$ociety for the Advancement of $cientific Hermeneutics

Interpreting the Evolution of Linguistics from Hippocrates to Hypocrises

FOR IMMEDIATE RELEASE

May 17, 2015

Washington, D.C., May 17, 2015: Medical abuse-victims' rights group Society for Advancement of Scientific Hermeneutics (SASH), announces its Occupy the USDOJ protest beginning June 1, in Washington, D.C. This Occupy movement, led by a group of chronically ill and disabled activists, is a direct result of the medical abuse they say has been inflicted upon them by government and the medical/pharmaceutical corporate complex.

The Occupy leaders state in their criminal charge sheets that there is a common disease mechanism linking Lyme disease, ME/CFS, Gulf War Illness, Fibromyalgia and Autism. Furthermore, to expose such mechanism would reveal rampant fraud and racketeering within the CDC and other entities, as well as the cause of the autism pandemic. All abused patient groups are encouraged to join this peaceful but passionate protest on the steps of USDOJ, 950 Pennsylvania Ave NW, Washington, DC, USA , from June 1 until July 4.

Through a massive compilation of published scientific research and public-record documents, SASH makes a convincing case for Lyme Disease, ME/CFS, Gulf War Illness, Fibromyalgia and Autism sharing a common mechanism of fungal-induced immunosuppression, known to the National Institutes of Health (NIH) as "Post-Sepsis Syndrome." They report that such immunosuppression leads to the chronic reactivation in the central nervous system of multiple viruses such as Epstein-Barr Virus, Cytomegalovirus and HHV-6, leading to cancers and an AIDS-like disease. SASH also shares evidence that the interaction of fungi with attenuated viruses in vaccine vials causes the reactivation of those viruses and ultimately, the diseases they are meant to prevent.

The group's primary charge centers on the USDOJ's failure to take action on a whistleblower complaint that was filed in July 2003 by Kathleen Dickson, a former analytical chemist at pharmaceutical giant Pfizer. Her complaint alleged that CDC officers, Yale University medical faculty and others committed research fraud to falsify the current, Dearborn case definition (2-tiered test standard) in order to falsify the outcomes of the OspA vaccines, namely LYMErix, which was pulled from the market after an FDA ultimatum to the manufacturer.

Ms. Dickson's complaint further alleged that the very same government employees who committed these crimes stood to gain substantial financial rewards from a monopoly on all tick-borne diseases, vaccines and test kits. Additionally, their falsification of the Lyme disease case definition and treatment guidelines have left 85% of actual Lyme sufferers unable to obtain diagnosis, treatment, or insurance coverage for their AIDS-and cancer-like illness.

An abundance of scientific and historical evidence is presented in the charge sheets. Many of the citations refer to the alleged criminals' own peer-reviewed, published research papers and patent documents, which paint a chilling picture of the extreme effort that SASH says has been made by the alleged criminals to deny basic healthcare to an estimated 30 million sufferers in the United States. They say that the extent of deceit and corruption, with intent to deny an illness, goes far beyond anything that occurred in the early days of AIDS activism.

They are calling on USDOJ to prosecute for the fraud and racketeering charges, which have left millions of people to suffer in isolation while being ridiculed by doctors, family members and employers as psychosomatic or lazy. The victims, often bankrupted by the high cost of out-of-pocket medical expenses, and unable to work due to illness, frequently commit suicide to escape their continuous denial of basic human rights.

For additional information and to view the charge sheets, visit ohioactionlyme.org.

The Criminal Charges Sheets:

$ociety for the Advancement of $cientific Hermeneutics

Interpreting the Evolution of Linguistics from Hippocrates to Hypocrises

ALDF-CDC Enterprise Conspires to Defraud USA in Dearborn-Vaccines Scam (18 U.S.C. § 371)

Falsifying the case definition - a CDC Staff Conspiracy; Steere, Barbour, and Johnson: The testing for Lyme disease was falsified to pass off bogus vaccines and test kits – a chronology:

Originally, Lyme borrelia were perceived by the 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 a flagellin method. In spite of this impossibility, 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 in association with Kaiser-Permanente, who are still at NYMC writing MD-training modules. (CDC is often found in collaboration with Kaiser-Permanente; we knew this even before their "Morgellon's investigation" scam.)

The ALDF.com is a False Claims-and-Racketeering 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 would be given some assurance against the prosecution of the testing scam necessary to pass off these bogus recombinant products. The Defendants, 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 infections; rodent brains used to be the storage media (Barbour, 1986) before the CDC learned how to freeze-dry spirochetes (1964):

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

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

The ALDF.com Defendants even changed the disease's name to "Lyme disease" from "Lyme borreliosis." The participants in the scam even referred to themselves as an "enterprise" (Arthur Weinstein, 1998).

Meet some of the founding members of the fake non-profit American Lyme Disease Foundation (ALDF.com), Valhalla, NY, in 1990, left to right: Edward McSweegan, Durland Fish, Gary Wormser, and John J. Connolly




If you are reading this from a paper copy, please visit actionlyme.org for full access to embedded hyperlinks.

The Defendants conspired to make Lyme disease largely undetectable. The plan 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. If Lyme disease is only 15% detectable, the Defendants 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 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."

The problem 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:

2000, ***Modulation of lymphocyte proliferative responses by a canine Lyme disease vaccine of recombinant outer surface protein A (OspA).***

"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.

THEY CHANGED IT? — Yes, They Changed the Diagnostic Standard for Lyme disease.

The following article by Allen Steere is the foundation of the CDC's original, 1990, "Lyme disease" "case definition" blood test (serology). It was later falsified at a farce of a serology conference put on by the CDC in 1994 in Dearborn, MI.

1986, Allen Steere says:

Appearance of a new immunoglobulin M response and expansion of the immunoglobulin G response late in the illness.

"… 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:

http://www.actionlyme.org/CDC_DOCUMENTS_1990.htm

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 or 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 only able to produce new, IgM bands if the organism is still alive and not killed by antibiotics.

Steere also wrote in that same 1986 report (above; basis of the 1990 CDC case definition) that all you need is band 41 to diagnose Lyme; just rule out syphilis:

sonicated whole spirochetes (11, 12). Furthermore, in this study, IgM antibodies to the 41-kD polypeptide were usually apparent by immunoblots before IgM titers were elevated by the current ELISA (12). Although antibodies reactive against this antigen may be present in patients with relapsing fever or syphilis (11, 14), these diseases can be distinguished clinically from Lyme disease and therefore should not cause diagnostic confusion. The

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 US 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 MS, not to mention the fact that Lyme and LYMErix both are known to cause cancer (and MS, Lupus, possibly RA) via the reactivation of latent herpes viruses. Mycoplasma are also known to be associated with the production of cancer. 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 Post-Sepsis Syndrome.

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, criteria is very different from the 1990 criteria and basically refers to only the late, HLA-linked, arthritis, hypersensitivity response. It came about as a result of research fraud committed by Allen Steere in Europe in 1992. OspA and B (bands 31 and 34) are notably absent.

As an aside, we can assume that the reason the IDSA/ALDF/CDC/Yale Lyme Defendants do not want anyone treated for Lyme is because late in the disease, it's really about fungal antigen tolerance and cross tolerance, as well as 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. See more at:

http://badlymeattitude.com/2015/01/05/m-e-cfsfibromyalgialymeautismgws-post-sepsis-syndrome/

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

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

"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."

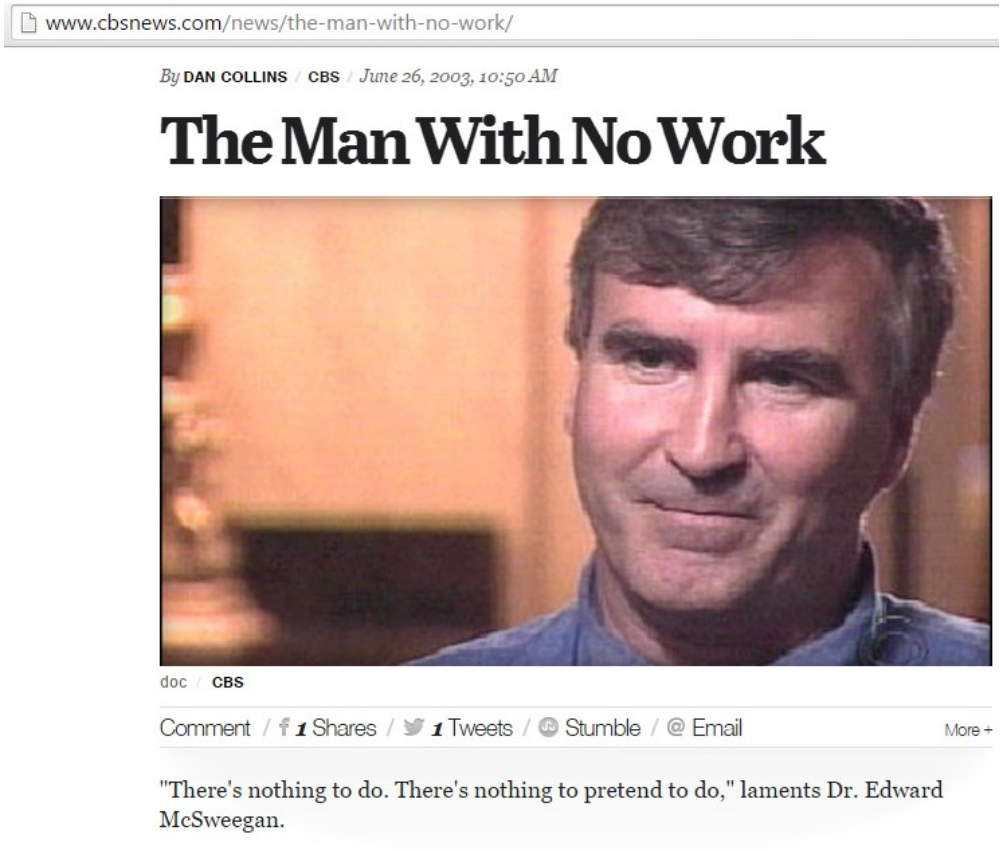
http://www.the-hospitalist.org/article/infectious-diseases-society-of-america-2014-practice-guidelines-to-diagnose-manage-skin-soft-tissue-infections/

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 (excludes the CDC and IDSA).

How Lyme and OspA cause disease we learned from the LYMErix fiasco, because the fungal OspA vaccines caused the same systemic, protean, post-sepsis syndrome, chronic active infections/disease (per Ben Luft and Dave Persing, and the vaccine victims themselves as reported to the FDA through the VAERS; see below for those links and quotes).

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, etc., Barbour and Fish, 1993). 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: "Lyme patients are not sick, and OspA was a vaccine."

The FDA ordered LYMErix off the market in February 2002, 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", and even after Senators Markey, Blumenthal, et al, ordered the FDA to assure Lyme testing was valid according to the FDA's criteria.


www.cbsnews.com/news/the-man-with-no-work/

By DAN COLLINS / CBS / June 26, 2003, 10:50 AM

The Man With No Work

doc / CBS

Comment / 1 Shares / 1 Tweets / Stumble / @ Email More +

"There's nothing to do. There's nothing to pretend to do," laments Dr. Edward McSweegan.

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, trashed U.S. Navy to divert their vector borne diseases funding to his buddies at the 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 paraphysical 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, as shown above where 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):

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

"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://actionlyme.org/DATTWYLER_NK_SUPPRESSION.htm

And:

Modulation of natural killer cell activity by Borrelia burgdorferi.

"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

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

http://www.actionlyme.org/CDC_DOCUMENTS_1990.htm

1990, Allen Steere reports that "chronic, neurologic Lyme won't test positive," uses Dattwyler and Volkman's Seronegative Lyme T Cell Assay
CHRONIC NEUROLOGIC MANIFESTATIONS OF LYME DISEASE (NEJM)
"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)"

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

http://www.actionlyme.org/STEERES_SERONEG_LYME_ASSAY.htm (**note "#26 reference in this link)

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

*****This is reference #26 from above (this is important): Seronegative Lyme disease; dissociation of the specific T- and B- lymphocyte responses to Borrelia burgdorferi*** - by Raymond Dattwyler, et al, see 1988 above.

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

1990, ALDF.com founded-- a self-proclaimed "entrepreneurial quartet." includes McSweegan, Fish, Wormser and Connolly. (*Please note the sponsors on their board.)

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

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

1992, CDC officer Allen Steere falsifies testing in Europe:

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

The PubMed links to those 2 reports – no full text available, that is why thy were scanned directly from the Yale Medical Library in 2002.

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

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

Western blotting in the serodiagnosis of Lyme disease.

http://www.ncbi.nlm.nih.gov/pubmed/8380611

Of those two reports of Steere's lab shenanigans in Europe, only the second one is 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 entities are 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. We will come to this later, as it is critical to the whole scam and shows the intent of their entire enterprise. Steere, in Europe used bogus "high-passage" borrelia 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.

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:

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

"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

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."

"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=%2Fnetahtml%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 borrelia that did not have the vaccine antigens in it would have to be used. 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 meant even more vaccine patents in the future. 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 Defendants 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:

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

http://www.ncbi.nlm.nih.gov/pubmed/8968914

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 opportunistics (NIH's "Post-Sepsis Syndrome") from the imunosuppression 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.

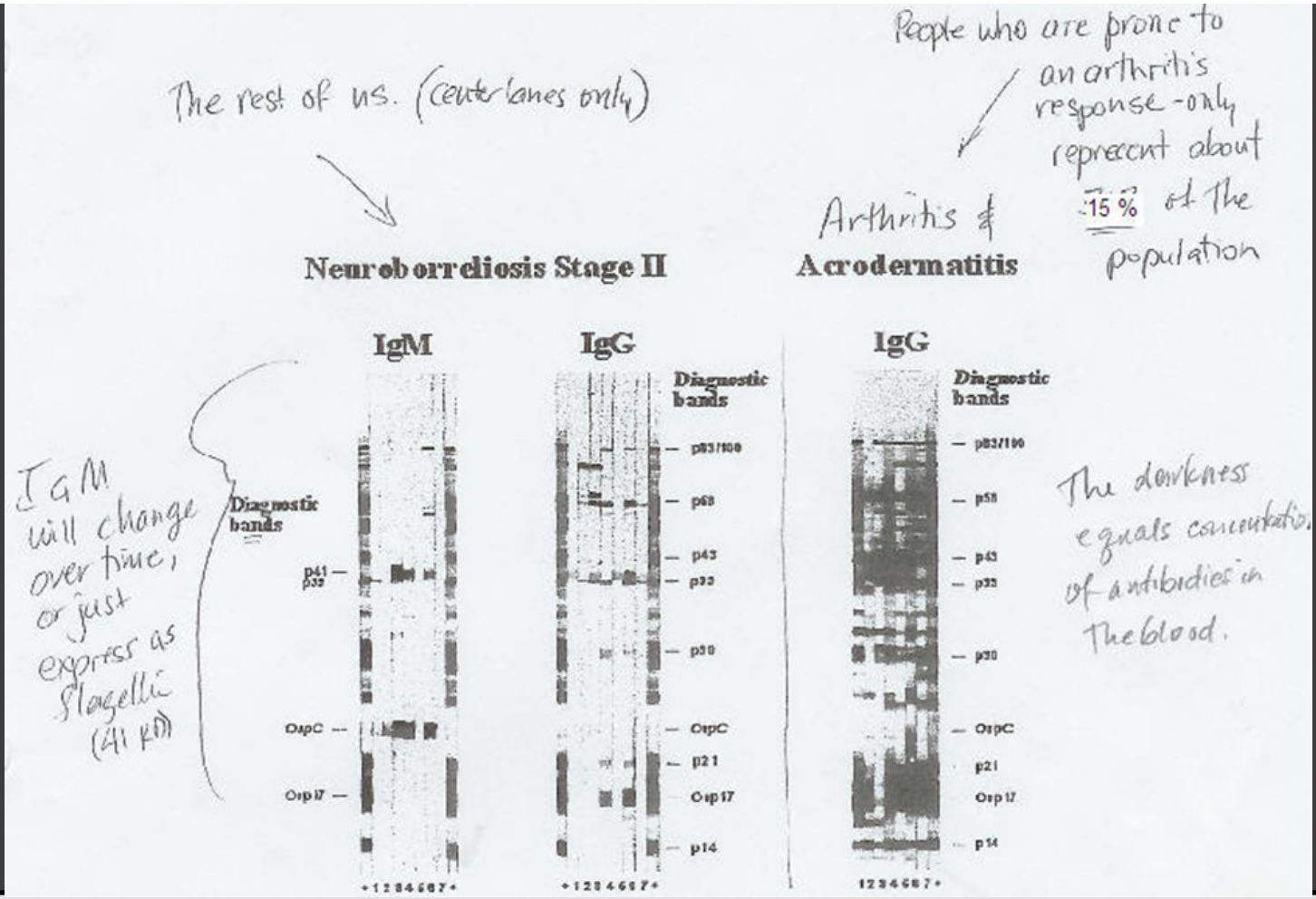
1993, Barbour and Fish slam Neurologic Lyme victims in:

The Biological and Social Phenomenon of Lyme Disease

Barbour and Fish admit 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:

http://actionlyme.org/BarbourFishpdf.pdf

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 Defendants 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 Defendants 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):

A Case-Control Study to Examine HLA Haplotype Associations in Patients with Posttreatment Chronic Lyme Disease

"…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."

