Editorial—Why is the Hepatitis B vaccine still mandated?

Andrew Maniotis^a, PhD; Rita Maniotis^b; N. Joseph Espat^c, MD; Xue Chen^d, MD; Peter Lycos^e, PhD

^aProgram Director in the Cell and Developmental Biology of Cancer Department of Pathology, Anatomy and Cell Biology, and Bioengineering, College of Medicine Research Building 909 South Wolcott Ave. University of Illinois at Chicago Chicago, IL 60607

^bPast President, Emerson PTA
President, Morton West Parent Teachers Association
Secretary, Illinois Vaccine Awareness Coalition
Berwyn, IL 60402

dOphthalmologist/Ophthalmic pathologist
Peking University Eye Center
49 North Gardon Road
Hai Dian District
Beijing, 100083, P. R. China

^cAssociate Professor of General Surgery, MC 958 435 CSB University of Illinois at Chicago Chicago, IL 60612

> ^ePhysical Chemist Illinois Institute of Technology Chicago IL, 60616-3793

Abstract

Evidence from many sources show the hepatitis B vaccine is not safe or effective, and is linked to many autoimmune syndromes. Hepatitis B syndrome is rare (0.00024%-hovering near zero percent for both adults and children), while over 10.4% of hepatitis B vaccine recipients experience adverse vaccine reactions. According to package inserts, 1% of these are serious enough for emergency room admission. France discontinued its vaccination of school-aged children because of the types and high rate of catastrophic illnesses linked to the vaccine. Molecular markers (HBsAg, anti-HBsAg, HbeAg, anti-HbeAg, or HBV-DNA) are not diagnostic or predictive of liver disease, and Down sydrome, leukemia, and genetic polymorphisms related to differences in disease susceptibility were central to the discovery of HBV. Cell culture, animal models, and human studies have failed to show cytopathic effects in liver consistent with the hypothesized pathogenicity of HBV infection. In regions where the HBV markers are endemic, long-term study has shown that the vaccine increased the rate of hepatitis B syndrome in teens. The claim that seropositivity for HBV markers is linked to liver cancer decades later may be a form of molecular mimicry, and it is unsupported by evidence: it is like claiming that a freckle on an infant signals that melanoma will develop decades later. To date, and despite congressional investigations and numerous studies questioning the safety of the hepatitis B vaccine, informed consent and the original trial safety data has not been provided by the public health service.

© Copyright 2006 Pearblossom Private School, Inc-Publishing Division. All rights reserved.

Keywords: Hepatitis B vaccine, HBsAg, HBeAg, adverse events, mimicry, HCC

The Vaccine Adverse Events Reporting System (VAERS) shows that the hepatitis B vaccine damages far more individuals than there are persons who exhibit the hepatitis B syndrome. Evidence obtained from the American Association of Physicians and Surgeons (AAPS) and other physicians, vaccinemonitoring agencies such as the National Vaccine Information Center (NVIC), the CDC and World Health Organization, the Illinois Vaccine Awareness Coalition, the hepatitis B vaccine manufacturers Merck and GallaxoSmithKline, and evidence from the peer reviewed scientific literature, all show that the risk of groups such as infants and children acquiring liver hepatitis associated with hepatitis B virus (HBV) is nearly 0%. In all comprehensive statistical surveys available, the actual incidence of the hepatitis B syndrome in the US has remained constant at about 2 to 4 cases/ 100,000 individuals despite widespread mandated and aggressive vaccination programs in all but 4 states [1].

The data also show that the hepatitis B syndrome, when it does occur in non-vaccinated individuals, spontaneously resolves in almost 100% of those who became seropositive for the HBV molecular markers (HBsAg, anti-HBsAg, HbeAg, anti-HbeAg, or HBV-DNA). The liver syndrome is quite rare (0.00024%-hovering near zero percent for both adults and children), while over 10% of hepatitis B vaccine recipients experience adverse reactions. According to the Merck package inserts,

10.4% experience adverse reactions, and 1% are serious enough for emergency room admission.

