure): “... human tissue-passaged vaccine isolates are highly related to parent vaccine strains (1, 15).

“...However, fatal infections have been documented in immunodeficient children vaccinated with these strains (1, 12, 14, 15). **The symptoms of infection occur many months after immunization, and the viruses isolated are similar to the original LA vaccine (1, 15), suggesting that in the absence of an effective host immune response, persistent infection with the vaccine strain can lead to fatal disease. Viruses isolated from these children could potentially represent virulent revertants of the original LA vaccine.**”

The following report refutes the entire concept that live, attenuated viral vaccines are preventing disease; one wonders whether in Lyme and Chronic Fatigue Syndrome these childhood vaccine diseases or naturally acquired infections are not reactivated, too, with the herpes:

“Subclinical [means no spots or lumps or immunosuppression-ish] Infection is Not Uncommon.”

[Ugeskr Laeger](#). 1992 Jul 13;154(29):2008-13.

[Duration of immunity and occurrence of secondary vaccine failure following vaccination against measles, mumps and rubella].

[Article in Danish]

“... In rare cases, rubella re-infection has resulted in infection in utero, so that a slight risk of congenital rubella cannot be entirely excluded after successful vaccination. No extensive systematic investigations of the effect of revaccination have been carried out and, similarly, **the optimal interval between two or more vaccinations has not been illustrated in more detail in the literature. Subclinical infection is not uncommon after all three vaccines.** Where measles is concerned, immunity may possibly be regarded as a continuum which, depending upon the antibody level, protects the individual from various degrees of clinical disease. If wild virus can be spread via individuals with subclinical infections, **it is doubtful** whether population immunity (herd immunity), which is necessary to eliminate the three diseases, can be attained in large populations.”

<https://www.ncbi.nlm.nih.gov/pubmed/1509566>

Wikipedia spells it out.

See the Wikipedia page on “attenuated vaccine.” You will see the exact same claims as above – the dangers are that the vaccines will fail and those children will GET those brain damaging viruses:

https://en.wikipedia.org/wiki/Attenuated_vaccine

<p>Disadvantages [edit]</p> <ul style="list-style-type: none"> • Secondary mutation can cause a reversion to virulence.^[7] • Can cause severe complications in immunocompromised patients.^[8] • Some can be difficult to transport due to requirement to maintain conditions (e.g. temperature) 	<p><i>Code Language for "You could actually GET these viruses."</i></p>
-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------	-------------------------------------------------------------------------

And Switzerland and the Netherlands say (March, 2017): The mother was immunosuppressed and may have passed along another vaccine virus or common virus, rendering the child too

immunosuppressed to get a fungal Tuberculosis vaccine on top of it:

Vaccine. 2017 Mar 1;35(9):1216-1226. doi: 10.1016/j.vaccine.2017.01.048. Epub 2017 Feb 3.

Safety of live vaccinations on immunosuppressive therapy in patients with immune-mediated inflammatory diseases, solid organ transplantation or after bone-marrow transplantation - A systematic review of randomized trials, observational studies and case reports.

Croce E1, Hatz C2, Jonker EF3, Visser LG3, Jaeger VK4, Bühler S5.

"... In most studies, the administration of live vaccines was safe. However, some serious vaccine-related adverse events occurred. 32 participants developed an infection with the vaccine strain; in most cases the infection was mild. However, in two patients fatal infections were reported: a patient with RA/SLE overlap who started MTX/dexamethasone treatment four days after the YFV developed a **yellow fever vaccine-associated viscerotropic disease (YEL-AVD)** and died. The particular vaccine lot was found to be associated with a more than 20 times risk of YEL-AVD. **One infant whose mother was under infliximab treatment during pregnancy received the BCG vaccine at the age of three months and developed disseminated BCG infection and died.** An immunogenicity assessment was performed in 43 studies. In most cases the patients developed satisfactory seroprotection rates. In the IMID group, YFV and VV demonstrated high seroconversion rates. MTX and tumor necrosis factor inhibitory therapy appeared to reduce immune responses to VV and HZ vaccine, but not to MMR and YF-revaccination. Seroconversion in SOT and BMT patients showed mostly higher rates for rubella than for measles, mumps and varicella."

<https://www.ncbi.nlm.nih.gov/pubmed/28162821>

These are many examples of how live attenuated vaccines fail by giving people the very diseases the vaccines were intended to prevent.

Policy Baloney and Circle Jerk "Reviews" (where they continually cite and recycle their own former scientific garbage, as seen in the Lyme crimes):

Vaccine. 2003 Sep 8;21(25-26):3954-60.

Unintended events following immunization with MMR: a systematic review.

Jefferson T1, Price D, Demicheli V, Bianco E; European Research Program for Improved Vaccine Safety Surveillance (EUSAFEVAC) Project.

"Public debate over the safety of the trivalent measles, mumps and rubella (MMR) vaccine and the drop in vaccination rates in several countries persists despite its almost universal use and accepted effectiveness. We carried out a systematic review to assess the evidence of unintended effects (beneficial or harmful) associated with MMR and the applicability of systematic reviewing methods to the field of safety evaluation. Eligible studies were comparative prospective or retrospective on healthy individuals up to 15 years of age, carried out or published by 2003. We identified 120 articles satisfying our inclusion criteria and included 22. MMR is associated with a lower incidence of upper respiratory tract infections, a higher incidence of irritability, similar incidence of other adverse effects compared to placebo and is likely to be associated with benign thrombocytopenic purpura (TP),

parotitis, joint and limb complaints and aseptic meningitis (mumps Urabe strain-containing MMR). Exposure to MMR is unlikely to be associated with Crohn's disease, ulcerative colitis, autism or aseptic meningitis (mumps Jeryl-Lynn strain-containing MMR). The design and reporting of safety outcomes in MMR vaccine studies, both pre- and post-marketing, are largely inadequate. The evidence of adverse events following immunization with MMR cannot be separated from its role in preventing the target diseases.”

<https://www.ncbi.nlm.nih.gov/pubmed/12922131>

Of course, *now* we know ^^ that is all FALSE since they exclude cases where the child should not have been vaccinated due to immunosuppression, especially with a concurrent infection. When they find the MMR viruses associated with encephalitis, they say, “Oh, it must be a wild type strain,” not mentioning that the wild type can revert from the vaccine strain and viruses mutate all the time – standard fare.

The issue is, how dangerous can it be to have three potentially immunosuppressive (we know measles is) vaccines injected several times into infants whose immune systems and especially brain are still developing with no prescreening for immune status? The Autism rate skyrocketing and there is a negative-benefit, a 1:68 brain damage rate.

They, the CDC and others in policy or “Public Health” say they don’t know the “mechanism” by which viruses become reactivated in immunosuppression but they certainly do know the mechanisms of sepsis or “overwhelming the immune system” and they know what “cytokine storm” means. They know what happens in the cases of multiple infections – the immune system shuts down. HLA molecules are down-regulated in the body but upregulated in the brain, there is tolerance and cross tolerance, meaning expanding infections and infections types. Etc. They know about the opposite of “autoimmunity” since we caught the CDC lying about it so many times.

We caught the CDC lying about so many things: Dearborn, OspA, Lyme, Chronic Fatigue Syndrome and Suzanne Vernon with the mycoplasma, and now here, blaming the baby-victims and their parents, saying, “Oh, if you got the live reactivated MMR viruses, you caught it from the field. Bad mommies. You did not protect your children.”

5) CDC’s Patent, US # 7,632,510, admitting people can get the diseases from the vaccines

5.A.) Methods of inducing flavivirus immune responses through the administration of recombinant flaviviruses comprising an engineered japanese encephalitis virus signal sequence

"Finally, there is the risk that the virus may not be fully or completely inactivated or attenuated and thus, the vaccine may actually cause disease."

<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=7,632,510.PN.&OS=PN/7,632,510&RS=PN/7,632,510>

CDC SAYS,...

5.B) Measles, Mumps, and Rubella -- Vaccine Use and Strategies for Elimination of Measles, Rubella, and Congenital Rubella Syndrome and Control of Mumps: Recommendations of the Advisory Committee on Immunization Practices (ACIP)

"Updated information on adverse events and contraindications, particularly for persons with severe HIV infection, persons with a egg allergy or gelatin allergy, persons with a history of thrombocytopenia, and persons receiving steroid therapy [are immunosuppressed – SASH]."

<http://www.cdc.gov/mmwr/preview/mmwrhtml/00053391.htm>

6) Pharma SAYS:

We learn from this MRSA vaccine patent (US patent 7,771,728, Intercell AG) that there is a risk of reversion to virulence if live attenuated viruses are injected into immunosuppressed persons:

Method for identification, isolation and production of antigens to a specific pathogen

"Several established vaccines consist of live attenuated organisms where the risk of **reversion to the virulent wild-type strain exists. In particular in immunocompromised hosts this can be a live (*sic*) threatening scenario.** Alternatively, vaccines are administered as a combination of pathogen-derived antigens together with compounds that induce or enhance immune responses against these antigens (these compounds are commonly termed adjuvant), since these subunit vaccines on their own are generally not effective."

<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=7,771,728.PN.&OS=PN/7,771,728&RS=PN/7,771,728>

Giving brain-damaging meningitis to children via mumps vaccine virus:

[Rev Med Virol.](#) 1998 Jul;8(3):129-142.

Genetic studies on a mumps vaccine strain associated with meningitis.

[Brown EG1, Wright KE.](#)

[Author information](#)

“Vaccination with mumps measles and rubella (MMR) vaccine containing the live attenuated mumps strain, Urabe AM9, is associated with an increased incidence of meningitis. The isolation

of mumps virus from CSF and subsequent identification as Urabe AM9-like by sequence analysis confirmed the causative role of Urabe AM9 vaccine in meningitis. To assess the role of genetic reversion in vaccine failure, sequence comparisons were made between several genes of Urabe AM9 vaccine and post-vaccination meningitis mumps isolates. An amino acid substitution in the Urabe AM9 HN gene Lys335Glu was not detected in the post-vaccination meningitis isolates suggesting that reversion to wild type sequence was associated with vaccine failure. However, further analysis showed that the vaccine was a mixture of viruses that differed at aa 335 of HN, possessing either the wild type Lys335 or the mutant Glu335, whereas the clinical isolates were homogeneous and possessed the wild type Lys335. Passage of the Urabe AM9 vaccine preparations in Vero cells resulted in the amplification of the Glu335 virus, however the post-vaccination meningitis isolates (Lys335) grew better in Vero cells than Urabe AM9 vaccine. A virus isolate, similar to the post-vaccination isolates was obtained from the vaccine suggesting that the strain responsible for vaccine failure was a pre-existing component of the vaccine and was not necessarily the result of reversion. The Urabe AM9 vaccine is a heterogeneous mixture of genotypes that differ in virulence with the HN Glu335 viruses being attenuated and at least a subset of the HN Lys335 viruses that are associated with disease. The Glu335 mutation may be among a class of attenuating mutations identified in several neurotropic viruses that involve charged amino acids in neutralising epitopes of receptor binding proteins. Copyright 1998 John Wiley & Sons, Ltd.”
<https://www.ncbi.nlm.nih.gov/pubmed/10398501>

And:

[Acta Neuropathol.](#) 2017 Jan;133(1):139-147. doi: 10.1007/s00401-016-1629-y. Epub 2016 Oct 21.

Deep sequencing reveals persistence of cell-associated mumps vaccine virus in chronic encephalitis.

[Morfopoulou S1](#), [Mee ET2](#), [Connaughton SM2](#), [Brown JR3](#), [Gilmour K4](#), [Chong WK5](#), [Duprex WP6](#), [Ferguson D2](#), [Hubank M7](#), [Hutchinson C8](#), [Kaliakatsos M9](#), [McQuaid S10,11](#), [Paine S8,12](#), [Plagnol V13](#), [Ruis C14](#), [Virasami A8](#), [Zhan H15](#), [Jacques TS8,16](#), [Schepelmann S2](#), [Qasim W17,18](#), [Breuer J14,3](#).

“Routine childhood vaccination against measles, mumps and rubella has virtually abolished virus-related morbidity and mortality. Notwithstanding this, we describe here devastating neurological complications associated with the detection of live-attenuated mumps virus Jeryl Lynn (MuVJL5) in the brain of a child who had undergone successful allogeneic transplantation for severe combined immunodeficiency (SCID). **This is the first confirmed report of MuVJL5 associated with chronic encephalitis and highlights the need to exclude immunodeficient individuals from immunisation with live-attenuated vaccines.** The diagnosis was only possible by deep sequencing of the brain biopsy. Sequence comparison of the vaccine batch to the MuVJL5 isolated from brain identified biased hypermutation, particularly in the matrix gene, similar to those found in measles from cases of SSPE. The findings provide unique insights into the pathogenesis of paramyxovirus brain infections.”

<https://www.ncbi.nlm.nih.gov/pubmed/27770235>

7) CDC on how humans can get diseases from immunosuppressed vaccinated animals:

Human Exposure to Brucella abortus Strain RB51 -- Kansas, 1997

<http://www.cdc.gov/mmwr/preview/mmwrhtml/00051495.htm>

[In the above, an immunosuppressed pregnant cow was given a Brucella (LYMERix-like) "live attenuated" vaccine and the baby cow ended up with the disease, which then was transferred to the humans handling the cow and her dead baby. This parallels what is happening to children who are vaccinated while immunosuppressed, or who receive mycoplasma (LYMERix-like) contaminated vaccines.]

More:

[Clin Infect Dis](#). 2003 Aug 1;37(3):407-14. Epub 2003 Jul 22.

Human illness associated with use of veterinary vaccines.

[Berkelman RL](#)1.

“Veterinary vaccines are being used with increasing frequency in the United States to protect the health of animals. However, humans may be inadvertently exposed to these products by means of unintentional inoculation or other routes of exposure. The potential for both exposure and for adverse consequences secondary to exposure to veterinary vaccines may be growing. With the exception of brucellosis vaccines, there have been few reports of suspected or confirmed adverse events in humans associated with the use of animal vaccines, but it is unclear whether that is because few adverse events occur or because adverse events are not recognized and/or reported. Results of a search for relevant literature and of communications with health officials at governmental and private institutions suggest that enhanced efforts are needed to recognize and to prevent human illness associated with use of veterinary vaccines.