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; http://www.actionlyme.org/PRIMERSHELLGAME.htm.) **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

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.

Above are the FDA's rules for the validation of an analytical method. These standards were met by Yale's 1991 Flagellin Method Patent US # 5,618,533 and this report: ***Molecular characterization of the humoral response to the 41-kilodalton flagellar antigen of Borrelia burgdorferi, the Lyme disease agent.***

"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

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 at: http://www.actionlyme.org/PRIMERSHELLGAME.htm for the phylogeny or the genetics that shows Lyme is closest to B. anserina (from Africa).

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:

Seronegative Lyme disease. Dissociation of specific T- and B-lymphocyte responses to Borrelia burgdorferi.
"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://actionlyme.org/DATTWYLER_NK_SUPPRESSION.htm

And:

Modulation of natural killer cell activity by Borrelia burgdorferi.
"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 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

Dearborn, Who Said What

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:

Serodiagnosis in Early Lyme Disease

"Overall, 51 of 59 (86%) convalescent phase serum specimens were reactive by IB [Dearborn criteria

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.

Wormser says that when using the Dearborn proposed criteria, only 9/59 cases of Lyme were detected, or is 15% accurate.

DISCUSSION

Immunoblot-KMD], 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…"

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.

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.

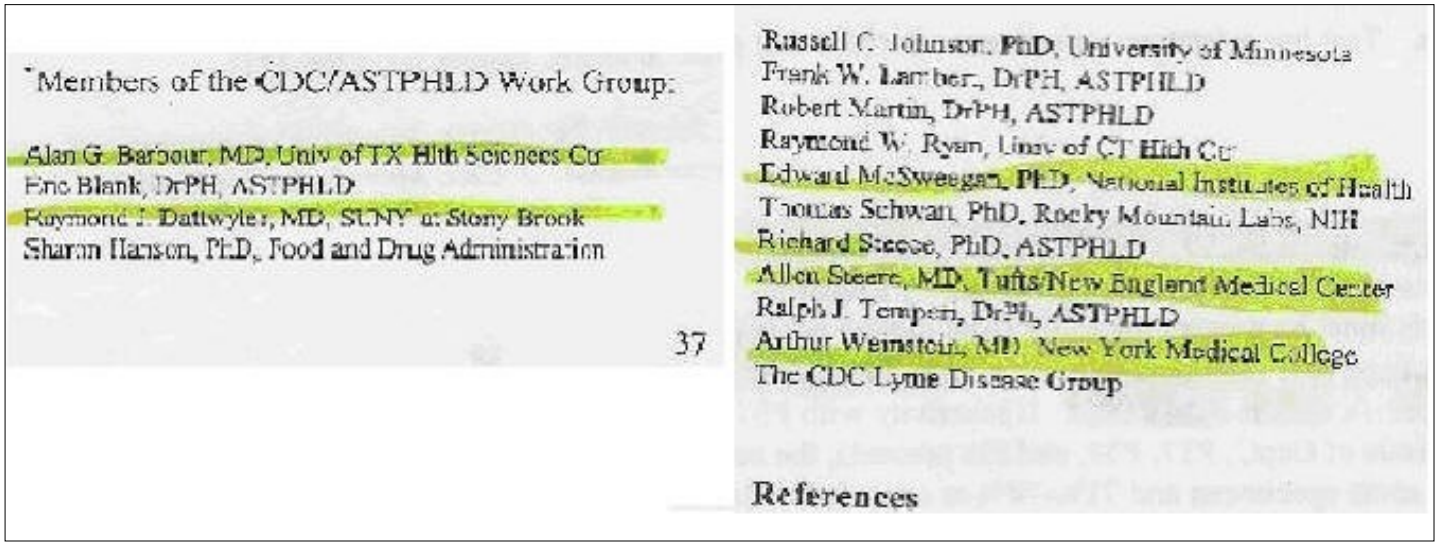
11) CDC Atlanta— talked about mice, not humans. The mouse criteria was 2 out of three from OspC, 16 kD, 17.9 kD, for the mice.

We got this standard anyway, even though none of the invited participants agreed - not by a long shot. See the Primers Shell Game reports here or at this link: http://www.actionlyme.org/PRIMERSHELLGAME.htm 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

37

Russell C. Johnson, PhD, University of Minnesota
Frank W. Lamber., 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 Steere, PhD, ASTPHLD
Allen Steere, MD, Tufts/New England Medical Center
Ralph J. Tempero, DrPh, ASTPHLD
Arthur Weinstein, MD, New York Medical College
The CDC Lyme Disease Group

References

"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 Defendants 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/transcpt/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=%2Fnetahtml%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.
The Biological and Social Phenomenon of Lyme Disease
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=/netahtml/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).

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."

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=%2Fnetahtml%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:

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.

====

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"):
http://content.nejm.org/cgi/content/abstract/339/4/209

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

1) Yale's Robert Schoen and Mayo's/Corixa's David Persing, with John Anderson,1995-6; the RICO report:
http://jcm.asm.org/cgi/reprint/35/1/233?view=long&pmid=8968914

2) Shoen 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=%2Fnetahtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=6045804.PN.&OS=PN/6045804&RS=PN/6045804

3) David Persing and Lenny Sigal explaining that the Western Blots of OspA-vaccine victims were not readable (which means whoever was in charge of data safety monitoring like Arthur Weinstein is in big trouble): http://www.journals.uchicago.edu/doi/pdf/10.1086/313920

4) Yale's Robert Schoen in the 1998 Munchausen's Book, instructing 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, and did not report that their Western Blots were unreadable. Each vaccine trial report and summary was 2 false claims.

====

In the fall of 1998, the LYMErix vaccine was approved, anyway, by the FDA (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: "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.

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."

Charge One: Falsifying the case definition- a CDC Staff Conspiracy; Steere, Barbour, and Johnson

A) CDC officers Allen Steere, Alan Barbour, Barbara Johnson conspired to falsify the case definition for Lyme disease. [Conspiracy to Defraud, see the ALDF.com as "Astroturf" or a fake non-profit.]
B) Barbour and Johnson own patents from which they stood to profit only if the testing case definition was falsified. [Theft of Honest Services.]

C) Steere falsified the Western Blot case definition panel of antibodies testing in Europe in 1992 via research fraud, leaving OspA and B out of the diagnostic standard using recombinant antigens and high-passage strains that drop plasmids. This would give the appearance that OspA and B (encoded on the same plasmid so they would have to be dropped together) were not "primary, immunodominant antigens" which was contrary to Steere, et al's previous claims and the very nature of their alphanumeric nomenclature ("OspA, OspB, OspC, OspD, OspE, OspF, etc" -antigens).

Steere, allegedly, stood to profit with the secondary outcome of falsified testing – testing with a method that was designed by Steere (in Europe) that would be necessary after an OspA vaccine was on the market. It left OspA and B out. OspA and B are encoded on the same plasmid. Steere's friends' companies were to be the only ones licensed to use this method.

Steere was involved in a monopoly with RICO entities David Persing (Mayo Clinic and Corixa), Robert Schoen (Yale's L2 Diagnostics), and Phillip Molloy (Imugen) to capture all the post-LYMErix testing for North America. They publicly claimed in an SEC filing and in public announcements/advertisements that they would be the only labs licensed to test for Lyme "when the vaccination status of the population was unknown" (US patent 6, 045,804), or if it was unknown if a person had had an OspA vaccine or not. [False Claims, Racketeering]

Charge Two: Steere added an unnecessary ELISA

Steere added an unnecessary ELISA screening test that only detects late Lyme arthritis in the first step and declared this to be a test for "early Lyme."

Steere not only added an ELISA as a screening test that of (falsely raising the bar on a total-antibody test) that left out all neurologic outcomes of Lyme as "cases," but the normal cut-off for a chromatography assay such as this is 3 standard deviations above baseline noise (that means the signal generated by a blank). Steere used 5 standard deviations for a cut-off - another act of fraud.

It was never necessary to use a total-antibody test such as an ELISA since Steere himself knew many patients produced low antibody concentrations, having used the Dattwyler-Halperin Seronegative T cell Proliferation Assay to assess "Chronic Neurologic Lyme" victims in 1990.

Charge Three: CDC officer Barbara Johnson hosted a fake consensus conference in Dearborn, MI

CDC officer Barbara Johnson hosted a fake consensus conference in Dearborn, MI, in October, 1994, subsequent to Steere falsifying the testing in Europe with Frank Dressler (a student in Germany). Johnson sent out invitations to labs across the country that were under the impression the conference would be about standardization of the METHOD of Western Blotting (e.g., what concentration of reagents and strains to use) rather than the interpretation of the Western Blots. Only MarDx agreed with the antibody panel proposed by A from his European research fraud criteria, but they, MarDx, had been given Lyme-arthritis -positive blood (HLA-linked hypersensitivity response) to qualify their Western Blot test strips. The average assessment of the ACCURACY (cases that were known to be positive with, for example a DNA method), excluding MarDx, that were shown to be positive with this falsified antibody panel for a "case" of Lyme was 15%.

Johnson ignored all those recommendations, despite inviting them to "participate in the proceedings."

Charge Four: Falsifying the OspA vaccines outcomes

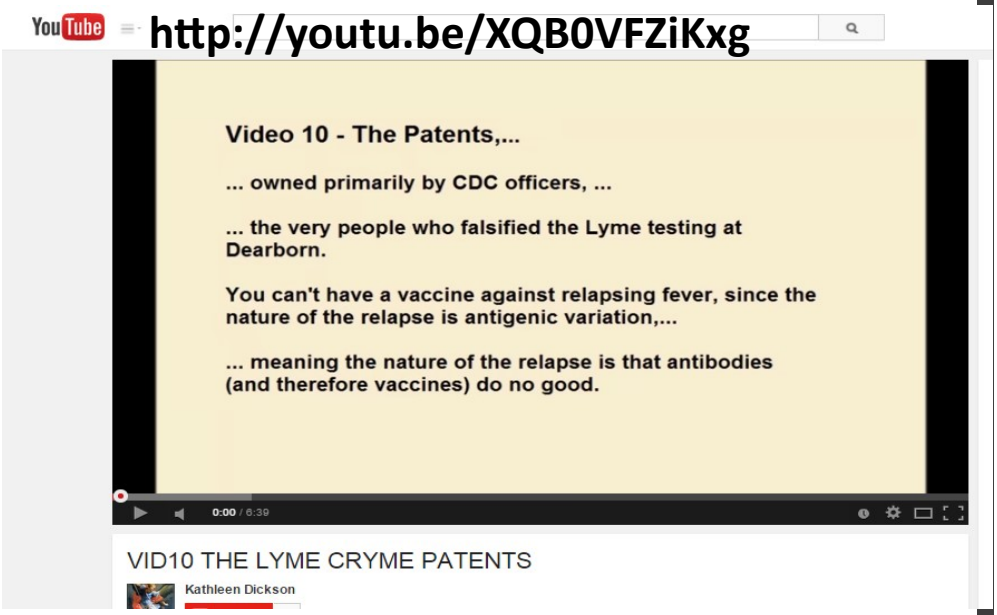
This gang then reported 76% and 92% "safe and effective" OspA vaccines (ImuLyme and LYMErix) when the Western Blots, they later reported, were unreadable. So, they used a bogus test, the Dearborn Method (they claimed), to assess the outcomes of their vaccines, but they later reported they actually had no idea if OspA vaccines prevented Lyme because they could not read their results.

The "Lyme Disease" Patents

Research fraud and racketeering complaint data to assist USDOJ in their prosecution of the criminals

This document is a companion to Video 10—The Patents and covers the following information: 1) lists the CDC officers and ALDF.com/Yale/NYMC associates who own patents related to Lyme and other tick-borne diseases (TBDs); and 2) the Dearborn event was not only research fraud but interest-conflicted, as were the FDA panels to approve LYMErix (they were all the same characters: Steere, Barbour, Schoen, Rahn, Johnson, Weinstein, McSweegan, etc).

This video teaches you how to use the patent databases to search for related conflict of interest or racketeering data. In the patents you will find all sorts of language contradictory to what the Lyme Disease "specialists" of today (IDSA, etc.) use publicly. Therefore, these patents MUST all be studied and examined by you. These are legal patent CLAIMS and therefore, for the most part, truthful. These individuals can't very well patent a non-truth or someone else will patent the actual truth, useful for scientific history as well as commercially.



A little background about the Dearborn/OspA-scam

A little background about this Dearborn/OspA-scam, since it is the central or essence of the crimes:

"Dearborn" refers to the 1994 United States Centers for Disease Control and Prevention (CDC) conference that took place in Dearborn, Michigan. This was the event where the testing for Lyme Disease was falsified. Prior to "Dearborn," the Lyme spirochete was regarded as just another relapsing fever-causing organism. The new, bogus definition and accompanying 2-tiered test was something else entirely contrived (not even empirically perceived) and false. The Dearborn event is discussed in this video and the other ones in the YouTube series called "Lyme Crymes."

...The "Lyme Disease" Patents, continued

For years, no one among this CDC/ALDF/Yale.NYMC 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 I made a recombinant measles vaccine based on a DNA sequence that coded for say, "XYZ" surface antigen, I would not use recombinant "XYZ" surface antigen in the testing schema to see if a person had measles because I 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 was it was straight up research fraud from these DNA patenteers. *This Dearborn event left the sickest people with no disease diagnosis.*

If I 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 I do? I'd make sure I got all of it: vaccines, test kits, grants, funding, all future blood testing for all future potentially patentable goodies in that blood, publicity, my name on plaques and statues (like Alan Barbour), awards like an "Astute Clinician Award,"… or, I 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," and US States naming, like, "Allen Steere Day" after me…

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, and with this cabal's chronic hystrionics over the development of other tests for Lyme, etc., because that, Dearborn, will be the most serious criminal charge – the homicide charge. All the ALDF.com and DNA patent-owners, here, have slandered and libeled against Lyme victims, making this not a simple negligent homicide charge. All of their derogatory slander and label and trash-talking Lyme and LYMErix victims show **"intent to cause harm,"** which is not negligent homicide or manslaughter, but murder and maiming.

