VACCINE INDUCED IMMUNODEFICIENCY

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CONCLUSIONS

Whole-population vaccination may be causing immunodeficiency that can be detected at the national level as increased unnecessary infections from vaccine-targeted and non-vaccine targeted pathogens.

Vaccination therefore cannot be expected to “protect” the immunodeficient.

In the long run, whole-population vaccination makes the use of vaccines a self-defeating prospect due to vaccine-induced immunodeficiency.
Retrospective Analyses of Health Outcomes in a
Variably Vaccinating Pediatric Practice

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Using these data, we will study three Specific Aims:

- **Specific Aim 1:** Is vaccination exposure associated with major health outcome categories?

  Patients will be stratified based on vaccine status: “fully vaccinated” per CDC pediatric schedule, partially vaccinated, and unvaccinated. Outcomes to be studied include, but are not limited to:
  - Any neurodevelopmental/neurobehavioral disorder diagnosis (autism spectrum disorder per DSM-5, ADHD, pervasive development disorder, tics, Tourette’s syndrome, other);
  - Any allergy or autoimmune disorder (eczema, allergies, asthma, arthritis, IDDM, other) after 18 months of age;
  - Speech-related disorders (apraxia, others)
  - Repetitive motion or movement disorders
  - Social disorders
Specific Aim 2: Within the vaccinated, are there indicators of risk of vaccine-related morbidity/mortality available in early medical reports (<2 years of age)?

To address this aim, Phased Biomarker Development integrative modeling will be used to study available predictor variables including family history of autoimmunity, birth weight, older sibling w/adverse events, eczema, vaccine-related fever, vaccine-related febrile seizure, ear infections (otitis media), early exposure to acetaminophen, gestational vaccination (none, single, double), and others.

In addition to outcomes noted in “Specific Aim 1” above, additional outcome variables will include, but are not limited to:
Specific Aim 3: To determine if these models are enhanced by including variables traditionally considered “confounders”, such as family history and other variables.

Here, the same model evaluation will be used but these additional variables will be examined as possible co-predictors of the health effects of vaccination. Our data analysis plan (DAPs) per aim will be published online prior to execution of any analysis. Per national research regulatory standards and requirements, all data will be de-identified by an Honest Broker who will not be involved in the data analysis.

Inclusion Criteria:
- Over 3,000 medical records were examined during the quality assurance audit at Dr. Paul Thomas’ Integrative Pediatrics practice.
Autism Rates for 3,344 Patients At Integrative Pediatrics

The Vaccine-Friendly Plan
1 in 438

Vaccines
1 in 715

Vs.

1 in 45

CDC

Centers for Disease Control and Prevention
One rationale used argue for 100% vaccination is "herd immunity will protect the "immunodeficient""

aka, the immunocompromised
CDC: Vaccination Coverage of Children Remains High

*Rates of Unvaccinated, Vaccine-exempt Children Increase Slightly*

October 19, 2018 02:13 pm News Staff – A pair of CDC Morbidity and Mortality Weekly Reports (MMWRs) released Oct. 12 offered an overview of vaccination status among young children in the United States, most of which was positive.

Vaccination Status of Kindergarteners

The first MMWR (www.cdc.gov) focused on vaccine coverage and exemption rates among kindergarteners for the 2017-18 school year and found the median vaccination coverage was 95.1 percent for the state-required dosage series for diphtheria, tetanus and acellular pertussis (DTaP) vaccine.

The report, which summarized vaccine coverage and exemption estimates collected by state and local immunization programs for kindergarteners in 49 states and the District of Columbia, as well as data...
40 cases of whooping cough diagnosed last week in Santa Clarita (VIDEO)
Fig. 1 Pertussis cases in the US, 1940–2012. Data are from the Centers for Disease Control and Prevention via the National Notifiable Diseases Surveillance System.
Immunodeficiency is the state in which an individual has a partially or entirely suppressed adaptive immune system.
HUMAN ADAPTIVE IMMUNE SYSTEM HAS MANY MOVING PARTS!
KNOWN CAUSES OF IMMUNODEFICIENCY

