March, 2017

Vaccine Dangers: Examine The Evidence

Scientific evidence published in recent years shows that vaccines have great potential for causing brain and immune system injury. The emerging understanding of vaccine injury mechanisms is only just beginning to be explored in human studies.

New scientific evidence strongly suggests that vaccines are at least partly responsible for the alarming increase in neurological and immune system disorders in children, including autism. The scientific evidence frequently cited as proof that vaccines do not cause autism is limited to the MMR vaccine and the preservative thimerosal. The MMR and thimerosal studies are not relevant to the emerging body of compelling science presented below.

Vaccine injury is a vast and complex topic. This document focuses on the three critical issues related to vaccine dangers: Healthy user bias, aluminum adjuvant toxicity, and immune activation injury.

The American Academy of Pediatrics (AAP) circulates a list of 42 studies as evidence that vaccines are safe and do not cause autism or neurological disorders. The AAP's list and associated arguments are flawed in these ways:

1) 25 of the 42 studies look only at the MMR or the measles vaccine. The AAP refers to these studies as evidence that no vaccine causes autism. This is illogical, unscientific, and a misuse of the science. Studies of MMR cannot be used as evidence of safety for other vaccines. Every vaccine contains different ingredients. For example, the MMR vaccine does not contain aluminum adjuvant, a dangerous ingredient proven to cause brain inflammation and cognitive and behavioral deficits in animals at the same dosages given to human infants.

2) The MMR-autism and other vaccine studies are susceptible to healthy user bias, a type of selection bias that conceals evidence of harm. Healthy user bias is a systematic source of error. It may affect all the MMR-autism studies and none adequately control for it. Healthy user bias renders many studies of vaccine safety incapable of actually establishing safety.

3) None of the studies listed by the AAP include a fully-unvaccinated control group.

4) None of the studies listed by the AAP support the safety of aluminum adjuvant.

5) None of the studies listed by the AAP provide evidence that vaccines do not cause immune activation injury.

6) Two studies (Smith 2010 and DeStefano 2013) purport to show that general vaccine exposure (number of vaccines or vaccine antigens) is not associated with neurological disorders. These studies are fatally flawed because they do not include an unvaccinated control group.
Smith 2010 for example includes an analysis comparing groups that received 10.1 and 11.8 vaccines. Such a small difference in exposure is not able to detect adverse effects. Hence, it cannot be used as evidence of safety.
DeStefano 2013 focuses on number of antigens, but there is no evidence the number of antigens is a rational metric for vaccine risk. It's the aluminum adjuvant that creates the danger, but adjuvant exposure is not considered in this study. Also, this study has no unvaccinated controls. The DeStefano 2013 study is essentially meaningless.

Below is a list of studies that raise serious concerns about vaccine safety. The issues of healthy user bias, aluminum adjuvant toxicity and immune activation injury have been almost completely overlooked by the medical community.

Citation List Provided by VaccinePapers.org

Contact: vaccinepapers@gmail.com

Healthy User Bias (HUB)

Healthy user bias is a type of selection bias that is particularly relevant to vaccine safety research. HUB is created when people with health problems avoid vaccination, or when healthy people choose vaccination. When this occurs, the vaccinated have better baseline health, which is erroneously credited to the vaccine.

Vaccine safety studies typically use administrative data (data collected by government agencies , HMOs and insurance companies), and HUB is present in this type of data. With administrative data, researchers can not control who receives the vaccine and who does not. HUB creates the appearance that vaccines have dramatic, diverse and implausible beneficial effects. For example, HUB is likely responsible for the dramatic low mortality associated with use of the influenza vaccine.

HUB is universally acknowledged as a problem (even by Dr Offit!), but is rarely addressed in vaccine research. Vaccine safety studies almost never attempt to control for this potent source of error. HUB can be strong enough to reverse a study outcome, making very dangerous vaccines appear to be safe or powerfully beneficial.