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

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

If you are reading this from a paper copy, please visit actionlyme.org for full access to embedded hyperlinks.

...The "Lyme Disease" Patents, continued

Barbour, Alan G., CDC officer and participant in the Dearborn conference, owns 30+ patents including the ImmuLyme OspA patent. The ImmuLyme 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 ImmuLyme patent (&Berstrom, Magnarelli) European Patent #(5092/88, DK)
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=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=%2Fnetahtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5747294.PN.&OS=PN/5747294&RS=PN/5747294

ImmuLyme trial result (falsified; used Dearborn, blots were unreadable):
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.
http://www.ncbi.nlm.nih.gov/pubmed/9673299

LYMErix trial result (falsified; used Dearborn, blots were unreadable):
Vaccination against Lyme disease with recombinant Borrelia burgdorferi outer-surface lipoprotein A with adjuvant. Lyme Disease Vaccine Study Group.
http://www.ncbi.nlm.nih.gov/pubmed/9673298

Barbour says OspA will not work as a vaccine due to antigenic variation, 1992:
Antibody-resistant mutants of Borrelia burgdorferi: in vitro selection and characterization.
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:
Selection of variant Borrelia burgdorferi isolates from mice immunized with outer surface protein A or B.
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)

Steere Falsifies Test in Europe

Antibody responses to the three genomic groups of Borrelia burgdorferi in European Lyme borreliosis.
http://www.ncbi.nlm.nih.gov/pubmed/8106763

"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

...The "Lyme Disease" Patents, continued

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

Dearborn: http://www.actionlyme.org/DEARBORN_PDF.pdf
See in the Dearborn booklet: that none of the labs agreed with this Dearborn proposal in the Dearborn pdf. (Exception: MarDx Labs, which was given arthritis-positive blood to qualify their Western Blot strips prior to Dearborn; MarDx was then was given both vaccine trial contracts; MarDx was then sold to an 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 (http://www.actionlyme.org/PRIMERSHELLGAME.htm) 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 flagellins from all Borreliae.

Telford Phylogeny with Masters' Amblyomma Borrelia:
Lone star tick-infecting borreliae are most closely related to the agent of bovine borreliosis.
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=%2Fnetahtml%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

...The "Lyme Disease" Patents, continued

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:
Molecular characterization of the humoral response to the 41-kilodalton flagellar antigen of Borrelia burgdorferi, the Lyme disease agent. http://www.ncbi.nlm.nih.gov/pubmed/1894359

Fikrig and Flavell say OspA will not work due to antigenic variation:
Selection of variant Borrelia burgdorferi isolates from mice immunized with outer surface protein A or B.
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.

...The "Lyme Disease" Patents, continued

Padula OspC patent: USPatent No. 5,620,862
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=5,620,862.PN.&OS=PN/5,620,862&RS=PN/5,620,862

Padula OspC – not in strain B31 PubMed report:
Molecular characterization and expression of p23 (OspC) from a North American strain of Borrelia burgdorferi.
http://www.ncbi.nlm.nih.gov/pubmed/8225587

Persing, Schoen and the RICO-within-the-RICO patent – this patent shows the intention of Steere's Dearborn, falsified case definition (you will see later, in an announcement by the Mayo Clinic, below).

US Patent # 6,045,804
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=6,045,804.PN.&OS=PN/6,045,804&RS=PN/6,045,804

This patent reveals they know LYMErix causes a systemic disease like chronic Lyme, and in this patent they reveal their intended monopoly on post-LYMErix testing for the USA and Canada as they claimed in their advertising: *"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."***

"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. "***

Persing and Sigal later reveal that the Western Blots in both vaccine trials were unreadable. Both vaccine trials used MarDx test strips and said they were using the Dearborn method to assess the efficacies of these vaccines. Arthur Weinstein was the Data Safety Monitor for one of those trials and was also a participant in the Dearborn scam. Obviously Weinstein never looked at any of the data he was safety-monitoring since the Western Blots were unreadable. In reality, neither OspA vaccine trial group could tell whether or not OspA prevented Lyme, so those vaccine trial reports were research fraud events and reports.
This Persing-Schoen RICO-RICO Patent 6,045,804, is also written up in this PubMed report, co-written by Yale's Robert Schoen:
Borrelia burgdorferi enzyme-linked immunosorbent assay for discrimination of OspA vaccination from spirochete infection. 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:

...The "Lyme Disease" Patents, continued

(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 sulflhydryl side chain of the adjacent cysteine (Cys) residue (at position 17). Following this attachment, cleavage by signal peptidase II and the attachment of a third fatty acid to the N-terminus occurs. The completed lipid moiety, a tripalmitoyl-S -glycerylcysteine modification, is termed Pam3Cys (or is sometimes referred to herein as Pam(3)Cys or Pam3Cys). It has been suggested that the lipid modification allows membrane localization of proteins, with polypeptide portions exposed as immune targets. In addition to serving as targets for the immune response, Pam3Cys-modified proteins, such as OspA, have been reported to act as potent inflammatory stimulants though the toll-like 2 receptor mechanism (TLR2).

http://patentscope.wipo.int/search/en/detail.jsf?docId=US42934470&recNum=9&maxRec=30&office&prevFilter&sortOption=Pub+Date+Desc&queryString=tripalmitoyl+cysteine+or+Pam3Cys+and+Epstein-Barr&tab=NationalBiblio

The Mayo Clinic advertised the RICO within the RICO – a patent they were the assignee and would have gotten royalties (6,045,804); Connecticut Attorney General 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/WupHjKwFilIJ

"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

...The “Lyme Disease” Patents, continued

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’. 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.

###

“Contact: “Tom Huyck “507-284-0003 (days) “507-284-2511 (evenings)”

Society for the Advancement of Scientific Hermeneutics

Interpreting the Evolution of Linguistics from Hippocrates to Hypocrises

Lyme Disease Biomarkers

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Post Sepsis Syndrome

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

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

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

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

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

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

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

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

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

Lyme Disease Biomarkers, continued

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

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

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

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

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

Here are those 2 reports:

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

and

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

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

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

The other biomarkers

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

A) MMP-130 - Klempner as shown above.

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

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

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

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

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

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

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

E) ALLEN STEERE and Brain SPECT or Hypoperfusion

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Lyme Disease Biomarkers, continued

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

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

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

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

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

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

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

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

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

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

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

FULL JOURNAL REPORT, snippet…

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

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

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

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

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

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

Lyme Disease Biomarkers, continued

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

Types of Biological Agents

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

---

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

[61] Ibid.

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

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

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

If you are reading this from a paper copy, please visit actionlyme.org for full access to embedded hyperlinks.

Lyme Disease Biomarkers, continued

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

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

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

150219,KMD,SASH

Society for the Advancement of Scientific Hermeneutics

Interpreting the Evolution of Linguistics from Hippocrates to Hypocrises

Patient's Guide to NIH's Post-Sepsis Syndrome

"It is an indescribable experience knowing that what you are doing will have an impact on the lives… of millions of people."

~ Anthony S. Fauci, M.D.
NIAID Director

In this publication by the Society for the Advancement of Scientific Hermeneutics (SASH), we discuss the National Institutes of Health (NIH) model of Post-Sepsis Syndrome (PSS), a disease of immunosuppression, which parallels what the Centers for Disease Control (CDC) is calling "fungal meningitis."

We also provide insight into how these agencies think such a disease may be treated. In this PSS / fungal meningitis model, human TLR2/1 agonists -- fungal antigens -- turn off the immune system to prevent death from sepsis. This is the main reason the mouse model of disease does not parallel human disease. Mice do not have human TLR2.

Fungal antigens/infections also reactivate herpesviruses, whose chronicity has been widely proven as leading to cancers and neurological diseases. Since there are many ways to "acquire" immunosuppression, we will focus on several well-known outcomes: Lyme borreliosis, Autism, Gulf War Syndrome, CFS/ME/SEID and Fibromyalgia, to show how they fit the model.

We will begin with Lyme disease, or borreliosis, since it is necessary to explain what OspA is, to understand how it fits this fungal model. Borreliosis is a multi-system disease caused by a spirochetal parasite. A spirochete is a spiral shaped parasite with a unique mechanism for movement that features a bundle of tail-like "flagella" which resides inside the cell wall. Borreliosis is transmitted primarily through the bite of an infected tick, but also can be transmitted in utero to an unborn fetus (according to Yale), and possibly through insect vectors. If not treated immediately with antibiotics, the infection can persist for years and cause neurological diseases such as MS, Lupus, cancer, Chronic Fatigue/ Myalgic Encephalomyelitis, ALS (Lou Gehrig's Disease) and Alzheimer's, according to IDSA and the CDC. Thus, borreliosis is commonly referred to as "The Great Imitator" or "New Great Imitator."

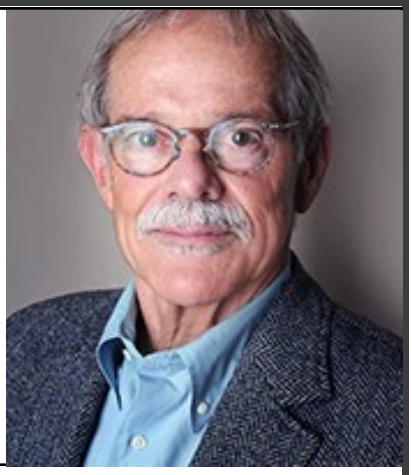
In a mechanism commonly known as blebbing, borreliae parasites have the ability to shed (bleb off) their outer membrane lipoproteins to evade detection by the immune system, per CDC officer Alan Barbour in (the probably mis-titled): "Researchers Finding Rewarding Careers As Software Entrepreneurs" "It's 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/

If you are reading this from a paper copy, please visit actionlyme.org for full access to embedded hyperlinks.

These outer surface lipoproteins, such as OspA (the Lyme "vaccine"-- LYMErix) are TLR2 agonists (fungal antigens) and also "undergo virtually limitless antigenic variation, leaving the immune system overwhelmed" says, again, CDC officer Alan Barbour 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=6,719,983.PN.&OS=PN/6,719,983&RS=PN/6,719,983 ; essentially, they disable or turn off the immune system.

"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.

~ Alan Barbour, MD, CDC Officer



Fungal antigens shed by Borrelia cause tolerance to other fungal antigens (TLR2/1-agonists such as those borne by mycoplasma and mycobacteria) as well as tolerance to other antigen types, managed by other TLRs. Tolerance means that the immune system stops recognizing fungal antigens such as:

1) inhibiting HLA-molecule function and therefore antibodies are no longer produced (Radolf and Harding), "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/content/167/2/910.full, and

2) exposure to Borrelial fungal antigens causes cross-tolerance to the TLRs that manage viral infections: "Because IRAK1 is required forTLR7/9-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." (CV Harding) http://www.ncbi.nlm.nih.gov/pubmed/22227568

This is very important, because often, when you are sick you get blood drawn to test for antibodies to various pathogens. We are generally led to believe that the higher the antibodies, the more advanced the infection. As we have seen, in diseases of immunosuppression, antibodies are not produced. This is one reason the Lyme Western blot is useless.

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

~Raymond Dattwyler, MD

At the 1994 Dearborn conference, Raymond Dattwyler, MD, agreed:

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 say, some people who mount better responses get worse disease. Did I hear you say that."

Dr. Dattwyler: "No, no, I said the reverse. The better responses tended to have a better response. And I should clarify where this came 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 didn't know that it was not optimal at the time--the ones that failed to mount a vigorous immune 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."

Exposure to shed borrelial lipoproteins causes fungal tolerance in the blood and the inability to get rid of mycoplasma/eperythrozoons from the blood, especially from the red blood cells (which causes fatigue). Also, there is cross-tolerance to TLR4 agonists from constant TLR2-agonism of shed borrelial antigens like OspA and vice-versa. This creates an environment where opportunistic infections can thrive and cause chronic, disabling disease.

A well known outcome of immunosuppression, regardless of how this is induced (e.g., Stelara, Humira, transplant drugs, methotrexate, HIV, etc) is the reactivation of the latent herpes viruses, particularly Epstein Barr Virus (EBV). Chronic EBV (or EBV in combination with CMV, HHV-6, or Varicella), is probably the main driver of all these New Great and Great Imitator diseases. That is the basic gist of the Post-Sepsis Syndrome model endorsed by the NIH, as demonstrated in these two studies:

1) NIH: profound immunosuppression is one of the chronic consequences of severe sepsis http://www.ncbi.nlm.nih.gov/m/pubmed/21048427/

2) Washington University in St. Louis: Reactivation of multiple viruses in patients with sepsis http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0098819

Borrelial- or OspA-induction of TLR2-antigen tolerance is one example of that model.

Other examples:

In Autism: Fungally contaminated vaccines reactivate the live, attenuated viruses and suppress the immune system (TLR2/1-agonist tolerance), causing children to get the disease instead of the protection.

In Gulf War Syndrome: Nerve agent antidote, DEET and hyper-vaccination [including fungally- (e.g. mycoplasma) contaminated vaccines] all work to suppress the immune system and reactivate latent herpesviruses.

In CFS/ME/SEID/Fibromyalgia: chronic mold exposure, OspA (Lyme or LYMErix vaccine), fungal infections, contaminated vaccines, or a septic event cause TLR2- agonist tolerance (immunosuppression) and reactivation of latent herpesviruses. It is well known that mycoplasmas (TLR2/1 agonists) adhere to, and go inside red blood cells. Mycoplasmas cause permeability issues with red blood cells in which oxygen cannot cross the cell wall, hence, fatigue because of low oxygen. Couple that with the reactivation of EBV and you get double fatigue--fatigue that is not acknowledged because typical lab tests to diagnose anemia only look for a reduced cell count--not impaired cell functionality.

Finally, everyone should know the CDC was aware that injecting fungal antigens directly into the bloodstream causes irreversible immunosuppression. Consequently, they later performed research fraud in order to deny that mycoplasma play any role in fatigue or the disease of immunosuppression. They did this by throwing out the red blood cells to which mycoplasma adhere, before looking for mycoplasma:

Absence of Mycoplasma Species DNA in Chronic Fatigue Syndrome, 2003: "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)." http://jmm.sgmjournals.org/content/52/11/1027.long

It is important to understand what the CDC did here. They do not want anyone to know that mycoplasma are involved in Chronic Fatigue Syndrome.

Why?

Because this is the mechanism behind the Autism pandemic:

NYTimes; Doctors admit Thimerosal is put in vaccines to prevent fungi: Vaccine Rule Is Said to Hurt Health Efforts (Dec, 2012)

They say: "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&]

What is the Treatment?

We have established that tolerance to fungal antigens causes immunosuppression, reactivation of viruses, (i.e. latent herpesviruses and live, attenuated viruses in vaccines), susceptibility to other types of infections, and the "New Great Imitator" diseases, including autism and GWS. Additionally, herpesviruses are known to lead to cancer.

At this point, the next question is inevitably, "what's the treatment?"

Anthony Fauci, head of the National Institute of Allergy and Infectious Diseases (NIAID), has patented a treatment for the immune suppression outcomes of chronic Lyme disease -- a condition he simultaneously denies. The CDC calls it "fungal meningitis": http://www.cdc.gov/meningitis/fungal.html

Notice that the CDC's diagnostic criteria there matches exactly the new Policy Paper by IDSA on using Mass-Spec-PCR to identify DNA pathogens, here: http://ein.idsociety.org/media/publications/papers/2014/Blaschke_DMID_14_Unmet_Diagnostic_Needs.pdf

Fauci's patent for the treatment of Fungal Meningitis (Chronic Fatigue, Chronic Lyme and LYMErix Disease): 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/%205,696,079

"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. ..."

