

## Review

# The danger of excessive vaccination during brain development: the case for a link to Autism Spectrum Disorders (ASD)

Russell L. Blaylock, MD

Website: [www.russellblaylockmd.com](http://www.russellblaylockmd.com)

### Abstract

The incidence of postnatal (regressive) autism has increased dramatically, since the mid-1980s. A number of studies have related this rise in incidence of Autism Spectrum Disorder (ASD) diagnoses to increases in the number of vaccines added to the childhood immunization schedule at this same time. Despite an intensive effort to identify the causation of this disorder, little has been offered in terms of a central causal mechanism. A number of observations have been made concerning alteration in immune system function, abnormal organic acid synthesis, mercury toxicity, and gliamorphin effects on cerebral function. Yet, none of these adequately explains the relationship to vaccinations. A compelling amount of research has shown that repeated stimulation of the systemic immune system results in first priming of the microglia in the developing brain, followed by an intense microglial reaction with each successive series of vaccinations. Because of the critical dependence of the developing brain on a timed sequence of cytokine and excitatory amino acid fluctuation, sequential vaccination can result in alterations in this critical process that will not only result in synaptic and dendritic loss, but abnormal pathway development. When activated, especially chronically, microglia, the resident immune cell of the brain, secretes a number of inflammatory cytokines, free radicals, lipid peroxidation products, and two excitotoxins—glutamate and quinolinic acid. This evidence suggests that this central mechanism results in the pathological as well as clinical features of autism.

© Copyright 2008, Medical Veritas International Inc. All rights reserved.

**Keywords:** adjuvants, autism, Autism Spectrum disorders (ASD), autoimmunity, celiac disease, glutamate, Hepatitis B vaccine, inflammatory cytokines, influenza vaccine, live virus vaccines, meningococcal vaccine, microglia, mercury toxicity, MMR vaccine, omega-6 oils, pneumococcal vaccine, seizures, T-helper lymphocytes, Th2 predominance, Thimerosal-preserved vaccines, neurodevelopmental disorders, vaccination, vaccines

### Introduction

In 1976, children received 10 vaccines before attending school. Today they will receive over 36 injections. The American Academy of Pediatrics and the Centers for Disease Control assured parents that it was safe to not only administer these vaccines, but that multiple vaccines could be given at one time with complete safety. Is this true? Or are we being lied to on a grand scale?

The medical establishment has created a set of terms that they use constantly to boost their egos and firm up their authority as the unique holders of medical wisdom—the mantra is “evidence-based medicine,” as if everything outside their anointing touch is bogus and suspect. A careful examination of many of the accepted treatments reveals that most have little or no scientific “evidence-based” data to support them. One often cited study found that almost 80% of medical practice had no scientific backing.

This is not to say that medical practice should be entirely based on pure and applied science, as understood in the fields of physics and chemistry. Medicine, as pointed out by many of the great men of medicine, is an art. For those interested, the paper, *Regimentation in Medicine and the Death of Creativity* (located on the Internet website <http://www.russellblaylockmd.com>) discusses the proper role of medicine.

Most medical practitioners recognize that some things are obvious without a placebo-controlled, double-blind, randomized study. For example, there has never been such a study to see if smashing your finger with a hammer will be painful, but we accept it without such pristine evidence. The same is true

with removing brain tumors or sewing up severe lacerations.

I find it interesting that there exists an incredible double standard when it comes to scientific evidence versus vaccination-supporting evidence. The proponents of vaccination safety can just say they are safe, without any supporting evidence whatsoever, and the public is expected to accept this without question. They can announce that mercury is not only safe but also that it seems to actually increase the IQ, and the public is to accept such pronouncements as the truth. They can proclaim Thimerosal safe to use in vaccines without ever having conducted a single study on its safety in over 70 years of use, and we are to accept it.

Yet, let anyone else suggest that excessive vaccination can increase the risk of not only autism but also schizophrenia and neurodegenerative diseases, and the vaccine apologists will scream like banshees: *Where is the evidence? Where is the evidence?* When independent researchers produce study after study questioning vaccination-program safety, the vaccine apologists always proclaim them either as presenting insufficient evidence or as having design “flaws.” More often than not, they just completely ignore the evidence. They have continued to do this even after independent researchers have produced dozens of published peer-reviewed studies that not only demonstrate clinical and scientific links between vaccination and/or vaccine ingredients and autism spectrum disorders (ASDs) but also clearly show the mechanism by which the damage is being done—even on a molecular level. These include cell culture studies, mixed cell cultures, organotypic tissue studies, *in vivo* animal studies using multiple species, and even human studies. To the defenders of vaccine safety, this ever-increasing body of

evidence is never sufficient and what independent researchers accept as a proven reality—the vaccine apologists either ignore or treat as a non-reality.

In the 1950s, there was no proof that cigarette smoking caused lung cancer. The connection was as obvious as the layman's observation that smashing your finger with a hammer would cause pain and even the town drunk knew it was true, but, to the medical establishment's position was, "there is no proof."

No one had ever produced lung cancer in animals by exposing them to cigarette smoke. In fact, my pathology professor, Dr. Jack Strong, had trained monkeys to chain smoke, and after years of smoking, none developed lung cancer. Yet, he was convinced that smoking caused lung cancer. Dr. Alton Oschner, founder of the famed Oschner Clinic in New Orleans, led the charge in proclaiming the link between cigarette smoking and lung cancer. It took almost another decade before the medical establishment was willing to admit that smoking caused most cases of lung cancer.

Almost 30 years passed from the time some iconoclastic men of medicine tried to convince the medical establishment that smoking caused most cases of lung cancer until it became generally accepted. The question that needs to be asked is: How many people died of lung cancer, the most prevalent cause of cancer death in the United States, during this time? Data from the National Cancer Institute estimated that in the year 2004, 157,000 people died of lung cancer. If 80% were secondary to smoking, that would be 125,000 dead. Over a ten-year period that would be over one million dead and, over 30 years, almost 4 million people would have died from a preventable cause of death that, at the time, was still being hotly debated by the medical establishment. Lung-cancer death rates were actually higher during that time period.

Thus, when questions of medical importance are obscured by the medical establishment's demands for conclusive causal proof that is acceptable to the establishment, the cost can be ongoing harm to the health of the public and millions of lives.

Today, there are over one million U.S. children and adults with autism, millions more with other identified neurological and behavioral disorders, and the numbers continue to grow. This is a medical disaster of monumental proportions. The link to the vaccine program is scientifically and logically compelling and, a recent vaccine-injury case, *Hannah Poling v. Sec. HHS*, has even been conceded by the medical personnel at the Division of Vaccine Injury Compensation, Department of Health and Human Services (DVIC). However, the vaccine apologists are still refusing to listen.

Like smoking and lung cancer, there is more than enough proof today to call a halt to the present excessive vaccine program and ban *any level* of mercury in vaccines. In 1983, before the autism epidemic began, children received 10 vaccinations prior to school entry and the U.S. autism rate was on the order of 1 in 10,000. Today, children receive 23 or more vaccines prior to the age 2 years and 36 or more by the time of school entry and the U.S. autism rate is now greater than 1 in 150. Medical "experts" have provided no plausible alternative explanation for this dramatic and sudden increase in ASD cases, despite their frantic efforts to find one.

Medical "experts" attempted to blame the increase on a ge-

netic factor, but independent geneticists were quick to respond that genetic disorders do not suddenly increase in such astronomical proportions. The vaccine apologists then said it was because of better diagnosis, despite the facts that: a) the diagnosis is obvious in virtually every case and b) the criteria officially accepted for diagnosis has become more, not less, restrictive.

When trapped by a lack of evidence, defenders of a nefarious position resort to their old standby—the epidemiological study. Statisticians will tell you that the least reliable type of study is an epidemiological study because it is easy to manipulate the data so that the study tells you anything you wish it to. Every justification offered by vaccine defenders is based on such studies and never the actual science. Moreover, the epidemiological studies conducted and/or pointed to by the vaccine apologists suffer from the post-publication refusal of the contact authors to provide the datasets they used so that independent researchers could confirm the validity of the design and findings of their studies. Then, the vaccination-safety defenders have had an Institute of Medicine review committee that they hired and charged to review the initial studies and announce that the issue is settled and no further studies need be done. In addition, in subsequent epidemiological studies conducted by those who defend vaccine safety, the papers have also made these "it is settled" claims even though all know that no epidemiological study can disprove the possibility of a link – it can only determine the statistical probability that there may be a link in the population studied. Of course, vaccine apologists tout their findings to the mainstream media who, because of the advertising dollars they receive from the healthcare establishment, are only too happy to publish such pro-vaccination propaganda as if it were factually accurate.

After the media has been informed that the issue has been settled, those who continue to present the evidence are considered kooks and the great unwashed ignorant.

### **The autism disaster: is it manmade?**

Today, specialists speak of the autism spectrum disorders (ASDs), which, in the U.S., currently includes a number of neurodevelopmental disorders such as autism (autistic disorder), Asperger's syndrome, and pervasive developmental disorders—not otherwise specified (PDD-NOS).

Historically, when specialists know very little about a "psychiatric" disorder, they spend an inordinate amount of time naming and sub-classifying it. In addition, they go to great lengths to define characteristics and symptoms of the disorder that must be present to meet the criteria of classification. Those patients who fail to meet these criteria tend to be ignored.

In the early 1980s, the incidence of children diagnosed with an ASD was about 1 in 10,000 children. By 2005, the incidence had leaped to about 1 in 250 and today it is more than 1 in 150 and appears to be still climbing—although this may be an underascertainment artifact. One of the strongest apparent causal links to the increase in ASD cases was a drastic change in the vaccine programs of the United States and many other countries, which included a dramatic increase in the number of vaccines being given at a very early age. No other explanation has been forthcoming from the medical establishment.

This paper presents evidence which has not been adequately discussed previously, that provides strong evidence for a link between excessive vaccination and neurodevelopmental disorders. A 2003 paper [55] stated that removing the mercury from vaccines would help relieve the problem, but it would not eliminate it. This observation was based on a number of studies in the neuroscience literature that indicated that excessive and especially repeated immune stimulation could result in severe disruption of brain development and even neurodegeneration.

In this 2003 paper as well as in another follow-up paper, the central mechanism for this harm was attributed to excessive and prolonged microglial activation with an interaction between inflammatory cytokines and glutamate receptor subtypes. The Vargas *et al.* study [54], published two years later in 2005, strongly supported my hypothesis, with the finding of elevated inflammatory cytokines as well as the presence of extensive, widespread activated microglia and astrocytes in the autistic brains of individuals aged 5 years to 44 years that were examined. These findings indicate that the brain's immune activation persists for decades. Recent research indicates that this phenomenon is not that uncommon and can be reproduced in the laboratory using a variety of immune stimulating agents and neurotoxins, including mercury and aluminum.

### Autoimmunity and vaccinations

A number of studies have suggested a link between autoimmune disorders and autism risk. Support comes from studies showing an increased risk of ASD in children of mothers with autoimmune disorders [1-3]. Yet, not all studies agreed, since at least one carefully done study found no strong link [4].

Other carefully performed studies provided evidence suggesting some link. For example, in one study serum from a mother with an autistic child was found to bind immunologically with specific brain cells (Purkinje cells) [5]. When this serum was injected into pregnant mice, their babies demonstrated neurological changes suggestive of autistic behavior, indicating a transfer of the autoantibodies into the developing baby mice.

