

THE DEARBORN CONFERENCE

~ How the test was spun ~

The current (2006-2007) CDC-sanctioned testing for hard-tick Relapsing Fever *had been changed* into the non-existent entity "Lyme Disease" in 1994 at the CDC's Dearborn, MI, meeting.

The CDC's 1994 Dearborn "two-tiered" testing criteria is meant for the diagnosis of "early Lyme," or within the second month of illness to less than six months into illness.

"Two tiered" testing means: First, one has an ELISA test or a general screening test to detect the concentration of antibodies in the blood to see if the victim has late stage chronic severe arthritis in a knee with the very, very high antibody concentration. (Steere's kind of autoimmune Lyme is a genetically-linked hypersensitivity reaction, like asthma or Type 1 diabetes). *Do keep in mind the ELISA test is 41years old, and there is newer, more specific testing available.*

If one has a positive ELISA result, one may then proceed to Western Blotting, but the problem is that the ELISA screens out all but the extraordinary cases of a loud, red, swollen arthritis in a knee and misses all of the neurological or regular Lyme cases.

That is, in order to have "Early Lyme," one must first have "Late Lyme Arthritis in a knee," according to Allen Steere and the American Lyme Disease Foundation and is now the CDC's accepted testing protocol.

We do not know of any more highly regulated testing anywhere in the US. If you, the lab owner, decided to perform actually scientifically valid testing for Lyme, the CDC will come after you and try to make you close down. We will refer an article from a Hartford Courant interview with Durland Fish:

"He proceeds down the list, name by name: "Totally bogus." "He killed one of his patients." "They tried to shut him down." Words like "crackpot," "wacko," "buffoon" and "fraud" pepper his discourse.

The Two Tiered testing schema was designed to SCREEN OUT all the neurological cases of Lyme with the first test. Some insurance companies even state that they will not pay for a Western Blot if the ELISA is negative. This is scientific fraud with intent to cause harm since Lyme progresses into all sorts of very severe neurological diseases and death.

The Lyme ELISA only detects late Lyme arthritis, with a full-blown swollen knee. Late Lyme arthritis in a knee is caused by a hypersensitivity or allergy reaction or too many antibodies to OspA. The too-many-antibodies against OspA allegedly cross reacted with knee tissue, but now Allen Steere's camp say that OspA is a superantigen for certain persons. That is, people with a certain genetic background will super-bind the OspA antigen into their HLA molecules, causing an almost toxic level of antibodies (chronic stimulation) and the associated immune response. Other researchers counter that if the Steere model was true, the phenomenon would not be limited to a knee.

If a Lyme victim passes *that* test, the ELISA, and has a late Lyme arthritis in a knee, they may graduate on to having a Lyme Western Blot test. One does not validate a test by saying, for example, "Only 7 ton elephants may be diagnosed as elephants, and that 200 pound baby elephants are not elephants." You can still see the baby elephant and know it is an elephant by its **specific** features, and know it is there. We will see that pathology associated with the presence of spirochetes is hardly limited to the production of cross-reacting antibodies and the associated strokes and clogged blood vessels. In other words, no one cares if they have "too many OspA antibodies," they only care to know if what is making them sick is something treatable with something so simple as antibiotics.

5 of the 10-11 early-Lyme, **pre-1987-Steere**, antibodies which occur "over months to years" must *now* - after 1994 and after the CDC Dearborn MI conference - occur within the first ~6 months of infection in order to be a case of "Lyme Disease. That is, where once Lyme was thought to be a Relapsing Fever, characterized by expanding IgM antibodies that occurred over a fairly long time and represented persisting infection, suddenly, 5 of 10 IgG bands *had to all occur at once* for a person to be able to have a diagnosable (treatable) case of "Lyme Disease."

When the conference was proposed and announced, some labs thought it was about standardizing the **method** and not to create a new interpretative platform. That is, to any bona fide scientist, to "standardize a method" means:

- 1) Everyone uses the same **equipment** and concentration of components in the electrophoretic gel.
- 2) the **standard** should be the same (use low passage strains of SPECIFIC organisms; Borrelia that express OspA, B, C, Borrelia-specific flagellin. and other specific known antigens that produce antibodies in most humans, 3) Everyone should agree that the assay of the proposed analytes is a method that does not co-elute more than one analyte. The method should be validated in that the concentration of one analyte is not erroneously added to at the same retention time or kilodalton apparent molecular weight by another (masked) analyte.