So, the question to Fauci is, why is this treatment not being used for all varieties of post-sepsis syndrome?

"It is an indescribable experience knowing that what you are doing will have an impact on the lives of tens, if not hundreds, of millions of people."

~ Anthony S. Fauci, M.D.

The Primers Shell Game – using the correct 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.

Or, "Science Made Stupid" by the CDC/ALDF, the Medical Mafia

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

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

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

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

V. SIDESTEPPING - Alert on "Biofilms"

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

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

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

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

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

BACKGROUND: The essence of these criminal charge sheets is that the Defendants make 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):

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 Lyme Cryme Defendants will probably attempt to say all this 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/ActionLyme criminal charge sheets and find how many ***other scientists*** referenced their work when these criminals were telling the truth. The CDC/ALDF criminal 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 Defendants either use 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 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 of 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. Please see:http://www.actionlyme.org/SASH_POLICYPAPER_MECFS.htm

"***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

Virological diagnosis of central nervous system infections by use of PCR coupled with mass spectrometry analysis of cerebrospinal fluid samples.
"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 Defendants: The Defendants deliberately use the wrong DNA to assess 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?

These victim-patients are deprived of their humanity, as well as functionality. They're tossed aside, sick, demoralized, ostracized, despised… and 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 Dearborn and Vaccines criminal charge sheet (http://www.ohioactionlyme.org/wp-content/uploads/2015/03/ALDF-CDC-Enterprise-Conspires-to-Defraud-USA-in-Dearborn-Vaccine-Scam.pdf) -, the actual number is 2 million per year. And that is a lot of human cost and disability that Uncle Sam will eventually have to pay for just so that a gang of low-lives could potentially capitalize on this new vaccines and test kits racket, the emerging, global pollution-related vector-borne-diseases, the ALDF.com. 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. Docs had their shot. They chose Kool-Aid and the age-old

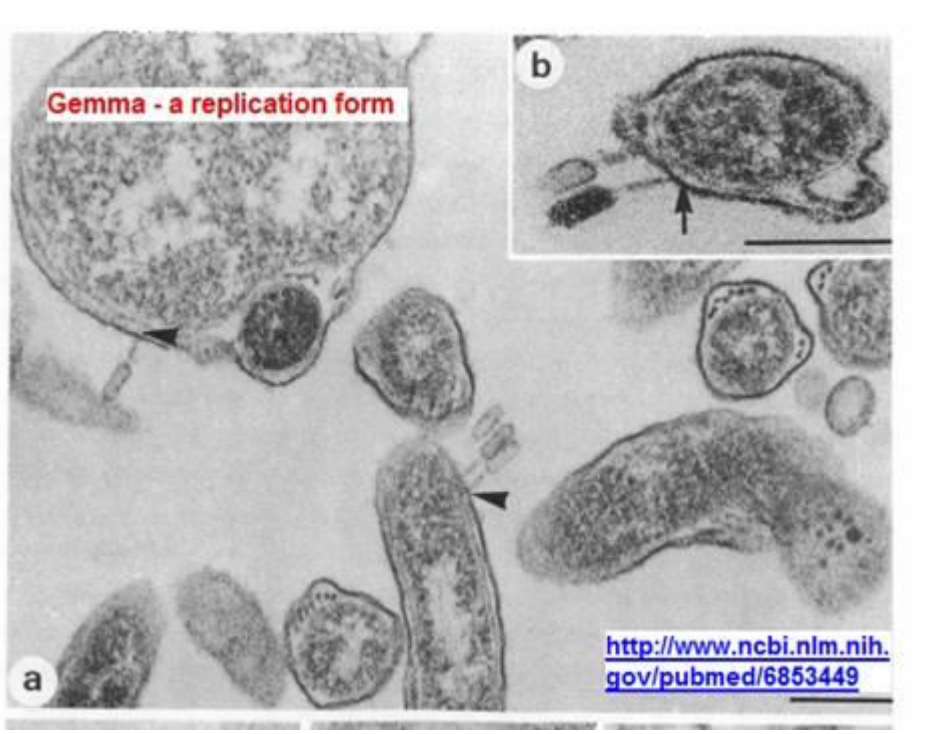
cliquish, clannish default position of looking down their noses and blaming their victims. Chose, people, and that's a spiritually dangerous thing from a bunch of First Do No Harm, oath-takers. 'Dangerous, also, because BS is never not a boomeranger. We saw that loud and clear with the 911 stunt and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

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 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):

1983 -- ***Bacteriophage in the Ixodes dammini spirochete, etiological agent of Lyme disease.***



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 encoded on those plasmid change all the time. So, there is only one species-determinant, flagellin. See also Casjens on this topic in the 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 have done all their damage early in the disease (matching the data from the U.S. Military's Jay Sanford in 1975, below), by shedding these varying, fungal antigens, as CDC officer Alan Barbour says in (the probably mis-titled) 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 secondaries, the latents (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 (Section IV, below):

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=%2Fnetahtml%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. The little one can learn about them is that they are apparently smaller than the Osps in molecular weight. 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.

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 and “vascular events” resulting from LYMErix or OspA vaccination.

Schoen on LYMErix Damage, the Phase IV data (strokes, cancer, “vascular events”):

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.
http://www.ncbi.nlm.nih.gov/pubmed/12637121
Full text at: http://www.actionlyme.org/OspA_4.htm

Korean Chemistry Journal on the Structure of OspA/Pam3Cys

Characterization of Extremely Hydrophobic Immunostimulatory Lipoidal Peptides by Matrix-Assisted Laser Desorption ionization Mass Spectrometry
"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."
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, vascular events described by Schoen and Steere in their Phase IV trial results (with a 9% cancer rate, also), 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:

2000 - ***Detection of multiple reactive protein species by immunoblotting after recombinant outer surface protein A lyme disease vaccination:***
"... 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

OspA 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 Defendants 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.
We don't know for sure if this particular ligand for plasminogen and chitinous tissue, OspA, was added 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

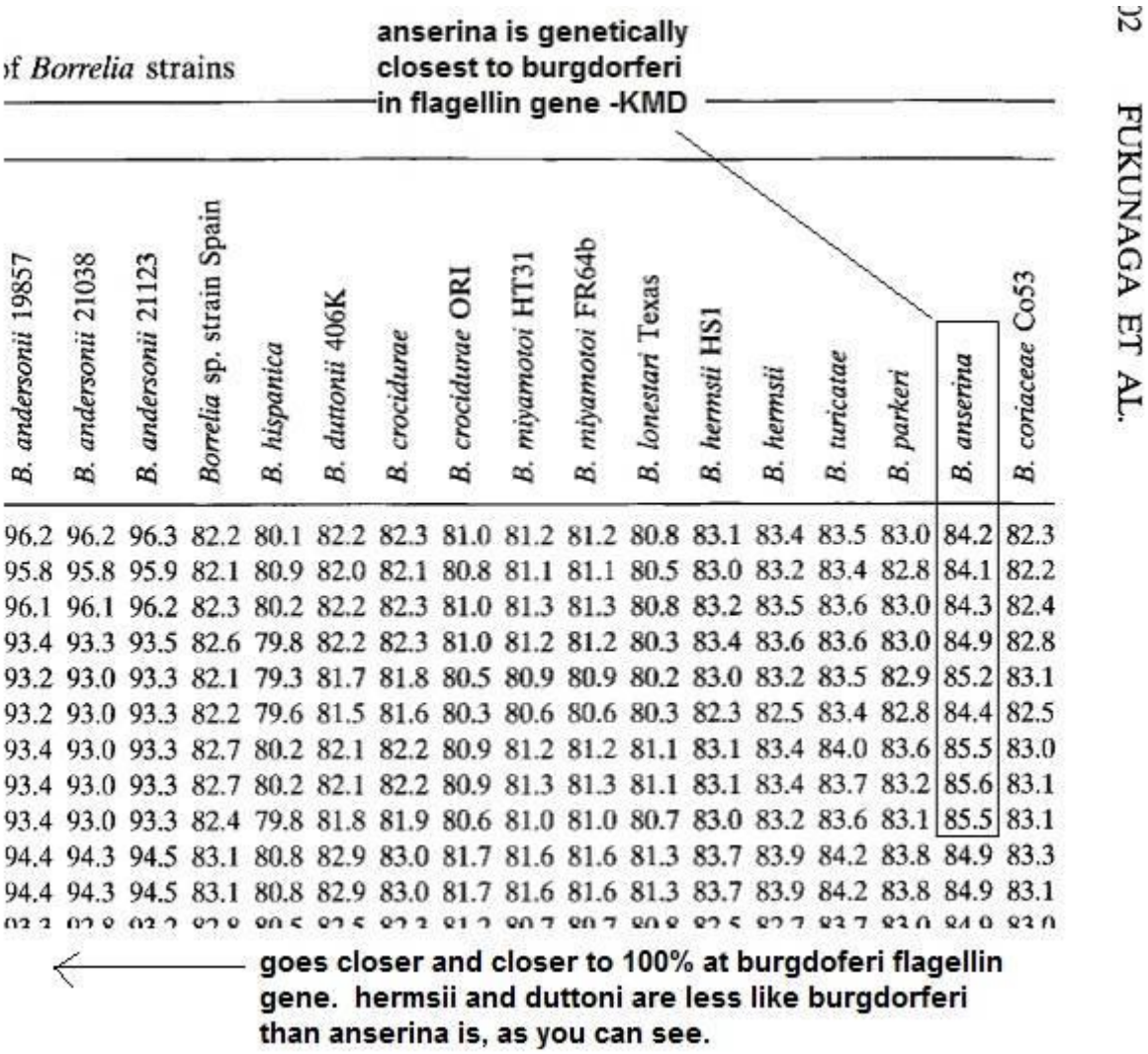
You can believe the CDC's theory that Lyme spirochetes/West Nile blew/flew from Africa to the northeastern United States on seabirds during hurricanes - actually what the CDC claimed/claims -, or, you can consider the circumstantial scientific evidence against the backdrop of CDC's other lies. For the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

"Migratory Birds and Spread of West Nile Virus in the Western Hemisphere
"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.

1996-- ***Phylogenetic Analysis of Borrelia Species Based on Flagellin Gene Sequences and Its Application for Molecular Typing of Lyme Disease Borreliae***



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).

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.

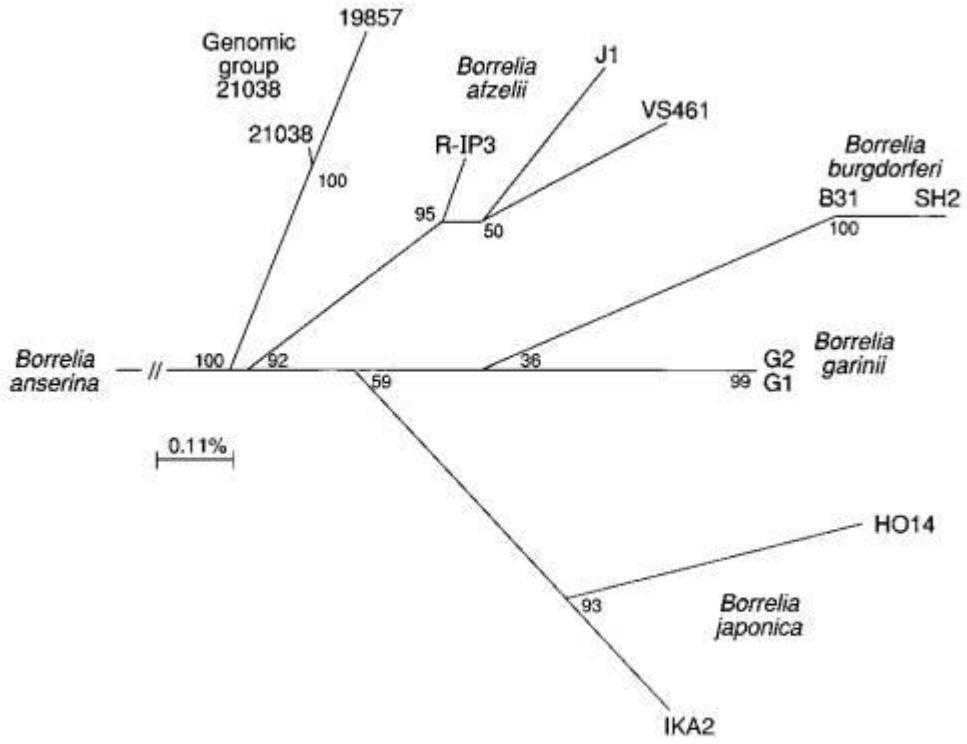


FIG. 7. Phylogenetic tree of 16S rRNA derived from LDS isolates. The phylogenetic tree was constructed as described in the text. Numbers at the branch nodes indicate the results of bootstrap analysis. The 16S rRNA sequence from *Borrelia anserina* served as an outgroup.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC228430/pdf/332427.pdf

UPenn on Lyme spirochetes being evolutionarily unlikely:

UNCOORDINATED PHYLOGEOGRAPHY OF BORRELIA BURGDORFERI AND ITS TICK VECTOR, IXODES SCAPULARIS:

”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-Stony Brook on Lyme/Plum Island as the original outbreak area (Ed Bosler):

Evolution of a focus of Lyme disease
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 SB1, Burrage TG, Scoles GA, Fish D, Rock DL.
1**Plum 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:

Immunogenic variation among the so-called LC strains of Mycoplasma mycoides subspecies mycoides.
“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.

So, challenging various vectors (bugs) with diseases from Africa is what Plum Island does. Naturally, an odd one 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 as "beyond a reasonable doubt," especially considering all the other lies about Lyme disease, like the hurricane fairy tale, the Idsociety.org'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 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***

1951: ***Relapse Phenomena in Rats with a Single Spirochete***
"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."
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 data: http://www.actionlyme.org/RICOCHRON.htm; and from the Defendants on the incurability of relapsing fever: http://www.actionlyme.org/BRAIN_PERMANENT.htm; an independent study on spirochetes in the brain from dentists and they say:

2002**--** ***Molecular and immunological evidence of oral Treponema in the human brain and their association with Alzheimer's disease.***
Riviere GR, Riviere KH, Smith KS.
Department of Pediatric Dentistry, School of Dentistry, Oregon Health and Sciences University, Portland, OR 97201-3097, USA.
"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 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 of Borrelia 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 1996,

> ***Biology of Borrelia Species***
> **"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 modem ultracold freezers, strains were kept in the brains of rodents and passed once or twice a year (92).
> 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. 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 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 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), at least, or current case definition in this way, using high-passage strains,
http://www.ohioactionlyme.org/wp-content/uploads/2015/03/ALDF-CDC-Enterprise-Conspires-to-Defraud-USA-in-Dearborn-Vaccine-Scam.pdf
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 in this report CDC officer Allen Steere playing 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 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 a third of his spinal-fluid, and synovial-fluid patients to the tune of at least a third of the patients.

1990 – Pachner, on human brain strains changing plasmid DNA code in mice:

> ***Borrelia burgdorferi infection of the brain: characterization of the organism and response to antibiotics and immune sera in the mouse model.***
> "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

The plasmid content was different, after a time, from the original strain says Pacher. 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. It's not an "end-stage," as some claim, it is a replication form. 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.

Where else to we find these fungal OspA like antigens?

