

Military Toxins, Autoimmune Disorders, The Gulf War Syndrome (GWS) and the Aluminum Adjuvant Vaccine Connection

By <u>Dr. Gary G. Kohls</u>
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Gulf War Syndrome refers to the complex of symptoms that affects veterans of the 1990-1991 Gulf War at significantly excess rates. It is characterized by multiple diverse symptoms not explained by established medical diagnoses or standard laboratory tests, symptoms that typically include a combination of memory and concentration problems, persistent headache, unexplained fatigue, and widespread pain, and can also include chronic digestive difficulties, respiratory symptoms, and skin rashes.

...the biological effects of different combinations of pyridostigmine bromide (PB), multiple pesticides, low-level nerve agents, oil and dense smoke from burning wells, depleted uranium (DU) weaponry dust, fuel vapors, exhaust from tent heaters, Chemical Agent Resistant Coating (CARC) paint, airborne particulates, infectious agents, and receipt of multiple vaccines, experienced concurrently or over a brief time period, are unknown. Many have suggested that unknown and difficult-to-characterize effects may have been precipitated by an 'exposure cocktail' or 'toxic soup' effect during Gulf War deployment.

"Non-deployed veterans who reported getting vaccines...had significantly higher rates of symptoms in several domains (chronic somatic pain, neurological, and gastrointestinal problems) and a nearly four-fold higher rate of Gulf War illness than non-deployed veterans who did not receive vaccines. Veterans who served in theater, by comparison, had Gulf War illness symptoms at 11 times the rate of non-deployed veterans who did not receive vaccines." –

The above three quotes have been excerpted from the 465 page VA scientific document concerning the soldier victims of Gulf War I. There was very little mention of the now-well-known toxic effects of aluminum adjuvants in the document, which can be accessed at:

http://www.va.gov/gulfwaradvisorycommittee/docs/GWlandHealthofGWVeterans_RAC-GWVIReport_2008.pdf)

* * *

Recently I attended a seminar at an area college that dealt with how such a college campus might be more welcoming to Gulf War veterans who are enrolling at relatively high rates, thanks to the GI Bill. The faculty did a good job of discussing the many obstacles that every returning veteran faces when he or she returns to domestic life, including academic life. I did notice that there were some important medical issues that were not discussed, but medical issues were beyond the areas of expertise of the seminar presenters and probably

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not expected to be part of the discussion.

I actually am quite familiar with the situations that colleges are facing when it comes to traumatized or toxified veterans in academia. Not only had I studied posttraumatic stress disorder (PTSD) for several decades as a part of my medical practice and teaching experiences, but I also practiced as a physician at a mental hospital for 2 ½ years in the late 1990s. Following that, I spent nearly a decade practicing holistic mental healthcare.

During that practice experience, I dealt with literally hundreds of patients with both fullblown and partial expressions of PTSD (domestic as well as military victims of severe psychological trauma). Significantly, most of those patients had never been previously diagnosed with PTSD, a very easily diagnosable disorder.

Simultaneous with the time that I had my independent holistic mental healthcare practice, I also taught – for 6 semesters – an upper level psychology class at the University of Minnesota-Duluth. The course was titled "The Science and Psychology of the Body-Mind Connection".

In that class, I spent a lot of time teaching my students (who were mostly juniors, seniors or graduate students (destined for psychology or sociology careers) about the realities of PTSD (especially the combat-induced variety). We also discussed the root causes of violence, the basic neuroscience of the brain, how neurotoxic psychiatric drugs work at the synapse level and the science and healing qualities of optimum brain nutrition.

During the course, I had my students watch and then write papers on "Beyond Vietnam" (a powerful Veterans for Peace video about the psychological consequences of combat war), "One Flew Over the Cuckoo's Nest" and Pink Floyd's "The Wall", all powerful films that nicely illustrated the realities of PTSD (which is all too-often mis-diagnosed as a mental illness "of unknown cause" and therefore mis-treated). The vast majority of my students rated the class mostly 5s out of 5 in their end-of-semester evaluations of the course.

Over those six semesters, two Gulf War I veterans (that I knew of) enrolled in my class. Both of them missed lectures and also missed handing in some papers. They usually failed to participate in class discussions and ultimately both abruptly withdrew before the end of the semester without warning or asking my counsel. I never found out the real reasons why they withdrew. I think that they both dropped out of college entirely.

