OPPOSE HB 87 and SB 135 Minor Consent Bill

I am testifying in OPPOSITION to HB 87 and SB 135: Minor Consent Bill

HB87 and SB135;

- Would strip Parental Rights and remove our ability to decide if our children get vaccines and some medical procedures.

As parents with children who have had adverse reactions, we know that all drugs and biologics carry some level of risk. These risks should be evaluated on each personal health history and the potential reward.

We parents are the best health advocates for our children – not the state – not the school - and not big pharma. We know their health history better than they do. My girls, and yours, at 16 may not know their full medical history to know if they are contraindicated, leading to an adverse reaction against HPV or Flu vaccines.

In Maryland, childhood and teen vaccination rates are already the highest in the Nation at between 90 and 99% compliance - except for the HPV vaccine. So, what is the big rush? Follow the money. This bill would result in significantly increasing damage from the All Risk / No Reward cash cow HPV vaccine.

Did you know that In the US, drug companies are free of liability for any childhood vaccine injury or death; by law, they can't be sued.

Consider just three of many reasons this bill should be stopped;

Safety Risks: All drugs and biologics carry some level of risk. For example, Gardasil and Gardasil 9 HPV vaccines have been shown to actually increase a girl's risk of cervical cancer by over 44% if she is HPV positive at the time of inoculation. A teenager is not going to know this. There is no screening. Parents need to be involved in these decisions.

Your child will not be told by the health care provider that the incidence of cervical cancer in the US is very low, **less than 1%**. The onset of such cancers are usually in **adults over 50**, so would occur long after any antibodies produced by the vaccine were gone.

Parental Rights: The minor and their parents must investigate the possible risks relative to prior health history and individual susceptibility. At 16 years, most minors will not know their history and could easily be subject to peer pressure or coercion without the parent's knowledge or consent.

Did you know the Gardasil vaccine was fast-tracked by the FDA and was approved in just six months. Because it was fast-tracked, there are no long-term safety studies, no studies about the interaction with other vaccines given at the same time, and no studies about the interaction with birth control pills. There is also no evidence that it actually prevents cervical cancer.

Cost Ineffective: For example, HPV vaccines are marketed to prevent Cervical Cancer with no evidence that they actually do! Meanwhile, slow growing cervical cancer comprises <1% of all cancers, usually in adults past 50 and is preventable with pap screening. There is < ¼ of 1% lifetime risk of dying of cervical

cancer. At about $750 per vaccine regiment, it costs $75million to vaccinate each 100,000 girls in the still unproven chance of preventing one death from cervical cancer. Who is going to pay for this? We all are.

Recent polls show that Big Pharma the most hated industry in the world. They outspend Oil & Gas 2 to 1 in lobbying. Why would they need to do that?

Why do people see drug companies' proven **malevolence** through repeated court case discovery but then accept the lie of **benevolence** when it comes to childhood vaccines where they have been immune from liability since 1986?

Parents need to be involved with health decisions. I believe Humans have a right to **Informed Consent, do you?** If people evaluate the risk and still want it – have at it!

This law would pave the way for drug companies to influence and coerce minors through more "incentives" and "giveaways" to get vaccines like HPV vaccines - presenting serious health risks for very little benefit.

I urge you to advocate for your children and oppose this bill.

Cliff McDougall

2309 Ridge Road

Reisterstown, MD 21136

410.935.2547

National Childhood Vaccine Injury Act

The 1986 Act

National Childhood Vaccine Injury Act

Consequences?

1. No incentive to conduct safety studies
2. Liability free market of 78 million American children
3. Strong incentive to develop more vaccines

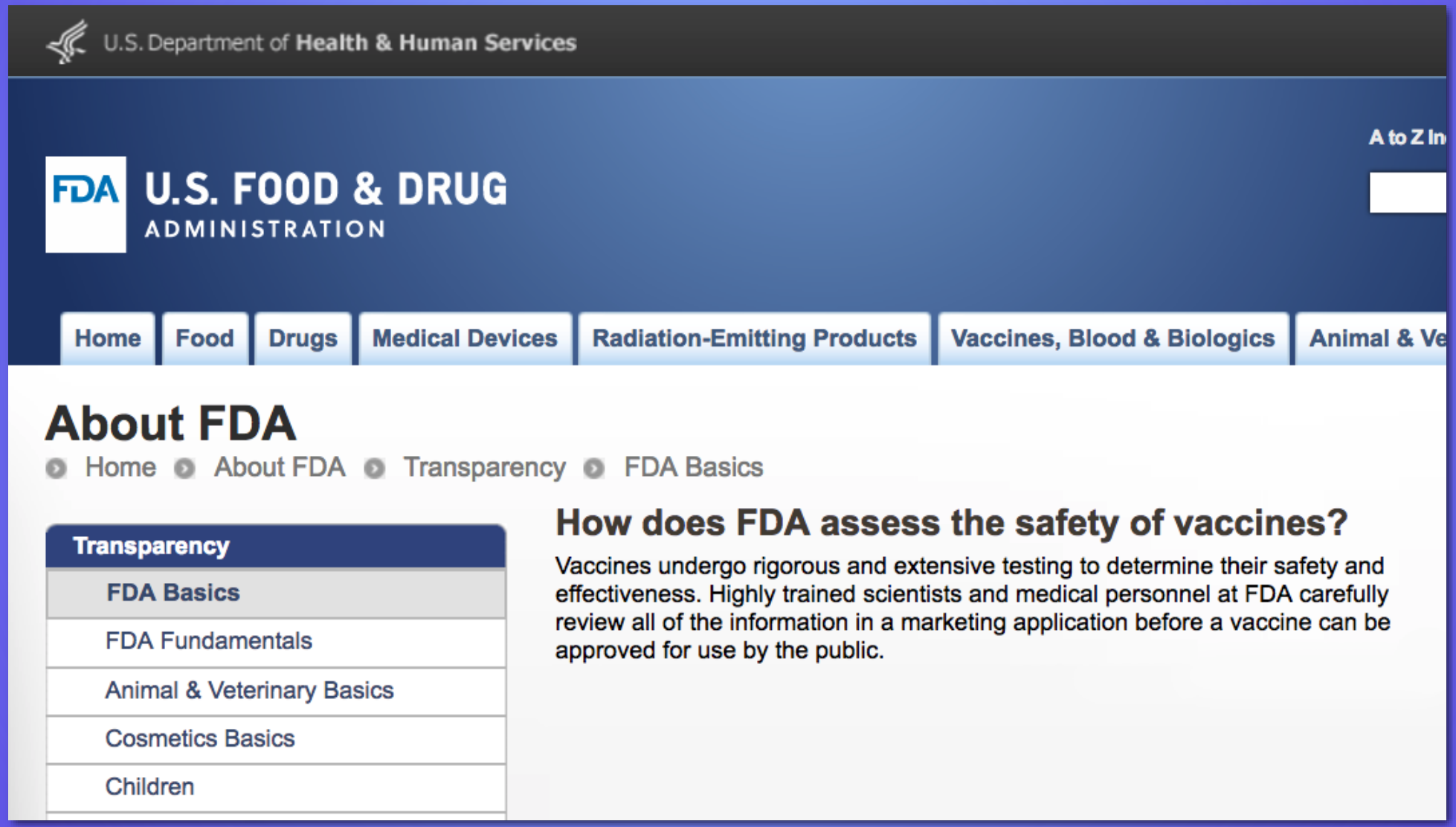
National Childhood Vaccine Injury Act

Who is responsible for vaccine safety?

HHS

FDA CDC HRSA NIH

How Vaccines are **Licensed**, Recommended, Promoted, Defended



1986
11 Vaccines

- DTaP (2 months)
- Polio (2 months)
- Hib (2 months)
- PCV (2 months)
- Rotavirus (2 months)
- DTaP (4 months)
- Polio (4 months)
- Hib (4 months)
- PCV (4 months)
- Rotavirus (4 months)

2017
54 Vaccines

- Influenza (pregnancy)
- Tdap (pregnancy)
- Hepatitis B (one day)
- Hepatitis B (one month)
- DTaP (2 months)
- Polio (2 months)
- Hib (2 months)
- PCV (2 months)
- Rotavirus (2 months)
- DTaP (4 months)
- Polio (4 months)
- Hib (4 months)
- PCV (4 months)
- Rotavirus (4 months)
- DTaP (6 months)
- Polio (6 months)
- Hepatitis B (6 months)
- Hib (6 months)
- PCV (6 months)
- Rotavirus (6 months)
- Influenza (6 months)
- MMR (12 months)
- Varicella (12 months)
- Hib (12 months)
- Hepatitis A (12 months)
- PCV (12 months)
- DTaP (15 months)
- Hepatitis A (18 months)
- Influenza (18 months)
- Influenza (2 years)
- Influenza (3 years)
- Influenza (4 years)
- DTaP (4 years)
- MMR (4 years)
- Rubella (4 years)
- Varicella (4 years)
- Influenza (5 years)
- Influenza (6 years)
- Influenza (7 years)
- Influenza (8 years)
- Influenza (9 years)
- Influenza (10 years)
- HPV (11 years)
- Influenza (11 years)
- TDaP (11 years)
- HPV (11 ½ years)
- Influenza (12 years)
- HPV (12 years)
- Influenza (13 years)
- Influenza (14 years)
- Influenza (15 years)
- MCV (16 years)
- Influenza (16 years)
- MenB (16 years)
- Influenza (17 years)
- Influenza (18 years)

Near Future
100s of Vaccines

- Influenza (pregnancy)
- Tdap (pregnancy)
- Hepatitis B (one day)
- Hepatitis B (one month)
- DTaP (2 months)
- Polio (2 months)
- Hib (2 months)
- PCV (2 months)
- Rotavirus (2 months)
- DTaP (4 months)
- Polio (4 months)
- Hib (4 months)
- PCV (4 months)
- Rotavirus (4 months)
- DTaP (6 months)
- Polio (6 months)
- Hepatitis B (6 months)
- Hib (6 months)
- PCV (6 months)
- Rotavirus (6 months)
- Influenza (6 months)
- MMR (12 months)
- Varicella (12 months)
- Hib (12 months)
- Hepatitis A (12 months)
- PCV (12 months)
- DTaP (15 months)
- Influenza (pregnancy)
- Tdap (pregnancy)
- Hepatitis B (one day)
- Hepatitis B (one month)
- DTaP (2 months)
- Polio (2 months)
- Hib (2 months)
- PCV (2 months)
- Rotavirus (2 months)
- DTaP (4 months)
- Polio (4 months)
- Hib (4 months)
- PCV (4 months)
- Rotavirus (4 months)
- DTaP (6 months)
- Polio (6 months)
- Hepatitis B (6 months)
- Hib (6 months)
- PCV (6 months)
- Rotavirus (6 months)
- Influenza (6 months)
- MMR (12 months)
- Varicella (12 months)
- Hib (12 months)
- Hepatitis A (12 months)
- PCV (12 months)
- DTaP (15 months)
- Hepatitis A (18 months)
- Influenza (18 months)
- Influenza (2 years)
- Influenza (3 years)
- Influenza (4 years)
- DTaP (4 years)
- MMR (4 years)
- Rubella (4 years)
- Varicella (4 years)
- Influenza (5 years)
- Influenza (6 years)
- Influenza (7 years)
- Influenza (8 years)
- Influenza (9 years)
- Influenza (10 years)
- HPV (11 years)
- Tdap (pregnancy)
- Hepatitis B (one day)
- Hepatitis B (one month)
- DTaP (2 months)
- Polio (2 months)
- Hib (2 months)
- PCV (2 months)
- Rotavirus (2 months)
- DTaP (4 months)
- Polio (4 months)
- Hib (4 months)
- PCV (4 months)
- Rotavirus (4 months)
- DTaP (6 months)
- Polio (6 months)
- Hepatitis B (6 months)
- Hib (6 months)
- PCV (6 months)
- Rotavirus (6 months)
- Influenza (6 months)
- MMR (12 months)
- Varicella (12 months)
- Hib (12 months)
- Hepatitis A (12 months)
- PCV (12 months)
- DTaP (15 months)
- Hepatitis A (18 months)
- Influenza (18 months)
- Influenza (2 years)
- Tdap (pregnancy)
- Hepatitis B (one day)
- Hepatitis B (one month)
- DTaP (2 months)
- Polio (2 months)
- Hib (2 months)
- PCV (2 months)
- Rotavirus (2 months)
- DTaP (4 months)
- Polio (4 months)
- Hib (4 months)
- PCV (4 months)
- Rotavirus (4 months)
- DTaP (6 months)
- Polio (6 months)
- Hepatitis B (6 months)
- Hib (6 months)
- PCV (6 months)
- Rotavirus (6 months)
- Influenza (6 months)
- MMR (12 months)
- Varicella (12 months)
- Hib (12 months)
- Hepatitis A (12 months)
- PCV (12 months)
- DTaP (15 months)
- Hepatitis A (18 months)
- Influenza (18 months)
- Influenza (2 years)

2013- Pharma 271 Under Dev
2017- 2300 Clinical Trials

According to the CDC:

1 in 6 children has a developmental disability*

ADD	Hearing Loss	Seizures
ADHD	Learning Disability	Stammering
Autism	Mental Disability	Stuttering

According to an HHS Funded Publication:

54% of children have chronic illness†

Anxiety Problems	Chronic Ear Infections	Environmental Allergies
Asthma	Depression	Epilepsy
Behavioral Problems	Diabetes	Rheumatoid Arthritis
Bone and Muscle Disorders	Digestive Allergies	Seizure Disorder

*Source: https://www.cdc.gov/ncbddd/developmentaldisabilities/about.html

†Bethel et. al, 2011, *A National and State Profile of Leading Health Problems and Health Care Quality for US Children: Key Insurance Disparities and Across-State Variations*, Academic Pediatrics.

Number of Childhood Vaccine Injections Administered

11
1986 schedule

54
2017 schedule

Childhood Chronic Illness & Developmental Disability Prevalence

12.8%*
1986 prevalence

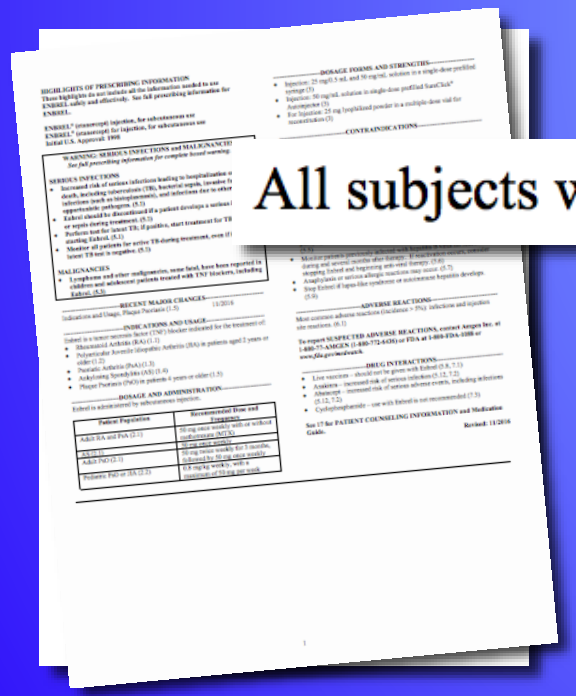
54%†
2011 prevalence

* Cleave et. al, 2010, *Dynamics of Obesity and Chronic Health Conditions Among Children and Youth*, JAMA.

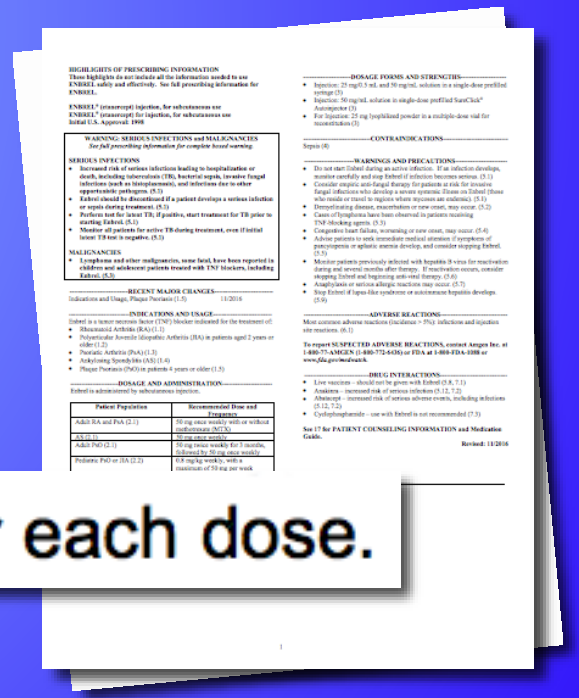
†Bethel et. al, 2011, *A National and State Profile of Leading Health Problems and Health Care Quality for US Children: Key Insurance Disparities and Across-State Variations*, Academic Pediatrics.

How Vaccines are Licensed, Recommended, Promoted, Defended

Recommended Age (First Dose)	Vaccine/ Manufacturer	Safety Review Period Prior to Licensure	Subject Group	Placebo Group
1 Day Old	Hep-B (Engerix)/ GlaxoSmithKline	4 Days	Hep-B	No Placebo
1 Day Old	Hep-B (Recombivax)/ Merck	5 Days	Hep-B	No Placebo



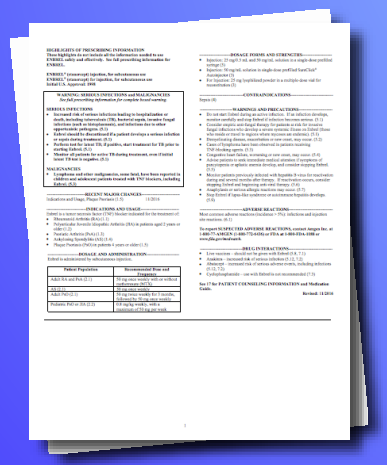
All subjects were monitored for 4 days post-administration.



monitored for 5 days after each dose.