“Veterinary vaccines are being used with increasing frequency in the United States to protect the health of animals. In addition to their direct benefit to animals, these vaccines have also markedly decreased the risk of transmission of many zoonotic infections (e.g., rabies and brucellosis) to humans. The US Department of Agriculture currently licenses >2000 vaccines for use in animals [1]. Most of these vaccines are inactivated formulations, but >500 live vaccine formulations for animals are also licensed. Veterinary vaccines are intended only for use in animals and are not tested for safety in humans. However, humans may inadvertently be exposed to these products by means of unintentional inoculation or other routes of exposure.

<https://www.ncbi.nlm.nih.gov/pubmed/12884166>

<https://academic.oup.com/cid/article/37/3/407/437242/Human-Illness-Associated-with-Use-of-Veterinary>

The point is that an immunosuppressed animal is infectious for the vaccine virus, and that, like we hear in the Humira and Stelara commercials, “don’t go near anyone who recently had a vaccine if you are taking these immunosuppressive drugs,” because the ***other person harbors live viruses, and you are immunosuppressed*** – a model to which the CDC does not otherwise admit. Why. Because then people will say, “Oh, the babies are getting the live viruses and may be immunosuppressed at the same time? Is that what’s happening with the MMR Autism vaccines?”

8.) CDC SAYS... stress hormones like cortisol activate viruses (but when fungi activate latent viruses it is not reversible, as is shown in other EBV-diseases such as Lupus, cancer, MS, and CFIDS/Lyme):

2012; *The effect of exogenous corticosterone on West Nile virus infection in Northern Cardinals (Cardinalis cardinalis)*

“Corticosterone was administered at levels that individuals enduring chronic stressors (i.e., long-term inclement weather, food shortage, anthropogenic pollution) might experience in the wild. Corticosterone greatly impacted mortality: half of the corticosterone-implanted cardinals died between five - 11 days post-inoculation whereas only one of nine sham-implanted (control) birds died. ... No differences were found in viral titer between corticosterone- and sham-implanted birds. However, cardinals that survived infections had significantly higher average body temperatures during peak infection than individuals that died... In sum, this study indicates that elevated corticosterone could affect the survival of WNV-infected wild birds, suggesting that populations may be disproportionately at-risk to disease in stressful environments.”

<http://7thspace.com/headlines/410671/>

[the-effect-of-exogenous-corticosterone-on-west-nile-virus-infection-in-northern-cardinals-cardinalis-cardinalis.html](http://7thspace.com/headlines/410671/the-effect-of-exogenous-corticosterone-on-west-nile-virus-infection-in-northern-cardinals-cardinalis-cardinalis.html)

The same is true for humans and cortisol and the activation of latent herpesviruses; just go to PubMed and look for **astronauts and EBV, or medical students and EBV**—you’ll see cortisol come up);); when astronauts or wannabe doctors are stressed out, they may have cortisol-activated EBV. We’ve made this information into a criminal charge sheet (Somatoform/Wessely) to show the slander and libel - and the CDC-associated perps *know* - yet claim that us regular humans, no, we’re assigned “**some psychiatric disorder**.” Why the big secret, no one knows since it’s common knowledge that arrogance is the cowardly calling card of walking anal sphincters. This harassment of the CDC’s victims is called a “Deprivation of Rights via Color of Law” crime.

9) IDSA admits vaccines not safe for babies:

“Amanda Jezek, the vice president of Public Policy and Government Relations at the Infectious Diseases Society of America (IDSA), in Arlington, Va., said there is concern that this push to recommend a vaccine before the ACIP has reviewed the evidence would completely “jeopardize the integrity of ACIP’s recommendations.”

“Most of the vaccinations given in this country are received by those younger than 2 years of age, so assuring the safety and efficacy of vaccines is paramount. Every year, more than 40 million vaccines are given to children younger than 1 year of age, usually between 2 and 6 months of age, Dr. Temte said. ***At this age, infants are at greatest risk for certain serious medical adverse events, including high fevers, seizures and sudden infant death syndrome,*** according to the U.S. Vaccine Adverse Event Reporting System. *Therefore, it is important for the ACIP to consider carefully the risks versus the benefits before making a recommendation rather than be on a forced schedule that suits the manufacturer as opposed to the patient.*”

http://www.idse.net/ViewArticle.aspx?d=Public%2BHealth&d_id=212&i=August+2015&i_id=1215&a_id=33373

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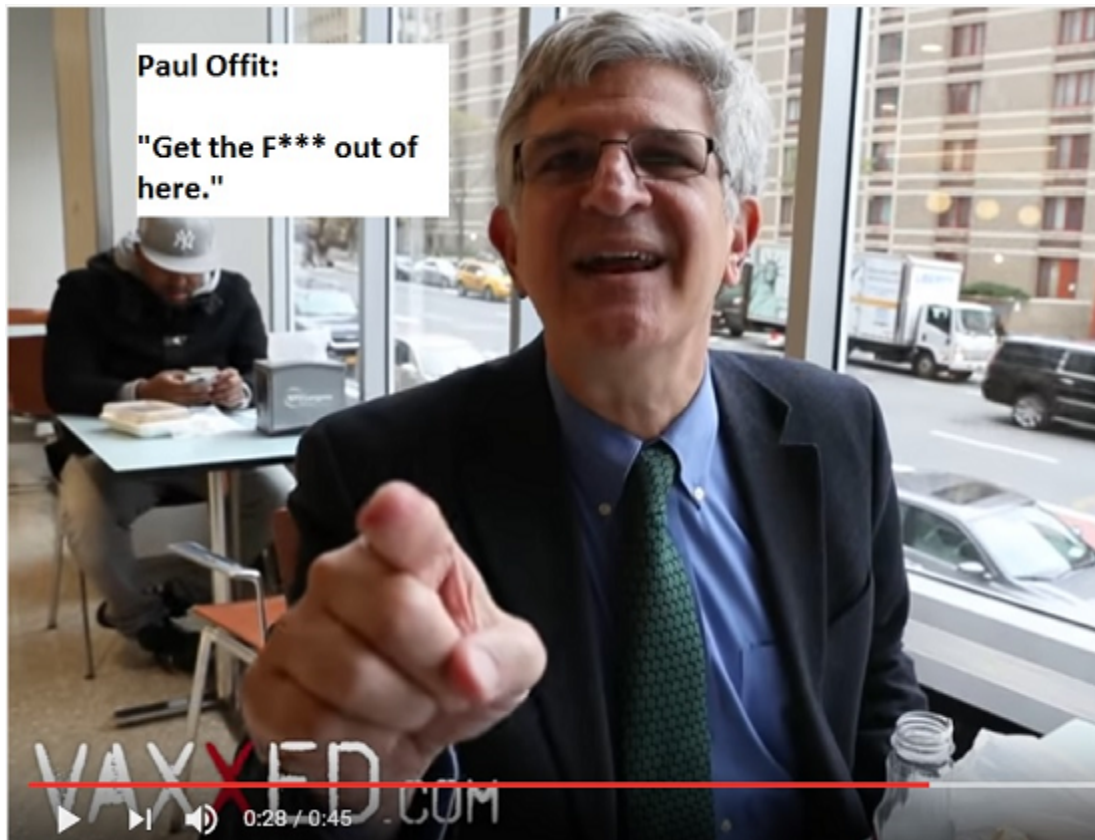
In addition, the ACIP is tasked with choosing the components of the annual influenza vaccination, which changes every year as the virus mutates throughout the season. Experts around the world track these mutations to predict which flu strains will be predominant in the following season. The ACIP makes recommendations about the strains to include in next year’s flu vaccination. Just under 1 million U.S. infants, children and adults received the influenza vaccine during the 2012-2013 flu season.

Every situation surrounding a recommendation is different, Dr. Temte said. For instance, the pneumococcal conjugate vaccine (Pneumovax 23, Pfizer) is recommended to protect against pneumococcal disease. The vaccine has been indicated for children for some time, but received a new indication for adults older than 50 years of age in December 2011.

The vaccine received accelerated approval by the FDA for the adult indication without clinical data to show efficacy in adults. Those data did not come until the company conducted a postlicensure trial involving 84,000 individuals. It was almost two years after the new indication was granted before the ACIP had the safety and efficacy data to make a good recommendation about the vaccine.

“I’d like to get some explanation about how we can compress the acquiring of information into a very limited time frame,” Dr. Temte said.

10) Offit reveals vaccines fail via immunosuppression via snarkasm (“how ironic, since the lady did not produce antibodies”), and Eugene Shapiro reveals you can’t have a “disease” unless it is “inflammatory” or “autoimmune” or results in “too many antibodies.”



In one of the most revealing of all anti-anti-vaxxer reports, **Paul Offit** shows that he absolutely knows vaccines can cause immunosuppression, and we know what happens in cases of immunosuppression from the sum of the data in the Occam’s Razor criminal charge sheet. **The woman he talks about acquired multiple sclerosis, which comes from what? Immunosuppression-reactivated Epstein-Barr, which pretty surely is associated with the development of Multiple Sclerosis (and post-sepsis Chronic Fatigue/Lyme):**

[N Engl J Med](#). 2008 May 15;358(20):2089-91. doi: 10.1056/NEJMp0802904.

Vaccines and autism revisited--the Hannah Poling case.

[Offit PA1](#).

“No case, however, represented a greater deviation from the VICP’s original standards than that of Dorothy Werderitsh, who in 2006 successfully claimed that a hepatitis B vaccine had caused her multiple sclerosis. By the time of the ruling, several studies had shown that hepatitis B vaccine neither caused nor exacerbated the disease, and the Institute of Medicine had concluded that “evidence favors rejection of a causal relationship between hepatitis B vaccine and multiple sclerosis.”² But the VICP was less impressed with the scientific literature than it was with an expert’s proposal of a mechanism

by which hepatitis B vaccine could induce autoimmunity (**an ironic conclusion, given that Dorothy Werderitsh never had a detectable immune response to the vaccine**).”
<http://www.nejm.org/doi/full/10.1056/NEJMp0802904>

Be sure to read the whole “report.” The woman mentioned here, Dorothy Werderitsch, had no immune response to the vaccine—that means she was immunosuppressed. Right, Offit, thanks for revealing it all in not only this example, but all your others in that report. Also, you will find, here, the Hepatitis B vaccines cause immunosuppression.

Again, **Yale’s Eugene Shapiro** (who assaulted Czech children with a known fake vaccine, LYMERix, which they knew would do those children no good because there is none of that kind of OspA in Europe, and was just an experiment to see how bad were the adverse events) saying you can’t have a disease without inflammation:

Having a “Disease” without classic “Inflammation” or “Autoimmunity” – This FRAUD has to do with the Autism pandemic:

The Lyme criminals claim you can’t have a “disease” unless you have inflammation or an autoimmune outcome. Of course, such a claim betrays the source of the Autism pandemic. The kids are getting the viruses instead of the protection and this is shown in many places and is called an “adverse event.”

Yale’s Eugene Shapiro in PBS’ “Life on Earth Series”:

“TOOMEY: But most physicians think there's good reason to discount the possibility of chronic Lyme Disease.

“SHAPIRO: What some people would have you believe is that there are two different diseases.

“TOOMEY: Yale physician Eugene Shapiro says first, take the obvious case of Lyme Disease that usually starts with a distinctive rash and can lead to arthritis and facial paralysis.

“SHAPIRO: Somehow, for that form of the disease, antibiotics are effective. They do fine. But then there's some other form of the disease which is, you can't put your hand around it. They don't have objective findings of inflammation, which is the way bacteria cause disease.

“TOOMEY: What these patients do have are symptoms that doctors call nonspecific. They span a broad spectrum. Emotional problems, as in the case of Lisa's daughter; or fatigue, muscle pains, depression. Doctor Shapiro helped write the Lyme treatment guidelines put out by the Infectious Diseases Society of America. Guidelines that state even the most advanced cases of infection can be eradicated with two months of oral or intravenous antibiotics. But for the patients with these ongoing, nonspecific symptoms, the maximum treatment didn't seem to work.

“SHAPIRO: They would have you believe that form of the disease, somehow this is, the bacteria knows to act differently and it doesn't respond to antibiotics in this sense. It really doesn't make any sense.

<http://loe.org/shows/segments.html?programID=00-P13-00037&segmentID=1>

Of course it does make sense: Spirochaeta are not bacteria, in the sense that 1) they are their own Phylum, 2) shed fungal antigens and 3) have no LPS. But, what would Shapiro know about science? His undergraduate degree is in English Literature. The point is, you see here, Shapiro, et al, claim that you can't have a disease unless you have inflammation or an autoimmune one. Spirochetes are notorious immune suppressors like Tuberculosis because the Osps are triacylated lipoproteins, which means they are managed by Toll Like Receptors 1 and 2, and which means they are FUNGAL. (Which means THEY ARE *NOT* REGULAR “BACTERIA.”) Thimerosal is put in vaccines to prevent the very shed fungal antigens from spirochetes or anything, obviously, fungal, like mycoplasma or chlamydia, etc.

11) Hep B associated with immunosuppression via TLR2 agonism, the vaccine antigen is called “HbsAg”)

<https://www.ncbi.nlm.nih.gov/pubmed/?term=Hepatitis+B+recombinant+antigen+and+tlr>

Hepatology. 2009 Apr;49(4):1132-40. doi: 10.1002/hep.22751.

Hepatitis B virus suppresses toll-like receptor-

mediated innate immune responses in murine parenchymal and nonparenchymal liver cells.

[Wu J1](#), [Meng Z](#), [Jiang M](#), [Pei R](#), [Tripller M](#), [Broering R](#), [Bucchi A](#), [Sowa JP](#), [Dittmer U](#), [Yang D](#), [Roggendorf M](#), [Gerken G](#), [Lu M](#), [Schlaak JF](#).