The following is the publication of the results of the CDC's Dearborn, MI, conference:

Notice to Readers Recommendations for Test Performance and Interpretation from the Second National Conference on Serologic Diagnosis of Lyme disease

The Association of State and Territorial Public Health Laboratory Directors, CDC, the Food and Drug Administration, the National Institutes of Health, the Council of State and Territorial Epidemiologists, and the National Committee for Clinical Laboratory Standards cosponsored the Second National Conference on Serologic Diagnosis of Lyme Disease held October 27-29, 1994. Conference recommendations were grouped into four categories: 1) serologic test performance

and interpretation, 2) quality-assurance practices, 3) new test evaluation and clearance, and 4) communication of developments in Lyme disease (LD) testing. This report presents recommendations for serologic test performance and interpretation, which included substantial changes in the recommended tests and their interpretation for the serodiagnosis of LD.

A two-test approach for active disease and for previous infection using a sensitive enzyme immunoassay (EIA) or immunofluorescent assay (IFA) followed by a Western immunoblot was the algorithm of choice. All specimens positive or equivocal by a sensitive EIA or IFA should be tested by a standardized Western immunoblot. Specimens negative by a sensitive EIA or IFA need not be tested further. When Western immunoblot is used during the first 4 weeks of disease onset (early LD), both immuno- globulin M (IgM) and immunoglobulin G (IgG) procedures should be performed. A positive IgM test result alone is not recommended for use in determining active disease in persons with illness greater than 1 month's duration because the likelihood of a false-positive test result for a current infection is high for these persons. If a patient with suspected early LD has a negative serology, serologic evidence of infection is best obtained by testing of paired acute- and convalescent-phase serum samples. Serum samples from persons with disseminated or late-stage LD almost always have a strong IgG response to *Borrelia burgdorferi* antigens.

It was recommended that an IgM immunoblot be considered positive if two of the following three bands are present: 24 kDa (*OspC*)*, 39 kDa (*BmpA*), and 41 kDa (*Fla*) (1). It was further recommended that an IgG immunoblot be considered positive if five of the following 10 bands are present: 18 kDa, 21 kDa (*OspC*)*, 28 kDa, 30 kDa, 39 kDa (*BmpA*), 41 kDa (*Fla*), 45 kDa, 58 kDa (not GroEL), 66 kDa, and 93 kDa (2).

The details of both plenary sessions and the work group deliberations are included in the publication of the proceedings, which is available from the Association of State and Territorial Public Health Laboratory Directors; telephone (202) 822-5227.

References

Engstrom SM, Shoop E, Johnson RC. Immunoblot interpretation criteria for serodiagnosis of early Lyme disease. *J Clin Microbiol* 1995;33:419-22.

Dressler F, Whelan JA, Reinhart BN, Steere AC. Western blotting in the serodiagnosis of Lyme disease. *J Infect Dis* 1993;167:392-400.

The apparent molecular mass of *OspC* is dependent on the strain of *B. burgdorferi* being tested. The 24 kDa and 21 kDa proteins referred to are the same.

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The CDC apparently thinks we can not see that the 1986-Steere out of "10-11 bands that occur over months to years" is not equal to the 1994 Dearborn version: "most of the 10-11 bands occur together in first few months of Lyme infection and we don't know what happens after that, since we're only talking about EARLY LYME diagnosis."

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

Antigens of Borrelia burgdorferi recognized during Lyme disease. Appearance of a new immunoglobulin M response and expansion of the immunoglobulin G response late in the illness.

Craft JE, Fischer DK, Shimamoto GT, Steere AC.

