

Chapter 9 – “FALSE” = the claim that OspA antibodies prevent spirochetes by disinfecting ticks.

Second of all, it is known that spirochetes stop expressing OspA at 37 C and instead express OspC, since OspC is a ligand for something on red blood cells. So, OspA was the wrong choice for a vaccine and this was known for a long time.

First of all, OspA is Pam3Cys or a triacyl lipoprotein, which means, as a fungal endotoxin, never could have been a vaccine.

The Yale, Alan Barbour, and SmithKline Cabal Claim: “Anti-OspA antibodies kill spirochetes in the tick”

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1988). The white-footed mouse (*Peromyscus leucopus*), however, is a highly competent reservoir host. It is infected easily by nymphs, is highly infectious for larvae, and remains infectious for life (Levine et al. 1985, Donahue et al. 1987, Davidar et al. 1989).

Vaccination with *B. burgdorferi* outer surface protein A (OspA) has been shown to protect experimental animals and humans from infection (Fikrig et al. 1990, Sigal et al. 1998, Steere et al. 1998). OspA is expressed when the spirochete is in the tick gut (Fikrig et al. 1992b, Burkot et al. 1994, de Silva et al. 1996). A vaccinated host produces anti-OspA antibodies that will kill the spirochetes in the gut of the tick and prevent migration to the salivary glands (de Silva et al. 1996, Nowling and Philipp 1999). Thus, the vaccine blocks transmission in the tick, and the spirochetes never enter the host. Furthermore, the vaccine can clear infection within infected ticks feeding upon immunized hosts (Fikrig et al. 1992b, de Silva et al. 1996).

<https://www.ncbi.nlm.nih.gov/pubmed/12653137>

And in....

the first two doses was 68% and 49% for ImuLyme and LYMERix, respectively. In the second year, following the 12 month booster vaccination, efficacy rose to 92% for ImuLyme and 76% for LYMERix. Overall, OspA-based vaccines appeared to be safe and well tolerated during phase III studies. Side effects were not remarkable and consisted of those typically associated with vaccination (e.g., minor soreness, redness, swelling at the injection site, low grade fever, chills). FDA approval was given for the LYMERix vaccine at the end of 1998; no application for ImuLyme approval was submitted (Nigrovic and Thompson, 2007). The LYMERix vaccine was subsequently recommended by the CDC Advisory Committee on Immunization Practices for use in populations at high risk for contracting Lyme disease (Anonymous, 1999).

OspA is a linear-plasmid-encoded 28kDa lipoprotein (Howe et al., 1986; Barbour and Garon, 1988) that structurally consists of two globular domains composed almost entirely of beta-sheets that are connected by a unique single-layer beta-sheet (Fig. 52.5) (Li et al., 1997; Makabe et al., 2006). Crystallographic structures have indicated that there is a possible ligand-binding site formed in the C-terminal globular domain (Li et al., 1997; Makabe et al., 2006). OspA is expressed in the tick vector (Fingerle et al., 1995; Leuba-Garcia et al., 1998), and may be responsible for adhesion of spirochetes to the wall of the tick midgut (Pal et al., 2004a). During transit from the midgut to the tick salivary glands, OspA expression is down-regulated; thus, spirochetes entering the mammalian

host do not express the OspA protein (Schwan et al., 1995). Vaccination with OspA induces an anti-OspA antibody response that blocks transmission of spirochetes from the tick to the mammalian host by killing or immobilizing the spirochetes within the tick midgut (de Silva et al., 1996, 1999). Since the spirochetes are, in large part, killed prior to entry into the mammalian host, this mechanism of action largely precludes the elicitation of anamnestic responses. The effectiveness of the vaccine is thus dependent on the presence of sufficient levels of circulating anti-OspA antibody. The requirement for high circulating antibody titer likely explains the need for yearly booster immunizations to maintain efficacy. During the second year of the ImuLyme trial, for example, there was no difference in protection between unvaccinated patients and vaccinated patients that had received the two initial vaccinations, but not the 12 month booster (Sigal et al., 1998). The incomplete (49% in year one; 76% in year two) protection provided by the LYMERix vaccine, the lack of safety and efficacy data for children, and apparent requirement for frequent booster immunizations to maintain protection are all factors that suggest that the development of new and improved Lyme disease vaccines are needed.

Within a year of its introduction, LYMERix was being anecdotally associated with development of arthritis. The possible association between the vaccine and these adverse events was well publicized, particularly by Lyme disease advocacy groups. Lawsuits, including a class-action, alleging vaccine-related harm

<https://www.amazon.com/gp/search?index=books&linkCode=qs&keywords=9780080919027>

That (above) was what they claimed publicly. “The OspA antibodies killed spirochetes in the tick,” but there was never any evidence for that; there were only more “bogus articles.”

The following 1995 report by the CDC and NIH (Piesman and Schwann) says that OspA is not expressed at 37 C (human body temp) but that OspC is upregulated. Therefore the better choice - if we must have an Osp toxin as a vaccine - would have been OspC to prevent dissemination throughout the body should a tick regurgitate its gut contents into a mammal.

Proc Natl Acad Sci U S A. 1995 Mar 28;92(7):2909-13.

Induction of an outer surface protein on Borrelia burgdorferi during tick feeding.

Schwan TG1, Piesman J, Golde WT, Dolan MC, Rosa PA.

Author information

Lyme disease spirochetes, *Borrelia burgdorferi* sensu lato, are maintained in zoonotic cycles involving ticks and small mammals. In unfed ticks, the spirochetes produce one outer surface protein, OspA, but not OspC. During infection in mammals, immunological data suggest that the spirochetes have changed their surface, now

expressing OspC but little or no OspA. **We find by in vitro growth experiments that this change is regulated in part by temperature; OspC is produced by spirochetes at 32-37 degrees C but not at 24 degrees C.** Furthermore, spirochetes in the midgut of ticks that have fully engorged on mice now have OspC on their surface. Thus two environmental cues, an increase in temperature and tick feeding, trigger a major alteration of the spirochetal outer membrane. This rapid synthesis of OspC by spirochetes during tick feeding may play an essential role in the capacity of these bacteria to successfully infect mammalian hosts, including humans, when transmitted by ticks.

