Criminal Charge Sheet # 5.) Common Mechanisms in ME/CFS and the Brain Damage we call Autism (and Chronic Lyme disease or post-sepsis syndrome)

This report is actually just a continuation of the Occam's Razor Criminal Charges Sheet: the mechanisms of post-sepsis syndrome, fungal exposures, how fungal antigens cause

immunosuppression, how there are no antibody markers for the diseases set we are talking about, and how Chronic Fatigue/ME and Fibromyalgia are essentially the same as Post Sepsis syndrome, with or without a tick bite. In Lyme, spirochetes are not what causing the disease except for the initial immunosuppression event. It is the secondary opportunistics, like the fatigue-causing reactivated herpes viruses, the TLR2/1 agonist-bearing, fatigue-causing mycoplasma, and the like. However there are a few independent data sets regarding Chronic Fatigue Syndrome that are worth reviewing.

But let's start with the very first three things everyone should know*:*

1) 2012, Dec, *New York Times*; Doctors admit Thimerosal is put in vaccines to prevent fungi:

Vaccine Rule Is Said to Hurt Health Efforts

"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.'"
http://www.nytimes.com/2012/12/17/health/experts-say-thimerosal-ban-would-imperil-global-health-efforts.html?_r=2&

2) A report from Denmark which says that once Thimerosal was removed from certain vaccines, Autism cases from vaccines skyrocketed, although the majority of Autism cases seem to have a closer relationship to the MMR vaccines:

Pediatrics. 2003 Sep;112(3 Pt 1):604-6.

Thimerosal and the occurrence of autism: negative ecological evidence from Danish population-based data.

Madsen KM1, Lauritsen MB, Pedersen CB, Thorsen P, Plesner AM, Andersen PH, Mortensen PB.

"OBJECTIVE: It has been suggested that thimerosal, a mercury-containing preservative in vaccines, is a risk factor for the development of autism. We examined whether discontinuing the use of thimerosal-containing vaccines in Denmark led to a decrease in the incidence of autism.
DESIGN: Analysis of data from the Danish Psychiatric Central Research Register recording all psychiatric admissions since 1971, and all outpatient contacts in psychiatric departments in Denmark since 1995.
PATIENTS: All children between 2 and 10 years old who were diagnosed with autism during the period from 1971-2000.
OUTCOME MEASURES: Annual and age-specific incidence for first day of first recorded admission with a diagnosis of autism in children between 2 and 10 years old.
RESULTS: A total of 956 children with a male-to-female ratio of 3.5:1 had been diagnosed with autism during the period from 1971-2000. There was no trend toward an increase in the incidence of autism during that period when thimerosal was used in Denmark, up through 1990. From 1991 until 2000 the incidence increased and continued to rise after the removal of thimerosal from vaccines, including increases among children born after the discontinuation of thimerosal.
CONCLUSIONS: The discontinuation of thimerosal-containing vaccines in Denmark in 1992 was followed by an increase in the incidence of autism. Our ecological data do not support a correlation

between thimerosal-containing vaccines and the incidence of autism…."

"A total of 956 children with a male to female ratio of 3.5:1 had been diagnosed with autism during the period 1971–2000. Figure 1 shows the incidence rates according to calendar year and age band. The incidence was stable until 1990 and thereafter it increased in all age groups until 1999. Generally, rates were lower in 2000 than in 1999. Further subdivision by gender had no impact on these results (data not shown). In additional analyses we examined data using inpatients only. This was done to elucidate the contribution of the outpatient registration to the change in incidence. The same trend with an increase in the incidence rates from 1990 until the end of the study period was seen (data not shown). There was no trend toward an increase in the incidence of autism during the period when thimerosal was used up to 1990. The incidence of autism began to increase in 1991, but continued to rise after the discontinuation of thimerosal (Fig 1), including increases among children born after 1992 (ie, the peak autism incidence in 1999 among children aged 2 to 4 and 5 to 6 years of age corresponds to children born in 1993–1997 after the introduction of thimerosal-free vaccines)."

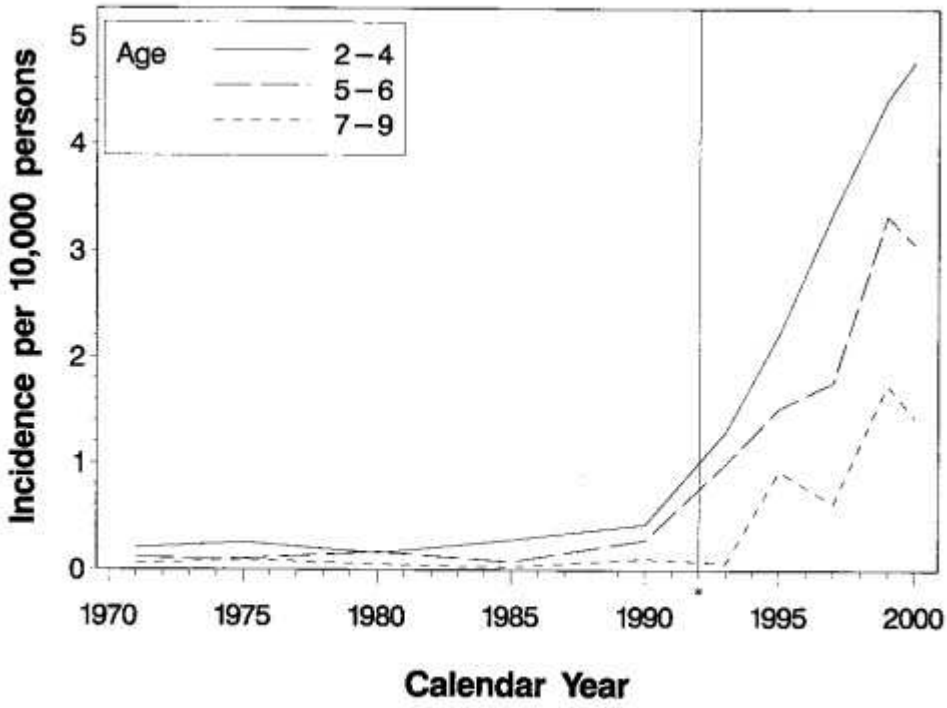


Fig 1. Incidence of autism by age and calendar year. The asterisk (*) indicates removal of thimerosal-containing vaccines in 1992.

https://www.ncbi.nlm.nih.gov/pubmed/12949291

And here, the Food and Drug Administration (FDA) is making fun of mothers for reporting an association between the MMR vaccines and Autism, especially when the MMR was given in combination with some other vaccine (when that is what we would expect, since the vaccine viruses and some of the antigens alone nearly all seem to cause immunosuppression, even if not contaminated with fungal antigens):

Am J Public Health. 2004 Jun;94(6):990-5.

Vaccine risk perception among reporters of autism after vaccination: vaccine adverse event reporting system 1990-2001.

Woo EJ1, Ball R, Bostrom A, Shadomy SV, Ball LK, Evans G, Braun M.

"OBJECTIVES: We investigated vaccine risk perception among reporters of autism to the Vaccine Adverse Event Reporting System (VAERS).
METHODS: We conducted structured interviews with 124 parents who reported autism and related disorders to VAERS from 1990 to 2001 and compared results with those of a published survey of parents in the general population.
RESULTS: Respondents perceived vaccine-preventable diseases as less serious than did other parents. Only 15% of respondents deemed immunization extremely important for children's health; two thirds had withheld vaccines from their children.
CONCLUSIONS: Views of parents who believe vaccines injured their children differ significantly from those of the general population regarding the benefits of immunization. Understanding the factors that shape this perspective can improve communication among vaccine providers, policymakers, and parents/patients.

"Vaccines.
Almost two thirds of the VAERS reports (81 reports, 65.3%) listed MMR or its component vaccines. MMR or measles–rubella (1 report) was the only vaccine listed on 22 reports (17.7%); on 59 reports (47.6%), it was listed in conjunction with other vaccines, the most common of which were *Haemophilus influenzae* type B, oral live polio, diphtheria–tetanus–acellular pertussis, and varicella. On the 43 reports (34.7%) that did not list MMR or any of its component vaccines, diphtheria–tetanus–pertussis, diphtheria–tetanus–acellular pertussis, *Haemophilus influenzae* type B, and oral live polio vaccine were the most commonly reported vaccines. Parent interviews confirmed which vaccines the child had received in relation to the reported symptoms. Reports received on March 1, 1998, or later were somewhat more likely to list MMR (67.0% vs 59.3%) than reports received earlier. Reports received on August 1, 1999, or later were more likely to list hepatitis B (18.1% vs 5.1%), *Haemophilus influenzae* type B (38.6% vs 28.2%), and diphtheria–tetanus–acellular pertussis (26.5% vs 12.8%) vaccines than reports received earlier. Because manufacturer names and lot numbers were missing from the reports, it was not possible to determine from these VAERS reports how many of the case-patients received thimerosal-containing vaccines that had been distributed to clinics before the request was issued.

"Making the Association Between Vaccination and Autism and Related Disorders
In response to the open-ended question, "What made you think that _____'s symptoms might be related to a vaccination?" reporters listed a variety of reasons (Table 1). **The most frequently volunteered reason was the temporal proximity of vaccination and symptom development…**"
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1448378/

Given the fact no one has any real or valid information on this association, given this condescension by the FDA, given our own empirical observations watching the before and after videos of children damaged by the MMR vaccine in particular, and given how the enture Health and Human Services (FDS CDC, NIH) treats their victims, we're going to believe these "EDUCATED" mothers.

3.) Borna virus is a model of the "neurodevelopmental brain damage" we call Autism. That is, a live virus infection which no doubt is responsible for the inflammation, SSPE, SIDS (warned about in the MMR monograph), is what deoes the damage; the active viruses destroy neurons, etc.

Proc Natl Acad Sci U S A. 1999 Oct 12;96(21):12102-7.
An infection-based model of neurodevelopmental damage.
Hornig M1, Weissenböck H, Horscroft N, Lipkin WI.

"Perinatal exposure to infectious agents and toxins is linked to the pathogenesis of neuropsychiatric disorders, but the mechanisms by which environmental triggers interact with developing immune and neural elements to create neurodevelopmental disturbances are poorly understood. We describe a model for investigating disorders of central nervous system development based on neonatal rat infection with Borna disease virus, a neurotropic noncytolytic RNA virus. Infection results in abnormal righting reflexes, hyperactivity, inhibition of open-field exploration, and stereotypic behaviors. Architecture is markedly disrupted in hippocampus and cerebellum, with reduction in granule and Purkinje cell numbers. Neurons are lost predominantly by apoptosis, as supported by increased mRNA levels for pro-apoptotic products (Fas, caspase-1), decreased mRNA levels for the anti-apoptotic bcl-x, and in situ labeling of fragmented DNA. Although inflammatory infiltrates are observed transiently in frontal cortex, glial activation (microgliosis > astrocytosis) is prominent throughout the brain and persists for several weeks in concert with increased levels of proinflammatory cytokine mRNAs (interleukins 1alpha, 1beta, and 6 and tumor necrosis factor alpha) and progressive hippocampal and cerebellar damage. The resemblance of these functional and neuropathologic abnormalities to human neurodevelopmental disorders suggests the utility of this model for defining cellular, biochemical, histologic, and functional outcomes of interactions of environmental influences with the developing central nervous system."
https://www.ncbi.nlm.nih.gov/pubmed/10518583 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC18419/

More:
https://www.ncbi.nlm.nih.gov/pubmed/?term=borna+virus+and+autism

The rubella vaccines were invented in the first place because rubella was known to cause "congenital Autism."

Stanley Plotkin article, next, and others show that rubella causes immunosuppression, infected infants shed the live viruses and give them to other people,…. while the CDC et al deny this, and say the people are not getting the viruses from the vaccinated person, but some other wild type strain (even though they are they same strain). And also, people taking immunosuppressing drugs are told not to be near someone "recently vaccinated."

Plotkin, 1975:

Am J Dis Child. 1975 Apr;129(4):444-9.
Routes of fetal infection and mechanisms of fetal damage.
Plotkin SA.

"… Once the rubella virus infects the fetus, a chronic, nonlytic infection is established. This was first demon¬ strated in vitro.27 Infection of strains of human fibroblasts, once estab¬ lished, persists for weeks or months in stationary cultures. When the cell cultures are placed in fresh vessels under conditions that allow uninfected control cells to divide, mitotic inhibition is observed.

Rubella virus carrier cultures derived from congeni¬ tally infected infants exhibit de¬ creased cell division rate, and are not susceptible to cure with antibody. They also show resistance to superinfection not mediated by Inter¬ feron.28 Crucial evidence was added when the number of cells in fetal or¬ gans was measured. There was a 50% decrease in rubella-infected fetuses compared to controls.29 The possibility that this inhibition of cell division is mediated by a soluble protein was suggested.30

"Four additional mechanisms of fe¬ tal damage by rubella virus remain to be considered. **First, it seems certain from histologie examination of the brain and the organ of Corti that much rubella damage is vascular in origin. Damage to endothelial cells leads to thrombosis of small blood vessels and surrounding tissue necro¬ sis.31**

"Second, some cells, particularly those in the lens of the eye, are proba¬ bly killed by rubella virus.

"Third, study of rubella carrier cell cultures from aborted fetuses shows increased incidence of chromatid breaks. Specific chromosomal anom¬ alies in fetuses with rubella syndrome have been reported or suggested,32 but the evidence that chromosomal abnormalities are a cause of rubella anomalies is not compelling.

"Fourth, there are many parts of the rubella syndrome that are the direct result of persistent infection. Among these are the encephalomeningitis, which often continues during the first year of life33; the cataracts, which may grow worse after birth and in which the virus survives for years34; the postnatal hepatitis; the thrombocytopenia, which is partly due to megakaryocyte destruction and which eventually resolves after birth; the pneumonias, which occur in the early months of postnatal life; the myocarditis, which may be present at birth35; and the osseous lesions.

The relationship between the per¬ sistent virus carrier state and func¬ tion of the immunologie system is dif¬ ficult to resolve, as the facts are somewhat confusing. **It is clear that (1) lymphocytes of normal individuals can be infected in vitro and show decreased phytohemagglutinin (PHA) response after infection36; (2) lympho¬ cytes from infants with rubella syn¬ drome often carry virus for long peri¬ ods after birth37**; (3) infants with congenital rubella syndrome usually have high titers of rubella antibody,34 particularly of the IgM type; (4) **humoral antibody responses to anti¬ gens such as diphtheria toxoid, teta¬ nus toxoid, blood group antigens, and types 1 and 3 poliovirus are decreased in infants with rubella syndrome when they are excreting virus, but not after they stop.**

"**Just recently, absence of cell-me¬ diated immunity to rubella was dem¬ onstrated in nine of 12 infants with rubella syndrome.38 One can formulate an explanation for viral persistence in the following way: 1. Antibody-forming cells (B lym¬ phocytes) are only partly damaged in infants with rubella syndrome.** 2. Thymus lymphocytes are them¬ selves infected with the virus, do not go into mitosis, and therefore have reduced competence to destroy in¬ fected cell clones. The occasional defective PHA response, the relative immune defects, and the slow conver¬ sion from IgM to IgG antibody would be explained by damage to lympho¬ cytes. 3. Antibody to rubella, secreted by uninfected lymphocytes, is stimu¬ lated in utero without the develop¬ ment of tolerance. When uninfected clones of lymphocytes become avail¬ able, they attach to and destroy in¬ fected cells, releasing virus that is then neutralized by the secretions of lymphocytes.

"It is difficult, however, to reconcile the absence of cell-medi¬ ated immunity to rubella in those in¬ fants with rubella syndrome who no longer excrete the virus. Since the In¬ terferon response remains intact in infants with rubella syndrome, per¬ sistence cannot be explained by fail¬ ure of this

mechanism. Patterns of acquisition of cytome¬ galovirus (CMV) antibodies vary from early seropositivity in many developing countries, to slow seroconversion with a high percentage of susceptible child-bearing women in urban centers of industrialized coun¬ tries. …

"… **Fetal brain damage may also result from selective lysis of dividing cells. The best example of this is provided by the H-l picornavirus, which de¬ stroys the cerebellar granular cells in immature cats or hamsters**.48 Other mechanisms that have been shown to operate in animals, but not yet in man, are alteration of neural tube closure by influenza virus, or cavitation of the brain through cell destruction in bluetongue disease of sheep. Recent support for the supposition that influenza A2 virus is teratogenic was provided by the development of hydrocephalus in monkeys inoculated intracerebrally with this virus during the fetal state.49 **Thus, some viruses may be capable of destroying certain brain cells during fetal development, leaving behind morphological de¬ rangement without inflammation."**
https://www.ncbi.nlm.nih.gov/pubmed/165711

Piconavirus, he says is a model of the brain damage we call Autism The vaccines are the live, attentuated viruses. We just wanted to make sure everyone knew that live viruses are the model for the brain damage, and that the rubella vaccine was invented specifically to prevent Autism. It seems most people don't know this.