*Using immunoblots, we identified proteins of Borrelia burgdorferi bound by IgM and IgG antibodies during Lyme disease. In 12 patients with early disease alone, both the IgM and IgG responses were restricted primarily to a 41-kD antigen. This limited response disappeared within several months. In contrast, among six patients with prolonged illness, the IgM response to the 41-kD protein sometimes persisted for months to years, and late in the illness during arthritis, a new IgM response sometimes developed to a 34-kD component of the organism. The IgG response in these patients appeared in a characteristic sequential pattern **over months to years to as many as 11 spirochetal antigens**. The appearance of a new IgM response and the expansion of the IgG response late in the illness, and the lack of such responses in patients with early disease alone, suggest that B. burgdorferi remains alive throughout the illness. PMID: 3531237 [PubMed - indexed for MEDLINE]*

As an aside, Mark Klempner, CDC "officer," and now a Boston University apprentice provost, alleges that the meaning of: "only 78 out of 1800 (4%) persons" who are "Chronic Lyme" victims, and who all have been treated before with antibiotics - yet still have the CDC bogus positive early Lyme/late Lyme IgG blood testing criteria of Allen Steere - means that the probability that a person who claims to have Chronic Lyme "is 96% likely to be a kook." An antibiomaniac. A Munchauser. A Lyme paranoiac. No one knows what kind of people Klempner used in his "Chronic Lyme is imaginary but cured by the placebo effect of antibiotics 'study,'" since the Steere early Lyme/Late Lyme criteria for a "positive case of Lyme," has never been studied, much less validated for late, treated chronic Lyme. The class of people Mark Klempner claimed to have assessed for antibiotic efficacy should have been "seronegative," due to the host adaptation of their own particular spirochetes.

Few people realize that the CDC IgG method to diagnose "Lyme Disease" is only to be used in the first few months after infection, but that also, "IgG means past infection and not a treatable case," according to Imugen Labs (Norwood, MA). That is, the 1994 Dearborn (Dressler/Steere) early Lyme criteria to be used to diagnose early Lyme comes from an imaginary standard that was revealed (to Allen Steere) as a series of antibodies developed against antigens "over months to years," but that IgG means old or previous, so most people will not have a treatable case of Lyme no matter what the circumstances. IgG means past infection. And no treatment. IgM means current. But almost no IgM bands may be counted as a positive test. Which means no treatment.

Most MDs do not realize that this inadequate testing standard is to not be used for persons who have been sick for more than a year because the spirochetes become “**host adapted.**” That is, spirochetes that arrive into a person fresh from a tick are expressing different surface antigens than those spirochetes which have been cohabitating with their human victims for years. Therefore, the long-infected person produces *different* antibodies than the newly-infected.

These late, undetectable, antibodies are not detectable because 1) everyone has their own special collection of spirochetes' antibodies (which have become host-adapted and are constantly undergoing antigenic variation), 2) everyone has their own genetically-determined propensity to find certain kinds of antigens irritating (HLA differences), and 3) most people, especially the sickest- usually "seronegative (seronegative to the imaginary Steere Dearborn criteria case)" - are likely to be multiply infected with the immune suppression organisms, Babesia and Ehrlichia (ref Dave Persing) and have the neurologic kind of Lyme, since only the knee-kind tests positive. Only the knee-kind of Lyme people make sufficient antibodies and may even be called immune-competent, according to Vikay Sikand, MD (East, Lyme, Connecticut, at the 1998 LYMERix FDA meeting).

According to CDC's officer Alan Barbour,

<http://patft1.uspto.gov/6,719,983>

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.**"*

TRANSLATION: "The Lyme infected person's immune system could be completely overwhelmed by their chronic infection with multiple "clones" of spirochetes, all of which continuously undergo antigenic variation (negating the validity of the term "clone") creating seventy five gazillion different antibodies which overwhelm the immune system.

Therefore, due to antigenic variation and likely being infected with multiple types of spirochetes (*burgdorferi*, or *lonestari*, or *afzeliii*, etc), the only way to see if a chronically infected person is chronically infected using an antibody method, is to capture some spirochetes from said chronically infected person, grow a few, and use that **current**-human-spirochete-slurry to Western Blot the same infected person and see if they have antibodies against their own spirochetes.

We could only expect such mediocre science from the Steere camp. We know that if they performed such a obtuse science experiment, the Steere camp would still try to tell the patient from whom

spirochetes were extracted that their antibodies were only IgG antibodies, and that that meant that the patient only has past-Lyme disease or is therefore no longer infected and does not need treatment.