A number of studies of autistic children have found significantly elevated numbers of autoantibodies to various brain structures, such as serotonin receptors, myelin basic protein, neuron axon filament protein, nerve growth factor, and cerebellar neurofilaments [6-10]. It should be understood that these autoantibodies are not found in all cases to cause the disease but, instead, they may develop as a result of the damage caused by the disease itself. For example, we know that a substantial number of stroke victims or individuals with head injury will develop autoantibodies to brain proteins. Nevertheless, the autoantibodies can worsen the damage and prolong the damaging pathology.

It has also been demonstrated that methylmercury (from fish) and ethylmercury (in Thimerosal) are both powerful immunosuppressants and are associated with a high incidence of autoimmunity [11]. In the 2005 study by Havarinasab *et al.*, researchers found that unlike methylmercury, thimerosal (ethylmercury) initially caused immune suppression and then strong TH2-induced autoimmunity. They attributed this to the higher conversion of ethylmercury to ionic mercury ( $Hg^{+}$ ) than seen

with methylmercury. In fact, one 2004 study by Hornig *et al.* found that strains of mice highly susceptible to developing autoimmune diseases were sensitive to the ASD-like behavioral effects upon mercury exposure; whereas, mouse strains genetically not susceptible to autoimmunity do not develop ASD behaviors [12]. It is obvious from the extremely high incidence of ASD that these autoimmune-related genes are very common, but they remain silent until triggered by vaccines or other environmental toxins.

Immunologists have now concluded that autoimmune disorders are not the result of excessive activation of a normal immune system, but rather activation of a dysfunctional immune system. The question remains: What is causing such widespread immune dysfunction among our population? Studies have shown that the number of autoimmune diseases has increased over the past 30 years, with asthma, type 1 diabetes, and eczema rates increasing over two fold. There is also compelling evidence to indicate that certain vaccinations are associated with these autoimmune-related conditions [13,14].

A compelling number of studies have shown an increased incidence of autoimmune reactions in children with ASD, especially involving measles antigens, milk antigens, and antibodies to gliadin and gluten [15-17]. Some of these antigens have been shown to cross-react with brain-derived proteins as well, especially those in the cerebellum, a major structure affected in these disorders [18].

Recently, neuroscientists have shown that much of the damage done in cases of autoimmunity is not due to direct immune reactions with brain structures, but rather results from the release of storms of free radicals and lipid peroxidation products during the immune reaction, something that can be called a "hand grenade in a shopping mall effect." If you use a hand grenade to target a single person in a crowd, you will not only kill and injure the intended target, but all of the bystanders as well.

Neuroscientists P. L. McGeer and E. G. McGeer have named this effect *bystander damage* [19]. The immune attack caused by the autoimmune reaction in the autistic person's brain damages a number of surrounding structures, especially brain connections called dendrites and synapses. Subsequent studies have confirmed that bystander damage is the most destructive reaction of autoimmunity.

Some studies referred to above have shown that autism is much more commonly diagnosed among children in families with a hereditary tendency for autoimmune diseases, which makes sense because one or both parents will manifest dysfunctional immune systems. There is also compelling evidence that vaccines themselves can damage the immune system of immature animals, leading to a higher incidence of autoimmunity and abnormal brain development [20-24]. Mercury, even in small concentrations, is also known to induce autoimmunity in a high percentage of those exposed [11].

Ironically, things that suppress a portion of the immune system, usually cellular type immunity, increase the likelihood of autoimmunity. Immunologists speak about a Th1 to Th2 shift and vice versa. This can occur with exposure to mercury as well as in response to vaccination [25]. A great number of autoimmune diseases are associated with a Th2 shift.

The immune system is a very complex system that is incom-

pletely formed at birth. This means, as has been confirmed in both animal and human studies, that the manner in which the immune system reacts to vaccinations differs according to age; so that small babies have a different reaction than adults. This has been shown with the hepatitis B vaccine now given to newborns. The rate of maturation of the immune system also differs considerably among babies and children, meaning we cannot say what effect will occur in all children. There are a great many variables, including diet.

The immune system's reaction to infection and immunization can be quite different. Normally the immune system relies on a shifting of T-lymphocyte function to determine which is better for the particular situation [26]. The T-helper lymphocytes (Th) can exist as Th1, Th0, or Th2 forms. When there is no infection, the system is in the Th0 mode (an uncommitted phase). If a virus invades, the T-helper lymphocytes quickly switch to the Th1 phase—allowing immune cells to secrete a group of cytokines that kill viruses. This Th1 phase also activates immune lymphocytes that kill viruses and bacteria. At other times, the immune system needs an entirely different set of immune signals and cells that are supplied by the Th2 phase. The Th2 phase in general reduces immune reaction and favors the production of antibodies mainly supplied by B-cells.

Infants remain in the Th2 mode during intrauterine life as a part of nature's immune-cloaking to prevent the fetus from being immunologically rejected by the mother during pregnancy (much like transplant rejection), since, if seen by the mother's immune system, the baby would be recognized as a foreign body. Upon birth, expecting to receive the needed antibodies from its mother's breast milk, the baby continues to remain primarily in a Th2 mode, but has a limited ability to switch to the Th1 defensive mode should the need arise—perhaps as the result of an infection. Months later the baby's tertiary immune system begins to switch to the adult primarily Th1 mode. If the baby's immune system remains in a Th2 mode for too long, it will exhibit a higher risk of developing an autoimmune disorder, such as eczema, asthma, or other allergies.

Presently, vaccine authorities recommend every baby be vaccinated with the Hepatitis B vaccine (HepB) at birth. But, is this safe? A recent study looked at the immune reaction in newborn infants up to the age of one year who had received the HepB vaccine to see if their immune reaction differed from that of adults getting the same vaccine [27]. The study found that infants, even after age one year, reacted differently than adults. HepB antibody levels of infants were substantially higher (about 3-fold) than those of the adults and HepB levels in infants remained higher throughout the study. In essence, babies responded to the vaccine by having an intense Th2 response that persisted long after it should have disappeared—a completely abnormal response.

Autistic children have been described as having a Th2 predominance, which would explain their propensity to develop autoimmune diseases and to be more susceptible to infections early in life [20, 28-30]. Elevated proinflammatory cytokines, particularly TNF- $\alpha$ , have been described in studies of the cytokine profile in autistic children. As we shall see later, an excess production of B-cell cytokines and suppression of T-lymphocyte TH1 activity, as seen in autism, is associated with a high incidence of neurological damage by excitotoxins.

Several things about these immune responses should be the concern to all parents, including effects of such immune overstimulation during pregnancy. For example, it has been shown that excess immune stimulation, as occurs with vaccination, can significantly increase the risk of a pregnant woman having a child with autism or schizophrenia later in life, depending on when the vaccine is given [31,32]. In addition, persistent Th2 responses caused by the HepB vaccine puts a child at a great risk of developing an autoimmune disorder and impairing the baby's ability to fight infections. This means that vaccinating an infant immediately following birth may place your child at a greater risk of all childhood related infections, including *H. influenza* meningitis, meningococcal meningitis, rotavirus, measles, chickenpox, etc. Additionally, numerous studies have shown that such immune suppression greatly increases the number of severe complications associated with these infections. Thus, should your child be exposed to measles or chickenpox, they are more likely to suffer neurological damage, seizures, or other systemic disorders [12,33,34]. When this occurs, rather than admit that the science indicates that the vaccine program is the cause of the complications and deaths, vaccine proponents consider this an opportunity to demonstrate the need for greater efforts to vaccinate our children.

### Immune suppression by live virus containing vaccines

It is also known that certain viruses, including the measles virus, powerfully suppress immunity [35]. The MMR vaccine contains live measles, mumps, and rubella viruses. Moreover, recent studies have shown that immune suppression after vaccination with this vaccine suppresses immunity in a profound way, lasting up to six months [36-41]. In fact, the CDC recommends separating the MMR vaccine from other live virus vaccines to prevent viral overgrowth even though the measles virus is combined with two other live viruses—rubella and mumps—in the MMR vaccine.

Public health officials never address the obvious question: wouldn't the MMR vaccine make the child more susceptible to other naturally occurring infections such as *hemophilus influenza* type B meningitis, meningococcal meningitis, persistent measles infection, influenza infection, and even chickenpox? This has been strongly suggested by a number of studies [42]. Not only would such children be more susceptible, but as previously discussed, severe complications and even death would be more prevalent as well.

When death and severe complications occur due to these infections, pediatricians, the CDC, and the American Academy of Pediatrics use this as a justification for more vaccines, never admitting that the increased incidence of these infections and complications was likely precipitated by their very own vaccine recommendations.

Increased risk of infection following vaccination is especially high for a child among other children in a household or in day care centers. With a prolonged suppressed immune system, exposure to other sick children can put a vaccinated child at a high risk of contracting the infection and experiencing complications including death from that subsequent infection.

Studies have also shown that vaccines that cover only a few specific strains of a virus or bacteria that naturally have a great

number of strains (some have over a hundred different strains), can cause a shift in strain dominance so that the strain not included in the vaccine emerges as the dominant disease-causing strain. We see this demonstrated with the meningococcal and pneumococcal vaccines [43–45]. While this adverse outcome is discussed in the scientific literature, the general public and most pediatricians seem to be unaware of this information.

When vaccine viral or biological components are combined with Thimerosal (50% mercury by weight)—which is also an immune-suppressing substance and bioaccumulative systemic poison—adverse effects are compounded. Fluoroaluminum, formed in fluoridated drinking water, is another substance that interferes with immune function, as are many insecticides and herbicides used around the home [46].

Often forgotten, is the substantial evidence that omega-6 oils powerfully induce inflammation and immune suppression when consumed in large amounts. Those eating a Western diet are consuming 50-fold higher amounts of this type of oil (called linoleic acid) than normally recommended. Through consumption of food containing these oils—including corn, safflower, sunflower, canola, peanut and soybean oils—children’s immunity can be altered, making them not only more susceptible to natural infection, but also prone to vaccine complications.

In essence, by over-vaccinating our children, public health officials are weakening their immune system, making them more susceptible to a number of infections, and less able to combat infections. This provides these officials an endless source of “horror stories” to justify additional vaccines. Remember also that mercury is an immune suppressant that accumulates in the body from Thimerosal-containing vaccines, seafood consumption, and environmental pollution.

It is not difficult to understand why a pregnant mother having dental amalgam fillings, on a diet high in methylmercury-containing seafood, and living in an area with high atmospheric mercury (e.g. West Texas), would be at a greater risk of having an autistic child than one not exposed to these sources of mercury. Contributions to the body’s mercury load from amalgams, diet, and the environment have never been a consideration to physicians who insist all children be given the same routine vaccines, including the Thimerosal-containing flu vaccine.

### The child prone to being diagnosed with an ASD

What is becoming increasingly obvious is that there are specific risk factors associated with certain children that develop autism. Some newborns and small children, because of a developmental immune deficiency, may be more vulnerable to developing infections at a higher rate than other less vulnerable children. Such developmental immune deficiencies can affect only a portion of the immune system and are often not diagnosed by a pediatrician. A great number of cases of childhood immune deficiencies go undiagnosed by practicing pediatricians, especially the more subtle cases, which may comprise the majority of ASD-prone children.

Many physicians treating autistic children have noted a high incidence of ear infections that are treated with broad-spectrum antibiotics. This, in turn, often leads to a high incidence of *Candida* overgrowth in the child’s body. Both infections prime the microglia (or specific resident immune cells) in the child’s

brain. This priming effect shifts these normally resting microglia immune cells into overdrive [47]. If stimulated again within weeks or even months, they generate extremely high levels of free radicals, lipid peroxidation products, inflammatory cytokines, and two excitotoxins—glutamate and quinolinic acid [48]. Studies have shown this to be a major mechanism underlying viral- and vaccine-related brain injury.