The CDC claims something to the effect that "the increasing Autism rate could be due to something in the environment," not mentioning which environment. The child's body with viruses from vaccines, congenital CMV, congenital other herpes, the contaminated vaccine vial - contaminated with fungal antigens? No one ever assesses the immune status of the child prior to MMR vaccination, and no one is instructed as to how this testing should be done. The MMR monograph merely claims immunosuppressed children should not be vaccinated.

Thimerosal is put in vaccines to prevent LYMErix, or the immune suppressing fungal endotoxin, OspA and here next we see fungi or fungal antigens injected into babies could be bad, but there is no way to assess the status of the MMR batch or individual vials for mishandling or contamination:

Brain Behav Immun. 2015 Aug;48:301-12. doi: 10.1016/j.bbi.2015.04.020. Epub 2015 May 27.
Postnatal TLR2 activation impairs learning and memory in adulthood.
Madar R1, Rotter A1, Waldman Ben-Asher H2, Mughal MR3, Arumugam TV4, Wood WH 3rd3, Becker KG3, Mattson MP5, Okun E6.

"Neuroinflammation in the central nervous system is detrimental for learning and memory, as evident form epidemiological studies linking developmental defects and maternal exposure to harmful pathogens. Postnatal infections can also induce neuroinflammatory responses with long-term consequences. These inflammatory responses can lead to motor deficits and/or behavioral disabilities. Toll like receptors (TLRs) are a family of innate immune receptors best known as sensors of microbial-associated molecular patterns, and are the first responders to infection. TLR2 forms heterodimers with either TLR1 or TLR6, is activated in **response to gram-positive bacterial infections, and is expressed in the brain during embryonic development. We hypothesized that early postnatal TLR2-mediated neuroinflammation would adversely affect cognitive behavior in the**

adult. Our data indicate that postnatal TLR2 activation affects learning and memory in adult mice in a heterodimer-dependent manner. TLR2/6 activation improved motor function and fear learning, while TLR2/1 activation impaired spatial learning and enhanced fear learning. Moreover, developmentalTLR2 deficiency significantly impairs spatial learning and enhances fear learning, stressing the involvement of the TLR2 pathway in learning and memory. Analysis of the transcriptional effects of TLR2 activation reveals both common and unique transcriptional programs following heterodimer-specific TLR2 activation. These results imply that adult cognitive behavior could be influenced in part, by activation or alterations in the TLR2pathway at birth."
http://www.ncbi.nlm.nih.gov/pubmed/26021559

Research Fraud by CDC officer Suzanne Vernon – trying to make it appear mycoplasma or global immunosuppression is not a factor in Chronic Fatigue Syndrome/Myalgic Encephalomyelitis:

J Med Microbiol. 2003 Nov;52(Pt 11):1027-8.
Absence of Mycoplasma species DNA in chronic fatigue syndrome.
Vernon SD, Shukla SK, Reeves WC.

"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)."
https://www.ncbi.nlm.nih.gov/pubmed/14532349 http://jmm.sgmjournals.org/content/52/11/1027.long

Vernon committed research fraud by centrifuging out the very cells to which mycoplasma adhere and then said, ***"How Amazing! There is no mycoplasma here!!"*** This is important because it shows the CDC does not want to admit to the mechanisms of immunosuppression especially via fungi (no antibodies, increased susceptibility to reactivating latent viruses) because that betrays the source of the Autism pandemic. The same thing is happening when you inject a child with a live attenuated vaccine that is contaminated with fungal antigens…. And against which Thimerosal was added to vaccines as a "preservative."

"Preservative" means "prevent the likes of fungal mycoplasmal antigens or LYMErix growing in vaccine vials with mercury."

---

4) Johns Hopkins' Paul Auwaerter says vaccines fail by becoming reactivated - by reverting to the virulent, active form; that measles itself causes immunosuppression (confirming the synergy with multiple infections); and that the virus symptoms occur "months later" (whereas none of the MMR qualifications followed these children for any of these outcomes much less more than a few weeks):

4.A) J Virol. 1999 Oct;73(10):8791-7.

Altered virulence of vaccine strains of measles virus after prolonged replication in human tis sue.
Valsamakis A1, Auwaerter PG, Rima BK, Kaneshima H, Griffin DE.

"…Our data suggest that the adverse outcomes associated with immunization of patients suffering from congenital and acquired immunodeficiency syndromes are due to the emergence of an MV strain with increased virulence in a host unable to mount a sufficient immune response to clear the originally inoculated vaccine virus. This situation is mimicked in the SCID-hu mouse. Sequence analyses of pMor-1 H and M and other isolates derived from immunodeficient patients demonstrate that these human tissue-passaged vaccine isolates are highly related to parent vaccine strains (1, 15).

"…However, fatal infections have been documented in immunodeficient children vaccinated with these strains (1, 12, 14, 15). **The symptoms of infection occur many months after immunization, and the viruses isolated are similar to the original LA vaccine (1, 15), suggesting that in the absence of an effective host immune response, persistent infection with the vaccine strain can lead to fatal disease. Viruses isolated from these children could potentially represent virulent revertants of the original LA vaccine."**
https://www.ncbi.nlm.nih.gov/pubmed/10482633

Fatal disease or disabling, like, with brain damage ("Autism"), ya mean, right, Paul?

4.B.) Paul Auwaerter *Also* Says (about how measles causes immunosuppression and that the fatal brain infections can come from the vaccines)…

"Increased virulence of vaccine strains isolated from immunocompromised infants with fatal infections was not evident."

J Infect Dis. 1999 Oct;180(4):950-8.
Measles virus infection in rhesus macaques: altered immune responses and comparison of the virulence of six different virus strains.
Auwaerter PG1, Rota PA, Elkins WR, Adams RJ, DeLozier T, Shi Y, Bellini WJ, Murphy BR, Griffin DE.

"Measles remains a major cause of childhood mortality, with questions about virus virulence and pathogenesis still requiring answers. Rhesus macaques were infected with 5 different culture-adapted strains of measles virus, including 2 from patients with progressive vaccine-induced disease, and a sixth nonculture-adapted strain, Bilthoven. All caused infection detectable by reverse transcriptase-polymerase chain reaction and induction of antibody. Chicago-1 and Bilthoven induced viremias detectable by leukocyte cocultivation. Bilthoven induced Koplik's spots, conjunctivitis, and rash. Lymphopenia and depressed interleukin (IL)-2 production were followed by monocytosis and eosinophilia. **All monkeys, including 41 involved in a primate facility outbreak, showed suppressed responses to phytohemagglutinin**. As the rash resolved production of IL-2, IL-1beta, tumor necrosis factor-alpha, IL-6, and IL-5 mRNA increased. Monkeys are useful for studies of measles immunopathogenesis, but virus strains must be carefully chosen. Increased virulence of vaccine strains isolated from immunocompromised infants with fatal infections was not evident.

"Measles is an important human disease that causes the death of □1,000,000 children each year. Most of these deaths are due to secondary infections [1]. This increase in susceptibility to other

pathogens is associated with a well-documented measles-induced immunosuppression [2]. This suppression of immune responses is incompletely understood and is probably multi-factorial: it is likely that different mechanisms are of primary importance in early and late phases of infection. Human studies of necessity focus on the time of the appearance of the rash and thereafter, because that is when measles is recognized clinically. Studies in primates offer the opportunity to look at all phases of infection.

"Many of the deaths associated with secondary infection could be prevented by more widespread application of measles immunization. The vaccine against measles is a live attenuated virus with an impressive record of efficacy and safety, although suppression of immune responses is often detectable after immunization [3]. …"
https://www.ncbi.nlm.nih.gov/pubmed/10479117

Notice he says:

The latest thing Auwaerter said this: "**This suppression of immune responses is incompletely understood and is probably multi-factorial: it is likely that different mechanisms are of primary importance in early and late phases of infection. Human studies of necessity focus on the time of the appearance of the rash and thereafter, because that is when measles is recognized clinically**."

So, inject 3 or more live, allegedly attenuated viruses into infants with immature immune systems, several of which are known to cause immunosuppression, and we know what happens in immunosuppression – reactivated viruses.

Auwaerter is also suggesting there is the non-spots form of the disease, or a non-inflammatory form of the disease. The Lyme criminals say "you cannot have a disease without inflammation," or that "there is no disease other than autoimmune," when we know the opposite is the most damaging outcome: live viruses and infections without immunity. See the quotes by Eugene Shapiro and Paul Offit, below where they reveal this "policy." A woman was vaccinated with immunosuppressing Hep B and then developed MS, yet Offit claims, how "ironic," basically, "since she had no immune response to the vaccine" (implying therefore, she could not possibly have a disease). It's not ironic, it's a ***fact*** and a phenomenon they're trying to hide with snarkasm, harassment of their victims and research fraud (CDC's Vernon, Lyme criminals, and here, in the MMRs, where the CDC and BigPharma throw out vaccine failure or injury data just as they did with LYMErix, claiming those injury/failure cases were "Unconfirmed Lyme").

Next: How convenient for Auwaerter to pretend to know nothing about fungal diseases/antigens or spirochetes that shed them, ***and the fact that they casue immunosuppression***. His office literally returned a phone call to us saying, "No, Auwaerter does not know what OspA is."

Medscape Infectious Diseases > Auwaerter on Infectious Diseases
COMMENTARY
***Candida auris*: Time to Prick Up Your Ears?**
Paul G. Auwaerter, MD

"Hello. This is Paul Auwaerter, speaking for Medscape Infectious Diseases and from Johns Hopkins University School of Medicine.

"Candida and antifungal resistance, to **me as a nonmycologist**, seems relatively static…"
http://www.medscape.com/viewarticle/872986

Remember from the Occam's Razor, what was unique about Paul Auwaerter was that he claimed on his webpage to have expertise in 2 areas: Lyme and EBV. Curious enough that he excludes this work he did on why the MMR vaccines fail. Auwaerter insists the Cabal is right, and that Lyme is only an autoimmune bad knee and that the post-sepsis Lyme outcome is due to some frail emotional status. Yet here we find him in 1999 reporting on how you should not vaccinate immunosuppressed people with live, attenuated viruses because those viruses could become reactivated (and clearly they did- the were the same "type"). So, while we have claimed that the reason the Cabal and the CDC do not want to admit to immunosuppression/post-sepsis outcomes as the actual diseases of Lyme, CFIDS, Fibro, etc., here we finally have the first proof that our theory was correct. The lies about Lyme and ME/CFS/Fibro have to do with how the pediatric vaccines fail and give these children the very brain damaging viruses claim to prevent - immunisuppression/sepsis.

Auwaerter also reveals two other aspects of these simultaneous scandals: the vaccine brain damaged children are not followed officially, ever, for more than a few weeks in the "safety and efficacy "studies. Secondly, it is very likely the only "adverse events" signs the pediatricians are allowed to report are the "autoimmune" ones, like rashes. We proposed there is a data set somewhere (hidden) of children known to have been vaccinated while immunosuppressed with each live viral vaccine type, which were excluded from the "safety and efficacy" results because the CDC and BigPharma would say, "Oh, well those children should not have been vaccinated in the first place, so we can't count them." And indeed at the end of February, 2017, we found that proof (below).

No Autism groups are asking for or showing the correct data. Most of them are on the Thimerosal-Go-Round – nowhere.

Thimerosal was put in vaccines to prevent LYMErix, really. Exactly. And no vaccine against spirochetal diseases ever prevented spirochetes. The Cabal does not even technically make this claim. They just say OspA or LYMErix creates antibodies, which is false. ***The previous lawsuit against SmithKline-Beecham (GSK) was about not warbning or pre-notifying people with the HLAs for autoimmune arthritis not to get the LYMErix vaccine. But the reality about the vast majority of the adverse events to LYMErix was that they were NON-HLA-LINKED immunosuppression outcomes. We're making the same claim with this paper: No children are assessed for existing co-infection or immunosuppression status prior to vaccination. Pediatricians are not informed as to how to assess immune status.***

5) The MMR Monograph warns against babies actually getting the live viruses or potentially pregnant women (clue), but essentially sloughs off (like a snake) responsibility/liability on the injecting pediatrician:

http://www.merck.com/product/usa/pi_circulars/m/mmr_ii/mmr_ii_pi.pdf

"CONTRAINDICATIONS Hypersensitivity to any component of the vaccine, including gelatin.{40} Do not give M-M-R II to pregnant females; the possible effects of the vaccine on fetal development are unknown at this time. If vaccination of postpubertal females is undertaken, pregnancy should be

avoided for three months following vaccination (see INDICATIONS AND USAGE, Non-Pregnant

”Adolescent and Adult Females and PRECAUTIONS, Pregnancy).
Anaphylactic or anaphylactoid reactions to neomycin (each dose of reconstituted vaccine contains approximately 25 mcg of neomycin). 4 Febrile respiratory illness or other active febrile infection.

”However, the ACIP has recommended that all vaccines can be administered to persons with minor illnesses such as diarrhea, mild upper respiratory infection with or without low-grade fever, or other low-grade febrile illness.{41} Patients receiving immunosuppressive therapy. This contraindication does not apply to patients who are receiving corticosteroids as replacement therapy, e.g., for Addison's disease.

”Individuals with blood dyscrasias, leukemia, lymphomas of any type, or other malignant neoplasms affecting the bone marrow or lymphatic systems. **Primary and acquired immunodeficiency states, including patients who are immunosuppressed in association with AIDS or other clinical manifestations of infection with human immunodeficiency viruses;{41-43} cellular immune deficiencies; and hypogammaglobulinemic and dysgammaglobulinemic states.**

”**Measles inclusion body encephalitis{44} (MIBE), pneumonitis{45} and death as a direct consequence of disseminated measles vaccine virus infection have been reported in immunocompromised individuals inadvertently vaccinated with measles-containing vaccine. Individuals with a family history of congenital or hereditary immunodeficiency, until the immune competence of the potential vaccine recipient is demonstrated.**

What they are saying is, don’t vaccinate someone who is immunosuppressed, but whose pediatrician ever pre-screens for immune incompetence prior to vaccination? We’ve heard of infants with cold viruses going to the pediatrician, being vaccinated, and then being carried out never the same again. You see clearly they write in the MMR “Contraindications” the same warnings we are proving to you – don’t vaccinate someone who is immunosuppressed and be sure the vaccine vials are not contaminated with fungal mycoplasma and the like, but how does anyone know what’re the states of the vaccine vial or the children?

IDSA believes (below) that there is a problem, here, especially regarding the AGE of the vaccinee and they CLAIM basically, that “this has killed some babies” - whose parents were probably blamed; let’s remember Roy Meadows, the original Munch-meister and SIDS deaths -, and that the vaccine schedule “***suits the manufacturers and not their victims.”***

From a book on Adverse Events – it indeed shows they threw out cases where the child should not have been vaccinated from the safety and efficacy data (*kids were immunosuppressed, presently infected with a cold or EBV, etc*) – just as we guessed. See those references they threw out:

Adverse Effects of Vaccines: Evidence and Causality.

Show details
Committee to Review Adverse Effects of Vaccines; Institute of Medicine; Stratton K, Ford A, Rusch E, et al., editors.
Washington (DC): National Academies Press (US); 2011 Aug 25.

”The committee identified 18 publications reporting encephalitis or meningoencephalitis after the administration of vaccines containing measles, mumps, and rubella alone or in combination. Mustafa et al. (1993) described one case of encephalitis developing after administration of a MMR vaccine; however, wild-type measles virus was demonstrated in the patient. Fourteen publications did not provide evidence beyond temporality (Ehrengut and Zastrow, 1989; Fescharek et al., 1990; Forster and Urbanek, 1982; Jagdis et al., 1975; Jorch et al., 1984; Kumar et al., 1982; Landrigan and Witte, 1973; Pollock and Morris, 1983; Ross and Yeager, 1977; Schneck, 1968; Schuil et al., 1998; Shuper, 2011; Wiersbitzky et al., 1992b, 1993a). In addition, five publications reported concomitant infections that could contribute to the development of symptoms (Ehrengut and Zastrow, 1989; Forster and Urbanek, 1982; Jorch et al., 1984; Wiersbitzky et al., 1992b, 1993a). **These publications did not contribute to the weight of mechanistic evidence**.”

