

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

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

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

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

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

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

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

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

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

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

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

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

2017 Update:

This charge sheet is a revision of the 2014 Criminal Charges Sheet called the “DNA or Primers Shell Game.” Since that time we learned from Nicole Baumgarth, et al, at UC Davis, and Chiu/Aucott at UCSF, that spirochetes go right to the lymph nodes and destroy the B cell maturation or germinal centers,... and that around half of all tick bite sepsis victims have long term changes to their immune

systems (despite claiming that it doesn't happen) and don't recover:

[PLoS Pathog.](#) 2015 Jul 2;11(7):e1004976. doi: 10.1371/journal.ppat.1004976. eCollection 2015.

Suppression of Long-Lived Humoral Immunity Following Borrelia burgdorferi Infection.

Elsner RA1, Hastey CJ1, Olsen KJ2, Baumgarth N3.

“Lyme Disease caused by infection with *Borrelia burgdorferi* is an emerging infectious disease and already by far the most common vector-borne disease in the U.S. Similar to many other infections, infection with *B. burgdorferi* results in strong antibody response induction, which can be used clinically as a diagnostic measure of prior exposure. However, clinical studies have shown a sometimes-precipitous decline of such antibodies shortly following antibiotic treatment, revealing a potential deficit in the host's ability to induce and/or maintain long-term protective antibodies. This is further supported by reports of frequent repeat infections with *B. burgdorferi* in endemic areas. The mechanisms underlying such a lack of long-term humoral immunity, however, remain unknown. We show here that *B. burgdorferi* infected mice show a similar rapid disappearance of *Borrelia*-specific antibodies after infection and subsequent antibiotic treatment. This failure was associated with development of only short-lived germinal centers, micro-anatomical locations from which long-lived immunity originates. These showed structural abnormalities and failed to induce memory B cells and long-lived plasma cells for months after the infection, rendering the mice susceptible to reinfection with the same strain of *B. burgdorferi*. The inability to induce long-lived immune responses was not due to the particular nature of the immunogenic antigens of *B. burgdorferi*, as antibodies to both T-dependent and T-independent *Borrelia* antigens lacked longevity and B cell memory induction.

Furthermore, influenza immunization administered at the time of *Borrelia* infection also failed to induce robust antibody responses, dramatically reducing the protective antiviral capacity of the humoral response. Collectively, these studies show that *B. burgdorferi*-infection results in targeted and temporary immunosuppression of the host and bring new insight into the mechanisms underlying the failure to develop long-term immunity to this emerging disease threat.”

<https://www.ncbi.nlm.nih.gov/pubmed/26136236>

And

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

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

Bouquet J1, Soloski MJ2, Swei A3, Cheadle C2, Federman S1, Billaud JN4, Rebman AW2, Kabre B1, Halpert R4, Boorgula M2, Aucott JN5, Chiu CY6.

“Lyme disease is a tick-borne illness caused by the bacterium *Borrelia burgdorferi*, and approximately 10 to 20% of patients report persistent symptoms lasting months to years despite appropriate treatment with antibiotics. To gain insights into the molecular basis of acute Lyme disease and the ensuing development of post-treatment symptoms, we conducted a longitudinal transcriptome study of 29 Lyme disease patients (and 13 matched controls) enrolled at the time of diagnosis and followed for up to 6 months. The differential gene expression signature of Lyme disease following the acute phase of infection persisted for at least 3 weeks and had fewer than 44% differentially expressed genes (DEGs) in common with other infectious or noninfectious syndromes. Early Lyme disease prior to antibiotic therapy was characterized by marked upregulation of Toll-like receptor signaling but lack of activation of the inflammatory T-cell apoptotic and B-cell developmental pathways seen in other acute infectious syndromes. Six months after completion of therapy, Lyme disease patients were found to have 31 to 60% of their pathways in common with three different immune-mediated chronic diseases. No differential gene expression signature was observed between Lyme disease patients with resolved illness to those with persistent symptoms at 6 months post-treatment. The identification of a sustained

differential gene expression signature in Lyme disease suggests that a panel of selected human host-based biomarkers may address the need for sensitive clinical diagnostics during the "window period" of infection prior to the appearance of a detectable antibody response and may also inform the development of new therapeutic targets.

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

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

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

"Following the acute phase of infection, recent treatment trials among patients with EM have estimated that approximately 10 to 20% of patients treated for Lyme disease experience lingering symptoms that may progress to PTLDS, although the incidence can be as high at 50% (4). The pathogenetic mechanisms of PTLDS remain unknown, but autoantigens and/or central nervous system sensitization have been postulated to play a role (10, 33–35). In our study, the relatively large proportion of posttreatment

Lyme disease patients with persistent symptoms of fatigue, widespread musculoskeletal pain, and/or cognitive dysfunction (13 [46.4%] of 28) can be potentially accounted for by more stringent enrollment criteria at the time of presentation (**requiring the presence of EM and concurrent influenza-like symptoms rather than EM alone**). This may have resulted in the selection of patients with more severe disease and thus with an increased likelihood of persistent symptoms (36). Of note, according to the proposed formal case definition for PTLDS, which requires a functional decline in patients in addition to lingering symptoms, only 4 (14.3%) of our 28 patients met all of the criteria, within the range of the 10 to 20% frequency reported in the literature (4).

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

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

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

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

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

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

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

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

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

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

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

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

Gerlic M1, Horowitz J, Farkash S, Horowitz S.

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

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

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

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

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

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

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

review. How viral and bacterial infections that target cells of the monocytic lineage affect the expression of anti-apoptotic genes is important in understanding the pathological mechanisms that lead to manifested disease. The latest therapeutic approaches that target anti-apoptotic genes will also be discussed.

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

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

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

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

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

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

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

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

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

Gary Wormser saying – while LYMERix was still on the market -, that how sick you become, depends on how much OspA you got stuck with, either by ticks/spirochetes or syringe. You cant make this up:

“The magnitude of modulation [immunosuppression – KMD] was directly dependent on the quantity of OspA. OspA interferes with the response of lymphocytes to proliferative stimuli

including a blocking of cell cycle phase progression.” – Gary Wormser:

FEMS Immunol Med Microbiol. 2000 Jul;28(3):193-6.

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

Chiao JW¹, Villalon P, Schwartz I, Wormser GP.

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

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

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

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

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

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

Duray in IDSA’s journal:

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

“Clinical pathologic correlations of Lyme disease.

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

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

In the text of that article:

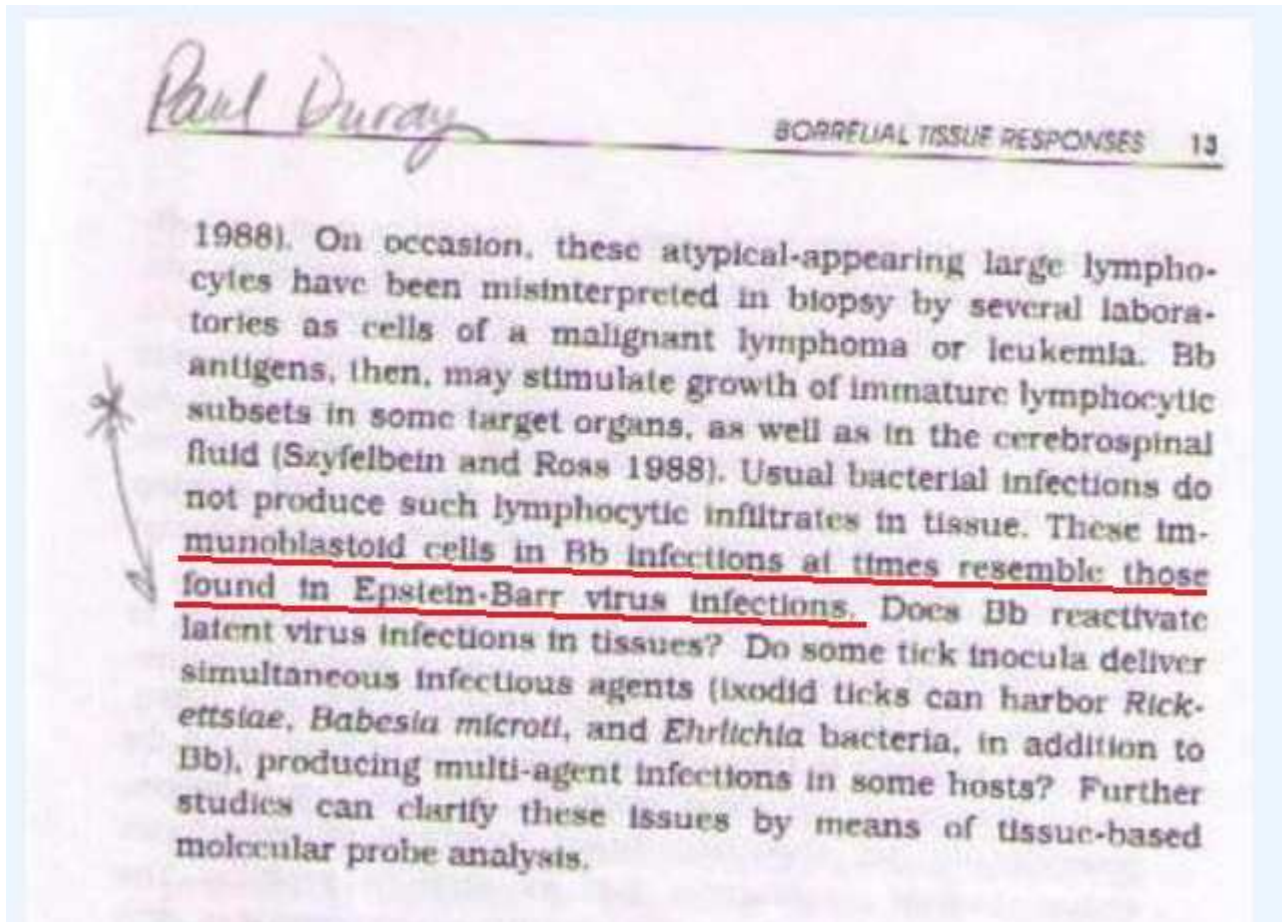
is usually no pleocytosis until the onset of stage II disease. Frank signs of meningeal irritation herald stage II illness, reflected by an increase in CSF lymphocytes and plasma cells and moderate increases in total protein in CSF [9, 16, 17]. Immature B cells can also be seen in the spinal fluid. These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

Duray in IDSA's journal, 1989.

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

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

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



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

Ann N Y Acad Sci. 1988;539:103-11.

Modulation of natural killer cell activity by Borrelia burgdorferi.

Golightly M1, Thomas J, Volkman D, Dattwyler R.

"Effect of B burgdorferi Culture on Normal PBL

"..when lymphocytes are cultured in the presence of growing Bb there is a marked inhibition ($p < .0005$) of NK activity on days 3, 5, and 7 when compared to lymphocytes cultured in BSKII media in the absence of spirochetes. This effect is not due to a selective depletion or toxicity to endogenous NK since viability studies and monoclonal antibodies demonstrate no significant changes after culture with the organism.

"The **inhibition** is directly attributable to the organism or its supernatants (data not shown)."

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

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

1922: Ancient history on how spirochetes target the lymph nodes

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

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

Pearce L, Brown WH.

A widespread dissemination of *Treponema pallidum* from a local focus of inoculation in the rabbit constantly occurs by way of the lymphatics. Spirochetes were regularly recovered from the satellite lymph nodes by animal inoculation after scrotal inoculation; they were present as early as 2 days, when no specific primary reaction was detected, and at later periods of from 5 to 61 days after inoculation. Other superficial nodes at remote sites such as the popliteals and with no syphilitic lesions in the drainage area have also been shown to harbor active organisms. Although spirochetes were found in relatively few of the lymph node emulsions, the orchitis resulting from their injection was of a rapidly progressive type with an incubation period but slightly longer than that produced by a testicular or skin nodule emulsion rich in spirochetes. It has further been shown that a syphilitic infection is sufficiently established in the rabbit body within 48 hours after scrotal inoculation so that the primary lesion is no longer essential for its maintenance. Active treponemata survive in the popliteal lymph nodes for long periods of time and have been regularly recovered from them in cases of true latency.

The lymph nodes, therefore, function as reservoirs of the organisms. The ability to recover the spirochetes from lymphoid tissue through successive generations is seen in the serial passage of lymph node emulsion to testicle during an 18 months period. The persistence of spirochetes in lymphoid tissue irrespective of the presence or absence of syphilitic lesions is a characteristic and fundamental feature of syphilis of the rabbit. The existence of infection, therefore, may be demonstrated at any time by the recovery of spirochetes from the popliteal lymph nodes by animal inoculation. This fact is of great practical importance in the therapy of the infection and may be profitably utilized in determining the ultimate effect of a therapeutic agent. These experiments demonstrate that the disease is not confined to the site of local inoculation but that lymphogenous dissemination of treponemata regularly takes place, and that during the course of this process organisms become localized in the lymph nodes and exist there indefinitely irrespective of the occurrence of manifestations of disease. The intimate relation of *Treponema pallidum* to lymphoid tissue is an essential concept of syphilis of the rabbit, and from this point of view, the infection is primarily one of lymphoid tissue.

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

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

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

Chesney AM, Kemp JE; Assistance of Allan K. Poole, M.D.

“Simultaneous transfers to the testes of normal rabbits of circulating blood, heart muscle, liver, brain, spleen and bone marrow (mixed), inoculated testicle, and popliteal lymph nodes from a series of untreated syphilitic rabbits, demonstrated the persistence of the original infection uniformly in the lymph nodes and less regularly in the liver, mixed spleen and bone marrow, and testis originally inoculated. In one instance the circulating blood was found to be infectious. Transfer of similar tissues from syphilitic rabbits treated with arsphenamine late in the course of the disease failed to disclose syphilitic infection of any of these tissues. In one animal, in which keratitis developed both before and

after treatment, the blood, internal organs, and lymph nodes were found to be non-infectious in spite of the fact that the cornea was shown to be the site of a syphilitic inflammation. Transfer of lymph nodes or internal organs of treated syphilitic rabbits is probably the best method of evaluating an antisymphilitic agent, but it must be supplemented by careful observation of treated animals over an appreciable interval of time following treatment. The results of this study support the idea that failure to reinoculate a treated syphilitic animal does not necessarily mean the existence of the first infection but might be interpreted as indicating the presence of an acquired resistance which persists in rabbits in which no trace of the first infection can be demonstrated.”

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

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

[Sex Transm Dis](#). 2014 Jul;41(7):440-6. doi: 10.1097/OLQ.0000000000000149.

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

[Marra CM](#)1, [Sahi SK](#), [Tantalo LC](#), [Ho EL](#), [Dunaway SB](#), [Jones T](#), [Hawn TR](#).

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

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

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

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

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

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

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

Reviews of Infectious Diseases

Volume 11 • Supplement 6 • September–October 1989

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

This is Yale:

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

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

Kang I1, Quan T, Nolasco H, Park SH, Hong MS, Crouch J, Pamer EG, Howe JG, Craft J.