The entire point of the erroneous and final solution dictated to us from Dearborn "conference," is that "We should look for 5 of 10 IgG bands and ignore most of the IgM bands, because the presence of IgM bands is a phenomenon that only occurs in persisting infections, according to the 1986 version of Allen Steere."

Examples of explanations of host-adapted spirochetes or the proof that late neurologic Lyme will never test positive to the CDC Dearborn method:

Pachner and Brains, 1990 "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."

Deliberate antibody selection using monoclonal antibodies to select "mutants" in vitro- Barbour

Antibody selection: Fikrig, over OspA's uselessness

<http://iai.asm.org/cgi/reprint/63/5/1658?view=long&pmid=7729870>

The exposure of spirochetes to borreliacidal antibodies has been used to isolate mutant organisms in vitro, suggesting that antibodies can elicit selective pressure (1, 17). The growth of *B. burgdorferi* in the presence of anti-*B. burgdorferi* serum or antibodies to OspA or OspB results in the inhibition of growth of a majority of the spirochetes (1, 17). However, the outgrowth of spirochetes that do not express OspA or OspB, have mutations in the *osp* genes resulting in mutant Osps, or have lost the 49-kb plasmid that contains the *ospAB* operon occurs occasionally (17). The frequency of recovery of these variant strains has

New antigen milieu from Firkig re brains <http://www.pnas.org/cgi/content/full/100/26/15953>

The usual case of a chronic Lyme victim is that they have had chronic fatigue for several years before finding their way to a specialist who understands that the blood testing schema proposed by the 1993 version of Allen Steere, fraudulently accepted by the Centers for Disease and accepted by the American Medical Association, is an elaborate concoction of foolery intended to set up a monopoly on testing for "Lyme Disease" around the OspA vaccine. Keeping in mind that the ALDF.com camp are interested only in the profit in vector borne diseases and not in anyone's better health outcome, "Lyme Disease" – the serologic definition previously established by Allen Steere and the CDC as a Relapsing Fever organism, the diagnosis of which was to be in the performance of serial Western Blots in order to look for "changing and expanding IgM and IgG antibodies" - was spun.

In 1992, Allen Steere went to Europe with an illegal high-passage strain of Borrelia- strain G39/40 from Guilford, Connecticut.

bite, and clinical manifestations of the infection.

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

Specific IgM and IgG antibodies to the spirochetal

"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 (21). 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."

The National Institutes of Health's Rocky Mountain Labs recommended **not using high passage strains to diagnose** "Lyme Disease" because they drop plasmid expression or drop the expression of specific diagnostic antigens (or fewer bands will show up in a Western Blot).

Spirochete cultures that have been passed many times in vitro without being pharmed back into ticks and rodents start growing populations which have lost the ability to infect, because they have lost the code for the surface antigens - the speckles or the Osps or the Vmps or the Vsps - which they use to bind surface molecules on the cells of their hosts. With fewer of them; with fewer surface antigens with which they can invade tissue and penetrate or adhere to cells, these spirochetes can cause less damage. Spirochetes are parasites and must take something from the host in order to survive. Logically, one would want to look at what is the makeup of spirochetes, since that would suggest what are its metabolic needs. However, these Borreliae spirochetes can apparently adapt to any environment, any mammalian environment, and any tissue type.

<http://iai.asm.org/cgi/reprint/56/8/1831?view=long&pmid=3397175> NIH Rocky Mountain Labs, including Willy Burgdorfer, say to use low passage strains to Western Blot because high passage strains drop plasmids- yet this is the high passage strain, G39/40, from which we got the Dearborn CDC criteria.

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Changes in Infectivity and Plasmid Profile of the Lyme Disease Spirochete, *Borrelia burgdorferi*, as a Result of In Vitro Cultivation

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In vitro cultivation of *Borrelia burgdorferi*, the etiologic agent of Lyme spirochetosis, allows for the isolation and growth of this bacterium from infected tissues. However, continuous cultivation in modified Kelly medium causes a reduction in the number of detectable plasmids and the loss of infectivity in the white-footed mouse, *Peromyscus leucopus*. In an unpassaged culture of *B. burgdorferi*, nine plasmids were present, including seven linear plasmids ranging in size from 49 to 16 kilobases (kb) and two circular plasmids of 27 and 7.6 kb. The 7.6-kb circular and 22-kb linear plasmids were no longer detectable in spirochetes noninfective in white-footed mice, suggesting that a gene(s) encoding for factors responsible for infection may be present on one or more of these extrachromosomal elements. Furthermore, changes in spirochetal proteins and lipopolysaccharide-like material were observed also during early cultivation and may be related to loss of infectivity.