> 1999-- ***Toll-like receptor 2 functions as a pattern recognition receptor for diverse bacterial products.***
> "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

These triacyl-lipopeptides only *initially* inflammatory. After a time, this same researcher, Radolf, wrote that these fungal lipoproteins cause immunosuppression and a lack of antibody production:

2001-- ***Toll-like receptor 2-dependent inhibition of macrophage class II MHC expression and antigen processing by 19-kDa lipoprotein of Mycobacterium tuberculosis.***
"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

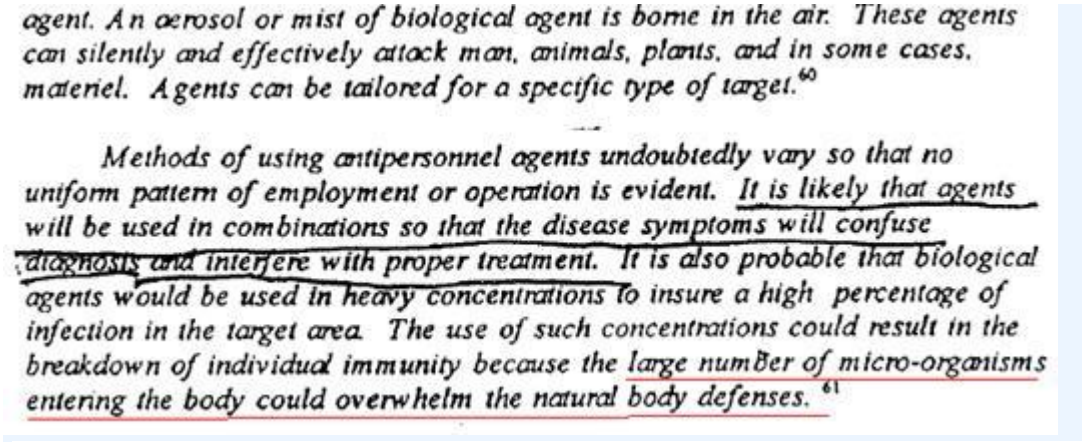
Different antipersonnel agents require varying periods of time before they take effect, and the periods of time for which they will incapacitate a person also vary. Most of the diseases having antipersonnel employment potential are found among a group of diseases that are naturally transmitted between 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 wary 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; 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 at:
http://www.ohioactionlyme.org/wp-content/uploads/2015/03/Patients-Guide-to-NIHs-Post-Sepsis-Syndrome.pdf

---

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):

> ***Demonstration of Spirochaetes in Patients with Lyme disease with a Modified Silver Stain***
> 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]:

> "***Morphology of Borrelia burgdorferi: structural patterns of cultured borreliae in relation to staining methods.***
> "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, and while they were always known to be covered in a slime layer, are *not* the main cause of the disease or the reason antibiotics fail. See more at http://www.ohioactionlyme.org/wp-content/uploads/2015/03/Patients-Guide-to-NIHs-Post-Sepsis-Syndrome.pdf for what "Chronic Lyme" is really all about.
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:
http://www.ohioactionlyme.org/wp-content/uploads/2015/03/ALDF-CDC-Enterprise-Conspires-to-Defraud-USA-in-Dearborn-Vaccine-Scam.pdf

---

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:

"***Molecular characterization of the humoral response to the 41-kilodalton flagellar antigen of Borrelia burgdorferi, the Lyme Disease spirochete.***"
"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

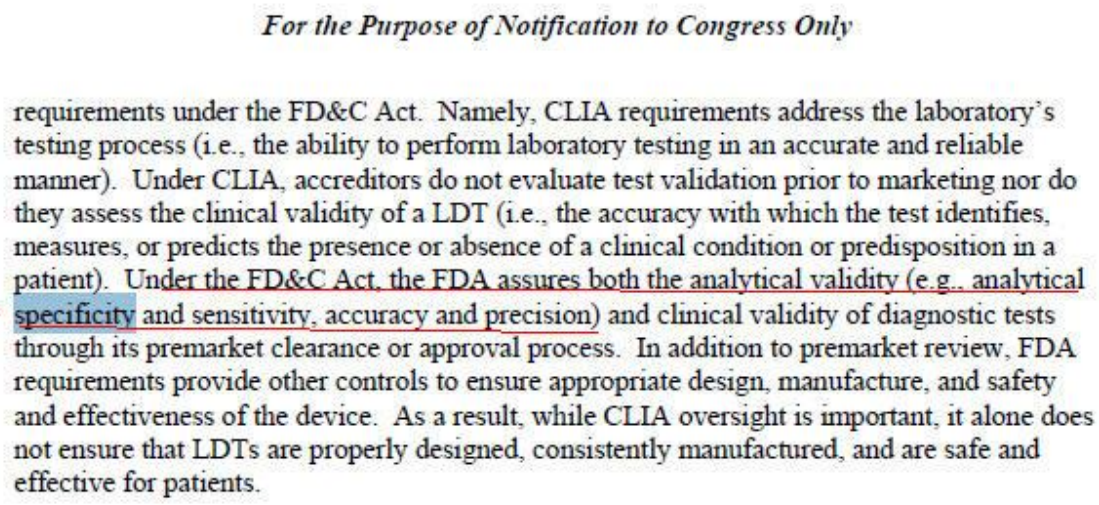
cases," says the FDA, verbatim), *and this method was made SPECIFIC, which means it does not detect any other flagellins from non-Borreliae organisms.*
When the FDA says "sensitivity," they really mean "LIMIT OF DETECTION" and refer to the METHOD and not the "CASES." "Accuracy" addresses cases. Yale, 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 flagellins 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 Defendants misuse 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.

> 1991-- "***Molecular characterization of the humoral response to the 41-kilodalton flagellar antigen of Borrelia burgdorferi, the Lyme Disease spirochete.***"
> 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-flagellins 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 use 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.”
See the ***The Patient’s Guide to NIH’s Post Sepsis Syndrome*** report by the Society for the Advancement of Scientific Hermeneutics (AH), http://www.ohioactionlyme.org/wp-content/uploads/2015/03/Patients-Guide-to-NIHs-Post-Sepsis-Syndrome.pdf and the Vaccine Scam report: http://www.ohioactionlyme.org/wp-content/uploads/2015/03/ALDF-CDC-Enterprise-Conspires-to-Defraud-USA-in-Dearborn-Vaccine-Scam.pdf. 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):

> ***RECOVERY OF TREPONEMA AND BORRELIA AFTER LYOPHILIZATION***
> http://www.ncbi.nlm.nih.gov/pmc/articles/PMC277387/pdf/jbacter00438-0287.pdf

Next, the CDC is throwing out the blood cells (throwing out whole cells of any kind), including immune cells or white cells to which mycoplasma adhere, while alleging to look for mycoplasma kin Chronic Fatigue Syndrome. Mycoplasma or epERTHROzoa are called epERYTHROzoa because they're known to be attached to red blood cells. Such epERYTHROzoa are famous for changing the erythrocyte membrane potential and this the ability of Oxygen to cross the membrane, causing tremendous fatigue even in amimals.
CDC did this to allegedly show Mycoplasma were not involved in Chronic Fatigue Syndrome:

> "***Absence of Mycoplasma species DNA in chronic fatigue syndrome***"
> "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."
> 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 extreme fatigue. They must be important bioweapons, a problem with the pediatric vaccines causing Autism, or both.

Next up the DNA Shell Game. 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 committing research fraud in other arenas.

IX. CDC and Associated Defendants 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:

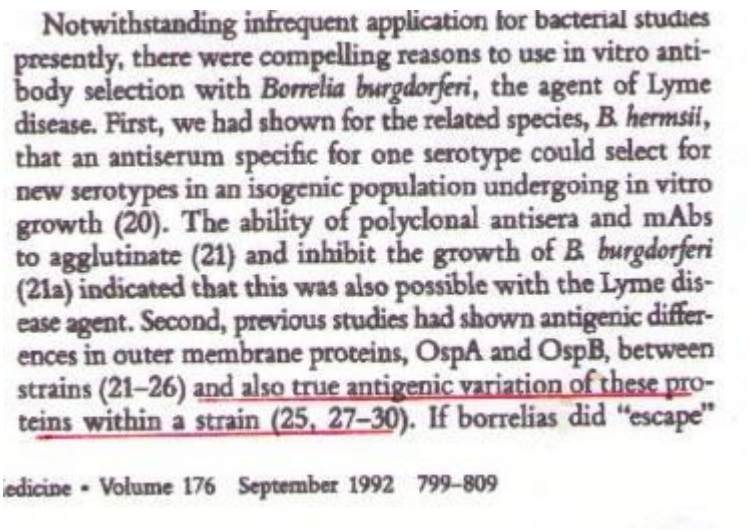
You will want to look at The Patents criminal charge sheet for Cryme Disease, http://www.ohioactionlyme.org/wp-content/uploads/2015/02/Lyme-Disease-Patents8.pdf to see that CDC officer and former head of the NIH's Rocky Mountain Bioweapons Montana Lab (you're familiar with Montana, the place where there 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; more can be seen on the Bioweapons pages of ActionLyme.org
(http://www.actionlyme.org/120702.htm and http://www.actionlyme.org/BIOWEAPONS_ATTRIBUTES.htm).

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 longed called the CDC

the Centers for Disease Confabulation.

Alan Barbour states below that OspA undergoes true antigenic variation and that you cannot use this as a vaccine (while he owns the patent for the ImuLyme OspA non-vaccine). If it undergoes "true antigenic variation," it certainly cannot be used for DNA diagnostics as Klempner 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.

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



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"

edicine • 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 every prevented Lyme in any animal, by the way. 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:

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

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=%2Fnetahtml%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 OspA for a vaccine, for post-treatment 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, likely making it spread around fastest.

1995 – Barbour's patent for Lyme in Missouri, using 16S RNA sequencing and flagellin primer probes that Gary Wormser did not use when trying to fool Edwin Masters. The Gary Wormser Chapter of the Primers Shell Game is below in this document. More background on the genetic relatedness and the history of discovery of various Borreliae has to be shown here, first.

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=%2Fnetahtml%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 prrimer 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.

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.

1992 -- ***Diagnosis of early Lyme disease by polymerase chain reaction amplification and culture of skin biopsies from erythema migrans lesions.***
*"*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. ansenina 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, Gl, 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 amplimer 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 amplimer 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.

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. Regardless, 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."**

Genetic diversity of Borrelia burgdorferi in lyme disease patients

"... 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):

Identification of an uncultivable Borrelia species in the hard tick Amblyomma americanum: possible agent of a Lyme disease-like illness.

"…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. miyamotae 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. miyamotae sp. nov. (accession no. 045192)."

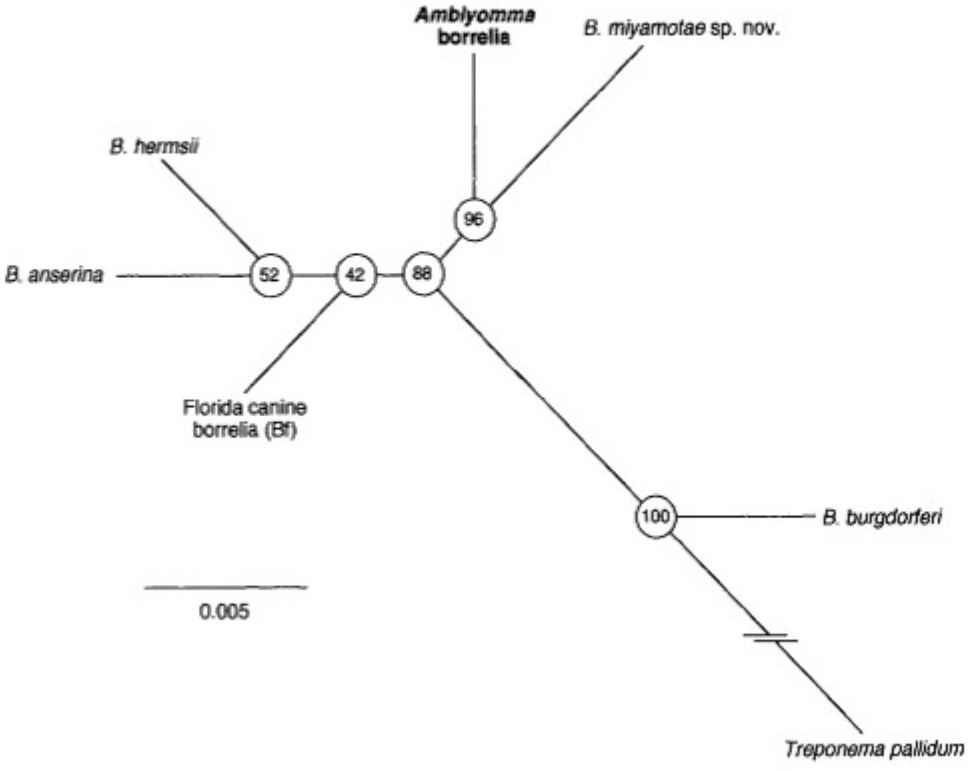


Figure 4. Unrooted distance matrix phylogenetic tree of *Borrelia* species with *Treponema pallidum* as outgroup. 16S rRNA sequences corresponding to base positions 36–1371 of *Borrelia burgdorferi* 16S rRNA gene were aligned and analyzed with PHYLIP program package. Exhibited tree in New Hampshire standard format is: (((Florida canine borrelia: 100, (*Borrelia anserina:* 100, *Borrelia hermsii,* 100): 52): 42, (borrelia from *A. americanum:* 100, *Borrelia miyamotae* 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. miyamotae:* 100): 94, *B. hermsii:* 100): 34, Florida canine borrelia: 100): 25, *B. anserina* 100): 81, *B. burgdorferi:* 100): 100, *T. pallidum:* 100).

”Figure 4. Unrooted distance matrix phylogenetic tree of Borrelia species with Treponema pallidum as out group. 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 miyamotae 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. miyamotae: 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

Barbour actually sequenced for flagellin and 16S RNA and found all kinds of spirochete in this way. Gary Wormser did no such thing when trying to find “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”

2005: ***Microbiologic evaluation of patients from Missouri with erythema migrans.***
“PCR amplifications were performed in a 50-μL 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 μmol/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′-GTTTTGTAATTTCAACTGCTGACCCCTC-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 the other relapsing fever groups) flagellin genes, or 16S rDNA specific to Borrelia species, nor did he actually try to sequence any of these Borrelia 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.

Glycerol-3-Phosphate Acquisition in Spirochetes: Distribution and Biological Activity of Glycerophosphodiester Phosphodiesterase (GlpQ) among* Borrelia*Species
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 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.)
Importantly, Wormser makes one more very incriminating revelation in his "No Lyme in Missouri" report:

> "One tick yielded a PCR product, which was cloned and subjected to DNA sequencing. DNA database analysis revealed the sequence to be most closely related to an uncharacterized a proteobacterium (GenBank accession number **AJ459874**), consistent with a contaminating soil bacterium or a tick endosymbiont."
> http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2773674/

Let's guess that that was a fungal type organism living in the soil, adding to the probability that fungi injected directly into the bloodstream, like the OspA vaccine, caused a chronic Lyme-like illness. We know this is supported by other data on failed Tuberculosis vaccines and is the reason – to prevent fungi – Thimerasol is put in vaccines: Fungal-Viral Synergy or Post-Sepsis Syndrome:
http://www.actionlyme.org/SASH_POLICYPAPER_MECFS.htm

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:

Two Controlled Trials of Antibiotic Treatment in Patients with Persistent Symptoms and a History of Lyme Disease
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

Detection of Borrelia burgdorferi DNA by Polymerase Chain Reaction in Cerebrospinal Fluid in Lyme Neuroborreliosis
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 μL of CSF, and the DNA extract was resuspended in 30 μL of ultrapure water. A 5-μL 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-μL 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, Klempner says he found none (2001 RI "Diseases of Summer" conference at South County Hospital, audiotaped).

The crooks – including Klempner 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 for16S (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.

2001 - ***A relapsing fever group spirochete transmitted by Ixodes scapularis.***
"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-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 Borrelia*burgdorferi*–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′-AACCCTTTACACTTTCTTCGATTGCGCT-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.