Primary

- Genetics
  - ataxia telangiectasia
  - common variable immunodeficiency
  - severe combined immunodeficiency
  - DiGeorge syndrome
  - Wiskott-Aldrich syndrome
  - X-linked agammaglobulinemia

Secondary

SECONDARY

- HIV INFECTION
- AGE (IMMUNOSENESCENCE)
- CHEMOTHERAPY
- TRANSPLANT IMMUNOSUPPRESSIVE DRUGS
- ANTIBIOTICS
- CHEMICALS
  - Perfluorooctanoate and Perfluorooctanesulfonate (WAS USED IN SCOTCHGUARD)
  - Thimerosal
EVIDENCE THAT VACCINES MAY INDUCE IMMUNODEFICIENCY

- THIMEROSAL SPECIFICALLY INHIBITS ERAP1
- MITOTOXICITY
- LINKED EPITOPE SUPPRESSION
1. THIMEROSAL

- 50% ETHYLMERCURY BY WEIGHT
- ANTI-FUNGAL ADDITIVE USED IN MULTI-DOSE VIALS
- ABOUT 80% OF INFLUENZA VACCINES IN THE US MARKET CONTAIN THIMEROSAL
- >250 MCG PER DOSE
THIMEROSAL &
ENDOPLASMIC RETICULUM AMINOPEPTIDASE 1 (ERAP1)

- ERAP1 shortens proteins *en route* to be presented on the surface of MHC Class 1 cells
GENETIC VARIANTS IN ERAP1 AND ERAP1 ACTIVITY CONFER AUTOIMMUNE RISK
"Cell-based analysis indicated that thimerosal can effectively reduce ERAP1-dependent cross-presentation by dendritic cells in a dose-dependent manner."

Screening Identifies Thimerosal as a Selective Inhibitor of Endoplasmic Reticulum Aminopeptidase 1

Athanasios Stamogiannos, Athanasios Papakyriakou, Francois-Xavier Mauvais, Peter van Endert

National Center for
Institut National de National de la Recherche
Supporting Info
THIMEROSAL-CONTAINING FLU VACCINES

INCREASED THE RISK OF NON-INFLUENZA RESPIRATORY VIRUS INFECTIONS BY $\frac{390}{88} = 4.43$ (p<0.01)

Increased Risk of Noninfluenza Respiratory Virus Infections Associated With Receipt of Inactivated Influenza Vaccine

Benjamin J. Cowling,1 Vicky J. Fong,1 Hiroshi Nishiura,1,2 Kwok-Hung Chan,1 Sophia Ng,1 Dennis K. M. Ip,1 Susan S. Chia,1 Gabriel M. Leung,1 and J. S. Malik Peiris1,3

1School of Public Health, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Pokfulam, Hong Kong SAR, China; 2PRESTO, Japan Science and Technology Agency, Saitama; 3Department of Microbiology, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Queen Mary Hospital, Department of Pediatrics and Adolescent Medicine, The University of Hong Kong, Queen Mary Hospital, and Centre for Influenza Research, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Pokfulam, Hong Kong SAR, China

We randomized 115 children to trivalent inactivated influenza vaccine (TIV) or placebo. Over the following 9 months, TIV recipients had an increased risk of virologically-
COWLING ET AL:

- Examined the efficacy of TCV’s on influenza and effect on other non-influenza RV infection

- **DID NOT** FIND A STATISTICALLY SIGNIFICANT REDUCTION OF INFLUENZA INFECTION FOLLOWING TCV

- **DID** FIND A SIGNIFICANT INCREASED RISK OF

Non-influenza respiratory virus infection among TCV recipients:

- rhinovirus infection

- coxsackie/echovirus infection
Financial support. This work was supported by the Area of Excellence Scheme of the Hong Kong University Grants Committee (grant number AoE/M-12/06), the Hong Kong University Research Council Strategic Research Theme of Public Health, the Harvard Center for Communicable Disease Dynamics from the National Institute of General Medical Sciences (grant number U54 GM088558), and the Research Fund for the Control of Infectious Disease, Food and Health Bureau, Government of the Hong Kong SAR (grant number PHE-2). The funding bodies had no role in study design, data collection and analysis, preparation of the manuscript, or the decision to publish.