<https://www.ncbi.nlm.nih.gov/pubmed/7708747>

Who quoted that article (373 citations as of this writing):

https://www.ncbi.nlm.nih.gov/pubmed?linkname=pubmed_pubmed_citedin&from_uid=7708747

Erol **Fikrig** and Richard Flavell (owner of LYMERix), Durland Fish, Justin Radolf, Sam **Telford**, Allen Steere, Linda Brockenstedt at Yale, **Alan Barbour** (owner of the ImmuLyme OspA patent), Gary Wormser, Dave Persing, Steve Malawista, CDC officer Barbara Johnson (ran the Dearborn stunt and is a multi-patent owner with SmithKline in Europe),... just about everyone in the RICO Cabal, the ALDF.com.

‘Same ones who said OspA was a vaccine. The Cabal. All the main perps.

Here Yale allegedly shows OspA antibodies disinfect ticks using a bogus method, rather a method that uses fluorescing anti-OspC antibodies and then some culture or DNA method (see the Primers Shell Game Charge Sheet) such as *Borrelia* flagellin DNA or a spacer gene like 16S or 23S RNA to see if the animals did not get Lyme after the rOspA injection, followed by infected tick attachment.

[J Exp Med.](#) 1996 Jan 1;183(1):271-5.

***Borrelia burgdorferi* OspA is an arthropod-specific transmission-blocking Lyme disease vaccine.**

[de Silva AM1](#), [Telford SR 3rd](#), [Brunet LR](#), [Barthold SW](#), [Fikrig E](#).

Author information

Borrelia burgdorferi, the spirochetal agent of Lyme disease, is transmitted by Ixodes ticks. A vaccine based on *B. burgdorferi* outer surface protein (Osp) A protects mice from spirochete infection. Here we report on the expression of OspA on spirochetes inside engorging ticks and relate OspA expression to antispirochetal immunity. Spirochetes in the gut of unfed nymphal ticks were stained by an OspA antibody, whereas in feeding ticks, the majority of spirochetes in the gut and salivary glands did not stain with the antibody. Thus, OspA was not expressed on most spirochetes during transmission from the vector to the vertebrate host. To examine the mechanism of protection afforded by OspA antibody, mice were passively immunized with OspA antibody at different times relative to tick attachment. When OspA antibody was administered to mice before or at the time of tick attachment, spirochetal development events in the vector, such as growth and salivary gland invasion, were blocked and the mice were protected from *B. burgdorferi* infection. When OspA antibody was administered to mice 48 h after tick attachment, spirochetes persisted in the nymphs and the mice were not protected despite the presence of circulating antibodies in the host as well as in the tick blood meal. Thus, OspA immunity appears to be effective only during a narrow window time at the beginning of the blood meal when antibodies bind to OspA-expressing spirochetes in the tick gut and block transmission from the vector to the host.

<https://www.ncbi.nlm.nih.gov/pubmed/8551231>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2192397/>

“DETECTION METHOD:

”Each slide was dipped in acetone for 5 min before staining with an anti-*B. burgdorferi* FITC-conjugated rabbit polyclonal antibody or polyclonal rabbit sera against OspA or OspB.”

(Knowing OspA is no longer going to be expressed, at 37C or will select for variants of OspA or B.)

“After nymphal tick attachment, spirochete transmission requires , (approx) 48 h, and during this time period, *B. burgdorferi* multiply and cross the gut epithelial barrier into the hemolymph, disseminate to the salivary glands, and infect the host via tick saliva (2, 3, 12).

”Our results document that during tick engorgement, OspA, which is abundantly expressed on spirochetes in unfed ticks, is no longer expressed on the majority of *B. burgdorferi* in the vector. The loss of OspA probably begins ~ 24h after tick attachment, since spirochetes resistant to OspA antiserum were first detected at this time.

“The absence of OspA appears to continue even after the spirochetes enter the mammalian host because mice were infected when OspA antiserum was administered 48 and 72 h after tick attachment (Table 2).

“A recent report demonstrated that OspC is absent from the spirochetes in unfed ticks, and is present in feeding ticks and infected mice (13). **During tick engorgement, we observed that spirochetes in the gut and salivary glands that do not express OspA react with OspC antiserum (data not shown).**

“Thus, as spirochetes move from the vector, many appear to be "changing their coat" by, at least in part, differential expression of OspA and OspC. During engorgement, temperature shifts and biochemical changes in the tick may directly or indirectly influence Osp expression (13, 14).

”The loss of OspA expression by spirochetes during transmission has revealed the mechanism by which the OspA vaccine protects vertebrate hosts: antibody binds to the OspA-expressing *B. burgdorferi* in the tick gut and prevents their replication and subsequent dissemination to the salivary glands and, ultimately, to the vertebrate host. This is the first example of a vaccine undergoing human clinical trials that protects by blocking transmission from the vector. “

Um, no, the loss of OspA expression happens at 37C and antibodies cause the spirochete to change surface proteins (the “data not shown” previously mentioned in that report), and relapsing fever spirochetes are quite famous for this, which is why they are called relapsing fever spirochetes - they undergo “selection pressure” as Fikrig wrote about himself a year earlier:

*Selection of variant *Borrelia burgdorferi* isolates from mice immunized with outer surface protein A or B.* *Infect Immun.* 1995 May;63(5):1658-62.

*Selection of variant *Borrelia burgdorferi* isolates from mice immunized with outer surface protein A or B.* *Fikrig E1, Tao H, Barthold SW, Flavell RA.*

Author information

A nonclonal population of *Borrelia burgdorferi* N40 (passage 3) that survived protective immunity following challenge inoculation of outer surface protein (Osp) A- or B-hyperimmunized mice were characterized for the molecular basis of evasion of immunity. Two of six *B. burgdorferi* isolates, cultured from OspA-immunized mice, had antigenic diversity in the carboxyl terminus of OspA and did not bind to the protective OspA monoclonal antibody designated IXDII. However, OspA-immunized mice challenged with

these variants were fully protected. Moreover, *B. burgdorferi* isolates with a point mutation in ospB, which results in a truncated OspB that does not bind to protective OspB monoclonal antibody 7E6C, were frequently enriched after infection of OspB-immunized mice. These studies suggest that the incomplete efficacy of an OspA- or OspB-based vaccine may be partly due to immunomediated in vivo selective pressure, resulting in the persistence of some spirochetes that do not bind to protective antibodies.