Some of the severe adverse effects include autism, Stevens-Johnson Syndrome, arthritis (both transient and permanent), Guillain-Barré Syndrome, myelitis including transverse myelitis, seizure, febrile seizure, peripheral neuropathy including Bell's palsy, diabetes mellitus, pancreatitis, encephalitis, multiple sclerosis, thrombocytopenia, systemic lupus erythematosus, lupus-like syndrome, vasculitis, optic neuritis, radiculopathy. Lesser vaccine effects include vomiting, abdominal pains, vertigo, dizziness, pruritus, angioedema, urticaria, lymphadenopathy, insomnia, dysuria, hypotension, increased risk of shingles, migraine, severe muscle pain and weakness, hypesthesia, alopecia, petechiae, increased sedimentation rate, tinnitus, conjunc vitis, visual disturbances, syncope, tachycardia, keratitis, irritability [2].

The practical and economic impact of the effects of this mandated recombinant hepatitis B vaccine policy is devastating infants, families, and the nation, because the nature and frequency of the vaccine damage is typically so debilitating. The damage experienced during the French mandatory hepatitis B program prompted France to discontinue its hepatitis B program several years ago, and a class action lawsuit compensated some 15,000 families that had been devastated from hepatitis B vaccine injury [3].

doi: 10.1588/medver.2006.03.00135

The efficacy of the vaccine has also been challenged. According to some long-term studies in populations said to exhibit "endemic" frequencies of markers indicating endemic hepatitis B "infection" such as Gambia and Egypt, antigenicity (the presence of the HbsAg antibody among the vaccinated) does not persist beyond about 5 years [4], yet expression of the hepatitis B syndrome confers immunity and antigenicity for life [5] in nearly 100% of those who are unvaccinated and experience the full-blown syndrome that spontaneously resolves without significant morbidity in almost all cases [1]. A study conducted with Egyptian children, reported a similar lack of long-term antigenicity as determined by antibody levels. The study population comprised six equal groups (30 children in each group, 15 boys and 15 girls) at different post-vaccination intervals following the completion of the third dose of HB vaccine: 1 month (group 1), 1 year (group 2), 2 years (group 3), 3 years (group 4), 4 years (group 5), and 5 years (group 6). "63.3% of the children in group 1 had a good immune response (anti-HBs > 100 mIU/mL), in groups 2 and 3 this had dropped to 43.3%, to 23.3% in group 4, 6.7% in group 5 and in group 6 (5 years post-vaccination) none of the children had a good immune response (0.0%)" [6].

The "cryptic argument," that every person on the planet must be vaccinated because the hepatitis B "virus" can hide in cells in "chronic carriers" for decades without causing clinically detectable disease, and then mysteriously, decades later, "cause" hepatocellular carcinoma, ignores the fact that seropositivity for the hepatitis B antigens may not have anything to do with serum hepatitis. In the vast majority of seropositive individuals without liver disease, the presence of the HBV markers may represent non-specific markers of immunological stress, or merely represent a normal genetic polymorphism, as was originally thought by Baruch Blumberg (who discovered the Au antigen, HbsAg, in the blood of a black Australian aboriginal, and was awarded the Nobel Prize that he shared with NIH's former Neurobiology Program director, D. Carlton Gajducek—the discoverer of the so-called "slow virus" prion diseases). For these discoveries, the doctors were jointly given The Nobel Prize in Physiology or Medicine in 1976 "for their discoveries concerning new mechanisms for the origin and dissemination of infectious diseases," because the infectious agents and mechanisms of disease causation were believed not to conform to the standards of accepted pathogen isolation, the idea of distinctive genetic (nucleic acid) identity, the timing of infection to demonstrable cell pathology or morbidity, or to the classic proofs of pathogenicity worked out by Koch. For instance, D. Carlton Gajducek championed the idea that "infectious proteins" devoid of nucleic acids were at the basis of slow, debilitating neurodegenerative disorders (e.g., kuru, CJD, Mad Cow, scrapie in sheep)—syndromes that are characterized by extremely long latency periods after initial "infection," and destruction of the brain tissue years or decades after "infection." Although the concept of slow viruses, and pathogens devoid of nucleic acids were vigorously challenged and rejected by many in the scientific establishment during the 1980's because the idea challenged the established biochemical chain of events worked out for all other infectious agents, and because these syndromes appeared to be both infectious and run in families, Stanley Pruisner believed Gajducek's hypotheses to be plausible, and found that the hypothesized disease-causing PRP protein was present in both diseased and healthy hamsters (for which another Nobel Prize was awarded).