“EBV infection is more common in patients with systemic lupus erythematosus (SLE) than in control subjects, suggesting that this virus plays an etiologic role in disease and/or that patients with lupus have impaired EBV-specific immune responses. In the current report we assessed immune responsiveness to EBV in patients with SLE and healthy controls, determining virus-specific T cell responses and EBV viral loads using whole blood recall assays, HLA-A2 tetramers, and real-time quantitative PCR. Patients with SLE had an approximately 40-fold increase in EBV viral loads compared with controls, a finding not explained by disease activity or immunosuppressive medications. The frequency of EBV-specific CD69+ CD4+ T cells producing IFN-gamma was higher in patients with SLE than in controls. By contrast, the frequency of EBV-specific CD69+ CD8+ T cells producing IFN-gamma in patients with SLE appeared lower than that in healthy controls, although this difference was not statistically significant. These findings suggest a role for CD4+ T cells in controlling, and a possible defect in CD8+ T cells in regulating, increased viral loads in lupus. These ideas were supported by correlations between viral loads and EBV-specific T cell responses in lupus patients. EBV viral loads were inversely correlated with the frequency of EBV-specific CD69+ CD4+ T cells producing IFN-gamma and were positively correlated with the frequencies of CD69+ CD8+ T cells producing IFN-gamma and with EBV-specific, HLA-A2 tetramer-positive CD8+ T cells. **These results demonstrate that patients with SLE have defective control of latent EBV infection that probably stems from altered T cell responses against EBV.**”

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

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

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

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

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

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

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

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

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

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

And here is the Cabal’s answer:

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

Division of the genus *Borrelia* into two genera (corresponding to Lyme disease and relapsing fever groups) reflects their genetic and phenotypic distinctiveness and will lead to a better understanding of these two groups of microbes (Margos et al. (2016) There is inadequate evidence to support the division of the genus *Borrelia*. Int. J. Syst. Evol. Microbiol. doi: 10.1099/ijsem.0.001717).

Barbour AG1, Adeolu M2, Gupta RS3.

“This rebuttal Letter responds to a Letter in the IJSEM by Margos et al. challenging division of the genus *Borrelia* into two genera. We discuss here point-by-point the issues raised by Margos et al. and show that much of their criticism is unfounded and in several cases based on misreading of the presented results. We summarize here the extensive evidence based on genomic, genetic and phenotypic properties showing that the members of the family *Borreliaceae* (containing mainly the genus *Borrelia*) comprises two distinct and cohesive groups of microbes, differing in diseases they cause and other phenotypes. Prior to the proposed division, *Borrelia* spp. causing Lyme disease (LD) were already functionally treated as a distinct group, referred to as “*B. burgdorferi sensu lato*” to distinguish them from the other cluster of *Borrelia* spp. which includes all known species causing

relapsing fever (RF). With the more explicit division of Borreliaceae species into two genus level groups, which are distinguishable from each other based on numerous unique genetic and molecular characteristics, the attention can now be focused on the biological significance of different molecular characteristics differentiating the two groups. The clear distinction of the LD and the RF groups of microbes based on numerous highly reliable markers, which are expected to be present even in uncharacterized members of these two groups, should aid in the improved diagnosis as well treatment of both these diseases, which is hindered by the conflation of a common name for agents causing two different types of diseases.”

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

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

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

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

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

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

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

These are the research-fraud “Guidelines,” the signers of which will be prosecuted among others

(CDC):

[Clin Infect Dis](#). 2006 Nov 1;43(9):1089-134. Epub 2006 Oct 2.

The clinical assessment, treatment, and prevention of lyme disease, human granulocytic anaplasmosis, and babesiosis: clinical practice guidelines by the Infectious Diseases Society of America.

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

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

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

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

- - - -

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

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

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

Primers are like a starting DNA or RNA sequence to look for a match in your sample. If you start with the wrong primer probes, you won't find what are looking for. When looking for spirochetes in humans, particularly when trying to claim "NO LYME," either in EM rashes in Missouri, or after "treatment," the Cabal either uses the wrong primers (they prefer to use OspA primers in particular, when trying to *not* find Lyme), or using inadequate primers such that only one or 2 species are probed for in humans, when there are probably a hundred *formal* different types of borrelia.

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

"Unmet diagnostic needs in infectious disease"

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

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

And

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

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

Lévêque N1, Legoff J, Mengelle C, Mercier-Delarue S, N'guyen Y, Renois F, Tissier F, Simon F, Izopet J, Andréoletti L.

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

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

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

These victim-patients are deprived of their humanity, as well as functionality. They're tossed aside, sick, demoralized, ostracized, and despised, yet they suffer a complex of several exhausting, neurologic diseases at the same time. While the CDC now claims that Lyme is 10 times underreported - meaning the new annual cases number around 300,000 rather than 30,000 because the falsified case definition misses 85% of the cases as shown in the other criminal charge sheets -, they of course never mention **it is only 15% reportable due to the fraud of Dearborn**. That is a lot of human cost and disability for which Uncle Sam will have to pay. Somehow a gang of low-lives was put in charge so they could potentially capitalize on this new vaccines and test kits racket, the emerging, global

pollution-related vector-borne-diseases. The ALDF's was a 50 year to roll-out plan for every new type of disease: rickettsia, babesia, borrelia, any new viruses they find, etc. Their model was to in each instance, invent a vaccine, and *then* the falsify the serological description of the disease. Whoever did not meet their Vaccine First disease definition was to be trashed. It's the same violence seen in any mob-related activity. **"You do it our way or we'll break your legs, we'll kill you or ruin your family, but you will be taken out. Silenced."**

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

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

Use Google Images to discover the basic structure of a spirochete; see the internal flagellar bundle that facilitates movement by expanding and contracting like a muscle; the organism borrows. There may be no help from "physicians" in this campaign, but that really doesn't even mean anything any more. The victims themselves have carried this campaign all these 20+ years and in the end, we'll probably welcome robot-doctor kiosks in the malls and at Walmart, perhaps with a nurse standing by to take blood and write the orders for the radioimaging. 'No need to overpay a middle-man for their incompetence. You'll at the end of this campaign be convinced no one needs a man with perverted, unscientific ideas about disease and medicine getting in the way of the machines. "Doctors" had their shot. They chose Kool-Aid and the age-old cliquish, clannish default position of looking down their noses and blaming their victims. "Chose," people, and that's a spiritually dangerous thing from a bunch of First Do No Harm, oath-takers. It was dangerous also, because BS is never not a boomeranger. We saw that loud and clear with the 911 stunt and then the subsequent quintuple financial and military superpower of Iran, Russia, Brazil, China, and South Africa (BRICS), not to mention ISIS and losing Syria and the Middle East oil wars.

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

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

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

officers like Alan Barbour or Yale staff patent borrelia species, they patent the specific flagellin that differentiates that particular bug from the other borrelia.

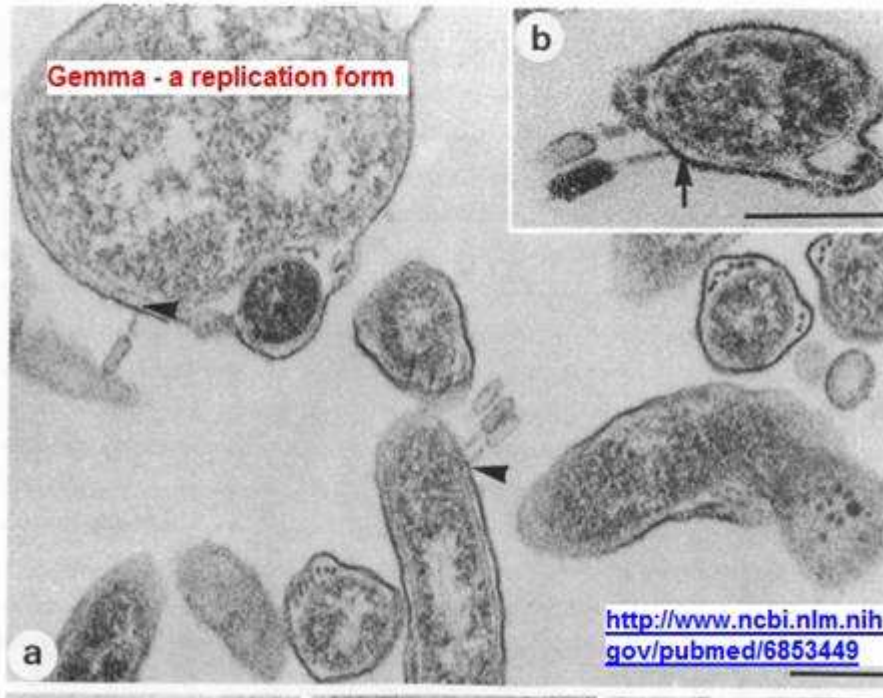
Plasmid content changes all the time *within* individual spirochetes and this is known as **antigenic variation**. CDC officer Alan Barbour is an expert on how this plasmid content changes and produces the well-known antigenic variation in spirochetes. Oscar Felsenfeld once said there was no point in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

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

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

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

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



<http://www.ncbi.nlm.nih.gov/pubmed/6853449>

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

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

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

“Researchers Finding Rewarding Careers As Software Entrepreneurs”

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

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

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

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

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

VMP-like sequences of pathogenic Borrelia

”2.1 Methods of Treatment

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

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

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

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

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

Catherine L. Lawson,^{1,*} Brian H. Yung,¹ Alan G. Barbour,² and Wolfram R. Zückert^{2,3,*}
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1482977/>

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

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

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

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

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

[Schoen RT1](#), [Deshefy-Longhi T](#), [Van-Hoecke C](#), [Buscarino C](#), [Fikrig E](#). or ([OspA_4.htm](#))

<https://www.ncbi.nlm.nih.gov/pubmed/12637121>

Korean Chemistry Journal on the Structure of OspA/Pam3Cys

Bull. Korean Chem. Soc. 1996, Vol. 17 Number 11

Characterization of Extremely Hydrophobic Immunostimulatory Lipoidal Peptides by Matrix-Assisted Laser Desorption ionization Mass Spectrometry

Jung-Suk Jang, Sung-Taek Lee, et al, Korea

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

Introduction

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

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

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

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

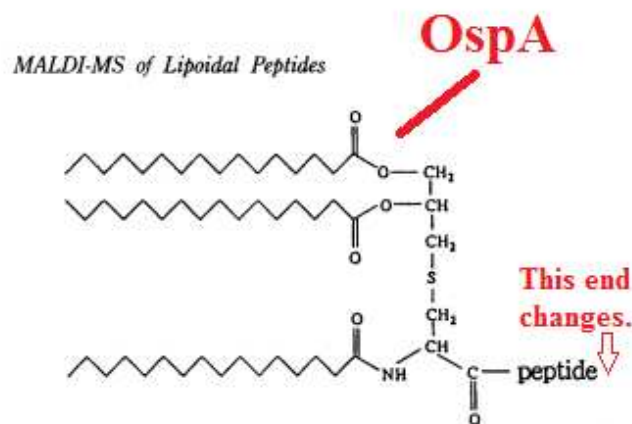


Figure 1. Structure of N-palmitoyl-S-[2,3-bis(palmitoyloxy)-propyl]-cysteinyl (Pam₃Cys) peptide, showing the modified cysteine residue with an N-palmitoyl ester and a dipalmitoylglycerol moiety connected via a thioether linkage. This amino acid is placed at the N-terminus of the synthetic peptide.

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

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

Results and Discussion

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

[Rappole JH](#)1, [Derrickson SR](#), [Hubálek Z](#).

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

”A very few birds, particularly seabirds, are carried by tropical storms across the Atlantic each summer from their normal environs on or near the coast of West Africa (39). A number of such storms form each summer and fall near the Cape Verde Islands off the western coast of Africa, travel across the Atlantic, and occasionally reach land along the East Coast of North America, depositing birds that were carried thousands of kilometers from their homes. Species known to have been infected by West Nile virus and whose habitat and distribution indicate that they might be affected by such displacement include the Gray Heron (*Ardea cinerea*), the Little Egret (*Egretta garzetta*), the Cattle Egret (*Bubulcus ibis*), the Black-headed Gull (*Larus ridibundus*), and the Yellow-legged Gull (*Larus cachinnans*) (Table 1). The same objections apply to this scenario for the introduction of the virus to the New World as for

normal migration, i.e., low numbers and the likelihood that a storm transported bird would be infected with the West African rather than the Middle Eastern form of the virus.”

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

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

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

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

[Fukunaga M1](#), [Okada K](#), [Nakao M](#), [Konishi T](#), [Sato Y](#).

anserina is genetically closest to burgdorferi in flagellin gene -KMD

of <i>Borrelia</i> strains															
<i>B. andersonii</i> 19857	<i>B. andersonii</i> 21038	<i>B. andersonii</i> 21123	<i>Borrelia</i> sp. strain Spain	<i>B. hispanica</i>	<i>B. duttoni</i> 406K	<i>B. crocidurae</i>	<i>B. crocidurae</i> ORI	<i>B. miyamotoi</i> HT31	<i>B. miyamotoi</i> FR64b	<i>B. lonestari</i> Texas	<i>B. hermsii</i> HS1	<i>B. hermsii</i>	<i>B. turicatae</i>	<i>B. parkeri</i>	<i>B. anserina</i>
96.2	96.2	96.3	82.2	80.1	82.2	82.3	81.0	81.2	81.2	80.8	83.1	83.4	83.5	83.0	84.2
95.8	95.8	95.9	82.1	80.9	82.0	82.1	80.8	81.1	81.1	80.5	83.0	83.2	83.4	82.8	84.1
96.1	96.1	96.2	82.3	80.2	82.2	82.3	81.0	81.3	81.3	80.8	83.2	83.5	83.6	83.0	84.3
93.4	93.3	93.5	82.6	79.8	82.2	82.3	81.0	81.2	81.2	80.3	83.4	83.6	83.6	83.0	84.9
93.2	93.0	93.3	82.1	79.3	81.7	81.8	80.5	80.9	80.9	80.2	83.0	83.2	83.5	82.9	85.2
93.2	93.0	93.3	82.2	79.6	81.5	81.6	80.3	80.6	80.6	80.3	82.3	82.5	83.4	82.8	84.4
93.4	93.0	93.3	82.7	80.2	82.1	82.2	80.9	81.2	81.2	81.1	83.1	83.4	84.0	83.6	85.5
93.4	93.0	93.3	82.7	80.2	82.1	82.2	80.9	81.3	81.3	81.1	83.1	83.4	83.7	83.2	85.6
93.4	93.0	93.3	82.4	79.8	81.8	81.9	80.6	81.0	81.0	80.7	83.0	83.2	83.6	83.1	85.5
94.4	94.3	94.5	83.1	80.8	82.9	83.0	81.7	81.6	81.6	81.3	83.7	83.9	84.2	83.8	84.9
94.4	94.3	94.5	83.1	80.8	82.9	83.0	81.7	81.6	81.6	81.3	83.7	83.9	84.2	83.8	84.9
92.2	92.0	92.2	82.0	80.5	82.5	82.2	81.2	80.7	80.7	80.8	82.5	82.7	82.7	82.0	84.0

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

22 FUKUNAGA ET AL.

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

1995 -- Next, New York Medical College (NYMC) and Marconi at Medical College of Virginia at Virginia Commonwealth University, Richmond, VA, say *anserina* is an “out-group” when

comparing *burgdorferi* or the Lyme group from other borrelia. It is not some random out-group. It is the origin of *burgdorferi* as you will see when we talk more about 1) Plum Island as the original outbreak area, where 2) UPenn says this vector-pathogen match-up was evolutionarily unlikely, and 3) where they just happen to do that kind of African-diseases-with-North-American Vectors kind of research on Plum, not to mention, 4) all the CDC's lies and attempts to have us believe "Lyme disease" is not even a spirochetal disease, but autoimmune arthritis (Dearborn).