The high incidence of infection in these children indicates the possibility of preexisting immune system dysfunction. As stated, this also increases the risk of an autoimmune reaction. The stage is then set for the autism cascade to develop and this can be triggered by early vaccination or a recurrent infection. Remember, the microglia have been primed, either by a natural infection or an earlier vaccination (such as the hepatitis B vaccine given soon after birth or a flu shot given to the mother prepartum). The vaccine is different from a natural infection in that the vaccine produces brain immune stimulation for very prolonged periods.

Both animal and human studies show that either systemic infections or immune activation by vaccines, rapidly activate the brain’s microglial system; moreover, vaccines can do so for prolonged periods [49–53]. Once the primed microglia are reactivated by a subsequent vaccination or infection, the microglia activate fully and secrete their destructive compounds as previously discussed.

The immune system quickly clears a natural infection and then shuts off the immune activation, thus allowing repair of what damage was done. This shutting down of the microglia is very important. By contrast, there is evidence that with repeated and excessive vaccine-triggered immune stimulation, the microglia do not shut down [47]. This difference in immune system function is supported by Vargas *et al.* In this study, dying brains of 11 autistic patients ranging in age from 5 years to 44 years without active infectious disease were compared to age matched controls [54]. Widespread activation of inflammatory cells (microglia and astrocytes) found in the brains of the autistic patients explained the widespread brain damage seen in all the autism cases.

The Vargas *et al.* study represented one of the most extensive examinations of the immune reactions in the autistic brain ever conducted, involving immunocytochemistry, cytokine protein assays and enzyme-linked immunosorbent assays of the brain tissue. Similar assays were performed on spinal fluid from an additional six living autistic patients, which confirmed the intense immune activation and inflammation.

The typical child will have received at least 24 inoculations by age two years and 36 by the time of school entry. Many of these will be spaced within a month or two of each other, which means the priming and activation cycle of the microglia will be virtually continuous. In addition, recent CDC recommendations specify children are to receive a flu vaccine every year starting at age 6 months through 18 years. In the U.S., the majority of the inactivated influenza vaccine doses still contain a full “preservative” dose of Thimerosal (i.e., 0.005% mercury—equivalent to 25 micrograms [ $\mu\text{g}$ ] of Hg per 0.5 mL, or 50  $\mu\text{g}$  of Hg per mL, which is the same as 50,000  $\mu\text{g}$  per liter; or 50,000 parts per billion [ppb] of mercury) and most of the rest contain a lower level of Thimerosal (i.e., these reduced-Thimerosal flu vaccine doses contain 0.0002% mercury—equivalent to 1 mi-

crogram [ $\mu\text{g}$ ] of Hg per 0.5 mL, or 2  $\mu\text{g}$  of Hg per mL, which is the same as 2000  $\mu\text{g}$  per liter; or 2000 ppb).

Additionally, we must consider the effect of the measles and rubella portions of the MMR vaccine that is administered beginning at age 1 year. The profound immune suppression, which lasts up to six months following vaccination, not only increases the risk of developing other infections, but increases the risk of an autoimmune reaction. Cytomegalovirus is also a powerful immune suppressing virus that commonly infects newborns and small children, especially if they are immune suppressed. So, we see that administering a live, immunosuppressant vaccine early in life can dramatically increase the risk of autoimmune disorders, increase microglial-induced brain injury, and increase the risk of infection by other immune-suppressing viruses and pathogenic organisms. Such vaccination dramatically increases the risk of a child developing one of the autism spectrum disorders (ASDs).

It should also be appreciated that the *Candida* infections in these children trigger a prolonged systemic immune reaction. This contributes to a prolonged brain immune response and worsens any autoimmune disorder that may have been produced.

### Seizures and autism

It is estimated that 30% to as high as 82% of autistic children develop seizures, depending on the sensitivity of the examination [55,56]. Growing evidence indicates there is a close correlation between brain inflammation (by microglial released inflammatory cytokines and glutamate) and seizures. This same correlation is seen between excessive brain immune stimulation with vaccines and seizures. Using lipopolysaccharide as a vaccine-based immune stimulant, scientists have induced seizures in experimental animals of various species [57,58].

A considerable amount of evidence links excitotoxicity and seizures. In addition, a number of the newer anti-seizure medications work by blocking glutamate receptors or preventing glutamate release. One of the central mechanisms linking excessive immune stimulation, as occurs with vaccines, with seizures, is the induced release of the excitotoxins glutamate and quinolinic acid from immune stimulated microglia and astrocytes [59-61].

In many cases, vaccine-triggered seizures are clinically silent or manifest as behavioral problems. Such seizure activity often goes unrecognized by pediatricians; yet, such seizures can alter brain function and eventually result in abnormal brain development. Even the CDC and American Academy of Pediatrics recognize that infants and children with a history of seizures should not be vaccinated.

It is also known that autistic children who regress, that is begin to show a sudden worsening of mental development, have a significantly higher incidence of both clinical and subclinical seizures compared to those children who do not regress. Interestingly, studies have shown that during early brain development after birth, the number of glutamate receptors (that trigger the seizures) increase steadily until they peak at the age of 2 years [62]. Thereafter, glutamate receptors decline in number. Thus, when a young child receives 24 vaccines, his or her immature brain is significantly more susceptible to seizures than

an older, more mature brain. There is a high incidence of seizure associated with vaccines administered to young children.

Consider the case of a mother who takes her one-year-old child to the pediatrician for vaccines. The pediatrician convinces the mother that all five vaccines be administered to the child during that single office visit as recommended for all children in that age group. After all, both the CDC and the American Academy of Pediatrics assure mothers and fathers that this practice is completely safe. This means that the child's immune system will be assaulted by 7 different antigens (viruses, three of which are alive) as well as five full doses of immune adjuvant—a powerful mix of immune-stimulating chemicals.

This intense immune stimulation not only produces a painful, swollen redness at the injection sites, but gives rise to a hyperintense activation of the brain's immune system. Mothers and fathers are familiar with the high-pitched crying their babies have produced following such a series of vaccinations. Often, this high-pitched crying, lethargy, and poor feeding continues for a duration ranging from weeks to months. This reaction is not due to injection pain as the pediatrician often tries to assure; rather, it is secondary to brain inflammation—what we call an encephalitic cry [63].

Recently, CDC modified its preference for the combination MMR-V (Measles, Mumps, Rubella, and Varicella) vaccine by Merck known by the trade name ProQuad. The administration of this quadravalent vaccine to 43,000 children resulted in twice the number of seizures compared to those children who were administered MMR and Varivax vaccines separately. The MMR-V vaccine contains MMR antigens as well as five times the varicella (chickenpox) viral antigen contained in the Varivax vaccine. Public health officials attributed the increased seizures to fever caused by the vaccine; however, this is only part of the story.

I have seen a number of febrile seizures during my neurosurgical practice and my research indicates that the reason some children are susceptible to febrile seizures and others are not, is that the susceptible ones are deficient in neuroprotective nutrients and are often exposed to neurotoxic substances, such as mercury and aluminum, that increase sensitivity to seizures. Consistently found in the studies of febrile seizures is the presence of low blood sodium levels (called hyponatremia) [64].

Physicians specializing in neurology know that very low sodium blood levels can trigger (even in normal individuals) seizures, rapid coma, and death—especially in children. In the presence of brain inflammation, the incidence of hyponatremic seizures is much higher. One of the major causes of hyponatremia in infants and small children is the doctor giving IV fluids that contain little or no sodium chloride (salt). During my medical practice, I constantly tried to convince pediatricians to stop using D5W (5% dextrose and water) as an IV solution in sick children, since this protocol induced seizures. I am convinced that a significant number of children who died following a meningitis infection, actually died of hyponatremia induced by a combination of the infection and the pediatrician specifying hypotonic IV fluids (D5W) during treatment.

I will always remember the case of a young girl who developed *H. influenzae* meningitis and slipped into a deep coma. The treating pediatricians consulted me, suspecting a brain abscess. This was quickly ruled out; however, I noticed the child was

getting D<sub>5</sub>W as an IV solution. A simple blood test demonstrated the girl had severe hyponatremia. Because she was comatose, the pediatricians urged me to let her die. When I refused, the pediatricians went so far as to approach my partners to have them remove me from the case. Fortunately, they refused to intervene. When I corrected her sodium deficiency, she made a good recovery and had no further seizures.

Studies in small animals with immature nervous systems have shown that glutamate, as MSG, increases the likelihood of seizures from other causes, such as fever [65,66]. Excess vaccination increases brain levels of glutamate.

Keep in mind that the child by age one will already have had 20 vaccine inoculations, each spaced no more than one or two months apart. This means the brain microglia are maintained in a constantly primed state. Each vaccine increases dramatically the damage done by the previous vaccine series. One should not be surprised to learn that many vaccinated children develop seizures, often repetitive seizures, or that vaccinated children exhibit a high incidence of autism. And I can assure the elite of the American Academy of Pediatrics and the CDC that over one million autistic children pose far more disease burden and medical costs than do measles, mumps, diphtheria, chickenpox, tetanus, rotavirus, HiB meningitis, and hepatitis combined. Also, please consider that for every fully autistic child, there are more than ten times that many children with lesser degrees of impairment.

Compelling evidence indicates that the death rates for diseases targeted by childhood vaccines fell dramatically, by over 90%, in developed countries prior to the mass vaccination programs. Documentation for this fact is found in Neil Z. Miller's book, *Vaccines: Are They Really Safe and Effective?* [67] Objective studies attribute the fall in death rates to better nutrition and improved public sanitation. So, when health authorities warn (or use the scare tactic) that stopping the present vaccine program will mean a return to millions of children dying from childhood diseases, this is simply untrue.

### Human brain development is different

Humans undergo unusual brain development in that there is a prolonged period of maturation and neuroanatomical pathway development occurring years after birth. The most rapid brain development occurs during the last trimester of intrauterine life and two years after birth—what is referred to as the brain growth spurt. Areas regulating higher brain functions, such as emotions, emotional control, thinking, complex memory, and language function, are last to develop.

Recent studies using functional MRI scans (fMRI) and PET (positron emission tomography) scans have shown that substantial brain development continues until approximately age 26 or 27 months. Using such brain mapping techniques as volumetric parcellations that give a 3-D view of the brain, researchers examined the brains of 13 children followed for 10 years with scans being done every 2 years [68]. It was found that there was an overdevelopment of synaptic connections after birth that was gradually removed (called pruning) in developmental cycles during early childhood and even adolescence. For example, around age 4 to 8 years, a thinning of the cortex in the language areas of the brain (parietal lobes) occurs that spreads to the tem-

poral lobes and finally to the frontal lobes. This thinning moves the brain into a more functional state of development, that is, it gets rid of unnecessary pathways and connections—sort of a final correction.

Further, these researchers found that language areas of the brain matured around age 11 to 13 years and the brain areas controlling higher brain function, the prefrontal cortex, matured in the mid-twenties [69,70]. Thus, during the first two years of life, the child's brain undergoes rapid and very critical development, with the more advanced cognitive portions of the brain continuing their development later—much later.

There is compelling evidence that the pruning of these excess synapses is essential; otherwise, the brain would be inundated with an enormous array of competing signals—that is, a lot of static and misinterpreted messages. This pruning process, as well as the growth, maturation, and migration of neurons, is carried out by a combination of signals, which include carefully controlled fluctuating glutamate brain levels and appearance of specific microglia-released cytokines in a timed sequence [63,71-75]. This is all very exacting and easily disturbed by a number of toxins, such as mercury and aluminum. It is also critically dependent on the presence of thyroid hormone.

