Hepatitis B Vaccine: Where is the Public Outrage?

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The polio vaccine was welcomed in 1956 (Salk) and then again in 1962 (Sabin) following 20-30 years of a polio epidemic in the western hemisphere. The HIB vaccine was introduced for administration to infants after thousands of children died or suffered permanent neurological damage from hemophilus influenzae B meningitis and epiglotitis in the 1970s and 1980s. Incidences of wild type polio and HIB infections are now at an all time low in the western hemisphere.

Universal hepatitis B vaccination was introduced in 1991 and is now mandated for administration to every newborn, infant and small child before entering school. Yet there has never been an epidemic of hepatitis B infections in newborns, infants or children. Whereas previous vaccines have been introduced in response to outbreaks of infectious diseases, the introduction of the hepatitis B vaccine sets a new precedent. For the first time, we are giving a vaccine to prevent a viral infection that has no history of causing an epidemic in infants and children.

Hepatitis B Infection: Who Gets It and How?

The populations most at risk for contracting hepatitis B infections are adolescents and adults who use intravenous drugs, engage in frequent, promiscuous, unprotected, multi-partnered sexual activities and those who either handle or are exposed to blood products. Any other suggested method of transmission in humans is rare and often incidental.

Infants born to mothers whose blood tests are positive for hepatitis B surface antigen are at an increased risk of contracting hepatitis B infection. These infants are in danger of becoming chronic hepatitis B carriers. It makes medical sense to administer the hepatitis B vaccine to these select infants. But does it make medical sense to administer the vaccine to all infants? What other groups of infants and children are at risk for hepatitis B infection? The answer is very few, if any. There is no valid medical explanation for vaccinating **all** infants and children with the hepatitis B vaccine.

Infants and small children, as a rule, do not engage in sexual intercourse or use intravenous drugs and are not exposed to blood products. Therefore, it is **medically contraindicated** to administer the hepatitis B vaccine to infants and small children. So, why is the hepatitis B vaccine mandated for infants and children if there has never been a hepatitis B epidemic and they are not at risk for contracting the infection? The short answer is because we can and a critical mass of protesters has yet to form to stop this unfortunate policy.

Before the vaccine was mandated for universal use, vaccine manufacturers invested a lot of money on research and development of the hepatitis B vaccine. To recoup their expenses and earn further profits on their product, they lobbied public health officials to implement a

mass hepatitis B vaccination program for newborns, infants and children.

However, hepatitis B vaccination may not prevent infection in individuals who do not achieve protective antibody titers.[1] There are no tests to determine who the non-responders will be and we have no way of knowing how many will fail to develop antibody titers. Despite progress in vaccinating children and adults in some occupational and racial/ethnic groups, approximately 1.2 million persons in the United States have chronic hepatitis B viral infection, and an estimated 4,000-5,000 persons die each year from hepatitis B virus related liver disease.[2] This represents a death rate of less than 0.5% in the population of people who suffer from hepatitis B related chronic liver disease. The death rate percentage drops even further if we factor in the number of people who each year contract hepatitis B infections and have full recoveries. (Almost 100,000 patients die each year in hospitals from medical error).

So how do we limit the number of cases of hepatitis B infections as an important public health policy and lessen the serious outcomes from hepatitis B infection? Vaccinating all infants and children when they are the most captive of audiences and the least likely to get or transmit the disease and contribute to the overall incidence of hepatitis B infections is the consensus response from public health officials, but this approach is neither medically nor scientifically sound.

Recent evidence from the CDC indicates a decline in the incidence of hepatitis B infection among adolescents and adults.[2] Health officials attribute this decline to changes in social behaviors and improvements in education of high risk populations along with the use of vaccinations. Yet, in the past, the sole policy of vaccinating high risk adolescents and adults had little impact on the overall incidence of hepatitis B viral infection in the general population. Many people in high risk populations do not seek medical attention and are not apt to receive preventive primary medical care. ***Previous vaccination strategies limited to high risk populations had failed to substantially lower the overall incidence of hepatitis B infection.***

The health care officials who approved the hepatitis B vaccine for all infants and children viewed universal vaccination as a golden opportunity and an administrative convenience to reach every member of society before they grow up to *possibly* become members of at risk populations who *might* contract hepatitis B infections which, in most cases, are non-fatal anyway.[3] It is a poor strategic policy to vaccinate an entire population against a virus that causes chronic infections and death in less than 0.5% of the nation's select at risk populations that **do not** include newborns, infants and children.

Health experts put forth the idea that hepatitis B vaccination would protect newborns, infants and children from contracting the infection when they grow up. However, studies

show that antibody protection from hepatitis B vaccination only lasts 10-12 years (a set up for health officials to recommend booster shots). Therefore, by the time newborns, infants and children are old enough to *possibly* become members of high-risk populations, their protection and their antibodies, if they have developed any, are gone. The antibodies for the first 10-12 years of life serve no purpose.

It is not clear how many parents are aware of these contradictions; nevertheless, the message from health officials is clear. The vaccine is presumed safe for children anyway so there is no harm in giving it to them along with the rest of their recommended childhood vaccines.

Manufacturer Reported Ingredients

The Energix-B hepatitis B vaccine, marketed by SmithKlineBeecham, is manufactured using a culture of genetically engineered Saccharomyces cerevisiae (yeast) cells which carry the gene for the surface antigen of the hepatitis B virus. The surface antigen is purified by several physicochemical steps (which are not clarified in the package insert) and formulated as a suspension of the antigen adsorbed on aluminum hydroxide (aluminum is used as an adjuvant and is a suspected cardiovascular, blood, nerve and respiratory poison).

Each neonatal and infant injection of 0.5ml contains 10 micrograms of hepatitis B surface antigen adsorbed on 0.25mg aluminum as aluminum hydroxide, 1:20,000 thimerosal concentration as a preservative, sodium chloride, phosphate buffers and, no more than 5% yeast protein.[1] Ethyl mercury, the main component of thimerosal, is a recognized developmental and nerve poison and a suspected skin and sense organ poison.

