

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 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 test *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-with-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 trashes, stalks, harasses and denies 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

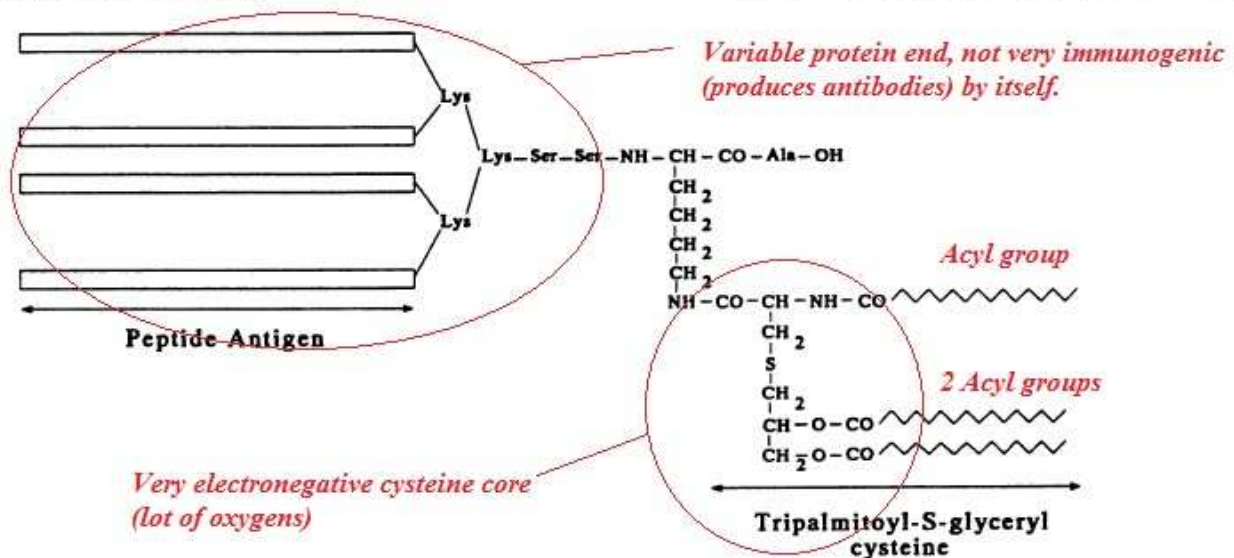


FIG. 1. Schematic representation of MAP-P3C.

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 intracorporeal 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 granulosa 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 numbers 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 tri-acylated 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. 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 these 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 vaccines was giving victims the same “multi-system” disease, was already known to both the Cabal and the U.S. Food and Drug Administration (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 RJ1, 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, 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, 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 RA](#)¹, [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://www.actionlyme.org/DEARBORN_PDF.pdf

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

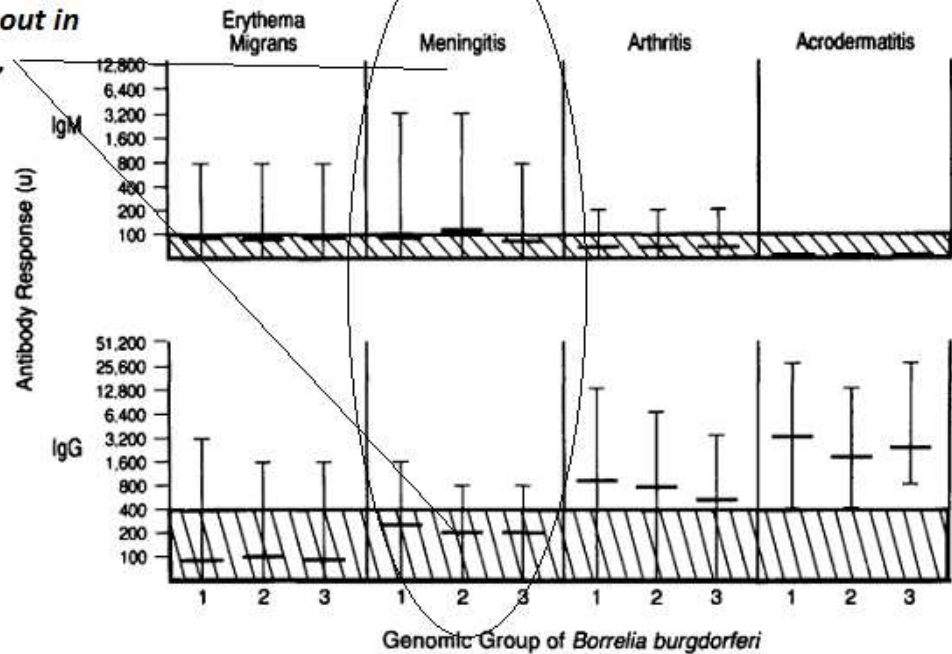
[J Infect Dis.](#) 1994 Feb;169(2):313-8.

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

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

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

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 opportunistic (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.

