Journal of Trace Elements in Medicine and Biology 55 (2019) 107–109

Contents lists available at ScienceDirect

Journal of Trace Elements in Medicine and Biology

journal homepage: www.elsevier.com/locate/jtemb

Letter to the Editor

Do the vaccines harm?

1. Introduction

The persistent claims of the WHO, CDC, FDA experts, vaccine manufacturers' experts and many other self-proclaimed experts, that vaccines do not contain toxic substances or that they contain them in physiologically negligible amounts and thus cannot endanger the health of children, is an assertion that threatens to question the validity of experimental medical science, its knowledge and its achievements. This incongruity makes us poses a certain question: what is the purpose of all these experiments if not to ultimately benefit and better life of experimental animals or to make the lives of children and adults better, safer, healthier and happier. Certainly, most reasonable, scientists, doctors, and even individuals with little knowledge medical field, will agree that experiments on animals are performed for the benefit of man. Why is it, then, that numerous experiments have revealed that aluminium (Al) i.e. adjuvant aluminium (AdAl), has very little benefit to man; it is, in fact, extremely toxic, primarily for the brain. In the following text, we will attempt dissuade the belief that AdAl is safe, harmless to the brain of the newborns, infants and young children. Our final, most noble goal is to advocate for an absolutely safe immunization regime – full prevention of disease without any harmful consequences.

2. The properties of experimental animals

In experiments examining the neurotoxicity of AdAl, usually mice seven weeks old are used, which means they are "neither newborns nor infants" [1]. If we compare the life span of mice, which is about 2.5 years with the life span of a man which is about an average of 75–80 years, by mathematical proportions, it can be calculated that a seven-week-old mice is approximately 4.0–4.5 in human years. Experiments on mice aged 7 weeks unambiguously showed AdAl neurotoxicity which would mean that AdAl would be toxic even for a child aged ˜4.5 years. From an evolutionary point of view, the brain and the body of mice at birth are mature enough to assist them in self-sustenance. In fact, the newborns of all mammals and primates are born mature and have a somewhat developed capability for survival right after coming into the world. On the other hand, logic tells us that the mature brain is more resistant to toxic substances (read adjuvant aluminium), and yet experiments with adult mice having mature brains have shown that their brain is also vulnerable to AdAl. If experiments with AdAl were performed on newborn mice (keeping in mind that newborn human babies receive AdAl on the first day of birth, the basic requirement is that body weight (BW) is more than 2000 g) certainly AdAl toxicity to the brain of newborn mice would be much more pronounced compared to the brain toxicity of adult mice. What would the results of AdAl neurotoxicity be if the experiments used mice whose brains and bodies are at the same level of maturity as that of a newborn child? In order to test this, it would be necessary to use mice with immature brains and bodies, which would mean that the mice in question should be obtained by Caesarean section, a day or two before their natural birth given that intrauterine mouse development lasts about 21 days.

3. The maturity of the human brain at birth

Evolution bestowed man with the capacity for bipedalism – the ability to walk on two legs. Owing to this evolutionary progress, humans have had to pay a great price; that price is the birth of a helpless infant, incapable of survival without someone else's help. Inarguably, human infants need many years of nurturing and cultivation before they are self-sufficient. In stark contrast, all other mammals bring their offspring capable on their own to get to the source of their food. Furthermore, the ability to walk upright has caused a significant shortening of intrauterine development in humans because the delivery channel is placed in a bone frame, which only allows a child to pass through with a maximum head circumference of roughly 36 cm, with mandatory pre-rotation. Because of this, the human brain as well as other organs, are extremely immature and sensitive to environmental factors postpartum. The maturity of the human brain, and of the whole organism itself, is in direct proportion to the length of the gestation. It has been found that premature newborns are extremely sensitive to environmental factors (toxic oxygen effect – retrolental fibroplasia, bronchopulmonary dysplasia). Therefore, if oxygen is toxic to immature newborn organs (CNS, eyes, lungs) and given the fact that life is near impossible without oxygen, then we only have to think about the possible toxicity of AdAl to the immature brain of the newborn child. Owing to current stipulations about vaccination, it is determined that every newborn with birth weight greater than 2000 g receives 0.25 mg of Al^{3+} parenterally on the first day. According to numerous studies of the aforementioned experts, this is a "physiologically negligible quantity" and consequently this suggests that 0.25 mg of Al^{3+} cannot endanger the health of children.