Citation	Summary	Citation Quotes
Confounding in Studies of Adverse Reactions to Vaccines Fine et al., American Journal of Epidemiology, Vol. 136, 1992	CDC researchers review healthy user bias, and show that this bias may explain why the studies of SIDS fail to consistently observe an association with vaccines. Calculations show that HUB can reverse study outcomes and make very dangerous vaccines appear safe. HUB can have a 5-10-fold impact on relative risk (RR). HUB can cause a true RR of 5 to become 1 or even less. HUB is a powerful bias that can completely conceal adverse effects of a vaccine.	"Confounding…is a general problem for studies of adverse reactions to prophylactic interventions, as they may be withheld from some individuals precisely because they are already at high risk of the adverse event." "If [epidemiological vaccine] studies are to prove useful, they must include strenuous efforts to control for such factors [i.e. HUB] in their design, analysis, and interpretation." "The magnitude of such confounding effects may be considerable."
Mortality Reduction with Influenza Vaccine in Patients with Pneumonia Outside "Flu" Season Eurich et al., AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE, Vol. 178, 2008.	This study looked at health outcomes in flu vaccine users when flu is <u>not</u> circulating (in the summer). Vaccinated subjects had far better health outcomes than unvaccinated controls, which cannot be caused by the vaccine. This result is likely due to the fact that the people in this study who chose vaccination were healthier to start with and had greater "health seeking" behavior. This is healthy user bias at work.	"The 51% reduction in mortality with vaccination initially observed in patients with pneumonia who did not have influenza was most likely a result of confounding." "…studies that have restricted their analyses to the influenza season have overestimated the potential mortality benefit of vaccination." "…our results empirically demonstrate that the mortality benefits of influenza vaccination may have been largely overestimated."

Healthy User and Related Biases in Observational Studies of Preventative Interventions: A Primer for Physicians Shrank et al., Journal of General Internal Medicine, Vol. 26, 2011.	This review considers a variety of evidence that non-randomized studies of preventative interventions (including vaccines) can be severely affected by healthy user bias (and related biases), it cites a strong effect of adherence in predicting health outcomes in the placebo arm of a drug trial.	"Clinicians should be skeptical when interpreting results of observational studies of preventive services that have not accounted for healthy user and related biases." "Another topic of recent debate is the magnitude of the benefit of influenza vaccination on mortality among elderly patients. Observational studies have typically reported 40%–50% reductions in all-cause mortality.[14,15] However, the observation that influenza vaccination appears to protect patients against mortality prior to the start of the flu season has cast doubt on these findings,[16] as have results indicating that improved statistical adjustment greatly reduces the apparent benefit.[17]"
The Introduction of Diphtheria-Tetanus-Pertussis and Oral Polio Vaccine Among Young Infants in an Urban African Community: A Natural Experiment Mogensen et al. EbioMedicine, January 31, 2017	The study has a clever design that eliminates healthy user bias. This study compared mortality among children receiving the DTP vaccine at older or younger ages. Variation of age at vaccination occurred because of infrequent (quarterly) vaccine clinic scheduling, and so is effectively random. Timing of vaccines is unrelated to child health. The study found a 5X higher mortality associated with the DTP vaccine.	"…the "unvaccinated" children in these [prior DTP vaccine] studies have usually been frail children too sick or malnourished to get vaccinated, and the studies may therefore have underestimated the negative effect of DTP." "When unvaccinated controls were normal children who had not yet been eligible for vaccination, mortality was 5 times higher for DTP-vaccinated children." "The negative effect of DTP was much worse in this natural experiment than has been reported in previous studies of DTP. This is presumably due to the "unvaccinated" control children in previous studies having been a frail subgroup too frail to get vaccinated."

Aluminum Adjuvant Toxicity

Most vaccines contain aluminum adjuvant, an ingredient necessary for inducing immunity. Al adjuvant comprises particles which are transported around the body by macrophages. Al adjuvant particles travel into the brain and other sensitive organs, where they cause chronic inflammation. Recent animal studies show that Al adjuvant can cause brain injury and behavioral disorders at dosages infants receive from vaccines (the same mcg/kg body weight). Al adjuvant causes microglial activation in the brain. Aluminum induces IL-6 expression in the brain; elevated IL-6 causes autism.

The CDC's case for Al adjuvant safety is based on two false claims: 1) that mice or rats ingesting 26 mg/kg/day Al do not experience adverse effects, and 2) that Al adjuvant particles have zero toxicity while in particulate form. These errors are fundamental to the fatally flawed Mitkus 2011 study used to defend aluminum adjuvant safety and cited by the CDC. Claims of Al adjuvant safety are indefensible in view of recent science on aluminum adjuvant kinetics and toxicity.