The 'Toxic Soup' Effect of Military Deployment is Partly latrogenic – and Very Long-lasting

As careful readers of my Duty to Warn column understand, I have over the last year become well-versed with the recent basic science-generated neuro-toxicology studies that indict any number of psychiatric drugs and also various common vaccine ingredients for contributing to autoimmune disorders, neurological disorders and mental dysfunction (and even damage to brain tissue).

One of the realities of American military life is the rather cavalier administration of cocktails of psychiatric drugs (during the deployment or post-deployment phases) and multiple vaccinations (pre-deployment and again during deployment). Usually, for military efficiency, vaccines are given in batches, often on the same day, with boosters often given later.

One of the vaccines that were routinely given to Gulf War I and II soldiers prior to deployment (in addition to many other vaccines), was an experimental anthrax vaccine,

which had never been approved for use in humans by the FDA. Some of the soldiers received multiple anthrax shots, each one containing an aluminum adjuvant and sometimes squalene (adjuvants are included in most vaccines to boost the immune response, which is generally weak without an adjuvant). Squalene is a 30 carbon Omega-2 fatty acid that has powerful adjuvant properties. It is extracted from the liver of deep water sharks in impure form.

Unfortunately (unappreciated by military authorities) aluminum adjuvants are well-known to unexpectedly cause a hyper-immune response in both animal lab subjects and humans, thus causing autoimmune disorders of various types. This reality is thought to explain the epidemic of autoimmune disorders in fully vaccinated individuals, including the epidemic of chronic illnesses in fully vaccinated children all across the nation. See one of the abstracts below that summarizes a well-designed study that introduces a newly-described syndrome, the Autoimmune/inflammatory Syndrome Induced by Adjuvants), aka ASIA (or Shoenfeld's Syndrome).

Other studies that are abstracted in the second half of this article explain why aluminum adjuvants could be expected to cause autoimmune disorders. (Recall that the aluminum and other adjuvants that are in vaccines are there specifically to increase the immunogenicity of the main vaccine ingredient, usually a viral protein antigen.)

The Definition of Autoimmune Disorder

An autoimmune disorder is a disease in which the body produces antibodies that attack its own tissues, leading to the deterioration or destruction of such tissue.

Vaccines are usually injected into muscle tissue (usually the thigh muscles of babies or the deltoid muscles of older folks) where there also happens to be a mixture of normal endothelial tissue, vascular tissue, nerve tissue, collagen, DNA, mitochondria, platelets, white blood cells and myelin (the fatty sheath that insulates some nerves).

Any adjuvants in the vaccines (which are intended to make the body's immune system build up antibodies against the antigen that is targeted, – such as HPV, DPT, influenza viruses, pneumococcal antigens, etc) can now inadvertently cause those previously normal tissues to be regarded as foreign by the immune system, which will then attack them (the very definition of autoimmune disorders above).

At any rate feeling qualified to offer my opinions to the above-noted college seminar audience, I spoke briefly to the group, hoping to shed some light on the worthy veteran's project.

Later in the day, in a follow-up email to the college's faculty, I tried to enlarge and clarify my concerns in the following statement:

Hello, I was the commenter that followed the seminar earlier today. As you probably could sense, I sensed an urgency to speak to the audience, especially the Veterans Task Force members that hosted the event and the veteran students in the audience (most of whom had unfortunately left by the time I spoke). I wanted to relay to you some details about the tremendous progress that is being made in understanding the root causes – and therefore the rational treatment – of the Gulf War Syndrome (GWS) that has been

published in the basic science research literature.

Many investigators into the GWS epidemic are becoming increasingly aware of the new research that concerns the adverse physical, neurological and psychiatric effects of the many military toxins that affected many, if not all, Gulf War vets. See the list above.

Among the examples of military toxins that I as a physician am most familiar with are the physician-prescribed psych drug cocktails (never proven for safety or efficacy in any population, including the military population) and the physician-prescribed vaccines (also never proven for safety or efficacy, especially when given in combinations).

Any of those 'drugs' can cause physical illnesses as well as neurotoxic illnesses (and thus psychotoxic illnesses) in some form or another. Given the multitude of toxic exposures that all soldiers experience after induction, one can expect that the academic preparedness of Gulf War veterans will also be adversely affected.

Of course, every investigator into the psychology and neurology of GWS understands that there are many potential contributing factors other than toxic drugs or toxic vaccines. Just consider the effects of the acute and chronic physical, emotional, psychiatric and neurological stresses that every combat vet experiences. Such stressors can be devastating to anybody, but each victim will react totally different from the next.