4. Postnatal human brain maturation [2]

Immediately after a neonate's first breath, an intensive development of the entire body ensues, especially of the central nervous system (CNS). During the first 6 months and the first year of an infant's life, the most intensive growth and development occurs within the CNS (head circumference at birth is 36 cm and at the age of one averages at 47 cm). Head size increases mostly due to increased brain mass that grows at the expense of multiplying of glial cells and the myelination of nerve fibers, and the establishment of the synapses that ought to have been established during intrauterine life were it not for the physiological shortening of the gestational period in humans. Owing to this synapsing, many various important brain functions are established shortly after birth. The most important, crucial CNS synapsing, is done during the first twelve months of the postnatal life (e.g.: central

https://doi.org/10.1016/j.jtemb.2019.06.006
Received 17 February 2019
0946-672X/

excitation-inhibition control, vision, hearing, speech, loss of the primitive reflexes, walk, …). The human brain has a specific, so-called, spatiotemporal development; we would add that this is strictly sequential-spatiotemporal development. Evolution has made this development genetically coded for each individual brain function to develop at a strictly defined time interval after birth, with about 700 new synapses per minute being established in the first days, weeks and even months after birth (often referred to as synaptic pruning). For example, the function "X" at the point "Y" must be established at time "Z" (ex. 6 h, 3 min, 35 s and 2 hundredths after birth). If for some reason the function of the enzyme responsible for the synthesis of the central neurotransmitter (ex. Serotonin, GABA, L-Dopa,…) responsible for the establishment of this function is disturbed, then the function "X" at the site "Y", at time "Z" will not be established and will cease to do so, due to the sequential shutting down of genes responsible for that function. In order to make it clear to the readers what this means, we ask them to read the mechanism and the consequences of intellectual functions damaging in phenylketonuria [3].

5. The data that all medical workers ought to learn [2]

A person is born regardless of their birth weight with approximately 14 billion cortical neurons. Cortical neurons, or rather Betz's neurons, are at the peak of human development; this text is essentially written owing to their existence. A healthy newborn with body weight of 2001 g (which is the basic condition for the use of AdAl, i.e. to be vaccinated against hepatitis B) has the same number of cortical neurons in comparison to newborns with a greater birth weight, for example with a birth-weight of 4000 g. This statement aims to point out the fact that the "participation" of cortical neurons' "mass" in the total BW of a newborn is higher for children born with 2001 g than in a child born with almost double that birth weight, carrying twice greater chances for toxicity in a child with lower birth weight. Nevertheless, both neonates, regardless of their birth weight, are exposed to 0.25 mg of Al^{3+} on the first day of birth which amounts to a total of 5.6×10^{18} ions of Al^{3+} (this number is obtained by direct proportion using Avogadro's law). A significant amount (possibly 100%) of this Al, bound to transferrin will be transported to the central nervous system. If we divide the number of Al particles-ions present in 0.25 mg Al^{3+} (5.6×10^{18}) with the number of cortical neurons (1.4×10^{10}), it follows that each cortical neuron could be potentially exposed to more than 500 million particles of trivalent Al. This is in accordance with a recent review [4] in which it has been calculated and shown that infants receive 17 times more Al than would be authorized if doses were adjusted according to BW. The levels of aluminium suggested by current guidelines, place infants at risk of acute, repeated and possibly chronic exposure to toxic levels of aluminium in modern vaccine schedules. Therefore, it has been suggested that vaccination in neonates and low birth-weight infants has to be reassessed regarding aluminium dosage reduction in vaccines according to birth-weight. The main obstacle facing this proposal is the unknown effect that the reduction may have on the final antigenicity of the vaccine [4]. It is interesting to note that according to Burnet's clonal evolution theory, each specific B cell clone, for a particular specific antigen is already formed in the human body during intrauterine development, long before it comes into contact with the antigen [5]. Since the human immune system is capable of mounting an immune response to at least 10^9 different antigens, it follows that humans possess at least 10^9 different B cell clones i.e. at least 10^9 different naïve B cells which are present in the body of each newborn baby at birth, with a birth-weight greater than 2000 g [5]. The same quantity of 10^9 different naïve B cells are present in the bodies of newborns with a birth weight of 2000 g and with a birth weight of 4000 g. Regardless of their birth weight, both newborn infants are capable of eliciting an ***equal immune response*** upon being loaded with an identical quantity of vaccinal antigens, including AdAl. Should the proposed suggestion for AdAl reduction according to the birth weights [4] be accepted, it seems that loading the infants with decreased vaccine antigens and decreased AdAl would almost certainly lead to decreased immune responses. Instead of decreasing AdAl, the following alternative solutions have already been proposed [6]:

1 All aluminum containing vaccines are to be immediately postponed until the time when the child's brain shows sufficient physiological maturation. This would coincide with the day when the child loses its last primitive reflex, corresponding to the age of roughly 6–7 months, ideally, after 12 months, or
2 AdAl must immediately be replaced with Calcium phosphate, since Calcium phosphate was used in France until the mid-1980s mainly for the diphtheria-pertussis-tetanus vaccine group without any mention of adverse reactions by physicians. Until the early-1970s it was also successfully used in pentavalent human vaccinations (smallpox, yellow fever, measles, BCG, and tetanus), also without any reported adverse reactions [7]. Furthermore, it has already been suggested that zinc could be the element of choice to replace aluminium in vaccines [6].

