Pediatric Dosing of Aluminum in Vaccines: Comparisons of Schedules

James Lyons-Weiler, PhD  
_The Institute for Pure and Applied Knowledge_

Collaborators: R. Richardson, G. McFarland, E. Lajoie & P. Thomas

1/5/2020  
at the  
_University of California Los Angeles_
Blinded peer-review
<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Aluminum Content (µg)* per dose</th>
<th>Birth</th>
<th>1 mo</th>
<th>2 mos</th>
<th>4 mos</th>
<th>6 mos</th>
<th>9 mos</th>
<th>12 mos</th>
<th>15 mos</th>
<th>18 mos</th>
<th>18-23 mos</th>
<th>2-3 yrs</th>
<th>4-6 yrs</th>
<th>7-10 yrs</th>
<th>11-12 yrs</th>
<th>13-15 yrs</th>
<th>15-18 yrs</th>
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<tbody>
<tr>
<td>Hepatitis B1 (HepB)</td>
<td>250</td>
<td>1st dose</td>
<td>2nd dose</td>
<td>3rd dose</td>
<td></td>
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<tr>
<td>Botavac2a (RV)</td>
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<td>1st dose</td>
<td>2nd dose</td>
<td></td>
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<tr>
<td>RV1 (2-dose series); RV5 (3 dose series)</td>
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<tr>
<td>Diphtheria, tetanus, &amp; acellular pertussis1 (DTPa)</td>
<td>0.25</td>
<td>1st dose</td>
<td>2nd dose</td>
<td>3rd dose</td>
<td>4-6th dose</td>
<td>5th dose</td>
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<td>Haemophilus influenzae type b1, (Hib)</td>
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<td>1st dose</td>
<td>2nd dose</td>
<td>3rd dose</td>
<td>4-3rd or 4th dose</td>
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<tr>
<td>Pneumococcal conjugate (PCV13)</td>
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<td>3rd dose</td>
<td>4-4th dose</td>
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<tr>
<td>Inactivated poliovirus (IPV&lt;18 yrs)</td>
<td>0</td>
<td>1st dose</td>
<td>2nd dose</td>
<td>3rd dose</td>
<td>4-5th dose</td>
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<td>Influenza7 (IIV; LAIV)</td>
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<td>Annual vaccination (IIV only) 1 or 2 doses</td>
<td>Annual vaccination (IIV only) 1 or 2 doses</td>
<td>Annual vaccination (IIV only) 1 or 2 doses</td>
<td>Annual vaccination (IIV only) 1 or 2 doses</td>
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<tr>
<td>Measles, rubella, mumps (MMR)</td>
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<td>1st dose</td>
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<tr>
<td>Varicella (VAR)</td>
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<tr>
<td>Hepatitis A10 (Hepa)</td>
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<td></td>
<td>1st dose</td>
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<tr>
<td>Meningooccal11 (Hib, MenCY a, &amp; 6 weeks; MenACWY D &lt; 6 mos; MenACWY CRM 6 &lt; 2 mos)</td>
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<td>1st dose</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>(Tdap)</td>
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<tr>
<td>Tetanus, diphtheria, &amp; acellular pertussis2 (Tdap ≥ 7 yrs)</td>
<td>0.319</td>
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<tr>
<td>Human papillomavirus13 (2yHPV/females only; 4yHPV, 6yHPV/males and females)</td>
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<tr>
<td>Meningooccal B11</td>
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<td></td>
<td>(3 dose series)</td>
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<tr>
<td>Pneumococcal polysaccharide3 (PPS103)</td>
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</tr>
</tbody>
</table>

* Total ag not adjusted to µg/kg

| 250 | 1225 | 975  | 1000  | 600  | 875  |
Vaccine Friendly Plan

Vaccine Plan
Dr. Paul Thomas, M.D., F.A.A.P.

Since 2008, Dr. Paul and the team at Integrative Pediatrics LLC have been using the plan outlined below. Combining this vaccine plan with exclusive breastfeeding, eating a diet of real food, getting enough vitamin D, exercising, and avoiding toxins like acetaminophen, aspartame, and glyphosate, the children in his practice have experienced superior health, and a significantly lower rate of autism (0 in 1176) than the national average, which is 1 in 45.

If you have autism in the family, a history of autoimmune disorders, or an MTHFR mutation: delay vaccines until at least age five.