Anything that alters these fluctuating glutamate and cytokine levels can affect, sometimes in drastic ways, the development of the brain which, as mentioned earlier, continues far into young adulthood [76-79]

Pathological studies of autistic brains demonstrate three areas that are especially affected: the cerebellum, the limbic system, and the prefrontal cortex [80-83]. There exist intimate connections between the cerebellum and prefrontal cortex and between the prefrontal cortex and limbic system—in particular the amygdalar nuclei. These areas are also frequently affected by inflammatory cytokines during immune stimulation, such as occurs following vaccination [84]. In the Vargas *et al.* study, the most intense microglial activation occurred in the cerebellum [54].

In low concentrations, both the cytokines and glutamate act to protect developing brain cells and promote brain development (neurotrophic function); but in high concentrations they can be very destructive, especially in combination. Of particular importance are the inflammatory cytokines interleukin 1 and 1 $\beta$  (IL-1 and IL-1 $\beta$ ), IL-6, and tumor necrosis factor-alpha (TNF- $\alpha$ ) [85-89].

Evidence that an alteration in these cytokines can cause developmental brain problems comes, in part, from studies of schizophrenia, a disorder that can be produced by stimulating inflammatory cytokine surges during pregnancy [90-92]. It is known, for example, that women who are infected with the flu during pregnancy are significantly more likely to give birth to an autistic child or a child with schizophrenia, depending on when the infection occurs. At first, they assumed this was due to the virus being passed to the fetus, but subsequent studies found that it was not the virus, but the mother's immune reaction that caused the problem—that is, it was the immune cytokines (IL-1, IL-2, IL-8, IL-6, and TNF- $\alpha$ ) that were causing the injury to the baby's developing brain.

The insane policy of having every pregnant woman vaccinated with an inactivated influenza vaccine dose, which often contains a preservative level of Thimerosal, is contrary to what

we know concerning the neurotoxic effect of excessive immune stimulation during pregnancy. Even if the vaccine prevented the flu (studies show it reduces it only in a select few), instead of a small percentage of pregnant women being at risk, vaccine policy insures every woman will be at risk. Keep in mind that some children will have been administered the flu shot (containing mercury) every year since age 6 months (according to present CDC recommendations), meaning they will have accumulated a significant amount of mercury and will, as a result, have a hyper-intense cytokine response should the flu vaccine have also been administered during pregnancy. In addition, they will have accumulated a significant amount of neurotoxic mercury and aluminum.

It is also important to keep in mind that the persistence of immune activation with vaccination is of longer duration than that resulting from natural immunity. Thus, immune activation with vaccination does not allow the brain time to repair itself either in the mother or in the fetus. In addition, recall that the way the immune system reacts to vaccination, differs between adults and the very young.

A new study from the Weizmann Institute in Israel by H. Schori *et al.* found that with a normally functioning immune system, the T-lymphocytes actually protected neurons from glutamate excitotoxicity, but if the immune system was dysfunctional, as is the case in most ASD children, the opposite occurred [93]. That is, stimulating the immune system was significantly destructive of the brain's cells. The Schori *et al.* study found that under conditions of immune dysfunction, B-cells predominated in invading the brain and this dramatically increased the destructive effect of excess glutamate.

Another study also found that mercury toxicity was greatest in mice prone to develop autoimmune diseases, thus confirming the findings reported in the Hornig *et al.* study [12]. Further, the Schori *et al.* study found that even in animals without an autoimmune-prone genetic makeup, suppression of T-lymphocyte function increased excitotoxic damage. Both the measles and cytomegalovirus inhibit T-cell function, as do mercury and the hepatitis B vaccine [11,27,35,41].

The Vargas *et al.* study also demonstrated that T-lymphocytes failed to infiltrate the autistic brains examined, meaning that the T-lymphocyte protection was not in evidence [54]. Under these conditions, systemic immune activation, as seen with multiple and sequential vaccinations, would increase the excitotoxic damage caused by the activation of the microglial cells, including the astrocytes.

When all the evidence is taken together, these studies provide powerful evidence that sequential, multiple vaccinations in newborns and small children maximize brain inflammation and, as a consequence, this dramatically enhances the excitotoxic pathology, doing so for prolonged periods (decades). As more vaccines are added to the recommended vaccination schedule, the devastating effects of excitotoxic pathology will be observed more frequently and the harm observed will be increasingly severe.

### What about the adjuvants used in vaccines?

While mercury has received much of the attention, aluminum (found in most vaccines) is also a major culprit in this

shocking saga. Added to most vaccine are a number of substances either used during manufacturing or designed as an immune booster (adjuvant). These substances include albumin, aluminum (either as hydrated aluminum hydroxide, aluminum phosphate or alum, also known as hydrated aluminum potassium sulfate), various amino acids, DNA residues, egg protein, gelatin, surfactants, monosodium glutamate (MSG), MRC-5 cellular protein, Thimerosal, and various antibiotics. Not listed on official lists are bacterial and viral contaminants, which can include their particulate, fragmented matter [94,95].

Aluminum compounds serve to dramatically boost and prolong the immune reaction to the vaccination. Some aluminum remains in the site of injection for years. Aluminum was first added to vaccines in 1926. Aluminum compounds as well as other vaccine components boost immunity—including some undesirable components of the immune system such as B-cells.

Because vaccine adjuvants are designed to produce prolonged immune stimulation, they pose a particular hazard to the developing nervous system. Studies have shown that immune activation following vaccination can last up to two years. This means that the brain's microglial cells are also primed for the same length of time or possibly longer.

A new emerging syndrome called *macrophagic myofasciitis* has been attributed to the aluminum adjuvant in vaccines and is especially associated with the hepatitis B vaccine and the adsorbed tetanus vaccine [100]. Victims of this syndrome suffer severe muscle and joint pains and severe weakness. Subsequent studies conducted since the syndrome was first described in France, indicate widespread, severe brain injury as well—confirmed by MRI scanning [101,102]. This brain syndrome has been described in American children as well.

It is known that aluminum can accumulate in the brain and that this accumulated aluminum is associated with neurodegeneration. The evidence for a link between aluminum neurotoxicity and Alzheimer's disease continues to mount. Aluminum, like mercury, activates microglia leading to chronic brain inflammation—a major event in both Alzheimer's disease and Parkinson's disease [103-110].

Flarend *et al.* conducted a study (using radiolabelled aluminum [<sup>26</sup>Al]) in which either of two approved forms of adjuvants (aluminum hydroxide or aluminum phosphate) used in vaccines were injected at a dose approved by the FDA (0.85 mg per dose) [111]. The results showed that aluminum was rapidly absorbed into the blood from both forms. However, aluminum phosphate was absorbed faster and produced tissue levels 2.9 times higher than aluminum hydroxide. Blood levels of aluminum remained elevated for 28 days with both adjuvants. Elevated aluminum levels were found in the kidney, spleen, liver, heart, lymph nodes, and brain.

Thus, aluminum from vaccines is redistributed to numerous organs including the brain where it can accumulate. Each vaccine adds to this tissue level of aluminum. A total dose of 30.6 mg (and not the 0.85 mg considered safe by the FDA) is available when we calculate the total aluminum dose available from 36 vaccinations. Of course, not all of this aluminum ends up in the tissues. However, aluminum can accumulate in substantial amounts from ingesting foods containing aluminum and from drinking water. When a number of aluminum-containing vaccines are administered to a child during a single office visit,



aluminum blood levels rise rapidly and can persist at a high elevation for over a month. During this period of high elevation, aluminum infiltrates the tissues, including the brain.

It is also known that aluminum enhances the toxicity of mercury and that aluminum, even from sources other than vaccines, increases inflammation in the body [106]. The question no one seems to be asking is this: Does the aluminum act as a constant source of brain inflammation? Research, especially focusing on aluminum-triggered microglial activation, seems to indicate it does [112]. Dr. Anna Strunecka, professor of physiology, found aluminum readily binds with fluoride to form fluoroaluminum and that this compound can activate G-protein receptors that control a number of neurotransmitters, including glutamate receptors [46]. Administering multiple aluminum-containing vaccines at once raises blood and tissue levels much higher than when these same vaccines are administered separately. Fluoride in drinking water, certain foods, and dental treatments can react with the brain aluminum, creating the neurotoxic fluoroaluminum combination. Studies have shown that fluoride also accumulates in the brain.

### The role of mercury in developmental brain damage

Mercury also activates microglia and does so in concentrations below 0.5 microgram (3 to 5 nanograms) per gram of wet tissue [113]. This is well below the concentration found in Thimerosal-containing vaccines administered to children. Ethylmercury hydroxide, like its cousin methylmercury hydroxide, enters the brain very easily, but once within the brain it is rapidly de-ethylated, forming tissue-retained inorganic mercury ( $\text{Hg}^{2+}$ ) species [114]. There is evidence that this “inorganic” mercury is significantly more neurotoxic than the organic mercury compounds from which it forms and more difficult to remove. Studies using monkeys demonstrated that ionic mercury is redistributed in the brain [115]. These same series of studies also demonstrated that there was extensive microglial activation in the monkey’s brain that persisted over 6 months after the mercury dosing was stopped. Thus, when the plasma mercury disappears, the brain mercury remains [116].

The preceding facts are important to remember when vaccine safety promoters tout findings of new studies showing that ethylmercury (in Thimerosal) disappears from the blood within several days. Actually, the mercury leaves the plasma and enters the brain, where it is de-ethylated and, based on human post-mortem research [174], remains with a half-life of about two decades. What is also conveniently hidden are the results of recent studies demonstrating that, within a short time, on average only about 7% of the methylmercury hydroxide administered orally was converted into brain-retained “inorganic” mercury; whereas 34% of the Thimerosal (ethylmercury compound) injected was similarly converted [117]. [Note: The value for “organic” mercury is calculated as the difference between the “total” mercury value and the “inorganic” mercury value based on sub-sample assays from a given homogenized sample. The two mercury assay procedures, “total” and “inorganic” only differ by the severity of the oxidation step used to liberate the mercury species – where the work-up for a “total” mercury determination is done under more severe conditions.] This means that a greater quantity of a more destructive form of

mercury is retained in the brain following administration of a Thimerosal-containing vaccine than from the methylmercury compounds present in fish.

Also consider that the vaccine-based mercury that was removed from the blood, enters the stool in high concentrations. Apparently, because little is excreted in the feces, this mercury can be reabsorbed through the colon, where it recirculates repetitively—meaning that with each cycle the mercury has access to the brain.

Mercury has another link to this immune/excitotoxic reaction. A number of studies have shown that mercury, in submicromolar concentrations, interferes with the removal of glutamate from the extracellular space, where it causes excitotoxicity [118-120]. This removal system not only plays a very important role in protecting the brain, but also in preventing abnormal alterations in brain formation [121]. As you will recall, it is the carefully programmed rise and fall in glutamate levels in the brain that allow the brain’s pathways to develop and allow for proper development of its connections (called synaptogenesis).

Mercury can also damage the brain is by interfering with its energy production. The mitochondria (the energy factory) of the neuron accumulate more mercury than any other part of the cell. It is known that interference with the neuron’s ability to produce energy, greatly magnifies its sensitivity to excitotoxicity—so much so, that even physiological concentrations of glutamate can become excitotoxic [124,125].

One of the destructive reactions of both excitotoxicity and mercury toxicity is the generation of storms of free radicals and lipid peroxidation products. Essential to the protection of brain cells is the antioxidant enzymes (catalase, glutathione peroxidase, and SOD). Mercury poisons these protective enzymes.

One of the most important protective systems is the glutathione molecule, which is present in every cell in the body. Mercury dramatically lowers glutathione levels by a number of mechanisms [126]. So, we see that mercury can greatly aggravate this entire destructive mechanism.