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

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC173206/pdf/631658.pdf>

From the full text:

This report describes the ability of OspA and OspB antibodies to cause the in vivo selection of *B. burgdorferi* organisms with subtle genetic alterations that result in the expression of OspA or OspB which do not bind to, or weakly bind with, antibodies that are protective in nature. These data suggest a potential reason for the lack of complete efficacy of an Osp-based Lyme disease vaccine. Over extended periods of time, the administration of an OspA- or OspB-based vaccine to hosts that are involved in the natural life cycle of the spirochete may result in the expansion of variant *B. burgdorferi* isolates within ticks at a higher frequency than would normally be found in the general population. If this selection pressure was to be maintained, the number of variant spirochetes could rise to a sig-

Fikrig says, above): “This report describes the ability of OspA and OspB antibodies to cause the in vivo selection of *B. burgdorferi* organisms with subtle genetic alterations that result in the expression of OspA or OspB which do not bind to, or weakly bind with, antibodies that are protective in nature. These data suggest a potential reason for the lack of complete efficacy of an Ospbased Lyme disease vaccine. Over extended periods of time, the administration of an OspA- or OspB-based vaccine to hosts that are involved in the natural life cycle of the spirochete may result in the expansion of variant *B. burgdorferi* isolates within ticks at a higher frequency than would normally be found in the general population.”

Fikrig says OspA vaccination does nothing except to cause spirochetes to change surface antigens, which is what *Borreliae* do for a living. That is why they are called relapsing fever organisms. This antigenic variation is the nature of the relapse.

Here, next, is a scientifically DNA method owned by Fikrig and developed and validated in 1991 that he could have used to assess whether or not OspA antibodies kill spirochetes in ticks (or as the recombinant protein to show whether or not people got Lyme after being vaccinated, or whether they have Lyme at all, really) that he could have used to assess the post-OspA vaccinated, then infected animals/humans to see if OspA antibodies prevented spirochetes (or an antibody method based on this):

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

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

[Berland R1](#), [Fikrig E](#), [Rahn D](#), [Hardin J](#), [Flavell RA](#).

Author information

The earliest humoral response in patients infected with *Borrelia burgdorferi*, the agent of Lyme disease, is directed against the spirochete's 41-kDa flagellar antigen. In order to map the epitopes recognized on this antigen, 11 overlapping fragments spanning the flagellin gene were cloned by polymerase chain reaction and inserted into an *Escherichia coli* expression vector which directed their expression as fusion proteins containing glutathione S-transferase at the N terminus and a flagellin fragment at the C terminus. Affinity-purified fusion proteins were assayed for reactivity on Western blots (immunoblots) with sera from patients with late-stage Lyme disease. The same immunodominant domain was bound by sera from 17 of 18 patients. This domain (comprising amino acids 197 to 241) does not share significant homology with other bacterial flagellins and therefore may be useful in serological testing for Lyme disease.

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

And here is Sam Telford, as shown in the previous charge sheets (Primers Shell Game), being well aware of how to use DNA and RNA primers to locate species:

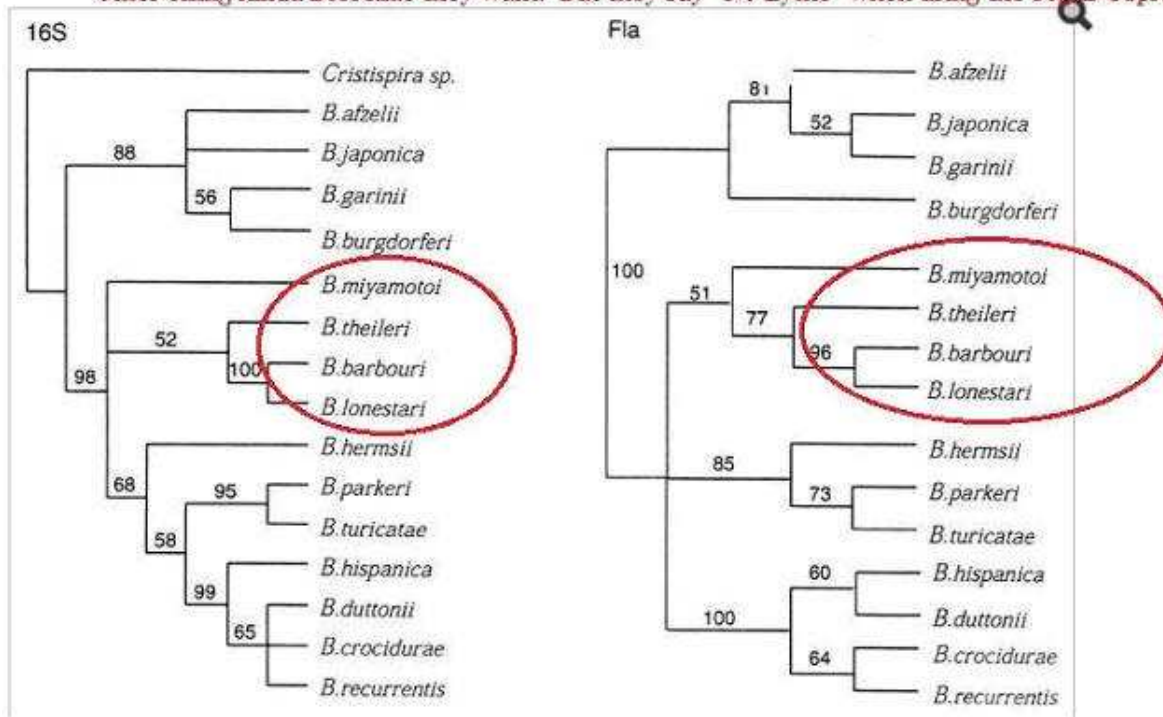
J Clin Microbiol. 2001 Feb;39(2):494-7.

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

Next, in 1990 Sam Telford was looking for spirochetes using an **OspA GENE**, meaning he knows how to pharm for species with a better method than immunofluorescing antibodies:

J Clin Microbiol. 1990 Mar;28(3):566-72.