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

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

and genomic group 21038 (*Borrelia andersonii* sp. nov.) isolates.

Marconi RT1, Liveris D, Schwartz I.

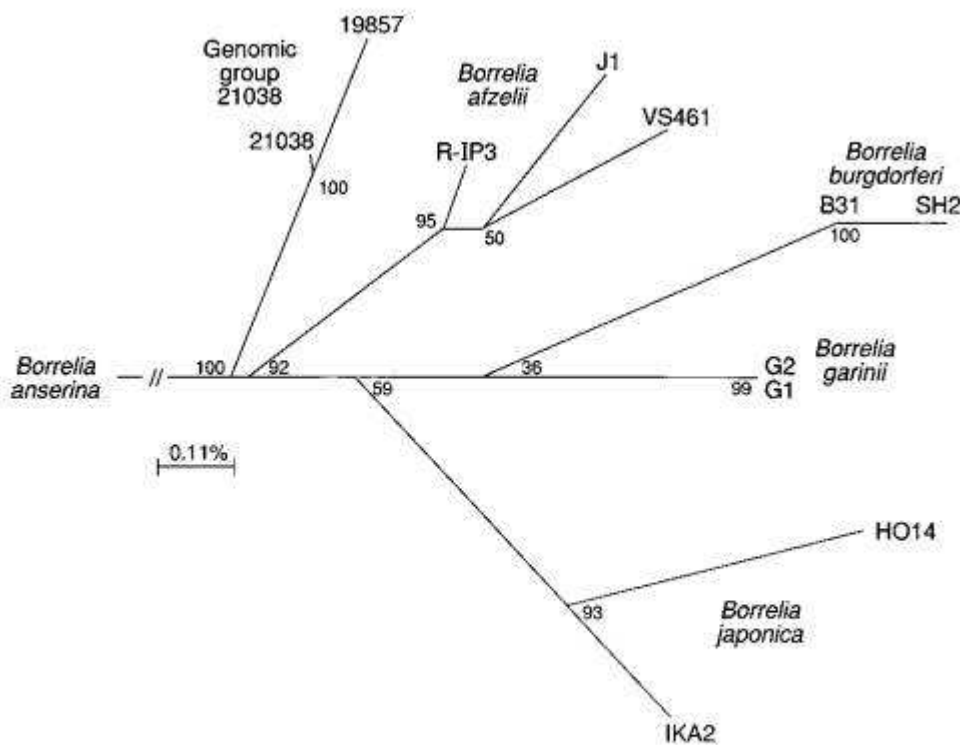


FIG. 7. Phylogenetic tree of 16S rRNA derived from LDS isolates. The phylogenetic tree was constructed as described in the text. Numbers at the branch nodes indicate the results of bootstrap analysis. The 16S rRNA sequence from *Borrelia anserina* served as an outgroup.

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

UPenn on Lyme spirochetes being evolutionarily unlikely:

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

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

Humphrey PT1, Caporale DA, Brisson D.

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

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

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

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

Evolution of a focus of Lyme disease.

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

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

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

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

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

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

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

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

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

<http://www.ncbi.nlm.nih.gov/pubmed/9499019>

Note that the end-point, here, slaughtering your infected livestock, is a Plum Island-, or as we call it, Von Traub Island-, goal. We should mention there is at least one “Plum Island” strain of *Mycoplasma*:

J Hyg (Lond). 1983 Jun;90(3):441-9.

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

Smith GR, Oliphant JC.

“Much evidence of immunogenic heterogeneity among the LC strains of **Mycoplasma mycoides** ssp. *mycoides* emerged from cross-immunization and -hyper-immunization experiments in mice in which three LC strains (Vom/**Plum** Island, 74/2488, and Mankefår 2833) were used for challenge purposes. All heterologous LC-strain vaccines cross-immunized against the three challenge strains, but protection was usually only 'partial', i.e. significantly less than that given by homologous vaccine. Cross-hyperimmunization with all heterologous LC but not SC strains produced protection against challenge with Vom/**Plum** Island that was virtually 'complete', i.e. similar to that produced by homologous vaccine. Challenge with 74/2488 gave generally similar results; but against Mankefår 2833 six heterologous LC vaccines gave complete protection and six did not. Vaccines prepared from the Smith (1423) strain of *M. mycoides* ssp. *capri* gave some protection against Vom/Plum Island but none against 74/2488 or Mankefår 2833. The cross-immunizing ability of three further *M. mycoides* ssp. *capri* strains appeared to resemble that of Smith (1423). In a cross-hyperimmunization experiment, vaccines prepared from SC strains of *M. mycoides* ssp. *mycoides* varied greatly in their ability to protect against challenge with strains 74/2488 and Mankefår 2833”

<http://www.ncbi.nlm.nih.gov/pubmed/6190898>

"Mycoplasma mycoides mycoides" = “Fungal-plasma fungal, fungal,” nice. Triple fungal mycoplasma on Plum Island. That’s adorable. ☺

So, challenging various vectors (bugs) with diseases from Africa is what Plum Island does. Naturally, an odd one could have escaped, one way or another – an African bird borreliosis -, genetically unlikely, and Plum Island was the original outbreak area. That’s all the real data we’ll ever have because we’ll never have the lab notebooks from Plum Island.

If we were prosecuting a murder trial, this all would probably fly circumstantial evidence case as “beyond a reasonable doubt,” especially considering all the other lies about Lyme disease, like the hurricane fairy tale, IDSA’s “Guidelines on the Diagnosis and Treatment of Lyme disease,” the Dearborn “case definition,” and most of all the very idea that everyone should get a vaccine against an imaginary disease. No one has ever met or heard from a person who can come up with a sound reason there would be a vaccine against a disease that does not exist and needs no treatment.

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

J Bacteriol. 1951 Aug;62(2):215-9.

Relapse phenomena in rats infected with single spirochetes (Borrelia recurrentis var. turicatae).

SCHUHARDT VT, WILKERSON M.

“Antigenic variation by the spirochete is generally believed to be responsible for the relapse phenomena in spirochetal relapsing fever. Schuhardt (1942) has reviewed the literature prior to 1942 on this subject, and little if any evidence has been presented subsequently to alter or extend this concept. Among the unanswered questions in spirochetal relapse phenomena are: (a) the antigenic variation capacity of a single spirochete, and (b) the capacity of an antigenic variety to recur in a series of relapses in a given animal. Although Cunningham, Theodore, and Fraser (1934) believe that antigenic varieties do not recur, other workers are not convinced that this possibility has been ruled out. Consequently we undertook a study of single spirochete infections in white rats in an effort to answer these two and possibly other questions related to the relapse phenomenon in spirochetal relapsing fever.”

<https://www.ncbi.nlm.nih.gov/pubmed/14861181>

<http://jlb.asm.org/cgi/reprint/62/2/215?view=long&pmid=14861181>

Oscar Felsenfeld, CDC officer Alan Barbour, Russell Johnson (ALDF member), and Diego Cadavid talking about/referencing this Single Spirochete Phenomena:

http://www.ncbi.nlm.nih.gov/sites/entrez?db=pubmed&cmd=link&linkname=pubmed_pubmed_citedin&uid=14861181

Oral spirochetes infecting Alzheimer's brains and traveling along inside nerves (this is not the only report that says this, you'll find it in syphilis reports too; from the older published data and from the Cabal on the incurability of relapsing fever; an independent study on spirochetes in the brain from dentists and they say:

Oral Microbiol Immunol. 2002 Apr;17(2):113-8.

Molecular and immunological evidence of oral *Treponema* in the human brain and their association with Alzheimer's disease.

Riviere GR1, Riviere KH, Smith KS.

“The purpose of this investigation was to use molecular and immunological techniques to determine whether oral *Treponema* infected the human brain. Pieces of frontal lobe cortex from 34 subjects were analyzed with species-specific PCR and monoclonal antibodies. PCR detected *Treponema* in 14/16 Alzheimer's disease (AD) and 4/18 non-AD donors ($P < 0.001$), and AD specimens had more *Treponema* species than controls ($P < 0.001$). PCR also detected *Treponema* in trigeminal ganglia from three AD and two control donors. Cortex from 15/16 AD subjects and 6/18 controls contained *Treponema pectinovorum* and/or *Treponema socranskii* species-specific antigens ($P < 0.01$). *T. pectinovorum* and/or *T. socranskii* antigens were also found in trigeminal ganglia and pons from four embalmed cadavers, and 2/4 cadavers also had *Treponema* in the hippocampus. **These findings suggest that oral *Treponema* may infect the brain via branches of the trigeminal nerve.**”

<http://www.ncbi.nlm.nih.gov/pubmed/11929559>

1975 -- Jay Sanford, Uniformed Services University School of Medicine, Bethesda, Maryland, page 391, in the book, The Biology of Parasitic Spirochetes, 1976 edited by ALDF.com's Russell C. Johnson

”The Biology of parasitic spirochetes” / edited by Russell C. Johnson

Bib ID 2160125, New York ; Academic Press, 1976, ISBN 012387050X

"The ability of the borrelia, especially tick-borne strains to persist in the brain and in the eye after

treatment with arsenic or with penicillin or **even after apparent cure is well known** (1). The persistence of treponemes after treatment of syphilis is a major area which currently requires additional study (3,5,10,11).”

http://www.actionlyme.org/Biology_of_Parasitic_Spirochetes1976.htm

See more at: The History of Relapsing Fever: <http://www.actionlyme.org/RICOCHRON.htm>

There was never any issue with persistence or neurotropism or even lymph node tropism of *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, naming **ALL** *Borrelia* (see the Taxonomy database), not *just burgdorferi*.

Says CDC officer Alan Barbour in 1986:

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

Biology of Borrelia species.

Barbour AG, Hayes SF.

”When relapsing fever *borreliae* are no longer detectable in the blood, they may still be found in organs (120). Although *borreliae* can usually be recovered from such organs as the spleen, liver, kidneys, and eyes of infected animals (37, 120), the organ usually with the most persistent infections is the brain. Humans with relapsing fever have had *borreliae* recovered from the cerebrospinal fluid (72). *Borrelia* can be recovered from the brains of animals that are immune to challenge with that strain (119, 127, 148, 178). Detection or isolation of *borreliae* from brains of animals that had been infected several months and up to 3 years previously has been reported (12, 181, 197, 223). Before the advent of modern ultracold freezers, strains were kept in the brains of rodents and passed once or twice a year (92).”

<https://www.ncbi.nlm.nih.gov/pubmed/3540570>

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

Rodent brains used to be the storage media says Barbour, above. And *borrelia* are often absent from blood even with valid DNA methods like flagellin DNA or species specific 16S genes, because, as Alan Barbour says, they are in the organs, especially the brain and lymph nodes. Obviously a culture method from blood can’t be used for the same reason – they’re not always in the blood.

CDC officer Alan Barbour also says in the same report:

”A strain of *B. duttonii* that had been passed many times in mice was found to have lost virulence for humans (212). When using *borreliae* for pyrotherapy of neurosyphilis, the authors of this report recommended that no more than 30 to 40 passages in mice be made before inoculation of the strain back into humans (212).”

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

It is fair to say this CDC officer, Alan Barbour, was not too confident in antibiotics if he suggested giving people a fever from a weakened (high passage) relapsing fever organism as a way to cure syphilis. Barbour shows us above that he is aware that one should not use high-passage strains – which Steere did to develop the Dearborn method -, since the point of high passages is to weaken the strain and have the organisms drop plasmids. We assume the reason Steere falsified the testing for the CDC’s Dearborn case definition panel (leaving *OspA* and *B* out; *OspA* and *B* are encoded on the same plasmid, so you can’t drop one without dropping the other), using high-passage strains, was that he and

his co-conspirators intended to **develop a test for Lyme that would be okay to use in a population “where the vaccination status was unknown.”** The Schoen-Persing-Steere RICO method patent, US 6,045,804, uses a strain of *Borrelia* that had dropped the OspA-B plasmid. It’s possible to do that with repeat passages; you can get the bugs to drop plasmids and “virulence determinants” in this way.

We will see from this report CDC officer Allen Steere played the shell game while he falsified the case definition strains, identifying *borrelia* using the correct primers when he developed that bogus Dearborn method in 1992. Later Steere used mainly the wrong DNA (OspA and in one instance 1 primer probe of 16S RNA) to assess human treatment results. Despite using the wrong primers, Steere found DNA persisted in 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:

Neurology. 1990 Oct;40(10):1535-40.

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

Pachner AR1, Itano A.

“To learn more about the neurologic involvement in Lyme disease, we inoculated inbred mice with the causative agent of Lyme disease, *Borrelia burgdorferi*. We cultured brains and other organs, and measured anti-B burgdorferi antibody titers. We further studied a brain isolate for its plasmid DNA content and its response in vitro to immune sera and antibiotics. One strain of B burgdorferi, N40, was consistently infective for mice, and resulted in chronic infection of the bladder and spleen. SJL mice developed fewer culture-positive organs and had lower antibody titers than Balb/c and C57Bl/6 mice. Organism was cultured from the brain early in the course of infection, and this isolate, named N40Br, was further studied in vitro. **The plasmid content of N40Br was different from that of the infecting strain, implying either a highly selective process during infection or DNA rearrangement in the organism in vivo.** N40Br was very sensitive to antibiotics, but only after prolonged incubation. Immune sera from both mice and humans infected with B burgdorferi were unable to completely kill the organism by complement-mediated cytotoxicity. These data demonstrate that B burgdorferi infects the brain of experimental animals, and is resistant to immune sera in vitro but sensitive to prolonged treatment with antibiotics.”

<http://www.ncbi.nlm.nih.gov/pubmed/2215944>

After a time the plasmid content was different from the original strain, says Pachner. That would be because Lyme is just another relapsing fever borreliosis. Antibiotics merely cause the organisms to convert into a spheroplast form, but that is a topic for another DOJ criminal charge sheet. The cyst or spheroplast form is not an “end-stage,” as some claim. It is a replication form. Previously we said, “The thing to do about Lyme is to catch it early before the shed Osp or fungal antigen-related immunosuppression invites (cross-tolerance) other pathogens or reactivates old, dormant ones like the herpes viruses, do most of the damage.” But apparently this damage is done right away, according to Baumgarth and Chiu.

Where else to we find these fungal, OspA-like antigens?

J Biol Chem. 1999 Nov 19;274(47):33419-25.

Toll-like receptor 2 functions as a pattern recognition receptor for diverse bacterial products.

[Lien E1](#), [Sellati TJ](#), [Yoshimura A](#), [Flo TH](#), [Rawadi G](#), [Finberg RW](#), [Carroll JD](#), [Espevik T](#), [Ingalls RR](#), [Radolf JD](#), [Golenbock DT](#).