“Described below are three publications reporting clinical, diagnostic, or experimental evidence that contributed to the weight of mechanistic evidence.
“Bakshi et al. (1996) described a 16-month-old boy presenting with a focal seizure on the right side and left hemipareses and a left gaze preference 5 months after receiving a measles, mumps, and rubella vaccine and 3 days after undergoing bone marrow transplantation. The patient was administered the vaccine prior to being diagnosed with sickle cell trait and a severe combined immunodeficiency. Serum and CSF were negative for bacteria and fungi. Mumps virus was demonstrated in the urine, serum, and CSF. The patient was diagnosed with meningoencephalitis and died 2 months after the onset of symptoms. Pathological examination of the leptomeninges showed chronic and focally prominent meningitis.
“Lacroix et al. (1995) describe a 5-year-old acquired immune deficiency syndrome (AIDS) patient presenting with fever, generalized seizures, and the inability to stand or walk approximately 2 years after vaccination against measles. The patient died months after presenting with neurological symptoms. Retrospective serum analysis showed measles antibody prior to vaccination. Viral cultures of brain samples were negative for measles virus. Frozen sections of basal ganglia, frontal cortex, and white matter were stained with antibodies against measles virus indicating the presence of measles virus in the brain.
Valmari et al. (1987) described a 7-year-old girl presenting with vomiting, headache, twitching of upper extremities, followed by coma lasting for several hours 54 days after receiving a measles, mumps, and rubella vaccine containing the Moraten measles strain and 5.5 years after receiving a measles vaccine containing the Schwarz measles strain. On the day the measles, mumps, and rubella vaccine was administered the patient complained of back pains leading to a diagnosis of acute lymphoblastic leukemia 23 days after vaccination. The patient presented with the symptoms described above 1 day after the fourth methotrexate treatment. Treatment with acyclovir was started and the patient seemed to improve. Measles virus was demonstrated in the CSF. The patient experienced a recrudescence of the neurological symptoms 58 days postvaccination and fever, photophobia, conjunctival inflammation, and a maculopapular rash 63 days postvaccination. Measles virus was demonstrated in the CSF again.

Weight of Mechanistic Evidence

“Encephalitis is considered a complication of infection with wild-type measles, mumps, and rubella viruses (Gershon, 2010a,b; Litman and Baum, 2010). Encephalitis develops in 1:1,000 to 1:2,000 patients infected with measles virus (Gershon, 2010a). In addition many patients upon recovering suffer from neurologic sequelae (Gershon, 2010a). Encephalitis develops in 1:400 to 1:6,000 patients infected with mumps virus (Litman and Baum, 2010). In patients developing early-onset encephalitis upon infection with mumps virus, the damage to the neurons is by direct viral invasion (Litman and

Baum, 2010). In patients infected with rubella virus, encephalitis develops in 1:5,000 patients (Gershon, 2010b). The committee considers the effects of natural infection one type of mechanistic evidence.
"The three publications described above, when considered together, did not present evidence sufficient for the committee to conclude the vaccine may be a contributing cause of encephalitis after administration of a measles or MMR vaccine. The patients described in the cases above had demonstrated immunodeficiencies. The publications presented evidence of the detection of viral antigens on frozen sections or the isolation of mumps or measles virus from the patients. However, the authors did not identify the virus as vaccine strain.

"The latency between vaccination and the development of encephalitis in the publications described above ranged from **5 months to 2 years**, suggesting persistent viral infection as the mechanism. Direct viral infection and viral reactivation may contribute to encephalitis; however, the publications did not provide evidence linking these mechanisms to MMR vaccine.

"The committee assesses the mechanistic evidence regarding an association between MMR vaccine and encephalitis as weak based on knowledge about the natural infection and three cases.
https://www.ncbi.nlm.nih.gov/books/NBK190025/

As you have just seen, it is precisely as we proposed. The kids being damaged from vaccines were immunosuppressed (or got a contaminated vaccine); there is a warning in the MMR about not vaccinating immunosuppressed children**; they are throwing out the vaccine failure cases by claiming the reversion to wild type can't be distinguished from natural infection**; and no doctor is given a tool for assessing immune status prior to vaccination.

These pediatric vaccines are One Size Fits All and the CDC says these "adverse events" are "a calculated risk." Right now the CDC calculates that the risk of 1:60 kids becoming brain damaged for life is a good risk ☺ The CDC/BigPharma make the claim that vaccines are safe – excluding the phrase "for children who are not already sick or immunosuppressed" - and they BLATANTLY claim that this is not a contributing mechanism. We have shown the mechanism of immunosuppression-reactivates-viruses in parallel with the LYMErix and Lyme, and CFIDS/ME post-sepsis syndrome.

Remember from Paul Auwaerter (and there are other reports on "reversion to wild type" in vaccine failure): "… human tissue-passaged vaccine isolates are highly related to parent vaccine strains (1, 15).
"…However, fatal infections have been documented in immunodeficient children vaccinated with these strains (1, 12, 14, 15). **The symptoms of infection occur many months after immunization, and the viruses isolated are similar to the original LA vaccine (1, 15), suggesting that in the absence of an effective host immune response, persistent infection with the vaccine strain can lead to fatal disease. Viruses isolated from these children could potentially represent virulent revertants of the original LA vaccine."**

The following report refutes the entire concept that live, attenuated viral vaccines is preventing disease; one wonders in Lyme, and Chronic Fatigue Syndrome these childhood vaccine disease or

naturally acquired infections are not reactivated, too, with the herpes:

"**Subclinical [means no spots or lumps or immunosuppression-ish] Infection is Not Uncommon."**

Ugeskr Laeger. 1992 Jul 13;154(29):2008-13.
[***Duration of immunity and occurrence of secondary vaccine failure following vaccination against measles, mumps and rubella].***
[Article in Danish]

"… In rare cases, rubella re-infection has resulted in infection in utero, so that a slight risk of congenital rubella cannot be entirely excluded after successful vaccination. No extensive systematic investigations of the effect of revaccination have been carried out and, similarly, **the optimal interval between two or more vaccinations has not been illustrated in more detail in the literature**. **Subclinical infection is not uncommon after all three vaccines.** Where measles is concerned, immunity may possibly be regarded as a continuum which, depending upon the antibody level, protects the individual from various degrees of clinical disease. If wild virus can be spread via individuals with subclinical infections**, it is doubtful** whether population immunity (herd immunity), which is necessary to eliminate the three diseases, can be attained in large populations."
https://www.ncbi.nlm.nih.gov/pubmed/1509566

Wikipedia spells it out.

See the Wikipedia page on "attenuated vaccine." You will see the exact same claims as above – the dangers are that the vaccines will fail and those children will GET those brain damaging viruses:
https://en.wikipedia.org/wiki/Attenuated_vaccine

Disadvantages [edit]

Code Language for "You could actually GET these viruses."

- Secondary mutation can cause a reversion to virulence.[7]
- Can cause severe complications in immunocompromised patients.[8]
- Some can be difficult to transport due to requirement to maintain conditions (*e.g.* temperature)

And Switzerland and the Netherlands say (March, 2017): The mother was immunosuppressed and may have passed along another vaccine virus or common virus, rendering the child too immunosuppressed to get a fungal Tuberculosis vaccine on top of it:

Vaccine. 2017 Mar 1;35(9):1216-1226. doi: 10.1016/j.vaccine.2017.01.048. Epub 2017 Feb 3.

Safety of live vaccinations on immunosuppressive therapy in patients with immune-mediated inflammatory diseases, solid organ transplantation or after bone-marrow transplantation - A systematic review of randomized trials, observational studies and case reports.
Croce E1, Hatz C2, Jonker EF3, Visser LG3, Jaeger VK4, Bühler S5.

"... In most studies, the administration of live vaccines was safe. However, some serious vaccine-related adverse events occurred. 32 participants developed an infection with the vaccine strain; in most cases the infection was mild. However, in two patients fatal infections were reported: a patient with RA/SLE overlap who started MTX/dexamethasone treatment four days after the YFV developed a **yellow fever vaccine-associated viscerotropic disease** (YEL-AVD) and died. The particular vaccine lot was found to be associated with a more than 20 times risk of YEL-AVD. **One infant whose mother was under infliximab treatment during pregnancy received the BCG vaccine at the age of three months and developed disseminated BCG infection and died**. An immunogenicity assessment was performed in 43 studies. In most cases the patients developed satisfactory seroprotection rates. In the IMID group, YFV and VV demonstrated high seroconversion rates. MTX and tumor necrosis factor inhibitory therapy appeared to reduce immune responses to VV and HZ vaccine, but not to MMR and YF-revaccination. Seroconversion in SOT and BMT patients showed mostly higher rates for rubella than for measles, mumps and varicella."
https://www.ncbi.nlm.nih.gov/pubmed/28162821

These are many examples of how live attenuated vaccines fail by giving people the very diseases the vaccines were intended to prevent.

Policy Baloney and Circle Jerk "Reviews" (where they continually cite and recycle their own former scientific garbage, as seen in the Lyme crymes):

Vaccine. 2003 Sep 8;21(25-26):3954-60.
Unintended events following immunization with MMR: a systematic review.
Jefferson T1, Price D, Demicheli V, Bianco E; European Research Program for Improved Vaccine Safety Surveillance (EUSAFEVAC) Project.
"Public debate over the safety of the trivalent measles, mumps and rubella (MMR) vaccine and the drop in vaccination rates in several countries persists despite its almost universal use and accepted effectiveness. We carried out a systematic review to assess the evidence of unintended effects (beneficial or harmful) associated with MMR and the applicability of systematic reviewing methods to the field of safety evaluation. Eligible studies were comparative prospective or retrospective on healthy individuals up to 15 years of age, carried out or published by 2003. We identified 120 articles satisfying our inclusion criteria and included 22. MMR is associated with a lower incidence of upper respiratory tract infections, a higher incidence of irritability, similar incidence of other adverse effects compared to placebo and is likely to be associated with benign thrombocytopenic purpura (TP), parotitis, joint and limb complaints and aseptic meningitis (mumps Urabe strain-containing MMR). Exposure to MMR is unlikely to be associated with Crohn's disease, ulcerative colitis, autism or aseptic meningitis (mumps Jeryl-Lynn strain-containing MMR). The design and reporting of safety outcomes in MMR vaccine studies, both pre- and post-marketing, are largely inadequate. The evidence of adverse events following immunization with MMR cannot be separated from its role in preventing the target diseases."
https://www.ncbi.nlm.nih.gov/pubmed/12922131

Of course, ***now*** we know ^^ that is all FALSE since they exclude cases where the child should not have been vaccinated due to immunosuppression, especially with a concurrent infection. When they find the MMR viruses associated with encephalitis, they say, "Oh, it must be a wild type strain," not mentioning that the wild type can revert from the vaccine strain and viruses mutate all the time – standard fare.

> The issue is, how dangerous can it be to have three potentially immunosuppressing (we know measles is) vaccines injected several times into infants whose immune systems and especially brain are still developing with no prescreening for immune status? The Autism rate skyrocketing and there is a negative-benefit, a 1:60 brain damage rate.

They, the CDC and others in policy or "Public Health" say they don't know the "mechanism" by which viruses become reactivated in immunosuppression but they certainly do know the mechanisms of sepsis or "overwhelming the immune system" and they know what "cytokine storm" means. They know what happens in the cases of multiple infections – the immune system shuts down. HLA molecules are down-regulated in the body but upregulated in the brain, there is tolerance and cross tolerance, meaning expanding infections and infections types. Etc. They know about the opposite of "autoimmunity" since we caught the CDC lying about it so many times.

We caught the CDC lying about so many things: Dearborn, OspA, Lyme, Chronic Fatigue Syndrome and Suzanne Vernon with the mycoplasma, and now here, blaming the baby-victims and their parents, saying, "Oh, if you got the live reactivated MMR viruses, you caught it from the field. Bad mommies. You did not protect your children."

---

6) CDC's Patent, US # 7,632,510, admitting <u>people can get the diseases from the vaccines</u>

5.A.) ***Methods of inducing flavivirus immune responses through the administration of recombinant flaviviruses comprising an engineered japanese encephalitis virus signal sequence***

"Finally, there is the risk that the virus may not be fully or completely inactivated or attenuated and thus, the vaccine may actually cause disease."

http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetahtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=7,632,510.PN.&OS=PN/7,632,510&RS=PN/7,632,510

CDC SAYS,…

5.B) ***Measles, Mumps, and Rubella -- Vaccine Use and Strategies for Elimination of Measles, Rubella, and Congenital Rubella Syndrome and Control of Mumps: Recommendations of the Advisory Committee on Immunization Practices (ACIP)***

"Updated information on adverse events and contraindications, particularly for persons with severe HIV infection, persons with a egg allergy or gelatin allergy, persons with a history of thrombocytopenia, and persons receiving steroid therapy [are immunosuppressed – SASH]."
http://www.cdc.gov/mmwr/preview/mmwrhtml/00053391.htm

---

7) Pharma SAYS:

We learn from this MRSA vaccine patent, that (US patent 7,771,728, Intercell AG) that there is a risk of reversion to virulence if live attenuated viruses are injected into immunosuppressed persons:

Method for identification, isolation and production of antigens to a specific pathogen

"Several established vaccines consist of live attenuated organisms where the risk of **reversion to the virulent wild-type strain exists. In particular in immunocompromised hosts this can be a live** *(sic)* **threatening scenario.** Alternatively, vaccines are administered as a combination of pathogen-derived antigens together with compounds that induce or enhance immune responses against these antigens (these compounds are commonly termed adjuvant), since these subunit vaccines on their own are generally not effective."
http://patft.uspto.gov/netacgi/nph-Parser?Sect1=PTO1&Sect2=HITOFF&d=PALL&p=1&u=%2Fnetahtml%2FPTO%2Fsrchnum.htm&r=1&f=G&l=50&s1=7,771,728.PN.&OS=PN/7,771,728&RS=PN/7,771,728

Giving brain-damaging meningitis to children via mumps vaccine virus:

Rev Med Virol. 1998 Jul;8(3):129-142.
Genetic studies on a mumps vaccine strain associated with meningitis.
Brown EG1, Wright KE.
Author information
"**Vaccination with mumps measles and rubella (MMR) vaccine containing the live attenuated mumps strain, Urabe AM9, is associated with an increased incidence of meningitis. The isolation of mumps virus from CSF and subsequent identification as Urabe AM9-like by sequence analysis confirmed the causative role of Urabe AM9 vaccine in meningitis**. To assess the role of genetic reversion in vaccine failure, sequence comparisons were made between several genes of Urabe AM9 vaccine and post-vaccination meningitis mumps isolates. An amino acid substitution in the Urabe AM9 HN gene Lys335Glu was not detected in the post-vaccination meningitis isolates suggesting that reversion to wild type sequence was associated with vaccine failure. However, further analysis showed that the vaccine was a mixture of viruses that differed at aa 335 of HN, possessing either the wild type Lys335 or the mutant Glu335, whereas the clinical isolates were homogeneous and possessed the wild type Lys335. Passage of the Urabe AM9 vaccine preparations in Vero cells resulted in the amplification of the Glu335 virus, however the post-vaccination meningitis isolates (Lys335) grew better in Vero cells than Urabe AM9 vaccine. A virus isolate, similar to the post-vaccination isolates was obtained from the vaccine suggesting that the strain responsible for vaccine failure was a pre-existing component of the vaccine and was not necessarily the result of reversion. The Urabe AM9 vaccine is a heterogeneous mixture of genotypes that differ in virulence with the HN Glu335 viruses

being attenuated and at least a subset of the HN Lys335 viruses that are associated with disease. The Glu335 mutation may be among a class of attenuating mutations identified in several neurotropic viruses that involve charged amino acids in neutralising epitopes of receptor binding proteins. Copyright 1998 John Wiley & Sons, Ltd."
https://www.ncbi.nlm.nih.gov/pubmed/10398501

And:

Acta Neuropathol. 2017 Jan;133(1):139-147. doi: 10.1007/s00401-016-1629-y. Epub 2016 Oct 21.
Deep sequencing reveals persistence of cell-associated mumps vaccine virus in chronic encephalitis.
Morfopoulou S1, Mee ET2, Connaughton SM2, Brown JR3, Gilmour K4, Chong WK5, Duprex WP6, Ferguson D2, Hubank M7, Hutchinson C8, Kaliakatsos M9, McQuaid S10,11, Paine S8,12, Plagnol V13, Ruis C14, Virasami A8, Zhan H15, Jacques TS8,16, Schepelmann S2, Qasim W17,18, Breuer J14,3.

"Routine childhood vaccination against measles, mumps and rubella has virtually abolished virus-related morbidity and mortality. Notwithstanding this, we describe here devastating neurological complications associated with the detection of live-attenuated mumps virus Jeryl Lynn (MuVJL5) in the brain of a child who had undergone successful allogeneic transplantation for severe combined immunodeficiency (SCID). **This is the first confirmed report of MuVJL5 associated with chronic encephalitis and highlights the need to exclude immunodeficient individuals from immunisation with live-attenuated vaccines.** The diagnosis was only possible by deep sequencing of the brain biopsy. Sequence comparison of the vaccine batch to the MuVJL5 isolated from brain identified biased hypermutation, particularly in the matrix gene, similar to those found in measles from cases of SSPE. The findings provide unique insights into the pathogenesis of paramyxovirus brain infections."
https://www.ncbi.nlm.nih.gov/pubmed/27770235

---

8) CDC on how humans can get diseases from immunosuppressed vaccinated animals:

Human Exposure to Brucella abortus Strain RB51 -- Kansas, 1997

http://www.cdc.gov/mmwr/preview/mmwrhtml/00051495.htm

[In the above, an immunosuppressed pregnant cow was given a Brucella (LYMErix-like) "live attenuated" vaccine and the baby cow ended up with the disease, which then was transferred to the humans handling the cow and her dead baby. This parallels what is happening to children who are vaccinated while immunosuppressed, or who receive mycoplasmally (LYMErix-like) contaminated vaccines.]