Blumberg termed the rare "hepatitis B" antigen, Au, for Australian antigen, because the Au antigen was first found in the blood of a healthy black, Australian aboriginal man, but he also detected it in the blood samples of Micronesians, Vietnamese, Taiwanese, Native Americans, and patients with Down syndrome, leukemia and transfusion patients. Blumberg acknowledged, however, that the vast majority of people, who test positive for HbsAg or HbeAg, never become sick, develop hepatitis, or cancer of any kind.

For instance, as the first to identify the hepatitis B antigen in their survey of genetic polymorphisms in blood samples, Blumberg and Alter reported that leukemia patients (not liver cancer patients), patients with Down syndrome, hemophiliacs, and blood transfusion recipients tested positive more than the general population for the hepatitis B antigen, yet rarely developed liver hepatitis [7], suggesting there is no specificity or pathogenicity with respect to Au marker and the appearance of the rare hepatitis B syndrome (because these syndromes are manifested due to vastly different etiologies).

Therefore, despite the presence of (Au) HBsAg antigen in a blood sample of a rare patient with full-blown hepatitis or liver cancer, the antigen did (does) not predict who would (will) develop clinically detectable hepatitis, and is not a specific marker for the development of liver cancer. Consequently, the genetic polymorphic "causes," or physiological stress "causes" of hepatitis as a form of autoimmune dysfunction or stress have been largely ignored, and instead, an infectious viral cause for hepatitis was advanced, as it was for pellagra, SMON, and a variety of other syndromes.

Blumberg and his colleagues reasoned that a virus might cause hepatitis because something smaller than bacteria that was associated with inducing transfusion hepatitis could pass through filter pores too small for bacteria to pass. Yet not only viruses, but foreign and antigenic proteins also can pass through these filters, and it has been well established in the medical literature since that era that foreign proteins can profoundly disturb the immune system, specific organs, and organ systems.

Although some agencies such as the World Health Organization and others claim that about 40-60 percent of liver cancer is attributable to HBV, how do we explain the fact that about 1/3 of Down patients also express the Au antigen, and 1 in 10 leukemia patients express the antigens, according to Blumberg, yet Down's syndrome isn't due to a virus – its due to chromosomal non-disjunction.

Regardless of what "causes" the rare hepatitis B syndrome or the appearance of the hepatitis B "markers" (which most physicians admit is likely due to an autoimmune disease process set into motion by a viral infection), and despite the ill-defined molecular markers that appear in 1/3 of Down syndrome children, and in 1 in 10 leukemia patients according to Blumberg [7], abundant evidence accumulated by the VAERS and the CDC shows that the hepatitis B vaccine is strongly associated with an unacceptable frequency of debilitating life-long illnesses.

Despite widespread mandated hepatitis B vaccines for more than a decade, and claims that it can prevent heptatocellular carcinoma, no evidence whatsoever exists linking hepatitis B causally with hepatocellular carcinoma, as no animal models have ever exhibited this carcinoma after experimental infections, and no liver cell culture of normal human or animal liver cells has ever been induced to change into cancerous cells after adding the "hepatitis B agent" to them.

As titles of papers about Hepatitis B published in journals as prestigious as *Science* sometimes suggest [8], it is reasonable to ask why neither chimps show liver pathogenicity, cellular damage, or develop anything resembling hepatitis in modern studies when they are experimentally infected with "hepatitis B," nor do humans show cytotoxic damage either [9]. In this respect, one might reasonably wonder why "hepatitis B" and "hepatitis C," are not considered primarily acquired autoimmune diseases, rather than infectious viral diseases, since cellular pathology in most cases is not present?