Citation	Summary	Quotes
Non-linear dose-response of aluminium hydroxide adjuvant particles: Selective low dose neurotoxicity Crepeaux et al., Toxicology, Vol. 375, 2016.	Shows that Al adjuvant at dosage of 200mcg/kg is transported into the brain, where it causes microglial activation (inflammation), and a 50-fold increase in brain aluminum concentration. Al adjuvant also caused behavioral abnormalities.	"In the context of massive development of vaccine-based strategies worldwide, the present study may suggest that aluminium adjuvant toxicokinetics and safety require reevaluation."
Neuroprotective Effect of Nanodiamond in Alzheimer's Disease Rat Model: a Pivotal Role for Modulating NF-κB and STAT3 Signaling Alawdi et al., Mol Neurobiol., Vol 2016.	Ingestion of 3.4 mg/kg/day Al caused cognitive impairment and a 4-fold increase of IL-6 in the brain. IL-6 causes autism. This is one of several studies disproving the foundation of the Mitkus 2011 analysis: that animals ingesting 26mg/kg/day Al do not experience adverse effects.	"The results also showed that aluminum administration increased the hippocampus pro-inflammatory cytokines TNF-α by 3.8-fold, IL-6 by 4-fold…compared to the normal control group."
Slow CCL2-dependent translocation of biopersistent particles from muscle to brain Khan et al. BMC Medicine, Vol. 11, 2013	Mice were injected intramuscularly with aluminum adjuvant, which was detected in the brain 1 year later, in particulate form. Transport into the brain is accelerated by the inflammatory chemokine CCL2 (also known as MCP-1). CCL2/MCP-1 is consistently elevated in autism and is induced by some vaccines. Transport effect of CCL2/MCP-1 indicates that macrophages are responsible for transporting the adjuvant particles.	"Intramuscular injection of alum-containing vaccine was associated with the appearance of aluminum deposits in distant organs, such as spleen and brain where they were still detected one year after injection." "…alum has high neurotoxic potential, and planning administration of continuously escalating doses of this poorly biodegradable adjuvant in the population should be carefully evaluated by regulatory agencies since the compound may be insidiously unsafe."

Aluminium-induced electrophysiological, biochemical and cognitive modifications in the hippocampus of aging rats Sethi et al., NeuroToxicology, Vol. 29, 2008	Rats ingested 5.6 mg/kg/day Al, which caused cognitive impairment and numerous adverse effects. This is one of several studies disproving the foundation of the Mitkus 2011 analysis: that animals ingesting 26mg/kg/day Al do not experience adverse effects.	"...aluminium intake impairs spatial learning abilities and increases anxiety by modifying brain functions at electrophysiological, biochemical and structural levels. We have also observed the magnitude of aluminium inflicted neurotoxicity was significantly higher in younger rats in comparison to older rats."
Curcumin attenuates aluminium-induced functional neurotoxicity in rats Sethi et al. Pharmacology, Biochemistry, and Behavior, Vol. 93, 2009.	Rats ingested 5.6 mg/kg/day Al, which caused behavioral abnormalities and neurotoxicity. This is one of several studies disproving the foundation of the Mitkus 2011 analysis: that animals ingesting 26mg/kg/day Al do not experience adverse effects.	"…aluminium enhances neurotoxicity by inflicting damage at sub-cellular structures. In accordance to previous reports we observed increased vacuolation, swollen mitochondria, and hyper-electron dense cells in Al-toxicated young and old rats compared to age-matched controls."
Administration of aluminium to neonatal mice in vaccine-relevant amounts is associated with adverse long term neurological outcomes Shaw et al. Journal of Inorganic Biochemistry, Vol. 128, 2013.	Mice were injected subcutaneously with at total of 550 mcg/kg Al adjuvant over the first 3 weeks of life. This dosage is approximately equal to the dosage received by infants according to the CDC vaccine schedule. Al adjuvant caused behavioral abnormalities and abnormal weight gain.	"…our current results are consistent with the existing evidence on the toxicology and pharmacokinetics of Al adjuvants which altogether strongly implicate these compounds as contributors to the rising prevalence of neurobehavioural disorders in children."
Aluminum hydroxide injections lead to motor deficits and motor neuron degeneration Shaw et al. Journal of Inorganic Biochemistry, Vol. 103, 2009.	Mice were injected with a total of 300mcg/kg Al adjuvant. The mice displayed pathological behavioral abnormalities and impaired learning and memory. The Al adjuvant caused brain inflammation and tau protein accumulation (associated with alzheimers disease). Aluminum was detected in the brain.	"…current results and our previous study have demonstrated significant behavioural and neuropathological outcomes with aluminum hydroxide."