Combat vets from each of America's most infamous and most tragic undeclared high tech wars (Vietnam and Gulf Wars I & II) commonly experience chronically elevated (and therefore potentially brain-altering) cortisol and adrenalin levels, but Vietnam vets also suffered from exposure to Agent Orange, a seriously neurotoxic and carcinogenic herbicide that seriously damages the body's mitochondria.

Gulf War vets were exposed to neurotoxic pesticides, just like Vietnam vets were, but they perhaps might have been somewhat less toxic and shorter-acting than the infamous dioxins that were in Agent Orange.

On the other hand, Vietnam vets were NOT exposed to the experimental Anthrax vaccines that even non-deployed Gulf War soldiers received, and Vietnam vets got far fewer vaccinations in general. GWS soldiers were heavily inoculated with vaccines that contained mercury [thimerosal] preservatives plus aluminum and squalene adjuvants) that Vietnamera soldiers did not get.

And, even though many Vietnam vets came back from war heroin-addicted and alcohol-dependent, they were not saturated with anywhere near the same number of potentially psychiatric drug cocktails that Gulf War vets did.

So the issue of comprehensively helping with the academic and social performance of military veteran students (who may or may not be experiencing full-blown or partial expressions of either GWS or PTSD) is a much more complex issue that any faculty member can be expected to comprehensively deal with, but the Task Force members must understand the situation as comprehensively as possible. I suspect that affected veteran students would thank you for doing so.

As I mentioned in my comments earlier today, fully understanding the implications of the new research into vaccine toxicity would take hours of study. My 10 minutes of commentary was insufficient to do more than perhaps whet your appetites to learn more, but I would

hope that there would be some attempt by the Task Force to be totally open to new information and to impart that information to the affected students so that they could make good use of it somehow.

Therefore, I attach below, in the form of a handful of abstracts from several basic neuroscience journals. This information is actually just a small fraction of the new information that, as I mentioned, is not being published in mainstream medical journals (and thus is likely unappreciated by mainstream physicians).

It needs to be said that most medical professionals don't willingly discuss iatrogenic (physician- or treatment-caused) diseases. Nor do most medical professionals want to discuss new illnesses that they don't yet know much about. That sort of avoidance response also seems to apply to most medical trade associations such as the AMA, the APA, the AAP, the AAFP, etc as well as most medical journal editors. But I think we all agree that any good liberal arts college should be open to – indeed should seek out – any and all new information that might assist in the solving of problematic situations that we all face daily, including what to do about the victims of Gulf War Syndrome.

Below are seven research study abstracts that might help you and your college's veteran's more fully understand how they might have been adversely affected long-term by potentially neurotoxic and autoimmunity-inducing substances that were in some of the vaccines that they received, especially the experimental anthrax vaccines. – Gary G. Kohls, MD, Duluth, MN

ANNEX

Seven Research Study Abstracts

Expert Rev Clin Immunol. 2013 Apr;9(4):361-73. doi: 10.1586/eci.13.2.

Autoimmune/inflammatory syndrome induced by adjuvants (Shoenfeld's syndrome): clinical and immunological spectrum.

Vera-Lastra O¹, Medina G, Cruz-Dominguez Mdel P, Jara LJ, Shoenfeld Y.

 Hospital de Especialidades Centro Médico La Raza, Instituto Mexicano del Seguro Social, Mexico City, Mexico.

http://www.ncbi.nlm.nih.gov/pubmed/23557271

Abstract

An adjuvant is a substance that enhances the antigen-specific immune response, induces the release of inflammatory cytokines, and interacts with Toll-like receptors and the NALP3 inflammasome. The immunological consequence of these actions is to stimulate the innate and adaptive immune response. The activation of the immune system by adjuvants, a desirable effect, could trigger manifestations of autoimmunity or autoimmune disease. Recently, a new syndrome was introduced, autoimmune/inflammatory syndrome induced by adjuvants (ASIA), that includes post-vaccination phenomena, macrophagic myofasciitis (MMF), Gulf War syndrome and siliconosis. This syndrome is characterized by nonspecific and specific manifestations of

autoimmune disease. The main substances associated with ASIA are squalene (Gulf War syndrome), aluminum hydroxide (post-vaccination phenomena, macrophagic myofasciitis) and silicone with siliconosis. Mineral oil, guaiacol and iodine gadital are also associated with ASIA. The following review describes the wide clinical spectrum and pathogenesis of ASIA including defined autoimmune diseases and nonspecific autoimmune manifestations, as well as the outlook of future research in this field.