“We have previously shown that Toll-like receptor (TLR)-activated murine nonparenchymal liver cells [(NPC); Kupffer cells (KC), liver sinusoidal endothelial cells (LSEC)] can suppress hepatitis B virus (HBV) replication. Therefore, the aim of this study was to investigate whether HBV has the ability to counteract the TLR-mediated control of its replication. Freshly purified murine hepatocytes and NPCs obtained from C57BL6 mice were stimulated by TLR 1-9 ligands in the presence or absence of hepatitis B surface antigen (HBsAg), hepatitis B e antigen (HBeAg), HBV virions, or supernatants from HBV-producing HBV-Met cells, and HBV replication was suppressed by anti-hepatitis B virus X protein (HBx) small interfering RNA (siRNA) in HBV-Met cells. Supernatants were collected and tested for antiviral cytokines by viral protection assay. HBV gene expression and replication was analyzed by southern blot. RNA and proteins were analyzed by quantitative reverse transcription polymerase chain reaction (RT-PCR) or western blot and enzyme-linked immunosorbent assay, respectively. **Pretreatment of hepatocytes and NPCs with HBV-Met cells supernatants, HBsAg, HBeAg, or HBV virions almost completely abrogated TLR-induced antiviral activity, which correlated with suppression of interferon beta (IFN-beta) production and subsequent interferon-**

stimulated gene induction as well as suppressed activation of interferon regulatory factor 3 (IRF-3), nuclear factor kappa B (NF-kappaB), and extracellular signal-regulated kinase (ERK) 1/2. In HBV-infected HBV-Met cells, TLR stimulation did not induce antiviral cytokines in contrast to primary hepatocytes. TLR-stimulated expression of proinflammatory cytokines [tumor necrosis factor alpha (TNF-alpha), interleukin-6 (IL-6)], and activation of IRF-3 was suppressed after up-regulation of HBV replication in HBV-Met cells. Accordingly, suppression of HBV replication by siRNA led to activation or expression of proinflammatory transcription factors and cytokines.

CONCLUSION:

Our data indicate that HBV can suppress the TLR-induced antiviral activity of liver cells. This has major implications for the interaction between HBV and the immune system.”

Here we have just shown that Hep B (HbsAg – the vaccine) causes immunosuppression, while Offit says: “But the VICP was less impressed with the scientific literature than it was with an expert's proposal of a mechanism by which hepatitis B vaccine could induce autoimmunity (**an ironic conclusion, given that Dorothy Werderitsh never had a detectable immune response to the vaccine**).”

Offit says, yes, she was immunosuppressed - probably from the vaccine since the Hep B vaccine causes immunosuppression. Or maybe she had a cold and should not have been vaccinated at the time.

And what happens with immunosuppression? Right, the reactivation of the herpes viruses which can lead to Multiple Sclerosis. It's hardly “ironic.” You can tell that **the denial of failed vaccines** that fail by giving people/children the actual viruses (the Hep B vaccine is a recombinant antigen and not a whole live virus), or cause immunosuppression and then the reactivation of latent herpesviruses (Lyme and ME/CFS),... is public policy. Auwaerter does it, and Offit does it.

The vaccines fail via immunosuppression then reactivation-of-latent viruses or activation-of-live-attenuated vaccine viruses – disease conditions not admitted to by the U.S. Government as policy. It's policy.

There is no NIIID. Not with 1:68 kids brain damaged for life from these insane non-vaccines. The whole country would come to a standstill if it was known by the general population that a program exists to throw out 25% or more of the humans in this country just to save the international reputations of BigPharma and the incompetent bioweaponeers of the CDC.

12) Claiming the Chicken Pox vaccine reactivates to live viruses in immunosuppressed individuals (how unsurprising)

[Nat Med](#). 2000 Apr;6(4):451-4.

Varicella vaccination: evidence for frequent reactivation of the vaccine strain in healthy children.

[Krause PR1](#), [Klinman DM](#).

Wild-type varicella zoster virus (VZV) causes chickenpox, a common childhood illness characterized by fever and a vesicular rash and rare serious complications. Wild-type VZV persists in a latent form in the sensory ganglia, and can re-activate to cause herpes zoster. More than 10 million American children have received the live attenuated Oka strain VZV vaccine (OkaVZV) since its licensure in 1995. Pre-licensure clinical studies showed that mean serum anti-VZV levels among vaccinees continued to increase with time after vaccination. This was attributed to immunologic boosting caused by exposure to wild-type VZV in the community. Here, we examine the alternative, that large-scale asymptomatic reactivation of OkaVZV might occur in vaccinees. We analyzed serum antibody levels and infection rates for 4 years of follow-up in 4,631 children immunized with OkaVZV. Anti-VZV titers decreased over time in high-responder subjects, but rose in vaccinees with low titers. Among subjects with low anti-VZV titers, the frequency of clinical infection and immunological boosting substantially exceeded the 13%-per-year rate of exposure to wild-type varicella. These findings indicate that OkaVZV persisted in vivo and reactivated as serum antibody titers decreased after vaccination. This has salient consequences for individuals immunized with OkaVZV.

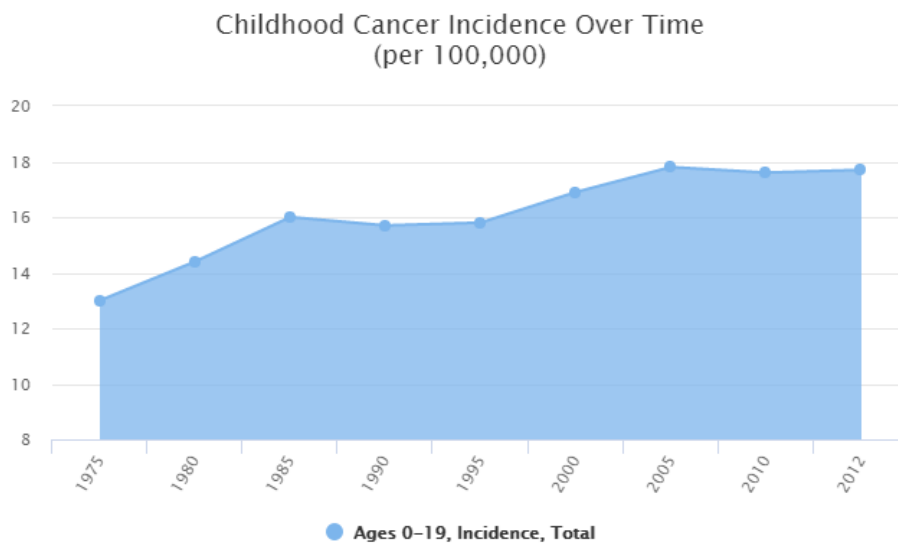
<https://www.ncbi.nlm.nih.gov/pubmed/10742154>

Many reports here:

https://www.google.com/?gws_rd=ssl#q=vaccine+virus+reactivation&*

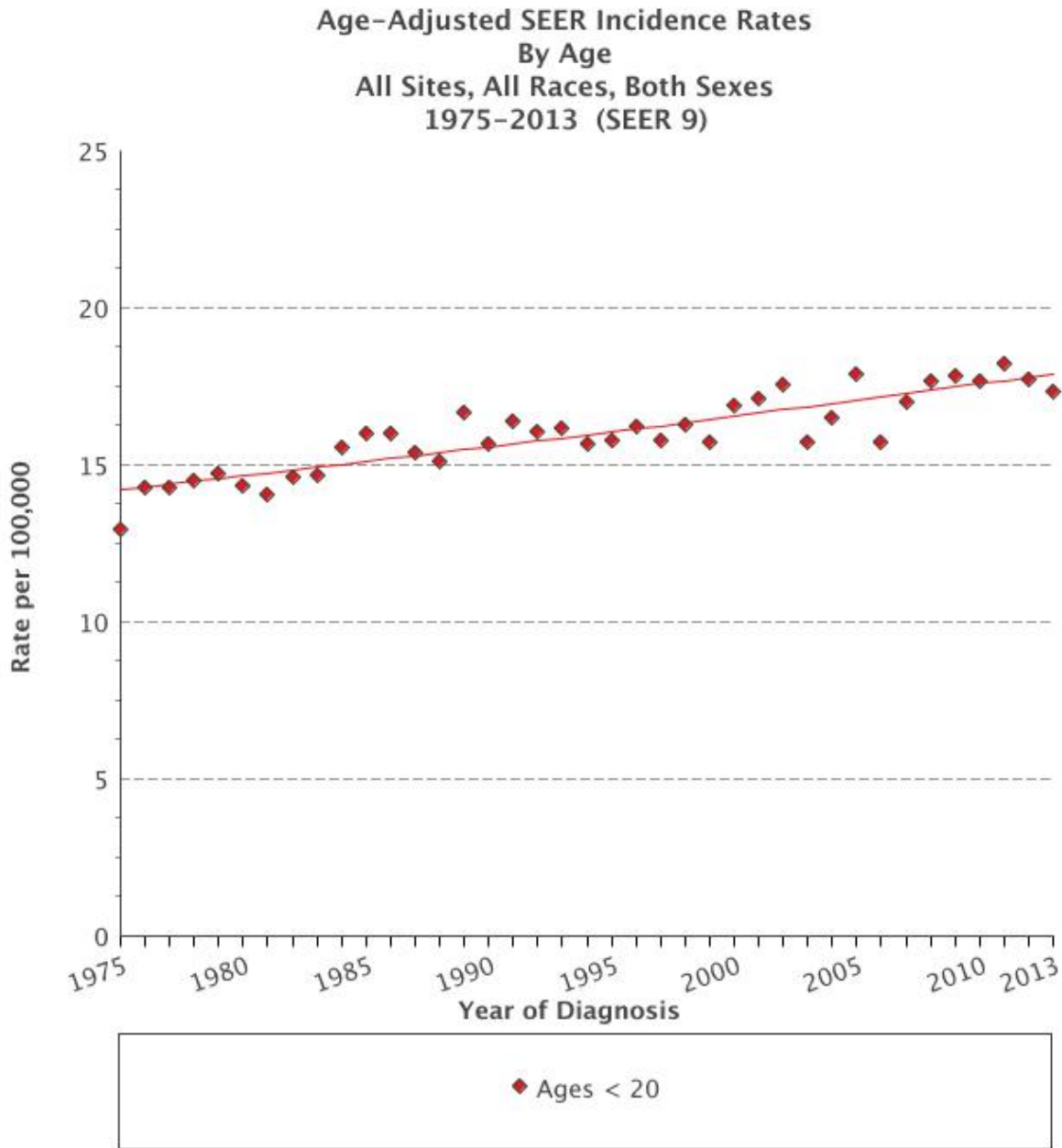
13) Cancer and Autism, naturally co-trending since hypervaccination causes immune-blunting just like old age immunity (cancer, like Lyme and CFIDS, is classified as "a failure of the immune system" which is at the other end of the immunity spectrum from autoimmunity):

Even as the cure rate continues to improve, the incidence of childhood [cancer](#) has been steadily increasing over the last few decades, from about 13 children per 100,000 in 1975 to over 17 children per 100,000 since 2005.



Source: Surveillance, Epidemiology, and End Results (SEER) Program (seer.cancer.gov) SEER 9 areas, 1975-2012, Age 0-19.

<https://curesearch.org/Incidence-Rates-Over-Time>



Cancer sites include invasive cases only unless otherwise noted.
Rates are per 100,000 and are age-adjusted to the 2000 US Std Population (19 age groups - Census P25-1130). Regression lines are calculated using the Joinpoint Regression Program Version 4.2.0, April 2015, National Cancer Institute.
Incidence source: SEER 9 areas (San Francisco, Connecticut, Detroit, Hawaii, Iowa, New Mexico, Seattle, Utah, and Atlanta).

<https://seer.cancer.gov/faststats/selections.php?#Output>

14) The effect and danger of “over-vaccination” with multiple live attenuated viruses. Here someone repeats our own proposal: the CDC is creating a new toxic cauldron in hypervaccinated humans in an environment of so many known immune suppressors, such as mold or Lyme, or stress or inhalation of diesel bus fumes, not to mention measles itself is immunosuppressive:

Evolution (N Y). 2011 Dec; 4(4): 635–643.

doi: [10.1007/s12052-011-0365-y](https://doi.org/10.1007/s12052-011-0365-y)

The double-edged sword: How evolution can make or break a live-attenuated virus vaccine

[Kathryn A. Hanley](#)

“Too much of a good thing: competition and facilitation among strains in multi-strain vaccines

“Vaccine viruses can exhibit quite different dynamics when administered in combination with other vaccine viruses than when administered alone. At one end of the spectrum, the individual components of a multi-strain vaccine can show “interference”, in which one or more strains replicate to lower levels or stimulate a poorer immune response in a combined vaccine than a single-strain vaccine. Interference can be the product of immunodominance, direct competition for cellular or viral replication machinery, or immune-mediated apparent competition. Such interference has long been recognized; Sabin and his colleagues [56] pointed out in 1960 that the individual components of OPV were more efficacious when administered individually (as monovalent vaccines) than when administered together in a trivalent vaccine. Subsequently, multiple instances of interference among live-attenuated vaccine viruses have been documented ([57] and references therein). At the other end of the spectrum, facilitation, in which the replication or immunogenicity of attenuated viruses is enhanced in combination, could also occur. While this phenomenon has not been documented in vaccine viruses to the best of my knowledge, previous studies have certainly shown facilitation between unrelated viruses during concurrent infection [46, 58-60], thus a similar interaction between vaccine viruses is possible. While the outcome of interference and facilitation have been described, the mechanisms driving these dynamics are poorly understood. **However as the number of new live-attenuated vaccines targeted to the already overscheduled child continues to increase, it is becoming increasingly important to gain a mechanistic understanding of the ecological interactions among these attenuated viruses.**

<https://www.ncbi.nlm.nih.gov/pubmed/22468165>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3314307/>

And as we know, the rubella vaccines were intended to defeat the brain damage we call congenital Autism, so we wonder how giving it to neurodeveloping babies could be a good thing. The proposal probably suits the manufacturers and not the victims.

The Herd Effect tall tale (or, half-tale) – has everything to do with vaccinated people giving those live viruses from the vaccines to the un-vaccinated. That means of course, the viral vaccines are active, have been active and may, as active, have brain damaged children at a too-early age.