The CDC's diagnostic standard at that time was based on Steere's own original 1986 observations that Lyme was a **Relapsing Fever organism** and that the antibody profile for Lyme would change over time, producing new (IgM) antibodies when viewed via Western Blotting. Under the pretense of "standardizing" all the US testing for Lyme in America, in 1994, the CDC sent out invitations to numerous labs across the country, inviting them to "contribute to the proceedings." However, apparently the decision had already been made that the CDC would adopt the new Steere method- the one he developed in Europe with a bogus strain that dropped plasmids, and thus, likely, the expression of the OspA-B plasmid. This would have the effect of creating a new sham profile from among Steere's patients that resulted in the current bogus CDC IgG criteria for a positive case of "Lyme Disease." that left OspA and B antibodies (band 31 and 34) out of the standard. What the labs invited to the farcical CDC 1994 Dearborn, MI, conference determined, in regard to Steere's new proposal for an IgG standard were:

- 1) Gary Wormser at New York Medical College- Steere's method detected 9/59 cases
- 2) Igenex- Steere's IgG detected 14% of the cases
- 3) Imugen – Steere's method detected 8% of the cases
- 4) Wisconsin—Steere's method was 15% accurate

- 5) UCONN- Larry Zemel was referring to Lyme as comparable to juvenile rheumatoid arthritis. Recommended adding band 50 for children's blots.
- 6) Roche—28% were positive for every Steere band.
- 7) Wadsworth – had some different scoring system. Did not report on accuracy of Steere
- 8) Ontario Ministry of Health
- 9) Lutheran Hospital— 14 % were accurate by Steere's IgG
- 10) MarDx Labs – recommended adding bands 31 and 34 were given CDC positive arthritis positive blood to qualify their test strips since these were used in both vaccine trials.
- 11) CDC Atlanta – talked about mice, not humans. The mouse criteria were 2 out of three from OspC, 16 kD, 17.9 kD, for the mice.

The reason Steere's Dressler/Steere Western Blotting recommendations performed so poorly was because Steere decided that "Lyme disease" would be a genetically linked autoimmune arthritis in knee. Because Lyme disease is not a genetically linked autoimmune arthritis in a knee, LYMERix was not a vaccine and came off the market.

The following is an excerpt from the Dressler/Steere report where Steere used high passage strain G39/40 to leave OspA and B out of the diagnostic standard:

Western Blotting in Lyme Disease

Table 1. Frequency of polypeptide responses in a retrospective analysis of patients with Lyme disease and control subjects.

kDa	IgM band present (%)				IgG band present (%)			
	Erythema migrans (n = 25)		Menin- gitis (n = 25)	Control (n = 125)	Menin- gitis (n = 25)	Arthritis (n = 25)	Late neuro (n = 25)	Controls (n = 125)
	Acute	Conv						
18	20	52	8	2	84	100	80	0
21	44	60	68	4	28	48	48	0
28	4	0	52	0	44	88	84	1
30	4	16	8	1	28	100	84	2
31	0	0	12	0	0	44	40	2
34	0	0	12	2	0	60	36	1
37	24	32	28	1	4	44	48	12
39	4	8	0	0	20	92	88	10
41	32	36	52	2	92	96	88	41
45	32	20	28	1	80	84	72	10
58	28	48	36	1	84	100	92	7
66	4	8	24	2	56	92	76	2
74	8	8	8	1	12	68	44	8
93	8	32	20	1	28	100	76	0

NOTE. Conv, convalescent phase; late neuro, encephalopathy and polyneuropathy.

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Presented in part: annual meeting of the American College of Rheumatology, Boston, November 1991 (abstract B94, *Arthritis Rheum* 1991;34:S113); International Conference on Lyme Borreliosis, Arlington, Virginia, May 1992 (abstract 15).