More:

Clin Infect Dis. 2003 Aug 1;37(3):407-14. Epub 2003 Jul 22.
Human illness associated with use of veterinary vaccines.
Berkelman RL1.
"Veterinary vaccines are being used with increasing frequency in the United States to protect the health of animals. However, humans may be inadvertently exposed to these products by means of unintentional inoculation or other routes of exposure. The potential for both exposure and for adverse consequences secondary to exposure to veterinary vaccines may be growing. With the exception of

brucellosis vaccines, there have been few reports of suspected or confirmed adverse events in humans associated with the use of animal vaccines, but it is unclear whether that is because few adverse events occur or because adverse events are not recognized and/or reported. Results of a search for relevant literature and of communications with health officials at governmental and private institutions suggest that enhanced efforts are needed to recognize and to prevent human illness associated with use of veterinary vaccines.

"Veterinary vaccines are being used with increasing frequency in the United States to protect the health of animals. In addition to their direct benefit to animals, these vaccines have also markedly decreased the risk of transmission of many zoonotic infections (e.g., rabies and brucellosis) to humans. The US Department of Agriculture currently licenses >2000 vaccines for use in animals [1]. Most of these vaccines are inactivated formulations, but >500 live vaccine formulations for animals are also licensed. Veterinary vaccines are intended only for use in animals and are not tested for safety in humans. However, humans may inadvertently be exposed to these products by means of unintentional inoculation or other routes of exposure.

https://www.ncbi.nlm.nih.gov/pubmed/12884166
https://academic.oup.com/cid/article/37/3/407/437242/Human-Illness-Associated-with-Use-of-Veterinary

The point is that an immunosuppressed animal is infectious for the vaccine virus, and that, like we hear in the Humira and Stelara commercials, "don't go near anyone who recently had a vaccine if you are taking these immunosuppressive drugs," because the ***other person harbor live viruses, and you are immunosuppressed*** – a model to which the CDC does not otherwise admit. Why. Because then people will say, "Oh, the babies are getting the live viruses and may be immunosuppressed at the same time? Is that what's happening with the MMR Autism vaccines?"

---

9) CDC SAYS… stress hormones like cortisol activate viruses (but when fungi activate latent viruses it is not reversible, as is shown in other EBV-diseases such as Lupus, cancer, MS, and CFIDS/Lyme):

2012; ***The effect of exogenous corticosterone on West Nile virus infection in Northern Cardinals (Cardinalis cardinalis)***
"Corticosterone was administered at levels that individuals enduring chronic stressors (i.e ., long-term inclement weather, food shortage, anthropogenic pollution) might experience in the wild. Corticosterone greatly impacted mortality: half of the corticosterone-implanted cardinals died between five - 11 days post-inoculation whereas only one of nine sham-implanted (control) birds died. … No differences were found in viral titer between corticosterone- and sham-implanted birds. However, cardinals that survived infections had significantly higher average body temperatures during peak infection than individuals that died… In sum, this study indicates that elevated corticosterone could affect the survival of WNV-infected wild birds, suggesting that populations may be disproportionately at-risk to disease in stressful environments."
http://7thspace.com/headlines/410671/the_effect_of_exogenous_corticosterone_on_west_nile_virus_infection_in_northern_cardinals_cardinalis_cardinalis.html

The same is true for humans and cortisol and the activation of latent herpesviruses; just go to PubMed and look for **astronauts and EBV, or medical students and EBV**,… – you'll see cortisol come up ;); when astronauts or wannabee doctors are stressed out, they may have cortisol-activated EBV. We've made this information into a criminal charge sheet (Somatoform/Wessely) to show the slander and

libel - and the CDC-associated perps ***know -*** yet claim that us regular humans, no, we're assigned "**some psychiatric disorder**." Why the big secret, no one knows since it's common knowledge that arrogance is the cowardly calling card of walking anal sphincters. This harassment of the CDC's victims is called a "Deprivation of Rights via Color of Law" crime.

---

10) IDSA admits vaccines not safe for babies:

"Amanda Jezek, the vice president of Public Policy and Government Relations at the Infectious Diseases Society of America (IDSA), in Arlington, Va., said there is concern that this push to recommend a vaccine before the ACIP has reviewed the evidence would completely "jeopardize the integrity of ACIP's recommendations."
"Most of the vaccinations given in this country are received by those younger than 2 years of age, so assuring the safety and efficacy of vaccines is paramount. Every year, more than 40 million vaccines are given to children younger than 1 year of age, usually between 2 and 6 months of age, Dr. Temte said. ***At this age, infants are at greatest risk for certain serious medical adverse events, including high fevers, seizures and sudden infant death syndrome****, according to the U.S. Vaccine Adverse Event Reporting System. Therefore, it is important for the ACIP to consider carefully the risks versus the benefits before making a recommendation rather than be on a forced schedule that suits the manufacturer as opposed to the patient."*
http://www.idse.net/ViewArticle.aspx?d=Public%2BHealth&d_id=212&i=August+2015&i_id=1215&a_id=33373

Amanda Jezek, the vice president of Public Policy and Government Relations at the Infectious Diseases Society of America (IDSA), in Arlington, Va., said there is concern that this push to recommend a vaccine before the ACIP has reviewed the evidence would completely "jeopardize the integrity of ACIP's recommendations."

Most of the vaccinations given in this country are received by those younger than 2 years of age, so assuring the safety and efficacy of vaccines is paramount. Every year, more than 40 million vaccines are given to children younger than 1 year of age, usually between 2 and 6 months of age, Dr. Temte said. At this age, infants are at greatest risk for certain serious medical adverse events, including high fevers, seizures and sudden infant death syndrome, according to the U.S. Vaccine Adverse Event Reporting System. Therefore, it is important for the ACIP to consider carefully the risks versus the benefits before making a recommendation rather than be on a forced schedule that suits the manufacturer as opposed to the patient.

In addition, the ACIP is tasked with choosing the components of the annual influenza vaccination, which changes every year as the virus mutates throughout the season. Experts around the world track these mutations to predict which flu strains will be predominant in the following season. The ACIP makes recommendations about the strains to include in next year's flu vaccination. Just under 1 million U.S. infants, children and adults received the influenza vaccine during the 2012-2013 flu season.

Every situation surrounding a recommendation is different, Dr. Temte said. For instance, the pneumococcal conjugate vaccine (Prevnar 13, Pfizer) is recommended to protect against pneumococcal disease. The vaccine has been indicated for children for some time, but received a new indication for adults older than 50 years of age in December 2011.

The vaccine received accelerated approval by the FDA for the adult indication without clinical data to show efficacy in adults. Those data did not come until the company conducted a postlicensure trial involving 84,000 individuals. It was almost two years after the new indication was granted before the ACIP had the safety and efficacy data to make a good recommendation about the vaccine.

"I'd like to get some explanation about how we can compress the acquiring of information into a very limited time frame," Dr. Temte said.

11) Offit reveals vaccines fail via immunosuppression via snarkasm ("how ironic, since the lady did not produce antibodies"), and Eugene Shapiro reveals you cant have a "disease" unless it is "inflammatory" or "autoimmune" or results in "too many antibodies."

In one of the most revealing of all anti-anti-vaxxer reports, **Paul Offit** shows that he absolutely knows vaccines can cause immunosuppression, and we know what happens in cases of immunosuppression from the sum of the data in the Occam's Razor criminal charge sheet. **The woman he talks about acquired multiple sclerosis, which comes from what? Immunosuppression-reactivated Epstein-Barr, which pretty surely is associated with the development of Multiple Sclerosis** (and post-sepsis Chronic Fatigue/Lyme):

N Engl J Med. 2008 May 15;358(20):2089-91. doi: 10.1056/NEJMp0802904.
Vaccines and autism revisited--the Hannah Poling case.
Offit PA1.

"No case, however, represented a greater deviation from the VICP's original standards than that of Dorothy Werderitsh, who in 2006 successfully claimed that a hepatitis B vaccine had caused her multiple sclerosis. By the time of the ruling, several studies had shown that hepatitis B vaccine neither caused nor exacerbated the disease, and the Institute of Medicine had concluded that "evidence favors rejection of a causal relationship between hepatitis B vaccine and multiple sclerosis."2 But the VICP was less impressed with the scientific literature than it was with an expert's proposal of a mechanism by which hepatitis B vaccine could induce autoimmunity **(an ironic conclusion, given that Dorothy Werderitsh never had a detectable immune response to the vaccine**)."
http://www.nejm.org/doi/full/10.1056/NEJMp0802904

Be sure to read the whole "report." The woman mentioned here, Dorothy Werderitsch, had no immune response to the vaccine - that means she was immunosuppressed. Right, Offit, thanks for revealing it all in not only this example, but all your others in that report. Also, you will find, here, the Hepatitis B vaccines cause immunosuppression.

Again, **Yale's Eugene Shapiro** (who assaulted Czech children with a known fake vaccine, LYMErix, which they knew would do those children no good because there is none of that kind of OspA in Europe, and was just an experiment to see how bad were the adverse events) saying you cant have a disease without inflammation from the Lobbyists Handbook:

Having a "Disease" without classic "Inflammation" or "Autoimmunity" – This FRAUD has to do with the Autism pandemic:

The Lyme criminals claim you can't have a "disease" unless you have inflammation or an autoimmune outcome. Of course, such a claim betrays the source of the Autism pandemic. The kids are getting the viruses instead of the protection and this is shown in many places and is called an "adverse event."

Yale's Eugene Shapiro in PBS' "Life on Earth Series":

"TOOMEY: But most physicians think there's good reason to discount the possibility of chronic Lyme Disease.

"SHAPIRO: What some people would have you believe is that there are two different diseases.

"TOOMEY: Yale physician Eugene Shapiro says first, take the obvious case of Lyme Disease that usually starts with a distinctive rash and can lead to arthritis and facial paralysis.

"SHAPIRO: Somehow, for that form of the disease, antibiotics are effective. They do fine. But then there's some other form of the disease which is, you can't put your hand around it. They don't have objective findings of inflammation, which is the way bacteria cause disease.

"TOOMEY: What these patients do have are symptoms that doctors call nonspecific. They span a broad spectrum. Emotional problems, as in the case of Lisa's daughter; or fatigue, muscle pains, depression. Doctor Shapiro helped write the Lyme treatment guidelines put out by the Infectious Diseases Society of America. Guidelines that state even the most advanced cases of infection can be eradicated with two months of oral or intravenous antibiotics. But for the patients with these ongoing, nonspecific symptoms, the maximum treatment didn't seem to work.

"SHAPIRO: They would have you believe that form of the disease, somehow this is, the bacteria knows to act differently and it doesn't respond to antibiotics in this sense. It really doesn't make any sense.

http://loe.org/shows/segments.html?programID=00-P13-00037&segmentID=1

Of course it does make sense: Spirochaeta are not bacteria, in the sense that 1) they are their own Phylum, 2) shed fungal antigens and 3) have no LPS. But, what would Shapiro know about science? His undergraduate degree is in English Literature. The point is, you see here, Shapiro, et al, claim that you can't have a disease unless you have inflammation or an autoimmune one. Spirochetes are notorious immune suppressors like Tuberculosis because the Osps are triacylated lipoproteins, which means they are managed by Toll Like Receptors 1 and 2, and which means they are FUNGAL. (Which means THEY ARE *NOT* REGULAR "BACTERIA.") Thimerosal is put in vaccines to prevent the very shed fungal antigens from spirochetes or anything, obviously, fungal, like mycoplasma or chlamydia, etc.

12) Hep B associated with immunosuppression via TLR2 agonism, the vaccine antigen is called "HbsAg")

https://www.ncbi.nlm.nih.gov/pubmed/?term=Hepatitis+B+recombinant+antigen+and+tlr

Hepatology. 2009 Apr;49(4):1132-40. doi: 10.1002/hep.22751.

Hepatitis B virus suppresses toll-like receptor-mediated innate immune responses in murine parenchymal and nonparenchymal liver cells.

Wu J1, Meng Z, Jiang M, Pei R, Trippler M, Broering R, Bucchi A, Sowa JP, Dittmer U, Yang D, Roggendorf M, Gerken G, Lu M, Schlaak JF.

"We have previously shown that Toll-like receptor (TLR)-activated murine nonparenchymal liver cells [(NPC); Kupffer cells (KC), liver sinusoidal endothelial cells (LSEC)] can suppress hepatitis B virus (HBV) replication. Therefore, the aim of this study was to investigate whether HBV has the ability to counteract the TLR-mediated control of its replication. Freshly purified murine hepatocytes and NPCs obtained from C57BL6 mice were stimulated by TLR 1-9 ligands in the presence or absence of hepatitis B surface antigen (HBsAg), hepatitisB e antigen (HBeAg), HBV virions, or supernatants from HBV-producing HBV-Met cells, and HBV replication was suppressed by anti- hepatitis B virus X protein (HBx) small interfering RNA (siRNA) in HBV-Met cells. Supernatants were collected and tested for antiviral cytokines by viral protection assay. HBV gene expression and replication was analyzed by southern blot. RNA and proteins were analyzed by quantitative reverse transcription polymerase chain reaction (RT-PCR) or western blot and enzyme-linked immunosorbent assay, respectively**. Pretreatment of hepatocytes and NPCs with HBV-Met cells supernatants, HBsAg, HBeAg, or HBV virions almost completely abrogated TLR-induced antiviral activity, which correlated with suppression of interferon beta (IFN-beta) production and subsequent interferon-stimulated gene induction as well as suppressed activation of interferon regulatory factor 3 (IRF-3), nuclear factor kappa B (NF-kappaB), and extracellular signal-regulated kinase (ERK) 1/2.** In HBV-infected HBV-Met cells, TLR stimulation did not induce antiviral cytokines in contrast to primary hepatocytes. TLR-stimulated expression of proinflammatory cytokines [tumor necrosis factor alpha (TNF-alpha), interleukin-6 (IL-6)], and activation of IRF-3 was suppressed after up-regulation of HBV replication in HBV-Met cells. Accordingly, suppression of HBV replication by siRNA led to activation or expression of proinflammatory transcription factors and cytokines.

CONCLUSION:

Our data indicate that HBV can suppress the TLR-induced antiviral activity of liver cells. This has major implications for the interaction between HBV and the immune system."

Here we have just shown that Hep B (HbsAg – the vaccine) causes immunosuppression, while Offit says: "But the VICP was less impressed with the scientific literature than it was with an expert's proposal of a mechanism by which hepatitis B vaccine could induce autoimmunity **(an ironic conclusion, given that Dorothy Werderitsh never had a detectable immune response to the vaccine**)."

Offit says, yes, she was immunosuppressed - probably from the vaccine since the Hep B vaccine causes immunosuppression. Or maybe she had a cold and should not have been vaccinated at the time.

And what happens with immunosuppression? Right, the reactivation of the herpes viruses which can lead to Multiple Sclerosis. It's hardly "ironic." You can tell that **the denial of failed vaccines** that fail by giving people/children the actual viruses (the Hep B vaccine is a recombinant antigen and not a whole live virus), or cause immunosuppression and then the reactivation of latent herpesviruses (Lyme and ME/CFS),… is public policy. Auwaerter does it, and Offit does it.

The vaccines fail via immunosuppression then reactivation-of-latent viruses or activation-of-live-attenuated vaccine viruses – disease conditions not admitted to by the U.S. Government as policy. It's policy.

There is no NIIID. Not with 1:60 kids brain damaged for life from these insane non-vaccines. The whole country would come to a standstill if it was known by the general population that a program exists to throw out a 25% or more of the humans in this country just to save the international reputations of BigPharma and the incompetent bioweaponeers of the CDC.

13) Claiming the Chicken Pox vaccine reactivates to live viruses in immunosuppressed individuals (how unsurprising)

Nat Med. 2000 Apr;6(4):451-4.
Varicella vaccination: evidence for frequent reactivation of the vaccine strain in healthy children.
Krause PR1, Klinman DM.