Manufacturer Reported Safety

Safety studies for the hepatitis B vaccine were limited to clinical observations of test subjects for up to 4 days after administration of the vaccine. Frequency of adverse experiences tended to decrease with successive doses of Energix-B. Of those adults and children studied, soreness and fatigue were the two most common adverse reactions. Fever, headache and dizziness were reported with a 1% to 10% incidence, the latter two predominantly reported in adults. Other adverse reactions reported with < 1% incidence were pain, pruritis, ecchymosis, sweating, malaise, chills, weakness, flushing, tingling, hypotension, influenza-like symptoms, upper respiratory illnesses, nausea, anorexia, abdominal pain, vomiting, constipation, diarrhea, lymphadenopathy, pain/stiffness in injection site, arthralgia, myalgia, back pain, rash, petechiae, somnolence, insomnia, irritability and agitation.[1]

Additional adverse reactions have been reported with the use of hepatitis B vaccine. These include anaphylaxis, Stevens-Johnson syndrome, angioedema, arthritis, serum-sickness

like illness within days to weeks of vaccination, tachycardia, palpitations, bronchospasm including asthma like symptoms, abnormal liver function tests, dyspepsia, migraines, syncope, paresis, neuropathy, Guillain-Barre syndrome, Bell's palsy, transverse myelitis, optic neuritis, multiple sclerosis, seizures, thrombocytopenia, eczema, purpura, herpes zoster, erythema nodosum, alopecia, conjunctivitis, keratitis, visual disturbances, vertigo, tinnitus and headache.[1]

What is Missing?

Important information is missing from the package insert. Most of these side effects are reported in adults. The occurrence of these medical problems in neonates, infants and children after hepatitis B vaccination is unknown. It is rare for neonates, infants and children to experience these conditions when not receiving the hepatitis B vaccine. It would seem that neonates, infants and children are more likely to experience any one of these medical problems from receiving the hepatitis B vaccine than if they do not receive the vaccine at all.

More important information is missing. There are **no** long term safety studies evaluating the health of infants and children beyond 4 days after the administration of the hepatitis B vaccine. Do the vaccine ingredients accumulate in neonates, infants and children? Do the ingredients interact? Do the vaccine ingredients adversely affect the developing immune, nervous and digestive systems? If so, do these adverse effects persist? Can the adverse effects be seen months or even years down the road? The answers to these questions remain unknown.

Do the medical experts associate the occurrence of serious adverse events after receiving the hepatitis B vaccine as a consequence of the vaccine? Adverse events such as fever, vomiting, diarrhea, seizures, bronchiolitis, pneumonia, asthma, apnea, vitreous hemorrhage or even death are often viewed by medical experts as symptoms and illnesses that would normally occur in a population of neonates, infants and children and are therefore judged not to be side effects from the vaccine.[4]

For neonates, infants and children, we cannot guarantee their development of antibodies against the hepatitis B antigen. Neither can we guarantee they will be protected from infection after they receive the hepatitis B vaccine. And we cannot guarantee their safety from short or long term adverse outcomes after we inject them with the hepatitis B vaccine ingredients.

There is no safety data addressing the effects of each vaccine ingredient (genetically engineered hepatitis B antigen, yeast cells, aluminum, ethyl mercury, other physicochemical steps and phosphate buffers) when injected into a newborn, infant, small child or adult. Specifically, the vaccine manufacturers have never looked at the effects of ethyl mercury or

aluminum, known neurotoxins, on maturing nerve tissue, while other scientists have published a plethora of articles on the dangers of these ingredients.

Do any of these vaccine ingredients enhance the activity of any of the other ingredients once injected into the body? Is the toxicity of ethyl mercury augmented by the presence of aluminum? Does the presence of mercury and aluminum, together with a mild viral or yeast infection, have any damaging effects on the immune systems of the people in whom these ingredients are injected? Is there any predictability as to whether an adverse outcome is more likely in boys vs. girls?

There are **no** conventionally supported scientific data that address how each vaccine ingredient bio-chemically interacts with one another inside the body of a newborn, infant, child or adult when they are completely healthy or when they are suffering from a mild infection or a chronic condition. Medical experts recommend vaccinating children without fevers who have mild infections or chronic conditions. These recommendations are based purely on medical opinion and not on conclusions from valid scientific studies.

Medical experts argue that the hepatitis B vaccine and other vaccines contain only minute amounts of heavy metals, adjuvants, solvents and other foreign materials that are well below the safety levels recommended by the EPA and well screened for adventitious agents. However, others argue that these safety levels are based on the study of ingested materials, not injected materials. Other studies show that even the tiniest amounts of these chemicals and agents are toxic to nerve, immune and developing cells. There is further evidence that vaccines **add** to the heavy metals and toxins that neonates, infants and children are already exposed to from breast milk, formula, food, water, air, soil, parks, clothes, toys and playgrounds.

Heavy metal detoxification capabilities of different immature immune systems of newborns and infants have not been evaluated either. Mercury is primarily removed through the biliary system (liver, gall bladder) and aluminum is removed by the renal (kidney) system. The inability of a newborn or infant to eliminate these toxicants greatly increases the potential for damage to their systems.[5] The biliary systems and kidney function of newborns and infants are immature. The maturation of these systems is individual to each infant and depends greatly on genetic factors, nutrition, other environmental exposures, the number and types of infectious illnesses, history of medications and stress. All of these factors influence how each newborn and infant handles the ingredients at the time of vaccination(s).

It remains unclear what level of each vaccine ingredient is potentially dangerous to neonates, infants and children. However, recent experiments by J. Curtis Pendergrass, PhD[5] show that human brain tissue exposed to mercury dramatically reduces the viability of tubulin, a major brain protein, but has little effect on actin, another major brain protein.

Tubulin and actin are vital for the growth and maintenance of neurons, especially in the central nervous system. Thimerosal, an organic mercury compound containing ethyl mercury, on the other hand, reduces the viability of tubulin **and** abolishes the viability of actin.

Boyd E. Haley, PhD, along with Mark Lovell, PhD, performed further experiments by adding pure thimerosal, thimerosal containing vaccines and thimerosal free vaccines to neurons grown in culture for 24 hours. Their results showed that thimerosal containing vaccines were as toxic to the neurons as they were to the brain tissues in Dr. Pendergrass' experiments and were much more toxic to the neurons than thimerosal free vaccines. The levels at which pure thimerosal was toxic to the neurons were 10,000 times **less than** the concentrations (of thimerosal) found in most vaccines.[7]

Aluminum, used as an adjuvant in vaccines, is also toxic to neurons, although to a lesser degree than thimerosal.[5] Further experiments by Dr. Haley showed that the presence of aluminum dramatically increases the rate of neuronal damage and death caused by the presence of low levels of thimerosal. Further experiments by Dr. Lovell showed that neurons pre-incubated with estrogen were substantially protected from neuronal death in the presence of thimerosal. However, the addition of testosterone caused a very large increase in neuronal death induced by thimerosal.[8] Parents, scientists and health care providers have witnessed greater numbers of boys with neurological damage, e.g., autism, learning disability, pervasive developmental disorder, behavioral and attention problems than girls.