As important role as mercury plays, it is not the lone essential element in this process. Rather, essential to this process is a combination of pre-existing or vaccine-induced immune dysfunction and excess immune stimulation by a crowded vaccine schedule. This is why autism will not go away, even if mercury were to be completely removed from all vaccines and other drugs. It is also important to appreciate that mercury can never be removed entirely from the picture because of the numerous sources of mercury in our environment, including but not limited to contaminated seafood, atmospheric mercury, and dental amalgams. However, were all use of mercury in medicine to be banned in the U.S. and all U.S.-approved drug products containing any level of any added mercury compound, including Thimerosal, to be recalled and destroyed, the overall incidence of autistic order should decline somewhat and the severity of the symptoms these cases exhibit should significantly decline.

### Why males are affected more often

One of the enigmas of autism is why it occurs more often in males than females. Actually there are a number of toxins that have this gender selectivity. For example, both mercury and monosodium glutamate (MSG) have greater neurotoxicity in

males than females [127]. The reason appears to be the enhancing effect of testosterone (and possibly other androgens) on the toxicity of both substances [128,129].

Glutamate is the most abundant neurotransmitter in the brain and operates through a very complex series of receptors (3 major ionotropic receptors [NMDA, AMPA, and kainite receptors] and 8 metabotropic receptors). The presence of glutamate outside brain neurons, even in very small concentrations, is brain cell toxic. Because of this, the brain is equipped with a very elaborate series of mechanisms to remove glutamate quickly, primarily by utilizing glutamate re-uptake proteins (EAAT1-5). Mercury, aluminum, free radicals, lipid peroxidation products, and inflammatory cytokines can easily damage these proteins [130,131].

One of the important ways glutamate regulates neuron function is by allowing calcium to enter the cell and by the release of calcium within cell storage depots. When calcium (glutamate-operated) channels are opened, the calcium flows in as a wave of concentrated calcium. These are referred to as calcium waves or oscillations and they regulate a number of neuron functions, one of which plays a vital role in brain development.

During brain development, the future neurons are lined up along membranes within the core of the undeveloped brain. These cells must migrate outwardly to reach their final destination. They do so by guided chemical signals mainly released by microglia and astrocytes. These trillions of connections also develop during a process called synaptogenesis and use many of the same signals.

Calcium waves cause developing brain cells to migrate, which is essential for development of the brain (forming the architectonic structures and functional columns of the brain) [132]. Interestingly, testosterone also affects embryonic brain cell migration by regulating calcium waves. Mercury has a similar effect, probably by stimulating glutamate release [133]. Estrogen reduces calcium oscillations and stops the migration. Other chemical signals in the brain also play a role (reelin).

If calcium oscillations are not properly regulated and too many occur, the brain develops abnormally. Testosterone and glutamate have an additive effect on these calcium waves. In this way, testosterone enhances the damaging effect of excessive glutamate and mercury.

Higher doses of MSG during brain formation can cause abnormalities of brain development that closely resemble mercury poisoning and the toxic effects of high levels of inflammatory cytokines [76]. Interestingly, vaccination has been shown to significantly increase the toxicity of several other neurotoxins, to the point that brain cell destruction or synaptic loss is triggered even when subtoxic concentrations of the toxicants are used. Testosterone aggravates this toxicity as well.

Studies of autistic children, including those who are female, show that most have elevated levels of androgens [134]. In general, androgens, such as testosterone, enhance neurological injury and estrogens tend to be protective of the brain [135].

### **The role of the leaky gut phenomenon and food intolerances**

Wakefield and his co-workers suggested a connection between the MMR vaccine and abnormal gut function in a landmark article appearing in the journal *Lancet* in 1998 [136]. In

this carefully conducted study, the lining of the intestines of autistic children having GI symptoms was biopsied and demonstrated lymphocytic infiltration as well as elevated levels of inflammatory antibodies and cytokines. TNF- $\alpha$  release was particularly high from these gut-based immune cells. The entire GI tract, from the stomach to the colon, was infiltrated by these immune cells.

Subsequent studies have shown a high incidence of abdominal pain, bloating, diarrhea and constipation in children with ASD [138,139]. A number of other studies have shown problems with digestive enzymes, defective detoxification, and an overgrowth of a number of pathogenic bacteria and fungi in the colon and intestine of ASD children [140,141].

Not surprisingly, a few studies have shown significant improvement in behavior when ASD children are placed on diets devoid of identified food allergens [142-144]. Antibodies to food components such as casein, gliadin, and gluten have also been described as well as cross-reactions between food antigens and brain components [145].

Celiac disease, in which there is an immune sensitivity to the food components gliadin and gluten, closely resembles the case of ASD in terms of brain injury associated with food allergens. Approximately 6% of such patients will demonstrate neurological damage, most frequently cerebellar ataxia [146]. Other studies have also found seizures, cranial nerve damage, dementia and impaired frontal lobe function [147-151].

Autopsy studies indicate that the most commonly found neurological damage occurs in the cerebellum, as we see in autism. Other studies have shown an immunologic cross-reactivity between gluten antibodies and Purkinje cells in the cerebellum [144]. Common to both celiac cases and to autism, the most intense microglia activation and neuronal loss occurred in the cerebellum. In many cases where autistic brains were examined, virtually all of the Purkinje cells were lost [54].

Studies investigating the incidence of GI symptoms in autistic children indicate that from 20% to 84% have complaints. It is interesting to note that in the studies on celiac-related neurological problems, only 13% complained of GI symptoms, so ASD children can have gut-related brain effects without obvious GI symptoms [154].

Some feel that the gliadin, casein, and gluten can be converted to opioid-like substances, such as gliadomorphin and casomorphin that can produce a morphine response in the brain, leading to abnormal behavior [142]. These opioids also suppress immunity and increase excitotoxicity [154]. While the opioid effect exists, it appears that the recurrent immune stimulation of primed microglia is the primary mechanism causing most of the damage seen in autism [155].

Studies have also found frequent dysbiosis in autistic children, that is, an overgrowth of pathogenic bacteria and fungi and a loss of beneficial probiotic organisms [138]. It has been demonstrated that *Candida* organisms can penetrate the gut wall and enter the blood stream where they can be distributed to all tissues and organs, including the brain [156]. The same is true for pathogenic bacteria and bacterial toxins. These brain implanted organisms act as continuous sources of immune stimulation, which is especially damaging to the brain because of vaccine-triggered microglia priming and/or activation occurring before the gut problem presents itself. Repeated vaccination

aggravates this injury.

With each subsequent vaccination, the microglia response is enhanced because of the recurrent immune activation by food antigens and microbiological antigens. It is interesting to note that trials of antibiotic vancomycin, which is not absorbed from the gut, objectively improved the cognitive function of a number of autistic children [157]. We also know that with children having celiac disease, even a very small amount of the offending food can have devastating neurological effects.

## Conclusion

I have presented a considerable amount of evidence implicating a connection between the present vaccine schedule and the development of autism spectrum disorders, yet even this paper serves only as a brief review of what is known. A more in-depth discussion will appear in a paper entitled, *Interaction of activated microglia, excitotoxicity, reactive oxygen and nitrogen species, lipid peroxidation products, and elevated androgens in autism spectrum disorders*, that is to be published in an upcoming special issue focusing on autism in the journal, *Alternative Therapies in Health and Medicine*.

Much of this information is being totally ignored by the medical elite and especially the media. The Simsonwood conference proceedings, in which over 50 scientists, vaccine pharmaceutical company representatives, and representatives from the World Health Organization met secretly in Norcross, Georgia, disclosed that the safety of your children is not their primary interest—their only interest is selling vaccines to the public. A friend of mine, while speaking to an audience of scientists and public health officials in Italy, was rudely told by a public health official that (paraphrased) “We all know that vaccines can cause neurological damage, but we must keep this from the public because it might endanger the vaccine program.”

It is also important to understand that most practicing pediatricians have never heard the preceding discussion. Most have very little understanding of immune function and have no idea of the pathological effect that giving multiple vaccines has on the brain. These effects are widely discussed in the neuroscience literature, but few practicing physicians, especially pediatricians, ever read such articles.

Immunology, like nutrition, gets only scant attention in medical school and even less in residency training of physicians. Older doctors have no concept of the newer discoveries in immunology, especially neuroimmunology. The human immune system is one of the most complex systems in physiology and our studies indicate an even greater complexity is to be found. Despite a renewed interest in the immune system’s function in neonates and small children, much remains unknown concerning the immune effects of exposing infants and small children to such a large barrage of vaccines early in life. Yet, what we do know is that young children with immature immune systems react quite differently than adults and, for some, pediatric vaccinations can have devastating consequences on brain development and function.

Vaccinating millions of children with the hepatitis B vaccine at birth can only be described as dangerous idiocy. The vast majority of infants, children and adolescents in the U.S. are in

no danger from hepatitis B infection—even the medical authorities agree on this point. It is also known that the effectiveness of this vaccine in young children probably lasts no more than two years and has little or no effectiveness in the immune suppressed child. Thus, it should be obvious to all that the HepB vaccination program is a reprehensible plan by the establishment vaccine to require (or force) vaccinations on all babies, since there is difficulty convincing adults (who are at greatest risk) to get the vaccine. In many hospital settings, on the day of birth, infants are automatically being administered the hepatitis B vaccine with no parental informed consent—since this procedure is now implicitly considered to be a part of the accepted birthing protocol.

The problem with this “plan” is that the vaccine is ineffective by the time the child reaches the age of risk. Now that public health officials in the U.S. have discovered this, the recommendation is that all children have a booster vaccine every two years. Japan, however, no longer requires the Hepatitis B vaccine. In fact, in Japan, where infant vaccinations are voluntary and the schedule generally permits delaying vaccination for up to 8 years, the first-year infant mortality rate in Japan (2.80 per 1,000) is currently less than one-half that of the U.S. (6.30 per 1,000) [175].

The American Academy of Pediatrics and the CDC, the most visible forces behind this vaccine mania, assure parents that giving all the required vaccines in any one office visit is perfectly safe. As we have seen, the scientific “evidence” does not support this policy. Following this policy actually exposes the child to a high concentration of immune-stimulating components that intensely activates the brain’s immune system (microglia) for prolonged periods during the brain’s most active growth period, that is, during the first 2 to 6 years of life and beyond since the maturation and development of the brain continues to a large degree throughout adolescence.

As we have seen, excessive vaccination can result in brain inflammation and brain swelling that can be prolonged, even lasting years, if not decades (as discussed in Vargas *et al.*). This can result in seizures, high pitched crying, severe lethargy, weakness, and behavioral problems, such as agitation, depression, anger, and other autistic behaviors.

In addition, giving the vaccines all at once, exposes the brain to higher levels of potentially neurotoxic aluminum as proven by the radiolabeled aluminum study discussed previously [111]. If a person were to rigorously follow the current recommended vaccine guidelines and live to be 80 years of age, they could receive more than 100 vaccinations in their lifetime. Because of the way the vaccines are given, this would not allow the essential shutting down of the brain’s microglial cells.

One of the effects of chronic microglial activation, other than brain inflammation, is an elevation in brain glutamate levels. Studies have shown this condition can lead to chronic neurodegeneration and is suspected as a common mechanism associated with neuropathic viruses, such as the measles and borna disease viruses [158-160]. In fact, blocking certain glutamate receptors can prevent brain damage by the measles virus, as well as other viruses [158]. We also know that the prognosis of spinal meningitis can be determined by the spinal fluid glutamate levels, with high levels having the worst prognosis [161]. Studies of autistic children have also shown elevated glutamate

levels in their blood and spinal fluid.