Aluminum Adjuvant Linked to Gulf War Illness Induces Motor Neuron Death in Mice Petrik et al., Nanomolecular Medicine, Vol. 9, 2007.	Mice were injected with a total of 100mcg/kg Al adjuvant. The adjuvant caused impaired neuromuscular function, inflammation and apoptosis in the nervous system, and death of motor neurons in the spinal cord. Squalene (an adjuvant) did not produce these effects.	"Aluminum hydroxide induced both behavioral and motor deficits, and the increased presence of apoptotic neurons and in various regions of the central nervous system with significant motor neuron loss in the lumbar spinal cord." "…the continued use of aluminum adjuvants in various vaccines for the general public may have widespread health implications."

Immune Activation Injury

It is generally accepted that immune activation during early development causes brain injury and mental illnesses, including autism and schizophrenia. Specifically, the brain injury is caused by cytokines; autism is caused by elevated interleukin-6 (IL-6) and interleukin-17a (IL-17). Immune activation experiments have been replicated in monkeys. Hundreds of studies have been published on the phenomenon. Al adjuvant and vaccine adverse reactions induce cytokines (including IL-6) in the brain. An important study (Li et al. 2015) demonstrated that the hepatitis B vaccine adversely affects brain development by an immune activation mechanism, thereby demonstrating that vaccines can impact brain development via immune activation.

Dr Paul Patterson, a pioneering immune activation and autism researcher at Caltech, wrote this in 2006:

"Should we really be promoting universal maternal vaccination? The flu vaccine has been recommended routinely to pregnant women in the United States since 1957. The official policy of the CDC states that "administration of vaccines to women seeking prenatal care is an opportunity for preventative intervention that should not be wasted." Now you might say, "Well, of course, you don't want to get the flu if you're pregnant!" But remember that double-stranded RNA experiment—we activated the immune system, and it caused all these downstream effects on the fetus. And what does a vaccination do? It activates the immune system. That's the *point* of vaccination.
I think that universal vaccination of pregnant women could get us into a whole new set of problems."
--"Pregnancy, Immunity, Schizophrenia and Autism", Engineering & Science (A CalTech magazine), No. 3, 2006.

Citation	Summary	Quotes
Neonatal vaccination with bacillus Calmette–Guérin and hepatitis B vaccines modulates hippocampal synaptic plasticity in rats Li et al., Journal of Neuroimmunology, Vol. 288, 2015.	Rats given BCG or Hep B vaccines showed long-term changes in brain development. Hep B vaccine caused adverse changes in neuron function, and induced IL-6 in the brain. Also shows that vaccines affect brain development by an immune activation mechanism, and can cause chronic brain inflammation. Also shows that vaccines can interact via effects on immune function. This study establishes that vaccines can cause immune activation injury.	"Our work highlights a critical role of neonatal vaccination in synaptic plasticity…which suggests the necessity of further studies on the association of vaccination with brain development" "Immune activation early in life can significantly affect the development of neural processes."
Maternal immune activation yields offspring displaying mouse versions of the three core symptoms of autism Malkova et al., Brain, Behavior and Immunity, Vol. 26, 2012	Mice exposed to immune activation in utero displayed abnormal behavior characteristic of autism at maturity. Mice displayed abnormal communication, social interaction and repetitive behavior, the three defining characteristics of autism.	"Maternal immune activation (MIA) yields male offspring with deficient social and communicative behavior, as well as high levels of repetitive behaviors, all of which are hallmarks of autism."
Maternal Immune Activation Alters Fetal Brain Development through Interleukin-6 Smith et al., J. Neurosci, 2007	This study was the first to show that the cytokine interleukin-6 (IL-6) causes autistic behavior in an animal model of autism.	"The data identify interleukin-6 (IL-6) as a key mediator of the effects of maternal immune activation on fetal brain development."