The synergistic action of other heavy metals with mercury, such as lead and cadmium, is well documented in the literature.[9] Additionally, the presence of certain antibiotics, such as tetracycline and ampicillin, enhances the toxicity of thimerosal on neuronal death.[5] Many mothers are given ampicillin in labor to treat Group B strep infections in their vaginal and cervical tissues. Soon after birth their newborns are vaccinated with a thimerosal and aluminum containing hepatitis B vaccine. Older infants may be prescribed amoxicillin, an ampicillin derivative, near or at the same time they are given hepatitis B and other mercury and aluminum containing vaccines. Unbeknownst to the mothers, they may have other heavy metals circulating in their bodies that enhance the toxicity of the mercury and aluminum in the hepatitis B vaccine on their newborns' central nervous systems.

In another experiment, mice with myocarditis from a coxsackie B3 viral infection were injected with methyl mercury (commonly found in the environment; a similar compound to the ethyl mercury found in thimerosal). A control group of mice with coxsackie B3 myocarditis was not exposed to mercury. The researchers were attempting to see if the presence of mercury affected the activity of the viral infection in the mice. The results showed that there was greater immune damage and chronicity of coxsackie B3 myocarditis

in the mercury exposed mice than in the control group.[10] In the presence of mercury, the coxsackie B3 viral infection was worsened. For infants and children, one or more of the hepatitis B doses are combined with viral and bacterial containing vaccines—DTaP, HIB, IPV and PCV7. In addition, many infants and children with simple, mild viral infections are given their vaccinations so long as they present to their physician without a fever.

Possible Dangers

If the above animal study is reproducible and can be extrapolated to humans, the presence of ethyl mercury and aluminum in the hepatitis B vaccine may affect the activity of the yeast cells that are present in the vaccine when injected into children. Mercury and aluminum from the vaccine may also affect the activity of yeast cells that already reside in newborns, infants, children and adults who receive the vaccine. Similarly, mercury and aluminum may affect the activity of bacteria or viruses that live in humans and are components of other vaccines. The yeast cells could conceivably become pathogenic in the presence of these metals leading to nervous, vascular and immune system damage, and possibly even death.

All newborns have immature immune systems. Regardless of whether they receive the hepatitis B vaccine or not, many newborns and infants have frequent yeast infections in the form of oral thrush and diaper rash. Each 0.5ml of hepatitis B vaccine may contain up to 5% of yeast cells. There are no experiments to test whether the injection of even the smallest amount of yeast cells, along with mercury and aluminum, can injure susceptible newborns and infants who have immature immune systems and an overgrowth of yeast. There are no experiments to test whether the injection of hepatitis B vaccine, alongside other childhood vaccines, can injure their immature immune systems in the presence of an overgrowth of yeast and/or other bacteria or viruses either.

In a study published in Lancet in 2002, the investigators measured the mercury concentrations in 40 full-term infants 6 months of age or younger who had received thimerosal containing DTaP, hepatitis B and HIB vaccines. They concluded that the measured mercury levels in these infants did not exceed the recommended guidelines and were, therefore, not likely to be harmful to infants.[11] However, the conclusions from the study are based on poor scientific methodology. And the guidelines to determine safe levels of mercury are suspect.

The Environmental Protection Agency (EPA) established safe levels of injected ethyl mercury (which is absurd as even the smallest exposures are known to be toxic) in vaccines for infants based on data obtained from fetuses who were neurologically damaged when pregnant Iraqi women ingested large quantities of methyl mercury-treated seed grain.[12] However, there are several problems with the model used by the EPA to establish these

levels which does not work.

The developing central nervous system of a fetus is more susceptible to environmental and toxic insults than that of a newborn.[13] As such, the central nervous systems of fetuses and newborns would not be equally susceptible to the harmful effects of mercury. Therefore, the fetus is not a fair model for establishing safe mercury levels for newborns even if exposures to the **same** mercury compound are compared.

The study is further invalidated because the EPA compared the exposure of **two** different types of mercury (methyl mercury and ethyl mercury) in two non-identical groups (fetuses and newborns).

Theoretically, a portion of the ingested methyl mercury should have been metabolized and eliminated by the detoxification pathways in the intestines and the livers of the Iraqi women before the mercury reached the blood stream of their fetuses. The amount of fetal methyl mercury exposure would have been lower than the amount ingested by their mothers. Therefore, the extent of their neurological damage can be based only on the levels of methyl mercury that were absorbed into the women's bloodstreams and not on the actual levels of ingested methyl mercury. The actual levels of fetal methyl mercury exposure cannot be calculated and are unknown. The methyl mercury levels used by the EPA to establish safety data are unreliable, at best.

With vaccines, infants and children are exposed to the **exact** amount of mercury that is present in the syringe. Mercury is injected directly into their lymph and vascular systems without the prior benefit of possible removal of some of this toxin through intestinal and liver detoxification pathways as if it were ingested.

Ethyl mercury is excreted from the body much more quickly than methyl mercury. Methyl mercury is expected to be more toxic to the central nervous system than ethyl mercury although other reports conclude that the more rapid clearance of ethyl mercury augments mercury toxicity in newborns, infants and children.[5] Either way, both mercury compounds are toxic and their toxicities are different.

To summarize, fetuses and newborns have distinct susceptibilities to the harmful effects of mercury **and** the toxicities and pharmacokinetics of methyl mercury and ethyl mercury are different. There are no data to explain how newborns and infants metabolize ingested mercury or injected mercury. There are no data to indicate whether newborns and infants metabolize injected mercury in a different way or at a different rate than ingested mercury. The investigators used a poor scientific model, with too many variables, to establish a credible safe level of mercury for newborns. Mercury is toxic, in whatever compound, in whatever exposure, ingestion or injection.

The EPA was essentially comparing apples and oranges to construct an imaginary safe number that omitted real differences between fetal and newborn susceptibilities to mercury. They omitted real scientific differences in methyl mercury and ethyl mercury metabolism and they failed to account for individual differences in detoxification and excretion capabilities from one infant to another. Therefore, the model of the fetus exposed to methyl mercury from maternal oral ingestion cannot be used to standardize safe levels of injected ethyl mercury in infants. The accepted safety level of mercury exposure for infants established by the EPA is meaningless.