How Vaccines are Licensed, Recommended, Promoted, Defended

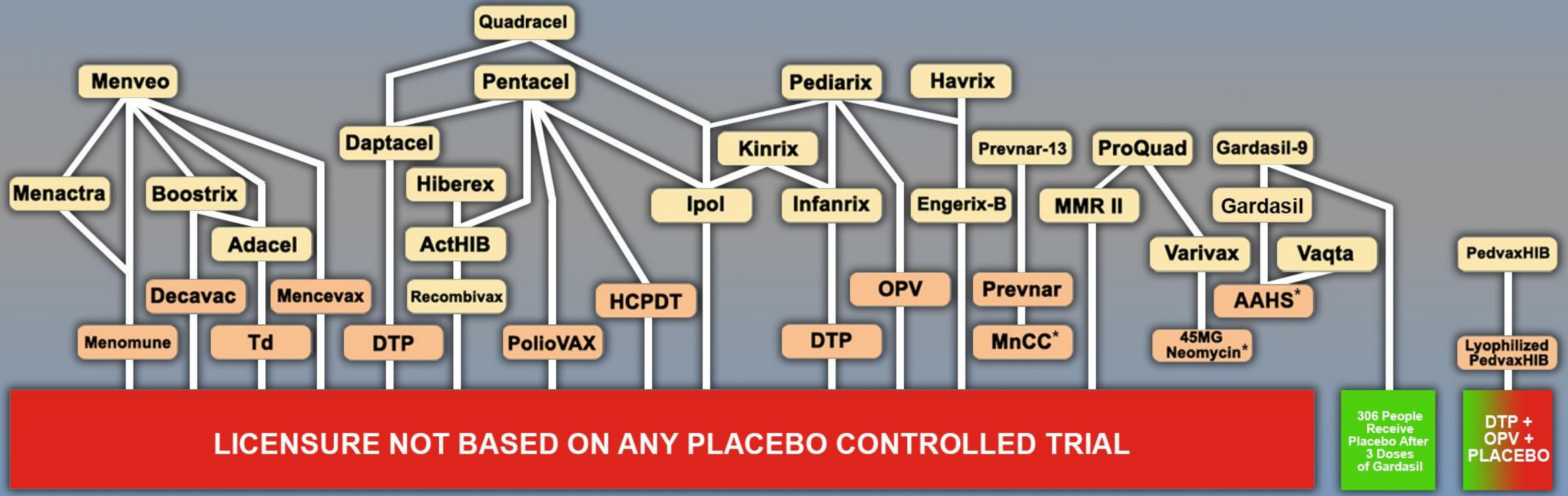
Recommended Age (First Dose)	Vaccine/ Manufacturer	Safety Review Period Prior to Licensure	Subject Group	Placebo Group
1 Day Old	Hep-B (Engerix)/ GlaxoSmithKline	4 Days	Hep-B	No Placebo
1 Day Old	Hep-B (Recombivax)/ Merck	5 Days	Hep-B	No Placebo
2 Month Old	Polio (PVI- Monkey Kidney)/ Sanofi Pasteur	48 hours	Polio + DTP	DTP



48 hours post-vaccination.

Because IPV was given in a different site but concurrently with Diphtheria and Tetanus Toxoids and Pertussis Vaccine Adsorbed (DTP), these systemic reactions could not be attributed to a specific vaccine.

PLACEBO PYRAMID SCHEME



Source: www.icandecide.org

Vaccination Status of Measles Outbreak Cases

- 82 (62%) cases had immunization status verified
  - 57 (70%) of these were unvaccinated
    - ✓ 28 (49%) personal beliefs exemptions
    - ✓ 16 (28%) too young
    - ✓ 2 (4%) missed dose/alternative schedule
    - ✓ 11 (19%) unknown reasons
  - Of the 25 (31%) who were vaccinated:
    - ✓ 10 (12%) had one dose of MMR vaccine
    - ✓ 13 (16%) had two doses of MMR vaccine
    - ✓ 2 (2%) had three doses of MMR vaccine

31% Vaccine Failure

- 49 (38%) of the 131 cases did not have immunization records; 48 of these were adults - 20 of whom self-reported being vaccinated; many others were unsure

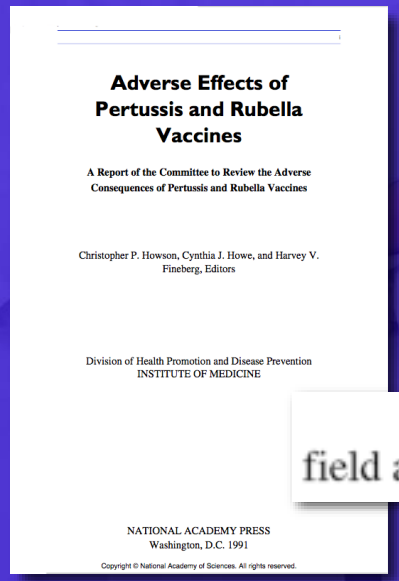
Lack of Vaccine Safety Science

Institute of Medicine Reports on Vaccine Safety

Year of IOM Report	Vaccines Reviewed	# of Conditions Studied	Literature Supports Causation	Liter Rej Caus
1991	DTP	22	6	

Literature Inadequate to Accept or Reject Causation

12



Adverse Effects of Pertussis and Rubella Vaccines

A Report of the Committee to Review the Adverse Consequences of Pertussis and Rubella Vaccines

Christopher P. Howson, Cynthia J. Howe, and Harvey V. Fineberg, Editors

Division of Health Promotion and Disease Prevention
INSTITUTE OF MEDICINE

NATIONAL ACADEMY PRESS
Washington, D.C. 1991

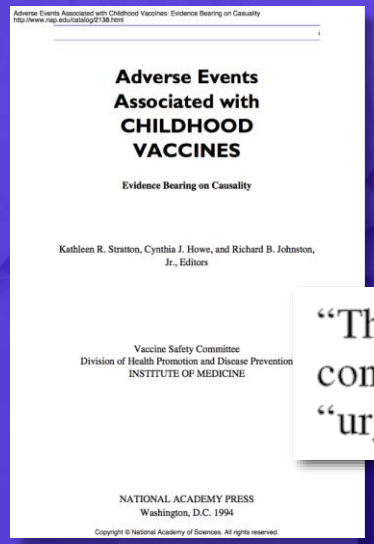
Aseptic meningitis; Chronic neurologic damage; Learning disabilities and attention-deficit disorder; Hemolytic anemia; Juvenile diabetes; Guillain-Barre syndrome; Erythema multiforme; Peripheral mononeuropathy; Radiculoneuritis and other neuropathies; Thrombocytopenia; Thrombocytopenic purpura

"If research capacity and accomplishment in this field are not improved, future reviews of vaccine safety will be similarly handicapped."

Lack of Vaccine Safety Science

Institute of Medicine Reports on Vaccine Safety

Year of IOM Report	Vaccines Reviewed	# of Conditions Studied	Literature Supports Causation	Literature Rejects Causation	Literature Inadequate to Accept or Reject Causation
1994	DT, MM, Hep-B & Hib	54	12	4	38



Adverse Events Associated with CHILDHOOD VACCINES

Evidence Bearing on Causality

Kathleen R. Stratton, Cynthia J. Howe, and Richard B. Johnston, Jr., Editors

Vaccine Safety Committee
Division of Health Promotion and Disease Prevention
INSTITUTE OF MEDICINE

NATIONAL ACADEMY PRESS
Washington, D.C. 1994

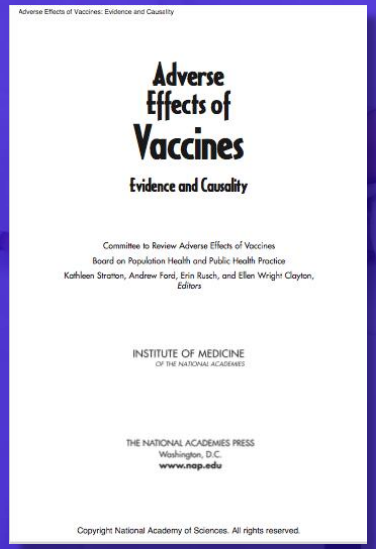
A partial list of the 38 conditions: Demyelinating diseases of the central nervous system, Sterility, Arthritis, Neuropathy, Residual seizure disorder, Transverse myelitis, Sensorineural deafness, Optic neuritis, Aseptic meningitis, Insulin-dependent diabetes mellitus, SIDS

"The lack of adequate data regarding many of the adverse events under study was of major concern to the committe." The IOM report somberly states it "regrets... this uncertainty" and "urge[s] that more definitive research be done."

Lack of Vaccine Safety Science

Institute of Medicine Reports on Vaccine Safety

Year of IOM Report	Vaccines Reviewed	# of Conditions Studied	Literature Supports Causation	Literature Rejects Causation	Literature Inadequate to Accept or Reject Causation
2011	Varicella, T, Hep-B, MMR	155	16	5	134



A partial list of the 134 conditions: Encephalitis, Encephalopathy, Infantile Spasms, Afebrile Seizures, Seizures, Cerebellar Antaxia, Anataxia, Autism, Acute Disseminated Encephalomyelitis, Transverse Myelitis, Optic Neuritis, Neuromyelitis Optica, Multiple Sclerosis, Guillain-Barre Syndrome, Chronic Inflammatory Demyelinating Polyneuropathy, Brachial Neuritis, Amyotrophic Lateral Sclerosis, Small Fiber Neuropathy, Chronic Urticaria, Erythema Nodosum, Systemic Lupus Erythematosus, Polyarteritis Nodosa, Psoriatic Arthritis, Reactive Arthritis, Rheumatoid Arthritis, Juvenile Idiopathic Arthritis, Arthralgia, Autoimmune Hepatitis, Stroke, Chronic Headache, Fibromyalgia, Sudden Infant Death Syndrome, Hearing Loss, Thrombocytopenia, Immune Thrombocytopenic Purpura

1986 Act: Vaccine Adverse Events Reporting System (VAERS)

5,911,700

In 2016, VAERS received ~~59,117~~ reports including:

43,200 ~~432~~ deaths,
109,100 ~~1,091~~ permanent disabilities,
413,200 ~~4,132~~ hospitalizations, and
1,028,400 ~~10,284~~ emergency room visits.

"fewer than 1% of adverse events are reported"
(Source: Report Funded by HHS)

"Former FDA Commissioner David A. Kessler has estimated that VAERS reports currently represent only a fraction of the serious adverse events."
(Source: U.S. Congressional Report)

1. The voluntary consent of the human subject is absolutely essential.

This means that the person involved should have legal capacity to give consent; should be so situated as to be able to exercise free power of choice, without the intervention of any element of force, fraud, deceit, duress, over-reaching, or other ulterior form of constraint or coercion; and should have sufficient knowledge and comprehension of the elements of the subject matter involved, as to enable him to make an understanding and enlightened decision. This latter

AUTISM & ALUMINUM ADJUVANTS IN VACCINES

How Aluminum Adjuvants in Vaccines Can Cause Autism

Published: August 18, 2017 (Version 1.0)

The Centers for Disease Control (CDC) asserts that vaccines and vaccine ingredients have been disproven as potential causes of autism. Statements by the CDC are generic and encompass all vaccines and vaccine ingredients. For example, the CDC states:

> *"Vaccines Do Not Cause Autism" "There is no link between vaccines and autism." "...no links have been found between any vaccine ingredients and autism spectrum disorder." (CDC website, August 2017)*

These statements are not supported by available science. The CDC's evidence supporting these statements is limited to the MMR vaccine (Taylor 2014), thimerosal preservative (Taylor 2014) and vaccine antigen exposure (DeStefano 2013).

Dr. Frank DeStefano of the CDC's Immunization Safety Office is co-author of a paper (Glanz 2015) which states:

> *"To date, there have been no population-based studies specifically designed to evaluate associations between clinically meaningful outcomes and non-antigen ingredients, other than thimerosal."*

This statement applies to, among other vaccine ingredients, aluminum adjuvant. Studies of MMR vaccine cannot be used as evidence of safety for other vaccines, for example vaccines that contain aluminum adjuvant. The overly-broad, generic assertions that no vaccines and no ingredients cause autism are thus not supported by scientific evidence. In fact, the CDC statements are contradicted by a large, consistent and growing body of scientific evidence, including:

1) studies showing neurotoxic and neuroinflammatory effects (e.g. microglial activation) from dosages of aluminum adjuvants lower than or approximately equal to dosages received by infants according to the CDC vaccine schedule (Crepeaux 2017, Petrik 2007, Shaw 2013, Shaw 2009);

2) studies linking vaccines to immune activation brain injury (Zerbo 2016, Li 2015);

3) studies showing that early-life immune activation is a causal factor in autism and other neurodevelopmental disorders and mental illnesses (e.g. schizophrenia) (Meyer 2009, Deverman 2009, Estes 2016, Kneusel 2014, Careaga 2017, Meyer 2014).

The accumulating evidence indicates that vaccine-induced immune activation, and aluminum adjuvants in particular, may cause mental illnesses and neurodevelopmental disorders, including autism.

In this paper, we present scientific evidence that aluminum adjuvants can cause autism and other brain injuries. Also, we explain why the studies allegedly supporting the safety of aluminum adjuvants do not show safety for adverse neurological outcomes.

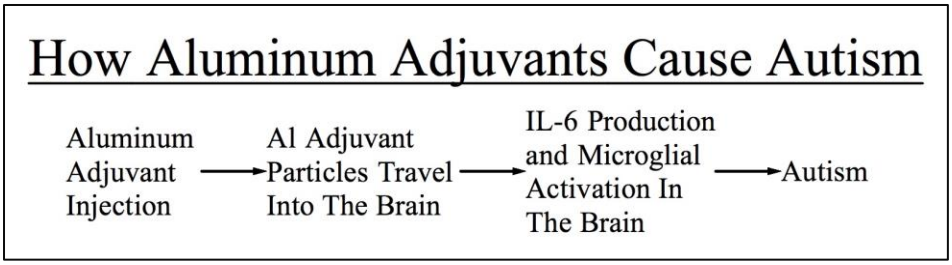


Fig 1: Proposed mechanism for how aluminum adjuvants cause autism. Each step is supported by replicated scientific studies.

Immune Activation: A Cause of Autism and Mental Illness

The term "immune activation" describes the activation of the cellular components of the immune system. The developing brain can be injured by immune activation, with life-long consequences (Meyer 2009, Deverman 2009, Estes 2016, Kneusel 2014, Careaga 2017, Meyer 2014). Immune activation injury is linked to autism, schizophrenia, depression and other mental illnesses or neurodevelopmental disorders. Immune activation effects on the brain are mediated by immune system signaling molecules, especially cytokines (Estes 2016, Meyer 2014, Smith 2007, Choi 2016, Pineda 2013).

It is generally accepted that immune activation (e.g., from infection) during pregnancy is a risk factor for autism and schizophrenia in the offspring (Ciaranello 1995, Atladottir 2010, Brown 2012). The intensity and duration of immune activation and cytokine expression appear to be important factors influencing autism risk (Meyer 2014). Intense immune activation is associated with greater risk of autism (Careaga 2017, Atladottir 2010). Chronic inflammation is associated with greater risk of autism (Jones 2016, Zerbo 2014). However, there is no evidence that short-duration, low-intensity immune activation resulting from common childhood illnesses increase autism risk. Timing of immune activation in relation to stages of brain development is also an important factor (Meyer 2006, Meyer 2009).

Animal experiments have tested the effects of immune activation during pregnancy and postnatally on the development of offspring (Meyer 2009, Deverman 2009, Estes 2016, Kneusel 2014, Careaga 2017, Meyer 2014). In these experiments, pregnant animals (mice, rats and monkeys) or neonates are injected with a non-infectious immune activating substance such as "poly-IC" (which mimics a viral infection) or lipopolysaccharide (LPS, which mimics a bacterial infection). These substances cause immune system activation without infection. They induce fever and cytokine production and can have substantial effects on brain development if activation is sufficiently intense or prolonged and if exposure occurs during vulnerable developmental stages.

Immune activation has been demonstrated in mice to cause the three core behavioral symptoms of autism: decreased socialization and communication, and increased repetitive behaviors (Malkova 2012). Immune activation has also been shown to cause neuropathology (Weir 2015) and behavioral abnormalities in monkeys that resemble behaviors in human schizophrenia and autism (Bauman 2014, Machado 2015). See Fig. 2.