This term is probably vague for many, yet here the Wikipedia page explains that it has to do with people who can’t be vaccinated because of conditions we are not allowed to mention—

(immunosuppression, post-sepsis syndrome, you know, the classes of people otherwise known as somatoform):

“**Herd immunity** (also called **herd effect**, **community immunity**, **population immunity**, or **social immunity**) is a form of indirect protection from infectious disease that occurs when a large percentage of a population has become [immune](#) to an infection, thereby providing a measure of protection for individuals who are not immune.^{[1][2]} In a population in which a large number of individuals are immune, chains of infection are likely to be disrupted, which stops or slows the spread of disease.^[3] The greater the proportion of individuals in a community who are immune, the smaller the probability that those who are not immune will come into contact with an infectious individual.^[1] Individual immunity can be gained through recovering from a natural infection or through artificial means such as [vaccination](#).^[3] Some individuals cannot become immune due to medical reasons and in this group herd immunity is an important method of protection.^{[4][5]} Once a certain threshold has been reached, herd immunity gradually eliminates a disease from a population.^[5] This elimination, if achieved worldwide, may result in the permanent reduction in the number of infections to zero, called eradication.^[6] This method was used for the eradication of [smallpox](#) in 1977 and for the regional elimination of other diseases.^[7] Herd immunity does not apply to all diseases, just those that are [contagious](#), meaning that they can be transmitted from one individual to another.^[5] [Tetanus](#), for example, is infectious but not contagious, so herd immunity does not apply.^[4]”

https://en.wikipedia.org/wiki/Herd_immunity

In other words, the idea that people are walking around with live unattenuated vaccine viruses such that they could give them to someone else...is never supposed to cross paths with the idea that infants are getting whacked with 3 or more viruses at the same time, one of which is Rubella and which is known to cause the brain damage we call Autism. And you have previously seen, they know the vaccine viruses are active, live and can cause disease. "Herd immunity" - which no one really understands - is about "everyone get vaccinated so the ones who can't get vaccine because they're immunosuppressed and might get those live viruses." Ahem, what about the babies who are never pre-screened for immune status?

15) Rubella & “Low Responders” ... are they actually the ones with chronic active disease?

[Nat Med](#). 2000 Apr;6(4):451-4.

Varicella vaccination: evidence for frequent reactivation of the vaccine strain in healthy children.

[Krause PR1](#), [Klinman DM](#).

”Here, we examine the alternative, that large-scale asymptomatic reactivation of OkaVZV might occur in vaccinees. We analyzed serum antibody levels and infection rates for 4 years of follow-up in 4,631 children immunized with OkaVZV. Anti-VZV titers decreased over time in high-responder subjects, but **rose in vaccinees with low titers**. Among subjects with low anti-VZV titers, the frequency of

clinical infection and immunological boosting substantially exceeded the 13%-per-year rate of exposure to wild-type varicella. These findings indicate that OkaVZV persisted in vivo and reactivated as serum antibody titers decreased after vaccination. This has salient consequences for individuals immunized with OkaVZV.”

<https://www.ncbi.nlm.nih.gov/pubmed/10742154>

Low responders, who later become higher antibody-responders, are responding to the reactivated viruses? At least in this case, Varicella...

... And here we show low antibody responders from a Rubella vaccine:

Calif Med. 1971 Nov;115(5):16-22.

Transmission of rubella vaccine virus from vaccinees to contacts.

Wilkins J, Leedom JM, Salvatore MA, Portnoy B.

“**The report presents evidence of the transmission of hpv-77 derived rubella vaccine virus from vaccinees to two susceptible contacts.** The first instance of transmission was to a child who served as a transmission control on a "closed" study ward, and the second was to an antibody-negative mother in an "open" family study. Neither of these persons had any clinical evidence of rubella. Both had significant increases in rubella hemagglutination inhibiting (hai) antibody titers, but detectable complement fixing (cf) antibodies did not develop in either. With the kind of antigen used in our rubella cf test, this pattern of serologic response is characteristic of, but not diagnostic of, infection with the rubella vaccine virus. The serological evidence which was compatible with rubella vaccine virus infection, the complete absence of serologic or clinical evidence of "wild" rubella virus infections among the other four rubella susceptible transmission control children and the security precautions employed to ensure isolation on the "closed" ward, make "wild" rubella virus infection extremely unlikely. The evidence for rubella vaccine virus infection in the other susceptible contact is not as conclusive, because "wild" rubella virus infection is difficult to rule out in any person living in an "open" family situation. Nevertheless the need for more data is emphasized by the virtual certainty that rubella vaccine virus transmission did occur in the subject on the isolation ward, plus the high probability that the infection observed in the family group setting also represented transmission of rubella vaccine virus. Such data can only come from close surveillance of recipients of live rubella virus vaccines and their contacts in the future.”

<https://www.ncbi.nlm.nih.gov/pubmed/5117594>

And another:

Calif Med. 1969 Mar;110(3):224-7.

Viremia in a recipient of HPV-77 Rubella virus vaccine.

Wilkins J, Salvatore MA, Leedom JM, Portnoy B.

Abstract

“A live rubella virus vaccine, HPV-77 (High Passage Virus - 77 tissue culture passages) was administered subcutaneously to eight rubella-susceptible children housed in an isolation ward. One blood specimen, taken on the tenth day after vaccination, from one of the eight vaccines, yielded a rubella virus. This virus had laboratory markers which were "vaccine-like." To our knowledge, this

represents the first isolation of rubella virus from the blood of a recipient of HPV-77 vaccine. **However, the consistent antibody responses among vaccinees and the regular presence of rubella virus in their pharynges argue that viremia occurs in almost every susceptible recipient. The most logical explanation for the failure to document viremia in other recipients of HPV-77 vaccine is that the viremia is ordinarily low grade or transient or both.**

<https://www.ncbi.nlm.nih.gov/pubmed/5773481>

We are merely making the point we see no one else making: The Rubella vaccine was invented in the first place to prevent congenital Autism. When the MMR vaccines fail because children are not pre-screened for immune status, could it be the Rubella virus from the vaccines causing the same brain damage?

No one seems to be talking about it. Mumps without the Lumps, Measles without the Spots, and Rubella without the rash. Why? Immunosuppression and vaccination is not the normal route of infection.

16) Synergism, and Chronic Fatigue Syndrome (post sepsis without the spirochetes)

You've already seen Auwaerter on how measles is immunosuppressive, and the book about failed vaccines say: "The latency between vaccination and the development of encephalitis in the publications described above ranged from **5 months to 2 years**, suggesting persistent viral infection as the mechanism. Direct viral infection and viral reactivation may contribute to encephalitis;"

That's basically the definition of post-sepsis Chronic Fatigue Syndrome, which is a condition simultaneously denied by the CDC. They say there is no such thing as chronic active viral and other infections because that would explain the Autism pandemic from vaccines.

But Dual Infections or multiple infections could be bad. "Doctors" are supposed to know this basic medical science—the synergy where **Malaria activated Epstein-Barr** and caused **Burkitt's Lymphoma** due to the immunosuppression from Malaria Plus Epstein-Barr virus. The following report could be a good cross over point between the failed vaccines that fail via immunosuppression or "overwhelming the immune system, or, "especially due to exposure to TLR2/1 agonists," and..., "**is** the model that parallels the post-septic shock outcomes of ME/CFS, Lyme and the other harassed victims groups."

We really don't want to say that "doctors are supposed to know this stuff," but doctors are supposed to know this stuff and it shouldn't have to be revealed by the crime victims. We're forced to live in an alternate universe. We're RICO organized crime victims like mob-poison-survivors or drive-by-shooting survivors solving our own case by hacking and taping and recording mob emails and phone calls, outlining the who/what/where of the crime for the stupid lazy cops or FBI. Yet, we're doing that exact thing while the dumb "doctors" look down their noses.

[Malar J.](#) 2010 Mar 1;9:64. doi: 10.1186/1475-2875-9-64.

Dual effect of Plasmodium-infected erythrocytes on dendritic cell maturation.

[Bettiol E1](#), [Carapau D](#), [Galan-Rodriguez C](#), [Ocaña-Morgner C](#), [Rodriguez A](#).

“It was found that intact erythrocytes infected with *P. yoelii* do not induce maturation of DC unless they are lysed, suggesting that accessibility of parasite inflammatory molecules to their receptors is a key issue in the activation of DC by *P. yoelii*. This activation is independent of MyD88. It was also observed that pre-incubation of DC with intact *P. yoelii*-infected erythrocytes inhibits the maturation response of DC to other TLR stimuli. The inhibition of maturation of DC is reversible, parasite-specific and increases with the stage of parasite development, with complete inhibition induced by schizonts (mature infected erythrocytes). **Plasmodium yoelii-infected erythrocytes induce a broad inhibitory effect rendering DC non-responsive to ligands for TLR2, TLR3, TLR4, TLR5, TLR7 and TLR9.**”

<https://www.ncbi.nlm.nih.gov/pubmed/20193084>

Immunosuppressing antigens in Malaria, again:

[FEBS J.](#) 2013 Dec;280(23):6196-212. doi: 10.1111/febs.12541. Epub 2013 Oct 16.

Structure and dynamic behavior of Toll-like receptor 2 subfamily triggered by malarial glycosylphosphatidylinositols of Plasmodium falciparum.

[Durai P1](#), [Govindaraj RG](#), [Choi S](#).

“The recognition of GPIs of the protozoans *P. falciparum* or *Toxoplasma gondii* appears to be via TLR2 and TLR4 [29](#). In an experimental study by Krishnegowda *et al.* [30](#), using mouse macrophages and human monocytes, ***P. falciparum* malarial GPIs consisting of three fatty acid chains were favourably recognized by human and mouse TLR2/TLR1** [30](#). Moreover, one of the derivatives of GPIs called *sn*-2-lyso GPI was the ligand for the hTLR2-hTLR6 complex. The above result was confirmed in another recent experimental study using macrophages from gene knockout mice, in addition to human monocytes and anti-human TLR1 and TLR6 sera [31](#). The ECD of TLR2 has the potential to recognize GPIs in the same binding sites of lipopeptides because the structural patterns of GPIs and lipoproteins are similar, although they are different classes of compounds [30](#). There is sufficient evidence for TLR2 recognition of GPIs; however, the binding site of GPIs and the interacting residues in the protein that would be useful for developing anti-malarial drugs or vaccines are still unknown.

“In the present study, we used some of the methods discussed below to determine the details of the interaction of the TLR2 subfamily with *P. falciparum* Man4-GPI and the *sn*-2 lyso GPI derivative. Molecular docking is a widely used modelling tool for predicting the exact positioning of a ligand in the active site of a protein [32](#). Hence, in the present study, we employed molecular docking to investigate the interactions between *P. falciparum* Man4-GPI and hTLR2-hTLR1 and between *sn*-2 lyso GPI and mTLR2-mTLR6. In addition, MD simulations that can report at the atomic level are appropriate for highlighting the dynamics of a given structure to validate the experimental studies on the ligand-induced dimerization analysis of TLRs [33](#). It is well known that ligands induce dimerization of the TLR2 subfamily [17](#); therefore, by utilizing MD techniques, we simulated the

subfamily of TLR2 for 15 ns as a monomer and dimer in the absence and presence of the GPI to better understand the ligand-induced dimerization and activation mechanism at the atomic level.
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4163636/>

We would expect, naturally, then to find quite a lot of Chronic Fatigue Syndrome in Africa and we do. As an aside, we know not to use antibody studies for finding the herpesviruses in diseases of immunosuppression like this, so any such studies will be thrown out.

J Health Psychol. 2007 May;12(3):461-74.

The prevalence of chronic fatigue syndrome in Nigeria.

Njoku MG1, Jason LA, Torres-Harding SR.

“The present study found adult rates of chronic fatigue syndrome (CFS) in Nigeria that were somewhat higher than rates from community-based CFS epidemiologic studies in the USA. The rates of chronic fatigue for both adults and children were also higher than in existing community-based studies. It is possible that the presence of several fatiguing illnesses such as malaria and typhoid, the lack of adequate healthcare resources and poverty in Nigeria, place individuals at greater risk for fatigue and its syndromes. There is a need for more epidemiologic studies on the prevalence and sociodemographic characteristics of CFS in developing countries.”

<http://www.ncbi.nlm.nih.gov/pubmed/17439996>

Among the other very first things we would like to say about ME/CFS and Fibromyalgia are:

Nat Commun. 2015 Dec 15;6:10145. doi: 10.1038/ncomms10145.

Sepsis induces long-term metabolic and mitochondrial muscle stem cell dysfunction amenable by mesenchymal stem cell therapy.

Rocheteau P1, Chatre L2,3, Briand D1, Mebarki M1, Jouvion G1, Bardon J1, Crochemore C2,3, Serrani P1, Lecci PP1, Latil M1, Matot B4,5, Carlier PG4,5, Latronico N6, Huchet C7, Lafoux A7, Sharshar T1,8,9,10, Ricchetti M2,3, Chrétien F1,10,11,12.

“Sepsis, or systemic inflammatory response syndrome, is the major cause of critical illness resulting in admission to intensive care units. Sepsis is caused by severe infection and is associated with mortality in 60% of cases. Morbidity due to sepsis is complicated by neuromyopathy, and **patients face long-term disability due to muscle weakness, energetic dysfunction, proteolysis and muscle wasting.** These processes are triggered by pro-inflammatory cytokines and metabolic imbalances and are aggravated by malnutrition and drugs. Skeletal muscle regeneration depends on stem (satellite) cells. Herein we show that mitochondrial and metabolic alterations underlie the sepsis-induced long-term impairment of satellite cells and lead to inefficient muscle regeneration. Engrafting mesenchymal stem cells improves the septic status by decreasing cytokine levels, restoring mitochondrial and metabolic function in satellite cells, and improving muscle strength. These findings indicate that sepsis affects quiescent muscle stem cells and that mesenchymal stem cells might act as a preventive therapeutic approach for sepsis-related morbidity.