In the Lancet study, mercury concentrations were measured in 40 full-term infants 6 months of age or younger who had received thimerosal containing DTaP, hepatitis B and HIB vaccines. There are three reasons the conclusions from this study are worthless. First, the measured mercury levels in the 40 infants, while below the recommended guidelines, might still cause damage to the newborns and infants. Second, measurements of mercury in the blood do not take into account the pharmacokinetics of mercury. Mercury can bind to the neurons of the central nervous system and remain in the intracellular, extracellular and interstitial spaces of newborns and children especially if detoxification pathways are inadequate and/or toxic loads are too great. The mercury blood level is not an accurate measurement of the newborns' total body content of mercury. Third, the study conclusions are fatally reductionistic. The real concern about the safety of mercury lies not just in how safe or harmful mercury alone may be to newborns and children, but how toxic it becomes when exposed to the other ingredients in vaccines, in humans and in the environment.

Heavy Metal Toxicity in Children

There are many physicians and practitioners who see children in their practice with mercury and heavy metal toxicity. They work primarily to chelate the metals from the children's bodies and replenish their often found nutritional and immunological deficiencies. Coincidentally, many of these children have neurological problems and neurodevelopmental delays. Mercury levels in hair, urine, blood and stool samples at the beginning of chelation are frequently low but commonly increase as chelation therapy proceeds. The practitioners find that the mercury binds tightly to their cell membranes, extracellular, intracellular and interstitial spaces and is usually the last and most difficult metal to be excreted from their bodies. Blood levels measured in the Lancet study are cannot be reflective of the total mercury burden in these children.

The Lancet study is used by experts in the conventional medical world as a way to support the pharmaceutical industry and substantiate the belief that thimerosal containing vaccines do not subject newborns and infants to harmful levels of mercury. However, the science disproves this assertion. Why haven't scientists and government agencies conducted the

proper studies to bear out the truth? The health and well being of children is at the heart of this matter.

Aluminum is the predominant adjuvant used in vaccine manufacturing in the United States. Its role in vaccines is meant to enhance antigen uptake by antigen presenting cells (e.g., dentritic cells), and to activate antigen-presenting cells. It may also induce production of cytokines and complement helping to initiate the cellular immune response. Antigen presenting cells are the immune cells that are supposed to begin the sequence of immune responses that leads to the eventual production of antibodies. ***The importance of each of these mechanisms in enhancing antigen-specific immune responses remains unclear.***[13]

Aluminum is extremely abundant in the environment. It can be found in the air, food, water, breast milk, formula, drugs, the home and vaccines. Large quantities of aluminum can cause serious neurological problems. As a result, the Agency for Toxic Substances Disease Registry (ATSDR) established guidelines for maximum safety levels for aluminum exposure. Lab mice were exposed to varying oral quantities of aluminum lactate and statistical analyses were done to conclude a minimum risk level for human exposure to aluminum. Using measured quantities of aluminum infants are exposed to in breast milk, formula and vaccines, health experts concluded that infants are exposed to quantities far below the ATSDR guidelines.[13]

The scientific problems with the experiments and conclusions in establishing safe levels of aluminum exposure for infants is similar to the problems mentioned above for figuring out safe levels of mercury. First, the investigators were using **oral** exposures of aluminum to conclude safety levels of **injected** aluminum. There are no data to conclude that oral aluminum is metabolized by infants in the same way as injected aluminum. Second, in order to find legitimacy in the investigators' conclusions, mice and infants would have to have the same set of adverse reactions to aluminum and at different levels of aluminum exposure and they do not. Third, aluminum lactate is not the same preparation of aluminum that is used in vaccines so metabolisms and eliminations may be different. Fourth, aluminum is one of many ingredients in vaccines. Toxicity may have more to do with the interaction with these other ingredients rather than from its singular toxic effects. Ultimately, the suggestion of a "safe" level of aluminum is meaningless.

We have **no** conventionally supported scientific data that address the effects of the hepatitis B vaccine, or any other vaccine, on the immature immune system of an infant or small child. Vaccine ingredients are injected past the major portion of a child's cellular immune defense system in the skin, exposing newborns, infants and children to ingredients that are never naturally introduced to the immune system in this way.

The Immune System of Infants and Children

Newborns begin life with immature immune systems which consist of cellular and humoral branches. They rely heavily on maternal antibodies to help protect them from the outside world during the first 12-18 months of their lives. Initially, their own humoral immune systems produce antibodies in response to foreign antigens. As they grow and the maternal antibodies diminish, their cellular immune systems mature from exposures to materials they ingest, inhale, absorb through their skin and receive as stimuli through their senses. The immune systems that reside in the intestines, the lining of the respiratory tract, the skin and the nervous system make up the major portion of their cellular immune systems.

A mature immune system in healthy children functions with balanced, equal responses from the cellular and humoral immune systems. Chronic conditions such as asthma, eczema, allergies, ADD/ADHD, autism, inflammatory bowel disease, lupus, diabetes, rheumatoid arthritis, multiple sclerosis and others have dysfunctional immune systems where cellular and humoral system responses are pathogenic and out of balance.

Immune cells in the cellular immune system are the first in line to begin the immune response when infants and children are challenged by exposures to viruses, fungi, parasites, cancer cells, protozoa, food, airborne particles, skin exposures and sensory stimuli.[13] These cells initiate the immune response by producing inflammatory chemicals, called cytokines, which begin the inflammatory process. The onset of the inflammatory process may come in the form of fever, body aches, malaise, vomiting, diarrhea, rash, sore throat, congestion, sneezing, mucus production, ear pain, irritability and others. In other instances, children have no symptoms or illnesses and are not cognizant of the inflammatory processes that are occurring in their bodies, although the immune system is still maturing.

The cytokines produced by the cells of the cellular immune system initiate the inflammatory process and, when unimpeded by suppressant over the counter drugs and prescription medications, stimulate the production of anti-inflammatory cytokines by the humoral immune system. Ultimately, the acute illness resolves with the production of antibodies. This process usually lasts from 3 to 10 days in infants and children. In most cases, this inflammatory and anti-inflammatory cascade goes smoothly.

The frequent stimulation of the cellular immune system from acute exposures to foreign antigens, followed by the initiation of a humoral immune response and the production of antibodies along with the resolution of an illness is the way in which the immune systems of infants and children mature throughout childhood. However, injected materials are presented to their immune systems in a manner that disrespects the genetic program of how their immune systems are biologically designed to mature.