Potential conflicts of interest. B. J. C. has received research funding from MedImmune. D. K. M. I. has received research funding from Roche. J. S. M. P. receives research funding from Crucell MV. All other authors report no potential conflicts.
INFLUENZA VACCINE RECORD

- USA
- 2004-2015
INFLUENZA VACCINE UPTAKE DECREASES EFFICACY OF FLU VACCINE… ???

- JLW RESULT
- IPAK
- THIMEROSAL?
- NON-INFLUENZA “FLU”?
- “FLU-SYNDROME”
Overall our findings suggest that Al induces ER stress and ROS generation which compromises the antioxidant defenses of neuronal cells thereby promoting neuronal apoptosis in p53 independent pathway.
Autism is an Acquired Cellular Detoxification Deficiency Syndrome with Heterogeneous Genetic Predisposition

James Lyons-Weiler
Institute for Pure and Applied Knowledge, USA

Abstract
Neurodevelopmental disorders, including autism spectrum disorders, have a complex biological and neurochemical basis, with diverse genetic risk and myriad environmental exposures. Teasing apart the role of specific stressors is difficult given the large number of apparently contributing associations, gene x environment interactions and phenotypic heterogeneity. These conditions have been rare, making causality assessment at the population level infeasible. Only a few studies have been able to test for association with autism, and it has been shown that improved diagnosis only explains a small percent of the variance. Now the rates are so high in some countries that public school programs cannot handle the large number of students with special needs, and professionals are quitting their jobs due to security concerns. Here, I present a mechanistic biomedical process model (theory) of the pathophysiology of autism that reconciles the apparent paradox between the high degree of causal
Figure 1: The canonical ER stress response pathway is activated in new cells due to the apoptotic release and redistribution of metals (and other toxins), spreading the ER response and initiating chronic microglial activation. With astrocytic dysfunction, the excess glutamate contributes to chronic gliosis, which is both a consequence and contributor to aberrant pruning during neurodegeneration, and ER stress include direct generation, and ER cal...
Reconsideration of the immunotherapeutic pediatric safe dose levels of aluminum

James Lyons-Weiler, Robert Ricketson

Toxicology

ARTICLE INFO

Keywords:
Aluminum
Minimum risk level
Provisional tolerable weekly intake
Regulatory elements
Pediatric dosing
No observed adverse effect level
Vaccines

ABSTRACT

FDA regulations require safety testing of constituent ingredients in drugs (21 CFR 610.15). With the exception of extraneous proteins, no component safety testing is required for vaccines or vaccine schedules. The dosing of aluminum in vaccines is based on the production of antibody titers, not safety science. Here we estimate a Pediatric Dose Limit that considers body weight. We identify several serious historical missteps in past analyses of provisional safe levels of aluminum in vaccines, and provide updates relevant to infant aluminum exposure in the pediatric schedule considering pediatric body weight. When aluminum doses are estimated from Federal Regulatory Code given body weight, exposure from the current vaccine schedule are found to exceed our esti-
J. Lyons-Weiler, R. Rickerson


Fig. 2. FDA Doses and exposures adjusted by body weight: Comparison between Infants and an Adult.
In a male child from birth through 36 months at the 50th percentile body weight, the FDA dose of 850 μg adjusted by body weight demonstrates that an adult weighing 60 kg receives significantly less aluminum per injection per kg compared to a child, particularly those children with lower body weights.

Table 1

FDA Dose Adjusted by Body Weight (μg/kg), Birth through Adulthood, US Population.

JECEFA provisional tolerable daily intake from dietary and additive exposures of 140 μg/kg/day and current provisional tolerable daily
when adjusted to body weight (µg/kg) and compared to a 60-80 kg adult, the aluminum load is significantly higher in the birth through 24-month age cohort.