<https://www.ncbi.nlm.nih.gov/pubmed/26666572>

And:

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[Cell cycle S phase markers are expressed in cerebral ne... Panleukopenia Virus.](#)

1. [Cell cycle S phase markers are expressed in cerebral ne... Panleukopenia Virus.](#)

Poncelet L, Garigliany M, Ando K, Franssen M, Desmecht
Cell Cycle. 2016 Dec 16;15(24):3482-3489. doi: 10.1080/15384101.2016.1278309. PMID: 27830988

We'd like to say "Fibro Herpes" and "Fibro Herpes living in the nerve root ganglia, messing with ion channels and perhaps due to ONGOING INFECTIONS," since duh. Imagine shingles, et al, without the typical, say, loud manifestations.

[Herpes](#). 2006 Nov;13(3):75-80.

Investigations of the pathogenesis of Varicella zoster virus infection in the SCIDhu mouse model.

[Arvin AM1.](#)

"Varicella zoster virus (VZV) is a medically important human herpesvirus that causes varicella, establishes latency in sensory ganglia and may reactivate to cause herpes zoster in healthy and immunocompromised patients. Experiments in the severe combined immunodeficiency (SCID) mouse model have provided new insights about VZV pathogenesis. In addition, the evaluation of VZV recombinant viruses, with targeted mutations of viral genes or their promoters in SCIDhu skin, T-cell and **dorsal root ganglia xenografts, has the potential to identify options for the design of a recombinant 'second-generation' VZV vaccine. This would be characterized by the retention of infectivity in skin combined with a restricted tropism for T-cells and neurons within sensory ganglia.**"

<https://www.ncbi.nlm.nih.gov/pubmed/17147912>

Un-latent herpesviruses might be painful and fatiguing illnesses. CDC officer Suzanne Vernon lies about mycoplasma playing a role in ME/CFS (Occam's Razor) – because such fungal induced immunosuppression reactivates viruses. Some herpes love ganglia, right where, “catastrophizing” “**pressure points**” are. You've already seen that EBV and other viruses may be antibody-negative due to the cross tolerance.

Here, next, a scientist makes a suggestion that infection may be triggering the swelling of the nerve root ganglia (after all, some of the herpesviruses are known to love nerve root ganglia):

Brain (2013) 136 (9): e246., 31 May 2013

Small fibre neuropathy, fibromyalgia and dorsal root ganglia sodium channels

[Manuel Martinez-Lavin](#) DOI: <https://doi.org/10.1093/brain/awt114>

“... Dorsal root ganglia contain the sensory fibres cell bodies. **Trauma and/or infection trigger sympathetic sprouting within dorsal root ganglia through nerve growth factor over-expression.** Such aberrant neuroplasticity enables catecholamines and sympathetic traffic to induce sensory neuron firing. Sodium channels play a pivotal role in this hyperexcitability. A sodium channel isoform (NaV1.7) encoded in gene *SCN9A* of chromosome 2q24.3 is predominantly expressed in the dorsal root ganglia pain-sensing neurons and sympathetic ganglia neurons and their fine-diameter axons... ” ... In a pilot study, we described a particular *SCN9A* sodium channel gene variant (rs6754031 GG genotype) associated with severe fibromyalgia (Vargas-Alarcon *et al.*, 2012). On the other hand, Faber *et al.* (2012) reported that a gain of function mutations in sodium channel NaV1.7, which render dorsal root ganglion neurons hyperexcitable, are present in a substantial proportion (28.6%; 8 of 28) of patients meeting strict criteria for small fibre neuropathy (Faber *et al.*, 2012). **This preliminary information raises the possibility that some cases of fibromyalgia and small fibre neuropathy may have underlying dorsal root ganglia sodium channelopathy.**

The Üçeyler *et al.* (2013) study reinforces our proposal of fibromyalgia as sympathetically maintained neuropathic pain syndrome. Sympathetic dysfunction provides a coherent explanation for the multiple non-pain related fibromyalgia symptoms (Martinez-Lavin, 2012).

<https://academic.oup.com/brain/article/136/9/e246/288267/Small-fibre-neuropathy-fibromyalgia-and-dorsal>

Lenny Sigal, one of the misogynistic Lyme criminals:

[Arthritis Rheum.](#) 2000 Nov;43(11):2493-500.

The role of catastrophizing in the pain and depression of women with fibromyalgia syndrome.

[Hassett AL](#)1, [Cone JD](#), [Patella SJ](#), [Sigal LH](#).

“OBJECTIVE: Although 2 recent studies have found associations between catastrophizing and poor medical outcomes in patients with fibromyalgia syndrome (FMS), neither assessed these findings in comparison with a similar group of patients with chronic pain. Our study examined the complex relationships between depression, catastrophizing, and the multidimensional aspects of pain in women with FMS and compared these relationships with those in women with rheumatoid arthritis (RA).

METHODS: Sixty-four FMS patients and 30 RA patients completed the Coping Strategies Questionnaire (CSQ), the Beck Depression Inventory II (BDI-II), and the McGill Pain Questionnaire.

RESULTS: Compared with subjects with RA, FMS subjects scored significantly higher on the catastrophizing subscale of the CSQ. FMS patients also earned higher scores on overall depression and on the cognitive subscale of the BDI-II. Furthermore, the relationship between catastrophizing and depression was significant in the FMS group only. Regression analyses revealed that in FMS, catastrophizing as a measure of coping predicted patients' perception of pain better than demographic variables such as age, duration of illness, and education.

CONCLUSION: Cognitive factors, such as catastrophizing and depressive self-statements, have a more pronounced role in the self-reported pain of patients with FMS than in patients with RA. Clinically, this indicates that treating pain and depression in FMS by adding cognitive therapy and coping skills components to a comprehensive treatment program may improve the outcomes obtained with pharmacologic interventions.”

<https://www.ncbi.nlm.nih.gov/pubmed/11083273>

[Catastrophizing. Think. We may have just discovered the brain magic behind somatoform illnesses. It must mean “very, very hard thinking and concentrating,” you know, like levitating gurus. We just wonder, why dismiss the magical Fibro-gurus instead of putting them to work for the CIA to stare at goats and discover Russia’s and China’s hidden submarines and underground bases?]

Again on mycoplasma (to which you probably have been tolerized if you have Chronic Fatigue or Fibromyalgia post sepsis syndrome) and how they can cause fatigue by damaging red blood cell membranes, **inhibiting the transfer of oxygen:**

[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>

[Ciba Found Symp.](#) 1981;80:98-118.

Adhesion of mycoplasmas to eukaryotic cells.

[Razin S](#), [Kahane I](#), [Banai M](#), [Bredt W.](#)

“Many pathogenic mycoplasmas are surface parasites, adhering to the epithelial linings of the respiratory and urogenital tracts. Since mycoplasmas lack cell walls their plasma membrane comes in close contact with that of their host, allowing exchange of components between the two membranes and possibly fusion. The tight association of the parasite with its host is illustrated in scanning electron micrographs of Mycoplasma pneumoniae and M. gallisepticum **adhering to human red blood cells.** Specialized structure at the tips of the mycoplasma cells appear to function as attachment organelles. Our main aim has been to chemically define the receptors on the host cell and the binding sites on the mycoplasma cells responsible for adhesion. Glycophorin (the major

sialoglycoprotein of human red blood cells) serves as the main or sole receptor for *M. gallisepticum* whereas *M. pneumoniae* binds to additional receptors on human red blood cells. Trypsin treatment of *M. pneumoniae* cells abolishes their ability to attach to human red cells, suggesting the protein nature of the binding sites. *M. pneumoniae* membranes solubilized by detergents were subjected to affinity chromatography on glycophorin-Sepharose so that membrane components with high affinity for glycophorin could be isolated. The fraction isolated consisted of several proteins (relative molecular mass 25 000 and 45 000). The binding of this fraction to red cells was relatively low but appeared to be specific, as it was inhibited by glycophorin but not by its hydrophobic moiety. **The possibility is discussed that the exposure of the binding sites on the mycoplasma cell surface is influenced by the electrochemical ion gradient across the membrane.**

<http://www.ncbi.nlm.nih.gov/pubmed/6790254>

Here we see again that such fungal antigens inhibit antigen presentation, or result in no antibodies, which is why there typically are no markers in Chronic Fatigue Syndrome or Fibromyalgia:

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.ncbi.nlm.nih.gov/pubmed/11441098>

You have already seen some of these reports, so we will just list a few to remind of the general concept that fungal antigens also **inhibit apoptosis in infected cells**, and mycoplasma, which were fraudulently thrown out by CDC’s Suzanne Vernon, do in fact cause “disease,” even though it might not be with classic “inflammatory” or “autoimmune” signs:

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](#).

“In conclusion, M. fermentans significantly inhibits TNFalpha-induced apoptosis in U937 cells, and its effect is upstream of the mitochondria and upstream of caspase-8.”

<http://www.ncbi.nlm.nih.gov/pubmed/15286682>

[Cell Microbiol](#). 2007 Jan;9(1):142-53. Epub 2006 Aug 2.

The inhibitory effect of Mycoplasma fermentans on tumour necrosis factor (TNF)-alpha-induced apoptosis resides in the membrane lipoproteins.

[Gerlic M1](#), [Horowitz J](#), [Farkash S](#), [Horowitz S](#).

“Mycoplasma have been shown to be involved in the alteration of several eukaryotic cell functions, such as cytokine production, gene expression and more. We have previously reported that infection of human myelomonocytic U937 cell line with live Mycoplasma fermentans (M. fermentans) inhibited tumour necrosis factor (TNF-alpha)-induced apoptosis.”

<http://www.ncbi.nlm.nih.gov/pubmed/16889623>

Mycoplasma cause disease by affecting red blood cells (oxygen) and they inhibit apoptosis in infected other blood immune cells, which is very close to a pre-cancer state. You'll remember from the Occam's Razor or your own casual reading that Rituximab was discovered to be a treatment for Chronic Fatigue/ME because those cancer patients recovered from their Chronic Fatigue Syndrome with that monoclonal antibody.

In other words, yes, Chronic Fatigue/Fibro waste-basketees not surprisingly developed cancer since we are talking about post-sepsis syndrome with the reactivated viruses of all kinds, especially the herpes.

The CDC (Vernon, wow) knows chronic mono or chronic EBV is a chronic fatiguing illness:

[BMC Infect Dis](#). 2006; 6: 15.

Preliminary evidence of mitochondrial dysfunction associated with post-infective fatigue after acute infection with Epstein Barr Virus

[Suzanne D Vernon](#),¹ [Toni Whistler](#),¹ [Barbara Cameron](#),² [Ian B Hickie](#),³ [William C Reeves](#),¹ and [Andrew Lloyd](#)²

BACKGROUND: Acute infectious diseases are typically accompanied by non-specific symptoms including fever, malaise, irritability and somnolence that usually resolve on recovery. However, in some individuals these symptoms persist in what is commonly termed post-infective fatigue. The objective of this pilot study was to determine the gene expression correlates of post-infective fatigue following acute Epstein Barr virus (EBV) infection.

METHODS: We followed 5 people with acute mononucleosis who developed post-infective fatigue of more than 6 months duration and 5 HLA-matched control subjects who recovered within 3 months. Subjects had peripheral blood mononuclear cell (PBMC) samples collected at varying time points

including at diagnosis, then every 2 weeks for 3 months, then every 3 months for a year. Total RNA was extracted from the PBMC samples and hybridized to microarrays spotted with 3,800 oligonucleotides.

RESULTS: 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.

In the full text they write:

“...Acute viral diseases such as infectious mononucleosis typically present clinically with a cluster of non-specific symptoms including; fever, an increased need to sleep, hyperalgesia, anorexia, loss of interest in usual activities, social interaction, body care, depressed mood, and impaired concentration [1-3]. This acute sickness behavior response comprises a highly organized and evolved disease-fighting strategy mediated by the action of pro-inflammatory cytokines [4-8]. In general, acute sickness behavior resolves in parallel with clearance or control of the infecting agent. However, some individuals exhibit prolonged illness with fatigue, mood changes and cognitive impairment. **Such prolonged illness following infectious mononucleosis has been recognized for at least half a century** [9]. Recent studies of infectious mononucleosis due to EBV infection demonstrated that fatigue, sore throat and malaise persisted for up to two months in approximately 40% of patients and for six or more months in approximately 10% [10,11].”

<https://www.ncbi.nlm.nih.gov/pubmed/16448567>

Everyone with Chronic Fatigue/ME and Fibromyalgia has known for years that the CDC pooh-pah'd the idea that CFIDS/ME was about chronic Epstein-Barr or about any chronic common viral infection. Yet, here they are saying, “Oh-yeah, this has been known for 50 years...”

Garth Nicolson on mycoplasma and chronic fatigue:

APMIS. 2003 May;111(5):557-66.

Multiple co-infections (Mycoplasma, Chlamydia, human herpes virus-6) in blood of chronic fatigue syndrome patients: association with signs and symptoms.

Nicolson GL1, Gan R, Haier J.

“Previously we and others found that a majority of chronic fatigue syndrome (CFS) patients showed evidence of systemic mycoplasmal infections, and their blood tested positive using a polymerase chain reaction assay for at least one of the four following Mycoplasma species: M. fermentans, M. hominis,

M. pneumoniae or *M. penetrans*. Consistent with previous results, patients in the current study (n=200) showed a high prevalence (overall 52%) of mycoplasmal infections. Using forensic polymerase chain reaction we also examined whether these same patients showed evidence of infections with *Chlamydia pneumoniae* (overall 7.5% positive) and/or active human herpes virus-6 (HHV-6, overall 30.5% positive). Since the presence of one or more infections may predispose patients to other infections, we examined the prevalence of *C. pneumoniae* and HHV-6 active infections in mycoplasma-positive and -negative patients. Unexpectedly, we found that the incidence of *C. pneumoniae* or HHV-6 was similar in Mycoplasma-positive and -negative patients, and the converse was also found in active HHV-6-positive and -negative patients. Control subjects (n=100) had low rates of mycoplasmal (6%), active HHV-6 (9%) or chlamydial (1%) infections, and there were no co-infections in control subjects. Differences in bacterial and/or viral infections in CFS patients compared to control subjects were significant. Severity and incidence of patients' signs and symptoms were compared within the above groups. Although there was a tendency for patients with multiple infections to have more severe signs and symptoms ($p < 0.01$), the only significant differences found were in the incidence and severity of certain signs and symptoms in patients with multiple co-infections of any type compared to the other groups ($p < 0.01$). There was no correlation between the type of co-infection and severity of signs and symptoms. The results indicate that a large subset of CFS patients show evidence of bacterial and/or viral infection(s), and these infections may contribute to the severity of signs and symptoms found in these patients.”