We have **no** conventionally recognized data that demonstrate how vaccines and their ingredients affect the growth and development of a balanced cellular and humoral immune

system either favorably or negatively. Different newborns, depending on their genetic history and random environmental exposures, will have varying cellular and humoral immune system responses to each vaccine and its ingredients.

Many of the vaccine ingredients are injected into infants and adults with the *hope* and *speculation* that there would be an identical response by their cellular and humoral immune systems as if they were naturally contracting the disease in the vaccine. ***Yet, to repeat, the exact mechanism by which the vaccine ingredients accomplish this remains unclear.***[13] This theory has not been tested by health officials on a practical level with lab examinations of children or adult immune systems. Each cellular and humoral immune response to a vaccine will vary depending on the chemical and physical state of the antigens, the adjuvant, the mode of administration, the catabolic rate of the antigens, the genetic characteristics of the child, host factors (e.g., age, nutrition, gender, pregnancy status, stress, concurrent infections), and the manner in which the antigen is presented. [14, 15]

We have a one size fits all mass hepatitis B vaccination program that runs contrary to the multiple variables that dictate each child's response to the vaccine. The results expected by administering the vaccine are based more on chance outcomes than on a known set of outcomes from proper scientific inquiries.

In the case of hepatitis B vaccine, with so many factors determining the nature of the immune response to the vaccine, it is nearly impossible to predict whether a child's immature immune system will stimulate the proper cellular immune response followed by the proper humoral response with the end result being the presence of an antibody to hepatitis B and adequate protection from an acquired infection. It is also difficult to predict whether children are susceptible to developing imbalances or damages to their cellular and humoral immune systems when exposed at such an early age to hepatitis B vaccine and its ingredients. Nevertheless, every child is vaccinated equally.

Children who get the hepatitis B vaccine have been evaluated only to see whether they respond properly to the vaccine, i.e., the development of hepatitis B antibodies. They have not been evaluated to see if their immune systems respond by producing abnormal levels or dysfunctions of cytokines and antibodies and whether these responses persist, taper off, get worse or improve with subsequent vaccinations and exposures to other environmental conditions. If their immune systems do produce abnormal levels or dysfunctions of cytokines and antibodies, what role do they play, if any, in the state of health or illness as the children grow and mature?

What remains clear is that children and adults with chronic conditions have abnormalities of their cellular and humoral immune systems—abnormal production and dysfunction of cytokines and antibodies. The effects of the hepatitis B vaccine and the effects of single and combination vaccines have not been studied on the cellular and humoral immune system

responses of different children with different backgrounds either acutely or within months to years following vaccination. Therefore, we cannot say for sure, one way or another, whether vaccines play a role in temporarily or permanently changing the balance of the cellular and humoral immune system responses in newborns, infants and children

Newborns and infants may develop an antibody to hepatitis B antigen from the vaccine. What remains unclear is what additional cellular immune system cytokines and humoral immune system antibodies and cytokines are produced in their bodies in response to the presence of yeast, mercury, aluminum, phosphates and physicochemical steps, especially since each child has a different genetic background. Ultimately, we could be altering and threatening the proper maturity of their immune systems from the very beginning of their lives with the introduction of the hepatitis B vaccine because of a lack of scientific study, inquiry and data.

We are just beginning to understand which branch of the immune system, cellular and/or humoral, are/is dysfunctional in each of the many chronic conditions we see in pediatrics and adult medicine. Without knowledge of whether vaccines affect the delicate balance of cellular and humoral immune system maturity, it is difficult to say whether or not they contribute to the development of any of these chronic conditions. There is much science that needs to be performed before we can say that the hepatitis B vaccine, or any other vaccine, is safe.

Hepatitis B Vaccine and SIDS

In December 1999, an article appeared in the *Archives of Pediatric and Adolescent Medicine* entitled **Neonatal Deaths after Hepatitis B Vaccine: The Vaccine Adverse Event Reporting System (VAERS), 1991-1998**.[16] VAERS is a passive surveillance system monitoring postmarketing vaccine safety. The authors write "despite limitations inherent in passive reporting systems such as biased reporting, incomplete reporting, and underreporting; lack of consistent diagnostic criteria; lack of a comparison group; and lack of data as to the number of vaccine doses administered, VAERS has proved a useful tool in identifying and evaluating vaccine-related events…".

Between 1991 and 1998, there were 1771 reports made to the VAERS system enumerating adverse events related to hepatitis B vaccination of neonates (infants 0-28 days). Eighteen deaths were reported. The list of adverse events and the persons making the reports are not made available. The article reviews the 18 neonates whose deaths were reported as possible adverse events related to hepatitis B vaccination which I will review as well. The authors address whether there is a relationship between hepatitis B vaccination of neonates and the development of adverse events. I will also address this relationship.

Seventeen of the eighteen neonates had autopsies performed. Twelve of the eighteen

neonates were reported at autopsy to die of SIDS (Sudden Infant Death Syndrome). Of those children reported to have died of SIDS, 9 had medical histories and autopsies reported in the article and 3 did not. The medical histories and salient autopsy findings of the 9 SIDS cases are as follows:

Case Number	Medical History	Autopsy Findings
Case 3/13/15	Found with blood @ nose	Moderate fatty changes of liver
Case 4/19/20	Found with blood @ nose	Recent focus alveolar hemorrhage; Liver: moderate microvascular changes; Thymus: moderate eosinophil deposition
Case 7/14/24	Found with blood in mouth and nose; co-slept with mother in bed	
Case 10/4/24		Occasional intra-alveolar hemorrhage, small bowel autolytic changes
Case 13/13/22	34 weeks; co-slept with mom, found on back on floor beside couch	Scattered acute intra-alveolar hemorrhage
Case 14/16/20	2 ½ hr post-vaccine vomiting, fever for 4 hrs treated with Tylenol, found limp, not breathing next am	Massive myocardial infection; CNS: diffuse ischemic encephalopathy; Lungs: small foci of hemorrhage
Case 15/1/16	Co-slept with mom in bed, found on left side	Liver: multifocal areas of extramedullary hematopoiesis, hepatic infarct with hyperemic border
Case 16/1/19	35 weeks, co-slept with mom in bed, found on left side, mom heavy smoker	Diffuse intra-alveolar acute hemorrhage; Liver, spleen, cerebellum: eosinophilia
Case 17/20/21		Liver: extramedulary hematopoiesis

The following is a brief summary of the remaining 6 infants in the report who died of causes other than SIDS. One neonate was reported to have bronchopneumonia on autopsy with a medical history of having died suddenly after vaccination, although an organism was not isolated. Another newborn, vaccinated soon after birth, had pneumonitis from

aspiration of amniotic sac contents (possibly infected) with a persistent fetal circulation. (Almost all newborns have persistent fetal circulation for a short time after birth). A third neonate was seen in the emergency room with fever, vomiting, bloody diarrhea and a swollen leg 2 hours after hepatitis B vaccination and died in the hospital of Enterobacter cloacae (E. cloacae) sepsis 16 days later.