Brain IL-6 elevation causes neuronal circuitry imbalances and mediates autism-like behaviors Wei et al., Biochimica et Biophysica Acta, Vol. 1822, 2012	Shows that chronic exposure of the brain to IL-6, beginning after birth, causes autistic behavioral abnormalities. IL-6 also created an excess of excitatory synapses, and a deficit of inhibitory synapses. This may explain hypersensitivity to lights and sounds in autism.	"IL-6 elevation in the brain could mediate autistic-like behaviors, possibly through the imbalances of neural circuitry and impairments of synaptic plasticity." "IL-6 elevation resulted in increased excitatory synaptic formation and a decreased number of inhibitory synapses."
Activation of the Maternal Immune System During Pregnancy Alters Behavioral Development of Rhesus Monkey Offspring Bauman et al., Biol Psychiatry, Vol. 75, 2014.	This study describes effects of maternal immune activation (MIA) in monkeys. MIA caused autistic behavioral abnormalties: social and communicative behavior deficits, and an increase in repetitive behavior. Findings match those observed in mice.	"In this rhesus monkey model, MIA yields offspring with abnormal repetitive behaviors, communication, and social interactions. These results extended the findings in rodent MIA models to more human-like behaviors resembling those in both autism and schizophrenia."
Maternal Immune Activation in Nonhuman Primates Alters Social Attention in Juvenile Offspring Machado et al., Biol Psychiatry, Vol. 77, 2015	This study reports that MIA causes abnormal visual scanning behavior in monkeys. Immune activation caused reduced visual scanning of images of faces and eyes. This is a characteristic of human autism. Human autistics avoid looking at faces and eyes.	"The use of noninvasive eye tracking extends the findings from rodent MIA models to more human-like behaviors resembling those in both autism spectrum disorder and schizophrenia."
Acquired Reversible Autistic Syndrome in Acute Encephalopathic Illness in Children Arch Neurol, Vol. 38, 1981	Describes 3 cases of sudden onset autism caused by infection and inflammation in the brain. Cases were 5, 7, and 11 years of age. This and similar case reports demonstrate that immune activation can cause autism in older children. This shows that the developing brain is sensitive to immune activation postnatally.	"…striking autistic features developed in previously normal children in the course of an acute encephalopathic illness…" "Cases are…reversible autistic syndrome…affording some insight into the neurological substrate of that syndrome."
Impaired synaptic development in a maternal immune activation mouse model of neurodevelopmental disorders Coiro, et al. Brain, Behavior, and Immunity, Vol. 50, 2015.	Behavioral abnormalities induced by MIA are blocked by an anti-inflammatory drug given postnatally. This demonstrates that the brain is impacted by inflammation in the postnatal period (when vaccines are given).	"Our results suggest that a possible altered inflammatory state associated with maternal immune activation results in impaired synaptic development that persists into adulthood but which can be prevented with early anti-inflammatory treatment."

Postnatal systemic inflammation exacerbates impairment of hippocampal synaptic plasticity in an animal seizure model Chen et al., Neuroimmunomodulation, Vol. 20, 2013.	Postnatal immune activation increased seizure susceptibility in rats. Other adverse brain effects were also reported. The postnatal timing is important because some vaccine advocates erroneously argue that the brain can only be injured during gestation.	"Central nervous system inflammation during critical stages of childhood development could disrupt the balance needed for neurophysiological actions of immune processes, producing direct, pernicious effects on memory, neural plasticity and neurogenesis into adulthood."
Maternal immune activation promotes hippocampal kindling epileptogenesis in mice Pineda et al., Ann Neurol, Vol. 74, 2013.	This study replicated the finding that IL-6 causes autism. It also shows that the cytokine combination IL-6 + IL-1B causes epilepsy. This explains the association between autism and seizure disorder.	"In addition to confirming previously established critical role of IL-6 in the development of autism-like behavior following MIA, the present study shows that concurrent involvement of IL-6 and IL-1β is required for priming the offspring for epilepsy. These data shed light on mechanisms of comorbidity between autism and epilepsy." "IL-6 is necessary and sufficient for causing autism in the offspring."
The maternal interleukin-17a pathway in mice promotes autismlike phenotypes in offspring Choi et al., Science, Vol. 351, 2016.	Shows that IL-6 causes autism by inducing the cytokine IL-17a. IL-6 and IL-17a are closely connected in a feedback loop. IL-6 induces IL-17a, and vice versa. Vitamin D reduces IL-17a production, which perhaps explains why vitamin D reduces autism severity and prevents autism.	"…in agreement with previous reports IL-6 injection into pregnant wild type (WT) mothers was sufficient to produce MIA-associated behavioral phenotypes." "…IL-6 injections into WT mothers were sufficient to induce IL-17a levels comparable to those of poly(I:C)-injected WT mothers." Note: Wild type=not genetically engineered.
Serum and CSF levels of cytokines in acute encephalopathy following prolonged febrile seizures Ichiyama et al., Brain and Development, Vol. 30, 2008.	Shows that febrile seizures strongly induce IL-6 in cerebrospinal fluid (CSF). IL-6 increase is comparable to levels that cause autistic behavioral abnormalities in animal studies. Vaccines cause seizures. Seizures always cause an IL-6 surge in the brain. Febrile seizures in infants are associated with regressive autism.	"CSF IL-6 levels were significantly higher in…febrile seizure compared with control subjects."

