



Society for the Advancement of Scientific Hermeneutics

Interpreting the Evolution of Linguistics from Hippocrates to Hypocrises

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

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

~ [Anthony S. Fauci, M.D.](#)
NIAID Director



it is necessary to explain what OspA is, to understand how it fits this fungal model. Borreliosis is a multi-system disease caused by a spirochetal parasite. A spirochete is a spiral shaped parasite with a unique mechanism for movement that features a bundle of tail-like "flagella" which resides inside the cell wall. Borreliosis is transmitted primarily through the bite of an infected tick, but also can be transmitted in utero to an unborn fetus (according to Yale), and possibly through insect vectors. If not treated immediately with antibiotics, the

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

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

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

We will begin with Lyme disease, or borreliosis, since

infection can persist for years and cause neurological diseases such as MS, Lupus, cancer, Chronic Fatigue/ Myalgic Encephalomyelitis, ALS (Lou Gehrig's Disease) and Alzheimer's, according to IDSA and the CDC. Thus, borreliosis is commonly referred to as "The Great Imitator" or "New Great Imitator."

In a mechanism commonly known as blebbing, borreliae parasites have the ability to shed (bleb off) their outer membrane lipoproteins to evade detection by the immune system, per CDC officer Alan Barbour in (the probably mis-titled): ["Researchers Finding Rewarding Careers As Software Entrepreneurs"](#) "It's using some sort of stealth-bomber-type mechanism," he says. Or, using another diversionary tactic called blebbing, the spirochete can pinch off bits of its membrane in order to release its surface proteins. Explains Barbour: "It's like a bacterial Star Wars defense program," in which released surface proteins might intercept incoming host antibodies, keeping the spirochete safe from immunological attack."

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

These outer surface lipoproteins, such as OspA (the Lyme "vaccine"-- LYMERix) are TLR2 agonists (fungal antigens) and also "undergo virtually limitless antigenic variation, leaving the immune system overwhelmed" says, again, CDC officer Alan Barbour

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

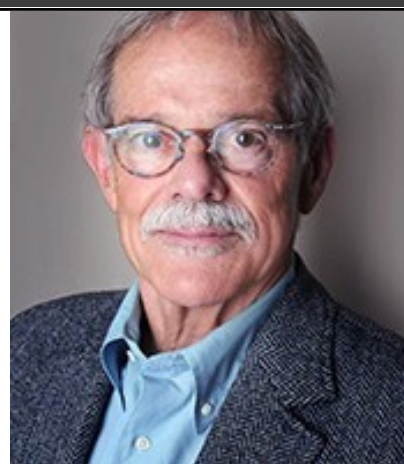
Fungal antigens shed by *Borrelia* cause tolerance to other fungal antigens (TLR2/1-agonists such as those borne by mycoplasma and mycobacteria) as well as tolerance to other antigen types, managed by other TLRs. Tolerance means that the immune system stops recognizing fungal antigens such as:

- 1) inhibiting HLA-molecule function and therefore antibodies are no longer produced (Radolf and Harding), "Despite the ability of MTB 19-kDa lipoprotein to activate microbicidal and innate immune functions early in infection, TLR 2-dependent inhibition of MHC-II expression and Ag processing by MTB 19-kDa lipoprotein during later phases of macrophage infection may prevent presentation of MTB Ags and decrease recognition by T cells. This mechanism may allow intracellular MTB to evade immune surveillance and maintain chronic infection."
<http://www.jimmunol.org/content/167/2/910.full>, and

- 2) exposure to *Borrelia* fungal antigens causes cross-tolerance to the TLRs that manage viral

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~ [Alan Barbour, MD](#), CDC Officer



infections: "Because IRAK1 is required for TLR7/9-induced IFN-I production, we propose that TLR2 signaling induces rapid depletion of IRAK1, which impairs IFN-I induction by TLR7/9. This novel mechanism, whereby TLR2 inhibits IFN-I induction by TLR7/9, may shape immune responses to microbes that express ligands for both TLR2 and TLR7/TLR9, or responses to bacteria/virus coinfection." (CV Harding) <http://www.ncbi.nlm.nih.gov/pubmed/2227568>