The scheduled pediatric vaccinations in 2016 have significantly particular the use of TdA, has changed. The combined doses of aluminum at 2, 4, and 6 months are 1225 µg, 975 µg, and 1225 µg respectively (Table 2), and are not determined considering infant and

![Graph](image)

**Fig. 4.** Comparison of the Calculated Pediatric MRL and the AL Exposures from DTaP Vaccine for Children (and Adults) using Clark’s Rule to Accommodate Pediatric Body Weights kg, per day, at 2 months and for Adult.
IN REVIEW
MITOTOXICITY

- THIMEROSAL (PRESERVATIVE)
- ALUMINUM (ADJUVANTS)
- AUTOIMMUNE MITOCHONDRIAL DEFICIENCY
Research Article

Thimerosal-Derived Ethylmercury Is a Mitochondrial Toxin in Human Astrocytes: Possible Role of Fenton Chemistry in the Oxidation and Breakage of mtDNA

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**SHARPE ET AL. 2013**

- B- Lymphocytes exposed to thimerosal
- 11 families
- ASD + Sibs
- Control (No ASD)
HUMAN ADAPTIVE IMMUNE SYSTEM HAS MANY MOVING PARTS!

- Combined with genetic mitochondrial impairment...
Aluminum-induced Defective Mitochondrial Metabolism Perturbs Cytoskeletal Dynamics in Human Astrocytoma Cells

J. Lemire, R. Mailloux, S. Puiseux-Dao, and V. D. Appanna

Department of Chemistry and Biochemistry, Laurentian University, Sudbury, Ontario, Canada
USM 505/EA 4105, Ecosystème et interactions toxiques, Département de régulations, développement, et diversité moléculaire, Muséum National d'Histoire Naturelle, Paris, France

Although aluminum (Al), a known environmental toxin, has been implicated in a variety of neurological disorders, the molecular mechanism responsible for these conditions is not fully understood. In this report, we demonstrate the ability of Al to trigger mitochondrial dysfunction and ineffective adenosine triphosphate (ATP) production. This situation severely affected cytoskeletal dynamics. Whereas the control cells had well-defined structures, the Al-exposed astrocytoma cells appeared as globular structures. Creatine kinase (CK) gradient, which is tapped to drive ATP formation (Yoshida et al., 2001). Complex eukaryotes also rely on other sources of ATP such as phosphagens in order to sustain energy demands (Sauer and Schlattner, 2004). Highly oxidative tissues such as the human brain and skeletal muscle invoke creatine kinase (CK) to produce ATP from phosphocreatine when energy is in high demand (Saks et al., 1996).

The brain consumes the most energy in the human body. Hence, neurons rely on a steady supply of ATP...
LEMIRE ET AL. ALUMINUM IMPAIRS CELLULAR ENERGETICS AND CYTOSKELETAL STRUCTURE

Fig. 9. Molecular link between Al toxicity and morphological perturbation in human astrocytoma cells. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]
ALUMINUM
HUMAN ADAPTIVE IMMUNE SYSTEM HAS MANY MOVING PARTS!

- Combined with thimerosal and genetic mitochondrial impairment…

![Diagram of the adaptive immune system](image-url)
How did the FDA determine that 850 ug of aluminum is safe for an adult?

Has the FDA published a minimum safe level (MSL) of aluminum doses in vaccines for children?

If not, can we estimate a MSL at a given body weight expressed as ug/kg/day?
FDA (Mitkus et al.) Claimed Aluminum in Vaccines is Safe
Their analysis (model-only) used estimates from:
**ORAL** forms of aluminum, not **INJECTED**
**ADULT** mice, not **INFANT** mice
**MICE**, not **HUMAN** studies

Expressed safe levels per dose independent of body weight or time, **not** *ug/kg/day*
Minimum AL dose ingested (mg/kg/day)

Golub et al., 1989
irregular feeding cycles

Cao et al., 2016
neuroinflammation, loss of dendritic spines (immunoneuroexcitotoxicity)

Bilkei-Gorzo, 1993
learning and memory impairment

Sethi et al. 2009
impaired spatial learning

Sethi et al. 2008
neurotoxicity

Dera, 2016
impaired kidney function

Borai et al., 2017
Purkunje fiber cell death, injury to cerebellum

Alawdi et al., 2016
brain inflammation, learning and memory impairment
Toxicology