Detection of *Borrelia burgdorferi* infection in *Ixodes dammini* ticks with the polymerase chain reaction.

Persing DH1, Telford SR 3rd, Spielman A, Barthold SW.

Author information

The polymerase chain reaction (PCR) was used to amplify DNA sequences of the etiologic agent of Lyme disease, *Borrelia burgdorferi*, and was applied to the detection of the spirochete in its tick vector. The target for PCR amplification was the **OSP-A gene** of strain B31; analysis of isolates from different geographical areas indicated that this gene could be used to identify most North American isolates. These methods were extended to the analysis of colony-derived and field-collected *Ixodes dammini*. OSP-A-specific sequences were identified in 15 of 15 colony-derived nymphal ticks that had fed previously on an infected animal; no such amplification products were detected in 8 control ticks. Segregated midgut tissues of field-collected adult and nymphal ticks from Nantucket Island, Mass., and the Crane Reserve, Ipswich, Mass., were examined by both direct fluorescent-antibody (DFA) staining and PCR. The DFA technique identified 16 infected ticks of 30 paired specimens; 15 of these specimens were positive by PCR. One specimen was positive by PCR that was DFA negative. Both live whole ticks and desiccated dead specimens were suitable for this analysis. Because only five ticks are suitable for DFA analysis, the use of PCR may extend the range of specimens that can be analyzed for the presence of the Lyme spirochete. <https://www.ncbi.nlm.nih.gov/pubmed/1969867>

So, Telford, Barbour, Fish, and Fikrig are well aware of real methods to determine if OspA antibodies killed or disinfected spirochetes in ticks and if OspA vaccinated animals ever then became infected with spirochetes.

OspA did not prevent spirochetes in monkeys:

Vaccine. 1997 Dec;15(17-18):1872-87.

The outer surface protein A (OspA) vaccine against Lyme disease: efficacy in the rhesus monkey.

Philipp MT1, Lobet Y, Bohm RP Jr, Roberts ED, Dennis VA, Gu Y, Lowrie RC Jr, Desmons P, Duray PH, England JD, Hauser P, Piesman J, Xu K.

Author information

The efficacy of an outer surface protein A (OspA) vaccine in three different formulations was investigated in the rhesus monkey. The challenge infection was administered using Ixodes scapularis ticks that were infected with the B31 strain of Borrelia burgdorferi. Protection was assessed against both infection and disease, by a variety of procedures. Some of the animals were radically immune suppressed, as an attempt to reveal any putative low level infection in the vaccinated animals. The significant difference found between the spirochaetal infection rates of ticks that had fed on vaccinated vs. control monkeys, lack of seroconversion in the vaccinated animals, and the absence of spirochaetal DNA in the skin of vaccinated animals in the weeks following the challenge, indicate that vaccinated monkeys were protected against tick challenge. **The post-mortem immunohistochemical and polymerase chain reaction analyses, however, suggest that these monkeys may have undergone a low-level infection that was transient.**

<https://www.ncbi.nlm.nih.gov/pubmed/9413097>

This, next, by Barbour, Telford, Fikrig and Fish was also a bogus method - their alleged “xenodiagnosis” with mice and ticks. They used this to assess the presence of spirochetes instead of a DNA method, knowing Borrelia spirochetes undergo antigenic variation under the “selection pressure” of antibodies:

Vector Borne Zoonotic Dis. 2001 Spring;1(1):65-74.

OspA immunization decreases transmission of Borrelia burgdorferi spirochetes from infected Peromyscus leucopus mice to larval Ixodes scapularis ticks.

Tsao J1, Barbour AG, Luke CJ, Fikrig E, Fish D.