"Toll-like receptors (TLRs) 2 and 4 are signal transducers for lipopolysaccharide, the major proinflammatory constituent in the outer membrane of Gram-negative bacteria. We observed that membrane lipoproteins/lipopeptides from *Borrelia burgdorferi*, *Treponema pallidum*, and *Mycoplasma fermentans* activated cells heterologously expressing TLR2 but not those expressing TLR1 or TLR4. These TLR2-expressing cells were also stimulated by living motile *B. burgdorferi*, suggesting that TLR2 recognition of lipoproteins is relevant to natural *Borrelia* infection. Importantly, a TLR2 antibody inhibited bacterial lipoprotein/lipopeptide-induced tumor necrosis factor release from human peripheral blood mononuclear cells, and TLR2-null Chinese hamster macrophages were insensitive to lipoprotein/lipopeptide challenge. The data suggest a role for the native protein in cellular activation by these ligands. In addition, TLR2-dependent responses were seen using whole *Mycobacterium avium* and *Staphylococcus aureus*, demonstrating that this receptor can function as a signal transducer for a wide spectrum of bacterial products. We conclude that diverse pathogens activate cells through TLR2 and propose that this molecule is a central pattern recognition receptor in host immune responses to microbial invasion."

<http://www.ncbi.nlm.nih.gov/pubmed/10559223> <http://www.jbc.org/content/274/47/33419.long>

What you should do when reading this general science material, particularly like the 1999 one above and see related and see cited by on PubMed.

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

[J Immunol](#). 2001 Jul 15;167(2):910-8.

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

[Noss EH1](#), [Pai RK](#), [Sellati TJ](#), [Radolf JD](#), [Belisle J](#), [Golenbock DT](#), [Boom WH](#), [Harding CV](#).

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

<http://www.ncbi.nlm.nih.gov/pubmed/11441098>

Spirochetes create multiple variants and all the individual spirochetes do their own thing, varying their

surface antigens on their own, shedding these fungal antigens in a process called blebbing, ruining a person's immune system. And a ruined immune system *is the DAMAGE* and *is the ILLNESS* and *is the specific goal of a bioweapon*:

Types of Biological Agents

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

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

⁶¹ Ibid.

⁶² Ibid, p. 9, section 1502.

AND

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

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

"Methods of using antipersonnel agents undoubtedly vary so that no uniform pattern of employment or operation is evident [make sure it does not produce antibodies, so assess the HLAs in the population you intend to abuse like the defecting Russian scientists at NYMC have been doing, is the short version- KMD]. It is likely that agents will be used in combinations so that disease symptoms will confuse diagnosis and interfere with proper treatment. It is also probable that biological agents would be used in heavy concentrations to insure a high percentage of infection [or just use the OspA vaccine-KMD] in the target area. The use of such concentrations [or the multiple infections it causes, due to the immunosuppression like HIV, Lyme, or LYMERix as acquired immune deficiencies - KMD] could

result in the breakdown of individual immunity because the large number of micro-organisms entering the body could overwhelm the natural body defenses [or just infect or inject people with an immune suppressor like OspA from a tick or a syringe, and the reverse will happen: people will acquire multiple infections because their immunity is trashed by fungal OspA- KMD].

Do you see the disease now? It's fungal (shed borrelial antigens are TLR2/1-agonists or fungal); it is about "overwhelming the immune system" (which is another way to say, "post-sepsis syndrome"); it is about not producing identifiable antibodies; your bioweapon should be like a Trojan Horse, setting off other latent infections; your immune system is now turned off ("overwhelmed" means "turned off"); you don't have "biofilms" at least of borrelia; Lyme was the "perfect stealth disabler." See more in the Occam's Razor chapter.

V. SIDESTEPPING - Alert on "Biofilms"

Use "Borrelia Staining" or "Borrelia Silver Staining" as search terms in PubMed to discover that Borrelia *in vivo* do not cluster at all, much less under a "biofilm." Here is one. Look closely for the "clustered spirochetes hiding under a biofilm" (there is no such thing):

[J Med Microbiol](#). 1987 May;23(3):261-7.

Demonstration of spirochaetes in patients with Lyme disease with a modified silver stain.

[De Koning J](#), [Bosma RB](#), [Hoogkamp-Korstanje JA](#).

<https://www.ncbi.nlm.nih.gov/pubmed/2438410> <http://jmm.sgmjournals.org/content/23/3/261.long>

Here is another one by Paul Duray [same guy who revealed that congenital Lyme brain damage kills babies and who revealed that Lyme- and LYMERix- diseases cause a leukemia-like illness and that the cells in the CSF of Lyme patients "look like Epstein-Barr transformed (mutated, pre-cancerous) cells]:

[J Clin Microbiol](#). 1991 Apr;29(4):764-72.

Morphology of Borrelia burgdorferi: structural patterns of cultured borreliae in relation to staining methods.

[Aberer E1](#), [Duray PH](#).

"The microscopic recognition of Borrelia burgdorferi in biologic fluids and tissues is difficult and challenging because of low numbers of organisms occurring as single isolated spirochetes, **the apparent lack of colony formation in tissues**, and differing lengths and structural morphologies."

<http://www.ncbi.nlm.nih.gov/pubmed/1716264>

Additionally, some biofilms are covered in TLR2/1 agonists so the body does not even see them at all any more, if they are there in this post-sepsis disease called Chronic Lyme, with the multiple reactivated herpes viruses, etc., and the expansion of tolerance to other toll-like-receptor-managed antigen types. The biofilms could be covering other organisms, but spirochetes are all independent operators and the illness and the damage is mainly from the secondary, "Post Sepsis Syndrome," infections.

REVIEW: Biofilms covering spirochetes are NOT responsible for the persistent symptoms in Chronic Lyme Disease. Spirochetes, while permanent, and while they have been shown to be draped in lymphocyte membrane, or have a "mucopeptide layer" (or while were always known to be covered in a

slime layer), are *not* the main cause of the disease or the reason antibiotics fail.

Yes, spirochetal diseases are incurable. No, the disease is not about spirochetes, since they shed fungal antigens and ruin the immune system, inviting in other opportunistics or reactivating old ones. We learned this from LYMERix disease where the vaccine gave people the same systemic disease we know of as Chronic Lyme or Chronic Fatigue Syndrome. It is a NO-IMMUNE disease, post-sepsis.

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

Says Yale:

[Infect Immun.](#) 1991 Oct;59(10):3531-5.

Molecular characterization of the humoral response to the 41-kilodalton flagellar antigen of *Borrelia burgdorferi*, the Lyme disease agent.

[Berland R I](#), [Fikrig E](#), [Rahn D](#), [Hardin J](#), [Flavell R A](#).

"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 Cabal misuses that word all the time.

FDA Rules on the Validation of an Analytical Method:

<http://www.fda.gov/downloads/drugs/guidancecomplianceregulatoryinformation/guidances/ucm368107.pdf>

Specificity (only detects one thing)

Accuracy (Should detect 100% of the instances where the analyte is present, and the concentration should be close to 100% of that known to be spiked in, and never should detect "none" as is the case with Lyme Western Blotting and the Lyme ELISA, especially)

Limit of Detection (means "What is the lowest concentration of the analyte in question does your method detect?")

Precision (system has integrity in performance)

Ruggedness (anyone can run the test with their own equipment and get the same results)

Linearity (concentration range of analyte for which the test is valid in and out of matrix or "inert ingredients")

Your test should primarily detect *all* the cases in question, - or be 100% ACCURATE - and that means, in the case of Lyme, the only analyte for which we can test is flagellin or anti-flagellar antibodies. Anti-flagellar antibodies can be found in probably 95% of Lyme cases. So, Yale went ahead and made that Specific (also described in US patent 5,618,533) in 1991, as shown previously, above.

Infect Immun. 1991 Oct;59(10):3531-5.

Molecular characterization of the humoral response to the 41-kilodalton flagellar antigen of *Borrelia burgdorferi*, the Lyme disease agent.

Berland R I, Fikrig E, Rahn D, Hardin J, Flavell RA.

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

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC258917/pdf/iai00046-0199.pdf>

For the other *Borrelia* in North America and Europe, at least, such a recombinant-specific-multiple-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 point to using a blood DNA method. Flagellin is the only reliable antibody and it can be made specific. There is no "end point" to treatment, since late Lyme is more about the other opportunistics. But early Lyme, all agree that the flagellar antibody test is the only test that captures the majority of cases and meets the FDA criteria for "ACCURACY."

Lyme and LYMERix cause immunosuppression and an AIDS-like disease or an acquired immune deficiency, or as the NIH describes it, post-sepsis with the all kinds of still-active herpes and other infections. It should be said that Lyme and LYMERix diseases are far worse than just spirochetes. Apparently that has always been the case. **The Great Imitator, Syphilis was probably really the Great Detonator of the latent herpes and other infections.** Syphilis was probably the original AIDS, via OspA-like or fungal-antigen-like immunosuppression and the reactivation of mostly Epstein-Barr.

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

CDC and IDSA claimed the cyst form was not viable, and that *Borrelia* DNA-positive human samples were "just dead DNA" (never happens, the body cleans up such debris). Yet here is the CDC in 1964 explaining how to dessicate and weaponize your *Borrelia* (freeze-drying – and good for at least a year, they say):

J Bacteriol. 1964 Sep;88:811.

RECOVERY OF TREPONEMA AND BORRELLIA AFTER LYOPHILIZATION.

HANSON AW, CANNEFAX GR.

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

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

Next we see the CDC is throwing out the blood cells (throwing out whole cells of any kind), including red blood cells and immune cells or white cells to which mycoplasma adhere, while alleging to look for mycoplasma in Chronic Fatigue Syndrome. Mycoplasma or epERTHROzoa are called epERYTHROzoa because they attach to red blood cells. Such epERYTHROzoa are famous for changing the erythrocyte membrane potential and the ability of oxygen to cross the red blood cell membrane, causing tremendous fatigue even in animals.

CDC did this to allegedly show Mycoplasma were not involved in Chronic Fatigue Syndrome:

J Med Microbiol. 2003 Nov;52(Pt 11):1027-8.

Absence of Mycoplasma species DNA in chronic fatigue syndrome.

Vernon SD, Shukla SK, Reeves WC.

"Plasma, the liquid portion of peripheral blood that is devoid of cells, is known to contain remnants of numerous physiological and disease processes. We used plasma DNA to detect and characterize bacterial 16S rDNA sequences in a group of individuals with CFS and a group of non-fatigued controls (Vernon et al., 2002). Whilst a variety of bacterial sequences were detected in both fatigued and non-fatigued groups, no Mycoplasma sp. 16S rDNA sequences were found."

<https://www.ncbi.nlm.nih.gov/pubmed/14532349>

<http://jmm.sgmjournals.org/cgi/pmidlookup?view=long&pmid=14532349>

That is important. The CDC does not want anyone to know fungal antigens and/or fungal antigen tolerance cause(s) extreme fatigue. They must be important bioweapons, a problem with the pediatric vaccines causing Autism, or both.

Next up the specific DNA Shell Game played by members of the Cabal. You will see it is almost entirely CDC officers committing this fraud. The data you have seen so far reveals 1) how to test for all Borrelioses, 2) how we got this particularly evolutionarily unlikely bird borreliosis in New England "on hurricanes?" and 3) catching the CDC staff committing research fraud in other arenas.

IX. The CDC Cabal Play the DNA and RNA Shell Game (we learned what is proper detection DNA: flagellin, and other non-variable specific RNA)

Alan Barbour playing the DNA/RNA shell game.

You will want to look at **The Patents** Criminal Charge sheet for this multi-Crymes Disease to see

that CDC officer and former head of the NIH's Rocky Mountain Bioweapons Montana Lab (you're familiar with Montana, the place where there are tons of relapsing fever borrelia but no "Lyme?"), **Alan Barbour**, reported that, basically, "antigenic variation in one spirochete, times all the spirochetes you have, leaves the immune system 'overwhelmed' with 'an infinite number of new antigens.'" This is a characteristic or attribute of bioweapons, well described by the US Army when speaking to Congress as shown previously in this document.

With all this malarkey about "Lyme disease" as opposed to relapsing fever, and how the pediatric Autism vaccines fail and give children the very brain infections they're meant to prevent (same mechanisms; immunosuppression either via fungal exposure or some other exposure, or genetic immune insufficiency, plus live, attenuated viruses that become un-attenuated), you get the impression that the CDC was never mentally or morally competent to maintaining theirs and the USDA's fallacies. We've longed called the CDC the Centers for Disease Confabulation.

Alan Barbour states below that OspA undergoes true antigenic variation and basically that, therefore you cannot use this as a vaccine (while he owns the patent for the ImmuLyme OspA non-vaccine). If it undergoes "true antigenic variation," it certainly cannot be used for DNA diagnostics as Klempner allegedly did in his "*BREAKING NEWS!!!*" bogus "re-treatment" "study" that is now the data used by IDSA for their "Guidelines on Lyme" from 2001 and 2006. Klempner said he looked for DNA of *Borrelia* in the spinal fluid of his victims – and used OspA primers (ones that will CHANGE and therefore not be there).

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

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

[Sädziene A1](#), [Rosa PA](#), [Thompson PA](#), [Hogan DM](#), [Barbour AG](#).

Notwithstanding infrequent application for bacterial studies presently, there were compelling reasons to use in vitro antibody selection with *Borrelia burgdorferi*, the agent of Lyme disease. First, we had shown for the related species, *B. hermsii*, that an antiserum specific for one serotype could select for new serotypes in an isogenic population undergoing in vitro growth (20). The ability of polyclonal antisera and mAbs to agglutinate (21) and inhibit the growth of *B. burgdorferi* (21a) indicated that this was also possible with the Lyme disease agent. Second, previous studies had shown antigenic differences in outer membrane proteins, OspA and OspB, between strains (21–26) and also true antigenic variation of these proteins within a strain (25, 27–30). If borrelias did "escape"

medicine • Volume 176 September 1992 799–809

Says Barbour above: "Second, previous studies had shown antigenic differences in outer membrane proteins, OspA and OspB, between strains (21–26) and also true antigenic variation of these proteins within a strain (25, 27–30)."

<http://www.ncbi.nlm.nih.gov/pubmed/1339462> , <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2119346/>

None of the OspA vaccines ever prevented Lyme or spirochetes in any animal. OspA vaccination may have prevented arthritis by tolerance, but no animal study showed prevention of spirochetes.

Remember, "mutants" is code language. They're all mutants. Antigenic variation or "selection pressure" is the nature of the relapse in relapsing fever. To call them mutants is silly and redundant,

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 potentially likely to spread around fast.

1995 – Barbour’s patent for Lyme in Missouri, using 16S RNA sequencing and flagellin primer probes that many of the Lyme Cabal members did not use when assessing human treatment outcomes.

Barbour says this is in Lone Star ticks in Missouri:

Diagnostic tests for a new spirochete, Borrelia lonestari sp. nov.

“Bites from Amblyomma americanum, a hard tick, have been associated with a Lyme disease-like illness in the southeastern and south-central United States. Present in 2% of ticks collected in four states were uncultivable spirochetes. Through use of the polymerase chain reaction, partial sequences of the flagellin and 16s rRNA genes of microorganisms from Texas and New Jersey were obtained. The sequences showed that the spirochete was a Borrelia sp. but distinct from other known members of this genus, including B. burgdorferi, the agent of Lyme disease. Species-specific differences in the sequences of the flagellin protein, the flagellin gene and the 16s rRNA gene between the new Borrelia species and previously known species provide compositions and methods for assay for determining the presence of this new spirochete, or for providing evidence of past or present infection by this spirochete in animal reservoirs and humans...