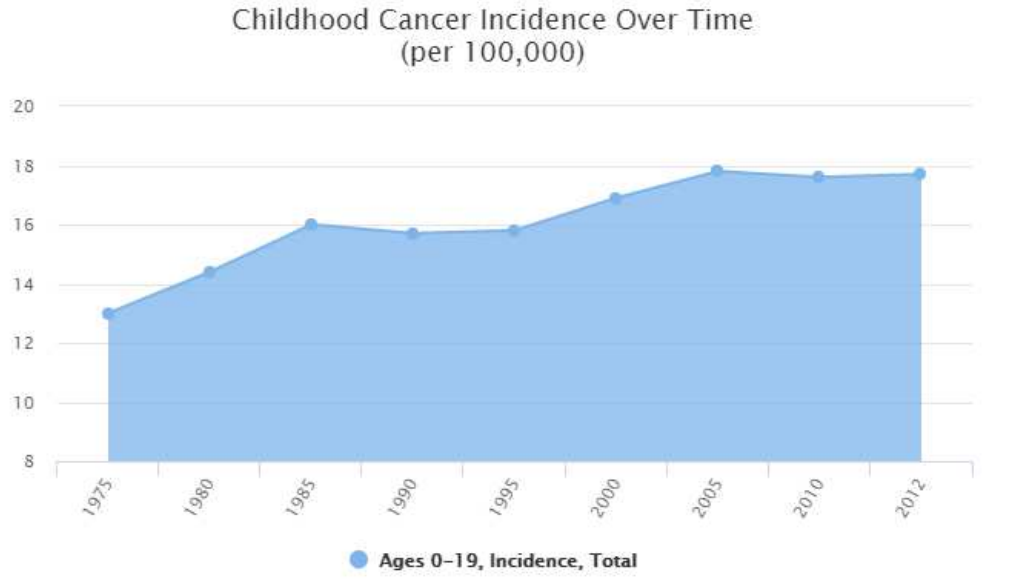
Wild-type varicella zoster virus (VZV) causes chickenpox, a common childhood illness characterized by fever and a vesicular rash and rare serious complications. Wild-type VZV persists in a latent form in the sensory ganglia, and can re-activate to cause herpes zoster. More than 10 million American children have received the live attenuated Oka strain VZV vaccine (OkaVZV) since its licensure in 1995. Pre-licensure clinical studies showed that mean serum anti-VZV levels among vaccinees continued to increase with time after vaccination. This was attributed to immunologic boosting caused by exposure to wild-type VZV in the community. Here, we examine the alternative, that large-scale asymptomatic reactivation of OkaVZV might occur in vaccinees. We analyzed serum antibody levels and infection rates for 4 years of follow-up in 4,631 children immunized with OkaVZV. Anti-VZV titers decreased over time in high-responder subjects, but rose in vaccinees with low titers. Among subjects with low anti-VZV titers, the frequency of clinical infection and immunological boosting substantially exceeded the 13%-per-year rate of exposure to wild-type varicella. These findings indicate that OkaVZV persisted in vivo and reactivated as serum antibody titers decreased after vaccination. This has salient consequences for individuals immunized with OkaVZV.
https://www.ncbi.nlm.nih.gov/pubmed/10742154

Many reports here:

https://www.google.com/?gws_rd=ssl#q=vaccine+virus+reactivation&*

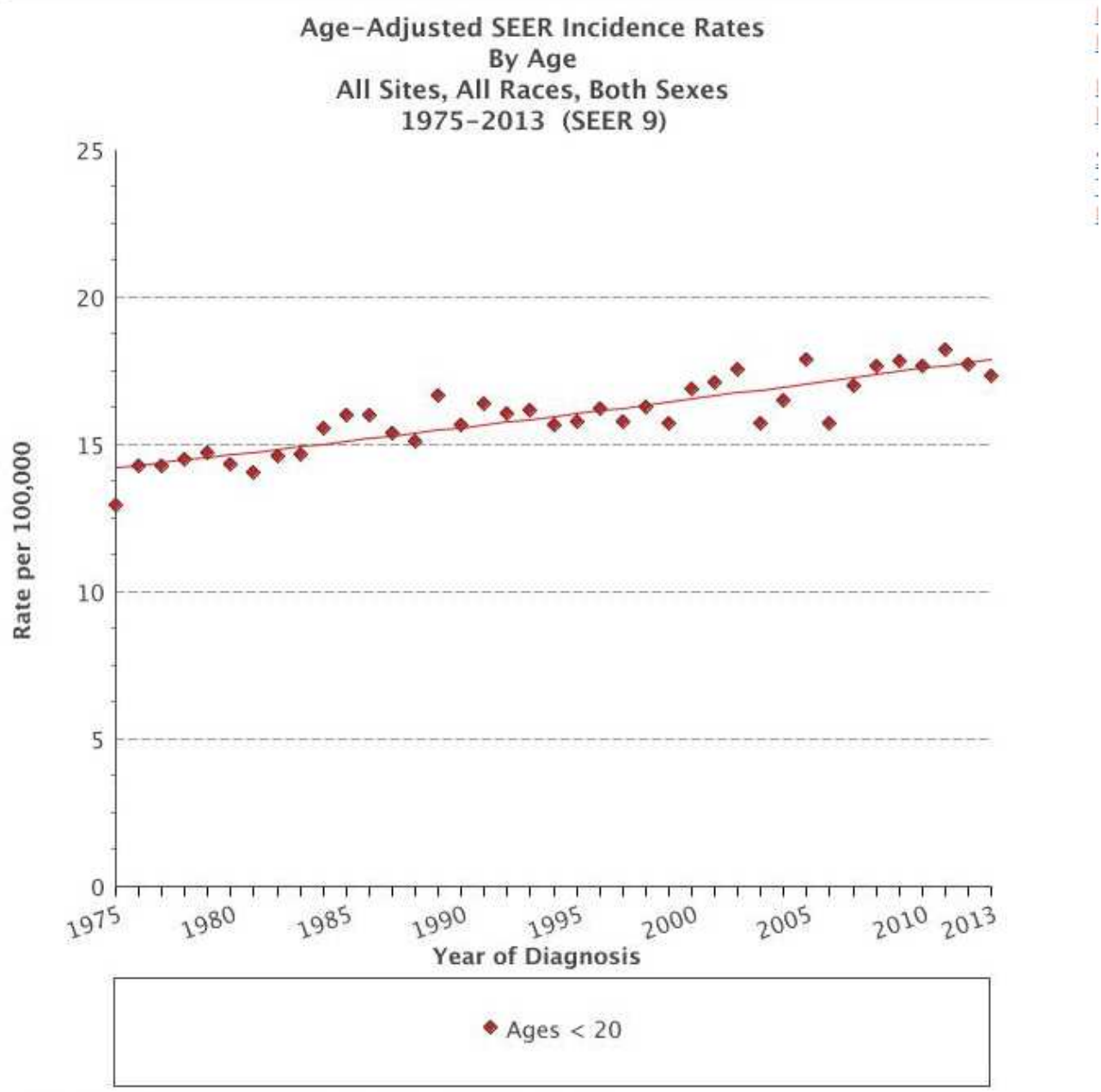
14) Cancer and Autism, naturally co-trending since hypervaccination causes immune-blunting just like old age immunity (cancer, like Lyme and CFIDS, is classified as "a failure of the immune system" which is at the other end of the immunity spectrum from autoimmunity):

Even as the cure rate continues to improve, the incidence of childhood cancer has been steadily increasing over the last few decades, from about 13 children per 100,000 in 1975 to over 17 children per 100,000 since 2005.



Source: Surveillance, Epidemiology, and End Results (SEER) Program (seer.cancer.gov) SEER 9 areas, 1975-2012, Age 0-19.

https://curesearch.org/Incidence-Rates-Over-Time



https://seer.cancer.gov/faststats/selections.php?#Output

15) The effect and danger of "over-vaccination" with multiple live attenuated viruses. Here someone repeats our own proposal: the CDC is creating a new toxic cauldron in hypervaccinated humans in an environment of so many known immune suppressors, such as mold or Lyme, or stress or inhalation of diesel bus fumes, not to mention measles itself is immunosuppressive:

Evolution (N Y). 2011 Dec; 4(4): 635–643.
doi: 10.1007/s12052-011-0365-y

The double-edged sword: How evolution can make or break a live-attenuated virus vaccine

Kathryn A. Hanley

"Too much of a good thing: competition and facilitation among strains in multi-strain vaccines

"Vaccine viruses can exhibit quite different dynamics when administered in combination with other vaccine viruses than when administered alone. At one end of the spectrum, the individual components of a multi-strain vaccine can show "interference", in which one or more strains replicate to lower levels or stimulate a poorer immune response in a combined vaccine than a single-strain vaccine. Interference can be the product of immunodominance, direct competition for cellular or viral replication machinery, or immune-mediated apparent competition. Such interference has long been recognized; Sabin and his colleagues [56] pointed out in 1960 that the individual components of OPV were more efficacious when administered individually (as monovalent vaccines) than when administered together in a trivalent vaccine. Subsequently, multiple instances of interference among live-attenuated vaccine viruses have been documented ([57] and references therein). At the other end of the spectrum, facilitation, in which the replication or immunogenicity of attenuated viruses is enhanced in combination, could also occur. While this phenomenon has not been documented in vaccine viruses to the best of my knowledge, previous studies have certainly shown facilitation between unrelated viruses during concurrent infection [46, 58-60], thus a similar interaction between vaccine viruses is possible. While the outcome of interference and facilitation have been described, the mechanisms driving these dynamics are poorly understood. **However as the number of new live-attenuated vaccines targeted to the already overscheduled child continues to increase, it is becoming increasingly important to gain a mechanistic understanding of the ecological interactions among these attenuated viruses**.
https://www.ncbi.nlm.nih.gov/pubmed/22468165
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3314307/

And as we know, the rubella vaccines were intended to defeat the brain damage we call congenital Autism, so we wonder how giving it to neurodeveloping babies could be a good thing. The proposal probably suits the manufacturers and not the victims.

The Herd Effect tall tale (or, half-tale) – has everything to do with vaccinated people giving those live viruses from the vaccines to the un-vaccinated. That means of course, the viral vaccines are active, have been active and may, as active, brain damaged children at a too-early age.

This term is probably vague for many, yet here the Wikipedia page explains that it has to do with people who cant be vaccinated because of conditions we are not allowed to mention (immunosuppression, post-sepsis syndrome, you know, the classes of people otherwise known as somatoform):

"**Herd immunity** (also called **herd effect**, **community immunity**, **population immunity**, or **social immunity**) is a form of indirect protection from infectious disease that occurs when a large percentage of a population has become immune to an infection, thereby providing a measure of protection for individuals who are not immune.[1][2] In a population in which a large number of individuals are immune, chains of infection are likely to be disrupted, which stops or slows the spread of

disease.[3] The greater the proportion of individuals in a community who are immune, the smaller the probability that those who are not immune will come into contact with an infectious individual.[1] Individual immunity can be gained through recovering from a natural infection or through artificial means such as vaccination.[3] Some individuals cannot become immune due to medical reasons and in this group herd immunity is an important method of protection.[4][5] Once a certain threshold has been reached, herd immunity gradually eliminates a disease from a population.[5] This elimination, if achieved worldwide, may result in the permanent reduction in the number of infections to zero, called eradication.[6] This method was used for the eradication of smallpox in 1977 and for the regional elimination of other diseases.[7] Herd immunity does not apply to all diseases, just those that are contagious, meaning that they can be transmitted from one individual to another.[5] Tetanus, for example, is infectious but not contagious, so herd immunity does not apply.[4]"
https://en.wikipedia.org/wiki/Herd_immunity

In other words, the idea that people are walking around with live unattenuated vaccine viruses such that they could give them to someone else,… is never supposed to cross paths with the idea that infants are getting whacked with 3 or more viruses at the same time, one of which is Rubella and which is known to cause the brain damage we call Autism. And you have previously seen, they know the vaccine viruses are active, live and can cause disease. "Herd immunity" - which no one really understands - is about "everyone get vaccinated so the ones who cant get vaccine because they're immunosuppressed and might get those live viruses." Ahem, what about the babies who are never pre-screened for immune status?

16) Rubella & "Low Responders" … are they actually the ones with chronic active disease?

Nat Med. 2000 Apr;6(4):451-4.
Varicella vaccination: evidence for frequent reactivation of the vaccine strain in healthy children.
Krause PR1, Klinman DM.

"Here, we examine the alternative, that large-scale asymptomatic reactivation of OkaVZV might occur in vaccinees. We analyzed serum antibody levels and infection rates for 4 years of follow-up in 4,631 children immunized with OkaVZV. Anti-VZV titers decreased over time in high-responder subjects, but **rose in vaccinees with low titers**. Among subjects with low anti-VZV titers, the frequency of clinical infection and immunological boosting substantially exceeded the 13%-per-year rate of exposure to wild-type varicella. These findings indicate that OkaVZV persisted in vivo and reactivated as serum antibody titers decreased after vaccination. This has salient consequences for individuals immunized with OkaVZV."
https://www.ncbi.nlm.nih.gov/pubmed/10742154

Low responders, who later become higher antibody-responders, are responding to the reactivated viruses? At least in this case, Varicella…

… And here we show low antibody responders from a Rubella vaccine

Calif Med. 1971 Nov;115(5):16-22.

Transmission of rubella vaccine virus from vaccinees to contacts.
Wilkins J, Leedom JM, Salvatore MA, Portnoy B.
"The report presents evidence of the transmission of hpv-77 derived rubella vaccine virus from vaccinees to two susceptible contacts. The first instance of transmission was to a child who served as a transmission control on a "closed" study ward, and the second was to an antibody-negative mother in an "open" family study. Neither of these persons had any clinical evidence of rubella. Both had significant increases in rubella hemagglutination inhibiting (hai) antibody titers, but detectable complement fixing (cf) antibodies did not develop in either. With the kind of antigen used in our rubella cf test, this pattern of serologic response is characteristic of, but not diagnostic of, infection with the rubella vaccine virus. The serological evidence which was compatible with rubella vaccine virus infection, the complete absence of serologic or clinical evidence of "wild" rubella virus infections among the other four rubella susceptible transmission control children and the security precautions employed to ensure isolation on the "closed" ward, make "wild" rubella virus infection extremely unlikely. The evidence for rubella vaccine virus infection in the other susceptible contact is not as conclusive, because "wild" rubella virus infection is difficult to rule out in any person living in an "open" family situation. Nevertheless the need for more data is emphasized by the virtual certainty that rubella vaccine virus transmission did occur in the subject on the isolation ward, plus the high probability that the infection observed in the family group setting also represented transmission of rubella vaccine virus. Such data can only come from close surveillance of recipients of live rubella virus vaccines and their contacts in the future."
https://www.ncbi.nlm.nih.gov/pubmed/5117594

And another:

Calif Med. 1969 Mar;110(3):224-7.
Viremia in a recipient of HPV-77 Rubella virus vaccine.
Wilkins J, Salvatore MA, Leedom JM, Portnoy B.
Abstract
"A live rubella virus vaccine, HPV-77 (High Passage Virus - 77 tissue culture passages) was administered subcutaneously to eight rubella-susceptible children housed in an isolation ward. One blood specimen, taken on the tenth day after vaccination, from one of the eight vaccines, yielded a rubella virus. This virus had laboratory markers which were "vaccine-like." To our knowledge, this represents the first isolation of rubella virus from the blood of a recipient of HPV-77 vaccine. **However, the consistent antibody responses among vaccinees and the regular presence of rubella virus in their pharynges argue that viremia occurs in almost every susceptible recipient. The most logical explanation for the failure to document viremia in other recipients of HPV-77 vaccine is that the viremia is ordinarily low grade or transient or both.**
https://www.ncbi.nlm.nih.gov/pubmed/5773481

We are merely making the point we see no one else making: The Rubella vaccine was invented in the first place to prevent congenital Autism. When the MMR vaccines fail because children are not pre-screened for immune status could it be the Rubella virus from the vaccines causing the same brain damage?

No one seems to be talking about it. Mumps without the Lumps, Measles without the Spots, and Rubella without the rash. Why? Immunosuppression and vaccination is not the normal route of infection.

17) Synergism, and Chronic Fatigue Syndrome (post sepsis without the spirochetes)

You've already seen Auwaerter on how measles is immunosuppressive, and the book about failed vaccines say: "The latency between vaccination and the development of encephalitis in the publications described above ranged from **5 months to 2 years**, suggesting persistent viral infection as the mechanism. Direct viral infection and viral reactivation may contribute to encephalitis;"

That's basically the definition of post-sepsis Chronic Fatigue Syndrome, which is a condition simultaneously denied by the CDC. They say there is no such thing as chronic active viral and other infections because that would expain the Autism pandemic from vaccines.

But Dual Infections or multiple infections could be bad. "Doctors" are supposed to know this basic medical science – the synergy where **Malaria-activated Epstein-Barr** and caused **Burkitt's Lymphoma** due to the immunosuppression from Malaria Plus Epstein-Barr virus. The following report could be a good cross over point between the failed vaccines that fail via immunosuppression or "overwhelming the immune system, or, "especially due to exposure to TLR2/1 agonists," and…, "***is*** the model that parallels the post-septic shock outcomes of ME/CFS, Lyme and the other harassed victims groups."