Informed consent was obtained from patients or their parents, and human experimentation guidelines of the US Department of Health and Human Services were followed.

Financial support: National Institutes of Health (AR-20358, AR-40576); Eshe Fund; Deutsche Forschungsgemeinschaft (1989-1990 research scholarship to F.D.); Becton Dickinson (1991 young investigator award to F.D.).

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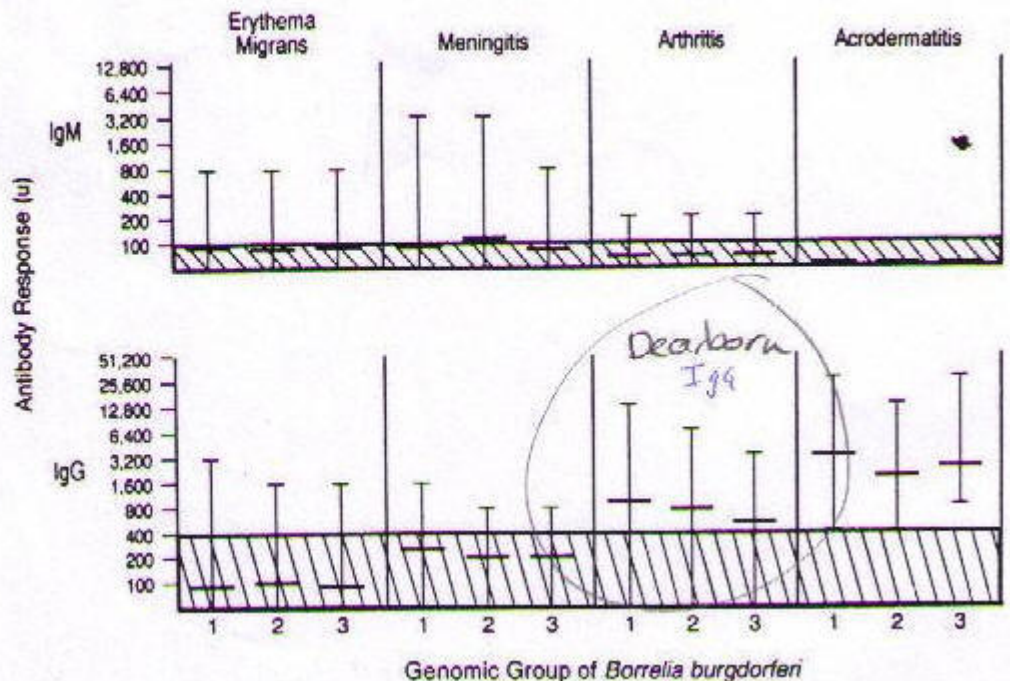
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In 1993, Dressler, Steere report where OspA and B were left out of the diagnostic standard by using a high passage strain G39/40 and is the basis for the current CDC IgG standard criteria to have a diagnosis of "Lyme Disease." This is part of a submission by a scientist in southern Connecticut to the FDA Vaccine Committee on January 31, 2001. It is part of the public record.

The following snippet comes out of the Steere in Europe report, where you can clearly see that each manifestation of the disease has a very different antibody panel. Note that meningitis has a higher across-the-board IgM spectrum, yet at Dearborn, almost all of these bands are left out of the potential contribution to identifying a case of Lyme. Lyme meningitis is the most awful case of Lyme:

Figure 2. Antibody responses to *B. burgdorferi*, group 1 strain G39/40, group 2 strain FRG, and group 3 strain IP3, by indirect ELISA. Horizontal bars show geometric mean response; vertical bars, range; hatched areas, range of values in normal control subjects. To calculate antibody response (units), value of each sample was adjusted from standard curve made from dilutions of known positive serum.



For the 1994 CDC Dearborn C, Outer Surface protein A, once thought to be the cause of Lyme Disease (autoimmune arthritis in a knee), was been left out of the standard. That was because it was going to be a vaccine, and in most cases of vaccination, there is a test for the disease that leaves out the vaccine antigen, since what would be the point of looking for an antibody that comes from vaccination? Vaccination affects the antibody the body would naturally make if it weren't vaccinated. What's different here is that there was a "controversy," over what Lyme was, ever since the publication of the Infectious Disease Reviews (in 1989) over what "Lyme Disease" is, when the ID Reviews Supplement 6, especially about chronic parasitic spirochetal infections, pretty much established that Lyme was a chronic parasitic spirochetal infection, like all the rest of the chronic parasitic spirochetal infections.