<http://www.ncbi.nlm.nih.gov/pubmed/12887507>

That sounds exactly like post-sepsis syndrome as shown in the Occam's Razor.

Next, suppression of immune markers and cytokines in Chronic Fatigue Syndrome, pointing to the disease not being about inflammation or autoimmunity, but the opposite, immunosuppression or post-sepsis syndrome; look at this chart:

[Clin Diagn Lab Immunol](#). 1999 Jan;6(1):6-13.

Changes in immune parameters seen in Gulf War veterans but not in civilians with chronic fatigue syndrome.

[Zhang Q1](#), [Zhou XD](#), [Denny T](#), [Otteweller JE](#), [Lange G](#), [LaManca JJ](#), [Lavietes MH](#), [Pollet C](#), [Gause WC](#), [Natelson BH](#).

Look closely at the Table 2 – all the markers are lower in Chronic Fatigue than normals. This is a disease of immune suppression and not inflammation or autoimmunity. This is post-sepsis syndrome, same as “Chronic Lyme.”

TABLE 2

Means and standard errors for cytokines and CD cell surface markers

Cell type, phenotype, or cytokine	Gulf War veterans						Civilians					
	n ^f	CFS		n	Control		n	CFS		n	Control	
		Mean	SE		Mean	SE		Mean	SE		Mean	SE
No. of ^a :												
WBC	42	6,564.29	262.54	33	6,233.33	253.67	43	6,516.28	284.89	39	6,064.1	213.08
Lymphocytes	42	2,119.76	103.81	33	1,918.33	96.03	43	1,826.63	75.93	39	1,874.36	68.69
CD(16+56) ^{+a}	42	261.10	28.72	33	290.70	26.13	43	180.74	13.69	39	196.08	15.11
CD19 ⁺	42	248.07	19.63	33	262.18	28.33	43	256.86	18.37	39	252.46	16.23
CD3 ⁺	42	1,613.62	78.65	33	1,373.58	72.47	43	1,385.86	63.84	39	1,425.95	57.89
CD3 ⁺ CD4 ⁺	42	1,014.69	51.62	33	809.15	45.34	43	889.93	41.29	39	927.26	40.77
CD3 ⁺ CD8 ⁺	42	567.62	34.95	33	515.33	42.20	43	458.37	28.99	39	476.85	32.11
% Lymphocytes ^b												
CD(16+56) ^{+c}	42	12.24	0.94	33	15.52	1.12	43	10.19	0.73	39	10.62	0.76
CD19 ^{+c}	42	11.55	0.67	33	13.03	1.02	43	14.23	0.80	39	13.38	0.66
CD3 ^{+c}	42	76.36	0.93	33	71.85	1.23	43	75.53	1.21	39	75.87	0.97
CD3 ⁺ CD4 ^{+c}	42	48.31	1.01	33	42.45	1.39	43	48.60	1.03	39	49.59	1.18
CD3 ⁺ CD8 ^{+c}	42	26.62	1.11	33	26.73	1.39	43	24.81	0.97	39	25.72	1.20
CD4 ⁺ CD45RO ^{+d}	40	71.40	1.85	33	72.18	2.12	43	67.83	2.26	39	70.51	2.44
CD4 ⁺ CD45RA ^{+d}	40	42.80	1.73	33	40.76	2.18	40	46.23	1.70	39	43.18	1.79
CD8 ⁺ CD28 ^{+d}	41	58.17	1.96	33	59.45	2.25	40	67.79	1.98	39	65.05	2.23
CD8 ⁺ CD38 ^{+d}	41	51.56	2.35	33	53.12	2.74	40	58.08	1.92	38	51.10	1.77
CD8 ⁺ HLA-DR ^{+d}	41	20.90	1.69	33	20.73	2.23	40	19.85	2.14	39	22.82	2.18
CD8 ⁺ CD11b ^{+d}	40	56.20	2.49	32	53.44	2.95	40	69.58	2.24	39	61.66	3.64
Cytokines ^e												
IL-2	43	430.95	140.23	34	251.97	61.17	68	77.93	13.02	53	95.48	17.24
IL-4	43	256.33	58.06	34	134.11	18.79	68	16.90	2.58	53	18.74	2.61
IL-6	43	2,882.11	505.21	34	1,710.95	337.08	68	98.21	32.49	53	281.42	112.87
IL-10	43	603.84	136.88	34	495.95	265.11	68	333.42	48.06	53	532.42	170.98
IL-12	43	299.55	84.64	34	136.37	38.89	68	463.22	66.90	53	656.63	159.95
TNF- α	43	288.62	48.37	34	166.35	27.28	68	140.84	16.80	53	182.93	19.90
IFN- γ	43	1,002.06	163.52	34	632.74	146.11	68	736.37	113.41	53	986.20	246.56

<https://www.ncbi.nlm.nih.gov/pubmed/9874656>
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC95652/>

Does this next report need narration or interpretation?

[Biochem Biophys Res Commun](#). 2005 Jul 29;333(2):438-42.

Epstein-Barr virus immediate-early proteins BZLF1 and BRLF1 alter mitochondrial morphology during lytic replication.

[LaJeunesse DR1](#), [Brooks K](#), [Adamson AL](#).

Epstein-Barr virus (EBV) is a human DNA virus that is responsible for the syndrome infectious mononucleosis, and is associated with several forms of cancer. During both lytic and latent viral infection, viral proteins manipulate the host's cellular components to aid in viral replication and maintenance. **Here, it is demonstrated that induction of EBV lytic replication results in a dramatic reorganization of mitochondria accompanied by a significant alteration of mitochondrial membrane potential and a rapid and transient increase in the microtubular cytoskeleton.** Moreover, we show that expression of the EBV immediate-early genes BZLF1 and BRLF1 contributes to the mitochondrial alteration but not the increase in the microtubule cytoskeleton, suggesting that the mechanism for the observed cytoplasmic restructuring involves a number of coordinated viral and host proteins.

<http://www.ncbi.nlm.nih.gov/pubmed/15950179>

No. Chronic Active EBV (CAEBV) could be, shall we say, fatiguing by damaging mitochondria. And not show any antibody signs.

17) Next, 4 reports on Seronegative Epstein-Barr (you have already seen this in the other charge sheets). It means you can have chronic active Epstein-Bar or other herpes viruses (some living in the ganglia, hello Fibromyalgia) but in these immunosuppression cases, you will not see the typical slightly elevated antibody titer associated with reactivated viruses in non-immunocompromised or non-post-sepsis individuals:

[From the Occam's Razor report, number 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 human 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.

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.

Resing 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.

AND, we know Lupus and MS are EBV-linked outcomes from post-Lyme sepsis. In those cases, those victims have the EBV-linked hypersensitivity association or some other mechanism that looks like those outcomes are “autoimmunity.” But as we know, Chronic Fatigue and Fibromyalgia are the same Lupus-and-MS-outcomes-of-the-Great-Imitators-Lyme-and-Syphilis, but without the autoimmunity.

This next report, of course says be careful when considering OspA as a chemo adjuvant because it is known to cause the same immunosuppression and inhibition of apoptosis as we mentioned here previously. What happens when OspA causes the inhibition of apoptosis especially in EBV infected cells? Right. The reactivation of those herpesviruses. Fungally contaminated vaccines? The kids are getting the viruses instead of the protection.

18) Here it says to please worry that too much TLR2 agonism such as Lyme, LYMERix, mycoplasma, etc. could be dangerous due to the immunosuppression, while Yale and the CDC said LYMERix or OspA was a vaccine, the opposite, ho hum:

[J Leukoc Biol](#). 2013 Jun;93(6):847-63. doi: 10.1189/jlb.1012501. Epub 2013 Mar 8.

TLR agonists: our best frenemy in cancer immunotherapy.

[Kaczanowska S1](#), [Joseph AM](#), [Davila E](#).

“TLR2 stimulation on human CD4+CD45RO+ memory cells also induces IFN- γ production, and these levels are increased when combined with IL-2 [43, 48]. Lipoproteins from *Mycobacterium tuberculosis*, a TLR2 agonist, can stimulate memory CD4+ T cells directly, resulting in enhanced proliferation, as well as IL-2 and IFN- γ production. Although resting CD4+ T cells responded to lipoproteins, as evidenced through NF- κ B activation, such as CD8 T cells, CD4 T cells also required concomitant TCR signaling to induce proliferation and cytokine production [69]. *** **In addition to enhancing T cell effector function, TLR2 agonists have been shown to promote T cell longevity and are associated with increased expression of antiapoptotic molecules A1 and Bcl-xL and down-regulation of the proapoptotic protein Bim [43, 53].** ***

<http://www.ncbi.nlm.nih.gov/pubmed/23475577>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3656332/>

Right, OspA acts like a BCL2 class molecule, inhibiting apoptosis, not to mention the intracellular damage and the reactivation of latent herpes viruses and what-not.

Fungal antigens straight up activate Epstein-Barr:

[J Virol](#). 2010 Apr;84(7):3612-23. doi: 10.1128/JVI.01400-09. Epub 2010 Jan 20.

Toll-like receptor agonists synergistically increase proliferation and activation of B cells by epstein-barr virus.

[Iskra S1](#), [Kalla M](#), [Delecluse HJ](#), [Hammerschmidt W](#), [Moosmann A](#).

“Epstein-Barr virus (EBV) efficiently drives proliferation of human primary B cells in vitro, a process relevant for human diseases such as infectious mononucleosis and posttransplant lymphoproliferative disease. Human B-cell proliferation is also driven by ligands of Toll-like receptors (TLRs), notably viral or bacterial DNA containing unmethylated CpG dinucleotides, which triggers TLR9. Here we quantitatively investigated how TLR stimuli influence EBV-driven B-cell proliferation and expression of effector molecules. CpG DNA synergistically increased EBV-driven proliferation and transformation, T-cell costimulatory molecules, and early production of interleukin-6. CpG DNA alone activated only memory B cells, but CpG DNA enhanced EBV-mediated transformation of both memory and naive B cells. Ligands for TLR2 or TLR7/8 or whole bacteria had a weaker but still superadditive effect on B-cell transformation. Additionally, CpG DNA facilitated the release of transforming virus by established EBV-infected lymphoblastoid cell lines. These results suggest that the proliferation of EBV-infected B cells and their capability to interact with immune effector cells may be directly influenced by components of bacteria or other microbes present at the site of infection.”

<http://www.ncbi.nlm.nih.gov/pubmed/20089650>

So, that is a fair amount of evidence for people dealing with what they think is ME/CFS or Fibromyalgia, it is basically the same as post sepsis syndrome or Lyme.

19) What about Diagnosing this/these. Welp, believe it or not, we can thank IDSA:

[J Clin Microbiol](#). 2014 Jan;52(1):212-7. doi: 10.1128/JCM.02270-13. Epub 2013 Nov 6.

Virological diagnosis of central nervous system infections by use of PCR coupled with mass spectrometry analysis of cerebrospinal fluid samples.

[Lévêque N1](#), [Legoff J](#), [Mengelle C](#), [Mercier-Delarue S](#), [N'guyen Y](#), [Renois F](#), [Tissier F](#), [Simon F](#), [Izopet J](#), [Andréoletti L](#).

“Viruses are the leading cause of central nervous system (CNS) infections, ahead of bacteria, parasites, and fungal agents. A rapid and comprehensive virologic diagnostic testing method is needed to improve the therapeutic management of hospitalized pediatric or adult patients. In this study, we assessed the clinical performance of PCR amplification coupled with electrospray ionization-time of flight mass spectrometry analysis (PCR-MS) for the diagnosis of viral CNS infections. Three hundred twenty-seven cerebrospinal fluid (CSF) samples prospectively tested by routine PCR assays between 2004 and 2012 in two university hospital centers (Toulouse and Reims, France) were retrospectively analyzed by PCR-MS analysis using primers targeted to adenovirus, human **herpesviruses 1 to 8** (HHV-1 to -8), polyomaviruses BK and JC, parvovirus B19, and **enteroviruses** (EV). PCR-MS detected single or multiple virus infections in 190 (83%) of the 229 samples that tested positive by routine PCR analysis and in 10 (10.2%) of the 98 samples that tested negative. The PCR-MS results correlated well with herpes simplex virus 1 (HSV-1), varicella-zoster virus (VZV), and EV detection by routine PCR assays (kappa values [95% confidence intervals], 0.80 [0.69 to 0.92], 0.85 [0.71 to 0.98], and 0.84 [0.78 to 0.90], respectively), whereas a weak correlation was observed with Epstein-Barr virus (EBV) (0.34 [0.10 to 0.58]). Twenty-six coinfections and 16 instances of uncommon neurotropic viruses (HHV-7 [n = 13], parvovirus B19 [n = 2], and adenovirus [n = 1]) were identified

by the PCR-MS analysis, whereas only 4 coinfections had been prospectively evidenced using routine PCR assays ($P < 0.01$). In conclusion, our results demonstrated that PCR-MS analysis is a valuable tool to identify common neurotropic viruses in CSF (with, however, limitations that were identified regarding EBV and EV detection) **and may be of major interest in better understanding the clinical impact of multiple or neglected viral neurological infections.**"

<http://www.ncbi.nlm.nih.gov/pubmed/24197874>

Neglected Viral Infections. Yes, thank you.