We really don't want to say that "doctors are supposed to know this stuff," but doctors are supposed to know this stuff and it shouldn't have to be revealed by the crime victims. We're forced to live in an alternate universe. We're RICO organized crime victims like mob-poison-survivors or drive-by-shooting survivors solving our own case by hacking and taping and recording mob emails and phone calls, outlining the who what where of the crime for the stupid lazy cops or FBI. Yet, we're doing that exact thing while the dumb "doctors" look down their noses.

Malar J. 2010 Mar 1;9:64. doi: 10.1186/1475-2875-9-64.
Dual effect of Plasmodium-infected erythrocytes on dendritic cell maturation.
Bettiol E1, Carapau D, Galan-Rodriguez C, Ocaña-Morgner C, Rodriguez A.

"It was found that intact erythrocytes infected with P. yoelii do not induce maturation of DC unless they are lysed, suggesting that accessibility of parasite inflammatory molecules to their receptors is a key issue in the activation of DC by P. yoelii. This activation is independent of MyD88. It was also observed that pre-incubation of DC with intact P. yoelii-infected erythrocytes inhibits the maturation response of DC to other TLR stimuli. The inhibition of maturation of DC is reversible, parasite-specific and increases with the stage of parasite development, with complete inhibition induced by schizonts (mature infected erythrocytes). **Plasmodium yoelii-infected erythrocytes induce a broad inhibitory effect rendering DC non-responsive to ligands for TLR2, TLR3, TLR4, TLR5, TLR7 and TLR9."**
https://www.ncbi.nlm.nih.gov/pubmed/20193084

Immunosuppressing antigens in Malaria, again:

FEBS J. 2013 Dec;280(23):6196-212. doi: 10.1111/febs.12541. Epub 2013 Oct 16.
Structure and dynamic behavior of Toll-like receptor 2 subfamily triggered by malarialglycosylphosphatidylinositols of Plasmodium f alciparum.

Durai P1, Govindaraj RG, Choi S.

"The recognition of GPIs of the protozoans *P. falciparum* or *Toxoplasma gondii*appears to be via TLR2 and TLR4 29. In an experimental study by Krishnegowda *et al*. 30, using mouse macrophages and human monocytes, ***P. falciparum* malarial GPIs consisting of three fatty acid chains were favourably recognized by human and mouse TLR2/TLR1** 30. Moreover, one of the derivatives of GPIs called *sn□2□*lyso GPI was the ligand for the hTLR2□hTLR6 complex. The above result was confirmed in another recent experimental study using macrophages from gene knockout mice, in addition to human monocytes and anti-human TLR1 and TLR6 sera 31. The ECD of TLR2 has the potential to recognize GPIs in the same binding sites of lipopeptides because the structural patterns of GPIs and lipoproteins are similar, although they are different classes of compounds 30. There is sufficient evidence for TLR2 recognition of GPIs; however, the binding site of GPIs and the interacting residues in the protein that would be useful for developing anti□malarial drugs or vaccines are still unknown.

"In the present study, we used some of the methods discussed below to determine the details of the interaction of the TLR2 subfamily with *P. falciparum* Man4□GPI and the *sn□2 lyso* GPI derivative. Molecular docking is a widely used modelling tool for predicting the exact positioning of a ligand in the active site of a protein 32. Hence, in the present study, we employed molecular docking to investigate the interactions between *P. falciparum* Man4□GPI and hTLR2□hTLR1 and between *sn□2 lyso* GPI and mTLR2□mTLR6. In addition, MD simulations that can report at the atomic level are appropriate for highlighting the dynamics of a given structure to validate the experimental studies on the ligand□induced dimerization analysis of TLRs 33. It is well known that ligands induce dimerization of the TLR2 subfamily 17; therefore, by utilizing MD techniques, we simulated the subfamily of TLR2 for 15 ns as a monomer and dimer in the absence and presence of the GPI to better understand the ligand-induced dimerization and activation mechanism at the atomic level.
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4163636/

We would expect, naturally, then to find quite a lot of Chronic Fatigue Syndrome in Africa and we do. As an aside, we know not to use antibody studies for finding the herpesviruses in diseases of immunosuppression like this, so any such studies will be thrown out.

J Health Psychol. 2007 May;12(3):461-74.

The prevalence of chronic fatigue syndrome in Nigeria.

Njoku MG1, Jason LA, Torres-Harding SR.

"The present study found adult rates of chronic fatigue syndrome (CFS) in Nigeria that were somewhat higher than rates from community-based CFS epidemiologic studies in the USA. The rates of chronic fatigue for both adults and children were also higher than in existing community-based studies. It is possible that the presence of several fatiguing illnesses such as malaria and typhoid, the lack of adequate healthcare resources and poverty in Nigeria, place individuals at greater risk for fatigue and its syndromes. There is a need for more epidemiologic studies on the prevalence and sociodemographic characteristics of CFS in developing countries."
http://www.ncbi.nlm.nih.gov/pubmed/17439996

Among the other very first things we would like to say about ME/CFS and Fibromyalgia are:

Nat Commun. 2015 Dec 15;6:10145. doi: 10.1038/ncomms10145.

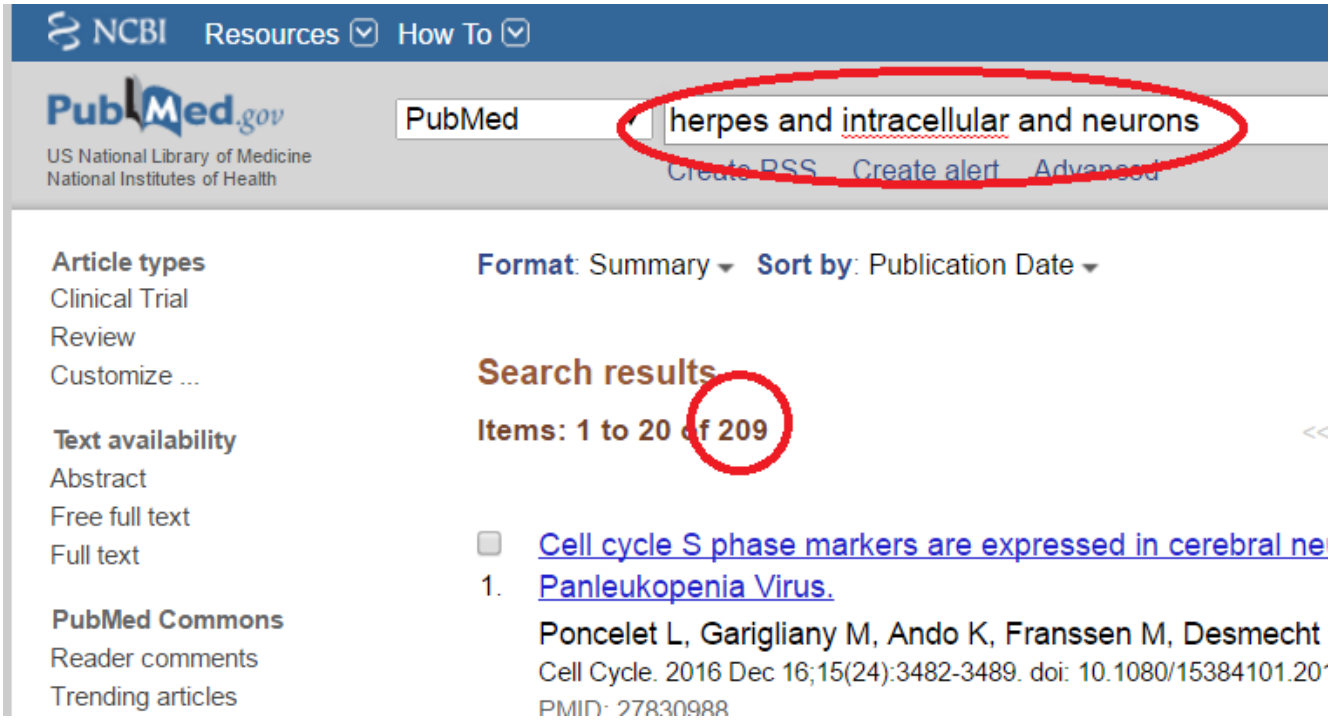
Sepsis induces long-term metabolic and mitochondrial muscle stem cell dysfunction amenable by mesenchymal stem cell therapy.

Rocheteau P1, Chatre L2,3, Briand D1, Mebarki M1, Jouvion G1, Bardon J1, Crochemore C2,3, Serrani P1, Lecci PP1, Latil M1, Matot B4,5, Carlier PG4,5, Latronico N6, Huchet C7, Lafoux A7, Sharshar T1,8,9,10, Ricchetti M2,3, Chrétien F1,10,11,12.

”Sepsis, or systemic inflammatory response syndrome, is the major cause of critical illness resulting in admission to intensive care units. Sepsis is caused by severe infection and is associated with mortality in 60% of cases. Morbidity due to sepsis is complicated by neuromyopathy, and **patients face long-term disability due to muscle weakness, energetic dysfunction, proteolysis and muscle wasting**. These processes are triggered by pro-inflammatory cytokines and metabolic imbalances and are aggravated by malnutrition and drugs. Skeletal muscle regeneration depends on stem (satellite) cells. Herein we show that mitochondrial and metabolic alterations underlie the sepsis-induced long-term impairment of satellite cells and lead to inefficient muscle regeneration. Engrafting mesenchymal stem cells improves the septic status by decreasing cytokine levels, restoring mitochondrial and metabolic function in satellite cells, and improving muscle strength. These findings indicate that sepsis affects quiescent muscle stem cells and that mesenchymal stem cells might act as a preventive therapeutic approach for sepsis-related morbidity.

https://www.ncbi.nlm.nih.gov/pubmed/26666572

And:



We’d like to say “Fibro Herpes” and “Fibro Herpes living in the nerve root ganglia, messing with ion channels and perhaps due to ONGOING INFECTIONS,” since duh. Imagine shingles, et al, without the typical, say, loud manifestations.

Herpes. 2006 Nov;13(3):75-80.

***Investigations of the pathogenesis of Varicella zoster virus infection in the SCIDhu mouse model*.**

Arvin AM1.

"Varicella zoster virus (VZV) is a medically important human herpesvirus that causes varicella, establishes latency in sensory ganglia and may reactivate to cause herpes zoster in healthy and immunocompromised patients. Experiments in the severe combined immunodeficiency (SCID) mouse model have provided new insights about VZV pathogenesis. In addition, the evaluation of VZV recombinant viruses, with targeted mutations of viral genes or their promoters in SCIDhu skin, T-cell and **dorsal root ganglia xenografts, has the potential to identify options for the design of a recombinant 'second-generation' VZV vaccine. This would be characterized by the retention of infectivity in skin combined with a restricted tropism for T-cells and neurons within sensory ganglia."**

https://www.ncbi.nlm.nih.gov/pubmed/17147912

Un-latent herpesviruses might be painful and fatiguing illnesses. CDC officer Suzanne Vernon lies about mycoplasma playing a role in ME/CFS (Occam's Razor) – because such fungal induced immunsouppression reactivates viruses. Some herpes love ganglia, right where, "catastrophizing" "**pressure points**" are. You've already seen that EBV and other viruses may be antibody-negative due to the cross tolerance.

Here, next, a scientist makes a suggestion that infection may be triggering the swelling of the nerve root ganglia (after all, some of the herpesviruses are known to love nerve root ganglia):

Brain (2013) 136 (9): e246., 31 May 2013

Small fibre neuropathy, fibromyalgia and dorsal root ganglia sodium channels

Manuel Martinez-Lavin **DOI:** https://doi.org/10.1093/brain/awt114

"… Dorsal root ganglia contain the sensory fibres cell bodies. **Trauma and/or infection trigger sympathetic sprouting within dorsal root ganglia through nerve growth factor over-expression.** Such aberrant neuroplasticity enables catecholamines and sympathetic traffic to induce sensory neuron firing. Sodium channels play a pivotal role in this hyperexcitability. A sodium channel isoform (NaV1.7) encoded in gene *SCN9A*of chromosome 2q24.3 is predominantly expressed in the dorsal root ganglia pain-sensing neurons and sympathetic ganglia neurons and their fine-diameter axons…
" …In a pilot study, we described a particular *SCN9A* sodium channel gene variant (rs6754031 GG genotype) associated with severe fibromyalgia (Vargas-Alarcon *et al.*, 2012). On the other hand, Faber *et al.* (2012) reported that a gain of function mutations in sodium channel NaV1.7, which render dorsal root ganglion neurons hyperexcitable, are present in a substantial proportion (28.6%; 8 of 28) of patients meeting strict criteria for small fibre neuropathy (Faber *et al.*, 2012). **This preliminary information raises the possibility that some cases of fibromyalgia and small fibre neuropathy may have underlying dorsal root ganglia sodium channelopathy.**
The Üçeyler *et al.* (2013) study reinforces our proposal of fibromyalgia as sympathetically maintained neuropathic pain syndrome. Sympathetic dysfunction provides a coherent explanation for the multiple non-pain related fibromyalgia symptoms (Martinez-Lavin, 2012).

https://academic.oup.com/brain/article/136/9/e246/288267/Small-fibre-neuropathy-fibromyalgia-and-dorsal

Lenny Sigal, one of the misogynistic Lyme criminals:

Arthritis Rheum. 2000 Nov;43(11):2493-500.
The role of catastrophizing in the pain and depression of women with fibromyalgia syndrome.
Hassett AL1, Cone JD, Patella SJ, **Sigal LH.**

"OBJECTIVE: Although 2 recent studies have found associations between catastrophizing and poor medical outcomes in patients with fibromyalgia syndrome (FMS), neither assessed these findings in comparison with a similar group of patients with chronic pain. Our study examined the complex relationships between depression, catastrophizing, and the multidimensional aspects of pain in women with FMS and compared these relationships with those in women with rheumatoid arthritis (RA).
METHODS: Sixty-four FMS patients and 30 RA patients completed the Coping Strategies Questionnaire (CSQ), the Beck Depression Inventory II (BDI-II), and the McGill Pain Questionnaire.
RESULTS: Compared with subjects with RA, FMS subjects scored significantly higher on the catastrophizing subscale of the CSQ. FMS patients also earned higher scores on overall depression and on the cognitive subscale of the BDI-II. Furthermore, the relationship between catastrophizing and depression was significant in the FMS group only. Regression analyses revealed that in FMS, catastrophizing as a measure of coping predicted patients' perception of pain better than demographic variables such as age, duration of illness, and education.
CONCLUSION: Cognitive factors, such as catastrophizing and depressive self-statements, have a more pronounced role in the self-reported pain of patients with FMS than in patients with RA. Clinically, this indicates that treating pain and depression in FMS by adding cognitive therapy and coping skills components to a comprehensive treatment program may improve the outcomes obtained with pharmacologic interventions."
https://www.ncbi.nlm.nih.gov/pubmed/11083273

[Catastrophizing. Think. We may have just discovered the brain magic behind somatoform illnesses. It must mean "very, very hard thinking and concentrating," you know like levitating gurus. We just wonder why dismiss the magical Fibro-gurus instead of putting them to work for the CIA to stare at goats and discover Russia's and China's hidden submarines and underground bases?]

Again on mycoplasma (to which you probably have been tolerized if you have Chronic Fatigue or Fibromyalgia post sepsis syndrome) and how they can cause fatigue by damaging red blood cell membranes, **inhibiting the transfer of oxygen**:

Berl Munch Tierarztl Wochenschr. 1992 Nov 1;105(11):380-3.
[The effect of Eperythrozoon suis infection on the osmotic fragility of erythrocytes].
[Article in German]
Heinritzi K1, Plank G.
"Osmotic fragility of erythrocytes was tested in weaned pigs experimentally infected with Eperythrozoon (E.) suis. Acute eperythrozoonosis of splenectomized pigs led to an increase of osmotic fragility. It is supposed that E. suis **infection causes a structural change in erythrocyte membrane**. Possible mechanisms of this cell membrane injury are discussed."
http://www.ncbi.nlm.nih.gov/pubmed/1471973

Ciba Found Symp. 1981;80:98-118.
Adhesion of mycoplasmas to eukaryotic cells.
Razin S, Kahane I, Banai M, Bredt W.