Neuromolecular Med. 2007;9(1):83-100.

Aluminum adjuvant linked to Gulf War illness induces motor neuron death in mice.

Petrik MS¹, Wong MC, Tabata RC, Garry RF, Shaw CA.

http://www.ncbi.nlm.nih.gov/pubmed/17114826

Abstract

Gulf War illness (GWI) affects a significant percentage of veterans of the 1991 conflict, but its origin remains unknown. Associated with some cases of GWI are increased incidences of amyotrophic lateral sclerosis and other neurological disorders. Whereas many environmental factors have been linked to GWI, the role of the anthrax vaccine has come under increasing scrutiny. Among the vaccine's potentially toxic components are the adjuvants aluminum hydroxide and squalene. To examine whether these compounds might contribute to neuronal deficits associated with GWI, an animal model for examining the potential neurological impact of aluminum hydroxide, squalene, or aluminum hydroxide combined with squalene was developed. Young, male colony CD-1 mice were injected with the adjuvants at doses equivalent to those given to US military service personnel. All mice were subjected to a battery of motor and cognitive-behavioral tests over a 6-mo period postinjections. Following sacrifice, central nervous system tissues were examined using immunohistochemistry for evidence of inflammation and cell death. Behavioral testing showed motor deficits in the aluminum treatment group that expressed as a progressive decrease in strength measured by the wiremesh hang test (final deficit at 24 wk; about 50%). Significant cognitive deficits in water-maze learning were observed in the combined aluminum and squalene group (4.3 errors per trial) compared with the controls (0.2 errors per trial) after 20 wk. Apoptotic neurons were identified in aluminum-injected animals that showed significantly increased activated caspase-3 labeling in lumbar spinal cord (255%) and primary motor cortex (192%) compared with the controls. Aluminum-treated groups also showed significant motor neuron loss (35%) and increased numbers of astrocytes (350%) in the lumbar spinal cord. The findings suggest a possible role for the aluminum adjuvant in some neurological features associated with GWI and possibly an additional role for the combination of adjuvants.

Exp Mol Pathol. 2002 Aug;73(1):19-27.

Antibodies to squalene in recipients of anthrax vaccine

Asa PB1, Wilson RB, Garry RF.

Abstract

We previously reported that antibodies to squalene, an experimental vaccine adjuvant, are present in persons with symptoms consistent with Gulf War Syndrome (GWS) (P. B. Asa et al., Exp. Mol. Pathol 68, 196-197, 2000). The United States Department of Defense initiated the Anthrax Vaccine Immunization Program (AVIP) in 1997 to immunize 2.4 million military personnel. Because adverse reactions in vaccinated personnel were similar to symptoms of GWS, we tested AVIP participants for anti-squalene antibodies (ASA). In a pilot study, 6 of 6 vaccine recipients with GWS-like symptoms were positive for ASA. In a larger blinded study, only 32% (8/25) of AVIP personnel compared to 15.7% (3/19) of controls were positive (P > 0.05). Further analysis revealed that ASA were associated with specific lots of vaccine. The incidence of ASA in personnel in the blinded study receiving these lots was 47% (8/17) compared to an incidence of 0% (0/8; P < 0.025) of the AVIP participants receiving other lots of vaccine. Analysis of additional personnel revealed that in all but one case (19/20; 95%), ASA were restricted to personnel immunized with lots of vaccine known to contain squalene. Except for one symptomatic individual, positive clinical findings in 17 ASA-negative personnel were restricted to 4 individuals receiving vaccine from lots containing squalene. ASA were not present prior to vaccination in pre-immunization sera available from 4 AVIP personnel. Three of these individuals became ASA positive after vaccination. These results suggest that the production of ASA in GWS patients is linked to the presence of squalene in certain lots of anthrax vaccine.

Mitochondrial Dysfunction in Gulf War Illness Revealed by 31Phosphorus Magnetic Resonance Spectroscopy: A Case-Control Study

Hayley J. Koslik, Gavin Hamilton, and Beatrice A. Golomb

Published: March 27, 2014

http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0092887

Of the 700,000 US troops deployed to the 1990-1 Persian Gulf theater, an estimated 175,000–250,000 (\sim 1/4–1/3 of those deployed), developed chronic multi-symptom health problems often termed "Gulf War illness" (GWI) [1]. GWI is characterized by protean symptoms spanning multiple symptom "domains" (such as cognitive, fatigue, musculoskeletal).