It also should be added that the antigenicity (the presence of the "hepatitis B" antibodies among the vaccinated) does not persist beyond about 5 years, yet hepatitis infections of all kinds confer immunity and antigenicity for life in those who are unvaccinated and experience a full-blown hepatitis B syndrome that spontaneously resolves in almost all cases. Moreover, despite widespread mandated hepatitis B vaccination in 47 states, liver cancer rates have increased in the US from 4 cases/100,000, in 1992, to 5.5 cases/100,000, since at the end of 1999, and leukemia rates have slightly decreased according to the NCI and CDC's official records.

One should rightly ask if a decade even qualifies as long enough to make such claims about a vaccine that prevents liver cancer, or which is associated with 10% of leukemia cases decades later. First of all, epidemiological studies cannot be used to claim a causal connection between the expression of a protein in a person's blood, and the development, or non-development of a cancer, decades after infection. In addition, a Japanese study" claims that heavy drinking rather than transfusions or cigarette smoking accounted for at least 41% of hepatocellular carcinoma patients harbouring antibodies against hepatitis B and C antigens in this country, 50 years after atomic bombs (not a risk factor for cancer?) were dropped by the U.S. on two of its civilian populations, such as Hiroshima, 175 miles away [10].

In this regard, the hepatitis B antigens may be only non-specific markers for some cancers or other grave medical conditions, as Blumberg first believed. The claim that seropositivity for HBV markers is linked to liver cancer decades later is unsupported by evidence, and, it is like claiming that a freckle on an infant signals that melanoma will develop decades later.

As scientists and physicians, or as concerned citizens, we should no longer allow this dangerous state-mandated vaccine program to proceed, while it continues to cause hundreds of vaccine damaged persons for every one person the vaccine supposedly protects.

By so doing, we are irresponsibly risking the health of a generation of infants and children, without providing parents with information about the adverse vaccine reactions, because of propaganda that suggests that by vaccinating them, we will insure that they will not contract hepatitis B or liver cancer if they grow up to become needle-using drug addicts, persons with multiple sex partners, prisoners, mental health patients, or health care workers exposed to human blood.

This kind of fear mongering and propaganda not only ignores evidence showing that these possibilities are without foundation, but functions to stifle legitimate questions about the biology of the hepatitis B syndrome, or legitimate questions concerning the benefits and consequences of vaccination that should have been addressed before this (or any) vaccine was mandated. The Advisory Committee on Immunization Practices (ACIP) should have asked the following legitimate questions:

- 1. Why is such alarm regarding hepatitis B sweeping across the planet now as a sexually transmitted syndrome, when jaundice (and assumed hepatitis) has been recorded in the medical literature since the time of the Ancient Greeks?
- 2. Because it is claimed that hepatitis B can only be spread through venereal contact or through exposure to infected fluids, are humans more promiscuous now than they were during the Eleusinian orgies and Roman bacchanals chronicled by the ancient poets?
- 3. Because the hepatitis B molecular markers are non-specific, what evidence is there to substantiate that 350,000,000 people in the world are "carriers" of HBV, and that 1,000,000 people in the US are carriers?
- 4. In this regard, why are the projected figures for hepatitis B syndrome given, when data recording the actual incidence of hepatitis B have been available for 20 or more years?
- 5. If the hepatitis B antigens are specific for the hepatitis B syndrome, and if these antigens don't simply represent markers for certain physiological stress responses such as cancer or long term alcohol or drug use, or if the presence of the hepatitis B antigens don't merely represent the different incidence and expression of certain Human genetic polymorphisms (differences in the kinds of molecules found in the blood of different peoples, as was originally thought by Blumberg), then why did Bluberg and his collaborators find the hepatitis B antigens present in a vast majority of healthy people who never develop hepatitis, or in patients experiencing other non-liver related illnesses or genetic disorders? In this same context, why did Blumberg clearly indicate that when Millman came to his laboratory in June of 1967, "and calculated the amount of Au in the serum of carriers and estimated that in some it amounted to about 1% of the serum proteins, that his immediate response was that if this was all virus it would be incompatible with the life of the carrier" (p7124, ref. 8)?
- 6. Do leukemia and hepatocellular carcinoma share something in common other than a high likelihood of generating molecular mimicry, or other types of mimicry, or is the antigen merely expressed in both diseases in persons whose immunology is altered by cancer, alcoholism, autoimmune stress, or altered for some other reason?
- 7. If a hepatitis B (or C) virus could by themselves cause liver cancer decades after infection, then why does the microscopic percentage of those who exhibit the hepatitis B antigens and who develop chronic clinically detectable liver disease, require carcinogenic co-factors "such as fungal afla-

toxins" (p. 7121 paragraph 4), or a lifetime of alcohol abuse or drug consumption to develop cancer [7]?