Because excitotoxicity plays such an important role in autism, parents of autistic children should avoid feeding their children foods containing excitotoxic additives, such as MSG, hydrolyzed protein, vegetable protein extracts, soy protein or soy protein isolates, natural flavoring, yeast enzymes, etc. There are many disguised names for high glutamate food additives. A recent study indicates that there is an interaction between certain food dyes and glutamate and aspartame that enhances neurotoxicity significantly.

Autistic children should also avoid immune suppressing oils, such as the omega-6 oils (corn, soybean, safflower, sunflower and peanut oils). As stated, people in this country eat 50-times the amount of this immune-suppressing oil than necessary for health.

While omega-3 oils are healthy, the EPA component is significantly immune suppressing and as a result, high intakes should be avoided. Suppressed lymphocyte function (NK cells) is associated with high intake of EPA [162]. It is the DHA component that has most of the beneficial effects, especially as regards brain repair and inflammation reduction [163]. DHA also inhibits excitotoxicity. Because the autistic child has intense brain inflammation, a combination of EPA and DHA is preferable, with a lower content of EPA (no more than 250 mg).

Milk and milk products should be avoided and foods containing gliadin and gluten should also be avoided. Soy foods are also responsible for a significant number of food allergies and are very high in glutamate, fluoride, and manganese. Fluoride should be avoided, especially in drinking water. Water is also a significant source of aluminum in the diet (it is added as a clarifying agent) and in fluoridated water the fluoride complexes with aluminum to form the highly neurotoxic fluoroaluminum compound. The greatest dietary sources of aluminum are biscuits, pancakes, black tea, and baked goods made with aluminum-containing baking powder.

Low magnesium intake, which is common in the United States, is associated with higher degrees of inflammation in the body and lower glutathione levels. Magnesium deficiency enhances excitotoxicity, because it is a natural modulator of the NMDA glutamate receptor. Low intakes of magnesium greatly enhance glutamate receptor sensitivity, worsening excitotoxicity. Low magnesium also lowers brain glutathione levels, which increases brain sensitivity to mercury toxicity. Increasing magnesium levels, reduces inflammation, raises glutathione levels, and reduces excitotoxic sensitivity.

A number of flavonoids are neuroprotective, especially against inflammation and excitotoxicity. These include curcumin, quercetin, ellagic acid, natural vitamin E (mixed tocopherol), epigallocatechin gallate (from white tea), theanine, DHEA, and hesperidin. All are available as supplements and most have a high safety profile.

The live virus vaccines for chickenpox (varicella), measles, mumps, and rubella, pose a special danger in the immunosuppressed child because some of these viruses can take up permanent residence in the body, including the brain. In one study, which examined the tissues of elderly dying of non-infectious causes, live measles virus was found in 45% of the bodies examined and 20% of the brains [164,165]. These measles viruses were highly mutated, meaning they could result in a number of

diseases not normally suspected with natural measles infection.

I have omitted discussions about another major problem—vaccine contamination. Several studies found a high incidence of microorganism contamination in vaccines made by a number of major pharmaceutical companies, with figures as high as 60% of the vaccines being contaminated [94-99]. Bacterial and viral fragments have also been found in a number of vaccines. While vaccine promoters were quick to assure us that these viral fragments *should* cause no problem, research indicates otherwise. In fact, a non-viable viral fragment implanted in microglia and astrocytes in the brain caused the devastating dementia associated with the HIV virus [167,168]. The virus does not infect the brain neurons themselves. The mechanism proposed is an immunological/excitotoxic-induced toxicity, just as we see with repeated vaccination. The same mechanism is seen with a number of viruses, including measles viruses, borna virus, and the herpes virus [168-172].

When brain glial cells or neurons are chronically infected with these viruses (called a persistent viral infection) the smoldering immune/excitotoxic reaction slowly destroys the brain cell connections because the immune system is attempting to destroy the infectious microorganism. Since it can never kill the organism, the destruction (and intense microglial activation) continues for decades, as we saw in the autistic brain [54]. The same can occur with viral fragments, the Lyme disease organism, aluminum and mercury that have accumulated in the brain from either contaminated vaccines or from vaccine additives. And because excessive vaccination, especially with immune-suppressive viruses, can depress proper immune function, children are at a greater risk of developing a persistent viral infection. Likewise, vaccinated children are at greater risk of developing deadly invasive bacterial infections, such as *H. influenza* meningitis, pneumococcal and meningococcal meningitis.

When these infections occur, the vaccine promoters scream that we need more vaccines to protect the children, never admitting that the vaccine program itself is to blame for destroying the lives of these children.

Parents must appreciate that those in positions of authority are, at best, unknowingly lying to them. Most pediatricians think they are doing what is right, because they too are victims of years of propaganda by the CDC and American Academy of Pediatrics. Most pediatricians truly believe what they are telling parents. These pediatricians, however, should wake up and join the fight to bring some sense to insane policy presented as the *Childhood Immunization Schedule*.

For more information on vaccine safety, please read the 2008 book, *The Vaccine Safety Manual*, by Neil Z. Miller [173].

## References

- [1] Money J, *et al.* Autism and autoimmune disease: A family study. *J Autism Child Schizophr* 1971;1:146–60.
- [2] Comi A, *et al.* Familial clustering of autoimmune disorders and evaluation of medical risk factors in autism. *J Child Neurology* 1999;14:388–94.
- [3] Sweetwen TL, *et al.* Increased prevalence of familial autoimmunity in probands with pervasive developmental disorders. *Pediatrics* 2003; 112:420.
- [4] Green LA, *et al.* Maternal autoimmune disease, asthma and allergies, and childhood autism spectrum disorders: a case-control study. *Arch Pediatr* 2005;159:151–7.
- [5] Dalton P *et al.* Maternal antibodies associated with autism and language

- disorders. *Ann Neurol* 2003;53:533–7.
- [6] Singh VK, Rivas WH. Prevalence of serum antibodies to caudate nucleus in autistic children. *Neuroscience Lett* 2004;355:53–6.
- [7] Singh VK *et al.* Antibodies to myelin basic protein in children with autistic behavior. *Brain Behavior Immunol* 1993;7:97–103.
- [8] Singer HS *et al.* Antibrain antibodies in children with autism and their unaffected siblings. *J Neuroimmunol* 2006;178:149–55.
- [9] Singh VK *et al.* Circulating autoantibodies to neural and glial filament proteins in autism. *Pediatr Neurol* 1997;17:88–90.
- [10] el-Fawal HA *et al.* Exposure to methylmercury results in serum autoantibodies to neurotypic and gliotypic proteins. *Neurotoxicology* 1996;17:531–9.
- [11] Havarinasab S *et al.* Immunosuppressive and autoimmune effects of thimerosal in mice. *Toxicol Appl Pharmacol* 2005;204:109–21.
- [12] Hornig M, Chian D, Lipkin WJ. Neurotoxic effect of postnatal thimerosal are mouse strain dependent. *Mol Psychiatry* 2004;9:833–45.
- [13] Tishler M, Shoenfeld Y. Vaccination may be associated with autoimmune disease. *Isr Med Assoc J* 2004;6:430–2.
- [14] Shoenfeld T, Aron-Maor A. Vaccination and autoimmunity-‘vaccinosis’ a dangerous liaison? *J Autoimmunity* 2000;14:1–10.
- [15] Vojdam A *et al.* Antibodies to neuron-specific antigens in children with autism: possible cross-reaction with encephalitogenic proteins from milk, *Chlamydia pneumoniae* and *Streptococcus* group A. *J Neuroimmunol* 2002;129:168–77.
- [16] Lucarelli S *et al.* Food allergy and infantile autism. *Panminerva Med* 1995;37:137–41.
- [17] O’Banion D *et al.* Disruptive behavior: a dietary approach. *J Autism Child Schizophr* 1978;8:325–37.
- [18] Vojdani A *et al.* Immune response to dietary proteins, gliadin and cerebellar peptides in children with autism. *Nutr Neuroscience* 2004; 7: 151–161.
- [19] McGeer PL and McGeer EG. Autotoxicity and Alzheimer Disease. 2000; 57:289–90.
- [20] Malek-Ahmadi P. Cytokines and etiopathogenesis of pervasive developmental disorders. *Med Hypothesis* 2001;56:321–4.
- [21] Weizman A *et al.* Abnormal responses to brain tissue antigen in the syndrome of autism. *Am J Psychiatry* 1982;139:1462–5.
- [22] Lee SC *et al.* Cytokine production by human fetal microglia and astrocytes. Differential induction by liposaccharide and IL-1beta. *J Immunol* 1993;150:2659–67.
- [23] Bauer S *et al.* The neuropoietic cytokine family in development, plasticity, disease and injury. *Nature Reviews/Neuroscience* 2007;8:221–32.
- [24] Boulanger LM, Shatz CJ. Immune signaling in neural development, synaptic plasticity and disease. *Nature Reviews/Neuroscience* 2004;5:521–31.
- [25] Agrawal A *et al.* Thimerosal induces TH2 responses via influencing cytokine secretion by human dendritic cells. *J Leukocyte Biol* 2007;81:1–9.
- [26] Kidd P. Th1/Th2 balance: The hypothesis, its limitations, and implication in health and disease. *Altern Medicine Rev* 2003;8:223–46.
- [27] Martin OC *et al.* Hepatitis B immunization induces higher antibody and memory Th2 responses in new-borns than adults. *Vaccine* 2004;22:511–9.
- [28] Cohly HH, Panja A. Immunologic findings in autism. In *Rev Neurobiol* 2005;71:317–41.
- [29] Singh VK. Plasma increase of interleukin-12 and interferon-gamma. Pathological significance in autism. *J Neuroimmunol* 1996; 66: 143–145.
- [30] Jyonouchi H *et al.* Proinflammatory and regulatory cytokine production associated with innate and adaptive immune responses in children with autism spectrum disorders and developmental regression. *J Neuroimmunol* 2001;120:170–9.
- [31] Pandey RS *et al.* Autoimmune model of schizophrenia with special reference to anti-brain antibodies. *Biol Psychiatry* 1981;16:1123–36.
- [32] Zhang XY *et al.* Elevated interleukin-2, interleukin-6 and interleukin-8 serum levels in neuroleptic-free schizophrenia: association with psychopathology. *Schizophr Res* 2002;57:247–58.
- [33] Turner W *et al.* Measles associated encephalopathy in children with renal transplants. *Am J Transplant* 2006;6:1459–65.
- [34] Larner AJ, Farmer SF. Myelopathy following influenza vaccination in inflammatory disorder treated with chronic immunosuppression. *Eu J Neurol* 2000;7:731–3.
- [35] Kerdlie YM *et al.* Immunosuppression by measles virus: role of viral proteins. *Rev Med Virol* 2006;16:49–63.
- [36] Abernathy RS, Spink WW. Increased susceptibility of mice to bacterial endotoxins induced by pertussis vaccine. *Fed Proc* 1956;15:580.
- [37] Auwaerter PD *et al.* Changes within T-cell receptor V beta subsets in infants following measles vaccinations. *Clin Immunol Immunopathol* 1996;79:163–7.
- [38] Hussey GD *et al.* The effect of Edmonston-Zagreb and Schwartz measles vaccines on immune responses in infants. *J Infect Dis* 1996;173:1320–6.
- [39] Hirsch RL *et al.* Measles virus vaccination of measles seropositive individuals suppresses lymphocyte proliferation and chemotactic factor production. *Clin Immunol Immunopathol* 1981;21:341–50.
- [40] Daum RS *et al.* Decline in serum antibody to the capsule of *Haemophilus influenzae* type b in the immediate postimmunization period. *J Pediatrics* 1989;114:742–7.
- [41] Pukhalsky AL *et al.* Cytokine profile after rubella vaccine inoculation: evidence of the immunosuppressive effect of vaccination. *Mediators Inflammation* 2003; 12: 203–7.
- [42] Miller NZ. *Vaccine Safety Manual: For Concerned Families and Health Practitioners.* New Atlantean Press, NM, 2008.
- [43] Pichichero ME *et al.* Pathogen shifts and changing cure rates for otitis media and tonsillopharyngitis. *Clin Pediatr* 2006;45:493–502.
- [44] Moore MR *et al.* Impact of conjugate vaccine on community wide coverage of nonsusceptible *Streptococcus* in Alaska. *J Inf Dis* 2004; 190:2031–8.
- [45] Pichichero ME, Cary JR. Emergence of a multiresistant serotype 19A pneumococcal strain not included in the 7-valent conjugate vaccine as an otopathogen in children. *JAMA* 2007; 298:1772–8.
- [46] Strunecka A, Patocka J, Blaylock RL *et al.* Fluoride interactions: from molecules to disease. *Current Signal Transduction Therapy* 2007; 2(3):190–213
- [47] Block ML, Zecca L, Hong J-S. Microglia-mediated neurotoxicity: uncovering the molecular mechanisms. *Nature Reviews/Neuroscience* 2007 Sept.;8:57–69.
- [48] Mandu P, Brown GC. Activation of microglial NADPH oxidase is synergistic with glial NOS expression in inducing neuronal death: a dual-key mechanism of inflammatory neurodegeneration. 2005;2:20.
- [49] Cagnin A *et al.* In vivo visualization of activated glia by [11C] (R)-PK11195-PET following herpes encephalitis reveals projected neuronal damage beyond the primary focal lesion. *Brain* 2001;124:2014–27.
- [50] Lemstra AW *et al.* Microglia activation in sepsis: a case-control study. *J Neuroinflamm* 2007;4:4
- [51] Buttini M, Lumonta S, Boddeke HW. Peripheral administration of lipopolysaccharide induces activation of microglial cell in rat brain. *Neurochem Int* 1996;29:25–35.
- [52] Cunningham C *et al.* Central and systemic endotoxin challenges exacerbate the local inflammatory responses and increased neuronal death during chronic neurodegeneration. *J Neurosci* 2005; 25:9275–84.
- [53] Godbout JP *et al.* Exaggerated neuroinflammatory and sickness behavior in aged mice following activation of the peripheral innate immune system. *FASEB J* 2005;19:1329–31.
- [54] Vargas DL *et al.* Neuroglial activation and neuroinflammation in the brain of patients with autism. *Ann Neurol* 2005;57:67–81.
- [55] Blaylock RL. Central role of excitotoxicity in autism. *JANA* 2003;6:7–19.
- [56] Lewine JD *et al.* Magnetoencephalographic patterns of epileptiform activity in children with regressive autism spectrum disorders. *Pediatrics* 1999; 104:405–15.
- [57] Auvin S *et al.* Inflammation exacerbates seizure-induced injury in the immature brain. *Epilepsia* 2007;48: 27–34.
- [58] Rizzi M *et al.* Glia activation and cytokines increased in rat hippocampus by kainic acid-induced status epilepticus during postnatal development. *Neurobiol Dis* 2003;4:94–103.
- [59] Eastman CL *et al.* Increased brain quinolinic acid production in mice infected with hamster neurotropic measles virus. *Exp Neurol* 1994; 125:119–24.
- [60] Heyes MP *et al.* Human microglia convert L-tryptophan into neurotoxin quinolinic acid. *Biochem J* 1996; 320: 595–597.
- [61] Ida T *et al.* Cytokine-induced enhancement of calcium-dependent glutamate release from astrocytes mediated by nitric oxide. *Neurosci Lett* 2008; 432: 232–236.
- [62] Ye GL *et al.* AMPA and NMDA receptor-mediated currents in developing dentate granule cells. *Brain Res Dev Brain Res* 2005; 155: 26–32.
- [63] Menkes JH, Kinsbourne M. Workshop on neurologic complications of pertussis and pertussis vaccinations. *Neuropediatrics* 1990; 21: 171–176.
- [64] Kiviravanta T, Airaksinen EM. Low sodium levels in serum are associated with febrile seizures. *Acta Paediatr* 1995;84:1372–4.