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," and Alan Barbour recommending infecting syphilis patients with old, wimpy, high-passage borrelia spirochetes to raise a fever. Spirochetal diseases are all incurable, as shown above. And surely it is true that older spirochete populations in the same host lose plasmids – another reason not to use plasmid DNA to assess Lyme in humans -, but the end game, and the point of all this crime, is that the Defendants are 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

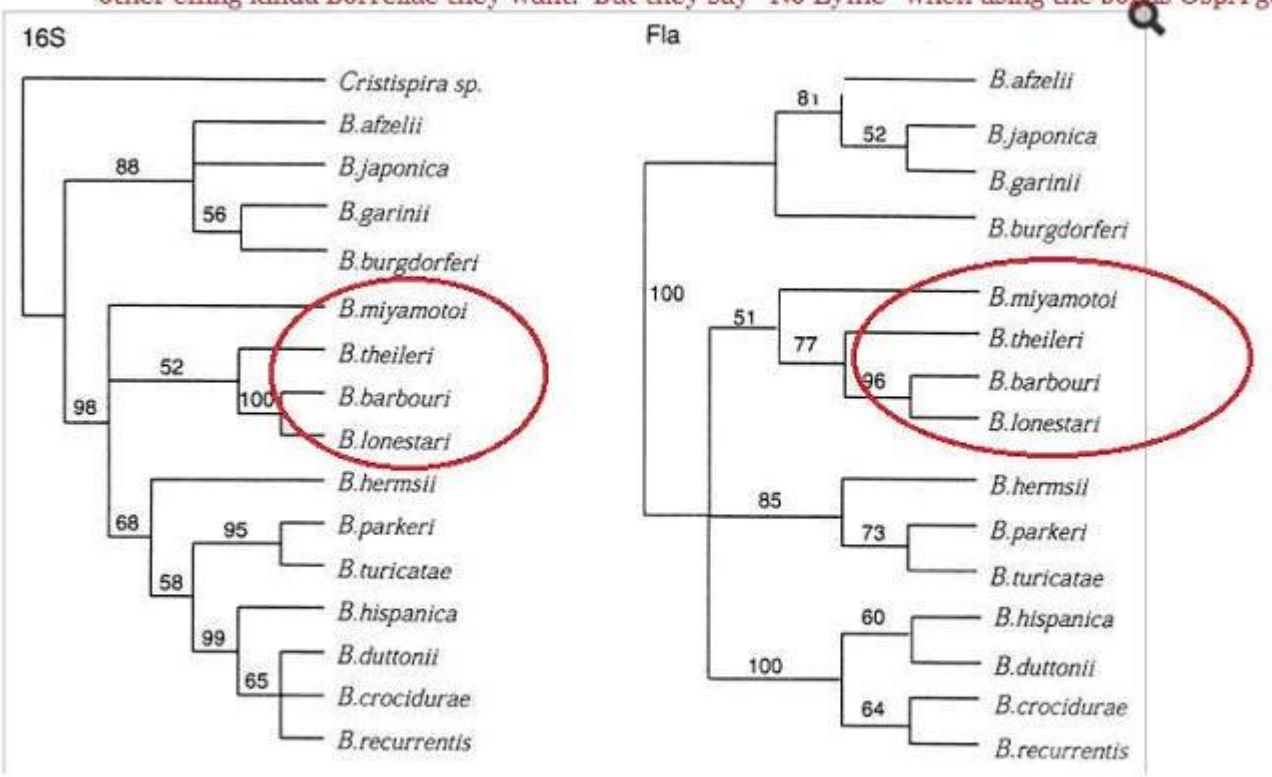
herpesviruses, Post-Sepsis Syndrome, and humoral immunosuppression with chronic inflammation in the brain due to all the neurotropic herpes viruses and Mycoplasma, etc., they exploded their scam. It was a very dumb choice for a vaccine.

All of what you see in these SASH criminal charge sheets is simply 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 Defendants so vigorously try to mask by issuing "Guidelines." The "Guidelines," based on Klempner, which is based on Dearborn, is a way of Offense being the Best Defense. The Defendants would have the world believe that *they* all believe the case definition was real and valid. We know for sure they know it is not valid, based on the consensus at Dearborn alone:
http://www.ohioactionlyme.org/wp-content/uploads/2015/03/ALDF-CDC-Enterprise-Conspires-to-Defraud-USA-in-Dearborn-Vaccine-Scam.pdf
To put such criminals 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):

> ***Lone star tick-infecting borreliae are most closely related to the agent of bovine borreliosis.***
> "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-uncultivatable 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 Defendants 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 flagellins 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: "OspA is bogus due to antigenic variation"

> ***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.***
> "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 and Barbour 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:

Borrelia burgdorferi enzyme-linked immunosorbent assay for discrimination of OspA vaccination from spirochete infection.
"…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 *Hin*dIII 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 - 1995-- ***Two geographically distinct isolates of Borrelia burgdorferi from the United States share a common unique ancestor.***
“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:

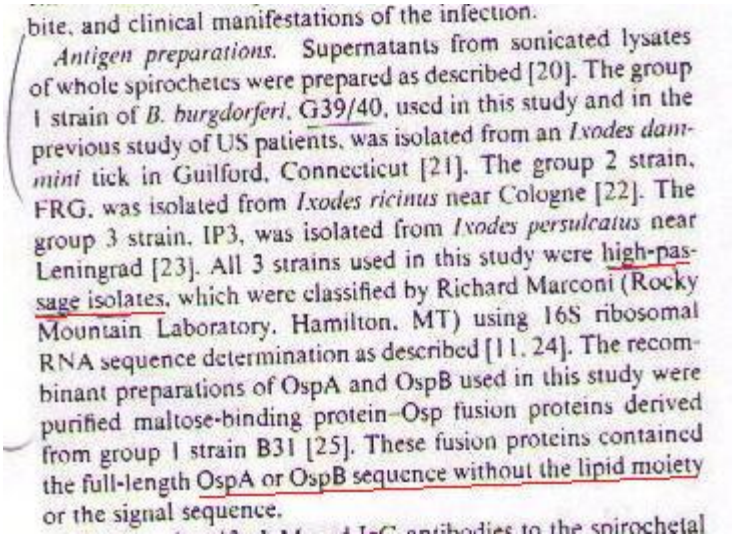
http://jid.oxfordjournals.org/cgi/crossref-forward-links/175/1/98

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

"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 -"

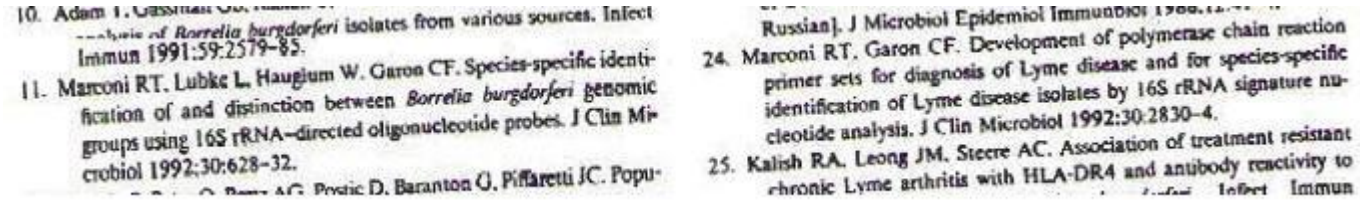


bite, and clinical manifestations of the infection.

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 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.

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/STEERE_IN_EUROPE.htm



10. Adam T. Classification of *Borrelia burgdorferi* isolates from various sources. Infect Immun 1991;59:2579–85.
11. Marconi RT, Lubke L, Haugium 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.

Russian]. J Microbiol Epidemiol Immunobiol
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--

Species-specific identification of and distinction between Borrelia burgdorferi genomic groups by using 16SrRNA-directed oligonucleotide probes.

"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:

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.
"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."

1994-- ***Detection of Borrelia burgdorferi DNA by polymerase chain reaction in synovial fluid from patients with Lyme arthritis.***

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 (> or = 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#t=articleResults

And

1996 -- ***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

Steere does not use the 16S RNA probe in this assessment, whereas he had before in knees and found more than a third of the patients were positive after treatment. OspA as a ligand for chitinous tissue, this tissue tropism driven by antigenic variation would certainly allow spirochetes to select for OspA expression. As shown by Pachner above using a human neuroinvasive strain (N40 was taken from the spinal fluid of a human patient), he found the plasmids had changed once transferred to mice. It is quite evident 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, CDC's goal from the beginning. The CDC deployed Allen Steere in the first place – a rheumatogist -- to manage a vector-borne, neurologic disease? Never made any sense.
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.

X. The Guidelines – Who signed on to this perverted science and are <u>therefore responsible</u> 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 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

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 IDSA's 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 criminal gang is guilty of the same thing – not sequencing for Borrelia DNA*** (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 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, and all their own bogus articles have to be retracted in addition to these criminals' 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.

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

Contents

I. Background on fungal antigens (mycoplasmal, eperythrozoal, mycobacterial, spirochetal) causing immunosuppression, changes to erythrocyte osmotic potential (hypoxic fatigue), and activating vaccine viruses:

> ***1953 IV. THE RELATIONSHIP OF EPERYTHROZOON COCCOIDES TO THE HEPATITIS VIRUS OF PRINCETON MICE***
> "In Swiss mice, animals with high natural resistance to hepatitis virus, the pathogenicity of this agent was markedly enhanced by combined infection with eperythrozoa. Eperythrozoa were maintained throughout 18 successive passages in normal Princeton and Swiss weanlings with intact spleens. The combined infection of Princeton mice with eperythrozoa and the virus component of Gledhill, Dick, and Andrewes, which is nearly inactive when injected alone, resulted in acute hepatitis with fatal outcome."
> http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2136329/?tool=pubmed
>
> ***[The effect of Eperythrozoon suis infection on the osmotic fragility of erythrocytes]***
> "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

So, what is a fungal antigen? What is "What's handled by TLR2 and TLR1 as a dimer?" Among them are tri-acylated antigens like OspA (there are others). Look up on Wikipedia or elsewhere (google images) these terms, before proceeding here. We are talking about how the immune system manages molecules that make up infectious disease pathogens. Science is all visual, so take it slow to absorb the images in your mind, or at least find a way to have easy access to these images and their structure-function again.

Remember that the entire HHS.gov and so called "medical" field and so-called "doctors" have entirely defaulted on all medical science. Therefore, this is a wide-open field for all well trained persons, especially in this business of immunosuppression-come-virus-reactivation that the authors are relating here.

There are 8 million people in America who have Fibromyalgia and 4 million who have Chronic Fatigue Syndrome according to the NIH, yet still, to this day (2015), we have to suffer idiots and low-lives with "MD" after their names, who in the media claim there are not valid biomarkers of Chronic Fatigue/ME/Fibromyalgia. Clearly such individuals gave up reading and rely totally on the drug reps for medical training. And there is no drug yet that reverses fungal antigen tolerance, and there are a huge number of people worldwide who have suffered through septic shock and they all have the same, "chronic poor health" outcome. Therefore if you are a human being and an English speaker or writer or have access to Western culture and science databases, all this belongs to you, and is your burden and your privilege to serve and share with others so that they no longer be slandered, libeled and abused.

Spirochetes bear and shed FUNGAL antigens of the "TLR2/1" type or are tri-acylated (have 3 fatty acid groups attached to a protein group with a very electronegative cysteine with 3 esters at its core). This type of antigen suppresses the immune system in most people -in 85% of us who do not have the arthritis prone HLAs. That means chronic Lyme - and all spirochetal diseases are

permanent - will not test as if it is a hypersensitivity response in most people. Yet, the Lyme ELISA only detects these HLA-linked hypersensitivity cases.

2001; ***Toll-like receptor 2-dependent inhibition of macrophage class II MHC expression and antigen processing by 19-kDa lipoprotein of Mycobacterium tuberculosis.***
Department of Pathology, Case Western Reserve University and University Hospitals of Cleveland, Cleveland, OH 44106, USA.
Noss EH, 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

The following is an example of Pam3Cys or OspA taken from a Korean journal article on HIV's and SIV's Pam3Cys:

"Characterization of Extremely Hydrophobic Immunostimulatory Lipoidal Peptides by Matrix-Assisted Laser Desorption Ionization Mass Spectrometry," Bull. Korean Chem. Soc.1996, Vol. 17, No. 11
http://newjournal.kcsnet.or.kr/main/j_search/j_download.htm?code=B961118

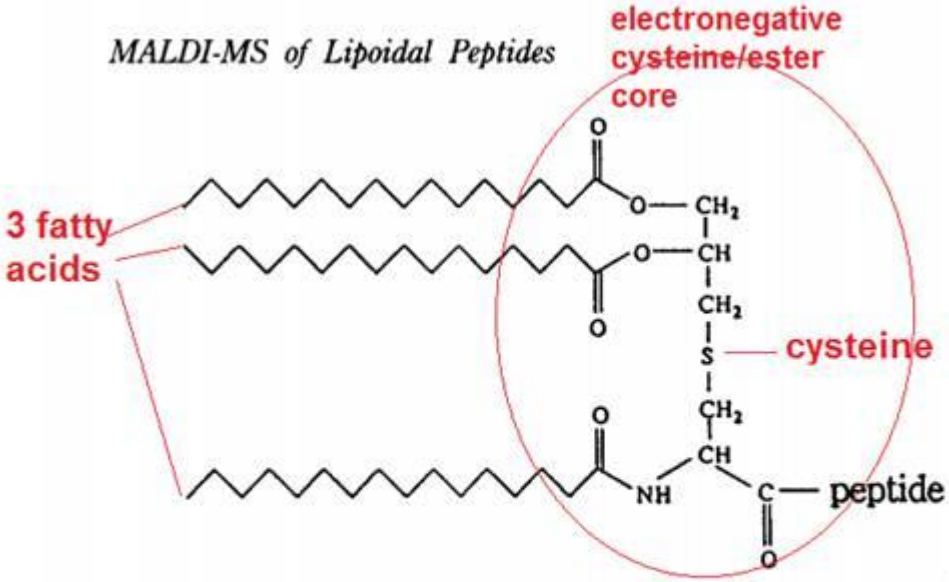


Figure 1. Structure of N-palmitoyl-S-[2,3-bis(palmitoyloxy)-propyl]-cysteinyl (Pam_3Cys) peptide, showing the modified cysteine residue with an N-palmitoyl ester and a dipalmitoylglyceryl moiety connected via a thioether linkage. This amino acid is placed at the N-terminus of the synthetic peptide.

We are currently using several mass spectral techniques to characterize the amino acid sequences of the Pam_3Cys 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,

"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."

Very much like what happened to OspA in the vaccine vials resulting in the blot-smudging that made the OspA vaccine trial results totally unreadable, which is another aspect of this Lyme fraud.

There are other TLR2/1 agonists besides triacyl lipoproteins from fungal pathogens. (In your PubMed research also use the term TLR1/2- agonist.)

II. SUNY-SB/Dattwyler/JJ Halperin/IDSA on seronegative, immunosuppressive Lyme and fungi, when discussing LYMErix, OspA or Borreliosis (1988)

Modulation of natural killer cell activity by Borrelia burgdorferi.

"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 supernatant would naturally contain the hydrophobic lipoproteins like OspA.