"Many pathogenic mycoplasmas are surface parasites, adhering to the epithelial linings of the respiratory and urogenital tracts. Since mycoplasmas lack cell walls their plasma membrane comes in close contact with that of their host, allowing exchange of components between the two membranes and possibly fusion. The tight association of the parasite with its host is illustrated in scanning electron micrographs of Mycoplasma pneumoniae and M. gallisepticum **adhering to human red blood cells**. Specialized structure at the tips of the mycoplasma cells appear to function as attachment organelles. Our main aim has been to chemically define the receptors on the host cell and the binding sites on the mycoplasma cells responsible for adhesion. Glycophorin (the major sialoglycoprotein of human red blood cells) serves as the main or sole receptor for M. gallisepticum whereas M. pneumoniae binds to additional receptors on human red blood cells. Trypsin treatment of M. pneumoniae cells abolishes their ability to attach to human red cells, suggesting the protein nature of the binding sites. M. pneumoniae membranes solubilized by detergents were subjected to affinity chromatography on glycophorin-Sepharose so that membrane components with high affinity for glycophorin could be isolated. The fraction isolated consisted of several proteins (relative molecular mass 25 000 and 45 000). The binding of this fraction to red cells was relatively low but appeared to be specific, as it was inhibited by glycophorin but not by its hydrophobic moiety. **The possibility is discussed that the exposure of the binding sites on the mycoplasma cell surface is influenced by the electrochemical ion gradient across the membrane.**
http://www.ncbi.nlm.nih.gov/pubmed/6790254

Here we see again that such fungal antigens inhibit antigen presentation, or result in no antibodies, which is why there typically are no markers in Chronic Fatigue Syndrome or Fibromyalgia:

J Immunol. 2001 Jul 15;167(2):910-8.
Toll-like receptor 2-dependent inhibition of macrophage class II MHC expression and antigen processing by 19-kDa lipoprotein of Mycobacterium tuberculosis.
Noss EH1, Pai RK, Sellati TJ, Radolf JD, Belisle J, Golenbock DT, Boom WH, Harding CV.
"Mycobacterium tuberculosis (MTB) induces vigorous immune responses, yet persists inside macrophages, evading host immunity. MTB bacilli or lysate was found to inhibit macrophage expression of class II MHC (MHC-II) molecules and MHC-II Ag processing. This report characterizes and identifies a specific component of MTB that mediates these inhibitory effects. The inhibitor was extracted from MTB lysate with Triton X-114, isolated by gel electroelution, and identified with Abs to be MTB 19-kDa lipoprotein. Electroelution- or immunoaffinity-purified MTB 19-kDa lipoprotein inhibited MHC-II expression and processing of both soluble Ags and Ag 85B from intact MTB bacilli. **Inhibition of MHC-II Ag processing** by either MTB bacilli or purified MTB 19-kDa lipoprotein was dependent on Toll-like receptor (TLR) 2 and independent of TLR 4. Synthetic analogs of lipopeptides from Treponema pallidum also inhibited Ag processing. Despite the ability of MTB 19-kDa lipoprotein to activate microbicidal and innate immune functions early in infection, TLR 2-dependent inhibition of MHC-II expression and Ag processing by MTB 19-kDa lipoprotein during later phases of macrophage infection may prevent presentation of MTB Ags and decrease recognition by T cells. This mechanism may allow intracellular MTB to evade immune surveillance and maintain chronic infection."
http://www.ncbi.nlm.nih.gov/pubmed/11441098

You have already seen some of these reports, so we will just list a few to remind of the general concept that fungal antigens also **inhibit apoptosis in infected cells**, and mycoplasma, which were

fraudulently thrown out by CDC's Suzanne Vernon do in fact cause "disease," even though it might not be with classic "inflammatory" or "autoimmune" signs:

Cell Death Differ. 2004 Nov;11(11):1204-12.
Mycoplasma fermentans inhibits tumor necrosis factor alpha-induced apoptosis in the human myelomonocytic U937 cell line.
Gerlic M1, Horowitz J, Horowitz S.

"In conclusion, M. fermentans significantly inhibits TNFalpha-induced apoptosis in U937 cells, and its effect is upstream of the mitochondria and upstream of caspase-8."
http://www.ncbi.nlm.nih.gov/pubmed/15286682

Cell Microbiol. 2007 Jan;9(1):142-53. Epub 2006 Aug 2.
The inhibitory effect of Mycoplasma fermentans on tumour necrosis factor (TNF)-alpha-induced apoptosis resides in the membrane lipoproteins.
Gerlic M1, Horowitz J, Farkash S, Horowitz S.
"Mycoplasma have been shown to be involved in the alteration of several eukaryotic cell functions, such as cytokine production, gene expression and more. We have previously reported that infection of human myelomonocytic U937 cell line with live Mycoplasma fermentans (M. fermentans) inhibited tumour necrosis factor (TNF-alpha)-induced apoptosis."
http://www.ncbi.nlm.nih.gov/pubmed/16889623

Mycoplasma cause disease by affecting red blood cells (oxygen) and they inhibit apoptosis in infected other blood immune cells, which is very close to a pre-cancer state. You'll remember from the Occam's Razor or your own casual reading that Rituximab was discovered to be a treatment for Chronic Fatigue/ME because those cancer patients recovered from their Chronic Fatigue Syndrome with that monoclonal antibody.

In other words, yes, Chronic Fatigue/Fibro waste-basketees not surprisingly developed cancer since we are talking about post-sepsis syndrome with the reactivated viruses of all kinds, especially the herpes.

The CDC (Vernon, wow) knows chronic mono or chronic EBV is a chronic fatiguing illness:

BMC Infect Dis. 2006; 6: 15.
Preliminary evidence of mitochondrial dysfunction associated with post-infective fatigue after acute infection with Epstein Barr Virus
Suzanne D Vernon,1 Toni Whistler,1 Barbara Cameron,2 Ian B Hickie,3 William C Reeves,1 and Andrew Lloyd2

BACKGROUND: Acute infectious diseases are typically accompanied by non-specific symptoms including fever, malaise, irritability and somnolence that usually resolve on recovery. However, in some individuals these symptoms persist in what is commonly termed post-infective fatigue. The objective of this pilot study was to determine the gene expression correlates of post-infective fatigue following acute Epstein Barr virus (EBV) infection.
METHODS: We followed 5 people with acute mononucleosis who developed post-infective fatigue of more than 6 months duration and 5 HLA-matched control subjects who recovered within 3 months. Subjects had peripheral blood mononuclear cell (PBMC) samples collected at varying time points

including at diagnosis, then every 2 weeks for 3 months, then every 3 months for a year. Total RNA was extracted from the PBMC samples and hybridized to microarrays spotted with 3,800 oligonucleotides.
RESULTS: Those who developed post-infective fatigue had gene expression profiles indicative of an altered host response during acute mononucleosis compared to those who recovered uneventfully. Several genes including ISG20 (interferon stimulated gene), DNAJB2 (DnaJ [Hsp40] homolog and CD99), CDK8 (cyclin-dependent kinase 8), E2F2 (E2F transcription factor 2), CDK8 (cyclin-dependent kinase 8), and ACTN2 (actinin, alpha 2), known to be regulated during EBV infection, were differentially expressed in post-infective fatigue cases. Several of the differentially expressed genes affect mitochondrial functions including fatty acid metabolism and the cell cycle.
CONCLUSION: These preliminary data provide insights into alterations in gene transcripts associated with the varied clinical outcomes from acute infectious mononucleosis.

In the full text they write:

"…Acute viral diseases such as infectious mononucleosis typically present clinically with a cluster of non-specific symptoms including; fever, an increased need to sleep, hyperalgesia, anorexia, loss of interest in usual activities, social interaction, body care, depressed mood, and impaired concentration [1-3]. This acute sickness behavior response comprises a highly organized and evolved disease-fighting strategy mediated by the action of pro-inflammatory cytokines [4-8]. In general, acute sickness behavior resolves in parallel with clearance or control of the infecting agent. However, some individuals exhibit prolonged illness with fatigue, mood changes and cognitive impairment**. Such prolonged illness following infectious mononucleosis has been recognized for at least half a century** [9]. Recent studies of infectious mononucleosis due to EBV infection demonstrated that fatigue, sore throat and malaise persisted for up to two months in approximately 40% of patients and for six or more months in approximately 10% [10,11]."
https://www.ncbi.nlm.nih.gov/pubmed/16448567

Everyone with Chronic Fatigue/ME and Fibromyalgia has known for years that the CDC pooh-pah'd the idea that CFIDS/ME was about chronic Epstein-Barr or about any chronic common viral infection. Yet, here they are saying, "Oh-yeah, this has been known for 50 years…"

Garth Nicolson on mycoplasma and chronic fatigue:

APMIS. 2003 May;111(5):557-66.
Multiple co-infections (Mycoplasma, Chlamydia, human herpes virus-6) in blood of chronic fatigue syndrome patients: association with signs and symptoms.
Nicolson GL1, Gan R, Haier J.
"Previously we and others found that a majority of chronic fatigue syndrome (CFS) patients showed evidence of systemic mycoplasmal infections, and their blood tested positive using a polymerase chain reaction assay for at least one of the four following Mycoplasma species: M. fermentans, M. hominis, M. pneumoniae or M. penetrans. Consistent with previous results, patients in the current study (n=200) showed a high prevalence (overall 52%) of mycoplasmal infections. Using forensic polymerase chain reaction we also examined whether these same patients showed evidence of infections with Chlamydia pneumoniae (overall 7.5% positive) and/or active human herpes virus-6 (HHV-6, overall 30.5%

positive). Since the presence of one or more infections may predispose patients to other infections, we examined the prevalence of C. pneumoniae and HHV-6 active infections in mycoplasma-positive and -negative patients. Unexpectedly, we found that the incidence of C. pneumoniae or HHV-6 was similar in Mycoplasma-positive and -negative patients, and the converse was also found in active HHV-6-positive and -negative patients. Control subjects (n=100) had low rates of mycoplasmal (6%), active HHV-6 (9%) or chlamydial (1%) infections, and there were no co-infections in control subjects. Differences in bacterial and/or viral infections in CFS patients compared to control subjects were significant. Severity and incidence of patients' signs and symptoms were compared within the above groups. Although there was a tendency for patients with multiple infections to have more severe signs and symptoms ($p<0.01$), the only significant differences found were in the incidence and severity of certain signs and symptoms in patients with multiple co-infections of any type compared to the other groups ($p<0.01$). There was no correlation between the type of co-infection and severity of signs and symptoms. The results indicate that a large subset of CFS patients show evidence of bacterial and/or viral infection(s), and these infections may contribute to the severity of signs and symptoms found in these patients."
http://www.ncbi.nlm.nih.gov/pubmed/12887507

That sounds exactly like post-sepsis syndrome as shown in the Occam's Razor.

Next, suppression of immune signs markers and cytokines in Chronic Fatigue Syndrome, pointing to the disease not being about inflammation or autoimmunity, but the opposite, immunosuppression or post-sepsis syndrome; look at this chart:

Clin Diagn Lab Immunol. 1999 Jan;6(1):6-13.

Changes in immune parameters seen in Gulf War veterans but not in civilians with chronic fatigue syndrome.

Zhang Q1, Zhou XD, Denny T, Ottenweller JE, Lange G, LaManca JJ, Lavietes MH, Pollet C, Gause WC, Natelson BH.

Look closely at the Table 2 – all the markers are lower in Chronic Fatigue than normals. This is a disease of immune suppression and not inflammation or autoimmunity. This is post-sepsis syndrome, same as "Chronic Lyme."

TABLE 2

Means and standard errors for cytokines and CD cell surface markers

Cell type, phenotype, or cytokine	Gulf War veterans						Civilians					
	n[f]	CFS		n	Control		n	CFS		n	Control	
		Mean	SE		Mean	SE		Mean	SE		Mean	SE
No. of[a]:												
WBC	42	6,564.29	262.54	33	6,233.33	253.67	43	6,516.28	284.89	39	6,064.1	213.08
Lymphocytes	42	2,119.76	103.81	33	1,918.33	96.03	43	1,826.63	75.93	39	1,874.36	68.69
$CD(16+56)^{+}$[a]	42	261.10	28.72	33	290.70	26.13	43	180.74	13.69	39	196.08	15.11
$CD19^{+}$	42	248.07	19.63	33	262.18	28.33	43	256.86	18.37	39	252.46	16.23
$CD3^{+}$	42	1,613.62	78.65	33	1,373.58	72.47	43	1,385.86	63.84	39	1,425.95	57.89
$CD3^{+}$ $CD4^{+}$	42	1,014.69	51.62	33	809.15	45.34	43	889.93	41.29	39	927.26	40.77
$CD3^{+}$ $CD8^{+}$	42	567.62	34.95	33	515.33	42.20	43	458.37	28.99	39	476.85	32.11
% Lymphocytes[b]	42	32.67	1.22	33	31.73	1.55	43	29.51	1.48	39	31.31	0.97
$CD(16+56)^{+}$[c]	42	12.24	0.94	33	15.52	1.12	43	10.19	0.73	39	10.62	0.76
$CD19^{+}$[c]	42	11.55	0.67	33	13.03	1.02	43	14.23	0.80	39	13.38	0.66
$CD3^{+}$[c]	42	76.36	0.93	33	71.85	1.23	43	75.53	1.21	39	75.87	0.97
$CD3^{+}$ $CD4^{+}$[c]	42	48.31	1.01	33	42.45	1.39	43	48.60	1.03	39	49.59	1.18
$CD3^{+}$ $CD8^{+}$[c]	42	26.62	1.11	33	26.73	1.39	43	24.81	0.97	39	25.72	1.20
$CD4^{+}$ $CD45RO^{+}$[d]	40	71.40	1.85	33	72.18	2.12	43	67.83	2.26	39	70.51	2.44
$CD4^{+}$ $CD45RA^{+}$[d]	40	42.80	1.73	33	40.76	2.18	40	46.23	1.70	39	43.18	1.79
$CD8^{+}$ $CD28^{+}$[d]	41	58.17	1.96	33	59.45	2.25	40	67.79	1.98	39	65.05	2.23
$CD8^{+}$ $CD38^{+}$[d]	41	51.56	2.35	33	53.12	2.74	40	58.08	1.92	38	51.10	1.77
$CD8^{+}$ HLA-DR^{+}[d]	41	20.90	1.69	33	20.73	2.23	40	19.85	2.14	39	22.82	2.18
$CD8^{+}$ $CD11b^{+}$[d]	40	56.20	2.49	32	53.44	2.95	40	69.58	2.24	39	61.66	3.64
Cytokines[c]												
IL-2	43	430.95	140.23	34	251.97	61.17	68	77.93	13.02	53	95.48	17.24
IL-4	43	256.33	58.06	34	134.11	18.79	68	16.90	2.58	53	18.74	2.61
IL-6	43	2,882.11	505.21	34	1,710.95	337.08	68	98.21	32.49	53	281.42	112.87
IL-10	43	603.84	136.88	34	495.95	265.11	68	333.42	48.06	53	532.42	170.98
IL-12	43	299.55	84.64	34	136.37	38.89	68	463.22	66.90	53	656.63	159.95
TNF-α	43	288.62	48.37	34	166.35	27.28	68	140.84	16.80	53	182.93	19.90
IFN-γ	43	1,002.06	163.52	34	632.74	146.11	68	736.37	113.41	53	986.20	246.56

https://www.ncbi.nlm.nih.gov/pubmed/9874656
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC95652/

Does this next report need narration or interpretation?

Biochem Biophys Res Commun. 2005 Jul 29;333(2):438-42.
Epstein-Barr virus immediate-early proteins BZLF1 and BRLF1 alter mitochondrial morphology during lytic replication.
LaJeunesse DR1, Brooks K, Adamson AL.

Epstein-Barr virus (EBV) is a human DNA virus that is responsible for the syndrome infectious mononucleosis, and is associated with several forms of cancer. During both lytic and latent viral infection, viral proteins manipulate the host's cellular components to aid in viral replication and maintenance**. Here, it is demonstrated that induction of EBV lytic replication results in a dramatic reorganization of mitochondria accompanied by a significant alteration of mitochondrial membrane potential and a rapid and transient increase in the microtubular cytoskeleton**. Moreover, we show that expression of the EBV immediate-early genes BZLF1 and BRLF1 contributes to the mitochondrial alteration but not the increase in the microtubule cytoskeleton, suggesting that the mechanism for the observed cytoplasmic restructuring involves a number of coordinated viral and host proteins.
http://www.ncbi.nlm.nih.gov/pubmed/15950179

No. Chronic Active EBV (CAEBV) could be, shall we say, fatiguing by damaging mitochondria. And not show any antibody signs.

18) Next, 4 reports on Seronegative Epstein-Barr (you have already seen this in the other charge sheets). It means you can have chronic active Epstein-Bar or other herpes viruses (some living in the ganglia, hello Fibromyalgia) but in these immunosuppression cases, you will not see the typical slightly elevated antibody titer associated with reactivated viruses in non-immunocompromised or non-post-sepsis individuals:

[From the Occam's Razor report, number DD] **Seronegative reactivated Epstein-Barr, and Clifford Harding again on how Pam3cys-ish molecules down-regulate the management of the TLRs that handle viruses**

Here are 4 examples from the literature of how Epstein-Barr also can be seronegative via the same mechanism of downregulation of antigen-presenting molecules or downregulation of HLA molecules (shows antigen so that B cells can make antibodies) or the MHC or "Major Histocompatibility Class" of cell components (all the same thing):

J Immunol. 2009 Feb 15;182(4):1799-809. doi: 10.4049/jimmunol.0802686.
Down-regulation of MHC class II expression through inhibition of CIITA transcription by lytic transactivator Zta during Epstein-Barr virus reactivation.
Li D1, Qian L, Chen C, Shi M, Yu M, Hu M, Song L, Shen B, Guo N.