Another neonate, who had a non-life threatening congenital heart disease, died in the car on the way home from receiving the hepatitis B vaccine at a well-baby visit. Another child, who co-slept with the parents and a one year old sibling on a sofa bed, died of accidental suffocation. The eighteenth neonate who died was fed on the same day of the hepatitis B vaccine, choked, stopped breathing and went limp. Parents supposedly "shook" the child, CPR was done and the baby was removed from life support 2 days later. Since this neonates' cerebrospinal fluid from the spinal tap was bloody, the child was given a provisional diagnosis of a subarachnoid hemorrhage, thought to be secondary to shaken baby syndrome. An autopsy was never performed on the neonate.

The occurrence of SIDS is rare during the first month of life. Only an estimated 1% of SIDS cases occur in neonates. The authors conclude that 1) the incidence of the 12 cases of SIDS in this report is no greater than what would be expected in the general population of neonates. 2) Coincidental SIDS deaths are expected following any infant vaccination given the extent of vaccine coverage and the incidence of SIDS. 3) The hepatitis B vaccination is not causing a clear increase in unexplained neonatal or infant deaths. 4) Several of the deaths were due to overwhelming sepsis, congenital heart disease and, where no other concrete reason for cause of death was found, an insinuation that co-sleeping with the family caused the babies to die of SIDS.

There are many weaknesses in the authors' conclusions.

The incidence of the 12 cases of SIDS in this report is no greater than what would be an expected incidence of SIDS in the general population of neonates.

First, based on the information provided, I do not believe we can say with certainty that the infants died of SIDS and not from other causes. One of the other causes could have been as a result of a reaction to the hepatitis B vaccine. Infants who die of SIDS do not have massive myocardial infarctions, liver hemorrhages, moderate fatty changes of the liver, marked eosinophilia and diffuse ischemic encephalopathy on autopsy. (See chart) Tiny hemorrhages on the surface of the heart, in the lungs and in the thymus are one of the two most common autopsy findings. Can they be seen as an adverse response to the hepatitis B vaccine? The other most common autopsy finding is an increased number of star-shaped cells in the brainstem, referred to as brainstem gliosis. Brainstem gliosis was not reported in any of the autopsies of the 12 children diagnosed with SIDS in the VAERS study. A very significant, common finding to help substantiate a SIDS death was missing.

SIDS is suspected when a previously healthy infant is found dead in bed, often after a normal feeding. SIDS is a diagnosis of exclusion when no other cause of death can be found or considered. In the majority of cases, there is no identifiable sign of distress. Blood is not found in the mouth or nose and vomiting and fever are not part of the histories of SIDS victims as reported in several of the cases listed above. The autopsy findings in SIDS infants are usually subtle and have not been of sufficient specificity and sensitivity to explain the disease.[16]

Second, let us assume that the infants died of SIDS and from no other cause. The authors make a distinction between the VAERS group and the general population of neonates and conclude that the incidence rate of SIDS cases in neonates in the VAERS group is the same as the incidence rate of SIDS in the general population of neonates. Both groups, however, are one in the same. The general population of neonates receives the hepatitis B vaccine as well. They are comparing the VAERS group to itself. If we assume, as the authors do, that SIDS was a coincidental finding in the infants and not related to the hepatitis B vaccination, we would have to look at the incidence of SIDS in a control group that did not receive the hepatitis B vaccination. This type of study would not only give us information about the rates of SIDS in hepatitis B vaccinated vs. non-hepatitis B vaccinated populations of neonates, but it would also give us information about whether the deaths of these infants may not be coincidental after all.

Third, the authors make the assumption that the VAERS data include **all** infants who "coincidentally" died after hepatitis B vaccination. We know that vaccine adverse events are underreported to the VAERS system. We do not know the full extent of the number of neonatal or infant deaths after hepatitis B vaccination because many parents and medical providers do not make the connection between the death of their children and the timely administration of the hepatitis B vaccine. If the medical community believed and publicly acknowledged the possibility of this relationship, based on the allowed publication of proper scientific studies, more parents and providers would make the connection. And more people might stand up and refuse this senseless policy of universal vaccination.

Coincidental SIDS deaths are expected following any infant vaccination given the extent of vaccine coverage and the incidence of SIDS.

This statement reflects the author's opinion and strong bias that deaths after hepatitis B or any other vaccination is simply coincidental. The editor of the article, Catherine D. DeAngelis, MD writes in her editor's note, "this report should help allay the fears of anti-vaccine groups; it should, but will it?" Perhaps, it would if the study were scientifically solid and non-biased. The fact is there are no legitimate scientific studies to show that SIDS deaths following hepatitis B or any infant vaccination **is not** coincidental and therefore not due to SIDS. Since the 1991 Advisory Committee on Immunization Practices (ACIP) recommended universal hepatitis B vaccination of infants, the authors state there has been

no evidence of either an increased trend in the overall number of neonatal deaths or in neonatal deaths after hepatitis B vaccination as reported to VAERS. To reiterate, reports to VAERS are underreported and parents and medical providers alike are not as likely to make the association.

As long as the authors, and those before and after them who analyze data from VAERS and the CDC, maintain the position that neonatal death from hepatitis B vaccination can only be coincidental, there will never be a report of a study or support for a study that shows a causal relationship between neonatal death and hepatitis B vaccination. As long as authors believe that infants in this study, as well as in subsequent studies, die from SIDS and not from any other cause, there will never be a report showing that this vaccine, or any other vaccine, can kill, or permanently damage, a subset of infants.

The public continues to witness the implementation of a policy that has poor medical reasoning and no scientific backing to support its safety. The data may show no increase in neonatal deaths after hepatitis B vaccination. Yet, just one single death after hepatitis B vaccination is more than the number of deaths we would see in newborns born to hepatitis B negative mothers who would otherwise not be at risk of dying from hepatitis B infections. Unless, of course, they were physically abused, stuck with multiple dirty needles and transfused with poorly screened blood products.