- 8. Why can't the hepatitis B virus (and C virus) be isolated according to standard isolation techniques, even after a Roman effort and after decades of trying, and why did Blumberg insist that nucleic acids were not recoverable from these "virus isolation" preparations [7:7124, para. 6], but were **inferred** to be a virus? In this same context, why did Blumberg clearly present the fact that "Millman and London found that partially purified Au particles that presumably also contained whole virus particles, which we had not yet visualized, could be transmitted by inoculation into experimental animals. The fully purified particles from which the whole virus had been removed were not infectious. The implication was that we could separate the noninfectious particles containing only the surface antigen from the pathogenic whole virus particles." Is this reason to mount a global vaccine campaign, against what may be inherited genetic and biochemical polymorphisms, that Blumberg believed were at the basis of Au-associated morbidity? Why did Blumberg state that "Additional studies, some of which are still in progress, were consistent with a genetic susceptibility to persistent infection with HBV, which is part of a complex interaction of polymorphic systems. Hence the research on genetic polymorphisms related to differences in disease susceptibility was central to the discovery of HBV" [7:7123, para. 9]?
- 9. What substance(s), then, were actually isolated from sick persons, and modified and developed by Merck as antigenic material to make hepatitis B the first "molecularly derived recombinant" vaccine?"
- 10. Why don't supposedly infectious and pathogenic hepatitis B isolates induce liver disease in chimpanzees, mice, or other organisms [8,9]? Why doesn't it induce either liver cancer or leukemia in animals? Why have there been no instances reported where "the hepatitis B virus" generated a pathological effect in animal models, or in liver cells infected in vitro that even remotely resembles the hepatitis B syndrome's hallmarks in those tiny fraction of seropositive individuals who exhibit morbidity consistent with the hepatitis B syndrome?
- 11. If the recombinant vaccine is molecularly specific against a hepatitis B virus and if the vaccine confers long-term immunity, then why does the vaccine 'wear off' after only several years? By contrast, when the real hepatitis B syndrome resolves in the vast majority of persons in nearly 100% of all cases who develop jaundice and demonstrable liver pathology, then why does this mild and transient syndrome provide lifetime immunity, and produce detectable antibody titres of the hepatitis B antibodies for at least 50 years [5]? If these data are correct and acknowledged even by the vaccine manufacturers, then what is the logic behind vaccinating newborns when their immune and digestive systems are developing and fragile, and when their often hypothesized membership into in IV injecting, multiple sex partner, or blood product exposure risk group might occur a decade or more after the antibodies generated by the vaccine can no longer be detected?

- 12. Why have some studies shown an increase in the hepatitis B syndrome when infants are vaccinated? (e.g., "Children vaccinated in infancy are at *increased risk* of hepatitis B virus infection in the late teens" [11]?)
- 13. Why is the hepatitis B vaccine still mandated after a congressional hearing that put its safety in question, and why aren't are parents given any information at all about the possible adverse effects of the hepatitis B vaccine that are listed on the manufacturer's package inserts?
- 14. Finally, is vaccine policy written according to politics rather than prudence?

The Illinois Department of Public Health versus the Parent Teachers Association of Illinois

A small group of physicians and scientists gained the support of the Illinois PTA in a unanimous decision to support a current halt to the current mandated hepatitis B vaccine. Another way of saying this is that every school representative present at the convention, when shown the data we had obtained, had agreed with our concerns, and immediately held a brief session to advance a motion to direct PTA funding to disseminate literature so that parents would be informed.