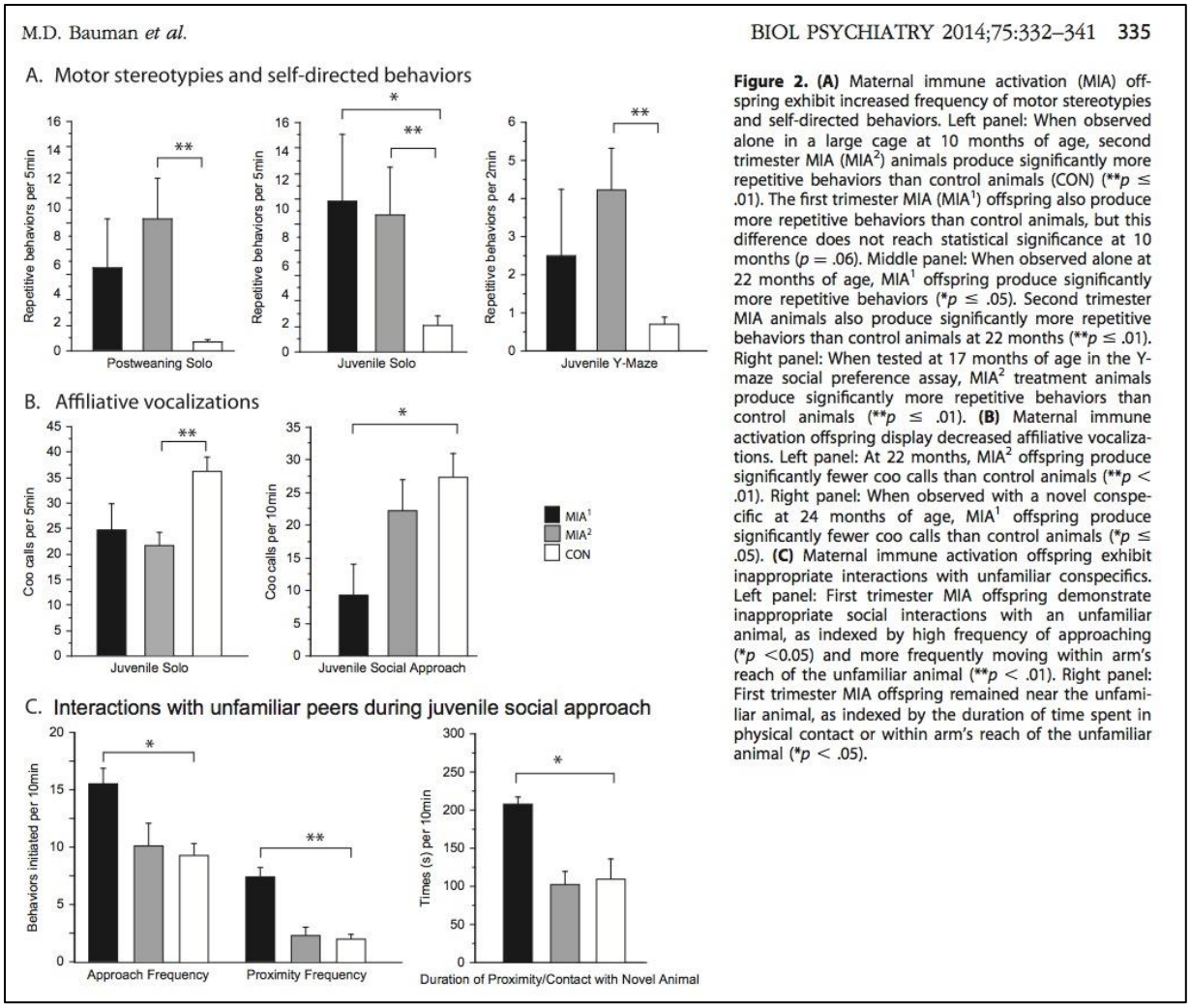


Figure 2. (A) Maternal immune activation (MIA) offspring exhibit increased frequency of motor stereotypies and self-directed behaviors. Left panel: When observed alone in a large cage at 10 months of age, second trimester MIA (MIA^2) animals produce significantly more repetitive behaviors than control animals (CON) (**$p \leq .01$). The first trimester MIA (MIA^1) offspring also produce more repetitive behaviors than control animals, but this difference does not reach statistical significance at 10 months ($p = .06$). Middle panel: When observed alone at 22 months of age, MIA^1 offspring produce significantly more repetitive behaviors (*$p \leq .05$). Second trimester MIA animals also produce significantly more repetitive behaviors than control animals at 22 months (**$p \leq .01$). Right panel: When tested at 17 months of age in the Y-maze social preference assay, MIA^2 treatment animals produce significantly more repetitive behaviors than control animals (**$p \leq .01$). **(B)** Maternal immune activation offspring display decreased affiliative vocalizations. Left panel: At 22 months, MIA^2 offspring produce significantly fewer coo calls than control animals (**$p < .01$). Right panel: When observed with a novel conspecific at 24 months of age, MIA^1 offspring produce significantly fewer coo calls than control animals (*$p \leq .05$). **(C)** Maternal immune activation offspring exhibit inappropriate interactions with unfamiliar conspecifics. Left panel: First trimester MIA offspring demonstrate inappropriate social interactions with an unfamiliar animal, as indexed by high frequency of approaching (*$p < 0.05$) and more frequently moving within arm's reach of the unfamiliar animal (**$p < .01$). Right panel: First trimester MIA offspring remained near the unfamiliar animal, as indexed by the duration of time spent in physical contact or within arm's reach of the unfamiliar animal (*$p < .05$).

Fig 2: Maternal immune activation in monkeys caused behavioral abnormalities in juvenile offspring resembling behaviors in both autism and schizophrenia. MIA1 (Black)= first trimester immune activation; MIA2 (grey) 2nd trimester immune activation; CON (white) saline control. From Bauman et al. 2014

Immune activation also causes non-behavioral effects associated with human autism (citations here link immune activation with these effects):

1) reduction in Purkinje cells (Shi 2009);
2) mitochondrial dysfunction (Giulivi 2013);
3) increase in brain volume (from IL-6 exposure, Wei 2012(b)) and neuron density in the brain (Smith 2012);
4) long term chronic brain inflammation (Garay 2012); and
5) microbiome disruption (dysbiosis) (Hsiao 2013).

These non-behavioral similarities further support the relevance of the immune activation models to human autism. The non-behavioral (e.g., physiological) effects of immune activation have been reviewed (Labouesse 2015).

The cytokines interleukin-6 (IL-6) and interleukin-17a (IL-17) have been identified as mediating the behavioral effects of immune activation (Smith 2007, Malkova 2012, Choi 2016, Pineda 2013, Wei 2012(a), Wei 2013, Parker-Athill 2010, Wei 2016). The IL-6 findings have been replicated by different researchers using a variety of experimental methods. For example, in an experiment with

poly-IC, abnormal behavior is almost completely prevented by simultaneous administration of IL-6-blocking antibody (Smith 2007, Pineda 2013). Injection of IL-6 by itself causes abnormal behavior that closely matches behavior resulting from poly-IC immune activation (Smith 2007). Inhibition of IL-6 signaling in a genetic autism model (BTBR mice) normalized social and repetitive behavior (Wei 2016). These results demonstrate that IL-6 is responsible for causing abnormal autism-like behavior.

The Patterson laboratory at CalTech was the first to report that IL-6 is responsible for causing the autism-like behavioral effects of immune activation (Smith 2007). Two papers from this research group state:

> *"IL-6 is central to the process by which maternal immune activation causes long-term behavioral alterations in the offspring." (Smith 2007)*

> *"…blocking IL-6 prevents >90% of the changes seen in offspring of poly(I:C)-injected females, showing that gene expression changes, as well as behavioral changes, are normalized by eliminating IL-6 from the maternal immune response." (Smith 2007)*

> *"IL-6 is necessary and sufficient to mediate these effects since the effects…are prevented by injection of pregnant mice with poly-IC combined with an anti-IL-6 antibody, and are mimicked by a single maternal injection of IL-6." (Garay 2013)*

Brain exposure to elevated IL-6 by engineered virus showed that IL-6 exposure, initiated after birth, caused autism-like behaviors (Wei 2012(a)). The Wei 2012(a) paper states:

> *"We demonstrated that IL-6 is an important mediator of autism-like behaviors. Mice with an elevated IL-6 in brain developed autism-like behaviors, including impaired cognition ability, deficits in learning, abnormal anxiety-like trait and habituation, as well as a decreased social interaction initiated at later stages. These findings suggest that an IL-6 elevation in the brain could modulate certain pathological alterations and contribute to the development of autism." (Wei 2012(a))*

More recent evidence shows that IL-17 acts downstream of IL-6 to cause autism-like behavioral abnormalities and atypical cortical development in mice (Choi 2016). Blocking either IL-6 or IL-17 prevents the autism-like behavior; an injection of IL-17 by itself causes the autism-like behavior (Choi 2016). IL-6 is known to induce IL-17 by promoting the development of Th17 cells which produce IL-17.

Immune activation animal models appear to be valid models for human neurological/psychiatric disorders, including autism (Estes 2016, Careaga 2017, Meyer 2014). The Estes 2016 review argues for the validity of the immune activation models to humans:

> *"These MIA (maternal immune activation) animal models meet all of the criteria required for validity for a disease model: They mimic a known disease-related risk factor (construct validity), they exhibit a wide range of disease-related symptoms (face validity), and they can be used to predict the efficacy of treatments (predictive validity)." (Estes 2016)*

Evidence suggests a mediating role for IL-6 and IL-17 in human autism. For example, IL-6 is significantly elevated in the cerebellum in human autism (Wei 2011) and is highly elevated in some brain regions of some autistic individuals (Vargas 2005). Treatment of human autistics with the anti-inflammatory flavonoid luteolin improves autistic behaviors in the individuals that also experience a decline in IL-6 blood levels (Tsilioni 2015). This result is consistent with a causal role for IL-6 in human autism. Also, IL-17 is elevated in human autism (Akintunde 2015, Al-Ayadhi

2012, Suzuki 2011). Vitamin D reduces IL-17 production (Bruce 2011, Wobke 2014, Drozdenko 2014) and improves autistic behaviors in humans (Saad 2016, Jia 2015). The vitamin D findings are consistent with a causal role for IL-17 in human autism.

IL-6 functioning appears to be similar or identical in mice and humans. No mouse-human differences in IL-6 functioning are described in a 2004 review (Mestas 2004). IL-6 functioning is quite conserved across species (Brown 2014). Central nervous system development in rodents and humans is governed by the same principles (Brown 2014). Hence, the fact that IL-6 causes autism-like behavioral abnormalities in animal models deserves a presumption of validity to humans.

Immune activation is a risk factor for autism, schizophrenia and other neurological/psychiatric disorders. The cytokines IL-6 and IL-17 are responsible for mediating the autism-like behavioral effects of immune activation in the animal models. The available evidence supports a causal role for IL-6 and IL-17 in human autism.

Maternal vs. Postnatal Immune Activation

The timing of immune activation is an important factor influencing effects on the brain. The developing brain is vulnerable to immune activation injury; the mature, adult brain is apparently not nearly as vulnerable. Sensitivity to immune activation likely declines as the brain matures (Meyer 2014, Meyer 2007).

In most immune activation experiments, the offspring are exposed to immune activation during gestation (by stimulating the maternal immune system). In contrast, most vaccines are administered postnatally. This raises the question of whether postnatal immune activation can have similar effects on the brain as maternal immune activation. Diverse evidence indicates that the brain can be adversely affected by postnatal immune activation. Postnatal immune activation experiments, human case reports, and consideration of brain development timelines suggest that the human brain is vulnerable to immune activation injury for years after birth.

In the maternal immune activation experiments, inflammatory signaling and some cytokines (e.g. IL-6) traverse the placenta into the fetus. Consequently, immune activation in the mother causes immune activation and elevated cytokines in the fetus, and in the fetal brain (Oskvig 2012, Ghiani 2011).

Postnatal immune activation can have adverse neurological effects, including increased seizure susceptibility (Chen 2013, Galic 2008), learning and memory deficits (Harre 2008), and an increase in excitatory synapse formation (Shen 2016). Seizure disorders, learning and memory dysfunction, and elevated excitatory signaling are associated with autism.

Elevated IL-6 in the brain in the postnatal period causes neuronal circuitry imbalance and mediates autism-like behaviors in mice (Wei 2012(a)). The circuitry imbalance observed in Wei 2012(a) was an excess of excitatory synapses and a deficit of inhibitory synapses. See Fig. 3. Excessive excitatory signaling is observed in human autism (Robertson 2016, Freyberg 2015). In fact, an imbalance between excitatory and inhibitory signaling (towards excess excitation) has been posited as a central characteristic of autism (Robertson 2016, Freyberg 2015).

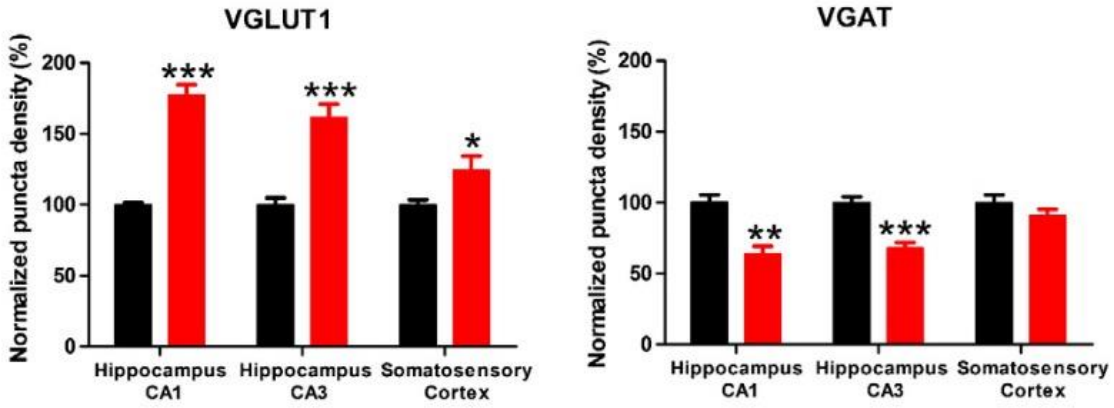


Fig 3: Elevation of IL-6 in the brains of mice (initiated shortly after birth) caused an increase in excitatory synapses (VGLUT1) and a decrease in inhibitory synapses (VGAT). Excessive excitatory signaling is observed in human autism. Red=Elevated IL-6; Black=Control. VGLUT1=excitatory synapses; VGAT=inhibitory synapses. *P<0.05, **P<0.01 and ***P <0.001. Adapted from Wei et al 2012(a).

In a maternal immune activation experiment with mice (Coiro 2015), autism-relevant behavior and dendritic spine abnormalities (relevant to autism and schizophrenia) were ameliorated by administering an anti-inflammatory drug postnatally. The drug was started at birth and continued for 2 weeks, which roughly corresponds to age 2 in humans (Semple 2013). This result indicates that brain development is affected by postnatal inflammation, at times corresponding to when vaccines are given to humans.

Several case reports describe previously-healthy children that displayed sudden-onset autistic behavior during or subsequent to infection in the brain. All the cases had signs of intense brain inflammation. Here are brief descriptions:

Delong 1981: describes 3 children, ages 5, 7 and 11 with full-blown autistic behavior associated with brain inflammation. Brain inflammation was presumed in two cases and confirmed in one. The 5 and 7 year olds recovered completely, and the 11-year recovered partially.

Marques 2014: describes a previously healthy 32-month-old girl that suffered autistic regression from a viral central nervous system infection with associated brain inflammation.

Ghaziuddin 2002: describes a previously healthy 11-year-old boy that suffered permanent autistic regression after sudden onset herpes brain infection with associated brain inflammation.

Gillberg 1986: describes a previously healthy 14-year-old girl with permanent autistic regression from herpes brain infection with associated brain inflammation.

The most parsimonious explanation for these cases is that autistic behavior resulted from intense inflammation and cytokine production in the brain. Accordingly, these cases indicate that the human brain remains vulnerable to immune activation injury well into childhood, though the vulnerability almost certainly decreases with maturation. The susceptibility of older children to inflammation-induced autistic behavior strongly suggests that younger infants, of 0-2 years of age, are also vulnerable. It is not reasonable to claim, and there is no evidence to suggest, that the age range of 0-2 years (when most vaccines are given) is uniquely resistant to immune activation

injury. All the available evidence indicates the opposite.

The immune activation experiments and case reports are consistent and indicate that immune activation and elevated cytokines in the postnatal period can cause brain injury.

The next critical question to consider is whether vaccines can cause immune activation and elevated cytokines in the brain.

Postnatal Vaccination Affects Brain Development in Animal Model

The first study to test the effect of postnatal vaccination on brain development was published in 2015 (Li 2015). In this experiment, neonatal rats were administered bacillus calmette-guerin (BCG) vaccine, hepatitis B (HBV) vaccine or a combination (BCG+HBV) timed to imitate human infant vaccination schedules. BCG and HBV vaccines produced opposite effects on the brain. Specifically, BCG enhanced synaptic plasticity and long-term potentiation (LTP, the basis for learning and memory); HBV inhibited synaptic plasticity and LTP. BCG and HBV vaccines also caused opposite changes in some synapse protein levels.

HBV vaccine (but not BCG vaccine) increased IL-6 gene expression in the brain; increased gene expression likely indicates an elevation in brain IL-6. The HBV vaccine contains aluminum adjuvant, and the BCG does not contain aluminum adjuvant. Hence, the aluminum adjuvant may be the ingredient responsible for the elevated IL-6 gene expression. See Fig. 4.

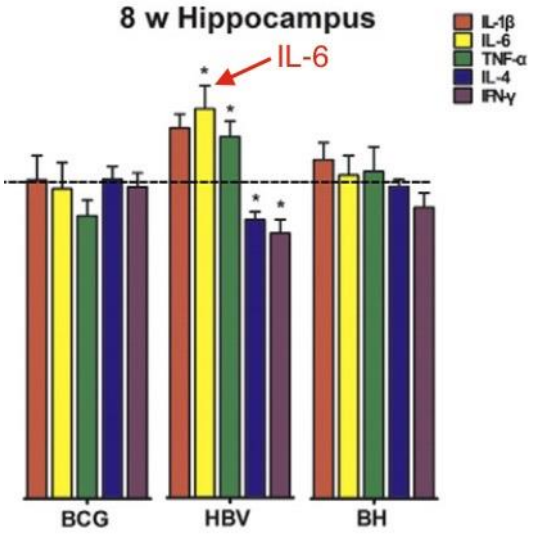


Fig. 4: Hepatitis B vaccine, but not BCG vaccine, increased IL-6 gene expression in the brain at 8 weeks after neonatal vaccination. Hepatitis B vaccine contains aluminum adjuvant; BCG vaccine does not. Elevated IL-6 causes autism-like behaviors in animal models. *P<0.05 Adapted from Li et al 2015.