I have seen 2 neonatal deaths after hepatitis B vaccination. The first was a healthy 5 week old girl who died "of SIDS" within 14 hours of her first hepatitis B vaccine. The autopsy report showed diffuse brain edema (which is **not** a SIDS related autopsy finding). Nevertheless, the authorities believed the death could not have been related to the vaccine, so it had to be SIDS. This was no comfort to a mom and dad who had no reason previously to question what the doctor was doing for their daughter by giving her the hepatitis B vaccine.

The other child was a 3 week old boy who was found to have significant frontal cerebral, retinal and subarachnoid hemorrhages within 9 days of his second hepatitis B vaccination. The parents were accused of shaken baby syndrome (SBS). Severe acute brain trauma cannot be produced in the infant by shaking alone. The mechanism of injury should more appropriately be referred to as shaking impact syndrome, requiring some form of blunt trauma to the head in combination with the shaking.[17]

While cerebral, retinal and subarachnoid/subdural hemorrhages are the sine qua non findings in SBS,[17, 18] this child had no external evidence of trauma on physical exam. All bone and skull X-rays were normal. No 'injuries' were found other than the findings in his brain. The autopsy report also showed no evidence of trauma. However, babies who are shaken have autopsies that show abnormal pathologies of their neck areas with findings of diffuse axonal injury in the cervical region.[19] There was no pathology on the neck autopsy

of this child. No hard evidence of impact and no hard evidence of shaking.

Cerebral hemorrhages in shaken babies most often appear as unilateral or bilateral high-density collections of fresh blood that are thin but extensive with a particular propensity for the spaces between the right and left brain hemispheres, especially in the posterior brain surrounding the brainstem.[19] Instead, this child had full, thick frontal lobe cerebral hemorrhages with large areas of necrosis. There was no report of hemorrhage or necrosis in the spaces between the brain hemispheres or in the posterior brain. However, previous reports of acute necrotizing encephalopathy (hemorrhaging and necrosis of brain tissue leading to coma and possibly death) related to vaccination have been reported.[20]

In order for this child to have had such significant findings in the frontal lobes as a result of trauma, he would have had to have sustained either an open head injury to the front of his head, or a severe closed head injury to the back of his head, transmitting the force to the frontal lobes of his brain. This child's physical exam, X-rays and autopsy reports indicated no evidence of external or internal trauma. There was no mechanism of injury to explain the frontal lobe findings. However, hemorrhages have been reported from an immune complex vasculitis as an adverse reaction to the hepatitis B vaccine.[21, 22]

It should also be mentioned that the infant was anemic on oral iron supplements, mostly formula fed and on treatment for oral candidiasis and a diaper rash at the time of his second hepatitis B vaccination. He did not feed well and had increased lethargy and sleepiness in the week following his hepatitis B vaccine.

So many pieces of the case did not fit the diagnosis of Shaken Baby Syndrome. The medical experts admitted there were inconsistencies in the history and the findings on physical exam, X-rays and autopsy. They could not explain how the child could have sustained frontal lobe hemorrhages without any physical or X-ray evidence of trauma. They could not explain how a child with such severe findings could have had a normal cervical region on autopsy. They knew the placement of the brain hemorrhages was different that what was normally expected. They could not explain why the child's cerebral hemorrhages with severe necrosis of the brain tissue were not thin but thick and massive in the frontal portion of the brain instead of near the brainstem.

Nevertheless, they repeated the mantra: subarachnoid, cerebral and retinal hemorrhages must equal shaken baby syndrome, end of story, parents are guilty. Why? Because it was impossible for them to fathom the relationship between these clinical findings and the possible bio-chemical, vascular and immunological consequences of a hepatitis B vaccine gone bad in a susceptible child. The authorities say it is not possible, and the experts agree.

One neonate in the VAERS study had a bloody spinal tap believed to be secondary to a subarachnoid hemorrhage. The experts attributed these findings to a probable case of

shaken baby syndrome. They were able to get away with making these two assumptions, despite not having an autopsy report. In the case of a bloody spinal tap, an autopsy is the only way to confirm the diagnosis of subarachnoid hemorrhage and shaken baby syndrome. The authors would not and could not hypothesize the relationship of a subarachnoid hemorrhage as an adverse effect of the hepatitis B vaccine, so they stretched and said it had to be shaken baby syndrome.

The hepatitis B vaccination is not causing a clear increase in unexplained neonatal or infant deaths.

Does this mean that the hepatitis B vaccine **is** causing neonatal or infant deaths, just not at a clear increasing rate? If it is not causing a clear increase, then is it causing an unclear increase in neonatal or infant deaths? What is an unclear increase? If hepatitis B vaccination is causing **any** neonatal or infant deaths, then they would be explained deaths, not unexplained deaths.

The reality is that hepatitis B vaccination **does** cause some explained neonatal and infant deaths; neonates and infants who do not have to die. The utilitarian theory states that it is morally right to sacrifice the few for the good of the many. The irony here is that neonates and infants are not susceptible to contracting hepatitis B infections, so how is this vaccine for the good of the many neonates and infants?

Several of the deaths were due to overwhelming sepsis, congenital heart disease and, where no other concrete reason for cause of death was found, an insinuation that co-sleeping with the family led to the diagnosis of SIDS.

In the VAERS group, a previously healthy infant went to the doctor for a hepatitis B vaccine and within 2 hours developed bloody diarrhea, fever, vomiting and swelling of the injected leg. The emergency staff treated the child supportively with Tylenol and fluids and sent the family home. Five days later, the child was hospitalized and placed on antibiotics for high fever and rapid breathing. The child's presentation and hospital course must have been serious enough for the intensive care staff to place central lines in his/her body for easier access to the veins and arteries. The child died 11 days later of Enterobacter cloacae (E. cloacae) sepsis.

Enterobacter cloacae are bacteria that naturally inhabit the intestines. There have been many reports by the CDC and in the medical literature describing contamination of central lines and central line sepsis from E. cloacae in ICU settings, usually in immunocompromised patients. The E. cloacae sepsis is a red herring and does not help explain why this child initially got sick. This child was immunocompromised on strong antibiotics with a central line in place and became infected and died from a contaminated line. While the sepsis may have been the cause of death, it was not the reason for his illness in the first place. The authors do not address his clinical course immediately after receiving the hepatitis B vaccine

or how he got sick enough to require intensive care.