This group of perhaps a thousand parents (mostly women), appeared to have only one concern: the total welfare, protection, and education of the school children of Illinois.

It should be stated emphatically, that the current hepatitis B mandate threatens not only our children's health, but also serves to threaten our children's education and admission to all kinds of institutions (day care and school admission), with the bluff that "if you don't get your kid vaccinated against this STD, that is detected only in subpopulations of injection drug users and perhaps highly promiscuous persons, healthy black Australian aboriginal men, Micronesians, Vietnamese, Taiwanese, Native Americans, patients with Down syndrome, leukemia and transfusion recipients, he or she cannot enter school to learn how to read and write." This is not overstating it. Children cannot gain admission into day-care, Kindergarten, elementary schools, junior highs, high schools, and now even colleges, without showing evidence of a mandated (federally-recommended), and dangerous vaccine (hepatitis B).

Pursuing these issues, we presented the current head of the IDPH (Illinois Department of Public Health Director Whitaker and his staff), with the same publicly available data from Medline, the vaccine manufacturer's package insert warnings, data from the Vaccine Adverse Events reporting System, the CDC, Vaccine-link, and other databases, that we had presented to the Illinois PTA convention. After visits with numerous Senators, and public officials during the past several years, over a year later, in June of 2005, we finally were granted a brief meeting with the IDPH, after they could put us off no longer.

As a response to our pleas to institute informed consent regarding the dangers of the hepatitis B vaccine's side effects and safety record as it appears on the Federal government's VAERS database, and after many weeks of deliberation, Dr. Whitaker and his staff emailed us a one paragraph letter stating:

"Parents are currently given enough informed consent."

Well, one may ask Dr. Whitaker, "how do threats that our children won't be admitted to school unless they are jabbed with the hepatitis B vaccine (a rare syndrome) and whose safety data we have yet to see, constitute, informed consent?"

Shouldn't parents at least be given a list of the adverse syndromes induced by the vaccines that are presented on the manufacturer's package inserts, as shown above on Merck's insert? Should parents be shown the VAERS data? Should a list of the hundred or so articles on Medline regarding adverse syndromes induced immediately after vaccination, by mostly private physicians? Shouldn't parents be informed that the data supposedly supporting the safety of the hepatitis B vaccine in neonates doesn't exist [12].

Somebody should tell the public, as we have tried to warn for the past several years, that parents have the right to refuse all vaccines or medical treatments on their children's behalf, with the aid of a publicly-available form on which either religious or philosophical objection to these experimental medical interventions can be declared.

The school nurse and Public Health Department, or school admittance policies should not be used to threaten you that you cannot enroll your kid, based on the madness surrounding the possibility that your 5-year-old will transmit a sexual, or needle-borne, or blood-product-transmitted "syndrome" that has a 95% or greater spontaneous resolution rate, to someone else's 5 year old, (when they have sex or shoot heroin in the gym locker-room, or if they share razor blades-are the reasons typically given to support mandatory vaccination) as the pharmaceutical company and Public Health Service logic goes.

We beg the Public Health Service to regard your own children as potentially at risk for becoming sexually promiscuous and needle-using drug addicts (or health care workers), and use them as experimental subjects of an expensive vaccine possessing a 10.4% rate of adverse events, so they won't contract a rare disease that poses almost 0 risk, that will resolve without treatment in most cases, that simply produces harmless antibodies as evidence of exposure, or that may represent immunological stress or a simple genetic polymorphism, as Blumberg first proposed. Please leave our infants and children alone.

Why in the face of all this damning evidence against the vaccine, does the hepatitis B vaccine mandate still stand with no end in sight? It is because new legislation has insured that there is no incentive, compensation laws, or mechanisms in place anymore to guard against dangerous universally mandated experiments.

