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

This is a continuation, really, of the Occam's Razor report. 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 since it does not matter. 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 thing everyone should know, since it was in the *New York Times:*

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&

Thimerosal is put in vaccines to prevent LYMErix, or the immune suppressing fungal endotoxin, OspA. And Fungi plus Babies = Bad:

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

Some examples of the CDC and BigPharma admitting to the bad, bad results of immunosuppression-plus-live-virus-or-bacterial-vaccines**, A through J.**

A.) **CDC's Patent**, US # 7,632,510,

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

B.) **CDC SAYS**,…

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

C.) **CDC SAYS**:

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 -SASH.]

D.) **Pharma SAYS:**

In the case of pandemic MRSA, as we have seen, the vaccine didn't work because TLR2 agonists (lipoproteins) suppress the immune system. We also learned 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 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

E.) **CDC SAYS**…
…that 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.

F.) **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

Stuff ya can't make up. I'll save a screen shot since this data has a way of disappearing ;)

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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.

G.) 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 children with cold viruses going to the pediatrician being vaccinated and then being carried out the same again. You see clearly they write in the 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?

As you've just seen, IDSA believes 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.***

H.) **Paul Auwaerter**. Boy does he keep turning up like an unmatched sock in the laundry…

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

Altered virulence of vaccine strains of measles virus after prolonged replication in human tissue.
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?

Now, 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. 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.

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. Secondly, the only "adverse events" signs the pediatricians are allowed to record are the "autoimmune" ones, like rashes.

If there exists a data set somewhere of children known to have been vaccinated while immunosuppressed with each live viral vaccine type, surely they are 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."

No Autism groups are asking for or showing the correct data. Most of them are on the Thimerosal-Go-Round. 'Nowhere, in other words.

Thimerosal was put in vaccines to prevent LYMErix, really. Exactly. And by the way, no vaccine against spirochetal diseases ever prevented spirochetes. The Cabal does not even technically make this claim. They just say it creates antibodies, which is, of course, false, but whatever, that's them.

I.) "**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

That's ^^^ long for "Baloney," in response to the "herd effect "claim, and they're also saying that, yes, it's not uncommon for people to simply acquire those live viruses all at once, in children too young (IDSA) and without proper vetting (IDSA).

J.) **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)

Synergism. Dual Infections could be bad. And "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. This report could be a good cross over point between the failed vaccines that fail via immunosuppression, especially due to exposure to TLR2/1 agonists, or "***is*** a model that parallels the post-septic shock (ME/CFS, Lyme, failed childhood vaccines)."

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. It's like 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, here we are, we're doing that exact thing.

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:

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 falciparum.
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/

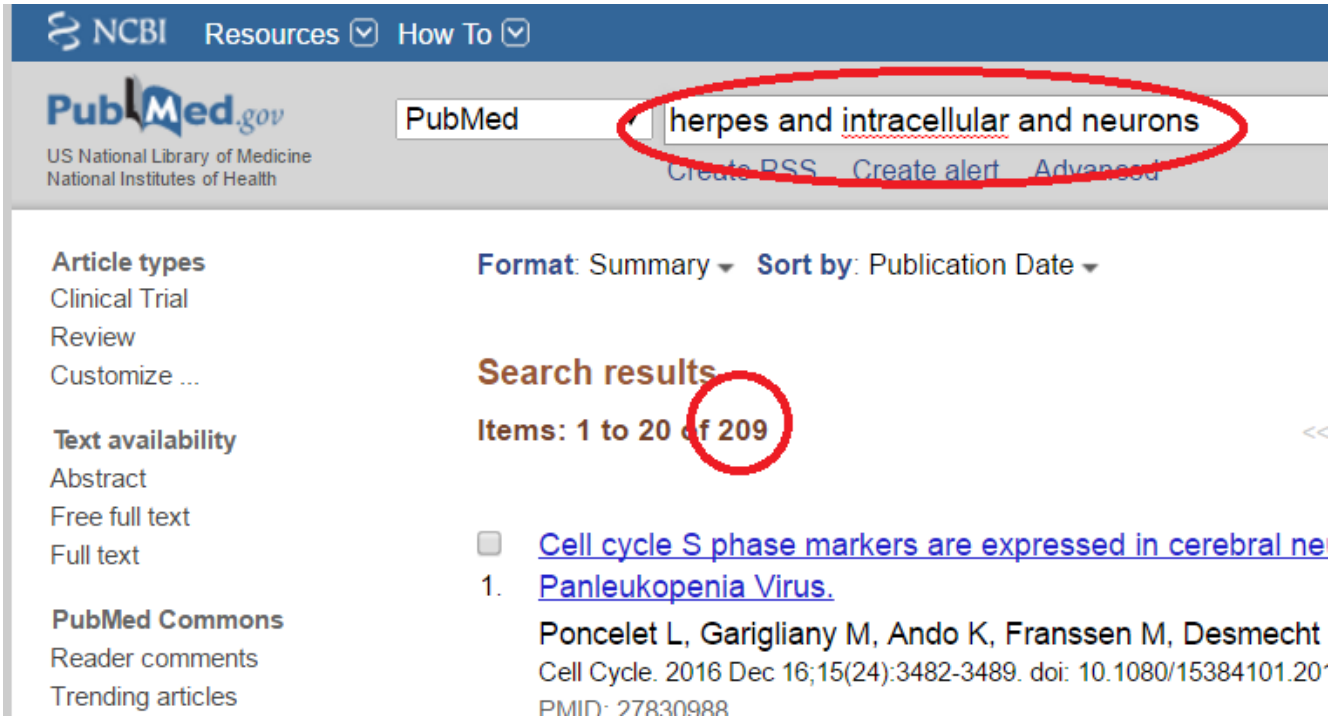
We would expect, naturally, then to find quite a lot of Chronic Fatigue Syndrome in Africa and we do. As an aside, we know not to use antibody studies for finding the herpesviruses in diseases of immunosuppression like this, so any such studies will be thrown out.

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

They might be painful and fatiguing illnesses since they are also post-sepsis syndrome with the reactivated herpes of all kinds. And CDC officer Suzanne Vernon lies about mycoplasma playing a role in ME/CFS (Occam's Razor). And the herpes love ganglia, right where the um, "catastrophizing" pressure points are. You've already seen in the Razor that EBV may be antibody-negative.

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:

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 (see the Occam's Razor) 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 and they inhibit apoptosis in infected cells, which is very close to a pre-cancer state. You'll remember from the Occam's Razor or your own discovery 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. 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 fatiguesyndrome.
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 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.

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.

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.

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."