Reconsideration of the immunotherapeutic pediatric safe dose levels of aluminum

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\begin{abstract}
FDA regulations require safety testing of constituent ingredients in drugs (21 CFR 610.15). With the exception of extraneous proteins, no component safety testing is required for vaccines or vaccine schedules. The dosing of aluminum in vaccines is based on the production of antibody titers, not safety science. Here we estimate a Pediatric Dose Limit that considers body weight. When aluminum doses are estimated from Federal Regulatory Code given body weight, exposure from the current vaccine schedule is found to exceed our estimate of a weight corrected Pediatric Dose Limit. Our calculations show that the levels of aluminum suggested by the currently used limits place infants at risk of acute, repeated, and possibly chronic exposures of toxic levels of aluminum in modern vaccine schedules. Individual adult exposures are on par with Provisional Tolerable Weekly Intake "limits", but some individuals may be aluminum intolerant due to genetics or previous exposures. Vaccination in neonates and low birth-weight infants must be re-assessed; other implications for the use of aluminum-containing vaccines, and additional limitations in our understanding of neurotoxicity and safety levels of aluminum in biologics are discussed.
\end{abstract}
The immune response to an antigen is driven by the context in which it was first encountered.

Tolerance induced to a single T cell epitope inhibits the response to all epitopes in the same protein.

Preferential responses of memory B cells following secondary exposure to vaccine components.

Memory B cells outcompete naive B cells for access to *Bordetella* epitopes.
Acellular pertussis vaccines protect against disease but fail to prevent infection and transmission in a nonhuman primate model

Jason M. Warfel, Lindsey I. Zimmerman, and Tod J. Merkel

Division of Bacterial, Parasitic and Allergenic Products, Center for Biologics Evaluation and Research, US Food and Drug Administration, Bethesda, MD, 20892

Edited by Rino Rappuoli, Novartis Vaccines and Diagnostics Srl, Siena, Italy, and approved October 22, 2013 (received for review August 5, 2013)

Pertussis is a highly contagious respiratory illness caused by the bacterial pathogen *Bordetella pertussis*. Pertussis rates in the United States have been rising and reached a 50-y high of 42,000 cases in 2012. Although pertussis resurgence is not completely understood, we hypothesize that current acellular pertussis (aP) vaccines fail to prevent colonization and transmission. To test our hypothesis, infant baboons were vaccinated at 2, 4, and 6 mo of age with aP or whole-cell pertussis (wP) vaccines and challenged with *B. pertussis* at 7 mo. Infection was followed by quantifying colonization in nasopharyngeal washes and monitoring leukocytosis and symptoms. Baboons vaccinated with aP were protected from severe pertussis-associated symptoms but not from colonization, did not clear the infection faster than naïve animals, and readily transmitted *B. pertussis* to unvaccinated contacts. Vaccination with wP induced a more rapid clearance compared with naïve and aP-vaccinated animals. By comparison, previously infected animals were not colonized upon secondary infection. Although all vaccinated and previously infected animals had robust serum antibody responses, we found key differences in T-cell immunity. Pre-therapeutic for established disease, and the highly contagious nature of pertussis. Although a variety of small-animal models have been used to study pertussis, none of them adequately reproduce the human disease (16). To address this gap, we recently developed a nonhuman primate model of pertussis using baboons (*Papio anubis*) and found the disease is very similar to severe clinical pertussis. Upon challenge, baboons experience 2 wk of heavy respiratory colonization and leukocytosis peaking between 30,000–80,000 cells/mL, similar to the range in pertussis-infected infants (1, 17). In addition, baboons experience a paroxysmal cough illness characterized by repeated fits of 5–10 coughs. The coughing fits last on average >2 wk in the baboon, although this is less than some severely infected children, where the cough can last up to 12 wk (1, 17). We also characterized airborne transmission of *B. pertussis* from infected to naïve animals, which is the route of transmission postulated to occur between humans (18). Because this is the only model of pertussis to reproduce the cough illness and transmission of the human disease, we believe it provides the unique opportunity to test our hypothesis that aP vaccines fail to prevent *B. pertussis* colonization, thus enabling transmission.
The Pertussis resurgence: putting together the pieces of the puzzle
Rotem Lapido1 and Christopher J. Gill2