The Li et al study showed that the vaccines caused other changes in the brain, including 1) changes in long-term potentiation (LTP) (Hep B decreased LTP), 2) changes in dendritic spines, and 3) changes in synapse protein expression. Changes in synapse proteins and dendritic spines have been observed in human brain disorders.

Li et al. attribute the brain effects to changes in cytokine levels and immune polarization (Th1/Th2 polarization) induced by the vaccines. Aluminum adjuvants cause

Th2 polarization. Li et al. state that the results suggest vaccines can interact by way of immune activation effects:

> *"…our data suggested that combinations of different vaccines can mutually interact (enhance or counteract). The mechanism of synaptic plasticity modulation through neonatal BCG/HBV vaccination may be via systemic Th1/Th2 bias accompanied by a specific profile of cytokines and neurotrophins in the brain." (Li 2015)*

Li 2015 demonstrates that vaccines affect brain development by an immune activation mechanism. Further, since aluminum adjuvants induce Th2 activation and long term Th2 polarization, the Li 2015 results suggest that all aluminum-adjuvanted vaccines may cause adverse effects similar to the HBV vaccine. Accordingly, the Li 2015 results suggest that studies showing that immune activation causes neurological/psychiatric disorders are relevant to vaccine adverse effects.

Vaccines Are Given During Synaptogenesis

Another way to answer the question of brain vulnerability to immune activation is to consider the types of brain development processes occurring when vaccines are administered. Vaccines are given primarily in the first 18 months after birth. The human brain undergoes intense and rapid development during this period. Synaptogenesis (formation of synapse connections between neurons) is especially intense in this period.

The vulnerability of the developing brain to immune activation is apparently related to the specific types of brain development processes occurring (Tau 2010, Meyer 2006, Meyer 2007). Such processes include migration (movement of neurons to final locations in the brain), adhesion (formation of chemical-mechanical attachments between brain cells), and synaptogenesis (formation of synapse connections between neurons), among others (neurogenesis, gliogenesis, myelination etc).

Cytokines affect brain development processes. For example, elevated IL-6 affects migration, adhesion and synaptogenesis (Wei 2011). Elevated IL-6 in the postnatal period promotes an excess of excitatory synapses and a deficit of inhibitory synapses, and mediates autism-like behaviors (Wei 2012(a)).

In humans, a dramatic increase in synaptogenesis begins around the time of birth, and continues until about age 3 (Huttenlocher 1997, Tau 2010, Stiles 2010, Semple 2013). Vaccines are administered during this intense synaptogenesis. See Figs. 5-6. Elevated brain IL-6 induced by vaccination during synaptogenesis may cause an excitatory-inhibitory imbalance, towards excitation. An excitatory imbalance has been observed in human autism (Robertson 2016, Freyberg 2015).

Synaptogenesis tapers off through childhood and adolescence. This fact may explain why some older children and teens can suffer autistic regression after intense brain inflammation, but apparently become less vulnerable to immune activation brain injury with age.

Intense synaptogenesis occurs at ages 0-18 months, when many vaccines are administered. Consequently, vaccines may adversely impact synaptogenesis if they induce inflammation or IL-6 in the brain.

The timing of brain development processes in humans supports the idea that the human brain is vulnerable to immune activation and cytokines in the first few years after birth, when vaccines are administered. Disruption of synaptogenesis by vaccine-induced immune activation is a particular concern.

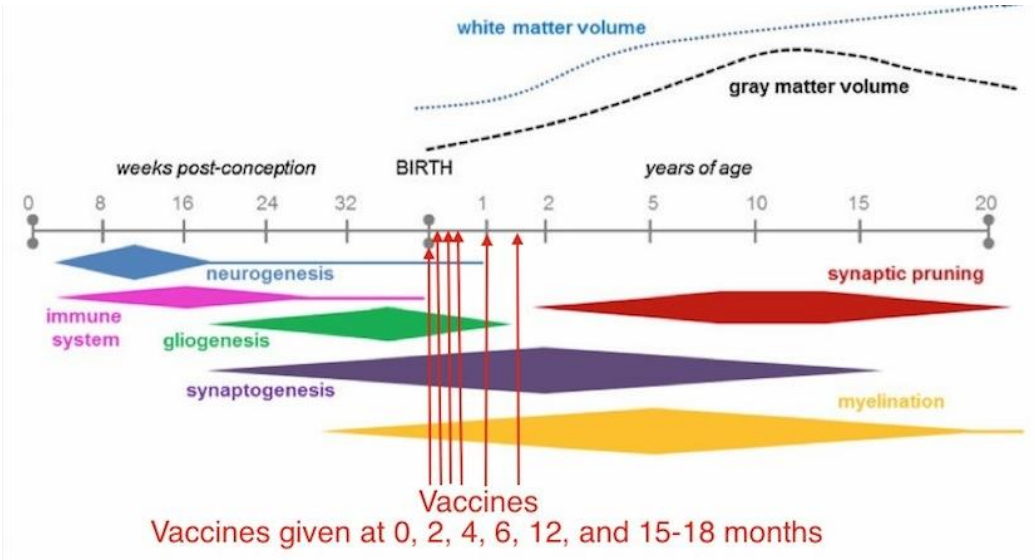


Fig. 5: Timeline of specific brain developmental processes in humans. Synaptogenesis is most intense during the first couple years of life, when vaccines are administered. Timing of vaccination according to the CDC vaccine schedule is shown. Elevated IL-6 during synaptogenesis may cause an excitatory-inhibitory synapse imbalance, towards excitation. Adapted from Semple 2013.

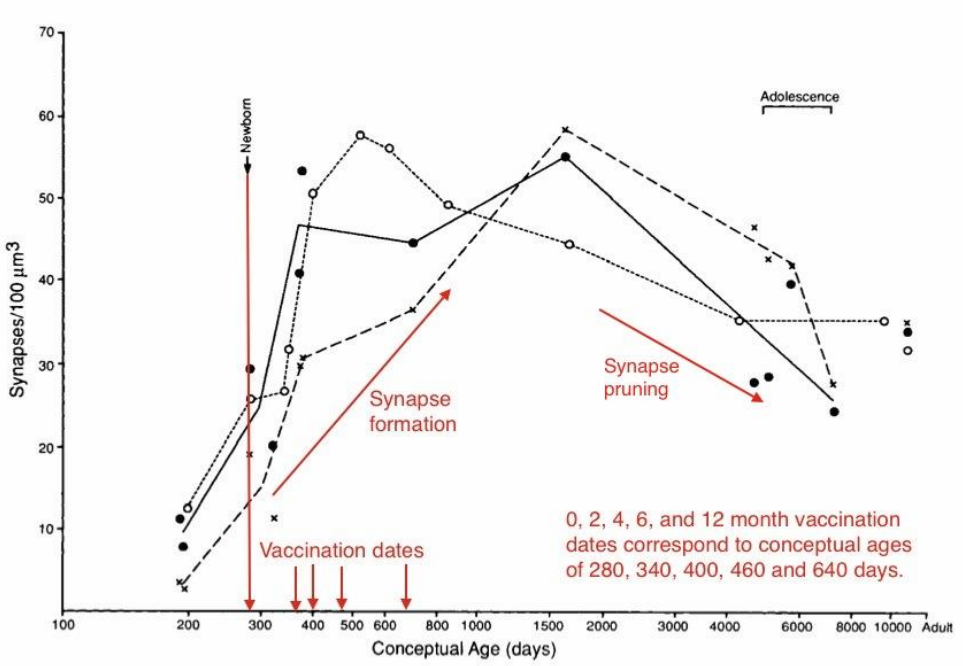


Fig. 2. Mean synaptic density in synapses/100 µm^3 in auditory, calcarine, and prefrontal cortex at various ages. Open circles, visual cortex (area 17); filled circles, auditory cortex; x, prefrontal cortex (middle frontal gyrus).

Fig. 6: Measurements of synapse density in human cadavers of various ages indicate a dramatic increase in synapses in the first few years of life. Vaccines are administered during intense synapse formation. Elevated IL-6 during synaptogenesis may cause an excitatory-inhibitory synapse imbalance, towards excitation. Image adapted from Huttenlocher and Dabholkar 1997.

Aluminum Adjuvants: Neurotoxic At Vaccine Dosages

Aluminum (Al) adjuvants have an essential role in many vaccines: to stimulate immune activation. Without Al adjuvants, these vaccines would have greatly reduced efficacy.

Aluminum adjuvants comprise sub-micron particles (primary particles) of aluminum compounds, typically AlOH, AlPO4, AlSO4 or mixtures. The primary particles are typically agglomerated into larger particles with sizes of about 2-20 microns (Harris 2012). The Al adjuvant materials have low solubility in water and body fluids. Al adjuvant particles are biopersistent and can remain in the body for months or years (Flarend 1997, Khan 2013, Gherardi 2001).

Aluminum ingested in the diet has low oral absorption (about 0.3%), is rapidly excreted by the kidneys, is (mostly) excluded from the brain by the blood-brain barrier, and is in a solubilized, Al3+ ionic form (not particulate). These defenses are adequate for protecting the brain from natural levels of aluminum exposure. These protective mechanisms are unable to protect the brain from injected aluminum adjuvant particles. Al adjuvant particles are too large to be removed by the kidneys, and are carried across the blood-brain barrier by macrophages.

Dosages of aluminum adjuvants received by infants according to the CDC vaccination schedule are:

Birth (Hep B):
74 mcg/kg (250 mcg for 3.4 kg infant)

2 month:
245 mcg/kg (1225 mcg for 5 kg infant)

4 month:
150 mcg/kg (975 mcg for 6.5 kg infant)

6 month:
153 mcg/kg (1225 mcg for 8 kg infant)

These are maximum-possible dosages (because different vaccine products have different amounts) for average-weight infants.

Accumulating evidence shows that aluminum adjuvants have adverse neurological effects at dosages lower than or approximately equal to dosages infants receive from vaccines. These effects appear to depend on the particulate nature and biopersistence of the aluminum adjuvant. Injected Al adjuvant has adverse effects that are apparently mediated by the particles and independent of solubilized Al3+ ions released by the slowly dissolving particles (Crepeaux 2017).

Al adjuvant injections in mice cause adverse effects at vaccine-relevant dosages of 100, 200, 300 and 550 mcg/Kg body weight (Crepeaux 2017, Shaw 2009, Petrik 2007, Shaw 2013). These include deficits in learning and memory (Shaw 2009), deficits in neuromuscular strength/function (Petrik 2007), and changes in locomotor activity and/or gait (Shaw 2009, Shaw 2013). Autism is associated with gait and movement abnormalities (Kindregan 2015) and memory dysfunction (Williams 2006).

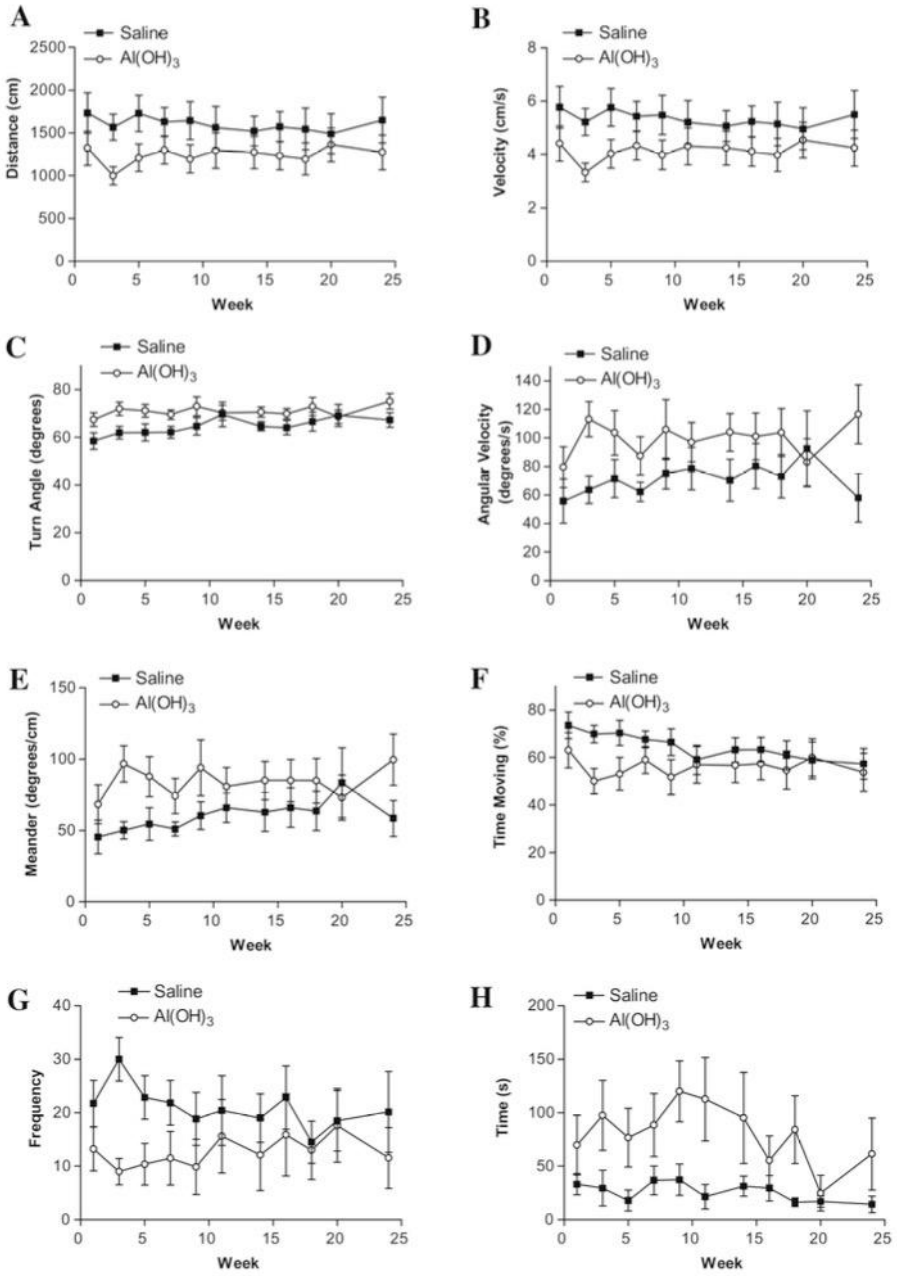


Fig. 4.
Open field movement analysis as an assessment of spontaneous activity and anxiety in control mice vs. mice injected six times with aluminum hydroxide. Aluminum hydroxide injected mice showed the following behavioural changes: (A) Shorter distances moved (***$p < 0.0001$). (B) Slower movement (***$p < 0.0001$). (C) Greater mean turn angle (***$p < 0.0001$). (D) More rapid turning (***$p < 0.0001$). (E) Greater meander (***$p < 0.0001$). (F) Smaller percentage of time in overall movement (**$p = 0.0030$). (G) Fewer entries into the centre of the open field (***$p < 0.001$). Late entry into centre (***$p < 0.0001$). (All measures, two-way ANOVA).

Fig. 7: Dosage of 300mcg/Kg AlOH adjuvant caused large and persistent changes in exploratory behavior and movement in open field tests. This is an indicator of neurotoxicity. Human autistics also display abnormal movement and exploratory behavior. Adapted from Shaw and Petrik 2009.

Al adjuvant dosages of 200mcg/Kg (as 3 x 66mcg/Kg) (Crepeaux 2017) and 300mcg/Kg (as 6 x 50mcg/Kg) (Shaw 2009) increased microglial activation in the ventral forebrain and lumbar spinal cord, respectively. The elevated microglial activation was measured about 6 months after Al adjuvant injection, which suggests that the microglial activation is chronic. Activated microglia indicate an ongoing inflammatory process and suggest the presence of elevated cytokines. Human autistics have activated microglia and elevated cytokines throughout the brain (Vargas 2005, Suzuki 2013, Li 2009).

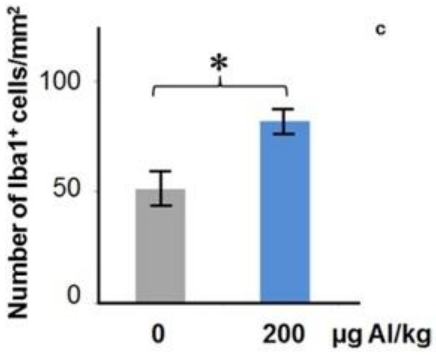


Fig. 8: Al adjuvant (200mcg/Kg) caused an increase in microglial activation in the brain of mice. The protein iba1 indicates activated microglia. Measurements were performed 6 months after Al adjuvant injection, indicating that the microglial activation is a chronic condition. * P<0.05. From Crepeaux et al., 2017.