”...SUMMARY OF THE INVENTION

”The present invention provides compositions, methods, and kits for the detection of a new spirochete that is associated with a Lyme disease-like illness. **The compositions are based on a Borrelia lonestari sp. nov.-specific allotype or combination of allotypes of the flagellin protein, or a Borrelia lonestari sp. nov.-specific allele or combination of alleles of the flagellin or 16s rRNA genes of the new spirochete.** The allotypes and alleles provided by the present invention have been determined by nucleic acid sequencing of portions of the flagellin and rRNA genes from this new spirochete. Detection of a species-specific amino acid or nucleotide as defined herein, or a species-specific combination of amino acids or nucleotides as defined herein, in a subject sample is indicative of infection with *Borrelia lonestari* sp. nov.”

<http://patft.uspto.gov/netacgi/nph->

[Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetacgi/nph-PTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5%2C932%2C220.PN.&OS=PN%2F5%2C932%2C220&RS=PN%2F5%2C932%2C220](http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetacgi/nph-PTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=5%2C932%2C220.PN.&OS=PN%2F5%2C932%2C220&RS=PN%2F5%2C932%2C220)

Barbour sequenced the RNA and DNA, obviously and did not use someone else’s primers. Using primer probes from Borreliae not expected to be there (burgdorferi flagellin and another specific lonestari gene) rather than sequencing. Therefore, Borreliae cannot be ruled out as Wormser did when assaying EM rashes in Missouri. This patent of Barbour’s also shows Barbour knows what are the species-distinguishers: NOT THE OSPS, VMPS or the PLASMID DNA. He then can’t sign on to any claims about Lyme that mimic the CDC’s and IDSA’s current fraudulent positions without expecting to be indicted. Barbour does claim however, that ***the species identifier is... FLAGELLIN.***

Gary Wormser playing the DNA/RNA shell game.

Next we are going to look at Gary Wormser who is in 1992 **using the correct primers**; this proves he knows exactly how to identify Borrelia species. Later, in order to “prove” there is no Lyme in

Missouri, he does not apply this same technique.

J Clin Microbiol. 1992 Dec;30(12):3082-8.

Diagnosis of early Lyme disease by polymerase chain reaction amplification and culture of skin biopsies from erythema migrans lesions.

Schwartz IL, Wormser GP, Schwartz JJ, Cooper D, Weissensee P, Gazumyan A, Zimmermann E, Goldberg NS, Bittker S, Campbell GL, Pavia CS.

"rRNA-based PCR detection assay for *B. burgdorferi*.

"The organization of the rRNA genes of *B. burgdorferi* and the sequences of the corresponding rRNAs have been determined (32). Figure 1 presents a schematic diagram of the rRNA operon and the positions of the primers and probes employed for PCR amplification and detection. The 23S rRNA sequence was compared for homology to other rRNA sequences in the GenBank data base. On the basis of these comparisons, a region near the 5' end of the 23S RNA sequence (nucleotides 689 through 948) was chosen as a likely target for amplification. The equivalent regions of the 23S rRNA genes in the related species *Borrelia hermsii* and *B. anserina* and several isolates of *B. burgdorferi* were also sequenced (Fig. 2). PCR primers (designated JS1 and JS2) were designed to contain perfect homology to the *B. burgdorferi* sequence but maximum mismatch at their 3' ends with the related *Borrelia* species (Fig. 2). The sensitivity of the PCR assay was determined with serially diluted, titered *B. burgdorferi* samples. Fewer than 10 spirochetes in a total sample could be detected efficiently (Fig. 3). The sensitivity and specificity of the assay were also investigated by performing PCR amplification with 10 different isolates of *B. burgdorferi*, *B. hermsii*, *B. anserina*, and *Borrelia turicatae*. Samples containing 50 spirochetes were subjected to PCR amplification, and one-fifth of the amplified product (equal to 10 spirochetes) was detected by hybridization with a radiolabeled probe (FS1) corresponding to a portion of the amplified sequence. All isolates of *B. burgdorferi* were detected by the procedure with essentially equal efficiency (Fig. 4). These included isolates from North America (isolates 24430, 24352, HK, B31, 297), Europe (20004, G1, 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. We'll see that report shortly.

In this next report, **Gary Wormser** in 1999, when using the correct primers (16S) finds some people are infected with more than one species of *Borrelia burgdorferi* (yet, ignoring the other *Borrelia*) and that you can't really use Barbour-Kelly-Stoener culture media, the only one anyone sells in the USA. It shows Wormser knows what DNA to use when looking for spirochetes, yet he is a signer of the IDSA "Guidelines" which are based on Klempner's bogus "re-treatment" "study," which is based on the

falsified Dearborn case definition, and which is based on the bogus OspA gene to determine “no Lyme” after “treatment.”

J Clin Microbiol. 1999 Mar;37(3):565-9.

1999 - Genetic diversity of *Borrelia burgdorferi* in lyme disease patients as determined by culture versus direct PCR with clinical specimens.

Liveris D1, Varde S, Iyer R, Koenig S, Bittker S, Cooper D, McKenna D, Nowakowski J, Nadelman RB, Wormser GP, Schwartz I.

“... The data confirmed the presence of the three major RFLP types previously described (17). Of 183 skin isolates, 46 (25.1%) were type 1, 70 (38.3%) were type 2, and 55 (30.1%) were type 3; ***the remaining 6.6% (12 of 183) were mixed cultures composed of at least two genotypically distinct isolates.***”

<http://www.ncbi.nlm.nih.gov/pubmed/9986813>

1995-6—Alan Barbour does proper sequencing for the analysis of the spirochetes in the Lone Star tick (compare to what Wormser does, following this report):

J Infect Dis. 1996 Feb;173(2):403-9.

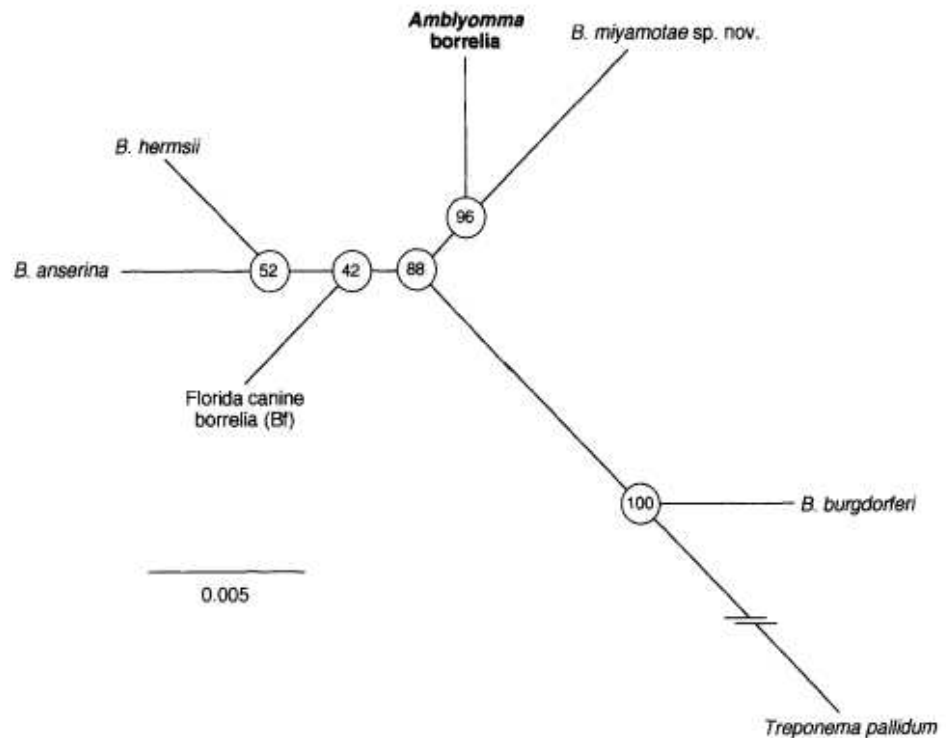
Identification of an uncultivable *Borrelia* species in the hard tick *Amblyomma americanum*: possible agent of a Lyme disease-like illness.

Barbour AG1, Maupin GO, Teltow GJ, Carter CJ, Piesman J.

“...The deduced amino acid sequences for flagellin proteins of the 2 microorganisms found in *A. americanum* were identical over 213 residues; the nucleotide differences between strains were synonymous. Figure 3 shows the alignment of part of the deduced flagellin sequences of the spirochetes found in *A. americanum* in Texas and New Jersey with the comparable variable regions of the flagellin proteins of 8 *Borrelia* species and *Treponema pallidum*, the spirochete that causes syphilis. The amino acid positions are numbered according to the full length *B. burgdorferi* flagellin protein. The flagellin proteins of microorganisms found in *A. americanum* differed from other borrelial flagellins at several positions and, uniquely among the *Borrelia* species, lacked most of a proline-alanine-rich region beginning around residue 220. The spirochetes found in *A. americanum* resembled *B. turicatae*, *B. hermsii*, *B. parkeri*, *B. crocidurae*, and *B. anserina* in being without the QAA at residues 204-206 of the Lyme disease agents *B. burgdorferi*, *B. garinii*, and *B. afzelii*...

“Analysis of 16S rRNA genes. Further phylogenetic classification was provided by comparison of 16S rRNA gene sequences (figures 4 and 5). The sequence of the spirochete found in *A. americanum* from Texas had the following identities with selected other spirochete 16S rRNA genes: *T. pallidum*, 79.6%; *B. burgdorferi*, 96.0%; *B. anserina*, 97.5%; *B. hermsii*, 97.8%; *B. miyamotoe* sp. nov., 98.3%; and the “Florida canine borrelia,” 98.4%. By distance matrix and parsimony analyses of the aligned sequences (figure 4), the spirochete found in *A. americanum* clustered with a group containing the relapsing fever species *B. hermsii*, *B. anserina*, the unnamed organism recovered from the blood of 2 dogs in Florida [25], and *B. miyamotoe* sp. nov. (accession no. 045192).”

Figure 4. Unrooted distance matrix phylogenetic tree of *Borrelia* species with *Treponema pallidum* as outgroup. 16S rRNA sequences corresponding to base positions 36–1371 of *Borrelia burgdorferi* 16S rRNA gene were aligned and analyzed with PHYLIP program package. Exhibited tree in New Hampshire standard format is: (((Florida canine borrelia: 100, (*Borrelia anserina*: 100, *Borrelia hermsii*: 100): 52): 42, (*borrelia* from *A. americanum*: 100, *Borrelia miyamotoe* sp. nov.: 100): 96): 88, *T. pallidum*: 100, *B. burgdorferi*: 100). Circled numbers indicate number of times (in 100) that particular node was supported by bootstrap analysis. Approximate evolutionary distances are measured along line segments; bar represents distance by Jukes-Cantor criteria of 0.005. Similar tree (not shown) was obtained by parsimony analysis of 100 bootstrapped datum sets: ((((((*borrelia* from *A. americanum*: 100, *B. miyamotoe*: 100): 94, *B. hermsii*: 100): 34, Florida canine borrelia: 100): 25, *B. anserina*: 100): 81, *B. burgdorferi*: 100): 100, *T. pallidum*: 100).



5). Among the 6 sequences represented in figure 5, there were 49 aligned positions at which only 1 of the 6 species differed; 27 (53%) of these differences were in *B. burgdorferi*. The following tree was produced with 100 bootstrapped data sets of these positions: ((((*borrelia* from *A. americanum*: 100, *B. miyamotoe*: 100): 94.4, (Florida canine borrelia: 100, (*B. hermsii*: 100, *B. anserina*: 100): 38): 64): 100, *B. burgdorferi*: 100).

Again, the borrelia from *A. americanum* clustered with the non-Lyme disease *Borrelia* species; it was most closely related to *B. miyamotoe* sp. nov. and the Florida canine borrelia.

Discussion

In this study, ~2% of *A. americanum* ticks from Missouri, New Jersey, New York, North Carolina, and Texas contained

”Figure 4. Unrooted distance matrix phylogenetic tree of *Borrelia* species with *Treponema pallidum* as 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 miyamotoe* sp. nov.: 100): 96): 88, *T. pallidum*: 100, *B. burgdorferi*: 100). Circled numbers indicate number of times (in 100) that particular node was supported by bootstrap analysis. Approximate evolutionary distances are measured along line segments; bar represents distance by Jukes-Cantor criteria of 0.005. Similar tree (not shown) was obtained by parsimony analysis of 100 bootstrapped datum sets: «««(*borrelia* from *A. americanum*: 100, *B. miyamotoe*: 1(0): 94, *B. hermsii*: 1(0): 34, Florida canine borrelia: 100): 25, *B. anserina* 100): 81, *B. burgdorferi*: 100): 100, *T. pallidum*: 100).”

<http://www.ncbi.nlm.nih.gov/pubmed/8568302> jid.oxfordjournals.org/content/173/2/403.full.pdf

Barbour actually sequenced for flagellin and 16S RNA and found all kinds of spirochete in this way.

For the Missouri Lyme Masters' Disease spirochete, Barbour said...

Again, the borrelia from *A. americanum* clustered with the non-Lyme disease *Borrelia* species; it was most closely related to *B. miyamotae* sp. nov. and the Florida canine borrelia.

... while Wormser tried to say "No Lyme In Missouri." (By the way, no one cares if they have *burgdorferi* or *antarcticii* or *siberii* or freakin *jupiterii*. They just want to know if the science shows they're sick.)

Here is Wormser trying to fool Edwin Masters, using the wrong DNA and RNA so he can say, "There is no Lyme in Missouri"

[Clin Infect Dis](#). 2005 Feb 1;40(3):423-8. Epub 2005 Jan 10.

Microbiologic evaluation of patients from Missouri with erythema migrans.

[Wormser GP](#), [Masters E](#), [Liveris D](#), [Nowakowski J](#), [Nadelman RB](#), [Holmgren D](#), [Bittker S](#), [Cooper D](#), [Wang G](#), [Schwartz I](#).

"PCR amplifications were performed in a 50-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'-CTGCAGCTTGAATTCAGGCACTTC-3'; reverse primer, 5'-GTTTTGTAATTCAACTGCTGACCCCTC-3') and/or recA [27]."

<http://www.ncbi.nlm.nih.gov/pubmed/15668867> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2773674/>

Wormser did not use the correct *Borreliae*-specific (for non-*burgdorferi* or from any other relapsing fever groups) flagellin genes, or 16S rDNA specific to *Borreliae* species, nor did he actually try to sequence any of these *Borreliae* as Barbour did (and Telford, below). Wormser would have known to use the correct method to detect spirochetes in Lone Star ticks, since he referenced Barbour's work (ref 11 was: ***Identification of an uncultivable Borrelia species in the hard tick Amblyomma americanum: possible agent of a Lyme disease-like illness.*** (shown above). Wormser knows how to do this kind of DNA analysis and that there are all sorts of *Borrelia* in Lone Star ticks – and ones that cause human disease.

The enzyme Wormser talks about, GlpQ (next reference here, by the NIH) is specific to *B. lonestari*, but that does not mean there are no disease-causing *Borreliae* in Lone Star ticks or Missouri.

J Bacteriol. 2003 Feb;185(4):1346-56.

Glycerol-3-phosphate acquisition in spirochetes: distribution and biological activity of glycerophosphodiester phosphodiesterase (GlpQ) among Borrelia species.

Schwan TG1, Battisti JM, Porcella SF, Raffel SJ, Schrumpf ME, Fischer ER, Carroll JA, Stewart PE, Rosa P, Somerville GA.