Abstract
Pertussis incidence is rising in almost every country whereacellular pertussis (aP) vaccines have been introduced, and is occurring across all age groups from infancy to adulthood. The key question is why? While several known factors such as waning of immunity, detection bias due to more sensitive tests and higher awareness of the disease among practitioners, and evolutionary shifts among B. pertussis all likely contribute, collectively, these do not adequately explain the existing epidemiologic data, suggesting that additional factors also contribute. Key amongst these is recent data indicating that the immune responses induced by aP vaccines differ fundamentally from those induced by the whole cell pertussis (wP) vaccines, and do not lead to mucosal immunity. If so, it appears likely that differences in how the two categories of vaccines work, may be pivotal to our overall understanding of the pertussis resurgence.

Keywords: Pertussis, Cellular pertussis vaccine, Resurgence, Epidemiologic modeling, Asymptomatic transmission, Pertussis vaccines, Review

Outcomes of exposure:

<table>
<thead>
<tr>
<th>Vaccine given</th>
<th>Disease</th>
<th>Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>No vaccine</td>
<td>Clinically ill Lymphocytosis</td>
<td>33-35 days, high density</td>
</tr>
<tr>
<td>aP vaccine</td>
<td>Asymptomatic No lymphocytosis</td>
<td>33-35 days, high density</td>
</tr>
<tr>
<td>wP vaccine</td>
<td>Asymptomatic No lymphocytosis</td>
<td>18 days, low density</td>
</tr>
</tbody>
</table>

Allowed time to seroconvert
Exposed to B. pertussis aerosols

Conclusion:
- Exposure to B. pertussis in absence of vaccines results in clinical disease and infection
- aP and wP vaccines both prevent clinical disease upon exposure to B. pertussis
- wP vaccines also shorten duration of infection
- aP vaccines have no impact on duration of infection
**Outcomes of exposure:**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinically ill</td>
<td>NP carriage within 10 days</td>
</tr>
<tr>
<td>Lymphocytosis</td>
<td></td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>NP carriage within 10 days</td>
</tr>
<tr>
<td>No lymphocytosis</td>
<td></td>
</tr>
</tbody>
</table>

**B. pertussis infected and symptomatic unvaccinated baboon**

**aP vaccinated**

**Cohoused in same cage**

**Conclusions:**
- Unvaccinated infected animals easily transmit *B. pertussis*
- Unvaccinated animals who are exposed to infected animals also become infected
- aP vaccinated animals are resistant to clinical disease
- However, aP vaccination does not prevent these animals from being infected

**Fig. 3** Outcome of exposure to infected animal by vaccination status. Here, an infected unvaccinated animal was co-housed with three initially uninfected animals, one of which was unvaccinated, while the other two had received aP vaccinations. All three animals became infected based on nasopharyngeal sampling, though only the unvaccinated animal showed signs of clinical illness. This showed that infection due to exposure to an infected animal can transmit *B. pertussis* (a more realistic model than exposure to aerosols). But again, while aP vaccinations blocked clinical disease, they did not prevent infection.
PERTUSSIS VACCINATION

- Does not lead to mucosal immunity
- Does not prevent infection
- Does not prevent transmission
- Linked epitope suppression is the most likely explanation
Original Antigenic Sin

May 15, 1958

DOI: 10.1056/NEJM195805152582014

Although numerous strains of influenza viruses may cause clinically indistinguishable forms of "grippe," the various agents may be reduced by serologic technics to two important types of virus, the influenza A's and their relatives, the B's; and two branches that are "country cousins," the C's and D's. Within the first two ancestral clans are perhaps a dozen or so families of the A strain and four or so major variants of B strains. Such genotypic distinctions are of more than academic interest since immunity established after infection by one type in no way protects against infection at a subsequent date . . .
Original Antigenic Sin Responses to Influenza Viruses