All Lyme Patients should know and understand:

1) Lyme Disease" was redefined by Allen Steere at the Dearborn Conference to an autoimmune arthritis in a knee where antibodies against OspA result in an autoimmune arthritis in a knee because OspA was intended to be the "vaccine."

2) No one is allowed to have "Lyme Disease," unless they have an autoimmune arthritis in a knee - an arthritis that is so obvious that it doesn't even need a blood test (red, raised, hot, and painful). It is usually restricted to one joint.

3) No one is allowed to have antibodies against OspA ("Lyme Disease") and have a diagnosis of "Lyme Disease." LYMERix is allegedly specific enough to *prevent* Lyme, but not specific enough to *detect* Lyme, even though it is 100% specific to Lyme.

4) Bb strain B31 is the "prototypical American strain," but it expresses no OspC- the brain invasion antigen.

Conclusion: "Lyme disease" is an autoimmune arthritis in a knee caused by the antibodies no one is allowed to have. It is not caused by OspA, B, or C, which are the "primary immunodominant antigens (produce the most antibodies)." It does not go into the brain or the knee or anywhere else, since there is no OspC in the prototypical strain. **This is what the Steere camp would like all the Doctors, citizens of America and the World to believe: that "Lyme Disease is a non-disease."**

For the Steere camp to further prove that "Lyme Disease" is a non-disease, Mark Klempner said at the 2001 Diseases of Summer Conference in Rhode Island that CDC does not recognize any other kind of Lyme except this imaginary kind of Lyme. One of the biggest problems in the Lyme crime world is that IDSociety.org is basing their "guidelines" on the very seriously invalid Klempner's Chronic Lyme Treatment report released in July 2001. For starters, Klempner says, "Only the Imaginary Criteria of Allen Steere Invented in Europe with high passage strain G39/40 may be called (diagnosed and therefore treated) 'Lyme Disease.'"

The following is an excerpt of the Question and Answer session that followed the Klempner Non-Diseases of Summer Conference at South Country Hospital, Rhode Island

Questioner8: I just have one more question for Dr. Klempner. Um, being that there are inadequacies, inaccuracies in the testing methods, seropositivity, etc, and the surveillance criteria that you used were just that, surveillance. And the CDC recognizes that there are so many more people that have Lyme disease who do not meet the CDC criteria. What's your feeling on what percentage of patients who have Lyme disease because they have not met the criteria for diagnosis?

*Klempner: I, um, I think there are a number of inaccuracies in what you just said. The CDC does **not** recognize that there are patients who have, um, that are seropositive that don't meet seropositive criteria. What they **say**, is that **these** are the **criteria**. I think what, the question, if I could reinterpret the question a little bit, is are there patients who are out there who had Lyme disease who continue to have symptoms and um, wouldn't fall into these categories. And the question is, how do you define patients who have had Lyme disease? You've gotta, you've gotta start with some agreed upon cohort, so what are the agreed upon criteria? And what we were trying to do, since we know this was a controversial topic to **start** with a group of patients who **no one would doubt** had had Lyme disease. And that was really the point of the study. Are there other patients who fall into equivocal groups that one could say, it's difficult to document that they had Lyme disease? Sure, but remember **we were about to do a study** that was very risky. Um, meaning giving people parenteral antibiotics, doing lumbar punctures, doing huge numbers of studies on these people. It was **very** important to start patients that **everybody** agreed had had acute Lyme disease. Are there lots of other people out there who say they have Lyme disease where the documentation is lacking? You know that better than I do. Of **course**, there are **lots** of people out there who says they have **lots** of things that you can't document.*

Questioner8: But, but according to what I've read is that not everyone is going to, number one, see an EM rash, um, so when you're are using entry criteria, diagnostic criteria, that you need to have an EM rash, and physician diagnosed...

Klempner: If you're're seronegative

Questioner8: Right, okay, but there are seronegative people that don't have the initial EM rash, And if they do, they may not see it, being on the back...