- [65] Bar-Peled O *et al.* Distribution of glutamate transporter subtypes during human brain development. *J Neurochem* 1997;69:2571–80.
- [66] Arauz-Contreras J, Feria-Velasco A. Monosodium-L-glutamate-induced convulsions I. Differences in seizure pattern and duration of effect as a function of age in rats. *Gen Pharmacol* 1984;15:391–5.
- [67] Neil Z. Miller. Vaccines: Are they Really Safe and Effective? A Parent's Guide to Childhood Shots. New Atlantean Press, NM 1999.
- [68] Toga Aw *et al.* Mapping brain maturation. *Trend Neurosci* 2006;29:148–59.
- [69] Gogtay N *et al.* Dynamic mapping of human cortical development during childhood and adolescence. *Proc Natl Acad Sci USA* 2006;101:8174–9.
- [70] Jerigan TL, Tallal P. Late childhood changes in brain morphology observable with MRI. *Dev Med Child Neurol* 1990;32:379–85.
- [71] Maslinska D *et al.* Morphological forms and localizations of microglial cells in the developing human cerebellum. *Folia Neuropathol* 1998;36:145–51.
- [72] Monier A *et al.* Entry and distribution of microglial cells in human embryonic and fetal cerebral cortex. *J Neuropathol Exp Neurol* 2007;66:372–82.
- [73] Schwab JM *et al.* IL-6 is differentially expressed in the developing human fetal brain by microglial cells in zones of neurogenesis. *In J Dev Neurosci* 2001;114:232–41.
- [74] Schlett K. Glutamate as a modulator of embryonic and adult neurogenesis. *Curr Top Med Chem* 2006;6:949–60.
- [75] Kumuro H, Rakic P. Modulation of neuronal migration by NMDA receptors. *Science* 1993;260:95–7.
- [76] Marret S *et al.* Arrest of neuronal migration by excitatory amino acids in hamster developing brain. *Proc Natl Acad Sci USA* 1996;93:15463–8.
- [77] Aarum J *et al.* Migration and differentiation of neural precursor cells can be directed by microglia. *Proc Natl Acad Sci USA* 2003;100:15983–8.
- [78] Ekdahl CT *et al.* Inflammation is detrimental for neurogenesis in adult brains. *Proc Natl Acad Sci USA* 2003;100:13632–5.
- [79] Chao CC *et al.* Tumor necrosis factor- $\alpha$  potentiates glutamate neurotoxicity in human fetal cell cultures. *Dev Neurosci* 1994;16:172–9.
- [80] Kemper TL *et al.* Neuropathology of infantile autism. *J Neuropathology Exp Neurol* 1998;57:645–52.
- [81] Bauman MI, Kemper TL. The neuropathology of autism spectrum disorders: What have we learned? *Novartis Foundation Symp* 2003;251:112–22.
- [82] Bauman M, Kemper TL. Developmental cerebellar abnormalities: a consistent finding in early infantile autism. *Neurology* 1986;36(Suppl 1):190.
- [83] Courchesne E. Brainstem cerebellar and limbic neuroanatomical abnormalities in autism. *Curr Opin Neurobiol* 1997;7:269–78.
- [84] Buller KM, Day TA. Systemic administration of interleukin 1 $\beta$  activates select populations of central amygdala afferents. *J Comp Neurol* 2002;452:288–96.
- [85] Taylor DL *et al.* Stimulation of microglial metabotropic glutamate receptor mGlu2 triggers tumor necrosis factor- $\alpha$ -induced neurotoxicity in concert with microglial-derived Fas ligand. *J Neurosci* 2005;25:2952–64.
- [86] Rothwell NJ. Cytokines—killers in the brain? *J Physiol* 1999;514:3–17.
- [87] Samland H *et al.* Profound increase in sensitivity to glutamatergic—but not to cholinergic agonist-induced seizures in transgenic mice with astrocytes production of IL-6. *J Neurosci Res* 2003;73:176–87.
- [88] Bernardino L *et al.* Modulator effects of interleukin-1 $\beta$  and Tumor necrosis factor- $\alpha$  on AMPA-induced excitotoxicity in mouse organotypic hippocampal slice cultures. *J Neurosci* 2005;25:6734–44.
- [89] Allan SM *et al.* Interleukin-1 and neuronal injury. *Nature Reviews/Immunol* 2005;5:629–40.
- [90] Burka SL *et al.* Maternal cytokine levels during pregnancy and adult psychosis. *Brain Behav Immunol* 2001;15:411–20.
- [91] Brown AS *et al.* Elevated maternal interleukin-8 levels and risk of schizophrenia in adult offspring. *Am J Psychiatry* 2004;161:889–95.
- [92] Ganguli R *et al.* Autoimmunity in schizophrenia: a review of recent findings. *Ann Med* 1993;25:489–96.
- [93] Schori H *et al.* Severe immunodeficiency has opposite effects in neuronal survival in glutamate-susceptible and resistant mice: adverse effect of B-cells. *J Immunol* 2002;169:2861–5.
- [94] Cutrone R *et al.* Some oral polio vaccines were contaminated with infectious SV-40 after 1961. *Can Res* 2005;65:10273–9.
- [95] Harasawa R, Tomiyama T. Evidence of pestivirus RNA in human virus vaccines. *J Clin Microbiol* 1994;32:1604–5.
- [96] Geier M *et al.* Endotoxins in commercial vaccines. *Appl Environ Microbiol* 1978;36:445–9.
- [97] Giangaspero M *et al.* Genotypes of pestivirus RNA detected in live virus vaccines for human use. *J vet Med Sci* 2001;63:723–33.
- [98] Potts BJ *et al.* Possible role of pestivirus in microcephaly. *Lancet* 1987;1:972–3.
- [99] Johnson JA, Heneine W. Characteristics of endogenous avian leukosis virus in chicken embryonic fibroblast substrates used in production of measles and mumps vaccine. *J Virol* 2001;75:3605–12.
- [100] Gherardi RK *et al.* Macrophagic myofasciitis lesion assess long-term persistence of vaccine-derived aluminum hydroxide in muscle. *Brain* 2001;124:1821–31.
- [101] Authier F-J *et al.* Central nervous system disease in patients with macrophagic myofasciitis. *Brain* 2001;124:974–83.
- [102] Bonnefont-Rousselot D *et al.* Blood oxidative status in patients with macrophagic myofasciitis. *Biomed Pharmacol* 2004;58:516–9.
- [103] Good PF *et al.* Selective accumulation of aluminum and iron in the neurofibrillary tangles of Alzheimer's disease: a laser microprobe (LAMMA) study. *Ann Neurol* 1992;31:286–92.
- [104] Esparza JL *et al.* Aluminum-induced pro-oxidant effect in rats: protective role of exogenous melatonin. *J Pineal Res* 2003;35:32–9.
- [105] Yokel RA *et al.* The distribution of aluminum into and out of the brain. *J Inorg Biochem* 1999;76:127–32.
- [106] Campbell A *et al.* Chronic exposure to aluminum in drinking water increases inflammatory parameters selectively in the brain. *J Neuroscience Res* 2004;75:565–72.
- [107] Bishop NJ *et al.* Aluminum neurotoxicity in preterm infants receiving intravenous feeding solutions. *N Engl J Med* 1997;336:1557–61.
- [108] Campbell A. Inflammation, neurodegenerative disease, and environmental exposures. *Ann NY Acad Sci* 2004;1035:117–32.
- [109] Shirabe T *et al.* Autopsy case of aluminum encephalopathy. *Neuropathology* 2002;22:206–10.
- [110] Armstrong RA *et al.* Hypothesis: Is Alzheimer's disease a metal-induced immune disorder. *Neurodegeneration* 1995;4:107–11.
- [111] Flarend RE, Hem SL, White JL, Elmore D, Suckow MA, Rudy AC, Dandashi EA. In vivo absorption of aluminum-containing vaccine adjuvants using <sup>26</sup>Al. *Vaccine* 1997 Aug.-Sept.;15(12-13):1314–8.
- [112] Platt B *et al.* Aluminum toxicity in the rat brain: histochemical and immunocytochemical evidence. *Brain Res Bull* 2001;55:257–67.
- [113] Brookes N. Specificity and reliability of the inhibition by HgCl<sub>2</sub> of glutamate transport in astrocytes cultures. *J Neurochem* 1988;50:1117–22.
- [114] Vahter ME *et al.* Demethylation of methylmercury in different brain sites of *Macaca fascicularis* monkeys during long-term subclinical methylmercury exposure. *Toxicol Appl Pharmacol* 1995;134:273–84.
- [115] Charleston JS *et al.* Changes in the number of astrocytes and microglia in the thalamus of the monkey *Macaca fascicularis* following long-term subclinical methylmercury exposure. *Neurotoxicology* 1996;17:127–38.
- [116] Charleston JS *et al.* Increase in the number of reactive glia in the visual cortex of *Macaca fascicularis* following subclinical long-term methylmercury exposure. *Toxicol Appl Pharmacol* 1994;129:196–206.
- [117] Burbacher TM *et al.* Comparison of blood and brain mercury levels in infant monkeys exposed to methylmercury or vaccines containing thimerosal. *Environ Health Perspect* 2005;113:1015–21.
- [118] Mutkus L *et al.* Methylmercury alters the in vitro uptake of glutamate and GLAST and GLT-1 transfected mutant CHO-K1 cells. *Biol Trace Elem Res* 2005;107:231–45.
- [119] Aschner M *et al.* Methylmercury alters glutamate transport in astrocytes. *Neurochem Int* 2000;37:199–206.
- [120] Kim P, Choi BH. Selective inhibitors of glutamate uptake by mercury in cultured mouse astrocytes. *Yonsi Med J* 1995;36:299–305.
- [121] Kugler P, Schleyer V. Developmental expression of glutamate transporters and glutamate dehydrogenase in astrocytes of the postnatal rat hippocampus. *Hippocampus* 2004;14:975–85.
- [122] Yel L *et al.* Thimerosal induces neuronal cell apoptosis by causing cytochrome C and apoptosis-inducing factor release from mitochondria. *In J Mol Med* 2005;16:971–7.
- [123] Humphrey ML *et al.* Mitochondria mediated thimerosal-induced apoptosis in a human neuroblastoma cell line (SK-N-SH). *Neurotoxicology* 2005;26:407–16.
- [124] Henneberry RC. The role of neuronal energy in neurotoxicity of excitatory amino acids. *Neurobiol Aging* 1989;10:611–3.
- [125] Zeevalk GD *et al.* Excitotoxicity and oxidative stress during inhibition of energy metabolism. *Dev Neurosci* 1998;20:444–5.
- [126] Haley BE. The relationship of the toxic effects of mercury to exacerbation of the medical condition classified as Alzheimer's disease. *Medical Veritas* 2007 Nov;4(2):1484–98.

