

# Re: More on toll-like receptors in Lyme, per NIH researchers

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Imagine a pyramid:

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C
CC
CCC
CCCC
*****
NNNNNNN
NNNNNNNNN
NNNNNNNNNNN
NNNNNNNNNNNNN
NNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNNNNNN
NNNNNNNNNNNNNNNNNNNNN
```

This pyramid reflects antibody response to Bb exposure among the population of exposed.  
At the top "C" reflects humans with CDC positive serology {a completely human construct}

The 'N's reflect humans with serology which is not CDC positive, yet demonstrate a lesser pattern of antibody binding on western blot. The cornerstone of all of these serologies is the 41KdA flagellin antibody, seen in the vast majority if not almost all cases.

As Bb disseminates in the human host, it alters its protein expression patterns – at first, it expresses ~150 proteins, but later in the infection this number decreases to around 30 as it settles into its niches in the brain and other protective nutrient rich environments.

What steere etc. studied was lyme arthritis, which can basically be regarded as an early inflammatory manifestation of disease. These are the "C"s, as all of the clinicians data is heavily biased toward arthritic presentations. They implicitly admit to this much, what they

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don't admit to but what can be demonstrated is a massive scheme to commit statistical and scientific fraud with respect to certain antigens especially 41 kDa.

In american DOGS, and in europe, 2 or 3 immunodominant IgG antibodies are enough to diagnose borreliosis. these criteria are less politicized, and in the case of europeans more inclusive of other manifestations of the disease. for instance in the absence of syphilis 39 kDa is diagnostic of exposure. really the same is true for 41 kDa.

Why would they need to define lyme disease serologically in this manner?

The answer is that the market for a vaccine would basically be focused on a hyperendemic area. Since probably >25% of the population of this area is already manifesting serological evidence of baseline exposure {and possibly nonarthritic manifestations of borreliosis}, this means that in a considerable portion of potential vaccine consumers AND clinical trial participants, it would be impossible to tell if the vaccine was effective based on a truthful serologic criteria for the disease, since large numbers of vaccinees **ALREADY HAVE THE DISEASE.** {and the vaccine doesn't eliminate latent infections – in fact it probably is harmful in this regard} In some of them, due to combinations of genotype and borrelia strain, this is likely a harmless exposure. However in large numbers for the exact same reason, this is not the case, and these individuals will go on to develop various late manifestations of this disabling and life-altering disease. Their lives sacrificed so that a few avaricious traitorous pigs could profit off of a vaccine, and insurance consultations for denying treatment to and otherwise ridiculing the vast numbers of non-steere serological exposures.

You might be interested to know that a subpopulation of asymptomatic or latent non-arthritic exposures are actually seropositive by CDC standards. This is reflective of the meaningless nature of the steere criteria. It cannot distinguish between asymptomatic and symptomatic infection either; it was simply a method of qualifying a vaccine, which by the way is only 70% effective anyway.

The steere criteria misses far more borreliosis cases than it catches.

Eliminating the deer in the early 90's would have meant that by now there was no lyme problem in the United States.

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