Author information

Abstract

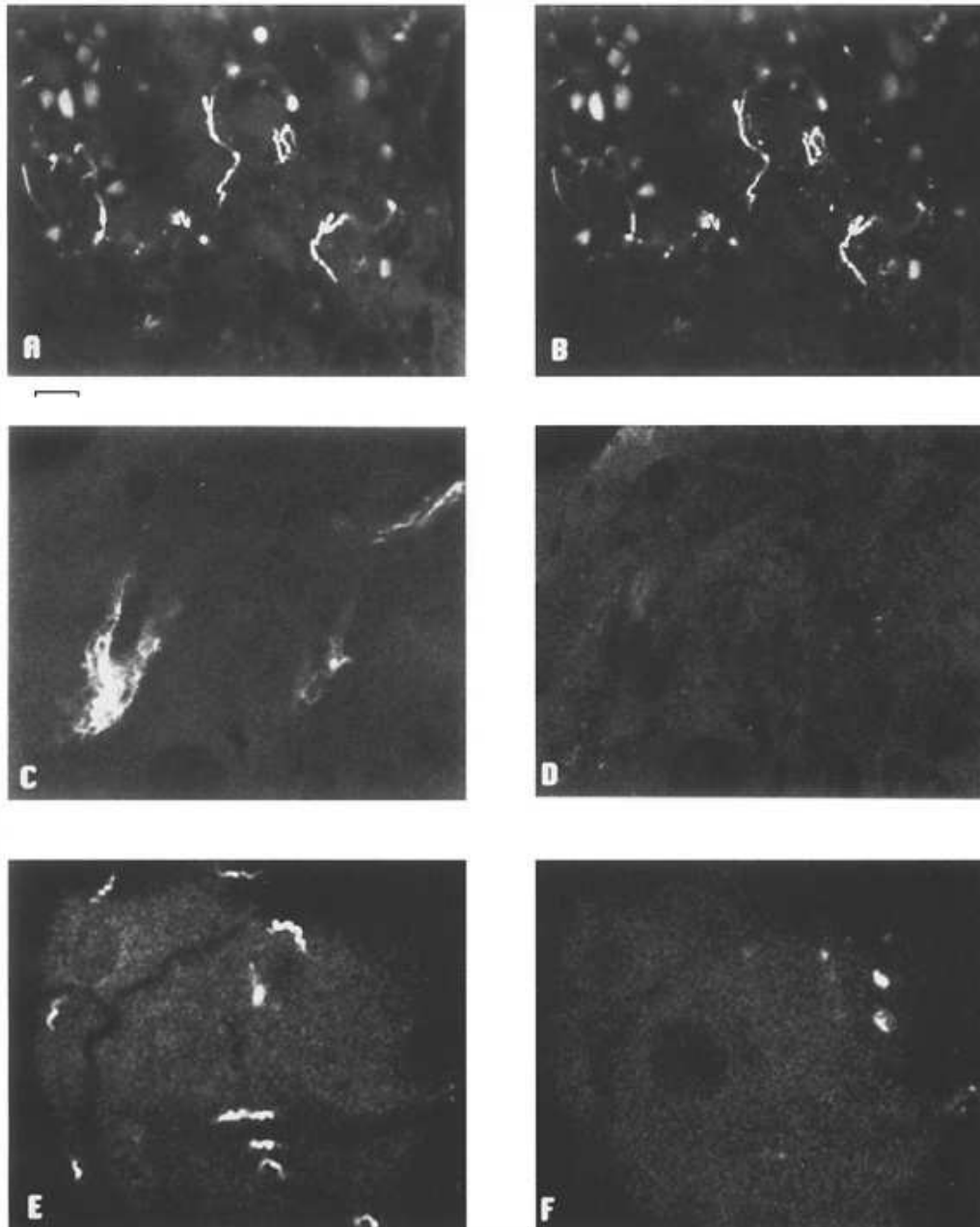
Recombinant outer surface protein A (OspA) vaccination of wild animal reservoirs has potential application for reducing Borrelia burgdorferi transmission in nature and subsequent risk of human infection. As a major reservoir host, the white-footed mouse (Peromyscus leucopus) is a candidate for a vaccination program designed to reduce infection prevalence in vector ticks. In this study we characterized the effect of various levels of immunization with recombinant OspA-glutathione transferase fusion protein on transmission dynamics from infected P. leucopus to larval ticks. Control mice were vaccinated with glutathione transferase alone. All mice were experimentally infected with B. burgdorferi before vaccination. The immune responses of the immunized mice were assessed by enzyme-linked immunosorbent assay for antibodies to OspA. Transmission of B. burgdorferi from infected mice was determined by xenodiagnosis with uninfected larval ticks. Spirochetes in ticks were counted by direct immunofluorescence assay. The concentration of antibody to OspA increased with each OspA vaccination but most markedly after the first and second vaccinations. In comparison with control mice, there was reduced transmission by OspA-vaccinated mice to uninfected ticks. One, two, or three doses of OspA reduced infection prevalence in xenodiagnostic ticks by 48%, 92%, or 99% and the numbers of spirochetes per tick by 84%, 98%, or 99%, respectively. This study suggests that vaccination of P. leucopus

with OspA could reduce transmission to the tick vector in nature despite prior infection of the reservoir host. <https://www.ncbi.nlm.nih.gov/pubmed/12653137>

In the full text article they say:

”Presence of spirochetes in the nymph midgut preparations was determined by DFA (direct immunofluorescent antibody) test using a fluorescein isothiocyanate-conjugated goat anti-*B. burgdorferi* antibody (Kirkegaard-Perry). Each nymph homogenate was overlaid with 0.2 mg of antibody”

Pretending to look for OspA and therefore spirochetes ... knowing OspA will not be expressed due to temperature or selection pressure (AKA antigenic variation), using fluorescing OspA antibodies, Wow.



Fikrig, Fish and Barbour using a bogus method to show no spirochetes. They could have used a DNA method.

Figure 1. Confocal fluorescence images of *B. burgdorferi* in tick organs stained for OspA expression. (A and B) The same field of a gut from an uninfected nymph. (C and D) The same field of a gut from an infected nymph that had fed for 60 h. (E and F) are the same field of a salivary acinus from an infected nymph that had fed for 60 h. A, C, and E display the FITC signal from a rabbit serum raised against whole spirochetes, while B, D, and F display the Texas red signal from an mAb that binds to OspA. OspA antibody staining was readily detected on spirochetes before tick feeding. During engorgement, the majority failed to stain with the OspA antibody. Bar, 12.5 μ m.

<http://online.liebertpub.com/doi/abs/10.1089/153036601750137705>

This, above, is research fraud or a “bogus article” by the criminals Durland Fish, Alan Barbour and Erol Fikrig. Why couldn't they just use a flagellin DNA or 16S DNA method to see if OspA vaccination of a mammal prevented ticks from acquiring spirochetes from said animal (xenodiagnosis)?

J Infect Dis. 1996 Oct;174(4):739-46.

Characterization of the protective borreliacidal antibody response in humans and hamsters after vaccination with a *Borrelia burgdorferi* outer surface protein A vaccine.

Padilla ML1, Callister SM, Schell RF, Bryant GL, Jobe DA, Lovrich SD, DuChateau BK, Jensen JR.

Author information

Abstract

Significant borreliacidal antibody was induced in volunteers and hamsters 60 days after primary and secondary vaccination with high concentrations of recombinant outer surface protein A (rOspA). However, the borreliacidal antibody response waned rapidly. Only 1 person had detectable cidal activity 180 days after vaccination. Similarly, the borreliacidal antibody response waned rapidly in hamsters by week 10 of vaccination. By contrast, the total anti-rOspA antibody response remained elevated in volunteers and hamsters. **When isolates of *Borrelia burgdorferi sensu lato* were incubated in sera from vaccinated humans or hamsters, only the vaccine-specific isolate was killed. These results were confirmed by challenging rOspA-vaccinated hamsters with different isolates of *B. burgdorferi sensu lato*. The results showed that monitoring total rOspA antibody is inappropriate for evaluating the efficacy of an rOspA vaccine.** The rOspA vaccine must be improved to yield comprehensive protection and maintain sustained levels of protective borreliacidal antibodies.

<https://www.ncbi.nlm.nih.gov/pubmed/8843211>

The above report shows OspA vaccination does not prevent spirochetes. It only downregulates that particular version of OspA, if it is even there. You can see the scam: These criminals said “Lyme disease” was only a bad knee caused by OspA autoimmunity. Thence, they have a vaccine that causes the downregulation of OspA in the spirochetes. So, if you don't have antibodies to OspA or T cells that cross react with human tissue from antibodies against spirochetes, you don't have “Lyme disease.”