<http://www.ncbi.nlm.nih.gov/pubmed/12562805> , <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC142843/>

Wormser's whole point was to say there are no *Borrelia* causing disease or "Lyme" in Missouri. The very last statement he makes in that report is: "Although it is unknown whether this rash illness has an infectious etiology, it is important to emphasize that this study does not indicate the absence of a therapeutic role for antibiotic treatment." (AKA, CYA, in the common vernacular.) **It could have been some other *Borrelia*, just not *burgdorferi* or *lonestari*. Check the Taxonomy database for all the *Borrelia* in the USA alone.**

Additionally, these were Wormser's actual results:

Table 1**Test results for specimens obtained from patients with erythema migrans (EM) or EM-like lesions**

Laboratory test	Missouri patients with EM-like lesions (n = 30)	New York State patients with EM (n = 143)	P ^a
Skin PCR/culture			
Positive <i>flaB</i> PCR	5/31 (16) ^b	70/139 (50)	<.001
Positive eubacterial 16S RNA PCR	0/20 (0) ^b	Not done	
Positive <i>glpQ</i> PCR	0/22 (0) ^b	Not done	
Positive <i>Borrelia burgdorferi</i> culture	0/19 (0)	89/142 (63)	<.001
Serum serologic analysis			
Acute phase, seropositive ^c	0/25 (0)	81/143 (57)	<.001
Convalescent phase, seropositive (1–3 months)	0/22 (0)	99/135 (73)	<.001
Seroconversion ^d	0/22 (0)	25/52 (48)	<.001
Acute or convalescent phase, seropositive	0/25 (0)	107/143 (75)	<.001
Overall ^e	5/31 (16) ^b	130/143 (90.9)	<.001

NOTE. Data are no. of specimens with positive test results/no. that were evaluable (%).^aBy 2-tailed Fisher's exact test.^bTwo biopsies of separate EM-like lesions were performed for 1 patient.^cBy ELISA.^dNegative-to-positive or equivocal-to-positive results ≤3 months after baseline.^ePositive *B. burgdorferi* culture, positive PCR result, or seropositive.**5 were positive
for a Bb fla gene.****Here is Wormser's abstract:**[Clin Infect Dis.](#) 2005 Feb 1;40(3):423-8. Epub 2005 Jan 10.

Microbiologic evaluation of patients from Missouri with erythema migrans.

[Wormser GP](#)¹, [Masters E](#), [Liveris D](#), [Nowakowski J](#), [Nadelman RB](#), [Holmgren D](#), [Bittker S](#), [Cooper D](#), [Wang G](#), [Schwartz I](#).

"*Borrelia lonestari* infects *Amblyomma americanum*, the tick species that is the most common cause of tick bites in southeast and south-central United States, and this spirochete has been detected in an erythema migrans (EM)-like skin rash in 1 patient. Therefore, *B. lonestari* is considered to be a leading candidate for the etiologic agent of EM in this region.

"Skin biopsy specimens obtained from patients from the Cape Girardeau area of Missouri who had EM-like lesions were cultured in Barbour-Stoenner-Kelly medium and evaluated by polymerase chain reaction (PCR) targeting multiple genes. Serum specimens were tested by enzyme-linked

immunosorbent assay for antibodies against sonicated whole-cell *Borrelia burgdorferi*. Results were compared with those obtained over the same period for patients from New York State who had EM. “*B. lonestari* was not detected by PCR in any of 31 skin biopsy specimens collected from 30 Missouri patients. None of 19 cultures of Missouri skin samples that were suitable for evaluation were positive for *B. burgdorferi*, compared with 89 (63%) of 142 cultures of samples collected from New York State patients ($P < .001$). None of the 25 evaluable Missouri patients were seropositive for antibodies against *B. burgdorferi*, compared with 107 (75%) of 143 New York State patients ($P < .001$). **“Neither *B. lonestari* nor *B. burgdorferi* is likely to be the cause of EM-like skin lesions in patients from the Cape Girardeau area of Missouri. The etiology of this condition remains unknown.** <https://www.ncbi.nlm.nih.gov/pubmed/15668867>

So, just to reiterate Wormser’s Take-Home Message here: ”There is no Lyme in Missouri except for those 5 out of ~30, and we were playing around with the DNA primers. (Shhh: It’s closer to *B. miyamotae* and Florida Canine Lyme disease *Borrelia*. Also, Shhhh, the Barbour culture medium is specific to *burgdorferi* spirochetes and does not grow all spirochetes.) The EM-Lyme illness in Missouri could have been from some other Relapsing Fever or Ehrlichial pathogen but we are pretending there is no such thing as an illness from a tick bite. Lyme is imaginary so get the placebo vaccine. We used no *hermsii* or other common spirochetes known to be in the west and south of the United States. The end game is to not find illness and to say that *there is no illness from a tick bite*, while Barbour patented the *lonestari* bug right under Masters’ nose for later use as a vaccine against this disease that does not exist and no one has so get the vaccine....especially in Missouri.”

It is, yes. This is a dizzying kaleidoscope of lies, is what you’re thinking now. The whole point of it all, and the reason this scam continues, is because the Cabal does not want anyone to know they falsified the case definition at Dearborn. They LIED to everyone and said, “No, ‘Lyme Disease’ is a self-limiting autoimmune monoarticular joint disease. It does not cause any illness.” (It’s “pretense.”)

Well, why do modern spirochetes not cause any illness? Because OspA injections had the same outcome. These criminal dummy wannabee scientists are mortified, really, over being discovered to be not only that stupid, but crooked. Think about it. They never *were* “the smartest guys in the room,” but they wanted to be seen as an authority on something, didn’t they? It’s obvious. Why else would they e-stalk us and e-trash us for going on 3 decades now, as way to allegedly be “scientific?”

Mark Klempner, playing the DNA/RNA shell game.

You have previously seen that the OspA gene undergoes antigenic variation and is not found in all *Borreliae*. You can’t use this DNA for anything, especially not vaccines or detection. We move on to the Klempner “study” which unfortunately resulted in the 2001 and 2006 IDSA “Guidelines” and where he references which DNA he used to assess “NO LYME IN LYME VICTIMS.” Klempner doesn’t actually say what DNA he uses (only by reference) to determine “No Lyme” in Lyme victims, and the peer reviewers at the New England Journal of Medicine (NEJM) never noticed he did not list his primers:

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

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

Klempner MS¹, Hu LT, Evans J, Schmid CH, Johnson GM, Trevino RP, Norton D, Levy L, Wall D, McCall J, Kosinski M, Weinstein A.
<https://www.ncbi.nlm.nih.gov/pubmed/11450676>
<http://www.nejm.org/doi/full/10.1056/NEJM200107123450202#t=articleMethods>

Laboratory Studies

Western blotting for IgG antibodies against *B. burgdorferi* antigens was performed with the IgG MarBlot (MarDx Diagnostics, Carlsbad, Calif.) according to the manufacturer's instructions.⁶ The intrathecal production of antibodies against *B. burgdorferi* was measured as previously described.²⁰ Base-line specimens of cerebrospinal fluid and plasma specimens obtained at base line and on days 3, 5, 21, and 45 were tested by PCR for the presence of *B. burgdorferi* DNA, as previously described.²¹ All samples of cerebrospinal fluid were cultured in Barbour–Stoenner–Kelly II medium to detect *B. burgdorferi* and were monitored by dark-field microscopy for six weeks.²² Some blood samples were cultured for *B. burgdorferi* in hypertonic medium.²³

“Laboratory Studies

“Western blotting for IgG antibodies against *B. burgdorferi* antigens was performed with the IgG MarBlot (MarDx Diagnostics, Carlsbad, Calif.) according to the manufacturer's instructions.⁶ The intrathecal production of antibodies against *B. burgdorferi* was measured as previously described.²⁰ Base-line specimens of cerebrospinal fluid and plasma specimens obtained at base line and on days 3, 5, 21, and 45 were tested by PCR for the presence of ***B. burgdorferi* DNA**, as previously described.²¹ All samples of cerebrospinal fluid were cultured in Barbour–Stoenner–Kelly II medium to detect *B. burgdorferi* and were monitored by dark-field microscopy for six weeks.²² Some blood samples were cultured for *B. burgdorferi* in hypertonic medium.²³”

So, what was that mysterious REFERENCE 21 above ^^ DNA that Klempner failed to report and the so-called peer-reviewers did not notice?

Steere's

[J Infect Dis.](#) 1996 Sep;174(3):623-7.

Detection of *Borrelia burgdorferi* DNA by polymerase chain reaction in cerebrospinal fluid in Lyme neuroborreliosis.

Nocton JJ¹, Bloom BJ, Rutledge BJ, Persing DH, Logigian EL, Schmid CH, Steere AC.

<https://www.ncbi.nlm.nih.gov/pubmed/8769624> <http://jid.oxfordjournals.org/content/174/3/623.full.pdf>

WHICH SAYS:

PCR assay. CSF samples from case and control patients were processed simultaneously in a blinded manner, as described [11]. Briefly, DNA was isolated from 100 μ L of CSF, and the DNA extract was resuspended in 30 μ L of ultrapure water. A 5- μ L aliquot from this suspension was amplified with primer-probe set 1, which targets base pairs 788–943 at the 3' end of the 50-kb plasmid of *B. burgdorferi* that encodes OspA [11]. This primer-probe set detects most strains of *B. burgdorferi* from New England. A second 5- μ L aliquot was amplified with primer-probe set 2, which targets base pairs 149–343 at the 5' end of the *ospA* gene [11]. This set detects all North American and European isolates tested to date, with the exception of rare natural isolates that lack the 50-kb plasmid. Amplification consisted of 45 cycles of denatur-

ONLY an OspA gene and later added an OspB gene (read the whole report). And where Steere found many positive patients, Klempner says he found none (2001 RI “Diseases of Summer” conference at South County Hospital, audiotaped).

The Cabal – 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 for 16S or flagellin DNA (non-variable, although species-specific and for which there are more copies) in the past, particularly to patent and therefore own species; 3) and referenced the NIH recommendation for using these 16 and 23S probes.

Durland Fish, using *the correct* primers to look for new species of Borreliae to patent in 2001, yet is a signer of the IDSA “Guidelines” once again, based only on an OspA gene and Dearborn as “Lyme” or the chronic illness caused by a tick bite.

Vector Borne Zoonotic Dis. 2001 Spring;1(1):21-34.

A relapsing fever group spirochete transmitted by Ixodes scapularis ticks.

Scoles GA1, Papero M, Beati L, Fish D.

"A 1,347-bp portion of 16S rDNA was amplified from a pool of infected nymphs, sequenced, and compared with the homologous fragment from 26 other species of Borrelia. The analysis showed 4.6% pairwise difference from *B. burgdorferi*, with the closest relative being Borrelia miyamotoi (99.3% similarity) reported from *Ixodes persulcatus* in Japan. Phylogenetic analysis showed the unknown Borrelia to cluster with relapsing fever group spirochetes rather than with Lyme disease spirochetes. A 764-bp fragment of the flagellin gene was also compared with the homologous fragment from 24 other Borrelia species. **The flagellin sequence of *B. burgdorferi* was 19.5% different from the unknown Borrelia and showed 98.6% similarity with *B. miyamotoi*.**"

<http://www.ncbi.nlm.nih.gov/pubmed/12653133>

What that means is the probably-Plum-Island-and-unlikely-hurricane-borrelia, *B. burgdorferi*, migrated to Japan and back to the United States again, “mutating” to adapt to a Japanese *Ixodes* tick. Yet, a year later, we see Durland Fish using the WRONG DNA (OspA gene again), to assess treated mice, to determine if there is any Borrelia, coming to the conclusion that there is pretty much no Borrelia:

Detection of Attenuated, Noninfectious Spirochetes in 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'-AACCCTTTACTTTCTTCGATTGCGCT-3'. The primer set for p37–47 has 100%

homology to sequences in both *B. burgdorferi* strains B31 and N40, and the gene has been localized to lp28-1 in both strains [26, 27]. The primer set for BBE21.1 amplifies a unique region in lp25 of *B. burgdorferi* strain B31 downstream of BBE21 (amplicon coordinates 13403–14530) [28]. BBE21 is located on a similar-size plasmid within *B. burgdorferi* strain N40 [29]. We have been able to amplify by PCR the region corresponding to GenBank accession number AE000785, product amplicon coordinates 14195–14921, indicating that BBE21 and BBE21.1 reside on the same plasmid in N40 (authors' unpublished data)"

<http://jid.oxfordjournals.org/content/186/10/1430.long>

Those are plasmids, those "lp" things. Plasmids are from where the variable surface protein antigens vary themselves. So, that is a classical Durland Fish type "bogus article." See: http://www.actionlyme.org/TICK_BITE_CONSPIRACY.htm where Durland admits that he writes "bogus articles," not that you're not already convinced. Anyway, previously he used the correct DNA, 16rDNA and flagellin, but then in a post-treated case or cases, he used a gene not likely to be there, OspA.

It probably is true that the spirochetes become attenuated as we have seen with Jay Sanford stating that spirochetes "persist in the brain and eye even after apparent cure" (or they do all their damage early), and Alan Barbour recommending infecting syphilis patients with old, wimpy, high-passage borrelia spirochetes to raise a fever. Spirochetal diseases are all un-eradicable, as shown above. And surely it is true that older spirochete populations in the same host lose plasmids [NEVER use plasmid DNA to assess Lyme in humans], but the end game, and the point of all this crime, is that the Cabal is trying to say that their chronically ill victims are not sick, just crazy. In the end it was OspA itself, a fungal antigen causing the reactivation of the herpesviruses, Post-Sepsis Syndrome, and humoral immunosuppression with chronic inflammation in the brain due to all the neurotropic herpes viruses and Mycoplasma, etc., that blew up their intended RICO "enterprise" scam (ALDF.com). It was a very dumb choice for a vaccine.

All of what you see in these SASH criminal charge sheets is evolving criminal fraud in an attempt to hide all their previous lies. The most serious offense, falsifying the case definition and rendering the 85% without the arthritis HLAs - the million or so per year - permanently disabled, is the one offense the Cabal so vigorously tries to mask by issuing "Guidelines." The "Guidelines," based on Klempner, which is based on Dearborn, is a way of Offense being the Best Defense. It's Pretense, or FRAUD. The Cabal would have the world believe that *they* all believe the case definition was real and valid. We know for sure they know it was not valid acceptable based on the non-consensus at Dearborn alone.

To put such debased individuals in charge of humans and vector-borne diseases? That's the United States; everything happens exclusively for profit. Greed is our nature. It is synonymous with Exceptional! It's ALL ABOUT ME!! And it's ALL ABOUT MONEY!! Hence, the new and totally novel in human history, the BS de-scrambler Society for the Advancement of Scientific Hermeneutics. Now we have Scientific Hermeneutics because this BS has become like a religion or belief system, a DOCTRINE, if you will, that to date, no "doctor" ever unscrambled on behalf of humanity.