- [127] Sun YM *et al.* Sex-specific impairment in sexual and ingestive behaviors of monosodium glutamate-treated rats. *Physiol Behavior* 1991;50:873–80.
- [128] Yang S-H *et al.* Testosterone increases neurotoxicity of glutamate in vitro and ischemia-reperfusion injury in an animal model. *J Appl Physiol* 2002; 92:195–201.
- [129] Estrada M *et al.* Elevated testosterone induces apoptosis in neuronal cells. *J Biol Chem* 2006;281:25492–501.
- [130] Aschner M *et al.* Involvement of glutamate and reactive oxygen species in methyl mercury neurotoxicity. *Braz J Med Biol Res* 2007;40:285–91.
- [131] Allen JM *et al.* The consequences of methylmercury exposure on interactive function between astrocytes and neurons. *Neurotoxicology* 2002; 23:755–9.
- [132] Lautermilch NJ, Spitzer NC. Regulation of calcineurin by growth cone calcium waves controls neurite extension. *J Neurosci* 2000; 20: 315–325.
- [133] Estrada M *et al.* Ca<sup>2+</sup> oscillations induced by testosterone enhance neurite outgrowth. *J Cell Sci* 2005;119:733–43.
- [134] Geier DA, Geier MR. A clinical trial of combined anti-estrogen and anti-heavy metal therapy in autistic disorder. *Neuroendocrinol Lett* 2006; 27:833–8.
- [135] Baker AE *et al.* Estrogen modulates microglial inflammatory mediator production via interactions with estrogen receptor  $\beta$ . *Endocrinology* 2004; 145:5021–32.
- [136] Wakefield AJ *et al.* Ileal-lymphoid nodular hyperplasia, non-specific colitis, and pervasive developmental disorders in children. *Lancet* 1998; 351:637–41.
- [137] Ashwood P, Wakefield AJ. Immune activation of peripheral blood and mucosal CD3+ lymphocyte cytokine profiles in children with autism and gastrointestinal systems. *J Neuroimmunol* 2006;173:126–34.
- [138] Horvath K *et al.* Gastrointestinal abnormalities in children with autistic disorder. *J Pediatr* 1999;135:559–63.
- [139] Afzal N *et al.* Constipation with acquired megacolon in children with autism. *Pediatrics* 2003;112:939–42.
- [140] Feingold SM *et al.* Gastrointestinal microflora studies in late-onset autism. *Clin Infect Dis* 2002;35:S6–16.
- [141] Vojdani A *et al.* Antibodies to neuron-specific antigens in children with autism: possible cross-reaction with encephalitogenic proteins from milk, Chlamydia pneumonia and Streptococcus group A. *J Neuroimmunol* 2002;129:168–77.
- [142] Lucarelli S *et al.* Food allergy and infantile autism. *Panminerva Med* 1995;37:137–41.
- [143] Knivsberg AM *et al.* A randomized, controlled study of dietary intervention in autistic syndrome. *Nutri Neurosci* 2002;5:251–61.
- [144] Vojdani A *et al.* Immune response to dietary proteins, gliadin and cerebellar peptides with autism. *Nutri Neurosci* 2004;7:151–61.
- [145] Whitely P *et al.* A gluten-free diet as an intervention for autism and associated disorders: preliminary findings. *Autism* 1999; m3:45–65.
- [146] Bushara KO. Neurologic presentation of celiac disease. *Gastroenterology* 2005;128: S92–7.
- [147] Kinney HC *et al.* Degeneration of the central nervous system associated with celiac disease. *J Neurol Sci* 1982;53:9–22.
- [148] DeSantis A *et al.* Schizophrenia symptoms and SPECT abnormalities in a coeliac patient: regression after gluten-free diet. *J Intern Med* 1997; 242:421–3.
- [149] Beyenberg S *et al.* Chronic progressive leukoencephalopathy in adult celiac disease. *Neurology* 1998;50:820–2.
- [150] Burk K *et al.* Sporadic cerebellar ataxia associated with gluten sensitivity. *Brain* 2001; 124:1013–9.
- [151] Hu WT *et al.* Cognitive impairment and celiac disease. *Arch Neurol* 2006;63:1440–6.
- [152] Wakefield AJ *et al.* Review article: The concept of entero-colonic encephalopathy, autism and opioid receptor ligands. *Aliment Pharmacol Ther* 2002;16:663–74.
- [153] Peterson PK *et al.* The opioid-cytokine connection. *J Neuroimmunology* 1998;83:63–9.
- [154] Zhu L *et al.* Enhancing effect of beta-endorphins on glutamate toxicity. *Zhongguo Yao Li Xue Bao* 1998;19:108–11.
- [155] Blaylock RL. Interaction of cytokines, excitotoxins, and reactive nitrogen and oxygen species in autism spectrum disorders. *JANA* 2003;6:21–35.
- [156] Rao S, Ali U. Systemic fungal infections in neonates. *J Postgrad Med* 2005;51(suppl 1):S27–9.
- [157] Sandler RH *et al.* Short term benefit from oral vancomycin treatment of regressive-onset autism. *J Child Neurol* 2000;15:429–35.
- [158] Anderson T *et al.* NMDA-receptor antagonist prevents measles virus-induced neurodegeneration. *Eur J Neurosci* 1991;3:66–71.
- [159] Eastman CL *et al.* Increased brain quinolinic acid production in mice infected with a hamster neurotropic measles virus. *Exp Neurol* 1994; 125:119–24.
- [160] Raslet A *et al.* Borrelia burgdorferi induces inflammatory mediator production by murine microglia. *J Neuroimmunol* 2002;130:22–31.
- [161] Ma W *et al.* Elevated cerebrospinal fluid levels of glutamate in children with bacterial meningitis as a predictor of the development of seizures or other adverse outcomes. *Pediatr Crit Care Med* 2003;4:170–5.
- [162] Zhao Y *et al.* Eicosapentaenoic acid prevents LPS-induced TNF- expression by preventing NF $\kappa$ B activation. *J Amer Coll Nutr* 2004;23:71–8.
- [163] Weldon SM *et al.* Docosahexaenoic acid induces an anti-inflammatory profile in liposaccharide-stimulated THP-1 macrophage mice more effectively than eicosapentaenoic acid. *J Nutr Biochem* 2007;18:250–8.
- [164] Katayama Y *et al.* Detection of measles virus nucleoprotein mRNA in autopsied brain tissue. *J Gen Virol* 1995;76:3201–4.
- [165] Katayama Y *et al.* Detection of measles virus mRNA from autopsied human tissues. *J Clin Microbiol* 1998;36:299–301.
- [166] Hult B *et al.* Neurobiology of HIV. *Int Rev Psychology* 2008;20:3–13.
- [167] Gonzales-Sarano F, Martin-Garcia J. The neuropathogenesis of AIDS. *Nat Rev Immunol* 2005;5:69–81.
- [168] Rubin SA *et al.* Viral teratogenesis: brain developmental damage associated with maturation state at time of infection. *Brain Dev Rev* 1999; 112:237–44.
- [169] Lellouch-Tubiana A *et al.* Immunocytochemical characterization of long-term persistent immune activation in human brain after herpes simplex encephalitis. *Neuropathology Appl Neurobiol* 2000;26:285–94.
- [170] Ovanesov MV *et al.* Activation of microglia by Borna disease virus infection: In vitro study. *J Virol* 2006;80:12141–8.
- [171] Volmer R *et al.* Borna disease virus infection impairs synaptic plasticity. *J Virol* 2007;81:8833–7.
- [172] De la Torre JC. Borna virus and the brain. *J Infect Dis* 2002; 186:(suppl. 2):S241–7.
- [173] Miller NZ. *The Vaccine Safety Manual for Concerned Families and Health Practitioners.* New Atlantean Press, Santa Fe, NM 2008.
- [174] Sugita M. The biological half-time of heavy metals. The existence of a third, “slowest” component. *Int Arch Occup Environ Health* 1978;41(1):25–40.
- [175] <https://www.cia.gov/library/publications/the-world-factbook/rankorder/2091rank.html>, last updated 15 April 2008; visited 19 April 2008.