The child's autopsy showed a necrotizing myocarditis and a right lateral ventricle hemorrhage involving the choroid plexus in the brain. While these findings may have occurred secondary to the effects of bacterial sepsis, necrotic tissue and hemorrhages in the brain can develop as a result of an adverse reaction to the hepatitis B vaccine.[20]

Hepatitis B vaccine has been previously found to induce an auto-immune response causing immune complex deposition and a vasculitis in other vaccinated subjects.[21,22] Therefore, it is scientifically and medically possible that the children who are described above as having died from shaken baby syndrome may have instead died from a severe auto-immune reaction with hemorrhaging of their brain tissue as a side effect of the hepatitis B vaccine.

It is also very possible that this child in the VAERS group who died of E. cloacae sepsis, could have died from a hepatitis B vaccine induced auto-immune response causing immune complex deposition. It would certainly help explain the deterioration of his clinical course soon after receiving the vaccine. However, the experts dismissed any idea that the hepatitis B vaccine could have played a role in the child's death. The final autopsy ruled that his death was due to E. cloacae sepsis.

Another neonate was vaccinated with the hepatitis B vaccine soon after birth and died. The autopsy showed persistent fetal circulation (which is a normal finding in neonates soon after birth). The autopsy also showed aspiration of amniotic sac contents felt to be 'possibly infected'. No **definitive** infection is noted on the autopsy yet the child was diagnosed as having an infection with aspiration of amniotic fluid as the cause of death. Most neonates have amniotic fluid in their lungs after birth. A child who aspirates sufficient amniotic fluid to cause concern is not well enough to receive a vaccine and would ordinarily require intensive care monitoring. If the child were well enough to receive a hepatitis B vaccine, the child could not have been sick from an aspiration of amniotic fluid. The cause of death as written on the autopsy is suspect leading to the questionable role the vaccine played in the child's death.

The third neonate in the study reported to have died from an infection was diagnosed with bronchopneumonia, although no organism was specified on the autopsy. However, the child died suddenly after receiving the hepatitis B vaccine. The authors seem satisfied that a child who dies suddenly after a hepatitis B vaccine, in truth, died from a bronchopneumonia that caused sudden death.

Another infant in the VAERS group with congenital heart disease received the hepatitis B vaccine at a well baby visit and died soon after in the car on the way home. The cause of death was listed as congenital heart disease. This is a child whose heart disease was clearly not serious enough to warrant constant in-hospital intensive care monitoring. Since the

child was living at home, there is a strong probability that there was no imminent danger of death from the anatomical defects in the heart. The child's congenital heart disease consisted of a coarctation of the aorta, mitral valve stenosis and chronic biventricular heart failure. More information about the child's cardiac history and medications is needed. Nevertheless, the child was visiting the doctor for a well-baby check-up.

The authors conclude that this child's death within 1 or 2 hours of leaving the doctor's office is just another coincidence. The experts fail to believe in the possible relationship between this child's death and the hepatitis B vaccine. A healthy, non-hospitalized, well-child with a congenital heart disease, with an unknown medical history dies soon after receiving the hepatitis B vaccine and no one thinks to investigate the possible role of the hepatitis B vaccine? A child with congenital heart disease who dies in a car accident does not necessarily die due to the congenital heart disease. How many babies need to coincidentally die after hepatitis B vaccination before it warrants further investigation and greater curiosity without bias?

There is a lot of controversy in the medical literature about the relationship between co-sleeping of infants and families and the incidence of SIDS. Recent reports by the SIDS Global Task Force, looking at childcare practices internationally, show that low SIDS awareness and low SIDS rates are associated with the highest co-sleeping/bed sharing rates.[23] SIDS rates are rare in many Asian cultures where co-sleeping is the norm.[23,24]

Co-sleeping is associated with increased risks to the infant when there are co-morbid factors like maternal smoking, drug or alcohol use, chaotic lifestyles, lack of education and opportunities, prone sleeping, and economically deprived, indigenous people.[25] Greater risk for SIDS may also be associated with soft mattresses and bottle feeding. Moreover, when the family chooses co-sleeping out of necessity rather than as a childcare strategy, an increased risk of SIDS is predicted.

Three of the infants in this study who were diagnosed with SIDS were also reported to have shared their beds with their mothers. The authors do not indicate whether the infants who died of 'SIDS' were co-sleeping at the time of their death although they do insinuate the relationship. Furthermore, the investigators would need to do a more thorough family history (perhaps difficult in this study due to a paucity of information from the VAERS reporting system) to detect any risk factors in the co-sleeping household that would increase the likelihood of a SIDS/co-sleeping relationship. Until a complete picture of the circumstances behind these children's deaths can be painted, the side effects of the hepatitis B vaccine remain a plausible factor in how these infants may have died so suddenly. It is good scientific inquiry to include this as a possibility. It is not enough to exclude it based on a biased belief system. But the real scientific studies need to be done.

In truth, how many of the 18 deaths in this study were as a result of the hepatitis B vaccine?

What about the 2 infants with whom I have had contact whose deaths were related to hepatitis B vaccination but not recognized by the experts? How many more unsuspecting parents and medical providers have incorrectly diagnosed the death of an infant when it may have been related to the hepatitis B vaccination? Until there is a shift in the belief system of the medical profession and the public, we will never know the true relationship between neonatal deaths and hepatitis B vaccination and the numbers will always appear as if there is no clear increase.

The authors of the study go on to say that there is no way to prove or disprove a causal relation between hepatitis B vaccination and SIDS using individual autopsy reports. After closer examination of the autopsies, I would have to disagree with the authors. There is enough suspicion in these reports to raise many questions as to whether some of these children died because of a reaction to the hepatitis B vaccine and not from SIDS. If a relationship between hepatitis B vaccination and neonatal death were to exist, the diagnosis would not be SIDS. It would be death secondary to a reaction to the hepatitis B vaccine.

With the strong beliefs and acceptance in the medical world that the hepatitis B vaccine is safe, neonatal and infant death after vaccination will always be viewed as coincidental and not causal. Unless, of course, you are the parent of one of these innocent, healthy newborns that has no risk for contracting or transmitting the hepatitis B virus, who watches your child die soon after getting a vaccine that is meaningless and senseless to your infant's life and well-being.

I can only hope that parents speak up enough to help abolish the universal hepatitis B vaccination policy for neonates, infants and children. It is remarkable that we in the medical profession have allowed the profit margin from administering the hepatitis B vaccine to cloud our judgment about what is clearly a bad medical decision.

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