Jin Hyang Kim, Ioanna Skountzou, Richard Comans, and Joshy Jacob

Most immune responses follow Burnet’s rule in that Ag recruits specific lymphocytes from a large repertoire and induces them to proliferate and differentiate into effector cells. However, the phenomenon of “original antigenic sin” stands out as a paradox to Burnet’s rule of B cell engagement. Humans, upon infection with a novel influenza strain, produce Abs against older viral strains at the expense of responses to novel, protective antigenic determinants. This exacerbates the severity of the current infection. This blind spot of the immune system and the redirection of responses to the “original Ag” rather than to novel epitopes was described fifty years ago. Recent reports have questioned the existence of this phenomenon. Hence, we revisited this issue to determine the extent to which original antigenic sin is induced by variant influenza viruses. Using two related strains of influenza A virus, we show that original antigenic sin leads to a significant decrease in development of protective immunity and recall responses to the second virus. In addition, we show that sequential infection of mice with two live influenza virus strains leads to almost exclusive Ab responses to the first viral strain, suggesting that original antigenic sin could be a potential strategy by which variant influenza viruses subvert the immune system. The Journal of Immunology, 2009, 183:3294–3301.

Influenza is the most recurring respiratory disease in humans. During the 20th century, influenza A viruses have afflicted the human race with three pandemics in 1918, 1957, and 1968, and numerous seasonal epidemics (1–3). Every year in the United States, 5–20% of the population gets infected with influenza viruses leading to over 200,000 hospitalizations and 36,000 deaths (4). Although a single influenza infection provides lifelong immunity against the homotypic strain, the public remains susceptible to infection with a novel flu variant (5). This is because the virus constantly undergoes genetic variation to avoid protective viruses that can no longer be neutralized by previous Abs (11). As a result, the variant viruses maintain shared epitopes with the parental strain but also have unique epitopes that allow escape from neutralizing Abs. When an immune host is exposed to this variant influenza virus, two things need to happen to ensure a successful protection: 1) activation of memory B cells that recognize shared epitopes and 2) activation of naïve B cells that recognize novel epitopes. In the case of repeated infection with variant influenza viruses, the latter response is not induced and this phenomenon is called original antigenic sin. Original antigenic sin was first described by Burnet in 1957, who postulated that vaccination with a related virus could lead to immune responses that were directed against conserved epitopes of different influenza subtypes.

“Humans, upon infection with a novel influenza strain, produce Abs against older viral strains at the expense of responses to novel, protective antigenic determinants”
Jin Hyang Kim, Ioanna Skountzou, Richard Compans, and Joshy Jacob

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INFLUENZA VACCINE UPTAKE DECREASES EFFICACY OF FLU VACCINE... !!!

- JLW RESULT
- IPAK
- THIMEROSAL?
- NON-INFLUENZA “FLU”?
- “FLU-SYNDROME”
Epidemic Pertussis and Acellular Pertussis Vaccine Failure in the 21st Century

James D. Cherry, MD, MSc

In this issue of Pediatrics Acosta et al1 present a tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis, adsorbed (Tdap) vaccine effectiveness study in adolescents in Washington State during the first 6 months of 2012. Their findings support the previous Tdap effectiveness data from Wisconsin.2 The duration of Tdap effectiveness is disappointing, particularly because case-control studies tend to inflate efficacy.3

In 4 recent publications (including 1 article in Pediatrics) I have discussed because of clear evidence of "observer bias" in both studies.4 In this present Washington State study, which involved adolescents 11 to 18 years of age, 81% of whom had received Tdap vaccines, the attack rate during the epidemic was only 182.3 per 100,000 for the one-half-year study period.1 This rate is no greater than that noted during nonepidemic periods in the pre-DTαP and Tdap eras.5,6

In 2012 in Pediatrics I discussed why pertussis vaccines fail7; however, new data have become available over the
### POPULATION-WIDE IMMUNODEFICIENCY

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<th>NATURAL IMMUNITY</th>
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<td>NO VACCINE FAILURE</td>
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<td>METALS IMMUNODEFICIENCY</td>
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Senator Richard Pan California, Earlier This Month

IT WORKS THE WAY
.. IT WORKS?

SB 276 Pan VAXXHOLE
CONCLUSIONS

Whole-population vaccination may be causing immunodeficiency that can be detected at the national level as increased unnecessary infections from vaccine-targeted and non-vaccine targeted pathogens.

Vaccination therefore cannot be expected to “protect” the immunodeficient.

In the long run, whole-population vaccination makes the use of vaccines a self-defeating prospect due to vaccine-induced immunodeficiency.