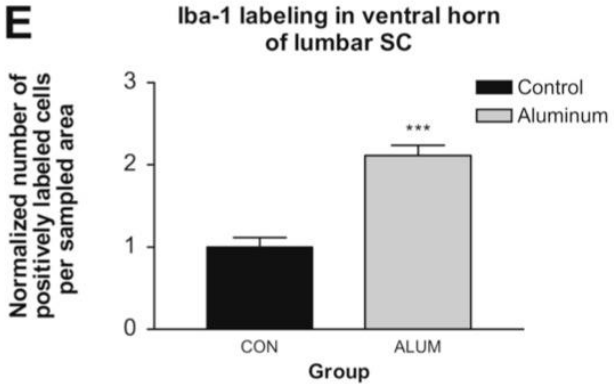


Fig. 9: Al adjuvant (300mcg/Kg) caused an increase in microglial activation in the lumbar spinal cord of mice. The protein iba1 indicates activated microglia. Measurements were performed 6 months after Al adjuvant injection, indicating that the microglial activation is a chronic condition. ***$p < 0.001$, one-way ANOVA. From Shaw and Petrik 2009.

Activated microglia are implicated as a causal factor in autism, because microglia mediate inflammation in the brain. Microglia can produce IL-6 when in an activated state. A recent review on microglia and autism (Takano 2015) states:

> *"…any factors that alter the number or activation state of microglia either in utero or during the early postnatal period can profoundly affect neural development, thus resulting in neurodevelopmental disorders, including autism." (Takano 2015)*

Microglia appear to play an important role in the causation of autism (Takano 2015, Kneusel 2014). Hence, the microglial activation caused by aluminum adjuvants suggests a role in autism.

Several studies show that Al adjuvants increase brain aluminum content (Crepeaux 2017, Flarend 1997, Shaw 2009, Khan 2013, Crepeaux 2015). A dosage of 200 mcg/Kg Al adjuvant caused a 50-fold increase in brain aluminum content in mice, from 0.02 ug/g to 1.0 ug/g dry weight of brain (Crepeaux 2017). These measurements were performed 6 months after the final injection, indicating that the Al persists in the brain long-term (Crepeaux 2017). See Fig. 10. Al adjuvants have been found to accumulate in the brain of mice up to one year after injection (Khan 2013). Crepeaux 2015 demonstrated persistence and increasing accumulation of Al adjuvant particles up to 270 days in spleen and lymph nodes of mice. Increasing accumulation of Al in distant organs over time suggests that toxic effects may increase with time, and may be delayed by months or years after exposure.

The 400 and 800 mcg/Kg doses used in the Crepeaux 2017 study did not cause adverse effects or elevated brain aluminum. The authors attribute this surprising inverted dose-response relationship to granulomas induced by the higher dosages. Granulomas trap the Al adjuvant at the injection site, thereby preventing its transport into the brain and other sensitive tissues. Granulomas occur after about 1% of vaccinations (Bergfors 2014). This is cause for concern because it indicates that, for 99% of vaccinations, the Al adjuvant can be transported around the body. It is not confined to a granuloma. See Fig. 11.

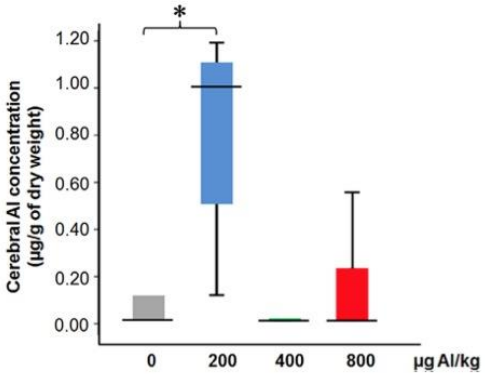


Fig. 10: Dosage of 200 mcg/Kg Al adjuvant caused a 50-fold increase in brain aluminum content, from 0.02 to 1.00 ug/g dry weight, in mice. Higher dosages (400 and 800 mcg/Kg) did not increase brain Al content, presumably because the higher dosages caused a granuloma at the injection site. A granuloma traps the Al adjuvant at the injection site, thereby preventing systemic dispersal and transport into the brain. These measurements were performed 6 months after the final injection, indicating that the Al persists in the brain long-term. *P<0.05. From Crepeaux et al., 2017.

Proposed Mechanism For Inverse Dose-Toxicity Relationship:

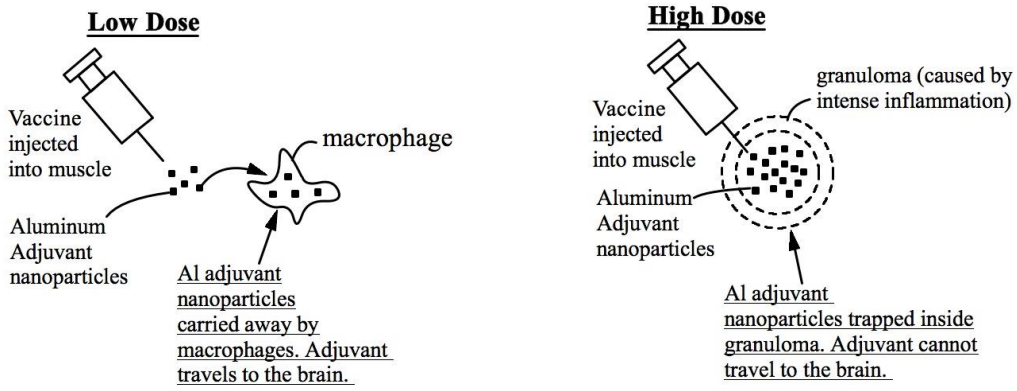


Fig. 11: High dose Al adjuvant injection into the muscle causes a granuloma, which traps the Al adjuvant and prevents it from traveling into the brain. Low dose does not form a granuloma. Hence, the lower dose is free to travel to the brain. Consequently, the lower dose is more toxic than the higher dose. This mechanism explains the surprising inverted dose-toxicity results of Crepeaux et al. 2017.

Particle Transport and Macrophage Chemotactic Protein (MCP-1)

Aluminum adjuvants travel into the brain (Khan 2013, Crepeaux 2015, Crepeaux 2017, Shaw 2009, Flarend 1997). Al adjuvant particles are carried through the blood-brain barrier and into the brain by macrophages (Khan 2013). Transport is promoted by macrophage chemotactic protein-1 (MCP-1) (Khan 2013). MCP-1 causes macrophages to travel around the body and into the brain. Particle transport into the brain by macrophages is well-established and has been investigated for therapeutic applications (Choi 2012, Pang 2016).

MCP-1 is elevated in the brains of human autistics (Vargas 2005) and is elevated in the blood of neonates later diagnosed with autism (Zerbo 2014). This suggests that neonates with high MCP-1 will experience elevated Al adjuvant transport into the brain when injected with Al adjuvanted vaccines. This is consistent with Al adjuvants causing autism by inducing immune activation and elevated cytokines in the brain.

Aluminum Induces IL-6 Expression In The Brain

Water-soluble aluminum salts (e.g. $AlCl_3$, Al lactate) induce elevated IL-6 in the brain and other tissues. In fact, aluminum appears to selectively induce IL-6 (Viezeliene 2013). Studies of aluminum exposure and IL-6 expression in the brain include:

Cao 2016: Ingestion of 30 or 90 mg/kg/day aluminum (as $AlCl_3$) for 90 days significantly increased gene expression of IL-6 and other cytokines in the brain (hippocampus).

Alawdi 2016: Ingestion of 3.4 mg/kg/day aluminum (as $AlCl_3$) for 6 weeks caused a 4-fold increase in IL-6 in the brain (hippocampus). This dosage is far lower than the outdated "no observed adverse effects level" (NOAEL) oral dosages (26 and 62 mg/kg/day) used as benchmarks for toxicity threshold (Mitkus 2011, Offit 2003).

In fact, other experiments show that oral dosages of 3.4, 4, 5.6, 6, and 20.2

mg/Kg/day aluminum cause numerous adverse effects in mice or rats and hence the NOAEL for orally ingested Al is currently unknown (Alawdi 2016, Dera 2016, Sethi 2008, Sethi 2009, Bilkei-Gorzo 1993).

The induction of IL-6 may occur because aluminum strongly induces oxidative stress (Exley 2003). Oxidative stress induces IL-6 expression (Viezeliene 2013).

CDC Website Cites Fatally Flawed Study Of Al Adjuvants (Mitkus 2011)

Dosages of Al adjuvants received by infants increased dramatically as the vaccine schedule was expanded in the 1980s and 1990s. However, as the vaccine schedule expanded, the increasing dosages of Al adjuvants were not tested for safety. Government agencies (HHS, NIH, CDC, FDA) have not pursued any new experimental work on Al adjuvant toxicity.

To support the safety of Al adjuvants at today's higher dosages, the CDC cites a 2011 FDA study of aluminum exposure from vaccines (Mitkus 2011). This study is the only scientific evidence cited by the CDC and FDA websites to support the safety of Al adjuvants.

The Mitkus 2011 study is a theoretical modeling study of Al adjuvant kinetics; it contains no new data concerning Al adjuvant toxicity (from animal models or epidemiology). Mitkus 2011 calculates a body burden of aluminum resulting from the slow dissolution of Al adjuvant particles, and compares the dissolved-aluminum body burden to a "minimal risk level" (MRL). The MRL is derived from a study of ingested Al toxicity in mice (Golub 2001). The Golub 2001 study provides the NOAEL (26 mg/kg/day ingested), which is converted into the MRL for human infants (based on 1mg/kg/day ingested) by using a safety factor of about 30.

The Mitkus study is fatally flawed for these reasons:

1) MITKUS ASSUMES AL ADJUVANT PARTICLES ARE HARMLESS

Mitkus makes an unstated assumption that Al adjuvants have zero toxicity while in particulate form. Mitkus only considers the potential toxicity of aluminum ions (Al3+) released by the slowly-dissolving Al adjuvant particles.

Al adjuvants comprise low-solubility and biologically-persistent microscopic particles. The Mitkus analysis assumes that the particles are absolutely nontoxic and perfectly harmless, even when present in the brain and other organs. Mitkus provides no justification for this unstated assumption. Further, the assumption is contradicted by recent findings on Al adjuvant toxicity (Crepeaux 2017) and particulate toxicity generally. Particles can have toxic effects mediated by surface chemistry (e.g. surface charge and surface catalytic activity) and particle shape, among other characteristics of solid particles (Sharifi 2012, Podila 2013).

Several studies show injected Al adjuvants cause behavioral abnormalities, abnormal weight gain, learning and memory impairment, motor neuron death/apoptosis, neuromuscular strength deficits, chronic microglial activation/brain inflammation, and large (e.g. 50X) increases in brain and spinal cord aluminum content (Petrik 2007, Shaw 2009, Shaw 2013, Crepeaux 2017). These adverse effects occur at dosages less than or approximately equal to dosages received by infants according to the CDC vaccine schedule.

2) NEW RESEARCH SHOWS INGESTED AL HARMFUL AT DOSAGES LOWER THAN 26 MG/KG/DAY

Mitkus assumes that Al adjuvant toxicity is mediated exclusively by solubilized Al (Al3+ ions) released by the slowly-dissolving Al adjuvant particles. To establish a threshold toxicity level from the solubilized Al, Mitkus relies on a mouse feeding study (Golub 2001) reporting a "no-observed adverse effects level" (NOAEL) oral dosage of 26 mg/Kg/day ingested aluminum. Mitkus

used a 30X safety factor for applying this dosage to humans, which is reasonable.

However, other experiments show that much lower oral dosages of 3.4, 4, 5.6, 6, and 20.2 mg/Kg/day aluminum cause adverse effects in mice or rats (Alawdi 2016, Dera 2016, Sethi 2008, Sethi 2009, Bilkei-Gorzo 1993). The adverse effects include chronic brain inflammation, learning and memory impairment, and kidney inflammation. So, the Mitkus analysis is wrong because 26 mg/kg/day is not a NOAEL. The "minimal risk level" (MRL) determined by Mitkus is too high by a factor of at least 26/3.4 = 7.6. Using a corrected NOAEL of 3.4 mg/Kg/day (based on Alawdi 2016) results in vaccine aluminum exposure exceeding the MRL for AlPO4 adjuvant, and approximately matching the MRL for AlOH adjuvant. The new, corrected MRL lines indicate that Al phosphate adjuvant (Fig. 12) and Al hydroxide adjuvant (Fig. 13) from the CDC vaccine schedule may cause toxicity from the solubilized Al per se.

Since 3.4mg/Kg/day is not a NOAEL (adverse effects were observed at this dosage) the true NOAEL is less than 3.4/mg/Kg/day. See Figs. 12-13.

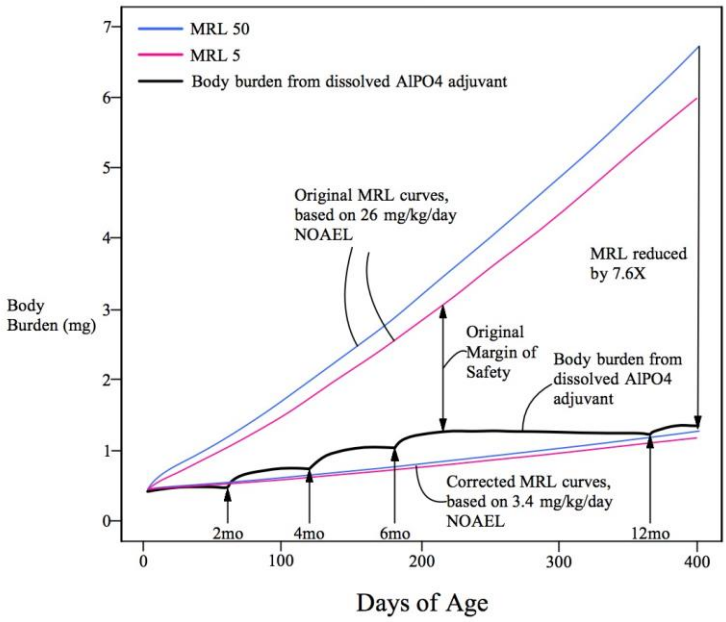


Fig. 12: Body burden vs. MRL comparison chart for Al <u>phosphate</u> adjuvant (AlPO4) corrected in accordance with the new discovery (Alawdi 2016) that ingestion of 3.4 mg/kg/day Al causes adverse effects. The body burden exceeds the corrected MRL curve for almost the entire first year of life, indicating toxicity. The toxicity of Al adjuvant <u>particles</u> is a separate, additional issue. MRL 50 and MRL 5 refer to two different infant growth rates. Adapted from Mitkus et al., 2011.

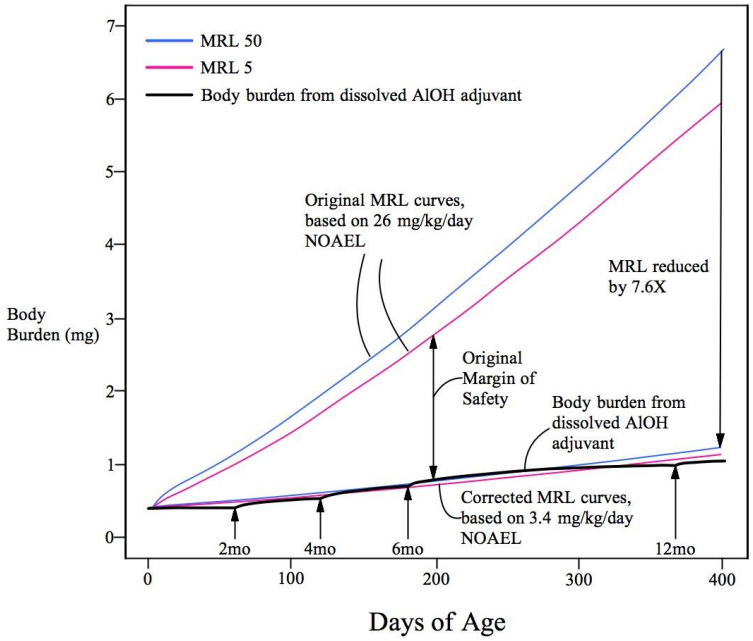


Fig. 13: Body burden vs. MRL comparison chart for Al hydroxide adjuvant (AlOH), corrected in accordance with the new discovery (Alawdi 2016) that ingestion of 3.4 mg/kg/day Al causes adverse effects. The body burden overlaps the new, corrected MRL, indicating borderline toxicity. The margin of safety is gone. MRL 50 and MRL 5 refer to two different infant growth rates. The toxicity of Al adjuvant particles is a separate, additional issue. Adapted from Mitkus et al., 2011.

3) NO AL ADJUVANT TOXICITY DATA CITED, DESPITE AVAILABILITY

Mitkus does not cite any toxicity data for injected Al adjuvants. Mitkus instead uses toxicity data for ingested, non-particulate, water-soluble Al (Golub 2001, which used Al lactate) to derive the MRL. This data comes from a single study (Golub 2001).

So, remarkably, Mitkus claims a safe level of injected Al adjuvant exposure, without citing any Al adjuvant toxicity data. The error is unnecessary and neglectful because at least two animal studies of injected Al adjuvant toxicity were available prior to the Mitkus publication in 2011 (Petrik 2007, Shaw 2009). These papers were not cited or mentioned by Mitkus 2011.

Each of these three flaws is fatal for the validity of the Mitkus study in establishing the safety of aluminum adjuvants. Hence, the CDC is completely lacking valid evidence for the safety of Al adjuvants. This is especially true for safety regarding neurological and long-term outcomes, because other available studies of Al adjuvant safety (e.g., Jefferson 2004) do not consider (or are incapable of detecting) these outcomes.

CDC Fails To Investigate Toxicity of Al Adjuvants

The CDC has conducted no epidemiological studies on long term safety (e.g. considering neurological outcomes) of Al adjuvants. There is one ecological study of country-level data, which reported an association between Al adjuvant exposure and autism (Tomljenovic 2011). However, being an ecological study, it is highly susceptible to confounding and biases.