Gulf deployed veterans on average have more symptom domains affected, and greater severity and multiplicity of symptoms within domains, than Gulf era veterans who were not deployed [2]. Fatigue, exercise intolerance, cognitive difficulties, muscle pain and weakness, shortness of breath, gastrointestinal problems, sleep problems, behavior change, neurological findings, and skin problems are all elevated. GWI is defined by symptoms; a number of objective findings have been replicated, such as autonomic dysfunction, increased autoantibodies, reduced natural killer cell activity, and increased coagulation activation among others [3]. Studies generally show that affected veterans have not improved with time [4], [5], [6]; Gulf War veterans (GWV) continue to experience symptoms and impaired function 23 years later.

GWI is not equivalent to signature conditions of subsequent deployments to the region, such as

posttraumatic stress disorder and traumatic brain injury. Indeed, stress and combat are demonstrably not the cause. While combat stress bears a dose-response relation to posttraumatic stress disorder (including in GWV); it bears no significant relationship to GWI in studies that adjust for other exposures [4].

Environmental factors are clearly inculpated in GWI. Many exposures were new, unique or excessive in the Gulf War. These include heat, sand, <u>depleted uranium tanks/munitions</u>, chemical agent resistant coating paint, and oil fires; as well as protections such as <u>high numbers of multiple vaccines</u>, <u>anthrax vaccine</u>, pyridostigmine bromide nerve agent pretreatment pills, pesticides and insect repellents, and permethrin-impregnated uniforms among others. Evidence most strongly implicates acetylcholinesterase inhibitor (AChEi) related exposures (which adhere to Hill's criteria for causality, include a dose-response relationship, and are buttressed by gene-exposure interaction data) [7].

Multiple vaccinations and anthrax vaccine also show relatively consistent epidemiological associations, but do not have the triangulating evidence for causality. AChEi related exposures include (carbamate) pyridostigmine bromide nerve agent pretreatment pills, given to an estimated 250,000 US troops [8]; carbamate and organophosphate pesticides [9], used aggressively and sometimes excessively to protect against insect vectors of disease [10], [11]; and organophosphate nerve gas, to which the Department of Defense estimates as many as ~100,000 US troops were exposed in association with the demolition of the Khamisiyah munitions depot [12], with other possible nerve agent exposures [13]. Number of exposures experienced has also been linked to illness [14]; and exposure interactions, conceptually and empirically, may produce more problems [15].

The involvement of AChEi provides important information regarding potential mechanisms. Whereas AChEi toxicity is often viewed in terms of acetylcholinesterase inhibition, evidence shows that toxicity and lethality in fact relate decisively to (intertwined) oxidative stress (OS) and mitochondrial dysfunction (MD) [16] (phenomena that are tightly intertwined because the mitochondria are a leading target and source of reactive oxygen species [17], [18], [19]): indeed, animal evidence shows that high quality antioxidants administered just before or just after organophosphate pesticide exposure protect against lethality and chronic sequelae [20],[21].

We have shown that a mechanism involving OS-MD would explain the symptom profile (including which symptoms dominate – fatigue as well as central nervous system and muscle symptoms dominate in MD), symptom multiplicity, protean symptom character, variable latency to onset of symptoms, and the objective markers linked to GWI [3]. This mechanism would also provide for a subsidiary role for multiple other exposures for which mechanisms of action are classically considered to be unrelated, but which share in common induction of OS – potentiating MD and further OS.

The entire article and references are posted at: http://journals.plos.org/plosone/article?id=10.1371/journal.pone.0092887

Front Neurol. 2015 Feb 5;6:4. doi: 10.3389/fneur.2015.00004. eCollection 2015.

Biopersistence and brain translocation of aluminum adjuvants of vaccines.