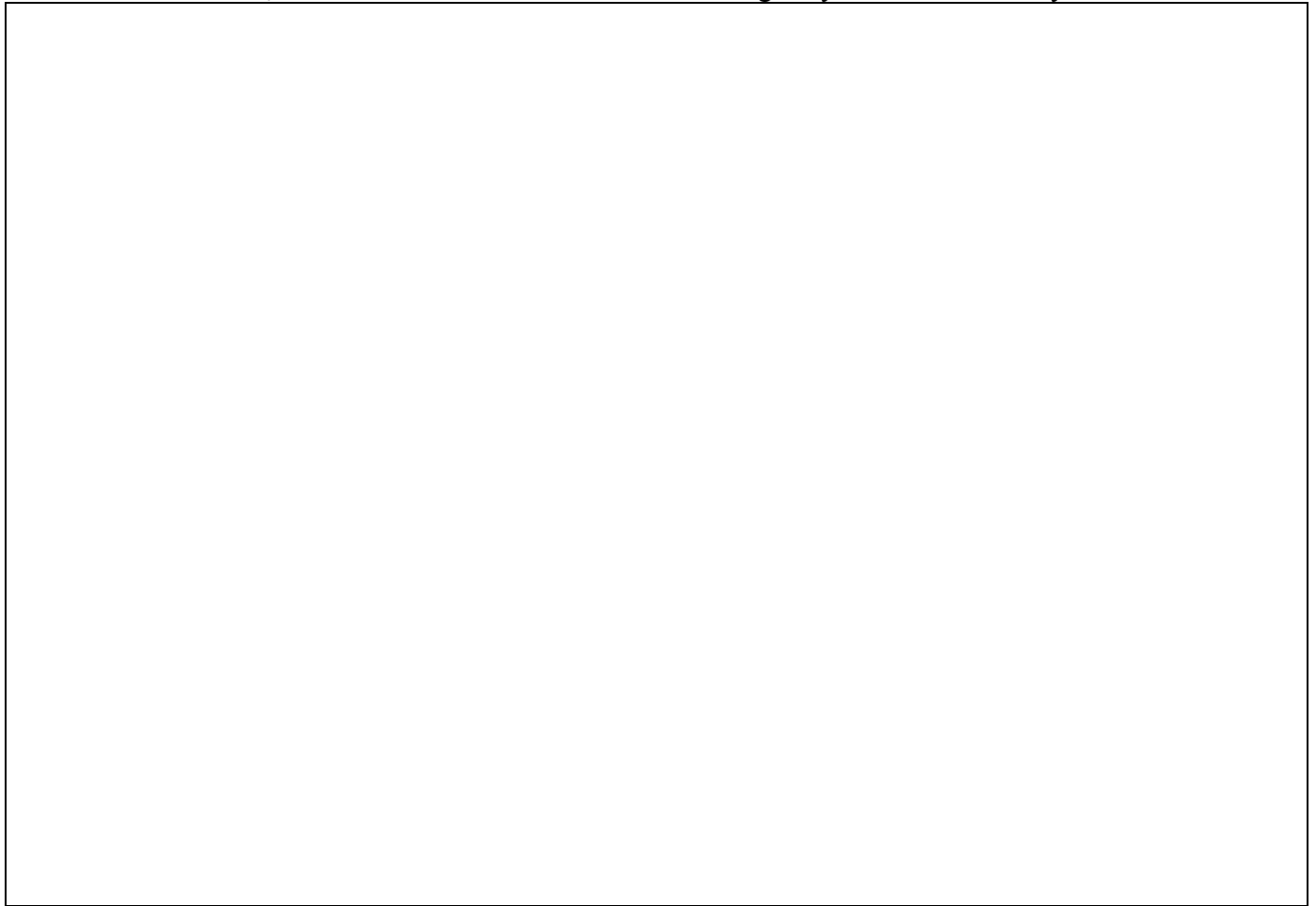
The biological and social phenomenon of Lyme disease.

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.

2. Evolution of LDT Technology, Marketing, and Business Models and the Need for Increased Regulatory Oversight of LDTs

From:

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

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*, filiarasis, 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 is 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_POLICYPAPER_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 ME](#)1, [Nowakowski J](#), [McKenna DE](#), [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..."

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

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

*% 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.

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.

Other's 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.

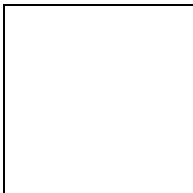
11) CDC Atlanta— talked about mice, not humans. The mouse criteria was 2 out of three from OspC, 16 kD, 17.9 kD, for the mice.

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



“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 (above),

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=%2Fnetahhtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804](http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetahhtml%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 (above), 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=/netahhtml/PTO/srchnum.htm&r=1&f=G&l=50&s1=6,800,613.PN.&OS=PN/6,800,613&RS=PN/6,800,613](http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=/netahhtml/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=%2Fnetacgi%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](#), [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](#), [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](#), [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=%2Fnetachtml%2FFPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804](http://patft1.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetachtml%2FFPTO%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 cant 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.”