Dr Frank DeStefano of the CDC's Immunization Safety Office is co-author of a

feasibility study (Glanz 2015) on using the Vaccine Safety Datalink (VSD) to investigate the safety of individual vaccine ingredients. The paper focuses on Al adjuvants. It acknowledges that thimerosal is the only vaccine ingredient studied for autism or neurological safety, and that a possible association between Al adjuvants and autism has not been explored in epidemiological studies. Glanz 2015 states:

> *"To date, there have been no population-based studies specifically designed to evaluate associations between clinically meaningful outcomes and non-antigen ingredients, other than thimerosal."*

The CDC has not investigated Al adjuvant safety concerns, despite the accumulating scientific evidence of harm and evidence linking Al adjuvants to immune activation mechanisms of brain injury.[1]

Conclusion

The science reviewed here tells a consistent and compelling story: that vaccines may cause autism by stimulating immune activation and elevated cytokines in the brain. Al adjuvants are implicated as a cause of autism because they can be transported into the brain, because they cause microglial activation at vaccine-relevant dosages, and because aluminum induces IL-6 in the brain.

In statements asserting no vaccine-autism link, the CDC cites scientific evidence that is not relevant to Al adjuvant safety or is incapable of disproving an Al adjuvant-autism link (Taylor 2014, DeStefano 2013, Mitkus 2011). In support of claims for Al adjuvant safety, the CDC relies on a profoundly flawed theoretical modelling study (Mitkus 2011). There is little scientific evidence supporting the safety of Al adjuvants, especially in relation to autism and other long term neurological outcomes.

[1] However, the Glanz paper notes that studies of aluminum adjuvants are problematic because of expected small differences in exposures in the low and high exposure groups. Glanz 2015 concludes: "*…children below the 10th percentile would be exposed to between 0 mg and 3.1mg, while children above the 90th percentile would be exposed to between 4.8 mg and 5.3 mg of aluminum from vaccines. It is unclear if such differences in aluminum exposure would be biologically meaningful.*" (Glanz 2015). So, epidemiological studies may not provide reliable evidence for safety or harm. Controlled, prospective human trials of aluminum adjuvant exposure from vaccines will likely be prohibited for ethical reasons. Also, Al adjuvants are essential ingredients for Al adjuvanted vaccines. Consequently, it will be challenging to design studies of long term adverse effects of Al adjuvants in humans. Experiments in animal models can provide valuable information. Al adjuvants should be tested for effects on: 1) excitatory/inhibitory imbalance; 2) core symptoms of autism (social, communicative and repetitive/stereotyped behaviors); 3) IL-6, IL-17, and other cytokine levels in the brain; 4) other physiological abnormalities associated with autism (e.g. mitochondrial dysfunction, microbiome dysbiosis, Purkinje cell loss, cerebellum abnormalities etc); and 5) microglial activation and immune activity in the brain. Investigating these outcomes can provide valuable information concerning the safety of Al adjuvants.

References

Akintunde et al., 2015 Increased production of IL-17 in children with autism spectrum disorders and co-morbid asthma, Journal of Neuroimmunology 286 (2015) 33-41.

Al-Ayadhi et al., 2012 Elevated serum levels of interleukin-17A in children with autism, Journal of Neuroinflammation 2012, 9:158.

Alawdi et al., Neuroprotective Effect of Nanodiamond in Alzheimer's Disease Rat Model: a Pivotal Role for Modulating NF-κB and STAT3 Signaling, Molecular Neurobiology, 54 (3):1906-1918.

Atladottir et al., Maternal infection requiring hospitalization during pregnancy and autism spectrum disorders, Journal of Autism and Developmental Disorders, 2010 Dec;40(12):1423-1430.

Bauman et al., 2014 Activation of the Maternal Immune System During Pregnancy Alters Behavioral Development of Rhesus Monkey Offspring, Biological Psychiatry, 2014;75: 332–341

Bergfors et al., 2014 How common are long-lasting, intensely itching vaccination granulomas and contact allergy to aluminium induced by currently used pediatric vaccines? A prospective cohort study, European Journal of Pediatrics, 173:1297–1307.

Bilkei-Gorzo, 1993, Neurotoxic effect of enteral aluminum, Food and Chemical Toxicology, 31(5):357-361.

Brown et al., 2014 Metabolic consequences of interleukin-6 challenge in developing neurons and astroglia, Journal of Neuroinflammation, 11:183.

Brown et al., Epidemiologic studies of exposure to prenatal infection and risk of schizophrenia and autism, Developmental Neurobiology, 2012 October ; 72(10): 1272–1276.

Bruce et al., 2011 Converging pathways lead to overproduction of IL-17 in the absence of vitamin D signaling, 2011 Aug; 23(8): 519–528.

Careaga et al 2017 Maternal Immune Activation and Autism Spectrum Disorder: From Rodents to Nonhuman and Human Primates, Biological Psychiatry, March 1, 2017; 81:391–401.

Chen et al., Postnatal systemic inflammation exacerbates impairment of hippocampal synaptic plasticity in an animal seizure model, Neuroimmunomodulation, 2013;20(4):223-32.

Choi et al., 2012, Delivery of nanoparticles to brain metastases of breast cancer using a cellular Trojan horse, Cancer Nanotechnology, 3:47–54.

Choi et al., 2016 The maternal interleukin-17a pathway in mice promotes autismlike phenotypes in offspring, Science, 2016 Feb 26; 351(6276): 933–939.

Ciaranello et a The Neurobiology of Infantile Autism, The Neuroscientist, 1:361-367

Coiro et al., Impaired synaptic development in a maternal immune activation mouse model of neurodevelopmental disorders, Brain, Behavior, and Immunity, Nov;50:249-258.

Crepeaux et al., 2015 Highly delayed systemic translocation of aluminum-based adjuvant in CD1 mice following intramuscular injections, Journal of Inorganic Biochemistry, 152:199-205.

Crepeaux et al., 2017 Non-linear dose-response of aluminium hydroxide adjuvant particles: Selective low dose neurotoxicity, Toxicology, 375 (2017) 48–57.

DeLong et al., 1981 Acquired reversible autistic syndrome in acute encephalopathic illness in children, Archives of Neurology, 36:191-194.

Dera 2016, Protective effect of resveratrol against aluminum chloride induced nephrotoxicity in rats, Saudi Medical Journal, 37 (4).

DeStefano et al., 2013 Increasing Exposure to Antibody-Stimulating Proteins and Polysaccharides in Vaccines Is Not Associated with Risk of Autism, The Journal of Pediatrics, 163 (2).

Deverman and Patterson, 2009 Cytokines and CNS Development, Neuron 64:61-78.

Drozdenko et al., 2014 Oral vitamin D increases the frequencies of CD38+ human B cells and ameliorates IL-17-producing T cells, Experimental Dermatology, 23: 107-112.

Estes and McAllister, 2016 Maternal immune activation: implications for neuropsychiatric disorders, Science, 353 (6301) 772-777.

Exley, 2003 The Pro-Oxidant Activity of Aluminum, Free Radical Biology and Medicine, 36(3): 380-387.

Flarend et al., 1997 In vivo absorption of aluminum-containing vaccine adjuvants using 26 Al, Vaccine, 15(12/13):1314-1318.

Freyberg et al., 2015 Reduced perceptual exclusivity during object and grating rivalry in autism, Journal of Vision, 15(13):11, 1–12.

Galic et al., 2008 Postnatal Inflammation Increases Seizure Susceptibility in Adult Rats, The Journal of Neuroscience, 2008, 28 (27) 6904-6913.

Garay et al., 2013 Maternal immune activation causes age- and region-specific changes in brain cytokines in offspring throughout development, Brain, Behavior, and Immunity, 31: 54-68.

Ghaziuddin et al., 2002 Autistic symptoms following herpes encephalitis, European Child and Adolescent Psychiatry, Vol. 11, No. 3:142-146.

Gherardi et al., 2001 Macrophagic myofasciitis lesions assess long-term persistence of vaccine-derived aluminium hydroxide in muscle, Brain, 124:1821-1831.

Ghiani et al., 2011 Early effects of lipopolysaccharide induced inflammation on foetal brain development in rat, ASN Neuro, 3 (4): 233-245.

Gillberg 1986 Brief Report: Onset at Age 14 of a Typical Autistic Syndrome. A Case Report of a Girl with Herpes Simplex Encephalitis, Journal of Autism and Developmental Disorders, VoL 16, No. 3:369-375.

Giulivi et al 2013 Gestational Exposure to a Viral Mimetic Poly(I:C) Results in Long-Lasting Changes in Mitochondrial Function by Leucocytes in the Adult Offspring, Mediators of Inflammation, Vol 2013:609602.

Glanz et al., 2015, Cumulative and episodic vaccine aluminum exposure in a population-based cohort of young children, Vaccine 33:6736–6744.

Golub et al., 2001 Long-term consequences of developmental exposure to aluminum in a suboptimal diet for growth and behavior of Swiss Webster mice, Neurotoxicology and Teratology 23 (2001) 365–372.

Gupta et al., 1998 Th1- and Th2-like cytokines in CD4+ and CD8+ T cells in autism, Journal of Neuroimmunology, 85:106-109.

Harre et al., 2008 Neonatal inflammation produces selective behavioural deficits and alters *N*-methyl-D-aspartate receptor subunit mRNA in the adult rat brain, European Journal of Neuroscience, 2008 Feb; 27(3): 644–653.

Harris et al., 2012 Alhydrogel® adjuvant, ultrasonic dispersion and protein binding: A TEM and analytical study, Micron, 43:192-200.

Hsiao et al., 2013 The microbiota modulates gut physiology and behavioral abnormalities associated with autism, Cell, 155(7): 1451-1463.

Huttenlocher and Dabholkar, 1997 Regional Differences in Synaptogenesis in Human Cerebral Cortex, Journal of Camparative Neurology, 387:167–178 (1997).

Jefferson 2004 Adverse events after immunisation with aluminium-containing DTP vaccines: systematic review of the evidence, The Lancet 4:84-90.

Jones et al., 2016 Autism with Intellectual Disability is Associated with Increased Levels of Maternal Cytokines and Chemokines During Gestation, Molecular Psychiatry, 22(2):273-279.

Khan et al., 2013 Slow CCL2-dependent translocation of biopersistent particles from muscle to brain, BMC Medicine, 11:99.

Kindregan et al., 2015 Gait Deviations in Children with Autism Spectrum Disorders: A Review, Autism Research and Treatment, ID:741480.

Knuesel et al., 2014, Maternal immune activation and abnormal brain development across CNS disorders, Nature Reviews 10:643-660.

Labouesse et al., 2015, Long-term pathological consequences of prenatal infection: beyond brain disorders, American Journal of Physiology, 309:1.

Li et al. 2009 Elevated Immune Response in the Brain of Autistic Patients, Journal of Neuroimmunology, 207(1-2): 111–116.

Li et al., 2015 Neonatal vaccination with bacillus Calmette–Guérin and hepatitis B vaccines modulates hippocampal synaptic plasticity in rats, Journal of Neuroimmunology, 288 (2015) 1-12.

Machado et al., 2015 Maternal Immune Activation in Nonhuman Primates Alters Social Attention in Juvenile Offspring, Biological Psychiatry, 2015 May 1;77(9):823-32.

Malkova et al., 2012 Maternal immune activation yields offspring displaying mouse versions of the three core symptoms of autism, Brain Behaviorand Immunity, 2012 May ; 26(4): 607–616.

Marques et al., 2014 Autism Spectrum Disorder Secondary to Enterovirus Encephalitis, Journal of Child Neurology, 2014, Vol. 29(5) 708-714.

Mestas et al., 2004 Of Mice and Not Men: Differences between Mouse and Human Immunology, Journal of Immunology, 0022-1767:2731-2738.

Meyer et al., 2006 The Time of Prenatal Immune Challenge Determines the Specificity of Inflammation-Mediated Brain and Behavioral Pathology, The Journal of Neuroscience, 26(18):4752– 4762.

Meyer et al., 2007 The neurodevelopmental impact of prenatal infections at different times of pregnancy: the earlier the worse?, Neuroscientist, Jun;13(3):241-56.

Meyer et al., 2009 In-vivo rodent models for the experimental investigation of prenatal immune activation effects in neurodevelopmental brain disorders, Neuroscience and Biobehavioral Reviews, 33 (2009) 1061–1079.

Meyer 2014, Prenatal Poly(I:C) Exposure and Other Developmental Immune Activation Models in Rodent Systems, Biological Psychiatry, 75:307-315.

Mitkus et al., 2011 Updated aluminum pharmacokinetics following infant exposures through diet and vaccination, Vaccine 29 (2011) 9538–9543.

Offit et al., 2003 Addressing Parents' Concerns: Do Vaccines Contain Harmful Preservatives, Adjuvants, Additives, or Residuals? Pediatrics, 112(6): 1394-1401.

Oskvig et al., 2012 Maternal immune activation by LPS selectively alters specific gene expression profiles of interneuron migration and oxidative stress in the fetus without triggering a fetal immune response, Brain Behavior and Immunity, 2012 May ; 26(4): 623–634.

Pang et al., 2016 Exploiting macrophages as targeted carrier to guide nanoparticles into glioma, Oncotarget 7(24):37081.

Parker-Athill and Tan, 2010 Maternal Immune Activation and Autism Spectrum Disorder: Interleukin-6 Signaling as a Key Mechanistic Pathway, NeuroSignals, 2010;18:113–128.

Petrik et al., 2007 Aluminum Adjuvant Linked to Gulf War Illness Induces Motor Neuron Death in Mice, NeuroMolecular Medicine, Vol. 9, 83-100.

Pineda et al., 2013 Maternal immune activation promotes hippocampal kindling epileptogenesis in mice, Annals of Neurology, 2013 July ; 74(1): 11–19.

Podila et al., 2013 Toxicity of Engineered Nanomaterials: A Physicochemical Perspective, Journal of Biochemical and Molecular Toxicology, 2013 January ; 27(1): 50–55.

Robertson et al., 2016 Reduced GABAergic Action in the Autistic Brain, Current Biology, 26, 1-6.

Saad et al., 2016 Vitamin D status in autism spectrum disorders and the efficacy of vitamin D supplementation in autistic children, Nutritional Neuroscience, 19 (8) 346-351.

Semple et al., 2013 Brain development in rodents and humans: Identifying benchmarks of maturation and vulnerability to injury across species, Progress in Neurobiology, Jul-Aug;106-107:1-16.

Sethi et al., 2008 Aluminium-induced electrophysiological, biochemical and cognitive modifications in the hippocampus of aging rats, Neurotoxicology 29, 1069-1079.

Sethi et al., 2009 Curcumin attenuates aluminium-induced functional neurotoxicity in rats, Pharmacology, Biochemisatry, and Behavior 93:31-39.

Shen et al., 2016 Postnatal activation of TLR4 in astrocytes promotes excitatory synaptogenesis in hippocampal neurons, Journal of Cell Biology, 215(5):719-734.

Sharifi et al., 2012 Toxicity of Nanomaterials, Chemical Society Reviews, 2012 Mar 21; 41(6): 2323–2343.

Shaw and Petrik, 2009 Aluminum hydroxide injections lead to motor deficits and motor neuron degeneration, Journal of Inorganic Biochemistry 103 (11).

Shaw and Tomljenovic, 2013 Administration of aluminium to neonatal mice in vaccine-relevant amounts is associated with adverse long term neurological outcomes, Journal of Inorganic Biochemistry, 128 (2013) 237–244.

Shi et al., 2009 Activation of the Maternal Immune System Alters Cerebellar Development in the Offspring, Brain, Behavior, and Immunity, January, 23(1): 116–123.

Smith et al., 2007 Maternal Immune Activation Alters Fetal Brain Development through Interleukin-6, Journal of Neuroscience, 2007 October 3; 27(40).

Smith et al., 2012, Maternal Immune Activation Increases Neonatal Mouse Cortex Thickness and Cell Density, Journal of Neuroimmune Pharmacology, 7(3):529-532.

Stiles et al., 2010 The Basics of Brain Development, Neuropsychology Reviews (2010) 20:327–348.

Suzuki et al., 2011 Plasma Cytokine Profiles in Subjects with High-Functioning Autism Spectrum Disorders, PloS ONE 6(5).

Suzuki et al., 2013 Microglial Activation in Young Adults With Autism Spectrum Disorder, JAMA Psychiatry 70(1): 49-58.

Takano 2015 Role of Microglia in Autism: Recent Advances, Developmental Neuroscience, 37:195-202.

Tau and Peterson, 2010 Normal Development of Brain Circuits, Neuropsychopharmacology, (2010) 35:147–168.

Taylor et al., 2014 Vaccines are not associated with autism: An evidence-based meta-analysis of case-control and cohort studies, Vaccine, 32:3623-3629.

Tomljenovic and Shaw, 2011 Do aluminum vaccine adjuvants contribute to the rising prevalence of autism? Journal of Inorganic Biochemistry 105.

Tsilioni et al., 2015 Children with autism spectrum disorders, who improved with a luteolin-containing dietary formulation, show reduced serum levels of TNF and IL-6, Translational Psychiatry, 5, 647.

Vargas et al., 2005 Neuroglial Activation and Neuroinflammation in the Brain of Patients with Autism, Annals of Neurology, 2005;57:67–81.

Viezeliene et al., 2013 Selective induction of IL-6 by aluminum-induced oxidative stress can be prevented by selenium, Journal of Trace Elements in Medicine and Biology, 27:226-229.