(Yet, antibodies against OspA and B were left out of the Dearborn standard – because that Dearborn test was to be used AFTER rOspA was on the mass market – see the other charge sheets).

All the lies – past and present -- about Lyme and LYMERix or OspA as a vaccine or “guidelines” or “case definitions” have to do with **preventing only jail** for these criminals. It's all pretense. Pretense is a legal word meaning fraud.

Here is Alan Barbour talking about how you cant have a vaccine against Lyme due to antigenic variation causing “mutants” which is redundant language since antigenic variation to avoid antibodies is what relapsing fever spirochetes (all *Borreliae*) do for a living:

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

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

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

Author information

We used polyclonal antisera and monoclonal antibodies (mAbs) to inhibit the growth of clonal populations of two strains of *Borrelia burgdorferi*, the Lyme disease agent, and thereby select for antibody-resistant mutants. mAbs were directed at the outer membrane proteins, OspA or OspB. Mutants resistant to the growth-inhibiting properties of the antibodies were present in the populations at frequencies ranging from 10^{-5} to 10^{-2} . The several escape variants that were examined were of four classes. Class I mutants were resistant to all mAbs; they lacked OspA and OspB and the linear plasmid that encodes them. Two other proteins were expressed in larger amounts in class I mutants; mAbs to these proteins inhibited the mutant but not the wild-type cells. Class II mutants were resistant to some but not all mAbs; they had truncated OspA and/or OspB proteins. Class III mutants were resistant only to the selecting mAb; they had full-length Osp proteins that were not bound by the selecting antibody in Western blots. In two class III mutants resistant to different anti-OspA mAbs, missense mutations were demonstrated in the ospA genes. Class IV mutants were likewise resistant only to selecting antibody, but in this case the selecting antibody still bound in Western blots.

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

~~basis for the escape mutations has not been determined.~~

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”

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There ^ is Alan Barbour talking about how you can create antigenic variation in spirochetes with antibodies. It's a thing. Anyone can do it. He also says there is true antigenic variation of OspA and B within a strain. This means using a fluorescing OspA antibody method to claim OspA vaccination prevented spirochetes is blatantly false or RESEARCH FRAUD. Barbour owns the OspA vaccine patent which was manufactured by Pasteur-Connaught, the results of which were falsified the same as with LYMERix. OspA as a vaccine especially from one strain does not do anything. Here is clearly admits it.

No one has ever actually shown vaccination prevents spirochetes, to date. Most people will not go for the idea of turning their bodies into walking canisters of spirochete antigenic variationators.

Now, importantly, since at least 1997, Alan Barbour said it was OspC and its ilk that was responsible for the dissemination of spirochetes to the brain, et al:

[Infect Immun.](#) 1997 Aug;65(8):3352-60.

Immunologic and genetic analyses of VmpA of a neurotropic strain of *Borrelia turicatae*.

[Cadavid D1](#), [Pennington PM](#), [Kerentseva TA](#), [Bergström S](#), [Barbour AG](#).

Author information

In mice infected with serotype A but not serotype B of the relapsing fever spirochete *Borrelia turicatae*, early invasion of the brain occurs. Serotypes A and B are further distinguished by the abundant surface protein they produce: VmpA and VmpB, respectively. Western blotting with monoclonal antibodies, one-dimensional peptide mapping, and partial amino acid sequencing demonstrated regions of the VmpA protein that differed from VmpB. Oligonucleotide primers based on the partial amino acid sequences of unique regions were used to amplify a portion of the VmpA gene (*vmpA*) by PCR, and the product was used as a probe in Southern blot and Northern blot analyses. These experiments showed that (i) expression of the *vmpA* sequence was determined at the level of transcription and (ii) the *vmpA* sequence was in two locations in serotype A and one location in serotype B. The *vmpA* gene at the expression-linked locus of serotype A was cloned and sequenced. An open reading frame would encode a polypeptide of 214 amino acids. The polypeptide expressed by *Escherichia coli* was bound by VmA-specific but not VmpB-specific antibody. Primer extension analysis identified a consensus sigma70-type promoter for *vmpA* at the expression locus. **Phylogenetic analysis revealed that VmpA is homologous to small Vmp (Vsp) proteins of *B. hermsii* and to OspC proteins of *B. burgdorferi*. These findings indicate that a function of the Vsp-OspC family of proteins of *Borrelia* spp. may be differential localization in organs, including the brain, during infection.**

<https://www.ncbi.nlm.nih.gov/pubmed/9234797>

OspA does not prevent spirochetes; it was the wrong antigen. Any Lyme vaccines should have at least been multiple types of OspC to prevent dissemination into the brain, lymph nodes, bone marrow and organs. As shown in the other charge sheets, it may tolerize dogs against arthritis - Gary Wormser also shows OspA causes this same tolerance/immunosuppression in dogs:

[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](#).

Author information

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

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

Next, the human OspA vaccine trials did not assess for protection against spirochetes, they only made the claim that the vaccines produced OspA antibodies and assessed for that. They also used the Dearborn method, threw out all the neurologic cases (85%) calling them “Unconfirmed Lyme” and then later claimed they actually could not even read their Western Blots on OspA vaccinated humans:

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

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

Steere AC1, Sikand VK, Meurice F, Parenti DL, Fikrig E, Schoen RT, Nowakowski J, Schmid CH, Laukamp S, Buscarino C, Krause DS.

Author information

BACKGROUND:

The risk of acquiring Lyme disease is high in areas in which the disease is endemic, and the development of a safe and effective vaccine is therefore important.

METHODS:

We conducted a multicenter, double-blind, randomized trial involving 10,936 subjects who lived in areas of the United States in which Lyme disease is endemic. Participants received an injection of either recombinant *Borrelia burgdorferi* outer-surface lipoprotein A (OspA) with adjuvant or placebo at enrollment and 1 and 12 months later. In cases of suspected Lyme disease, culture of skin lesions, polymerase-chain-reaction testing, or serologic testing was done. Serologic testing was performed 12 and 20 months after study entry to detect asymptomatic infections.