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

"The diagnosis of Lyme disease often depends on the measurement of serum antibodies to Borrelia burgdorferi, the spirochete that causes this disorder. Although prompt treatment with antibiotics may abrogate the antibody response to the infection, symptoms persist in some patients.
We studied 17 patients who had presented with acute Lyme disease and received prompt treatment with oral antibiotics, but in whom chronic Lyme disease subsequently developed. Although these patients had clinically active disease, none had diagnostic levels of antibodies to B. burgdorferi on either a standard enzyme-linked immunosorbent assay or immunofluorescence assay. On Western blot analysis, the level of immunoglobulin reactivity against B. burgdorferi in serum from these patients was no greater than that in serum from normal controls.
"The patients had a vigorous T-cell proliferative response to whole B. burgdorferi, with a mean (±SEM) stimulation index of 17.8±3.3, similar to that (15.8±3.2) in 18 patients with chronic Lyme disease who had detectable antibodies. The T-cell response of both groups was greater than that of a control group of healthy subjects (3.1+0.5; $P<0.001$).
"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 sero-negative patients with clinical indications of chronic Lyme disease. (N Engl J Med 1988; 319:1441–6.)…
"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 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

This was one of JJ Halperin's and Raymond Dattwyler's references for the above Seronegative Lyme Assay:

1976; ***Suppressor thymus-derived lymphocytes in fungal infection.***

"Thymus-derived lymphocyte (T-cell) function, as determined in vivo by cutaneous reactivity to several antigens and in vitro by responsiveness to mitogens and antigens, was assessed in 14 patients infected with a variety of fungal organisms. While all patients manifested a normal frequency of peripheral blood T cells, only seven patients reacted to at least one of the antigens used for cutaneous testing and demonstrated normal in vitro T proliferative responses. Three patients exhibited cutaneous anergy but normal in vitro T-cell reactivity while four patients demonstrated persistent anergy and marked in vitro T-cell hyporeactivity which was independent of activity of infection, concurrent medication, or any associated disorders. The marked diminution of in vitro T-cell reactivity noted for these later four patients was not due to a deletion of antigen- or mitogen-reactive cells. Thus, patients' cells which had been initially cultured for 7 days without any mitogenic or antigenic stimulus and which were subsequently washed and recultured with phytohemagglutinin, concanavalin A, or histoplasmin demonstrated a marked increase in their responsiveness. Moreover, this reactivity noted for recultured cells could be suppressed by a nonphagocytic, nonadherent, nonimmunoglobulin-bearing, sheep red blood cell rosette-forming population of cells isolated from the fresh peripheral blood mononuclear cells of the same patient. While these "regulator" T cells were capable of suppressing T-proliferative responses to antigens and mitogens, they did not diminish pokeweed mitogen-induced immunoglobulin synthesis by normal bone marrow-derived lymphocytes. Patients in whom suppressor "T" cells were found were at risk for relapsing, disseminated fungal infection."

Look at the references here (dates, topics):
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC436656/pdf/jcinvest00145-0081.pdf
http://www.ncbi.nlm.nih.gov/pubmed/1082888

III. Thimerosal is put in vaccines to prevent fungi because they help activate viruses via immunosuppression, and inhibition of apoptosis of fungally infected B cells in particular.

2012, Dec, NYTimes; 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&

IV. CDC and BigPharma in fungal-viral synergy / vaccine-failure conditions and vaccines (On Kynurenines, serotonin and melatonin synthesis, ROS by-products and intracellular damage resulting in pain & other symptomology without classical "autoimmunity" and "inflammation" – SSRIs and Lyrica as treatments come to mind)

CDC's Patent, US # 7,632,510,
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,…***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 - KMD]."
http://www.cdc.gov/mmwr/preview/mmwrhtml/00053391.htm

CDC SAYS: ***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 mycoplasmally (LYMErix-like) contaminated vaccines -KMD.]

In the case of pandemic MRSA, as we have seen, the vaccine didn't work because TLR2 agonists (lipoproteins) suppress the immune system. We also learned from this MRSA vaccine patent, that (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 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

CDC SAYS that 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
[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 wannabee doctors are stressed out, they may have cortisol-activated EBV. But regular humans no, they have some psychiatric disorder. Why the big secret, no one knows since it's common knowledge that arrogance is the cowardly calling card of assholes.]

1981; ***Adhesion of mycoplasmas to eukaryotic cells.***

"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

RELATED ARTICLES TO THE ONE IMMEDIATELY ABOVE (Read!!, 240):
http://www.ncbi.nlm.nih.gov/pubmed?linkname=pubmed_pubmed&from_uid=6790254

AND this is on oxidized lipoproteins

http://www.ncbi.nlm.nih.gov/pubmed?linkname=pubmed_pubmed&from_uid=20036735

Remember ROS are by-products of lipids broken down by kynurenines/quinolinic acid, the production of which makes L-tryptophan less available to produce serotonin and melatonin.

Quinolinic acid – intracellular ROS from mycoplasma lipids:
http://en.wikipedia.org/wiki/Kynurenine_pathway#mediaviewer/File:KP_pathway.jpg

This is might be one of the reasons you get pain without classic "autoimmune" humoral, "inflammation." It's a cellular response, not humoral (as seen in classic autoimmune diseases) as explained by Dattwyler, above.

Finally, everyone should know that the CDC, knowing fungal antigens injected directly into the bloodstream causes irreversible immunosuppression and "immune damage," later performed research fraud in order to deny that mycoplasma play any role in the chronic immune-suppression disease or fatigue: Throwing out the RBCs to which mycoplasma adhere before looking for mycoplasma, clue:

> 2003; ***Absence of Mycoplasma species DNA in chronic fatigue syndrome***
>
> "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)."
> http://jmm.sgmjournals.org/content/52/11/1027.long

So, it is pretty important that everyone know what the CDC did here. They do not want anyone to know mycoplasma are involved in Chronic Fatigue Syndrome.

V. Fungal antigens inhibit apoptosis – the first step in chronic disease and dysimmunity; seen again recently in Treg boosters in chemo. State of the Art mentions Pam3Cys inhibits apoptosis (2013)

> 2004, Israel, ***Mycoplasma fermentans inhibits tumor necrosis factor alpha-induced apoptosis in the human myelomonocytic U937 cell line.***
> ***"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

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

2000; Gary Wormser and his exact language when he writes about how OspA inhibits the immune response:

Modulation of lymphocyte proliferative responses by a canine Lyme disease vaccine of recombinant outer surface protein A (OspA).

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

By the way, no dog vaccine ever prevented spirochetes. The vaccines may have blunted the arthritis result, but they never prevented spirochetes. You'll notice in their language that they measure their fake vaccines efficacies in the production of antibodies rather than whether there is any spirochetal flagellin DNA to be found in their mammalian victims.

VI. Fungi/ TLR2/1-agonists, as are found in mycoplasma and spirochetes, cause immunosuppression in the form of a lack of antibodies as shown in the downregulation of the HLA/MHC molecules and cytokine production. (In parallel = failed fungal vaccines)

1999, Radolf, et al:
Toll-like receptor 2 functions as a pattern recognition receptor for diverse bacterial products.
"Lyme disease and syphilis are acute and chronic inflammatory disorders caused by the spirochetal pathogens *Borrelia burgdorferi* and *Treponema pallidum* subsp. *pallidum*, respectively (15, 16). Both spirochetes lack LPS (**17**, **18**); however, they do possess abundant membrane lipoproteins (**19**). There now exists a large body of evidence that spirochetal lipoproteins and synthetic lipohexapeptide analogs are potent activators of monocytes/macrophages, neutrophils, lymphocytes, endothelial cells, and fibroblasts and that acyl modification of the peptides is essential for these proinflammatory activities (**20-29**). More recent

observations suggest that the mechanisms underlying monocytic cell activation by motile ***B. burgdorferi*** and ***T. pallidum*** are identical to those employed by their purified membrane constituents (**30**). These results support the notion that lipoproteins are the principle component of intact spirochetes driving the host immune response during Lyme disease and syphilis. Similarly, lipoproteins and lipopeptides derived from the human pathogen ***Mycoplasma fermentans*** are also potent activators of monocytes/macrophages and may play an important role in the inflammatory response during infection (**31-33**).

"The cellular activation induced by the lipoproteins or lipoprotein-derived lipopeptides from *B. burgdorferi* and *T. pallidum* resembles that of the LPS signaling pathway, as CD14 appears to facilitate cellular activation by both types of pathogenic membrane structures (21, 25). However, several differences have been observed between LPS and lipoprotein cellular activation, indicating the utilization of different signaling elements. For example, spirochetal and mycoplasma lipoproteins and lipopeptides activate macrophages from LPS hyporesponsive C3H/HeJ mice (23, 24, 27,31). In addition, whereas Chinese hamster ovary (CHO)-K1 cells become remarkably sensitive to LPS after transfection with CD14 (34-36), they remain insensitive to the lipoproteins, lipopeptides, and motile *B. burgdorferi* (21, 30, 32). These observations led us to hypothesize that differences in main signaling components exist between lipoproteins and LPS.

"We have recently found that CHO-K1 cells do not express an mRNA transcript for full-length and functional TLR2 (37). This observation raised the possibility that the lack of functional TLR2 might account for the failure of CHO/CD14 cells to respond to bacterial structures other than LPS. To test this hypothesis, we engineered stable CHO/CD14 fibroblast cell lines that express TLR2. The transfected cells were highly susceptible to activation by lipoproteins and lipopeptides from *B. burgdorferi*, *T. pallidum*, and *M. fermentans*, as well as to activation by live motile *B. burgdorferi*. In contrast, cells expressing TLR1 or TLR4 did not acquire responsiveness to bacterial lipoproteins/lipopeptides. Moreover, we observed a TLR2-mediated cell activation by ***Mycobacterium avium***, an important pathogen in AIDS. Similar studies have documented inducible responses to other bacteria as well, including staphylococci, listeria, tuberculosis, and the pneumococcus, suggestive of wide-spread recognition of bacteria by TLR2 (**10**, **11**,**38**,**39**).**2** **3** We propose that TLR2 mediates cellular responses to structures from numerous microbial cell wall constituents and may thus be central in host recognition of diverse bacterial pathogens. Therapies directed at the TLRs may be useful anti-inflammatory agents for a large variety of chronic and acute bacterial infections.
http://www.jbc.org/content/274/47/33419.long
http://www.ncbi.nlm.nih.gov/pubmed/10559223

Mycobacterium tuberculosis LprG (Rv1411c): A Novel TLR-2 Ligand That Inhibits Human Macrophage Class II MHC Antigen Processing1
http://www.jimmunol.org/cgi/content/full/173/4/2660

The 19-kD antigen and protective immunity in a murine model of tuberculosis.
"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."
http://www.ncbi.nlm.nih.gov/pubmed/10792376

Mycobacterium tuberculosis 19-kilodalton lipoprotein inhibits Mycobacterium smegmatis-induced cytokine production by human macrophages in vitro.
http://www.ncbi.nlm.nih.gov/pubmed/11179309

LUFT and Schoen/Persing on OspA – fungal - causing systemic disease (parallels the above TB vaccine failure and the failed childhood vaccines failure):

1998, FDA Minutes (read all of it, it is pretty awesome):
http://www.fda.gov/ohrms/dockets/ac/98/transcpt/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 [means not limited to "bad knees- KMD]. 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 sero positive, you can start to have a different take on when someone has an adverse event of 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. I can only say from my own ..."

1996; Persings RICO-RICO patent developed with Robert Schoen on LYMErix causing systemic disease like chronic Lyme (unsurprising since it is a TLR2/1 agonist and therefore fungal like the failed Tb vaccines):

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

Chronic Fatigue Data, independent from the CDC. In it you can see a generalized immune deficiency and low cytokines production.

***Changes* in *immune parameters* seen in *Gulf War* veterans but not in civilians with *chronic fatigue* syndrome.**

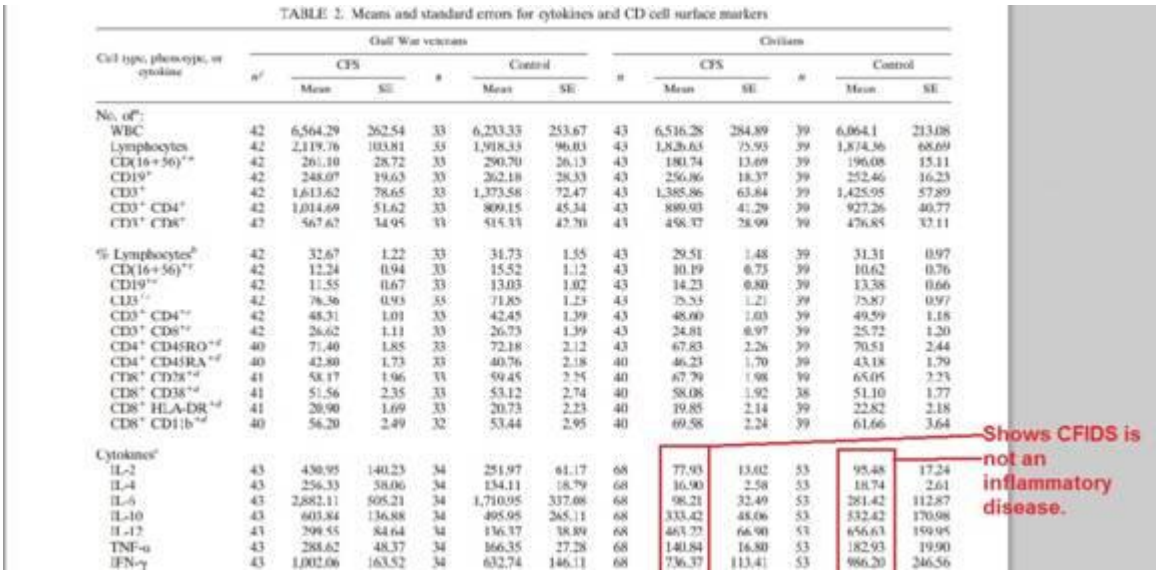


TABLE 2. Means and standard errors for cytokines and CD cell surface markers

Cell type, phenotype, or cytokine	Gulf War veterans						Civilians					
	CFS			Control			CFS			Control		
	n[a]	Mean	SE	n	Mean	SE	n	Mean	SE	n	Mean	SE
No. of[b]:												
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)$^{+}$	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	567.67	34.95	33	515.33	42.70	43	458.37	28.99	39	476.85	32.11
% Lymphocytes[b]	42	32.67	1.22	33	31.73	1.55	43	29.51	1.48	39	31.31	0.97
CD(16+56)$^{+}$	42	12.24	0.94	33	15.52	1.12	43	10.19	0.75	39	10.62	0.76
CD19^{+}	42	11.55	0.67	33	13.03	1.02	43	14.23	0.80	39	13.38	0.66
CD3^{+}	42	76.36	0.93	33	71.85	1.23	43	75.53	1.21	39	75.87	0.97
CD3^{+} CD4^{+}	42	48.31	1.01	33	42.45	1.39	43	48.60	1.03	39	49.59	1.18
CD3^{+} CD8^{+}	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	2.55	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[c]												
IL-2	43	450.95	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	463.77	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

http://www.ncbi.nlm.nih.gov/pubmed/9874656
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC95652/pdf/cd000006.pdf

---

VII. Expansion of Tolerance to viruses and LPS (matches the new NIH/wustl definition, post-sepsis syndrome):

2003; Medvedev and Cross-Tolerance TLR4 – to LPS, or plain old regular bacteria

Induction of in vitro reprogramming by Toll-like receptor (TLR)2 and TLR4 agonists in murine macrophages: effects of TLR "homotolerance" versus "heterotolerance" on NF-kappa B signaling pathway components.
http://www.ncbi.nlm.nih.gov/pubmed/12496438

2012; Harding and Cross Tolerance TLR7/9 agonists such as viruses

TLR2 signaling depletes IRAK1 and inhibits induction of type I IFN by TLR7/9.
"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/pmc/articles/PMC3262948/

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

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

FULL JOURNAL REPORT, snippet…

Reactivation of Multiple Viruses in Patients with Sepsis
"Sepsis is the host's non-resolving inflammatory response to infection that leads to organ dysfunction [1], [2]. A current controversial hypothesis postulates that if sepsis pursues a protracted course, it progresses from an initial primarily hyper-inflammatory phase to a predominantly immunosuppressive state [3]–[7]. 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]. …"
http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0098819

NEW, by the NIH (agreeing with the above description of ME/CFS and Chronic Lyme/Fibro):

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

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

VIII. CDC on chronic EBV causing fatigue via mitochondrial dysfunction (Suzanne Vernon)

Preliminary evidence of mitochondrial dysfunction associated with post-infective fatigue after acute infection with Epstein Barr virus.