The future is here: Medical Terrorism into law

From the "Biodefense and Pandemic and Vaccine and Drug Development Act of 2005—a bill to amend the Public Health Service Act to enhance biodefense and pandemic preparedness activities, and for other purposes [13] **SEC. 319F-3**:

"(a) Authority- As provided in subsection (b), and subject to subsection (b)(1)(C), a manufacturer, distributor [sic; distributor], or administrator of a security countermeasure, or a qualified pandemic and epidemic product, described in subsection (b)(1)(A) or a health care provider shall be immune from suit or liability caused by or arising out of the design, development,

clinical testing and investigation, manufacture, labeling, distribution, sale, purchase, donation, dispensing, prescribing, administration, or use of a security countermeasure, or a qualified pandemic and epidemic product, described in subsection (b)(1)(A)."

Further, subsection (b)(1)(A)(i) reads:

"(i) IN GENERAL- No cause of action shall exist against a person described in subsection (a) for claims for loss of property, personal injury, or death arising out of, reasonably relating to, or resulting from the design, development, clinical testing and investigation, manufacture, labeling, distribution, sale, purchase, donation, dispensing, prescribing, administration, or use of a security countermeasure or qualified pandemic or epidemic product distributed, sold, purchased, donated, dispensed, prescribed, administered, or used in anticipation of and preparation for, in defense against, or in response to, or recovery from an actual or potential public health emergency that is a designated security countermeasure or a qualified pandemic or epidemic product by the Secretary in a declaration described in paragraph (2).

What's being described here is almost carte-blanche freedom to use untested vaccines, drugs, medical products, or "security countermeasures". And there is nothing you, or we, can do about it because it is in the interest of "National Security."

References

- Weinbaum C, Lyerla R, Margolis HS. Prevention and Control of Infections with Hepatitis Viruses in Correctional Settings. CDC MMWR 2003 Jan. 24; 52(RR01):1-33.
- [2] Merck and GallaxoSmithKline package inserts.
- [3] Marshall E. A shadow falls on hepatits B vaccination effort. Science, 1998 Jul. 31;281(5377):630–1.
- [4] Whittle H, Jaffar S, Wansbrough M, Mendy M, Dumpis U, Collinson A, Hall A. Observational study of vaccine efficacy 14 years after trial of hepatitis B vaccination in Gambian children. BMJ, 2002 Sept. 14; 325(7364):569.
- [5] Black FL, Jacobson DL. Hepatitis A antibody in an isolated Amerindian tribe fifty years after exposure. J Med Virol, 1986 May; 19(1):19–21.
- [6] el-Sawy IH, Mohamed ON. Eastern Mediterranean Health Journal 1999;5(5):922–32.
- [7] Blumberg BS. Hepatitis B virus, the vaccine, and the control of primary cancer of the liver. PNAS, 1997; 94:7121–5.
- [8] Guidotti LG, Rochford R, Chung J, Shapiro M, Purcell R, Chisari FV. Viral clearance without destruction of infected cells during acute HBV infection. Science. 1999 Apr. 30; 284(5415):825–9.
- [9] Ordog K, Szendroi A, Szarka K, Kugler Z, Csire M, Kapusinszky B, Xie J, Csizmadia K, Brojnas J, Rusvai E, Tempfli A, Berencsi G. Perinatal and intrafamily transmission of hepatitis B virus in three generations of a low-prevalence population J Med Virol., 2003 Jun;70(2):194–204.
- [10] Pyong SJ, Tsukuma H, Hiyama T. Case-control study of hepatocellular carcinoma among Koreans living in Osaka, Japan. Jpn J Cancer Res. 1994 Jul; 85(7):674–9.
- [11] Hilton Whittle, Shabbar Jaffar, Michael Wansbrough, Maimuna Mendy, Uga Dumpis, Andrew Collison, Andrew Hall. Observational study of vaccine efficacy 14 years after trial of hepatitis B vaccination in Gambian children. BMJ vol 325, 14 September, 2002.
- [12] Lewis E, Shinefield HR, Woodruff BA, Black SB, Destefano F, Chen RT, Ensor R; Vaccine Safety Datalink Workgroup. Safety of neonatal hepatitis B vaccine administration. Pediatr Infect Dis J. Nov;20(11):1049-54, 2001; Also, Testimony of Dr. Marc Geier at IOM hearing, Aug. 2004.
- [13] http://thomas.loc.gov/ Search Bill Title or Number S.1873RS click 'enter bill number.'