RESULTS:

In the first year, after two injections, 22 subjects in the vaccine group and 43 in the placebo group contracted definite Lyme disease ($P=0.009$); vaccine efficacy was 49 percent (95 percent confidence interval, 15 to 69 percent). In the second year, after the third injection, 16 vaccine recipients and 66 placebo recipients contracted definite Lyme disease ($P<0.001$); vaccine efficacy was 76 percent (95 percent confidence interval, 58 to 86 percent). The efficacy of the vaccine in preventing asymptomatic infection was 83 percent in the first year and 100 percent in the second year. Injection of the vaccine was associated with mild-to-moderate local or systemic reactions lasting a median of three days.

CONCLUSIONS:

Three injections of vaccine prevented most definite cases of Lyme disease or asymptomatic *B. burgdorferi* infection.

<https://www.ncbi.nlm.nih.gov/pubmed/9673298>

<http://www.nejm.org/doi/pdf/10.1056/NEJM199807233390401>

In the text of the report they claim:

Laboratory Methods

Serologic testing was done exclusively by Western blotting (Mardex, San Diego, Calif.), since the standard enzyme-linked immunosorbent assay would be expected to give positive results in patients who had been vaccinated with OspA. The blots were read by experienced technicians according to the CDC criteria¹⁷; reactivity with the vaccine-induced 31-kd band was not reported, so that all investigators remained unaware of the subjects' treatment assignments. Serologic support for the diagnosis of Lyme

And what was that CDC criteria?

17. Recommendations for test performance and interpretation from the Second International Conference on Serologic Diagnosis of Lyme Disease. MMWR Morb Mortal Wkly Rep 1995;44:590-1.

The Dearborn ^^ method, which only detects late Lyme arthritis as you have seen previously.

And here is the CDC officer and Dearborn Stunt participant Alan Barbour OspA / Pam3Cys Triacyl-Lipoprotein (Fungal Endotoxin), TLR2/1 agonist non-vaccine trial:

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

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

[Sigal LH1](#), [Zahradnik JM](#), [Lavin P](#), [Patella SJ](#), [Bryant G](#), [Haselby R](#), [Hilton E](#), [Kunkel M](#), [Adler-Klein D](#), [Doherty T](#), [Evans J](#), [Molloy PJ](#), [Seidner AL](#), [Sabetta JR](#), [Simon HJ](#), [Klempner MS](#), [Mays J](#), [Marks D](#), [Malawista SE](#).

Author information

Erratum in

N Engl J Med 1998 Aug 20;339(8):571.

BACKGROUND:

Lyme disease is a multisystem inflammatory disease caused by infection with the tick-borne spirochete *Borrelia burgdorferi* and is the most common vector-borne infection in the United States. We assessed the efficacy of a recombinant vaccine consisting of outer-surface protein A (OspA) without adjuvant in subjects at risk for Lyme disease.

METHODS:

For this double-blind trial, 10,305 subjects 18 years of age or older were recruited at 14 sites in areas of the United States where Lyme disease was endemic; the subjects were randomly assigned to receive either placebo (5149 subjects) or 30 microg of OspA vaccine (5156 subjects). The first two injections were administered 1 month apart, and 7515 subjects also received a booster dose at 12 months. The subjects were observed for two seasons during which the risk of transmission of Lyme disease was high. The primary end point was the number of new clinically and serologically confirmed cases of Lyme disease.

RESULTS:

The efficacy of the vaccine was 68 percent in the first year of the study in the entire population and 92 percent in the second year among the 3745 subjects who received the third injection. The vaccine was well tolerated. There was a higher incidence of mild, self-limited local and systemic reactions in the vaccine group, but only during the seven days after vaccination. There was no significant increase in the frequency of arthritis or neurologic events in vaccine recipients.

CONCLUSIONS:

In this study, OspA vaccine was safe and effective in the prevention of Lyme disease.

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

<http://www.nejm.org/doi/pdf/10.1056/NEJM199807233390402>

And what did *they* use to show whether or not OspA prevented spirochetes or Lyme?

Case Definitions

Data on all subjects with symptoms that the local investigator decided were consistent with the presence of Lyme disease were reviewed by an independent data and safety monitoring board whose members were unaware of subjects' treatment assignments. The diagnosis was confirmed only if it met the strict CDC case definition,²⁰ which requires the presence of erythema migrans or later manifestations of Lyme disease, with laboratory confirmation in either case. Erythema migrans had to be documented by a photograph or a definitive descriptive clinical note clearly stating the size of the lesion. Laboratory confirmation consisted of proof of seroconversion (from seronegative results at base line to seropositive results at a subsequent analysis). In the absence of laboratory confirmation, the presence of erythema migrans, regardless of the size of the lesion, or of late manifestations of Lyme disease was insufficient for a diagnosis of Lyme disease.

Reference Number 20:

They used the old diagnostic standard, from 1990:

20. Lyme disease. MMWR Morb Mortal Wkly Rep 1990;39(RR-13):19-21.

In the same report they show **when** the vaccine trial started, which was before Dearborn (Oct 1994). But clearly they knew there was a problem with OspA vaccination because Barbour and Fish trashed the victims in 1993, admitting the vaccine Phase I and II trials were underway. This is from the text of the above vaccine trial report (Barbour's OspA patent):

Barbour's phase III OspA trial started before the Dearborn stunt (Oct 1994), which proves his victim-trashing article with Fish was the result of known OspA caused the same Chronic NeuroLyme.

Vaccination Protocol

Before receiving the first injection, all subjects provided written informed consent by signing a document approved by the institutional review board at each participating site and were counseled on Lyme disease and its prevention. A single dose (0.5 ml) of vaccine or placebo was injected intramuscularly into the deltoid. A second dose was administered 30 days after the first dose. The timing of entry into the study was such that both inoculations occurred before the end of May 1994 — before the start of the "Lyme disease season" in the areas included in the study. Subjects who enrolled in the extension study received a single booster dose of vaccine or placebo approximately 12 months after the first dose. All these subjects received the booster dose before the end of May 1995.

And here are Persing and Sigal (one of the vaccine administrators of the above Barbour ImmuLyme OspA vaccine trial report) admitting the Western Blots from OspA vaccination were unreadable:

[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 PJ1](#), [Berardi VP](#), [Persing DH](#), [Sigal LH](#).