Wei et al., 2011 IL-6 is increased in the cerebellum of autistic brain and alters neural cell adhesion, migration and synaptic formation, Journal of Neuroinflammation 2011, 8:52.

Wei et al., 2012 (a) Brain IL-6 elevation causes neuronal circuitry imbalances and mediates autism-like behaviors, Biochimica et Biophysica Acta, 1822 (2012) 831–842.

Wei et al. 2012 (b) Alteration of brain volume in IL-6 overexpressing mice related to autism, International Journal of Developmental Neuroscience, 30:554-559.

Wei et al., 2013 Brain IL-6 and autism, Neuroscience 252 (2013): 320–325.

Wei et al., 2016 Inhibition of IL-6 trans-signaling in the brain increases sociability in the BTBR mouse model of autism, Biochimica et Biophysica Acta, 1862(10):1918-1925.

Weir et al., 2015 Preliminary evidence of neuropathology in nonhuman primates prenatally exposed to maternal immune activation, Brain, Behavior, and Immunity, 48,139–146.

Williams et al., 2006 The Profile of Memory Function in Children With Autism, Neuropsychology, 20(1): 21-29.

Wobke et al., 2014 Vitamin D in inflammatory diseases, Frontiers in Physiology, 5: 244.

Zerbo et al., 2014 Neonatal cytokines and chemokines and risk of Autism Spectrum Disorder: the Early Markers for Autism (EMA) study: a case-control study, Journal of Neuroinflammation, 11:113.

Zerbo et al., 2017 Association Between Influenza Infection and Vaccination During Pregnancy and Risk of Autism Spectrum Disorder, JAMA Pediatrics, 171(1).

Vaccines: What About Immunocompromised Schoolchildren?

PHYSICIANS FOR INFORMED CONSENT
Delivering Data on Infectious Diseases & Vaccines™

Available in other languages at:
physiciansforinformedconsent.org/
immunocompromised-schoolchildren

1. WHAT DOES IT MEAN TO BE IMMUNOCOMPROMISED?

Immunocompromised children have weakened immune systems that prevent them from optimally fighting infections on their own. Consequently, they may be at increased risk of complications from infectious diseases and require additional precautions and treatments.

2. CAN IMMUNOCOMPROMISED CHILDREN ATTEND SCHOOL?

The Immune Deficiency Foundation states, "Years ago, a diagnosis of a PI [primary immune deficiency] meant extremely compromised lives... Today, with early diagnosis and appropriate therapies, many patients diagnosed with a PI can live healthy, productive lives." Modern treatments have reduced the risk of many immunocompromised children so that they are able to attend school.[1]

Children who are not severely immunocompromised can attend school with the approval of their doctor.

3. CAN IMMUNOCOMPROMISED SCHOOLCHILDREN BE VACCINATED?

Immunocompromised schoolchildren have the option to receive all the vaccines licensed for children in the United States, except for the live virus vaccines (such as vaccines targeting measles, mumps, rubella, or varicella infections).[2] Although vaccination often results in protective levels of antibodies in immunocompromised children,[3-7] clinical vaccine safety trials typically exclude immunocompromised subjects.[8] In addition, vaccines have not been evaluated for their potential to cause cancer, genetic mutations or impaired fertility in the general or immunocompromised population.[9] Due to these limitations, it is not known whether the benefit of vaccinating an immunocompromised child outweighs the risk of vaccine injury to that child.

4. DOES THE VACCINATION STATUS OF OTHER SCHOOLCHILDREN POSE A SIGNIFICANT RISK TO IMMUNOCOMPROMISED SCHOOLCHILDREN?

The vaccination status of other schoolchildren does not pose a significant risk to immunocompromised schoolchildren for the following reasons (Table 1):

- Some vaccines cannot prevent the spread of the bacteria or viruses they target.
- Immune globulin (plasma containing antibodies) is available for immunocompromised children exposed to certain infectious diseases.
- Some infectious diseases rarely cause complications in immunocompromised schoolchildren.
- Not all infectious diseases are contagious.
- Some infectious diseases are not spread in schools.

Immunocompromised schoolchildren are not put at significant risk by the vaccination status of other schoolchildren.

Table 1: Why the Vaccination Status of Other Schoolchildren Is Not a Significant Risk to Immunocompromised Schoolchildren

Some vaccines cannot prevent the spread of the bacteria or viruses they target.

Children vaccinated with the diphtheria, tetanus, and pertussis (whooping cough) vaccine (DTaP) or the inactivated polio vaccine (IPV) can still be infected with diphtheria-causing bacteria, pertussis bacteria, or poliovirus and spread them to others, even with mild or no symptoms of their own.[10-13] The influenza vaccines (TIV and LAIV) have not been observed to significantly reduce the spread of influenza.[14,15] About half of schoolchildren vaccinated with the measles, mumps, and rubella (MMR) vaccine can still be infected with measles virus and spread it to others, even with mild or no symptoms of their own.[16-19]

Immune globulin (plasma containing antibodies) is available for immunocompromised children exposed to certain infectious diseases.

Immune globulin (IG) is available for the prevention of severe symptoms in immunocompromised children exposed to measles or rubella (IG does not provide protection for fetuses of expectant mothers infected with rubella).[20,21] Varicella-zoster immune globulin (VIG) is available for the prevention of severe symptoms in immunocompromised children exposed to varicella (chickenpox).[22] Hepatitis B immune globulin (HBIG) and tetanus immune globulin (TIG) are also available for immunocompromised children.[2]

Some infectious diseases rarely cause complications in immunocompromised schoolchildren.

Fatal cases of mumps are very rare in schoolchildren (1 mumps death per 100,000 mumps cases),[23] and immunocompromised children have been observed to recover just as well from mumps as the general population.[24] Severe cases of pertussis or rubella rarely occur in schoolchildren, and being immunocompromised has not been observed to be a significant risk factor for complications of pertussis or rubella in schoolchildren.[25,26]

Not all infectious diseases are contagious.

Tetanus is not a communicable disease; that is, it cannot spread from person to person under any circumstances.[27]

Some infectious diseases are not spread in schools.

Hepatitis B is not spread by kissing, hugging, holding hands, coughing, sneezing, or sharing eating utensils,[28] and the main routes of hepatitis B transmission (sexual contact, injection drug use, or being born to an infected mother)[29] do not occur in school. Nearly all cases of *Haemophilus influenzae* type b (Hib) occur among children younger than 5 years of age; therefore, nearly all Hib transmission does not occur in school.[30] Human papillomavirus (HPV) is sexually transmitted and is therefore not spread in school.[31]

All references are available at physiciansforinformedconsent.org/immunocompromised-schoolchildren.

REFERENCES

1. Blaese RM, Ludwig M, Buckley R, Seymour JW, Dodds M. Immune Deficiency Foundation school guide for students with primary immunodeficiency diseases. 3rd ed. Towson (MD): Immune Deficiency Foundation; 2014. 6.
2. Centers for Disease Control and Prevention. Recommendations of the Advisory Committee on Immunization Practices (ACIP): use of vaccines and immune globulins in persons with altered immunocompetence. MMWR. 1993 Apr;42(No. RR-04).
3. Ercan TE, Soycan LY, Apak H, Celkan T, Ozkan A, Akdenizli E, Kasapçopur O, Yildiz I. Antibody titers and immune response to diphtheria-tetanus-pertussis and measles-mumps-rubella vaccination in children treated for acute lymphoblastic leukemia. J Pediatr Hematol Oncol. 2005 May;27(5):273-7.
4. Feldman S, Gigliotti F, Shenep JL, Roberson PK, Lott L. Risk of *Haemophilus influenzae* type b disease in children with cancer and response of immunocompromised leukemic children to a conjugate vaccine. J Infect Dis. 1990 May;161(5):926-31.
5. Hodges GR, Davis JW, Lewis HD Jr, Siegel CD, Chin TD, Clark GM, Noble GR. Response to influenza A vaccine among high-risk patients. South Med J. 1979 Jan;72(1):29-32.
6. Moss WJ, Clements CJ, Halsey NA. Immunization of children at risk of infection with human immunodeficiency virus. Bull of the World Health Organ. 2003;81(1):62,64.
7. Barbi M, Bardare M, Luraschi C, Zehender G, Clerici Schoeller M, Ferraris G. Antibody response to inactivated polio vaccine (E-IPV) in children born to HIV positive mothers. Eur J Epidemiol. 1992 Mar;8(2):211-6.
8. Centers for Disease Control and Prevention. Manual for the surveillance of vaccine-preventable diseases. 5th ed. Miller ER, Haber P, Hibbs B, Broder K. Chapter 21: surveillance for adverse events following immunization using the Vaccine Adverse Event Reporting System (VAERS). Atlanta: Centers for Disease Control and Prevention; 2011. 1,2.
9. U.S. Food and Drug Administration. Silver Spring (MD): U.S. Food and Drug Administration. Vaccines licensed for use in the United States; [updated 2018 Feb 14; cited 2018 Feb 27]. https://www.fda.gov/BiologicsBloodVaccines/Vaccines/ApprovedProducts/Ucm093833.htm.
10. Miller LW, Older JJ, Drake J, Zimmerman S. Diphtheria immunization. Effect upon carriers and the control of outbreaks. Am J Dis Child. 1972 Mar;123(3):197-9.
11. Warfel JM, Zimmerman LI, Merkel TJ. Acellular pertussis vaccines protect against disease but fail to prevent infection and transmission in a nonhuman primate model. Proc Natl Acad Sci USA. 2014 Jan 14;111(2):787-92.
12. Cuba IPV Study Collaborative Group. Randomized, placebo-controlled trial of inactivated poliovirus vaccine in Cuba. N Engl J of Med. 2007 Apr 12;356(15):1536-44.
13. Centers for Disease Control and Prevention. Washington, D.C.: U.S. Department of Health and Human Services. U.S. National Authority for Containment of Poliovirus: the need for containment; [cited 2019 Jul 21]. https://www.cdc.gov/cpr/polioviruscontainment/containment.htm.
14. Thomas RE, Jefferson T, Lasserson TJ. Influenza vaccination for healthcare workers who care for people aged 60 or older living in long-term care institutions. Cochrane Database Syst Rev. 2016 Jun 2;(6) CD005187:2.
15. Ohmit SE, Petrie JG, Malosh RE, Cowling BJ, Thompson MG, Shay DK, Monto AS. Influenza vaccine effectiveness in the community and the household. Clin Infect Dis. 2013 May;56(10):1363.
16. **Children with measles antibody levels less than 900 mIU/mL are susceptible to subclinical infection with measles virus but not to clinical infection. About 35% of vaccinated children 7 years of age have a measles antibody level less than 900 mIU/mL. This level steadily declines through childhood, resulting in about 60% of children 15 years of age with a measles antibody level less than 900 mIU/mL. Consequently, about half of schoolchildren are susceptible to infection with measles virus.**
    - LeBaron CW, Beeler J, Sullivan BJ, Forghani B, Bi D, Beck C, Audet S, Gargiullo P. Persistence of measles antibodies after 2 doses of measles vaccine in a postelimination environment. Arch Pediatr Adolesc Med. 2007 Mar;161(3):294-301.
17. Pedersen IR, Mordhorst CH, Glikmann G, von Magnus H. Subclinical measles infection in vaccinated seropositive individuals in arctic Greenland. Vaccine. 1989 Aug;7(4):345-8.
18. Chen RT, Markowitz LE, Albrecht P, Stewart JA, Mofenson LM, Preblud SR, Orenstein WA. Measles antibody: reevaluation of protective titers. J Infect Dis. 1990 Nov;162(5):1036-42.
19. Mizumoto K, Kobayashi T, Chowell G. Transmission potential of modified measles during an outbreak, Japan, March–May 2018. Euro Surveill. 2018 Jun 14;23(24):1800239.
20. McLean HQ, Fiebelkorn AP, Temte JL, Wallace GS; Centers for Disease Control and Prevention. Prevention of measles, rubella, congenital rubella syndrome, and mumps, 2013: summary recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR. 2013 Jun;62(RR-04):17,24.
21. Young MK, Cripps AW, Nimmo GR, van Driel ML. Post-exposure passive immunisation for preventing rubella and congenital rubella syndrome. Cochrane Database Syst Rev. 2015 Sep 9;(9)CD010586:3.
22. Centers for Disease Control and Prevention. Varicella-zoster immune globulin for the prevention of chickenpox: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR. 1984 Feb;33(7):84-90,95-100.
23. **Before the mumps vaccine was licensed in 1967, nearly everyone contracted mumps in childhood. In 1966, there were 43 mumps deaths out of 4 million cases (the average size of a birth cohort in the 1960s): about 1 mumps death per 100,000 mumps cases.**
    - Wagenvoort JH, Harmsen M, Boutahar-Trouw BJ, Kraaijeveld CA, Winkler KC. Epidemiology of mumps in the Netherlands. J Hyg (Lond). 1980 Dec;85(3):313-26.
    - Centers for Disease Control and Prevention. Reported cases and deaths from vaccine preventable diseases, United States, 1950-2013. Epidemiology and prevention of vaccine-preventable diseases. Hamborsky J, Kroger A, Wolfe S, eds. 13th ed. Washington, D.C.: Public Health Foundation; 2015. Appendix E3.
24. de Boer AW, de Vaan GA. Mild course of mumps in patients with acute lymphoblastic leukaemia. Eur J Pediatr. 1989 Jun;148(7):618-9.
25. Centers for Disease Control and Prevention. Epidemiology and prevention of vaccine-preventable diseases. 13th ed. Hamborsky J, Kroger A, Wolfe S, editors. Washington, D.C.: Public Health Foundation; 2015. 262,263,265.
26. Centers for Disease Control and Prevention. Epidemiology and prevention of vaccine-preventable diseases. 13th ed. Hamborsky J, Kroger A, Wolfe S, editors. Washington, D.C.: Public Health Foundation; 2015. 325,326.
27. Centers for Disease Control and Prevention. Epidemiology and prevention of vaccine-preventable diseases. 13th ed. Hamborsky J, Kroger A, Wolfe S, editors. Washington, D.C.: Public Health Foundation; 2015. 345.
28. Centers for Disease Control and Prevention. Washington, D.C.: U.S. Department of Health and Human Services. Hepatitis B questions and answers for the public; [cited 2019 Jul 15]. https://www.cdc.gov/hepatitis/hbv/bfaq.htm#bFAQc01.
29. Centers for Disease Control and Prevention. Epidemiology and prevention of vaccine-preventable diseases. 13th ed. Hamborsky J, Kroger A, Wolfe S, editors. Washington, D.C.: Public Health Foundation; 2015. 154-5.
30. Centers for Disease Control and Prevention. Epidemiology and prevention of vaccine-preventable diseases. 13th ed. Hamborsky J, Kroger A, Wolfe S, editors. Washington, D.C.: Public Health Foundation; 2015. 120.
31. Centers for Disease Control and Prevention. Epidemiology and prevention of vaccine-preventable diseases. 13th ed. Hamborsky J, Kroger A, Wolfe S, editors. Washington, D.C.: Public Health Foundation; 2015. 177.

THE DANGER OF ELIMINATING VACCINE EXEMPTIONS & CURTAILING VACCINE CRITICISM

ICAN
Informed Consent Action Network

Prior to any medical procedure, the U.S. Department of Health & Human Service ("**HHS**") explains that the "voluntary consent of the human subject is absolutely essential."[1] **Coercion invalidates informed consent.**[2] Infringing this right by eliminating vaccine exemptions and curtailing criticism is unethical and un-American given the following facts:

PHARMA HAS NO INCENTIVE TO ASSURE VACCINE SAFETY

1. Immunity from Liability for Vaccine Harms. By the early 1980s, pharmaceutical companies were facing crippling liability for injuries to children caused by their vaccines.[3] Instead of letting these market forces drive them to develop safer vaccines, Congress passed the National Childhood Vaccine Injury Act (the "**1986 Act**") which eliminated pharmaceutical company liability for injuries caused by their vaccine products.[4]

2. Pharmaceutical Company Misconduct. Since 1986, Merck, GSK, Sanofi and Pfizer have paid billions of dollars for misconduct and injuries related to their drug products.[5] These same companies manufacture almost all childhood vaccines, but because of the 1986 Act, cannot similarly be held accountable for misconduct and injuries related to their vaccine products.

HHS CONFLICTED FROM ASSURING VACCINE SAFETY

3. HHS Must Defend Against Any Claim of Vaccine Injury. After eliminating liability for pharmaceutical companies, the 1986 Act established the Vaccine Injury Compensation Program ("**Vaccine Court**"), part of the U.S. Court of Federal Claims, to compensate people injured by vaccines.[6] Under the 1986 Act, HHS is the defendant in Vaccine Court and is legally obligated to defend against any claim that a vaccine causes injury.[7] There is no right to discovery in Vaccine Court and HHS is represented by the formidable resources of the U.S. Department of Justice ("**DOJ**").[8] In nearly every case the injured person bears the burden to prove causation.[9] Despite these hurdles, since 1986, HHS has paid over $4 billion for vaccine injuries.[10]

4. HHS Incriminates Itself if it Publishes or Admits a Vaccine Can Cause a Harm. If HHS publishes any study supporting that a vaccine causes a harm, that study will then be used against HHS in Vaccine Court.[11] This greatly limits HHS's incentive to publish safety studies.