<table>
<thead>
<tr>
<th>Age</th>
<th>Vaccines</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pregnancy</td>
<td>No vaccines (No Tdap, No flu)</td>
</tr>
<tr>
<td>Birth</td>
<td>No Hep B</td>
</tr>
<tr>
<td>2 months</td>
<td>Hib, DTap (No Hep B, Rotavirus, IPV)</td>
</tr>
<tr>
<td>3 months</td>
<td>Pevnar</td>
</tr>
<tr>
<td>4 months</td>
<td>Hib, DTap (No Rotavirus, IPV)</td>
</tr>
<tr>
<td>5 months</td>
<td>Pevnar</td>
</tr>
<tr>
<td>6 months</td>
<td>Hib, DTap (No Hep B, Rotavirus, IPV)</td>
</tr>
<tr>
<td>7 – 9 months</td>
<td>Pevnar,</td>
</tr>
<tr>
<td>1 year</td>
<td>Hib, Pevnar (No MMR, Hep A, Varicella)</td>
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<tr>
<td>18 months</td>
<td>DTap,</td>
</tr>
<tr>
<td>2 years</td>
<td>(No Hep A)</td>
</tr>
<tr>
<td>3 years</td>
<td>Consider MMR (always give MMR by itself)</td>
</tr>
<tr>
<td>4 - 6 years</td>
<td>DTap, (consider Varicella, IPV)</td>
</tr>
<tr>
<td>10 years</td>
<td>Tdap (boost every 5 – 10 years)</td>
</tr>
<tr>
<td>11 years</td>
<td>Menneo or Menactra (meningococcal), Varicella</td>
</tr>
<tr>
<td>12-14 years</td>
<td>Hepatitis B (3 dose series)</td>
</tr>
<tr>
<td>16 – 18 Years</td>
<td>Menneo or Menactra &amp; consider meningococcal B, Hepatitis A</td>
</tr>
</tbody>
</table>
Why Reduce Aluminum?

• Aluminum is found in amyloid, the protein/non-protein aggregates in Alzheimer’s brain
• Aluminum hydroxide is used to routinely and reliably induce autoimmune conditions in animals (mice & rats)

Disruption of erythropoiesis and RBC function
• Osteomalacia in bone
• Cholestasis in liver
• Aluminum inhibits defensive mechanisms connected with white blood cells and macrophages
• https://www.ncbi.nlm.nih.gov/pubmed/16146022
Aluminium Induced Endoplasmic Reticulum Stress Mediated Cell Death in SH-SY5Y Neuroblastoma Cell Line Is Independent of p53

Syed Husain Mustafa Rizvi¹, Arshiya Parveen¹, Anoop K. Verma², Iqbal Ahmad³, Md Arshad⁴, Abbas Ali Mahdi¹*  

¹Department of Biochemistry, King George’s Medical University, Lucknow, Uttar Pradesh, India, ²Forensic Medicine & Toxicology, King George’s Medical University, Lucknow, Uttar Pradesh, India, ³Fibre Toxicology Division, CSIR-Indian Institute of Toxicology Research, Lucknow, Uttar Pradesh, India, ⁴Department of Zoology, Lucknow University, Lucknow, Uttar Pradesh, India

Abstract

Aluminium (Al) is used as a house-hold utensils, media and packaging material. It is known to induce ER stress and ROS generation which compromises the antioxidant defenses of neuronal cells thereby promoting neuronal apoptosis in p53 independent pathway.

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.
Aluminum-induced Defective Mitochondrial Metabolism Perturbs Cytoskeletal Dynamics in Human Astrocytoma Cells

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

1Department of Chemistry and Biochemistry, Laurentian University, Sudbury, Ontario, Canada
2USM 505/EA 4105, Ecosystème et interactions toxiques, Département de regulations, development, 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 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
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.]
<table>
<thead>
<tr>
<th>Citation (et al.)</th>
<th>Condition</th>
<th>Animal</th>
<th>bw (g)*</th>
<th>bw (kg)</th>
<th>AL dose (mcg)</th>
<th>AL dose (mg)</th>
<th>mcg/kg</th>
<th>mg/kg</th>
<th>human max exposure (mcg/kg)</th>
<th>x human max exposure</th>
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<td>Zhu[37]</td>
<td>Atherosclerosis</td>
<td>apoE null C57BL/6 mice</td>
<td>20</td>
<td>0.02</td>
<td>25</td>
<td>0.025</td>
<td>1250</td>
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<td>230</td>
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<td>Zhu[37]</td>
<td>Atherosclerosis</td>
<td>LDLR null C57BL/6 mice</td>
<td>20</td>
<td>0.02</td>
<td>25</td>
<td>0.025</td>
<td>1250</td>
<td>1.25</td>
<td>230</td>
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<td>Kelly-Scumpia[38]</td>
<td>Lupus</td>
<td>C57bl/6 mice</td>
<td>20</td>
<td>0.02</td>
<td>50</td>
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<td>Yasar[39]</td>
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<td>Rats</td>
<td>250</td>
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<td>1000</td>
<td>1</td>
<td>4000</td>
<td>4.00</td>
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<td>CD1 mice (male)</td>
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<td>292</td>
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<td>CP/CPPS</td>
<td>Wistar rats</td>
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<td>1000</td>
<td>1</td>
<td>50000</td>
<td>50.00</td>
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<tr>
<td>Qi[41]</td>
<td>CP/CPPS</td>
<td>Wistar rats</td>
<td>250</td>
<td>0.25</td>
<td>12500</td>
<td>2.5</td>
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<td>10.00</td>
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<td>BALB/c mice (female)</td>
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<td>2222222</td>
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</table>