COMPARE that to this IDSociety.org position paper on the issue of using rapid mass-spec PCR on spinal fluid samples for rapid detection of the CNS infections the NIH knows is driving Chronic Fatigue and Chronic Lyme:

"Unmet diagnostic needs in infectious disease"

"1. Introduction

"The importance of diagnostic testing in the management of infectious diseases (ID) was recently highlighted in the report of the Infectious Diseases Society of America's (IDSA) Diagnostics Task Force report: "Better Tests: Better Care: Improved Diagnostics for Infectious Diseases" (Caliendo et al., 2013). Similar sentiments are expressed in the report on Antibiotic Resistance Threats in the United States Centers for Disease Control (2013) from the Centers for Disease Control and Prevention (CDC). ******A number of new diagnostic technologies for ID are rapidly emerging: e.g., broad-range PCR, next-generation sequencing, and matrix-assisted laser desorption/ionization time of flight mass spectrometry.***** The reports from the IDSA and the CDC highlight deficiencies in current diagnostic methods and call for approval and access to methods that are rapid and available at the point of care, use direct-from-specimen analysis, and demonstrate high levels of sensitivity and specificity across a wide range of disease syndromes. The importance of syndrome-based panels (e.g., for central nervous system, bloodstream and respiratory tract infections) is highlighted in the IDSA report (Caliendo et al., 2013). Both the IDSA and CDC emphasize the critical need for culture-independent testing for specific pathogens and their pattern of susceptibility to antimicrobial agents...."

http://ein.idsociety.org/media/publications/papers/2014/Blaschke_DMID_14_Unmet_Diagnostic_Needs.pdf

Idsociety's "Policy Paper" on the same, rapid diagnostics (MassSpec-PCR. But that can't fit in a test kit, see, so there is no profit in it for the IDSA and CDC DNA profiteers. Superbugs will continue to kill people and there will be more calamities of the hospital acquired and new infection sort. And more of the Ebola and MERS and SARS sort.... If there is no money to be made, IDSA is not interested.

Better Tests, Better Care: Improved Diagnostics for Infectious Diseases

Angela M. Caliendo,¹ David N. Gilbert,^{2,3} Christine C. Ginocchio,^{4,5,6} Kimberly E. H...

http://www.idsociety.org/uploadedFiles/IDSA/Policy_and_Advocacy/Current_Topics_and_Issues/Diagnostics/Clin%20Infect%20Dis.-2013-Caliendo-S139-70.pdf

[“Won-der-ful,” as the rich people in Fairfield county, Connecticut say.]



TRUTHCURES.ORG
healthcare justice

CHARGE SHEET 7: WESSELY & PSYCH ABUSE AS "MEDICINE"

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

© 2017, February, Society for the Advancement of Scientific Hermeneutics, (\$A\$H).

Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 7: Wessely & Psych Abuse as "Medicine"

7.) Simon Wessely, Gulf War Illness, and Somatoformia, the current real definition.

CONJURMANIA!!

As long as psychiatry is completely made up and the entire vocabulary of the DSM is made up, it's a free for all, right?

So, Wessely claims Gulf War Illness Veterans are just plain cowards, basically.

The British "psychiatrist," Simple Simon, was hired by the U.S. Pentagon to trash Gulf War Illness veterans, while he totally knew otherwise:

<http://www.gresham.ac.uk/lectures-and-events/something-old-something-new-something-borrowed-something-blue-the-true-story-of>

Yet, this is a report Wessely wrote for the Pentagon:

BMJ. 2000 May 20;320(7246):1363-7.

Role of vaccinations as risk factors for ill health in veterans of the Gulf war: cross sectional study.

Hotopf M1, David A, Hull L, Ismail K, Unwin C, Wessely S.

"Among veterans of the Gulf war there is a specific relation between multiple vaccinations given during deployment and later ill health. Multiple vaccinations in themselves do not seem to be harmful but combined with the "stress" of deployment they may be associated with adverse health outcomes. These results imply that every effort should be made to maintain routine vaccines during peacetime."

<http://www.ncbi.nlm.nih.gov/pubmed/10818024>

Wessely later (see above, "Something Old, Something New...") made these statements about the sick veterans:

"One way of doing that is through neuro-imaging, but we didn't get the money to do that, so instead we have used sophisticated neuro-psychological testing."

"Those tablets, the NAPS tablets, it's just not possible to study. Pesticides, we don't find evidence. Chemical weapons, well, we don't think that for the British armed forces that was a big issue. But we do think there is a relationship between a **particular pattern of protection** and what happened later."

Neither of those claims are scientifically valid. "We didn't get the money to do real science testing so we just used the invalid subjective nonsense tests."

And, "We just plain can't be bothered to look at the very things that may cause immunosuppression on top of the hypervaccination, so we'll just call the veterans cowards instead. Me, Simon Wessely,

calling soldiers cowards. I know it's hilarious but I am paid very well for this slander and libel, plus it's hazard pay. Probably these vets and CFIDS/ME victims I abuse similarly would like to punch me in the face."

Kinda, yeah.

Those nerve agent antidote tablets? They cause immunosuppression, as does DEET.

(Sounds familiar, though, right? Hypervaccination in the presence of immune suppressors like fungi?)

[Immunopharmacol Immunotoxicol](#). 2004 Feb;26(1):1-15.

Pyridostigmine bromide (PYR) alters immune function in B6C3F1 mice.

[Peden-Adams MM1](#), [Dudley AC](#), [EuDaly JG](#), [Allen CT](#), [Gilkeson GS](#), [Keil DE](#).

"Pyridostigmine bromide (PYR) is an anticholinesterase drug indicated for the treatment of myasthenia gravis and neuromuscular blockade reversal. It acts as a reversible cholinesterase inhibitor and was used as a pretreatment for soldiers during Operation Desert Storm to protect against possible nerve gas attacks. Since that time, PYR has been implicated as a possible causative agent contributing to Gulf War Illness. PYR's mechanism of action has been well-delineated with regards to its effects on the nervous system, yet little is known regarding potential effects on immunological function. To evaluate the effects of PYR on immunological function, adult female B6C3F1 mice were gavaged daily for 14 days with PYR (0, 1, 5, 10, or 20mg/kg/day). Immune parameters assessed were lymphoproliferation, natural killer cell activity, the SRBC-specific antibody plaque-forming cell (PFC) response, thymus and spleen weight and cellularity, and thymic and splenic CD4/CD8 lymphocyte subpopulations. Exposure to PYR did not alter splenic and thymus weight or splenic cellularity. However, 20 mg PYR/kg/day decreased thymic cellularity with decreases in both CD4+/CD8+ (20 mg/kg/day) and CD4-/CD8- (10 and 20 mg/kg/day) cell types. Functional immune assays indicated that lymphocyte proliferative responses and natural killer cell activity were normal; whereas exposure to PYR **significantly decreased primary IgM antibody responses to a T-cell dependent antigen at the 1, 5, 10 and 20 mg/kg treatment levels for 14 days. This is the first study to examine the immunotoxicological effects of PYR and demonstrate that this compound selectively suppresses humoral antibody responses.**"

<http://www.ncbi.nlm.nih.gov/pubmed/15106728>

Repeat: "This is the first study to examine the immunotoxicological effects of PYR and demonstrate that this compound selectively **suppresses humoral antibody responses.**"

DEET and Immunosuppression:

[Toxicol Sci](#). 2009 Mar;108(1):110-23. doi: 10.1093/toxsci/kfp001. Epub 2009 Jan 13.

N,N-diethyl-m-toluamide (DEET) suppresses humoral immunological function in B6C3F1 mice.

[Keil DE1](#), [McGuinn WD](#), [Dudley AC](#), [EuDaly JG](#), [Gilkeson GS](#), [Peden-Adams MM](#).

"Significant decreases were observed in the percentage of splenic CD4-/CD8- and CD4+/CD8-

lymphocytes but only at the 62 mg DEET/kg/day treatment level and not in absolute numbers of these cells types. Additionally, significant decreases in the antibody PFC response were observed following treatment with 15.5, 31, or 62 mg DEET/kg/day. Pharmacokinetic (PK) data from the current study indicate 95% bioavailability of the administered dose. Therefore, it is likely that DEET exposure ranges applied in this study are comparable to currently reported occupational usage. Together, the evidence for immunosuppression and available PK data suggest a potential human health risk associated with DEET in the occupational or military environments assuming similar sensitivity between human and rodent responses.”

<http://www.ncbi.nlm.nih.gov/pubmed/19141786>

DEET and Immunosuppression, especially combined with Nerve Agent Antidote:

[Toxicol Ind Health](#). 2001 Jun;17(5-10):192-209.

Evaluation of immunotoxicity induced by single or concurrent exposure to N,N-diethyl-m-toluamide (DEET), pyridostigmine bromide (PYR), and JP-8 jet fuel.

[Peden-Adam MM1](#), [Eudaly J](#), [Eudaly E](#), [Dudley A](#), [Zeigler J](#), [Lee A](#), [Robbs J](#), [Gilkeson G](#), [Keil DE](#).

“Approximately 5,000 to 80,000 of the US service personnel involved in the Persian Gulf War have complained of a variety of nonspecific symptoms since their return in 1991. These symptoms have been collectively labeled Gulf War Illness and include muscle fatigue, general malaise, myalgia, impaired cognition, ataxia, headaches, fever, joint pain, skin rash, gastrointestinal disturbances, sleep disturbances, and respiratory difficulties. **Exposures of military and service personnel were diverse and included the prescribed anti-nerve gas agent pyridostigmine bromide (PYR), N.N-diethyl-m-toluamide (DEET) insect repellent, and environmental exposures to jet fuel.** Thus, studies in our laboratory were undertaken to determine if concurrent exposure to these agents, singly or in combination, would contribute to significant alterations in immunological function and disease susceptibility. To assess immune status, eight-week old B6C3F1 female mice were exposed for 14 days to single compounds or tertiary mixtures of 15.5 mg/kg DEET, 2 mg/kg PYR, and 500 mg/kg JP-8 (termed low dose), or 31 mg/kg DEET, 5 mg/kg PYR, and 1,000 mg/kg JP-8 (termed high dose). Immunosuppression was assessed 24 h after the last exposure. No remarkable alterations were evident in hematological parameters, spleen and thymus organ weight and total cellularity, natural killer (NK) cell activity, cytotoxic T-cell activity, or mitogen-induced lymphocyte proliferation after exposure to either single or tertiary mixtures at low or high doses. A few changes in CD4/CD8 flow cytometric lymphocyte subpopulations were detected after exposure to the tertiary mixture at the high dose. Delayed type hypersensitivity (DTH) was decreased by 88% after exposure to the high-dose mixture, and suppression of antibody-specific IgM immune responses (plaque-forming cell, PFC) occurred after exposure to all single and tertiary mixtures at both dose levels. In the PFC response, antagonism was apparent in the mixture, while coexposure to these agents resulted in a synergistic effect in the DTH response. Susceptibility to B16F10 tumor or *Listeria monocytogenes* challenge was not affected after single or tertiary exposures. **These data suggest that combined exposure to DEET, PYR, and JP-8 does not profoundly alter many immunological endpoints, but does selectively target functional endpoints such as the PFC and DTH response. This should be considered when assessing human health risks in the military environment.**”

<https://www.ncbi.nlm.nih.gov/pubmed/12539864>

Amazing, someone got the money to do real science and were not too cowardly to slough it all off (like a snake) as somatochondria and then say, “Knight me.” And, oh, guess what, about the veterans (I know, sorry to take the attention off Silly Simon), they can have an immunological effect from hypervaccination or too-many-not-really vetted vaccines.

More scientifically valid data: Garth Nicolson on Mycoplasma (the fungal contaminant against which they put Thimerosal in vaccines) in Gulf War Illness veterans:

[Science](#). 2001 May 4;292(5518):853.

Continuing research into Gulf War illness.

[Nicolson G.](#)

Summary The presence of systemic mycoplasmal infections in the blood of Gulf War veterans ($n=8$) and civilians ($n=28$) with Amyotrophic Lateral Sclerosis (ALS) and age matched controls ($n=70$) was investigated by detecting mycoplasma gene sequences with forensic Polymerase Chain Reaction (PCR) and back hybridization with a radiolabeled internal oligonucleotide probe. Almost all ALS patients (30/36 or ~83%) showed evidence of *Mycoplasma* species in blood samples, whereas <9% of controls had blood mycoplasmal infections ($P<0.001$). Using PCR ALS patients with a positive test for any mycoplasmal infection were investigated for the presence of *M. fermentans*, *M. pneumoniae*, *M. hominis* and *M. penetrans* in their blood. All Gulf War veterans with ALS were positive for *M. fermentans*, except one that was positive for *M. genitalium*. In contrast, the 22/28 civilians with detectable mycoplasmal infections had *M. fermentans* (13/22, 59%) as well as other *Mycoplasma* species in their blood, and two of the civilian ALS patients had multiple mycoplasma species (*M. fermentans* plus *M. hominis*). Of the few control patients that were positive, only two patients (2/70, 2.8%) were positive for *M. fermentans* ($P<0.001$). The results support the suggestion that infectious agents may play a role in the pathogenesis and/or progression of ALS, or alternatively ALS patients are extremely susceptible to systemic mycoplasmal infections.
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“**Summary** The presence of systemic mycoplasmal infections in the blood of Gulf War veterans ($n=8$) and civilians ($n=28$) with Amyotrophic Lateral Sclerosis (ALS) and age matched controls ($n=70$) was investigated by detecting mycoplasma gene sequences with forensic Polymerase Chain Reaction (PCR) and back hybridization with a radiolabeled internal oligonucleotide probe. Almost all ALS patients (30/36 or ~83%) showed evidence of *Mycoplasma* species in blood samples, whereas <9% of controls had blood mycoplasma infections ($P<0.001$). Using PCR ALS patients with a positive test for any mycoplasmal infection were investigated for the presence of *M. fermentans*, *M. pneumoniae*, *M. hominis*, and *M. penetrans* in their blood. All Gulf War veterans with ALS were positive for *M. fermentans*, except one that was positive for *M. genitalium*. In contrast, the 22/28 civilians with detectable mycoplasmal infections had *M. fermentans* (13/22, 59%) as well as other *Mycoplasma* species in their blood, and two of the civilian ALS patients had multiple mycoplasma species (*M. fermentans* plus *M. hominis*). Of the few control patients that were positive, only two patients (2/70, 2.8%) were positive for *M. fermentans* ($P<0.001$). The results support the suggestion that infectious agents may play a role in the pathogenesis and/or progression of ALS, or alternatively ALS patients are extremely susceptible to systemic mycoplasmal infections.”