Risk Factors for Autistic Regression: Results of an Ambispective Cohort Study Zhang et al., Journal of Child Neurology, Vol. 27, 2012.	Shows that febrile seizure is associated with regressive autism. Odds ratio (OR) =3.53, which is comparable to the OR for family history of autism (OR=3.62). The finding is expected because febrile seizures always induces IL-6 in the brain.	"…febrile seizures were associated with a significantly increased risk of regression."
Brain IL-6 and Autism Wei et al., Neuroscience, Vol. 252, 2013.	Reviews the evidence that autism is caused by elevated IL-6 in the brain. Autisic behavioral abnormalities are caused by either acute or chronic IL-6 elevation. Aluminum causes chronic IL-6 elevation in the brain.	"All these evidences suggest that brain IL-6 may play an important role in the development of autism." "Many studies show IL-6 dysregulation in individuals with autism…"
Maternal immune activation in late gestation increases neuroinflammation and aggravates experimental autoimmune encephalomyelitis in the offspring Zager et al. Brain, Behavior, and Immunity, Vol. 43, 2015.	MIA increased the severity of multiuple sclerosis (MS) in a mouse model. Susceptibility to MS was tested at maturity (60 days of age). So the results suggest that early life immune activation increases disease risk long term, perhaps for a lifetime.	"…maternal immune activation during mice late gestation influences the development of offspring's immune system, an effect that persists until adulthood. Specifically, the off-spring…showed aggravated clinical manifestations and immune responses during the course of EAE." Note: EAE=multiple sclerosis.
The microbiota modulates gut physiology and behavioral abnormalities associated with autism Hsiao et al. Cell, Vol. 155, 2013.	MIA caused adverse changes to the gastrointestinal microbiome. Neurotoxic substances produced by a pathogenic microbiome caused autistic behavioral abnormalities. Administering the beneficial, anti-inflammatory probiotc B. Fragilis completely alleviated some of the behavioral abnormalities. This study supports Dr Wakefield's 1998 hypothesis that vaccines cause autism by trigging an inflammatory gastrointestinal disorder.	"…these findings support a gut-microbiome-brain connection in autism and identify a potential probiotic therapy for gastrointestinal and behavioral symptoms of autism." "Remarkably, *B. fragilis* treatment ameliorates abnormal communicative, stereotyped, sensorimotor and anxiety-like behaviors in MIA offspring, supporting emerging evidence for a gut-brain link in autism."

Association Between Influenza Infection and Vaccination During Pregnancy and Risk of Autism Spectrum Disorder Zerbo et al. JAMA Pediatrics, Vol. 171, 2017.	This is perhaps the first human study looking for immune activation brain injury by vaccination. The authors inappropriately used a bonferroni correction to discount a significant association between 1st-trimester influenza vaccination and autism. Hazard ratio was 1.2, equivalent to about 4 autism cases per 1000 vaccinations. This represents a severely inverted risk/benefit, since 1000 vaccinations should be expected to prevent about 10-20 influenza illnesses, and cause a comparable number of non-influenza illnesses.	"If influenza vaccination during the first trimester of pregnancy causes ASD, our results suggest that it would amount to 4 additional ASD cases for every 1000 women vaccinated. Our finding of a possible association between maternal influenza vaccination in the first trimester and increased ASD risk parallels previous studies reporting an association between maternal viral infection or fever and increased ASD risk in the first trimester."