** estimated at 1225 mcg AL/5.326 kg (median body weight @ 2 mos.). Meant to be typical.
Observed Adverse Effects Level (LOAEL).
Each of the established FDA-approved doses of 850 μg, 1250 μg were converted to the equivalent dose expressing Clark’s Rule [28,39]:

\[ \text{Child’s Dose (mg)} = \text{Adult Dose (mg)} \times \frac{BW \text{(Child)} \text{lbs}}{BW \text{(Adult)} \text{lbs}} \]

The body weights for infants from birth through 24 months, the Clark’s Rule calculation were obtained using calculated growth velocities obtained from Weight for Age standards females from the 5th to the 95th percentile [40,41]. These pediatric doses were compared to the same doses in an adult by the body weight of 60 kg.

*Minimal risk level of aluminum in children

Clark’s Rule

- FDA Adult Limit 850 mcg per dose
- FDA Pediatric Limit: ?????????????
Mitkus et al. – The FDA’s Paper on Aluminum

- Used a model by Priest et al. which is based on human clearance of citrated aluminum forms (form in vaccines are not citrated)
- Data for the models in two studies were from 1 and 6 males (adults)
- Mitkus adjusted the model using data from creatine in infants
- MRL was derived from ATSDR oral limits
- ATSDR had picked 1 study and misrepresented the findings of Golub et al. [Next Slide]

- In P/K, the focus tends to be on plasma/serum clearance
- Mitkus assumes excretion via the kidney is similar to creatine (aluminum damages animal tissue) [Incorrect]
- Our concern is the accumulation (body burden) originally realized in the first Priest et al. study [because aluminum has known toxic effects in human tissue]
In 1996, the Committee on Nutrition in their article on neurotoxicity in children reported the 1989 JECFA provisional tolerable weekly intake of 1000 µg/kg [12] as a provisional daily intake [14]. Unfortunately, that error overestimates the provisional tolerable daily intake of aluminum from all sources in adults by a factor of at least 2. As the 1000 µg/kg/week PTWI was in fact replaced with a PTWI of 2000 µg/kg/week in 2011, the daily provisional tolerable intake should be around 286 µg/kg per day, and in consideration that the highest mean intake of a child at 2 years is 500 µg/kg per day [15]. The value 1000 µg/kg/day would seem to bring the 850 µg per dose into range, but it is off by a factor of at least 2 and perhaps seven. The role of the reliance on the incorrect public health may be significant, especially for infants, especially preterm infants and those born prematurely.
Disagreement Between Two Committees

**JEFCA**
Joint FAO/WHO Expert Committee on Food Additives

- 1989. Provisional Tolerable Weekly Intake (PTWI) established at 1 mg/kg all dietary sources and additives. Mean highest daily intake US children 0.5 mg Al/kg per day\(^1\)
- 2011. Previous PTWI of 1 mg Al/kg withdrawn. Revised PTWI to **2 mg/kg** (adults)\(^2\)

**ATSDR**
Agency for Toxic Substances Disease Registry

- 2008 (CAS ID #: 7429-90-5)\(^3\)
- Daily dietary intake of Al 2 mg/kg-day in adults
- Minimal Risk Level (MRL) 1 mg/kg-day (adults) same as No Observed Adverse Effect Level (NOAEL)

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\(^3\)Agency for Toxic Substances and Disease Registry (ATSDR) CAS ID #: 7429-90-5
How did 1 mg Al/kg week become 1 mg/kg –day and 850 mcg per dose regardless of body weight