<https://www.ncbi.nlm.nih.gov/pubmed/11341275>

<http://science.sciencemag.org/content/292/5518/853.2.long>

<http://www.actionlyme.org/GARTHNICOLSON.pdf>

Naturally we wonder if any of those experimental vaccines the Gulf War veterans were stabbed with were contaminated.

We've seen in previous SASH criminal charge sheets for the Justice Department, that mycoplasma causes fatigue via hypoxia via erythrocyte membrane osmotic changes and changes to mitochondria, etc.

More scientifically valid data – seen previously:

[Clin Diagn Lab Immunol](#). 1999 Jan;6(1):6-13.

Changes in immune parameters seen in Gulf War veterans but not in civilians with chronic fatigue syndrome.

[Zhang Q1](#), [Zhou XD](#), [Denny T](#), [Otteweller JE](#), [Lange G](#), [LaManca JJ](#), [Lavietes MH](#), [Pollet C](#), [Gause WC](#), [Natelson BH](#).
<https://www.ncbi.nlm.nih.gov/pubmed/9874656>
<http://www.pubmedcentral.nih.gov/picrender.fcgi?artid=95652&blobtype=pdf>

Next, ... and this is all not to mention that during the first Iraq War in 1991, CNN showed a video clip where the soldiers were all standing around unprotected as they blew up the buried chemical and biological weapons... and the earth moved and the dust rose...:

2014: *New research links Iraq dust to ill soldiers*

"An Armed Forces Health Surveillance Center report from 2012 also showed a 150 per 1,000 rate of clinic visits for respiratory diseases before the wars in Iraq and Afghanistan, and a rate of 173 per 1,000 rate during the war years."

<http://www.usatoday.com/story/news/nation/2014/06/02/lung-study-va/9771237/>

Now let's take a look at how you can have cortisol-reactivated Epstein-Barr **virus if you are an astronaut or medical school student** (clue, sleep-wake cycle) in the National Library of Medicine/pubmed:

<http://www.ncbi.nlm.nih.gov/pubmed/?term=epstein-barr+and+astronauts>

Results: 9

[Multiple latent viruses reactivate in astronauts during Space Shuttle missions.](#)

Mehta SK, Laudenslager ML, Stowe RP, Crucian BE, Sams CF, Pierson DL.
 BrainBehav Immun. 2014 Oct;41:210-7. doi:10.1016/j.bbi.2014.05.014. Epub 2014 Jun 2.

[Latent and lytic Epstein-Barr virus gene expression in the peripheral blood of astronauts.](#)

Stowe RP, Kozlova EV, Sams CF, Pierson DL, Walling DM.
 Brain Behav Immun. 2005 May;19(3):235-42.

[Epstein-Barr virus shedding by astronauts during space flight.](#)

Pierson DL, Stowe RP, Phillips TM, Lugg DJ, Mehta SK.
BrainBehav Immun. 2005 May;19(3):235-42.

[Immune function during space flight.](#)

Sonnenfeld G, Shearer WT.
Nutrition. 2002 Oct;18(10):899-903. Review.

[Elevated stress hormone levels relate to Epstein-Barr virus reactivation in astronauts.](#) Stowe RP,

Pierson DL, Barrett AD.
PsychosomMed. 2001 Nov-Dec;63(6):891-5.

[Immune responses and latent herpesvirus reactivation in spaceflight.](#)

Stowe RP, Mehta SK, Ferrando AA, Feedback DL, Pierson DL.
AviatSpace Environ Med. 2001 Oct;72(10):884-91.

[Space analogue studies in Antarctica.](#)

Lugg D, Shepanek M.
ActaAstronaut. 1999 Apr-Jun;44(7-12):693-9.

[Stress-induced reactivation of Epstein-Barr virus in astronauts.](#)

Stowe RP, Pierson DL, Feedback DL, Barrett AD.
Neuroimmunomodulation.2000;8(2):51-8.

[Incidence of Epstein-Barr virus in astronaut saliva during spaceflight.](#)

Payne DA, Mehta SK, Tyring SK, Stowe RP, Pierson DL.
AviatSpace Environ Med. 1999 Dec;70(12):1211-3.

Simon Says: if you're an astronaut, you may have a real disease. If a soldier or some other commoner, you may have a Salem Witch trial (no, really, wait until you see the exact definition of Somatoform).

Medical School Stress (it's baffling why they'd be stressed, after all, there is no science involved):

[Health Psychol.](#) 1993 Nov;12(6):435-42.

Stress and the memory T-cell response to the Epstein-Barr virus in healthy medical students.

[Glaser R](#), [Pearson GR](#), [Bonneau RH](#), [Esterling BA](#), [Atkinson C](#), [Kiecolt-Glaser JK](#).

“This study investigated the memory T-cell proliferative response to several early and late Epstein-Barr virus (EBV) polypeptides. Blood samples were collected twice, 1 month before a 3-day block of examinations and again on the last day of the exam series. Ss were 25 healthy, EBV seropositive medical students. The proliferative response to 5 of the 6 EBV polypeptides significantly decreased during examinations. In addition, Ss high (above the median) in seeking support, as measured by the COPE, had lower proliferative responses to 3 EBV polypeptides (p17, p52/50, and p85), as well as higher levels of antibody to EBV virus capsid antigen. **The data provide further evidence that psychological stress can modulate the cellular immune response to latent EBV.**”

<http://www.ncbi.nlm.nih.gov/pubmed/8293726>

The above report was cited by... 12 more reports:

http://www.ncbi.nlm.nih.gov/pubmed?linkname=pubmed_pubmed_citedin&from_uid=8293726

This one among them:

[Interdiscip Perspect Infect Dis](#). 2011;2011:571340. doi: 10.1155/2011/571340. Epub 2011 Dec 20.

Fatigue in medical residents leads to reactivation of herpes virus latency.

[Uchakin PN1](#), [Parish DC](#), [Dane FC](#), [Uchakina ON](#), [Scheetz AP](#), [Agarwal NK](#), [Smith BE](#).

“The main objective of this study was to detect fatigue-induced clinical symptoms of immune suppression in medical residents. Samples were collected from the subjects at rest, following the first night (low-stress), and the last night (high-stress) of night float. Computerized reaction tests, Epworth Sleepiness Scale, and Wellness Profile questionnaires were used to quantify fatigue level. DNA of human herpes viruses HSV-1, VZV, EBV, as well as cortisol and melatonin concentrations, were measured in saliva. Residents at the high-stress interval reported being sleepier compared to the rest interval. EBV DNA level increased significantly at both stress intervals, while VZV DNA level increased only at low-stress. DNA levels of HSV-1 decreased at low-stress but increased at high-stress. Combined assessment of the viral DNA showed significant effect of stress on herpes virus reactivation at both stress intervals. Cortisol concentrations at both stress intervals were significantly higher than those at rest.”

<http://www.ncbi.nlm.nih.gov/pubmed/22229027>

The Poor Things.

Conclude: If you are a medical school student or astronaut, your stress will produce a real disease (use Pubmed to discover cortisol does this even independently of stress), but if you are a plain old commoner or a soldier, no, you are having a “pattern of protection” (are scared), or are somatizing or producing scientifically valid illness biomarkers with your magical brain—the definition of somatization disorders.

Look next at this description of somatization illnesses (also called “medically unexplained”) from an “expert” seen on Fox “News” describing the **Justina Pelletier CPS kidnap case**, which became an international scandal revealing what knuckleheads make decisions in New England “hospitals.”

This psychiatrist does not even question the illogic of claiming that people can actually produce valid medical illness biomarkers with their psychogenic powers alone, when, we know that if anyone had such abilities they would not inflict the disease on themselves, but upon people like the “ChIID pRoTeCtIvE sErViCeS (CPS), psychiatry, or the CDC. At the same time, he claims there is no scientific evidence for how someone could produce such scientifically valid illness signs in themselves with their magical brains:

Follow: **"... causing her to believe she is medically ill, when she is not—that they have kindled in her a 'somatoform disorder' in which bodily symptoms actually have purely psychological roots, not anatomic ones..."**

And: **"First, we lack sufficient research data to back up my clinical experience and professional opinion (which some psychiatrists would agree with and some would disagree with)."**

<http://www.foxnews.com/health/2014/03/28/dr-ablow-sure-parents-can-make-their-kids-sick/>

Whaaaa ??

Conclude: A person can have a real disease, but not a real disease, and no one knows how they do it.

This sounds exactly like “magic” to a normal human.

We could take this one step further: Send Justina Pelletier and her kind to the CIA and see if she can do **remote viewing or kill goats with her eyeballs**, alone. If yes, she is a good witch. If not, she is a bad witch. If she can only inflict illness on herself, she must be a bad witch. Fair? Only a not-very-good witch would issue backfiring incantations.

This is America. We have to listen to that kind of crazy malarkey on the “news,” not to mention the horrors of those who experience Wessely-ish, Munchausery-CPS-psychiatric Witch Trials, personally.

And the somatoformizing, cowardly soldiers ... against the backdrop of known disease and known biomarkers, bad vaccines, bad advice about vaccines (or no advice as is the case with children and the MMR vaccine), Wessely’s fairy tales about not enough money to perform real experiments, and mini-witches still terrorizing the Boston area.

This paper does not intend to list all the data available on the First Gulf War Illness. Some people have evidence for other exposures. However, this vaccination business that Wessely first reported explains how people who were not even deployed might have acquired an illness. We know the mechanism of fungal contamination (mycoplasma) of the vaccines or the vaccination of an immune suppressed person can result in the live viruses being reactivated as shown in the SASH charge sheets.

We know from the cytokines study listed here, the Gulf War Illness veterans seemed to have overall higher markers of immune activation than the ME/CFS people tested, which conflicts with the other immunosuppression data, but we do know there are scientific realities to be had and acquired.

And the Pentagon hired Simon Wessely to not only trash the sick veterans, but people with ME/CFS, too.

No one asks Simon Wessely (or anyone else) how in the hell people can magically produce real signs of real illness in themselves and *ONLY* themselves. It is logical to assume some of these people may have stress induced Epstein-Barr like the astronauts and medical students, but what kind of arrogance blames the victim in this 21st Century, and makes them suffer every physical, social, and financial deprivation and humiliation, and does it for money?

There is a new definition of **WHORE** we would like to enter in the next DSM, 5.1:

”One who debases their profession to the point where they would declare their victims ‘conjurers’;

“The WHORES have no awareness of their illness, it’s along the lines of a Delusional Disorder and one of the signs is that they deny they’re mentally ill;

“They continually claim *other people* are not sick, either.”

No one is ever sick, and there are no doctors. There is no medicine, and there is not even a DSM or PDR.

(Well, *that* part is kinda true.)



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healthcare justice

CHARGE SHEET 8: THE STATE OF CONNECTICUT & YALE
ASSAULT CZECH CHILDREN

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

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Descrambling the Centers for Disease Control and Prevention's (CDC's) For-Profit scientific nonsense.

\$A\$H, 2017, Charge Sheet 8: The State of Connecticut & Yale Assault Czech Children

The State of CT and Yale assaulted Czech children with a vaccine that they knew would do them no good, as there is none of the B31 version of OspA (LYMErix) in Europe.

They simply assaulted these children to see how severe would be the adverse events.

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.

“The antibody responses to the three genomic groups of *Borrelia burgdorferi* (*B. burgdorferi sensu stricto*, *Borrelia garinii*, and *Borrelia afzelii*) were determined in 97 German patients with various manifestations of Lyme borreliosis. The geometric mean antibody titers in each patient group, determined by ELISA, were similar with each antigen preparation. By Western blotting, however, patients with meningopolyneuritis tended to respond to more spirochetal polypeptides of *B. garinii*, the group 2 strain, whereas those with arthritis recognized more antigens of *B. afzelii*, the group 3 strain ($P < .03$), as did those with acrodermatitis. **Only 1 patient each with erythema migrans, arthritis, or acrodermatitis had weak reactivity with outer surface protein A (OspA), and none responded to OspB.** It is concluded that differences among the three groups of *B. burgdorferi* may result in variations in the antibody response in European Lyme borreliosis.

<http://www.ncbi.nlm.nih.gov/pubmed/8106763>

J Pediatr. 1999 Nov;135(5):575-9.

Immunogenicity of a recombinant Borrelia burgdorferi outer surface protein A vaccine against Lyme disease in children.

Feder HM Jr1, Beran J, Van Hoecke C, Abraham B, De Clercq N, Buscarino C, Parenti DL.

“A recombinant lipoprotein vaccine against Lyme disease, containing 30 microg of *Borrelia burgdorferi* outer surface protein A (OspA) with aluminum adjuvant, has been shown in a large US field trial of subjects ≥ 15 years of age to offer 76% efficacy against clinical Lyme disease after 3 injections given at 0, 1, and 12 months. Lyme disease is also an important problem in children; thus, OspA vaccine trials in children are needed. The purpose of this study was to investigate the safety and immunogenicity of 2 different doses of lipoprotein OspA with aluminum adjuvant vaccine in healthy children 5 to 15 years of age in a double-blind, randomized study. In a double-blind study, 250 children from the Czech Republic were randomly assigned to receive 15 microg or 30 microg of OspA vaccine at 0, 1, and 2 months. Serum samples, obtained before vaccination and 1 month after the second and third doses, were analyzed for anti-OspA antibody. Solicited and unsolicited symptoms were collected from diary cards. Local pain at the injection site was reported by approximately 76% of the 250 children. Headaches (after 5% to 18% of the injections) and malaise (after 2% to 16% of the injections) were the most frequently reported general symptoms. Local and generalized symptoms were not different between the 15 microg and 30 microg groups, and all symptoms resolved within 4 days. Both

doses were highly immunogenic, with the 30 microg dose eliciting higher antibody levels. Seroconversion occurred in 99% of the 250 children. The OspA vaccine against Lyme disease was well tolerated and highly immunogenic in children.”

<https://www.ncbi.nlm.nih.gov/pubmed/10547245>

This was not even a vaccine as previously demonstrated in the other charge sheets. There is none of the LYMERix kind of B31 strain of Borrelia in Europe. So, this experiment was simply “assault.”