"The presentation of peptides to T cells by MHC class II molecules is of critical importance in specific recognition to a pathogen by the immune system. The level of MHC class II directly influences T lymphocyte activation. The aim of this study was to identify the possible mechanisms of the down-regulation of MHC class II expression by Zta during EBV lytic cycle. The data in the present study demonstrated that ectopic expression of Zta can strongly inhibit the constitutive expression of MHC class II and CIITA in Raji cells. The negative effect of Zta on the CIITA promoter activity was also observed. Scrutiny of the DNA sequence of CIITA promoter III revealed the presence of two Zta-response element (ZRE) motifs that have complete homology to ZREs in the DR and left-hand side duplicated sequence promoters of EBV. By chromatin immunoprecipitation assays, the binding of Zta to the ZRE(221) in the CIITA promoter was verified. Site-directed mutagenesis of three conserved nucleotides of the ZRE(221) substantially disrupted Zta-mediated inhibition of the CIITA promoter activity. Oligonucleotide pull-down assay showed that mutation of the ZRE(221) dramatically abolished Zta binding. Analysis of the Zta mutant lacking DNA binding domain revealed that the DNA-binding activity of Zta is required for the trans repression of CIITA. The expression of HLA-DRalpha and CIITA was restored by Zta gene silencing. The data indicate that Zta may act as an inhibitor of the MHC class II pathway, suppressing CIITA transcription and thus interfering with the expression of MHC class II molecules."
http://www.ncbi.nlm.nih.gov/pubmed/19201831

How many "doctors" know you can't rely on antibody testing to know if EBV has been reactivated?

Right, I never met one or heard of one, either.

Herpesviridae. 2011 Jan 5;2(1):1. doi: 10.1186/2042-4280-2-1.
Innate immune modulation in EBV infection.
Ning S1.

"Dysregulation of EBV-specific immune responses is also characteristic of EBV-associated autoimmune diseases such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE). CTL response to EBV infection has been well documented since the discovery of EBV [11]. However, significant progresses in characterizing individual viral proteins involved in evasion of the T cell-mediated adaptive immune response have only been made in the last decade [12-16]. For example**, the functional homologue of human IL10,** BCRF1, elicits CD8+ T cell responses, and can be processed and presented to CD8+ CTLs through a TAP-independent pathway [17]."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3063194/?tool=pubmed

'A functional homolog of IL-10, the human immune-suppressing cytokine. Awesome.

J Virol. 2002 Aug;76(16):8179-88.
The lytic cycle of Epstein-Barr virus is associated with decreased expression of cell surface major histocompatibility complex class I and class II molecules.

Keating S1, Prince S, Jones M, Rowe M.

Human herpesviruses utilize an impressive range of strategies to evade the immune system during their lytic replicative cycle, including reducing the expression of cell surface major histocompatibility complex (MHC) and immunostimulatory molecules required for recognition and lysis by virus-specific cytotoxic T cells. Study of possible immune evasion strategies by Epstein-Barr virus (EBV) in lytically infected cells has been hampered by the lack of an appropriate permissive culture model. Using two-color immunofluorescence staining of cell surface antigens and EBV-encoded lytic cycle antigens, we examined EBV-transformed B-cell lines in which a small subpopulation of cells had spontaneously entered the lytic cycle. Cells in the lytic cycle showed a four- to fivefold decrease in cell surface expression of MHC class I molecules relative to that in latently infected cells. Expression of MHC class II molecules, CD40, and CD54 was reduced by 40 to 50% on cells in the lytic cycle, while no decrease was observed in cell surface expression of CD19, CD80, and CD86. Downregulation of MHC class I expression was found to be an early-lytic-cycle event, since it was observed when progress through late lytic cycle was blocked by treatment with acyclovir. The immediate-early transactivator of the EBV lytic cycle, BZLF1, did not directly affect expression of MHC class I molecules. However, BZLF1 completely inhibited the upregulation of MHC class I expression mediated by the EBV cell-transforming protein, LMP1. This novel function of BZLF1 elucidates the paradox of how MHC class I expression can be downregulated when LMP1, which upregulates MHC class I expression in latent infection, remains expressed in the lytic cycle.
http://www.ncbi.nlm.nih.gov/pubmed/12134023

Remember, Chiu and Aucott says there is no change to immune genes expression. There is just the down-regulation of all mechanisms related to immune competence in the Post-Sepsis outcome of Lyme and LYMErix disease. Tardmerica may be stupid, but it's not boring.

Semin Cancer Biol. 2008 Dec;18(6):397-408. doi: 10.1016/j.semcancer.2008.10.008. Epub 2008 Oct 25.
Epstein-Barr virus evasion of CD8(+) and CD4(+) T cell immunity via concerted actions of multiple gene products.
Ressing ME1, Horst D, Griffin BD, Tellam J, Zuo J, Khanna R, Rowe M, Wiertz EJ.

"Evidence is accumulating that this paradoxical situation is the result of actions of multiple viral gene products, inhibiting discrete stages of the MHC class I and class II antigen presentation pathways. Immediately after initiation of the lytic cycle, BNLF2a prevents peptide-loading of MHC class I molecules through inhibition of the Transporter associated with Antigen Processing, TAP. This will reduce presentation of viral antigens by the large ER-resident pool of MHC class I molecules. Synthesis of new MHC class I molecules is blocked by BGLF5. Viral-IL10 causes a reduction in mRNA levels of TAP1 and bli/LMP2, a subunit of the immunoproteasome. MHC class I molecules present at the cell surface are downregulated by BILF1. **Also the antigen presenting capacity of MHC class II molecules is severely compromised by multiple EBV lytic gene products, including gp42/gH/gL, BGLF5, and vIL-10.** In this review, we discuss how concerted actions of these EBV

lytic proteins result in highly effective interference with CD8(+) and CD4(+) T cell surveillance, thereby providing the virus with a window for undisturbed generation of viral progeny."
http://www.ncbi.nlm.nih.gov/pubmed/18977445

Therefore, *never use antibody testing* to show an association between an illness and an infectious disease.

Clifford Harding says the chronic agonism of TLR2/1 by these lipoproteins also inhibit TLR7/9 function (manages the viruses like EBV); people want to know how Lyme and LYMErix activate EBV, besides that being about what happens commonly, in all general immunosuppression such as Humira and Stelara and post-transplant patients who acquired EBV-induced lymphoma, which we will get to:

J Immunol. 2012 Feb 1;188(3):1019-26. doi: 10.4049/jimmunol.1102181. Epub 2012 Jan 6.
TLR2 signaling depletes IRAK1 and inhibits induction of type I IFN by TLR7/9.
Liu YC1, Simmons DP, Li X, Abbott DW, Boom WH, Harding CV.

"Pathogens may signal through multiple TLRs with synergistic or antagonistic effects on the induction of cytokines, including type I IFN (IFN-I). IFN-I is typically induced by TLR9, but not TLR2. Moreover, we previously reported that TLR2 signaling by Mycobacterium tuberculosis or other TLR2 agonists inhibited TLR9 induction of IFN-I and IFN-I-dependent MHC-I Ag cross processing. The current studies revealed that lipopeptide-induced TLR2 signaling inhibited induction of first-wave IFN-α and IFN-β mRNA by TLR9, whereas induction of second-wave IFN-I mRNA was not inhibited. TLR2 also inhibited induction of IFN-I by TLR7, another MyD88-dependent IFN-I-inducing receptor, but did not inhibit IFN-I induction by TLR3 or TLR4 (both Toll/IL-1R domain-containing adapter-inducing IFN-β dependent, MyD88 independent). The inhibitory effect of TLR2 was not dependent on new protein synthesis or intercellular signaling. IL-1R-associated kinase 1 (IRAK1) was depleted rapidly (within 10 min) by TLR2 agonist, but not until later (e.g., 2 h) by TLR9 agonist. 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."**
http://www.ncbi.nlm.nih.gov/pubmed/22227568

OspA and Borrelia render you unable to manage viral infections by the viral-managing TLRs.

AND, we know Lupus and MS are EBV-linked outcomes from post-Lyme sepsis. In those cases, those victims have the EBV-linked hypersensitivity association or some other mechanism that looks like those outcomes are "autoimmunity." But as we know, Chronic Fatigue and Fibromyalgia are the same Lupus-and-MS-outcomes-of-the-Great-Imitators-Lyme-and-Syphilis, but without the autoimmunity.

This next report, of course says be careful when considering OspA as a chemo adjuvant because it is known to cause the same immunosuppression and inhibition of apoptosis as we mentioned here previously. What happens when OspA causes the inhibition of apoptosis especially in EBV infected cells? Right. The reactivation of those herpesviruses. Fungally contaminated vaccines? The kids are getting the viruses instead of the protection.

19) Here it says to "**please worry that too much TLR2 agonism such as Lyme, LYMErix, mycoplasma etc could be dangerous due to the immunosuppression**," while Yale and the CDC said LYMErix or OspA was a vaccine, the opposite, ho hum:

J Leukoc Biol. 2013 Jun;93(6):847-63. doi: 10.1189/jlb.1012501. Epub 2013 Mar 8.
TLR agonists: our best frenemy in cancer immunotherapy.
Kaczanowska S1, Joseph AM, Davila E.

"TLR2 stimulation on human CD4+CD45RO+ memory cells also induces IFN-γ production, and these levels are increased when combined with IL-2 [43, 48]. Lipoproteins from *Mycobacterium tuberculosis*, a TLR2 agonist, can stimulate memory CD4+ T cells directly, resulting in enhanced proliferation, as well as IL-2 and IFN-γ production. Although resting CD4+ T cells responded to lipoproteins, as evidenced through NF-κB activation, such as CD8 T cells, CD4 T cells also required concomitant TCR signaling to induce proliferation and cytokine production [69]. ***** In addition to enhancing T cell effector function, TLR2 agonists have been shown to promote T cell longevity and are associated with increased expression of antiapoptotic molecules A1 and Bcl-xL and down-regulation of the proapoptotic protein Bim [43, 53]. *****
http://www.ncbi.nlm.nih.gov/pubmed/23475577
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3656332/

Right, OspA acts like a BCL2 class molecule, inhibiting apoptosis, not to mention the intracellular damage and the reactivation of latent herpes viruses and what-not.

Fungal antigens straight up activate Epstein-Barr:

J Virol. 2010 Apr;84(7):3612-23. doi: 10.1128/JVI.01400-09. Epub 2010 Jan 20.
Toll-like receptor agonists synergistically increase proliferation and activation of B cells by epstein-barr virus.
Iskra S1, Kalla M, Delecluse HJ, Hammerschmidt W, Moosmann A.

"Epstein-Barr virus (EBV) efficiently drives proliferation of human primary B cells in vitro, a process relevant for human diseases such as infectious mononucleosis and posttransplant lymphoproliferative disease. Human B-cell proliferation is also driven by ligands of Toll-like receptors (TLRs), notably viral or bacterial DNA containing unmethylated CpG dinucleotides, which triggers TLR9. Here we quantitatively investigated how TLR stimuli influence EBV-driven B-cell proliferation and expression of effector molecules. CpG DNA synergistically increased EBV-driven proliferation and transformation, T-cell costimulatory molecules, and early production of interleukin-6. CpG DNA alone activated only memory B cells, but CpG DNA enhanced EBV-mediated transformation of both memory and naive B cells. Ligands for TLR2 or TLR7/8 or whole bacteria had a weaker but still superadditive effect on B-cell transformation. Additionally, CpG DNA facilitated the release of transforming virus by established EBV-infected lymphoblastoid cell lines. These results suggest that the proliferation of EBV-infected B cells and their capability to interact with immune effector cells

may be directly influenced by components of bacteria or other microbes present at the site of infection."
http://www.ncbi.nlm.nih.gov/pubmed/20089650

So, that is a fair amount of evidence for people dealing with what they think is ME/CFS or Fibromyalgia, it is basically the same as post sepsis syndrome or Lyme.

20) What about Diagnosing this/these. Welp, believe it or not, we can thank IDSA:

J Clin Microbiol. 2014 Jan;52(1):212-7. doi: 10.1128/JCM.02270-13. Epub 2013 Nov 6.
Virological diagnosis of central nervous system infections by use of PCR coupled with mass spectrometry analysis of cerebrospinal fluid samples.
Lévêque N1, Legoff J, Mengelle C, Mercier-Delarue S, N'guyen Y, Renois F, Tissier F, Simon F, Izopet J, Andréoletti L.

"Viruses are the leading cause of central nervous system (CNS) infections, ahead of bacteria, parasites, and fungal agents. A rapid and comprehensive virologic diagnostic testing method is needed to improve the therapeutic management of hospitalized pediatric or adult patients. In this study, we assessed the clinical performance of PCR amplification coupled with electrospray ionization-time of flight mass spectrometry analysis (PCR-MS) for the diagnosis of viral CNS infections. Three hundred twenty-seven cerebrospinal fluid (CSF) samples prospectively tested by routine PCR assays between 2004 and 2012 in two university hospital centers (Toulouse and Reims, France) were retrospectively analyzed by PCR-MS analysis using primers targeted to adenovirus, human **herpesviruses** 1 to 8 (HHV-1 to -8), polyomaviruses BK and JC, parvovirus B19, and **enteroviruses** (EV). PCR-MS detected single or multiple virus infections in 190 (83%) of the 229 samples that tested positive by routine PCR analysis and in 10 (10.2%) of the 98 samples that tested negative. The PCR-MS results correlated well with herpes simplex virus 1 (HSV-1), varicella-zoster virus (VZV), and EV detection by routine PCR assays (kappa values [95% confidence intervals], 0.80 [0.69 to 0.92], 0.85 [0.71 to 0.98], and 0.84 [0.78 to 0.90], respectively), whereas a weak correlation was observed with Epstein-Barr virus (EBV) (0.34 [0.10 to 0.58]). Twenty-six coinfections and 16 instances of uncommon neurotropic viruses (HHV-7 [n = 13], parvovirus B19 [n = 2], and adenovirus [n = 1]) were identified by the PCR-MS analysis, whereas only 4 coinfections had been prospectively evidenced using routine PCR assays ($P < 0.01$). In conclusion, our results demonstrated that PCR-MS analysis is a valuable tool to identify common neurotropic viruses in CSF (with, however, limitations that were identified regarding EBV and EV detection) **and may be of major interest in better understanding the clinical impact of multiple or neglected viral neurological infections."**
http://www.ncbi.nlm.nih.gov/pubmed/24197874

Neglected Viral Infections. Yes, thank you.

COMPARE that to this IDSociety.org position paper on the issue of using rapid mass-spec PCR on spinal fluid samples for rapid detection of the CNS infections the NIH knows is driving Chronic Fatigue and Chronic Lyme:

"Unmet diagnostic needs in infectious disease"
"1. Introduction
"The importance of diagnostic testing in the management of infectious diseases (ID) was recently

highlighted in the report of the Infectious Diseases Society of America's (IDSA) Diagnostics Task Force report: “Better Tests: Better Care: Improved Diagnostics for Infectious Diseases” (Caliendo et al., 2013). Similar sentiments are expressed in the report on Antibiotic Resistance Threats in the United States Centers for Disease Control (2013) from the Centers for Disease Control and Prevention (CDC**). ****A number of new diagnostic technologies for ID are rapidly emerging: e.g., broad-range PCR, next-generation sequencing, and matrix-assisted laser desorption/ionization time of flight mass spectrometry.*** The reports from the IDSA and the CDC highlight deficiencies in current diagnostic methods and call for approval and access to methods that are rapid and available at the point of care, use directfrom-specimen analysis, and demonstrate high levels of sensitivity and specificity across a wide range of disease syndromes. The importance of syndrome-based panels (e.g., for central nervous system, bloodstream and respiratory tract infections) is highlighted in the IDSA report (Caliendo et al., 2013). Both the IDSA and CDC emphasize the critical need for culture-independent testing for specific pathogens and their pattern of susceptibility to antimicrobial agents...."
http://ein.idsociety.org/media/publications/papers/2014/Blaschke_DMID_14_Unmet_Diagnostic_Needs.pdf

Idsociety’s “Policy Paper” on the same, rapid diagnostics (MassSpec-PCR. But that can’t fit in a test kit, see, so there is no profit in it for the IDSA and CDC DNA profiteers. Superbugs will continue to kill people and there will be more calamities of the hospital acquired and new infection sort. And more of the Ebola and MERS and SARS sort…. If there is no money to be made, IDSA is not interested.

Better Tests, Better Care: Improved Diagnostics for Infectious Diseases
Angela M. Caliendo,1 David N. Gilbert,2,3 Christine C. Ginocchio,4,5,6 Kimberly E. H…
http://www.idsociety.org/uploadedFiles/IDSA/Policy_and_Advocacy/Current_Topics_and_Issues/Diagnostics/Clin%20Infect%20Dis.-2013-Caliendo-S139-70.pdf
[“Won-der-ful,” as the rich people in Fairfield county, Connecticut say.]