- 1981-CFR amended to include 1250 µg/dose
- 1996-2007 PTWI estimated at 1 mg/kg/week; 0.5 mg/kg-day US child > 2 years of age (WHO Evaluation and Certain Food Additives and Contaminants. Section 4.1 Aluminum, 1996-2007)
- 1996 Committee on Nutrition Aluminum Neurotoxicity in Infants and Children (J Pediatrics), 1 mg/kg-day (in error as to PTWI- “provisional tolerable intake”)
- 2001 0.85 mg "selected empirically from data because it enhance the antigenicity and effectiveness of the vaccine" (Baylor et al 2001)
- 2001-2008 ATSDR set MRL/NOAEL to 1 mg/kg-day from all sources based on Golub 26 mg/kg-day NOAEL (ATSDR references Baylor et al (2001),
- 2001 MRL/NOAEL 2 mg/kg-day in adult humans from dietary sources (Golub et al 2001 (62 mg/kg-day, Keith et al)
- 2011 MRL=1 mg/kg bw/day (ATSDR, 2008), Mitkus (2011)
- 2017: CFR is 850 µg/DOSE.
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: Purkinje fiber cell death, injury to cerebellum
- Alawdi et al., 2016: Brain inflammation, learning and memory impairment

Minimum oral dose (mg/kg/day)
# Minimalna spożyta dawka glinu (mg/kg/dzień)

<table>
<thead>
<tr>
<th>Autorzy</th>
<th>Opis</th>
</tr>
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<tbody>
<tr>
<td>Golub et al., 1989</td>
<td>Żarliwość układu nerwowego, utrata kolców dendrytycznych (ekscytotoksyczność immuno-neurologiczna)</td>
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<tr>
<td>Cao et al., 2016</td>
<td>Upośledzenie zdolności uczenia się i pamięci</td>
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<tr>
<td>Bilkei-Gorzo, 1993</td>
<td>Upośledzenie zdolności nauki przestrzennej</td>
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<td>Sethi et al. 2009</td>
<td>Neurotoksyczność</td>
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<td>Sethi et al. 2008</td>
<td>Nieprawidłowa praca nerek</td>
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<td>Dera, 2016</td>
<td>Śmierć komórek włókna Purkiniego, uszkodzenie mózgu</td>
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<tr>
<td>Borai et al., 2017</td>
<td>Stan zapalny mózgu, upośledzenie zdolności uczenia się i pamięci</td>
</tr>
<tr>
<td>Alawdi et al., 2016</td>
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Mitkus’ Three-Compartment Model

\[ c_{Cr}(t) = \hat{a} + \hat{b} \left( \frac{t}{\hat{c}} \right) = 50.871 + 90.044 \left( \frac{t}{31.462} \right) \]

Fig. 1. Three-compartment model of aluminum disposition in adults. Rate constants were derived from the retention equation of Priest [5].
Flarend Rabbit Study (N=6)

- Injected radioactive ALPO4 and ALOH3 into rabbits
- Only 5.6% of the ALOH3 had been cleared from the body after 28 days (22 days for ALPO4)
Flarend found low clearance rate from blood

• “The aluminum concentration [in blood] produced by AH [Al hydroxide] adjuvant at 1 hour was similar to the concentrations found from 2 to 28 days.”

Clearly something is different between rabbits and humans?
Movsas Infant Study (N=15)

*Found aluminum serum levels rose 1% immediately after vaccination.*

“We were reassured to find no significant postvaccine rise in serum aluminum level after vaccination of preterm infants with vaccines containing a total of 1200 μg of aluminum.“ (JAMA Pediatrics, Letter)
Our Question

• If 850-1150 mcg is a regulated level per dose in human adults (FDA), since FDA has never published a pediatric dose limit (PDL) for humans, what might a scaled human dose limit look like?

• We considered bodyweight and allometric scaling (Clark’s Rule)

• We adapted Priest’s model as a liberal model of excretion.
Fig. 1. FDA Adult aluminum per dose limit scaled to child’s weight (Clark’s Rule) following Lyons-Weiler and Ricketson [27].
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.
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 (μg/kg, per day, at 2 months and for Adult).
Priest model

Clearance of single doses in a few individuals

The biological behaviour and bioavailability of aluminium in man, with special reference to studies employing aluminium-26 as a tracer: review and study update†

N. D. Priest

Professor of Environmental Toxicology, Middlesex University, Queensway, Enfield, UK EN3 4SF

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Until 1990 kinetic studies of aluminium metabolism and bioinerties in man and other animals had been substantially inhibited by analytical and practical difficulties. Of these, the most important are the difficulties in differentiating between administered aluminium and endogenous aluminium - especially in body fluids and excreta and the problems associated with the contamination of samples with environmental aluminium. As a consequence of these it was not possible to detect small, residual body burdens of the metal following experimental administrations. Concomitantly, means believed aluminium to be carcinogenic, exerted within a...
Toxicology