Author information

Abstract

Laboratory confirmation of the diagnosis of Lyme disease is based on the detection of an immune response to *Borrelia burgdorferi*. The serodiagnosis of *B. burgdorferi* infection is complex and may be further confounded by the immune response to the recombinant outer surface protein A (OspA) Lyme disease vaccine. To describe how the serological response to the recombinant OspA Lyme disease vaccine affects testing for antibody to *B. burgdorferi*, 240 specimens from 80 study subjects were obtained at defined intervals after recombinant OspA Lyme disease vaccination. Samples were tested by indirect enzyme-linked immunosorbent assay (ELISA), antibody capture enzyme immunoassay (EIA), and Western blotting (WB). After recombinant OspA Lyme disease vaccination, ELISA for 98% of the study subjects revealed reactivity. WB with use of OspA-containing *B. burgdorferi* strains as sources of antigens demonstrated multiple bands. Results of testing with a US Food and Drug Administration-approved WB kit showed homogeneous reactivity in the molecular weight region >30 kDa. Testing with OspA-free strains completely eliminated all vaccine-associated reactivity by both antibody capture EIA and WB.

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

From the full text:

study subjects. 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.

“The manufacturer of the only currently FDA approved (and released) recombinant OspA Lyme 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.”

In other words, not only was OspA a fungal endotoxin (Pam3Cys) that causes immunosuppression, the wrong vaccine choice because OspC is upregulated at 37C (human body temperature) instead, Dearborn a fraud, OspA antibodies never were shown to disinfect ticks, and OspA antibodies never proven to prevent spirochetes,.... here one of the OspA vaccine trial administrators says none of the 2 OspA vaccine trial administrators could actually read their smudged Western Blots. They actually **HAD NO IDEA** if anyone who was injected with OspA got Lyme.

Yet, they both reported “76%” and “92% “safe and effective” vaccines.

All “bogus articles.”

rOspA was never shown to prevent spirochetes in dogs, monkeys, humans or any other animal.

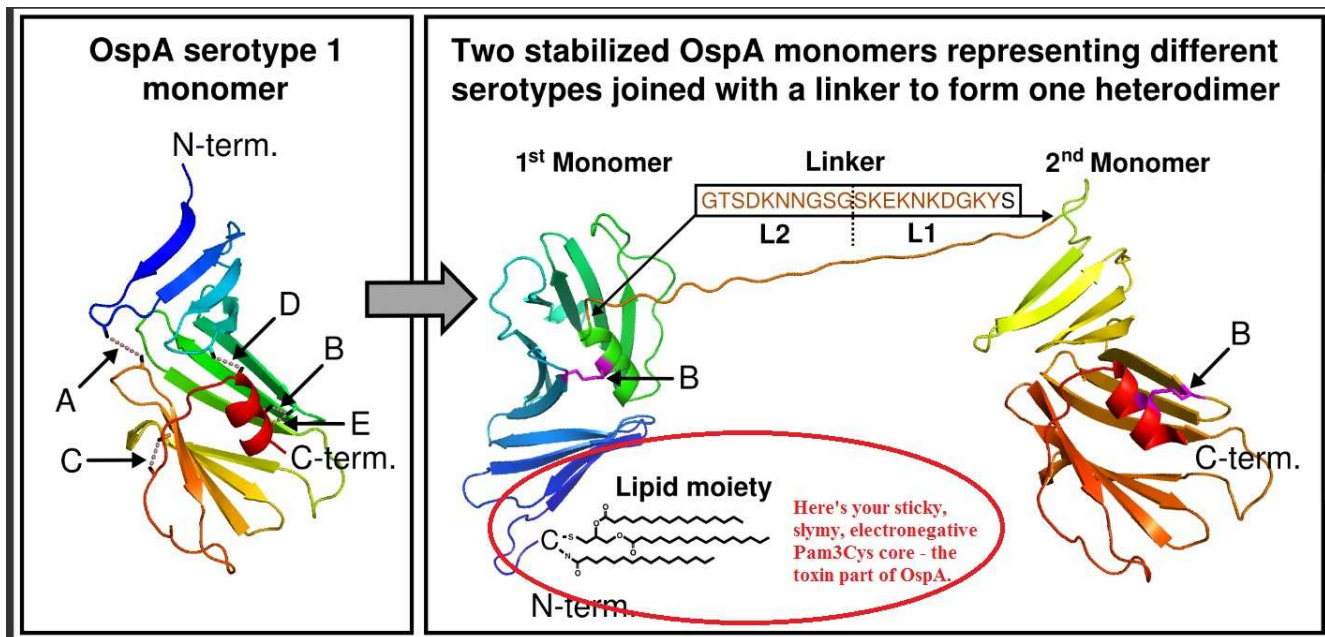
You've seen what OspA (Pam3Cys) is/does in the other charge sheets. It is a fungal endotoxin that causes an AIDS-like outcome in humans, the same as Lyme or Borreliosis itself in 85% of us.

Here, with rOspA as a "vaccine," the Cabal tried to say that humans should be walking canisters of tick disinfectant as a way to *conform* all their wasted efforts on OspA on a new, false and totally ridiculous hypothesis.

What's next? How about they inject us or have us drink **Round-Up** so we can all go piss on all the bad bad weeds.

These people were well aware by 1993 that OspA was a mistake. Yet as recently as late September 2017, Erol Fikrig was giving a talk about resurrecting LYMERix at a hospital in Long Island. He was heckled down by a doctor who has many LYMERix injured patients, still.

And here is the latest idea for an OspA vaccine (it still has sticky, endotoxic TLR2/1 agonist, immunosuppressive Pam3Cys stuck on it, yikes):



<http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0113294>

“More “pretense” by the Cabal. Let’s guess they intend to qualify it by the Dearborn method and claim it prevents bad knees. Who’s going to stop them? ILADS.org? The ones who think you can cure post-sepsis with antibiotics? They’ve had 18 years to get their act together. They never *wanted* to try to understand Lyme or OspA disease. ILADS.org has no actual data that says they ever cured anyone, and Lymedisease.org does not even know what “data” means.