5. CDC's Childhood Vaccine Schedule Was Created by Pharma Insiders. Congress has repeatedly found that the members of the FDA and CDC committees responsible for approving most of the currently licensed and recommended childhood vaccines had serious conflicts of interests with pharmaceutical companies.[12]

VACCINE SAFETY: CONCERNS & LIMITATIONS

6. HHS Fails to Perform Basic Vaccine Safety Requirements. After eliminating the market forces that assured vaccine safety, Congress made HHS directly responsible for vaccine safety pursuant to a section of the 1986 Act entitled the "Mandate for safer childhood vaccines."[13] As HHS recently

[1] https://ori.hhs.gov/chapter-3-The-Protection-of-Human-Subjects-nuremberg-code-directives-human-experimentation
[2] https://www.utcomchatt.org/docs/biomedethics.pdf
[3] https://www.nap.edu/read/2138/chapter/2#2 ("The litigation costs associated with claims of damage from vaccines had forced several companies [by 1986] to end their vaccine … programs as well as to stop producing already licensed vaccines.")
[4] 42 U.S.C. § 300aa-11 ("No person may bring a civil action for damages in the amount greater than $1,000 or in an unspecified amount against a vaccine administrator or manufacturer in a State or Federal court for damages arising from a vaccine-related injury or death."); Bruesewitz v. Wyeth LLC, 562 U.S. 223, 243 (2011) ("the National Childhood Vaccine Injury Act preempts all design-defect claims against vaccine manufacturers brought by plaintiffs who seek compensation for injury or death caused by vaccine side effects")
[5] https://www.citizen.org/sites/default/files/2408.pdf
[6] 42 U.S.C. § 300aa-12 ("In all proceedings brought by the filing of a petition [in Vaccine Court] the Secretary [of HHS] shall be named as the respondent."); https://www.congress.gov/106/crpt/hrpt977/CRPT-106hrpt977.pdf (HHS amended the Vaccine Court rules to make it extremely difficult to obtain compensation and "DOJ attorneys make full use of the apparently limitless resources available to them," "pursued aggressive defenses in compensation cases," "establish[ed] a cadre of attorneys specializing in vaccine injury" and "an expert witness program to challenge claims.")
[7] Ibid.
[8] Ibid.
[9] The 1986 Act created a Vaccine Injury Table (the "**Table**") which was intended to permit the Vaccine Court to quickly compensate certain common vaccine injuries. 42 U.S.C. § 300aa-12. For Table injuries, the burden shifts to HHS to prove the vaccine is not the cause. 42 U.S.C. § 300aa-13. After passage of the 1986 Act, almost 90% of claims were Table claims and quickly settled. Stevens v. Secretary of HHS, No. 99-594V (Office of Special Masters 2001). However, in the 1990s, HHS amended the Table such that now 98% of new claims are off-Table. http://www.gao.gov/assets/670/667136.pdf. As a result, injured children "must prove that the vaccine was the cause" in almost all cases. https://www.ncbi.nlm.nih.gov/nlmcatalog/101633437
[10] https://www.hrsa.gov/sites/default/files/hrsa/vaccine-compensation/data/monthly-stats-february-2019.pdf
[11] *See fn.* 6 and 9.
[12] http://vaccinesafetycommission.org/pdfs/Conflicts-Govt-Reform.pdf
[13] 42 U.S.C. § 300aa-27

conceded in federal court, it has not performed even the basic requirements of this section, such as submitting reports to Congress on how HHS has improved vaccine safety.[14]

7. Pediatric Vaccine Clinical Trials (i) Lack Placebos and (ii) Are Too Short. The pivotal clinical trials relied upon to license childhood vaccines do not include a placebo-control group and safety review periods in these clinical trials are typically only days or months.[15] The safety profile for a pediatric vaccine is therefore not known before it is licensed and routinely used in children.[16]

8. Post-Licensure Safety. After licensure and use by the public, federal law requires that the package insert for each vaccine include "*only* those adverse events for which there is some basis to believe there is a *causal* relationship between the drug and the occurrence of the adverse event."[17] Inserts for childhood vaccines include over one hundred serious immune, neurological and other chronic conditions that their manufacturers had a basis to believe are caused by their vaccines.[18]

9. Prevalence of Vaccine Harm. The CDC's Vaccine Adverse Events Reporting System ("**VAERS**"), to which doctors and patients may *voluntarily* report adverse vaccine events, received 58,381 reports in 2018, including 412 deaths, 1,237 permanent disabilities, and 4,217 hospitalizations.[19] An HHS-funded three-year review by Harvard Medical School of 715,000 patients stated that "fewer than 1% of vaccine adverse events are reported" to VAERS.[20] This could mean there are a hundredfold more adverse vaccine events than are reported to VAERS. The CDC has nonetheless refused to mandate or automate VAERS reporting.[21]

10. Children Susceptible to Vaccine Injury. While the Institute of Medicine ("**IOM**") has explained that "most individuals who experience an adverse reaction to vaccines have a preexisting susceptibility," HHS and CDC have failed to conduct studies to identify children susceptible to vaccine harms while at the same time recommending vaccines for all children.[22]

11. Carcinogenicity, Mutagenicity & Infertility. Most vaccines have never been evaluated for their potential to cause cancer, mutate genes or cause infertility.[23]

12. Autism. Autism is the most controversial of the claimed vaccine injuries and the one HHS and CDC declare they have thoroughly studied. Most parents with autistic children claim vaccines (including DTaP, Hep B, Hib, PCV13, and IPV, each injected 3 times by 6 months) are a cause of their child's autism.[24] The CDC tells these parents that "Vaccines Do Not Cause Autism."[25] However, there is no science to support this claim for almost all vaccines. For example, reports from the IOM in 1991 and 2012, and HHS in 2014, tried but failed to identify any study to support that DTaP does not cause autism.[26] The same is true for Hep B, Hib, PCV 13, and IPV.[27] The only vaccine actually studied with regard to autism is MMR, and a Senior CDC Scientist claims the CDC did find an increased rate of autism after MMR in the only MMR/autism study ever conducted by the CDC with American children.[28] Moreover, HHS's primary autism expert in Vaccine Court recently provided an affidavit explaining that vaccines can cause autism in some children.[29] Given the lack of studies regarding vaccines and autism, it should come as no surprise that there is a dearth of scientific studies that support the CDC's other claims regarding vaccine safety.

13. HHS Refuses to Conduct Vaccinated Vs. Unvaccinated Studies of Vaccine Schedule. A true epidemic in the U.S. is the fact that 1 in 2 children have an autoimmune, developmental, neurological, or chronic disorder.[30] These conditions have sharply

[14] http://icandecide.org/government/ICAN-HHS-Stipulated-Order-July-2018.pdf
[15] https://icandecide.org/hhs/ICAN-Reply.pdf (see Section I)
[16] Ibid.
[17] https://icandecide.org/hhs/ICAN-Reply.pdf (see Appendix B)
[18] Ibid.
[19] https://wonder.cdc.gov/vaers.html
[20] https://healthit.ahrq.gov/sites/default/files/docs/publication/r18hs017045-lazarus-final-report-2011.pdf
[21] https://icandecide.org/hhs/ICAN-Reply.pdf (see Section III)
[22] https://icandecide.org/hhs/ICAN-Reply.pdf (see Section V)
[23] https://www.fda.gov/biologicsbloodvaccines/vaccines/approvedproducts/ucm093833.htm
[24] https://www.ncbi.nlm.nih.gov/pubmed/16685182; https://www.ncbi.nlm.nih.gov/pubmed/25398603; https://www.ncbi.nlm.nih.gov/pubmed/16547798; https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1448378/
[25] https://www.cdc.gov/vaccinesafety/concerns/autism.html
[26] https://www.nap.edu/read/1815/chapter/2#7; https://www.nap.edu/read/13164/chapter/12?term=autism#545; https://www.ncbi.nlm.nih.gov/books/NBK230053/pdf/Bookshelf_NBK230053.pdf
[27] https://icandecide.org/hhs/ICAN-Reply.pdf (see Section VI)
[28] http://www.rescuepost.com/files/william-thompson-statement-27-august-2014-3.pdf; https://soundcloud.com/fomotion/cdc-whistle-blower-full-audio; https://www.c-span.org/video/?c4546421/rep-bill-posey-calling-investigation-cdcs-mmr-reasearch-fraud
[29] http://icandecide.org/documents/zimmerman.pdf
[30] https://www.ncbi.nlm.nih.gov/pubmed/21570014

risen in lock-step with the increases in the CDC's recommended vaccine schedule.[31] That schedule has risen from 7 injections of just 2 vaccines in 1986 to the current total of 50 injections of 12 different vaccines.[32] The need to compare health outcomes of vaccinated and unvaccinated children is urgent. In 2017, a seminal study found that babies receiving the DTP vaccine died at 10 times the rate of unvaccinated babies.[33] In another study, children received influenza vaccine or a saline placebo; while both groups had a similar rate of influenza, the vaccinated group had a 440% increased rate of non-influenza infections.[34] A recent pilot study from the School of Public Health at Jackson State University found that 33% of vaccinated preterm babies had a neuro-developmental disorder compared to 0% of the unvaccinated preterm babies; and vaccinated children in this study had an increased risk of 290% for eczema, 390% for allergies, 420% for ADHD, 420% for autism, and 520% for learning disabilities.[35] Nonetheless, HHS and CDC refuse to publish any studies comparing the health outcomes between vaccinated and unvaccinated children.[36]

MMR VACCINE

14. Measles is a Mild Childhood Illness. The mortality rate from measles declined by over 98% between 1900 and 1962 as living conditions improved in this country.[37] In 1962, a year before the first measles vaccine, the CDC reported a total of 408 deaths.[38] That amounts to 1 in 500,000 Americans at a time when measles infected nearly every American.[39]

15. Eliminating Measles Has Increased Cancer Rates. Eliminating measles has increased cancer rates. For example, the International Agency for Research on Cancer found that individuals who never had measles had a 66% increased rate of Non-Hodgkin Lymphoma and a 233% increased rate of Hodgkin Lymphoma.[40] Combined, these cancers killed 20,960 Americans in 2018.[41] As another example, individuals who never had measles, mumps or rubella had a 50% increased rate of ovarian cancer.[42] In 2018, ovarian cancer killed 14,070 Americans.[43] Eliminating measles in this country has caused more deaths from cancer.

16. Eliminating Measles Has Increased Heart Disease. A 22-year prospective study of over 100,000 individuals in Japan revealed that "measles and mumps, especially in case of both infections, were associated with lower risks of mortality from atherosclerotic CVD [heart disease]."[44] Heart disease killed 610,000 Americans in 2018.[45] Eliminating our ecological relationship with measles, mumps and rubella has had serious unintended consequences.

17. Side effects from MMR vaccine. The MMR vaccine has serious risks. For example, the MMR vaccine causes seizures in about 1 in 640 children, five times the rate from measles, as well as "thrombocytopenic purpura," "chronic arthritis," and "brain damage."[46] However, because the MMR was not licensed based on a placebo-controlled clinical trial and post-licensure studies are limited, there are many suspected harms the CDC has yet to confirm or rule out, such as those listed on Merck's package insert for the MMR.[47]

18. Waning Immunity. While the vaccination rate for measles in the United States has been stable over the last 20 years, what has changed is that Americans who have had measles (which confers lifetime immunity) are being replaced by those vaccinated with MMR (which does not typically confer lifetime immunity).[48] MMR produces no immunity in 2% to 10% of vaccinees; and 22 years after two doses of MMR approximately 33% of vaccinees are again

[31] https://www.ncbi.nlm.nih.gov/pubmed/20159870
[32] https://www.cdc.gov/vaccines/schedules/images/schedule1983s.jpg; https://www.cdc.gov/vaccines/schedules/downloads/child/0-18yrs-child-combined-schedule.pdf
[33] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5360569/
[34] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3404712/
[35] http://www.oatext.com/pdf/JTS-3-186.pdf; http://www.oatext.com/pdf/JTS-3-187.pdf
[36] https://icandecide.org/hhs/ICAN-Reply.pdf (see Section VII)
[37] https://www.cdc.gov/nchs/data/vsus/vsrates1940_60.pdf; https://www.cdc.gov/nchs/data/vsus/VSUS_1962_2A.pdf
[38] https://www.cdc.gov/nchs/data/vsus/VSUS_1962_2A.pdf
[39] Ibid.; https://www.census.gov/library/publications/1962/compendia/statab/83ed.html
[40] https://www.ncbi.nlm.nih.gov/pubmed/16406019
[41] https://seer.cancer.gov/statfacts/html/nhl.html; https://seer.cancer.gov/statfacts/html/hodg.html
[42] https://www.ncbi.nlm.nih.gov/pubmed/16490323
[43] https://seer.cancer.gov/statfacts/html/ovary.html
[44] https://www.ncbi.nlm.nih.gov/pubmed/26122188
[45] https://www.cdc.gov/heartdisease/facts.htm
[46] https://www.hrsa.gov/sites/default/files/vaccinecompensation/vaccineinjurytable.pdf; https://www.cdc.gov/vaccines/hcp/vis/vis-statements/mmr.pdf; https://physiciansforinformedconsent.org/measles/vrs/ (since the measles death from 1959 to 1962 was appx. 400 per 4 million cases https://www.cdc.gov/vaccines/pubs/pinkbook/downloads/appendices/e/reported-cases.pdf and death to seizure ratio is appx. 3.25 https://www.cdc.gov/vaccines/pubs/pinkbook/meas.html this amounts to 1 seizure in 3,095 measles cases).
[47] https://www.fda.gov/downloads/BiologicsBloodVaccines/UCM123789.pdf
[48] https://www.cdc.gov/vaccines/pubs/pinkbook/downloads/appendices/G/coverage.pdf

potentially susceptible to measles.[49] The proportion after 30 years is even higher.[50] Yet the only focus is on children whose parents have reason to believe the MMR may cause them harm, while ignoring the efficacy issues with this vaccine.

OTHER VACCINES

19. DTaP Vaccine. According to the FDA, those vaccinated with DTaP will have fewer symptoms of pertussis, but will become infected and transmit pertussis, and "will be more susceptible to pertussis throughout their lifetimes."[51] This means the children vaccinated for pertussis are more likely to catch and spread pertussis as asymptomatic carriers, while the unvaccinated are less likely to catch pertussis (and when they do will have symptoms and know to stay home).[52] Since pertussis is very common and more of a concern than measles, as long as children vaccinated for pertussis are permitted to attend school, children not vaccinated for measles should also be permitted to attend school. In any event, the immunity provided by DTaP for pertussis, tetanus, and diphtheria wanes within a few years.[53]

20. Inactivated Polio Vaccine. For the last 20 years, the only polio vaccine used in the U.S. is inactivated polio vaccine ("**IPV**"), which is injected intramuscularly, after it was determined that the oral polio vaccine can cause paralysis.[54] Polio is spread through fecal to oral contamination, and IPV does not prevent colonization and transmission of polio; it only potentially prevents polio from traveling to the spinal column.[55] Hence, those vaccinated or not vaccinated with IPV can equally become infected and transmit polio; but, it is the vaccinated who are considered less likely to have symptoms and thus more likely to spread polio.

21. Chicken Pox Vaccine. Children vaccinated for chicken pox can spread chicken pox virus for six weeks after vaccination.[56] Moreover, the immunity from this vaccine wanes and, absent natural boosting from exposure to chicken pox virus, can lead to shingles.[57] The increased risk of shingles from use of this vaccine is why countries, such as the United Kingdom, have not added it to their routine vaccine schedule.[58]

22. Note. There are additional efficacy and safety issues with the above vaccines and other vaccines not addressed due to space constraints. For example, aluminum adjuvant particles in vaccines, which animal studies reveal deposit in brain and bones, or the millions of snippets of human DNA cultured from the cell lines of aborted fetuses in certain vaccines.[59]

ADDITIONAL INFORMATION

The foregoing highlights a few of the vaccine safety and efficacy issues necessitating the need for informed consent for vaccination and the ability to openly criticize our vaccine policies.

At the least, the following should occur before censoring concerns regarding vaccine safety:

a. Vaccine safety duties should be removed entirely from HHS and placed into an independent board;

b. Pharmaceutical companies should be liable for injuries caused by their vaccine products; and

c. The childhood vaccine schedule and each vaccine should be safety tested in a properly sized long-term placebo-controlled clinical trial.

For additional information or to arrange a presentation, please contact Cat Layton at cat@icandecide.org

[49] https://www.ncbi.nlm.nih.gov/pubmed/17339511
[50] Ibid.
[51] https://www.ncbi.nlm.nih.gov/pubmed/24277828; https://www.ncbi.nlm.nih.gov/pubmed/30793754; https://www.ncbi.nlm.nih.gov/pubmed/29180031 ("neither DTP, nor DTaP or Tdap prevent asymptomatic infection and silent transmission of the pathogen")
[52] Ibid.
[53] Ibid.
[54] http://polioeradication.org/polio-today/polio-prevention/the-vaccines/ipv/
[55] Ibid.
[56] https://www.fda.gov/downloads/BiologicsBloodVaccines/Vaccines/ApprovedProducts/UCM142813.pdf
[57] https://www.ncbi.nlm.nih.gov/pubmed/22659447; https://www.ncbi.nlm.nih.gov/pubmed/24275643
[58] https://www.nhs.uk/common-health-questions/childrens-health/why-are-children-in-the-uk-not-vaccinated-against-chickenpox/
[59] http://vaccinepapers.org/wp-content/uploads/vaccine_papers_brochure_8.5x11.pdf; https://www.cdc.gov/vaccines/pubs/pinkbook/downloads/appendices/B/excipient-table-2.pdf; https://www.ncbi.nlm.nih.gov/pubmed/5949788; https://www.ncbi.nlm.nih.gov/pmc/articles/PMC274969/; https://www.ncbi.nlm.nih.gov/pubmed/29108182