Acute exposure and chronic retention of aluminum in three vaccine schedules and effects of genetic and environmental variation

Grant McFarland\textsuperscript{a}, Elaine La Joie\textsuperscript{a}, Paul Thomas\textsuperscript{b}, James Lyons-Weiler\textsuperscript{a,\*}

\textsuperscript{a} The Institute for Pure and Applied Knowledge, Pittsburgh, PA, 15101, United States
\textsuperscript{b} Integrative Pediatrics, Portland, OR, 97225, United States

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ABSTRACT

Like the mechanisms of action as adjuvants, the pharmacodynamics of injected forms of aluminum commonly used in vaccines are not well-characterized, particularly with respect to how differences in schedules impact accumulation and how factors such as genetics and environmental influences on detoxification influence clearance. Previous modeling efforts are based on very little empirical data, with the model by Priest based on whole-body clearance rates estimated from a study involving a single human subject. In this analysis, we explore the expected acute exposures and longer-term whole-body accumulation/clearance across three vaccination schedules: the current US Centers for Disease Control and Prevention (CDC) schedule, the current CDC schedule using low aluminum or no aluminum vaccines, and Dr. Paul Thomas’ “Vaccine Friendly Plan” schedule. We then study the effects of an implicit assumption of the Priest model on whether clearance dynamics from successive doses are influenced by the current level of aluminum or modeled by the assumption that a new dose has its own whole-body dynamics “reset” on the day of injection. We model two additional factors: variation (deficiency) in aluminum detoxification, and a factor added to the Priest equation to model the potential impact of aluminum itself on cellular and whole-body detoxification. These explorations are compared to a previously estimated...
Fig. 2. Aluminum Content in Body over First Two Years for Three Vaccine Schedules.
Fig. 3. Percent Days Over aluminum Limit (%alumTox) Birth to 7 Months and 2 Years.
Fig. 6. Aluminum for different schedules with “slow” aluminum clearing.
Fig. 8. Slowdown model results in higher retention and increased expected toxicity.
Fig. 3. Percent Days Over aluminum Limit (%alumTox) Birth to 7 Months and 2 Years.
Recommendations

• **Perform** per body-weight dose escalation studies in mice using injected forms of aluminum in infant mice

• **Re-evaluate** the use of aluminum in vaccines altogether

• **Encourage** awareness of the differences between *vaccine risk awareness*, which can lead to improvements in vaccines, and “Anti-vaccination” positions
Next Steps

- IPAK VAXXED VS. UNVAXXED STUDY
- ANALYSIS OF HEALTH OUTCOMES IN >3300 CHILDREN BORN INTO DR. PAUL’S PRACTICE
- 681 UNVACCINATED, 2647 VARIABLY VACCINATED (VFP)
- IRB APPROVED
- PHASE 1 IS COMPLETE, UNDER REVIEW
- RESULTS INCLUDE HEALTH OUTCOMES RISK FROM ACV’S CF. ALL VACCINES (% LIABILITY)
Science, Public Health Policy & the Law

An IPAK PHPI Publication

In spite of billions of dollars invested into biomedical research and practice, western civilizations are characterized by chronic illness. Papers published in this journal will focus on the nexus between science, public health policy, biomedical practice and the laws and regulations that govern health care practices. Solution-focused improvements in translation of science into improved human life are welcome.

Science, Public Health Policy & the Law: Convergence

James Lyons-Weiler, Editor-in-Chief

Our Flawed Mental Health Policies

Jennifer Giustra-Kozek
IPAK to 474747

IPAK Launches New Open Access Peer-Reviewed Journal
August 1, 2019 - Pittsburgh, PA

IPAK, The Institute for Policy and Applied Knowledge, today launched a new open access, peer-reviewed journal, "Sage Open: Open Access Policy in the News." The first issue contains four articles on issues impacting public health policy. Specifically, the journal will publish articles examining the governance of public health policies, laws, and biotechnological and medical products. The first issue is now available online.

Wearing Immunogenicity

IPAK Merch

IPAK Files

IPAK Launches a Public Health Policy Research Partnership Drive
1/19/2019

IPAK, The Institute for Policy and Applied Knowledge, today announced a drive to fund a Public Health Policy Research Partnership focused on building a pipeline to address the risk for and mitigation of emerging public health threats. The initiative builds on the success of the Institute’s previous "Open Access for Policy" program and is designed to support the development of tools that will enable policy makers and others to better understand and respond to emerging health threats.

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