Sam Telford's 2001 report saying "Southern Lyme" is closest to *theileri* or bovine relapsing fever (the former "Tick Fever" that the cowboy/farmer wars were all about):

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

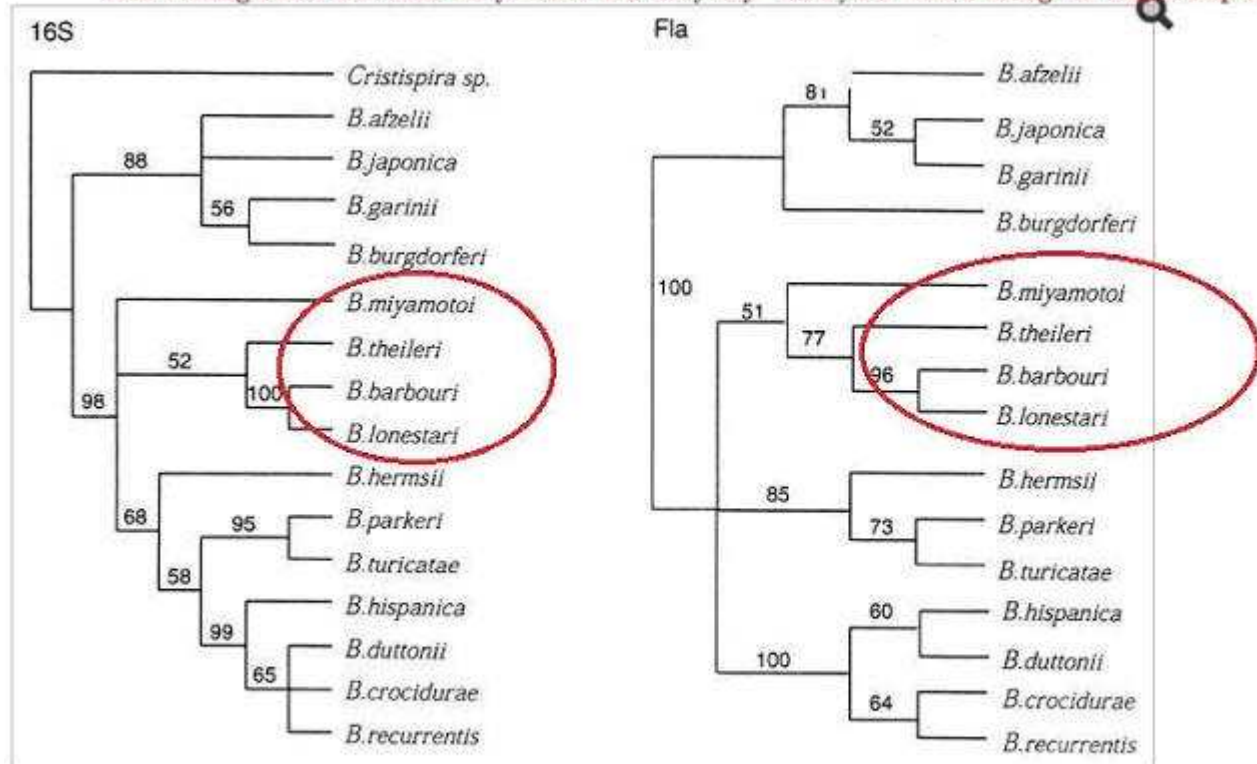
Lone star tick-infecting borreliae are most closely related to the agent of bovine borreliosis.

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

“Although *Borrelia theileri*, the agent of bovine borreliosis, was described at the turn of the century (in 1903), its relationship with borreliae causing Lyme disease or relapsing fever remains undescribed. We tested the previously published hypothesis that spirochetes infecting Lone Star ticks (*Amblyomma americanum*) may comprise *B. theileri* by analyzing the 16S ribosomal DNAs (rDNAs) and flagellin genes of these spirochetes. 9, the *Amblyomma* agent, and *B. miyamotoi* formed a natural group or clade distinct from but most closely related to that of the relapsing fever spirochetes. *B. theileri* and the *Amblyomma* agent were 97 and 98% similar at the nucleotide level within the analyzed portions of the 16S rDNA and the flagellin gene respectively, suggesting a recent divergence. The agent of bovine borreliosis might be explored as a surrogate antigen for the as-yet-uncultivable *Amblyomma* agent in studies designed to explore the etiology of a Lyme disease-like infection associated with Lone Star ticks.”

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

FIG. 1 When using the correct DNA or RNA primers, crooks find Masters' Disease or Southern Lyme or any other effing kinda *Borreliae* they want. But they say "No Lyme" when using the bogus *OspA* gene.



Bootstrap consensus (1,000 times) of neighbor-joining trees of *Borrelia* spirochetes using both 16S rDNA (left) and flagellin (right) loci. *Cristispira sp.* was used as an outgroup. Distances were derived from the Tamura-Nei algorithm (27a). Numbers indicate the bootstrap s

You can see that the Cabal had already sequenced the 3 similar strains of flagellar and genus specific 16S RNA spacer genes that are derived from a cow or bovine relapsing fever (*theileri*, *barbouri*, and *lonestari*). But there is no “Lyme” in Missouri. You can see it is a shell game.

TO THIS DATE – from 1995 to 2015 - still, no one is using any other of this proper DNA or RNA or SEQUENCING rather than using bogus primer probes they know will not be found in humans to detect human illness. *They all know* the only way to detect Lyme/Relapsing Fever is with specific recombinant 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 say, essentially, "OspA is bogus due to antigenic variation"

J Immunol. 1995 Dec 15;155(12):5700-4.

An ospA frame shift, identified from DNA in Lyme arthritis synovial fluid, results in an outer surface protein A that does not bind protective antibodies.

Fikrig E1, Liu B, Fu LL, Das S, Smallwood JI, Flavell RA, Persing DH, Schoen RT, Barthold SW, Malawista SE.

"Passive immunization with murine or human Abs to outer surface protein A (OspA) can protect mice against *Borrelia burgdorferi*, but OspA Abs elicited during natural infection in mice or humans are unable to clear the spirochete from the infected host. To examine Ab binding by OspA during the course of human infection, we amplified the operon encoding full-length ospA and ospB from synovial fluids of a patient with chronic Lyme arthritis, the first such recoveries from human material, at four separate time points over 4.5 mo, and expressed OspA in *Escherichia coli*. OspA mAbs that passively protected mice from infection did not bind one of the expressed OspAs, because of a deletion in ospA that resulted in a frame shift and premature stop codon near the carboxyl terminus. However, expressed OspA from a later synovial fluid sample did not contain this deletion. Thus, although altered forms of OspA, which potentially can influence host immune effectiveness, do occur in the human host, they cannot be the only factors responsible for microbial persistence."

<http://www.ncbi.nlm.nih.gov/pubmed/7499856>

Oh, you mean Lyme is a Relapsing Fever organism, so you can't use the OspA gene for human treatment outcomes assessment or vaccines, huh Mr. Schoen, or to detect "Lyme" in EM rashes in Missouri?

Telford, Fish, Schoen, Steere, Persing, Barbour, etc., were able to find any kind of *Borrelia* anywhere America – and they are everywhere, North, South, Central, West -, sequencing for species-specific non-variable, non-plasmid DNA.

Yale's Robert Schoen (who says Lyme is not a real disease, says, "I call it Lyme paranoia," and needs no treatment) using 23S RNA primers to assure his RICO monopoly strain (and later patent with Dave Persing, US Patent 6,045,804) is *burgdorferi*. On page 235 of the .pdf, Schoen says:

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

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

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

"...Subsequent evaluation of this isolate in our laboratory showed that this strain was nonreactive with

an OspA-based PCR assay designed to detect all North American and European isolates of *B. burgdorferi* but that it contained **23S ribosomal DNA** sequences indistinguishable from those of most North American strains of *B. burgdorferi* sensu stricto such as strains B31 and N40 (22). Genomic macrorestriction analysis of this isolate by PFGE is shown in Fig. 1. By PFGE, the isolate is related to *B. burgdorferi* N40, relatives of which are widely distributed in the northeastern United States, the Upper Midwest, and California (22). These isolates are also closely related to type strain B31, in contrast to isolates from moderate-climate regions of the southeastern and southwestern United States, which are often related to strain 25015 (19, 22). However, in contrast to strain N40, strain 49736 apparently lacked the ca. 53-kb linear plasmid species presumed to encode OspA and B. To verify this observation, we hybridized Southern blots of the *Mlu*I digest with a probe specific for the OspA gene. In contrast to strains N40 and B31, which were strongly OspA probe positive, no detectable signal was observed in the digest derived from strain 49736 (not shown). This observation was consistent with the absence of the 53-kb plasmid species. Similar results were obtained from N40-like isolates 46047, 48510, and B31-like isolates 46794 and 50772 (1)."

<http://www.ncbi.nlm.nih.gov/pubmed/8968914> <http://jcm.asm.org/content/35/1/233.full.pdf>

FIG. 1. PFGE analysis of *Mlu*I-digested genomic DNA from *B. burgdorferi* B31, N40, and 49736. The unmarked lane contains a mixture of lambda DNA *Hind*III fragments, lambda DNA, and lambda concatemers (Sigma) used as a molecular size marker. Southern blotting of this gel followed by hybridization with an OspA probe (OspA6s-3a) also showed that isolate 49736 lacked OspA (data not shown).

profile by protein gel electrophoresis (1). Subsequent evaluation of this isolate in our laboratory showed that this strain was nonreactive with an OspA-based PCR assay designed to detect all North American and European isolates of *B. burgdorferi* but that it contained 23S ribosomal DNA sequences indistinguishable from those of most North American strains of *B. burgdorferi* sensu stricto such as strains B31 and N40 (22). Genomic macrorestriction analysis of this isolate by PFGE is shown in Fig. 1. By PFGE, the isolate is related to *B. burgdorferi* N40, relatives of which are widely distributed in the northeastern United States, the Upper Midwest, and California (22). These isolates are also closely related to type strain B31, in contrast to isolates from moderate-climate regions of the southeastern and southwestern United States, which are often related to strain 25015 (19, 22). However, in contrast to strain N40, strain 49736 apparently lacked the ca. 53-kb linear plasmid species presumed to encode OspA and B. To verify this observation,

RNA/DNA Shell Game

Here Yale's Robert Schoen (who says Lyme is not a real disease and needs no treatment) using 23S RNA primers to assure his RICO monopoly strain (and later patent with Dave Persing) is related to *burgdorferi*,...

and also reveals there is "Lyme" in the Southern and Western states in 1996.

PubMed ID # 8968914
US Pat No. 6,045,804

and also reveals there is "Lyme" in the Southern and Western states in 1996:

Says Schoen, "These isolates are closely related to type strain B31, in contrast to isolates from moderate-climate regions of the southeastern and southwestern United States which are often related to strain 25015 (19,22)."

And what are those references, 19 and 22?

REF 19 - [Res Microbiol](#). 1995 Jun;146(5):415-24.

Two geographically distinct isolates of Borrelia burgdorferi from the United States share a common unique ancestor.

[Kolbert CP](#)¹, [Podzorski DS](#), [Mathiesen DA](#), [Wortman AT](#), [Gazumyan A](#), [Schwartz I](#), [Persing DH](#).

“The genetic diversity of *Borrelia burgdorferi* isolates from several geographic regions was evaluated by nucleotide sequence analysis of the genes encoding **23S ribosomal RNA** and outer surface protein A. Comparison of nucleotide sequences spanning 738 bp of the 23S ribosomal DNA from two unusual isolates, DN127 (Del Norte County, California) and 25015 (Millbrook, New York), to homologous sequences from other *B. burgdorferi* isolates from the United States and Russia identified several nucleotide sequence polymorphisms that are unique to these two isolates. Sequence analysis of a 615 nucleotide segment of the gene encoding outer surface protein A also revealed greater similarity of strains DN127 and 25015 (94.1%) compared to other US and Eurasian isolates. These data were further corroborated by genomic macrorestriction analysis, in which DN127 and 25015 demonstrated unique restriction digestion patterns. Our findings suggest that substantial genetic diversity of *B. burgdorferi*, rivaling that of European strains, exists among isolates from the United States. Strains DN127 and 25015 are unique among all *B. burgdorferi* isolates tested to date, and though isolated from opposite longitudinal extremes of the North American continent, are closely related.”

<http://www.ncbi.nlm.nih.gov/pubmed/8525058>

In other words, this funny-, like, accidental- Ixodes-come-Plum Island borrelia had already reached the American West, not to mention Europe, by 1995. Are we to believe Missouri has an invisible anti-bird and anti-rodent barrier?

REF 22- 1997 – **Persing and Telford**, again (and you’ll just have to look at the full text pdf of this article because you’re not going to believe how many different kinds of *Borrelia* are found in just about every state):

[J Infect Dis](#). 1997 Jan;175(1):98-107.

Genetic heterogeneity of Borrelia burgdorferi in the United States.

[Mathiesen DA](#)¹, [Oliver JH Jr](#), [Kolbert CP](#), [Tullson ED](#), [Johnson BJ](#), [Campbell GL](#), [Mitchell PD](#), [Reed KD](#), [Telford SR](#)^{3rd}, [Anderson JF](#), [Lane RS](#), [Persing DH](#).

"To examine in detail *Borrelia burgdorferi* strain diversity in the United States, 186 isolates from human, tick, and rodent sources were analyzed from multiple distinct geographic regions of the United States and abroad. Strains were characterized by genomic macrorestriction analysis and *ospA* and **23S rDNA gene** sequencing followed by phylogenetic analysis. Results indicate that spirochetal isolates from the United States fall into two major divisions and nine or more subdivisions; human isolates fell into five of these subdivisions. Greater genetic diversity was observed among *B. burgdorferi* isolates from moderate climatic regions, consistent with increased tick vector and reservoir diversity. All of the *Borrelia* isolates were reactive by *ospA* polymerase chain reaction except for *Borrelia hermsii* controls and several tick isolates from the Northeast, which were shown to lack the 49-kb plasmid encoding outer surface protein A (*OspA*). The data suggest that US *B. burgdorferi* isolates demonstrate substantial genetic heterogeneity, with regional differences in spirochete populations.

<http://www.ncbi.nlm.nih.gov/pubmed/8985202>

<http://jid.oxfordjournals.org/content/175/1/98.long>

Citing Authors:

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

This is all in comparison to what IDSA says about Lyme and particularly what Klempner did with his research-fraud re-treatment study published in 2001, which is now the basis of the IDSA "Guidelines." Klempner allegedly looked for the OspA gene in people, so he could declare that no one had Lyme after "re-treatment": <http://www.ohioactionlyme.org/wp-content/uploads/2015/02/Biomarkers1.pdf>

Allen Steere playing the DNA-RNA Shell Game; from 1992 when he falsified the Dearborn case definition (ref Marconi and the NIH re 16S probes), his 2 DNA analyses of post-treatment of humans where he found treatment failed in at least a third of the cases, and in the spinal fluid analysis where he used only an OspA probe, dropping the 16S probe he used in bad knees. Steere signs the "Guidelines" anyway and denies that treatment fails.

Allen Steere in 1992 when he falsified the Dearborn case definition, see his reference to Marconi and assessments of strains with 16S RNA; notice references 11, 24...

1992-1994 -- [J Infect Dis](#). 1994 Feb;169(2):313-8.

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

[Dressler F1](#), [Ackermann R](#), [Steere AC](#).