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

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

~ [Raymond Dattwyler, MD](#)



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

Dr. O'Brien: "I was concerned about your last slide where you said there was a poor correlation between serologic response and clinical

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

Dr. Dattwyler: "No, no, I said the reverse. The better responses tended to have a better response. And I should clarify where this came from. This is from antibiotic trials. These are treatment trials of

erythema migrans, in which individuals given an antibiotic regimen which was not optimal--we didn't know that it was not optimal at the time--the ones that failed to mount a vigorous immune response tended to do worse, clinically. So, there was an inverse correlation between the degree of serologic response and the outcome. So, individuals with a poor immune response tend to have worse disease."

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

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

- 1) NIH: profound immunosuppression is one of the chronic consequences of severe sepsis <http://www.ncbi.nlm.nih.gov/m/pubmed/21048427/>
- 2) Washington University in St. Louis: Reactivation of multiple viruses in patients with sepsis <http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0098819>

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

Other examples:

In Autism: Fungally contaminated vaccines reactivate the live, attenuated viruses and suppress

the immune system (TLR2/1-agonist tolerance), causing children to get the disease instead of the protection.

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

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

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

Absence of Mycoplasma Species DNA in Chronic Fatigue Syndrome, 2003: "Blood was collected in sodium citrate Vacutainer tubes (Beckton Dickinson) and shipped by overnight courier to the Centers for Disease Control (CDC), where plasma was collected by separation on lymphocyte separation medium (LSM; ICN Biomedicals). Plasma (1 ml) was concentrated to approximately 250 µl in a Centricon centrifugal filter unit YM-100 (Millipore). ***Cell-free plasma DNA was extracted by using a QIAamp DNA Mini kit (Qiagen) according to the manufacturer's instructions and quantified by using a DyNA Quant 200 fluorometer*** (Amersham Biosciences)." <http://imm.sgmjournals.org/content/52/11/1027.long>

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

Why?

Because this is the mechanism behind the Autism pandemic:

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

They say: "But a proposal that the ban include thimerosal, which has been used since the 1930s to prevent bacterial and fungal contamination in multidose vials of vaccines, has drawn strong criticism from pediatricians."

"They say that the ethyl-mercury compound is critical for vaccine use in the developing world, where multidose vials are a mainstay."

"Banning it would require switching to single-dose vials for vaccines, which would cost far more and require new networks of cold storage facilities and additional capacity for waste disposal, the authors of the articles said."

What is the Treatment?

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

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

Anthony Fauci, head of the National Institute of Allergy

and Infectious Diseases (NIAID), has patented a treatment for the immune suppression outcomes of chronic Lyme disease -- a condition he simultaneously denies. The CDC calls it "fungal meningitis":

<http://www.cdc.gov/meningitis/fungal.html>

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

[Blaschke_DMID_14_Unmet_Diagnostic_Needs.pdf](http://ein.idsociety.org/media/publications/papers/2014/Blaschke_DMID_14_Unmet_Diagnostic_Needs.pdf)

Fauci's patent for the treatment of Fungal Meningitis (Chronic Fatigue, Chronic Lyme and LYMERix Disease): <http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=/netahtml/PTO/srchnum.htm&r=1&f=G&l=50&s1=5,696,079.PN.&OS=PN/5,696,079&RS=PN/%205,696,079>

"BACKGROUND OF THE INVENTION "....Illustrative of specific disease states in treatment of which the present invention can be applied are HIV infection and other diseases characterized by a decrease of T-cell immunity, for example, mycobacterial infections like tuberculosis and fungal infections such as cryptococcal disease. This method also can be used in the treatment of secondary infections that occur in patients with suppressed immune systems, such as the opportunistic infections that occur in AIDS patients. ..."

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

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

~ [Anthony S. Fauci, M.D.](#)