"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

And the CDC admits in this same report, full text, that it has been known for 50 years that Chronic EBV is associated with Chronic Fatigue:

"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]."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1373655/?tool=pubmed

---

IX. Mycoplasma TLR2/1- agonist lipids affecting/inhibiting cell metabolism including transmembrane potential (This report is already seen above, re apoptosis, but the other important observation is in what mycoplasmal TLR2/1-agonists lipids do to intracellular organelles and membranes)

Mycoplasma fermentans inhibits tumor necrosis factor alpha-induced apoptosis in the human myelomonocytic U937 cell line.

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

http://www.ncbi.nlm.nih.gov/pubmed/15286682
http://www.nature.com/cdd/journal/v11/n11/full/4401482a.html

X. Tregs and Pam3Cys - "Our Best Frenemy"

Without sounding alarmed, we notice that some researchers think a mouse model of inhibiting the inhibitor Tregs in humans where the Tregs' suppressive activity can be over-run with Pam3Cys as sort of an immune boost. We certainly hope they know humans do not have the same TLR2s as mice.

2011; ***TLR1/TLR2 Agonist Induces Tumor Regression by Reciprocal Modulation of Effector and Regulatory T Cells***
http://www.jimmunol.org/content/186/4/1963.long

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. And of course, what happens when OspA causes the inhibition of apoptosis especially in EBV infected cells? Right. The reactivation of those herpesviruses, just as seen with fungally contaminated pediatric vaccines - the kids are getting the viruses instead of the protection.

TLR agonists: our best frenemy in cancer immunotherapy.
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-κB 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.

XI. Lyme, OspA and Epstein-Barr/similar herpes reactivation, Great Imitator, "L2 Diagnostics," NINDS' MS-Lyme Group (Martin & Marques), Duray on EBV, Halperin, Schoen and Luft on LYMErix causing the same disease as Chronic Neurologic Lyme

First of all let's remember that Lyme was the New Great Imitator after the first Great Imitator, Syphilis, since they both are known to cause cancer, MS, Lupus, RA, many of the leukemias, etc. And we know most of those diseases are actually chronic active Epstein-Barr and/or similar herpesviruses. So, later in the disease, the Chronic Fatigue-like, immunosuppressed, seronegative Lyme victims are dealing with a post-sepsis-like outcome which could be seen as a slow septic shock result. Regardless, once Lyme is advanced into the chronic neurologic stage, it's about more than spirochetes – it's about the reactivation of the herpes.

2010, Iksra, et al, Germany, ***Toll-like receptor agonists synergistically increase proliferation and activation of B cells by epstein-barr virus.***

"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

NIH, in the NYTimes once again, Lyme reactivates at least the herpes:

> ***When Lyme Disease Lasts and Lasts – Jane Brody***
> "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/

Why would Marques say that? She and Martin were in charge of the "MS-Lyme" Division of NINDS. We remind everyone, especially people with "MD" after their names, that "MS-Lyme" is not a disease of the knee or a Dearborn kind of Lyme. It is a secondary, AIDS-like outcome, where the secondaries or the opportunistics themselves have an HLA-linked hypersensitivity response. But obviously not in every case will the non-HLA-knees people have a subsequent non-HLA-knees-plus-yes-HLAs-for-the-opportunistics outcome.

1989, NIH, IDSA, National Cancer Institute and US Army's Paul Duray:

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

> 1992, Paul Duray, in Lyme Disease: Molecular and Immunologic Approaches. – 1992 book.
> "On occasion, these atypical-appearing large lymphocytes have been misinterpreted in biopsy by several laboratories as cells of a malignant lymphoma or leukemia. Bb antigens, then, may stimulate growth of immature lymphocytic subsets in some target organs, as well as in the cerebrospinal fluid (Szyfelbein and Ross 1988). Usual bacterial infections do not produce such lymphocytic infiltrates in tissue. These immunoblastoid cells in Bb infections at times resemble those found in Epstein-Barr virus infections. Does Bb reactivate latent virus infections in tissues? Do some tick inocula harbor simultaneous infectious agents (ixodid ticks can harbor Rickettsiae, Babesia microti, and Ehrlichia bacteria, in addition to Bb), producing multi-agent infections in some hosts? Further studies can clarify these issues by mans of tissue-based molecular probe analysis." -
>
> Paul Duray, NCI, NIH, Ft. Detrick, at the 1992 ALDF Cold Spring Harbor Conference
> http://www.amazon.com/Lyme-Disease-Immunologic-Approaches-Communications/dp/0879693770

MINDS' MS-Lyme's Martin and Marques, 2006, on how Lyme causes humoral immunosuppression, but with chronic inflammation in the brain, and on how fungal lipoproteins shed by these spirochetes might leave you with not even anti-flagellar antibodies:

> 2006; ***Borrelia burgdorferi Induces TLR1 and TLR2 in human microglia and peripheral blood monocytes but differentially regulates HLA-class II expression.***
> "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."
http://www.ncbi.nlm.nih.gov/pubmed/16783164

This report by 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):

2006; ***Borrelia burgdorferi lipoprotein-mediated TLR2 stimulation causes the down-regulation of TLR5 in human monocytes.***
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.
http://www.ncbi.nlm.nih.gov/pubmed/16479520

So, while some people say "you can't have a disease without inflammation or antibodies," here, clearly with fungal diseases like Lyme or Relapsing Fever, or Post-Sepsis Chronic Fatigue Syndrome, are diseases with none of the kinds of markers with which any "MD" with a low IQ and who likely graduated at the bottom of his class would be satisfied. Why "introduce complicating variables like the blood brain barrier," they've been known to say. (That is a quote from Steve Malawista, Yale, 2001, Rheumatology in the 21stCentury, if you can believe it. Maybe they meant the 15th century.)

2003, Hulinska***, Interaction of Borrelia burgdorferi sensu lato with Epstein-Barr virus in lymphoblastoid cells.***

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

That's reminiscent of CV Harding, Medvedev, and wustl & the NIH's "post sepsis syndrome" right?

Basically we should be calling spirochetes, myco-chetes since the MSMedia and MSMedicine seem to think spirochetes are not their own ancient phylum – shedders of fungal lipoproteins -, but plain old regular bacteria:

2003; ***Borrelia burgdorferi-induced tolerance as a model of persistence via immunosuppression.***

"If left untreated, infection with Borrelia burgdorferi sensu lato may lead to chronic Lyme borreliosis. It is still unknown how this pathogen manages to persist in the host in the presence of competent immune cells. It was recently reported that Borrelia suppresses the host's immune response, thus perhaps preventing the elimination of the pathogen (I. Diterich, L. Härter, D. Hassler, A. Wendel, and T. Hartung, Infect. Immun. 69:687-694, 2001). Here, we further characterize Borrelia-induced immunomodulation in order to develop a model of this anergy. We observed that the different Borrelia preparations that we tested, i.e., live, heat-inactivated, and sonicated Borrelia, could desensitize human blood monocytes, as shown by attenuated cytokine release upon restimulation with any of the different preparations. Next, we investigated whether these Borrelia-specific stimuli render monocytes tolerant, i.e. hyporesponsive, towards another Toll-like receptor 2 (TLR2) agonist, such as lipoteichoic acid from gram-positive bacteria, or towards the TLR4 agonist lipopolysaccharide. Cross-tolerance towards all tested stimuli was induced. Furthermore, using primary bone marrow cells from TLR2-deficient mice and from mice with a nonfunctional TLR4 (strain C3H/HeJ), we demonstrated that the TLR2 was required for tolerance induction by Borrelia, and using neutralizing antibodies, we identified interleukin-10 as the key mediator involved. Although peripheral blood mononuclear cells tolerized by Borrelia exhibited reduced TLR2 and TLR4 mRNA levels, the expression of the respective proteins on monocytes was not decreased, ruling out the possibility that tolerance to Borrelia is attributed to a reduced TLR2 expression. In summary, we characterized tolerance induced by B. burgdorferi, describing a model of desensitization which might mirror the immunosuppression recently attributed to the persistence of Borrelia in immunocompetent hosts.
http://www.ncbi.nlm.nih.gov/pubmed/12819085
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC162029/?tool=pubmed

From the Lyme and Lupus Clinic at Yale (now "L2 Diagnostics")– whoops, it's really about EBV:

2004***, Defective control of latent Epstein-Barr virus infection in systemic lupus erythematosus.***

"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."

http://www.ncbi.nlm.nih.gov/pubmed/14707107

XII. A Parallel Dynamic: Malaria and EBV and the production of Burkitts Lymphoma; and we expect to find high rates of Chronic Fatigue Syndrome in Africa and we do. Chronic Active EBV suppresses HLA expression, so we never associate pathologies such as ME/CFS with antibody studies; all of such reports have to be discarded from data summaries and analysis.

Burkitts Lymphoma caused by Malaria and EBV, Does the model of OspA and Borreliosis activating EBV fit?

Structure and dynamic behavior of Toll-like receptor 2 subfamily triggered by malarial glycosylphosphatidylinositols of Plasmodium falciparum.

"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.

2007; ***The prevalence of chronic fatigue syndrome in Nigeria.***
"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

Don't expect antibodies, you have to do proper DNA analysis such as proposed by IDSA with MassSpec DNA – say the next 2 reports:

2009***; Down-regulation of MHC class II expression through inhibition of CIITA transcription by lytic transactivator Zta during Epstein-Barr virus reactivation.***
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

2002; ***The lytic cycle of Epstein-Barr virus is associated with decreased expression of cell surface major histocompatibility complex class I and class II molecules.***

"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

XIII. IDSA's policy papers on rapid diagnosis of CNS diseases, 7X more accurate and complete diagnoses on all sorts of samples. Will never be deployed not because it is too costly to purchase Mass Spec instrumentation, but because no IDSA or CDC member can sell an office test kit. It's not about humans or health, after all, it's just about the money, the royalties.

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

"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

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

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.

1993 - 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

1999- 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.
1Department of Family Medicine, ***University of Connecticut*** Health Center, Farmington, Connecticut 06030-1406, USA.
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 OspAvaccine at 0, 1, and 2 months. Serum samples, obtained before vaccination and 1 month after the second and third doses, were analyzed for antiOspA 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.

22% of the adverse events were rated as “SEVERE,” and there was no real, long term follow up, just like the MMR vaccines. See the SASH criminal charge sheet on Yale’s and the CDC’s Vaccine and Dearborn Scam to see that they all knew LYMErix never prevented Lyme and caused a chronic Lyme-like systemic disease.

"Gulf War Illness victims are just plain cowards" - Simon Wessely

So says the British "psychiatrist," Simple Simon, 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

The following is a report Wessely wrote for the Pentagon:

> 2000- ***Role of vaccinations as risk factors for ill health in veterans of the Gulf war: cross sectional study***
> **Conclusion**
> "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/22039471

Yet, Wessely had this to say, later (see above, "Something Old, Something New…"):

> "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,"… -- [NOT SCIENTIFICALLY VALID-SASH].
>
> "Those tablets, the NAPS tablets, it's just not possible to study. Pesticides, we don't find evidence. [The antidotes to nerve agents given to veterans are a problem- SASH] 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** [long for "cowardice"-SASH] and what happened later."—[NOT SCIENTIFICALLY VALID-SASH]

Those nerve agent antidote tablets: they cause immunosuppression, as does DEET.
(Sounds familiar, though, right? Hypervaccination in the presence of immune suppressors like fungi?)

> 2004 -- ***Pyridostigmine bromide (PYR) alters immune function in B6C3F1 mice.***
> 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 20 mg/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."

Maybe not. Maybe some such studies were conducted by the CDC or DARPA and were not published.

DEET and Immunosuppression:

N,N,-diethyl-m-toluamide (DEET) suppresses humoral immunological function in B6C3F1 mice. http://www.ncbi.nlm.nih.gov/pubmed/19141786

DEET and Immunosuppression, especially combined with Nerve Agent Antidote:

Evaluation of immunotoxicity induced by single or concurrent exposure to N,N-diethyl-m-toluamide (DEET), pyridostigmine bromide (PYR), and JP-8 jet fuel.
http://www.ncbi.nlm.nih.gov/pubmed/12539864

More scientifically valid data: Garth Nicolson on Mycoplasma (the fungal contaminant for which they put Thimerosal in vaccines) in Gulf War Illness veterans:

Continuing research into Gulf War illness.
http://www.sciencemag.org/cgi/pmidlookup?view=long&pmid=11341275
http://www.actionlyme.org/GARTHNICOLSON.pdf

We've seen from the previous SASH criminal charge sheets for the Justice Department (sic), that mycoplasma causes fatigue via hypoxia via erythrocyte membrane osmotic changes and changes to mitochondria.

More scientifically valid data:

Changes in Immune Parameters Seen in Gulf War Veterans but Not in Civilians with Chronic Fatigue Syndrome
http://www.pubmedcentral.nih.gov/picrender.fcgi?artid=95652&blobtype=pdf

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.
> Brain Behav 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.
> Brain Behav 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.
> Psychosom Med. 2001 Nov-Dec;63(6):891-5.
>
> Immune responses and latent herpesvirus reactivation in spaceflight.
> Stowe RP, Mehta SK, Ferrando AA, Feeback DL, Pierson DL.
> Aviat Space Environ Med. 2001 Oct;72(10):884-91.
>
> Space analogue studies in Antarctica.
> Lugg D, Shepanek M.
> Acta Astronaut. 1999 Apr-Jun;44(7-12):693-9.
>
> Stress-induced reactivation of **Epstein-Barr** virus in **astronauts**.
> Stowe RP, Pierson DL, Feeback 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.
> Aviat Space Environ Med. 1999 Dec;70(12):1211-3.

So if you're an astronaut, you may have a real disease. If a soldier or some other commoner, no, it's the Salem Witch Trials for you:

> 1993-***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 seropositivemedical 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/pubhmed/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:

> 2011-- ***Fatigue in medical residents leads to reactivation of herpes virus latency.***
> "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

So, 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 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/

So, a person can have a real disease, but not a real disease, and no one knows how they do it. Sounds exactly like "magic" to a normal human. We could take this one step further: Send Justina and her kind to the CIA and see if she can do **remote viewing of Putin's submarines 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. And we have to listen to that crazy bullshit on the "news," not to mention the horrors of those who experience CPS-psychiatric Witch Trials, personally. Somatizing, cowardly soldiers … against the backdrop of known disease and known biomarkers. …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 of the vaccines or the vaccination of an immune suppressed person can result in the live viruses being reactivated. We know from the Cytokines study, the Gulf War Illness veterans seemed to have overall higher markers of immune activation, which conflicts with the other immunosuppression data, but we do know there are scientific realities to be had and acquired. Yet, the Pentagon hired Simon Wessely to not only trash the sick veterans, but people with 'Chronic Lyme" and 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;"
> "They have no awareness of their illness (this psychopathy is evident the whole world for to see yet they insist on being interviewed as 'experts');
> "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. In fact, no one has ever heard of mammals or biology or chemicals. Everything is, well, conjured.