Gherardi RK¹, Eidi H¹, Crépeaux G¹, Authier FJ¹, Cadusseau J¹.

http://www.ncbi.nlm.nih.gov/pubmed/25699008

Abstract

Aluminum oxyhydroxide (alum) is a crystalline compound widely used as an immunological adjuvant of vaccines. Concerns linked to the use of alum particles emerged following recognition of their causative role in the so-called macrophagic myofasciitis (MMF) lesion detected in patients with myalgic encephalomyelitis/chronic fatigue/syndrome. MMF revealed an unexpectedly long-lasting biopersistence of alum within immune cells in presumably susceptible individuals, stressing the previous fundamental misconception of its biodisposition. We previously showed that poorly biodegradable aluminum-coated particles injected into muscle are promptly phagocytosed in muscle and the draining lymph nodes, and can disseminate within phagocytic cells throughout the body and slowly accumulate in brain. This strongly suggests that long-term adjuvant biopersistence within phagocytic cells is a prerequisite for slow brain translocation and delayed neurotoxicity. The understanding of basic mechanisms of particle biopersistence and brain translocation represents a major health challenge, since it could help to define susceptibility factors to develop chronic neurotoxic damage. Biopersistence of alum may be linked to its lysosome-destabilizing effect, which is likely due to direct crystal-induced rupture of phago-lysosomal membranes. Macrophages that continuously perceive foreign particles in their cytosol will likely reiterate, with variable interindividual efficiency, a dedicated form of autophagy (xenophagy) until they dispose of alien materials. Successful compartmentalization of particles within double membrane autophagosomes and subsequent fusion with repaired and re-acidified lysosomes will expose alum to lysosomal acidic pH, the sole factor that can solubilize alum particles. Brain translocation of alum particles is linked to a Trojan horse mechanism previously described for infectious particles (HIV, HCV), that obeys to CCL2, signaling the major inflammatory monocyte chemoattractant.

Med Hypotheses. 2009 Feb;72(2):135-9. doi: 10.1016/j.mehy.2008.09.040. Epub 2008 Nov 11.

A role for the body burden of aluminium in vaccine-associated macrophagic myofasciitis and chronic fatigue syndrome.

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http://www.ncbi.nlm.nih.gov/pubmed/19004564

Abstract

Macrophagic myofasciitis and chronic fatigue syndrome are severely disabling conditions which may be caused by adverse reactions to aluminium-containing adjuvants in vaccines. While a little is known of disease aetiology both conditions are characterised by an aberrant immune response, have a number of prominent symptoms in common and are coincident in many

individuals. Herein, we have described a case of vaccine-associated chronic fatigue syndrome and macrophagic myofasciitis in an individual demonstrating aluminium overload. This is the first report linking the latter with either of these two conditions and the possibility is considered that the coincident aluminium overload contributed significantly to the severity of these conditions in this individual. This case has highlighted potential dangers associated with aluminium-containing adjuvants and we have elucidated a possible mechanism whereby vaccination involving aluminium-containing adjuvants could trigger the cascade of immunological events which are associated with autoimmune conditions including chronic fatigue syndrome and macrophagic myofasciitis.

J Investig Med High Impact Case Rep. 2014 Mar 18;2(1):2324709614527812. doi: 10.1177/2324709614527812.

Postural Orthostatic Tachycardia With Chronic Fatigue After HPV Vaccination as Part of the "Autoimmune/Auto-inflammatory Syndrome Induced by Adjuvants": Case Report and Literature Review.

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http://www.ncbi.nlm.nih.gov/pubmed/26425598

Abstract

We report the case of a 14-year-old girl who developed postural orthostatic tachycardia syndrome (POTS) with chronic fatigue 2 months following (aluminum-adjuvanted) Gardasil vaccination. The patient suffered from persistent headaches, dizziness, recurrent syncope, poor motor coordination, weakness, fatigue, myalgias, numbness, tachycardia, dyspnea, visual disturbances, phonophobia, cognitive impairment, insomnia, gastrointestinal disturbances, and a weight loss of 20 pounds. The psychiatric evaluation ruled out the possibility that her symptoms were psychogenic or related to anxiety disorders. Furthermore, the patient tested positive for ANA (1:1280), lupus anticoagulant, and antiphospholipid. On clinical examination she presented livedo reticularis and was diagnosed with Raynaud's syndrome. This case fulfills the criteria for the autoimmune/auto-inflammatory syndrome induced by adjuvants (ASIA). Because human papillomavirus vaccination is universally recommended to teenagers and because POTS frequently results in long-term disabilities (as was the case in our patient), a thorough follow-up of patients who present with relevant complaints after vaccination is strongly recommended.

Dr Kohls is a retired physician who practiced holistic, non-drug, mental health care for the last decade of his family practice career. He now writes a weekly column for the Reader Weekly, an

alternative newsweekly published in Duluth, Minnesota, USA. Many of Dr Kohls' columns are archived at http://duluthreader.com/articles/categories/200 Duty to Warn.

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