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

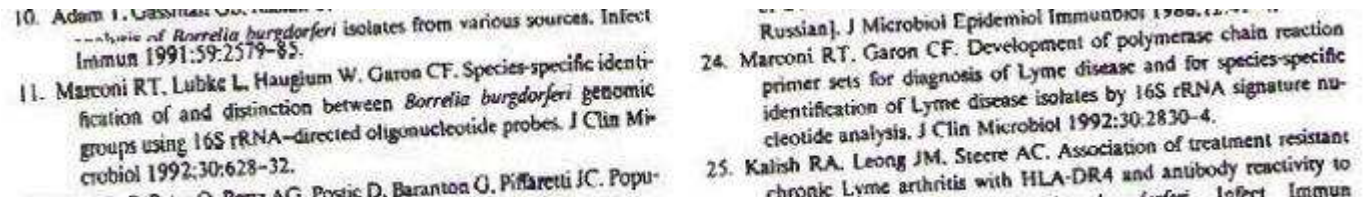
bite, and clinical manifestations of the infection.

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

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

And what are those references, 11 and 24? See the full text at:

<http://www.actionlyme.org/dressler1994.pdf>

- 
10. Adam T, Cassman G. Analysis of *Borrelia burgdorferi* isolates from various sources. Infect Immun 1991;59:2579-85.
11. Marconi RT, Lubke L, Hauglum W, Garon CF. Species-specific identification of and distinction between *Borrelia burgdorferi* genomic groups using 16S rRNA-directed oligonucleotide probes. J Clin Microbiol 1992;30:628-32.
24. Marconi RT, Garon CF. Development of polymerase chain reaction primer sets for diagnosis of Lyme disease and for species-specific identification of Lyme disease isolates by 16S rRNA signature nucleotide analysis. J Clin Microbiol 1992;30:2830-4.
25. Kalish RA, Leong JM, Steere AC. Association of treatment resistant chronic Lyme arthritis with HLA-DR4 and antibody reactivity to

Steere's Dearborn Reference 11: 1992, Marconi and Garon, NIH Bioweapons Lab, Montana--

[J Clin Microbiol](#). 1992 Mar;30(3):628-32.

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

[Marconi RT](#)1, [Lubke L](#), [Hauglum W](#), [Garon CF](#).

“Examination of a number of previously published aligned *Borrelia* 16S rRNA sequences revealed the presence of regions which could serve as oligonucleotide probe targets for both species-specific identification of *Borrelia burgdorferi* and distinction between genomic groups. Total cellular RNA isolated from *Borrelia* cultures was used in slot blot analysis. Radiolabeled oligonucleotides designed to hybridize to specific 16S rRNA targets were used as probes. These probes allowed for both species-specific identification and genomic group typing of *B. burgdorferi*...

“... Using *Borrelia* 16S rRNA sequences, we constructed probes that serve to distinguish *B. burgdorferi* from other *Borrelia* species and to distinguish between the genomic groups of *B. burgdorferi*. Other groups have developed *B. burgdorferi* species-specific probes by using polymerase chain reaction amplification (13, 15, 19, 22). We chose rRNA as the target molecule since it is present in large quantities within a cell, so rRNA targets can be considered to be naturally highly amplified. In addition, rRNA molecules are highly conserved and presumably are subject to a very low mutation frequency. The specificity of the probes was demonstrated through the use of slot blots with total cellular RNA as the target. This approach allows the reliable identification and genomic typing of *B. burgdorferi* from cultures, typically within 36 h.

<http://www.ncbi.nlm.nih.gov/pubmed/1372620>

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC265123/pdf/jcm00027-0106.pdf>

Steere's Dearborn Reference 24: 1992, Marconi and Garon, NIH:

[J Clin Microbiol](#). 1992 Nov;30(11):2830-4.

Development of polymerase chain reaction primer sets for diagnosis of Lyme disease and for species-specific identification of Lyme disease isolates by 16S rRNA signature nucleotide analysis.

[Marconi RT](#)1, [Garon CF](#).

“We have determined and compared partial **16S rRNA** sequences from 23 Lyme disease spirochete isolates and aligned these with 8 sequences previously presented. The 16S rRNA signature nucleotide compositions were defined for each isolate and compared with the genomic species signature nucleotide sets previously established. To identify positions truly indicative of species classification which could serve as targets for polymerase chain reaction species-specific identification primers, 16S rRNA-based phylogenetic analyses were conducted. On the basis of the identified signature nucleotides, we designed polymerase chain reaction primer sets which (i) amplify all spirochete species associated with Lyme disease and (ii) differentiate between these species. The primer sets were tested on 38 *Borrelia* isolates associated with Lyme disease and were found to be sensitive and specific. All Lyme disease isolates tested were amplification positive. These primers allow for the rapid species identification of Lyme disease isolates.”

<http://www.ncbi.nlm.nih.gov/pubmed/1280643> <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC270537/>

Steere’s Treatment DNA/RNA Shell Game, synovial (knees) and spinal fluid.

In the first report, knees, Steere finds treatment fails. He is using 3 *OspA* probes and one **16S rDNA** probe. He finds about 1/3 of the patients were positive with these probes after treatment and concludes longer than 30 days is necessary, as the longer the treatment, the lower the frequency of DNA-positive cases. After he makes these claims, he never again says treatment fails, only that everyone is cured and there are no positive cases after treatment by signing the “Guidelines”:

[N Engl J Med.](#) 1994 Jan 27;330(4):229-34.

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

[Nocton JJ1](#), [Dressler F](#), [Rutledge BJ](#), [Rys PN](#), [Persing DH](#), [Steere AC](#).

”BACKGROUND:

”*Borrelia burgdorferi* is difficult to detect in synovial fluid, which limits our understanding of the pathogenesis of Lyme arthritis, particularly when arthritis persists despite antibiotic therapy.

”METHODS:

”Using the polymerase chain reaction (PCR), we attempted to detect *B. burgdorferi* DNA in joint-fluid samples obtained over a 17-year period. The samples were tested in two separate laboratories with four sets of primers and probes, three of which target plasmid DNA that encodes outer-surface protein A (*OspA*).

”RESULTS:

”*B. burgdorferi* DNA was detected in 75 of 88 patients with Lyme arthritis (85 percent) and in none of 64 control patients. Each of the three *OspA* primer-probe sets was sensitive, and the results were moderately concordant in the two laboratories ($\kappa = 0.54$ to 0.73). Of 73 patients with Lyme arthritis that was untreated or treated with only short courses of oral antibiotics, 70 (96 percent) had positive PCR results. In contrast, of 19 patients who received either parenteral antibiotics or long courses of oral antibiotics ($> \text{or} = 1$ 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>

And

J Infect Dis. 1996 Sep;174(3):623-7.

Detection of *Borrelia burgdorferi* DNA by polymerase chain reaction in cerebrospinal fluid in Lyme neuroborreliosis.

Nocton JJ1, Bloom BJ, Rutledge BJ, Persing DH, Logigian EL, Schmid CH, Steere AC.

"A polymerase chain reaction (PCR) assay that detects *Borrelia burgdorferi* DNA in cerebrospinal fluid (CSF) was evaluated as a diagnostic test for acute or chronic Lyme neuroborreliosis. In one laboratory, 102 samples were tested blindly, and 40 samples were retested in a second laboratory. In the first laboratory, *B. burgdorferi* DNA was detected in CSF samples in 6 (38%) of 16 patients with acute neuroborreliosis, 11 (25%) of 44 with chronic neuroborreliosis, and none of 42 samples from patients with other illnesses. There was a significant correlation between PCR results and the duration of previous intravenous antibiotic therapy. The overall frequency of positive results was similar in the second laboratory, but concordance between the laboratories and among primer-probe sets was limited because many samples were positive with only one primer-probe set. Thus, PCR testing can sometimes detect *B. burgdorferi* DNA in CSF in patients with acute or chronic neuroborreliosis, but with current methods, the sensitivity of the test is limited.

"...Previous studies using PCR to detect *B. burgdorferi* DNA in cerebrospinal fluid (CSF) have been done primarily in small numbers of patients with early, acute neuroborreliosis [5-10]. In these studies, which have used several different probes and techniques, the PCR test had sensitivities of 24%-100%. We previously reported that a PCR assay targeting outer surface protein A (OspA) DNA is highly sensitive and specific for the detection of *B. burgdorferi* DNA in joint fluid of patients with Lyme arthritis [11]. We report here on the evaluation of this assay as a diagnostic test for the detection of spirochetal DNA in CSF in a large number of patients with **acute or chronic Lyme**

neuroborreliosis.”

<http://www.ncbi.nlm.nih.gov/pubmed/8769624> <http://jid.oxfordjournals.org/content/174/3/623.long>

From this report:

PCR assay. CSF samples from case and control patients were processed simultaneously in a blinded manner, as described [11]. Briefly, DNA was isolated from 100 μ L of CSF, and the DNA extract was resuspended in 30 μ L of ultrapure water. A 5- μ L aliquot from this suspension was amplified with primer-probe set 1, which targets base pairs 788–943 at the 3' end of the 50-kb plasmid of *B. burgdorferi* that encodes *OspA* [11]. This primer-probe set detects most strains of *B. burgdorferi* from New England. A second 5- μ L aliquot was amplified with primer-probe set 2, which targets base pairs 149–343 at the 5' end of the *ospA* gene [11]. This set detects all North American and European isolates tested to date, with the exception of rare natural isolates that lack the 50-kb plasmid. Amplification consisted of 45 cycles of denaturation at 94°C for 45 s, annealing at 50°C for 45 s, and extension at 72°C for 1 min. The cycles were preceded by a 4-min phase at 94°C and followed by a final 7-min phase at 72°C. Amplified products were resolved by 4% agarose gel electrophoresis and blotted onto nylon membranes, which were hybridized with the appropriate ³²P–5' end-labeled internal oligonucleotide probe [11] and exposed to film for 4–72 h.

You can see that Steere does not use the 16S RNA probe in this Neuroborreliosis assessment, whereas he had before in knees and found more than a third of the patients were positive after treatment. The *OspA* gene was seen more frequently in those arthritis knees patients probably due to the exosomes or blebs, which contain DNA (Dorward, 1990, PMID: 16348232). And as shown by Pachner, above, using a human neuroinvasive strain (N40 was taken from the spinal fluid of a human patient), the plasmids change. It is quite evident that Steere does not want to find Neuroborreliosis. He referenced Garon and Marconi's work and recommendations for using the 16S probe to assure the Dearborn strains were *burgdorferi*. Steere used DNA he knew would not likely be found in human brains to falsely show that people are not infected with spirochetes, the CDC's goal from the beginning. Not every strain of *Borrelia* has *OspA*, and one should use DNA that detects the GENUS, or at least more of the common species, flagellin or the spacer DNAs.

The CDC deployed Allen Steere in the first place – a rheumatologist -- to manage a vector-borne, neurologic disease? This never made any sense unless the CDC wanted to spin it from the beginning, once they found out, “Oops (Plum Island).” Nevertheless, here in these two reports, despite the shell game, Steere found treatment fails at least a third of the time in both knees and spinal fluid. Yet, he never mentioned this again and signed the “IDSA Guidelines” that state that spirochetes do not persist after 2 weeks to 30 days.

Surely by the late 1980s, this Cabal knew antibiotic treatment failed because spirochetes cause damage via activating Epstein-Barr and the like, and that no spirochetal infection has ever been “curable,” or eradicable. **All of their nonsense is about the *OspA* vaccines causing the same immunosuppression-and-not-arthritis disease and that they performed the Dearborn stunt to hide this fact.**

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

The IDSA “Guidelines” are based on the Dearborn-Falsified case definition, Klemmpner’s 2001, bogus “re-treatment” “study” where Klemmpner 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, Klemmpner 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 “Klemmpner” in the above report. You will see the basis of the “Guidelines” is Klemmpner’s research-fraud article on the non-retreatment of 2/3rds of his victims, using the falsified case definition and a bogus psychiatric check-list instead of the Cabal’s own valid biomarkers.

Notice that the authors say:

”Another study similarly was unsuccessful in recovering *B. burgdorferi* from the blood of 12 patients with chronic post-Lyme disease symptoms, using both conventional and hypertonic media (M.S.K., unpublished data) [288]. The latter study also cultured 128 CSF specimens for *B. burgdorferi* and evaluated blood specimens and CSF specimens by PCR. None of the 843 specimens tested in total was either culture or PCR positive [288, 289]. Therefore, the most plausible explanation for the positive results using the novel blood culture method reported by a single group of investigators [303] is that the microscopic findings were not, in fact, due to *B. burgdorferi*.

“In another study, *B. burgdorferi* DNA was detected by PCR in urine samples of 74.2% of 97 United States patients who were diagnosed as having “chronic Lyme disease” and who were previously treated with antibiotics for extended periods of time [306]. Few additional details were provided by the authors as to the characteristics of the patient population. ****Because the authors did not sequence the amplicons to confirm their identity, the results should be regarded as questionable in the absence of confirmation by other investigators.*”***

Klemmpner in his bogus non-retreatment article (Ref 288) used bogus OspA (which were not listed, one had to dig and find he used OspA) primers, *and this entire criminal gang is guilty of the same thing – not sequencing for Borrelia DNA or using proper primers* (Wormser in Missouri, for instance).

Wormser just used bogus probes of DNA not necessarily expected to be there (*burgdorferi* Fla and a specific *lonestari* enzyme, knowing there were plenty of other borrelia in ticks in Missouri, as shown above by Telford, Schoen, and Barbour).

This proves Wormser, et al, know they should have done the same thing. **All their own bogus articles have to be retracted in addition to the Cabal’s prosecution.**

And the 2001 “Guidelines” signers:

<http://www.guideline.gov/content.aspx?id=9537>

Guideline Title

Infectious Diseases Society of America practice guidelines for clinical assessment, treatment and prevention of Lyme disease, human granulocytic anaplasmosis, and babesiosis.

Bibliographic Source(s)

Wormser GP, Dattwyler RJ, Shapiro ED, Halperin JJ, Steere AC, Klemperer MS, Krause PJ, Bakken JS, Strle F, Stanek G, Bockenstedt L, Fish D, Dumler JS, Nadelman RB. The clinical assessment, treatment, and prevention of lyme disease, human granulocytic anaplasmosis, and babesiosis: clinical practice guidelines by the Infectious Diseases Society of America. Clin Infect Dis. 2006 Nov 1;43(9):1089-134. PubMed

Guideline Status

This is the current release of the guideline.

This guideline updates a previous version: Wormser GP, Nadelman RB, Dattwyler RJ, Dennis DT, Shapiro ED, Steere AC, Rush TJ, Rahn DW, Coyle PK, Persing DH, Fish D, Luft BJ. Practice guidelines for the treatment of Lyme disease. Clin Infect Dis 2000 Jul;31(Suppl 1):1-14.

The guideline was reaffirmed for currency by the developer in 2010.

What else can you say? The Cabal and the CDC do not want anyone to discover a Borreliosis infection much less treat it. If you understand that the disease is far more devastating than a common infection with a common bacteria – it’s functionally and physiologically like Post Sepsis Syndrome -, there really is no treatment for it at this time. But there is the threat that these walking cesspools of disease called humans with Lyme or Chronic Fatigue Syndrome might just be the cauldron from which emerges the next pandemic. There are a million such new cases of Tick Bite Sepsis (Disability) per year in the USA alone.

It’s going to be pretty bad Karma to abuse and neglect (Deprivation of Rights Under Color of Law) the very sick, one way or another. Either by natural disaster or the other nations will decide to boycott American businesses and especially ban the CDC staff from traveling to their countries. Are they not criminal terrorists? Who else but the NAZIs and the Japanese during World War II repeatedly and blatantly experimented on races and or with bioweapons?

Right. The CDC.

Tuskegee.

Guatemala.