*Klempner: So, then how do you know what they have, that is anybody who walks in the door who says, "I don't feel well", with this set of symptoms. Um, that is **not** a group of people that **I** would be comfortable putting an intravenous catheter in, an LP on, doing all this very complex study. I needed to be assured that those patients had had Lyme disease. You're describing a group of patients who cluster by virtue of symptoms. No different from the symptom complex that was given here [previous (Fibromyalgia) talk]. This is a very different symptom complex, very different patient population. **Very well documented Lyme disease**. And I just wanted to be sure. Um, that's where I started my talk, at that point. That is, there are a **lot** of people all over this world that claim to have **lots of different things**. But for doing studies, I think it's very important that we cluster patients by objective findings, strict criteria.*

For doing studies with invalid tests where the outcome is predetermined, one should start out with the assumption that the only kind of Lyme is the imaginary Steere kind. The same thing happened with the "vaccines" for Lyme: No one had the imaginary Steere kind of Lyme, therefore no Lyme emerged in vaccinated people, therefore the "vaccines" were "safe" and "effective."

**There are three essential reports that Lyme Patients
and Doctors absolutely should obtain to understand the stratagem:**

- (1) 1986 original standard development
- (2) 1993 Dressler/Steere or the Dearborn IgG method
- (3) 1992 Steere in Europe, Antibodies in Europe.

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

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

Dressler F, Ackermann R, Steere AC.

Division of Rheumatology/Immunology, New England Medical Center, Tufts University School of Medicine, Boston, Massachusetts 02111.

The antibody responses to the three genomic groups of Borrelia burgdorferi (B. burgdorferi sensu stricto, Borrelia garinii, and Borrelia afzelii) were determined in 97 German patients with various manifestations of Lyme borreliosis. The geometric mean antibody titers in each patient group, determined by ELISA, were similar with each antigen preparation. By Western blotting, however, patients with meningopolyneuritis tended to respond to more spirochetal polypeptides of B. garinii, the group 2 strain, whereas those with arthritis recognized more antigens of B. afzelii, the group 3 strain ($P < .03$), as did those with acrodermatitis. Only 1 patient each with erythema migrans, arthritis, or acrodermatitis had weak reactivity with outer surface protein A (OspA), and none responded to OspB. It is concluded that differences among the three groups of B. burgdorferi may result in variations in the antibody response in European Lyme borreliosis. PMID: 8106763 [PubMed - indexed for MEDLINE]

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

Comment in: J Infect Dis. 1993 Oct; 168(4):1073.

Western blotting in the serodiagnosis of Lyme disease.

Dressler F, Whalen JA, Reinhardt BN, Steere AC.

Division of Rheumatology/Immunology, Tufts University School of Medicine, New England Medical Center, Boston, Massachusetts 02111.

There are currently no accepted criteria for positive Western blots in Lyme disease. In a retrospective analysis of 225 case and control subjects, the best discriminatory ability of test criteria was obtained by requiring at least 2 of the 8 most common IgM bands in early disease (18, 21, 28, 37, 41, 45, 58, and 93 kDa) and by requiring at least 5 of the 10 most frequent IgG bands after the first weeks of infection (18, 21, 28, 30, 39, 41, 45, 58, 66, and 93 kDa). When these definitions were tested in a prospective study of all 237 patients seen in a diagnostic Lyme disease clinic during a 1-year period and in 74 patients with erythema migrans or summer flu-like illnesses, the IgM blot in early disease had a sensitivity of 32% and a specificity of 100%; the IgG blot after the first weeks of infection had a sensitivity of 83% and a specificity of 95%. Among patients with indeterminate IgG responses by ELISA, 6 of 9 patients with active Lyme disease had positive blots compared with 2 of 34 patients with other illnesses ($P < .001$). Thus, Western blotting can be used to increase the specificity of serologic testing in Lyme disease. PMID: 8380611 [PubMed - indexed for MEDLINE]

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

Antigens of Borrelia burgdorferi recognized during Lyme disease. Appearance of a new immunoglobulin M response and expansion of the immunoglobulin G response late in the illness.

Craft JE, Fischer DK, Shimamoto GT, Steere AC.

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

PMID: 3531237 [PubMed - indexed for MEDLINE]