6. Our explanation of AdAl neurotoxicity

All metabolic cell functions, including those of cortical neurons, are related to enzyme function. Most enzymes are metaloenzymes, which means that metal i.e. biometal (Fe, Cu, Zn, Mn, Co, Se) is incorporated in the active site of the enzyme. Moreover, each enzyme has its own half-life, which means that after some time of active work the enzyme is degraded, metabolized and subsequently synthesized again. If in the resynthesis of a given enzyme, instead of a particular biometal, some other metal (non-biometal such as are Al, Pb, Hg, Ni, Cd … and AdAl is currently employed in vaccines) were to be used, then an afunctional enzyme would be formed. This will certainly cause a failure of particular metabolic functions. In order to understand this, we refer the readers to a toxicology or paediatrics textbooks explaining the mechanism of the generation of microcytic anaemia in chronic lead poisoning [8].

7. Children with the highest exposure to AdAl

According to world literature, children in the United States are the most exposed to AdAl. By the 18th month, US children parenterally receive 4925 μg of AdAl [9]. Through the use of Avogadro's number, it can be calculated that 4925 μg of Al^{3+} contain 1.09×10^{20} of Al^{3+}. This means that potentially each cortical neuron could be exposed to 7.78×10^9 of Al^{3+}.

In Serbia, according to the immunization schedule, children are given about 1.7 mg of Al^{3+} (two doses of hepatitis B vaccine and 4 doses of Pentaxim®) by the 18th month of life. This 1.7 mg of Al^{3+} contains 3.79×10^{19} Al^{3+} ions, which means that in children of Serbia aged up to 18 months, around 2.7×10^9 Al^{3+} ions, potentially circulate around each cortical neuron. Keeping in mind all of these facts and calculations, and all that we have already addressed human evolutionary features and their consequences, the maturity of the CNS of a newborn, the characteristics of the maturation of the human brain after birth, the metabolism of metals and our examination of the mechanism of AdAl neurotoxicity, we now seek to pose a question. Where would one find higher incidences of AdAl neurotoxicity (read autism spectrum disorder – ASD) in children, among children of the USA or among Serbian children? According to data from world literature, the USA has the highest incidences of ASD; every 59th child has some form of ASD [10,11]. Perhaps, it is because of these 1.09×10^{20} aluminium ions, which according to the experts mentioned at the beginning of this text, is a "physiologically negligible quantity" of toxic aluminium and thus cannot endanger the health of children…

8. The issues regarding mercury

To make the things a bit more complicated we have to say something about mercury. Mercury as a divalent metal (Hg^{2+}) undergoes the same metabolic routes as all other biometals (Fe^{2+}, Cu^{2+}, Zn^{2+}, Mn^{2+}). During intrauterine development, fetal exposure to mercury occurs through transplacental transfer and after birth via breastfeeding. The intensity of both of these exposures depend on the maternal exposure to mercury, mainly through diet. Mercury present in the blood of pregnant women is easily transferred into fetal circulation through the same routes which are operative in the metabolism of biometals. Mercury present in food and in human milk in the form of methyl mercury as a divalent metal (Hg^{2+}) is adsorbed by an enterocytic divalent metal transporter (DMT1) [12]. As was previously reported [6], the only "physiological" access to Al into the bodies of newborns, infants and children is through vaccines in the form of AdAl. Alternatively, current immunization schedules with vaccines containing AdAl are given to infants, but thimerosal (as a vaccine preservative) is found mostly in vaccines used in non-industrialized countries [13]. Depending on manufacturers the Hg^{2+} concentrations in vaccines varies between 12.5 μg/0.5 ml and 25 μg/0.5 ml [13]. Both metals, Al and Hg are themselves toxic (neurotoxic). Mercury (Hg^{2+}) i.e. ethylmercury, which is derived from thimerosal in aqueous solutions is a potent mitochondrial toxin [14]. When found together in the same tissue at the same time, Al and Hg exert synergistic toxic effects, causing significant inflammatory neurodegeneration [15].

References

[1] G. Crépeaux, H. Eidi, M.-O. David, Y. Baba-Amer, E. Tzavara, B. Giros, F.-J. Authier, C. Exley, C.A. Shaw, J. Cadusseau, R.K. Gherardi, Non-linear dose-response of aluminium hydroxide adjuvant particles: selective low dose neurotoxicity, Toxicology 375 (2017) 48–75.

[2] J.J. Volpe, T. Inder, B. Darras, L.S. de Vries, A. du Plessis, J. Neil, J. Perlman, Volpe's Neurology of the Newborn, 6th edition, Elsevier, 2017 November Hardcover ISBN: 9780323428767.

[3] I. Rezvani, C.H. Ficicioglu, Defects in metabolism of amino acids – phenylalanine, in: R.M. Kliegman, B.F. Stanton, N.F. Schor, J.W. St GemeIII (Eds.), Nelson Textbook of Pediatrics, Elsevier, 1600 John F. Kennedy Blvd. Ste. 1800 Philadelphia, PA 19103-2899, 2016, pp. 637–640.

[4] J. Lyons-Weiler, R. Ricketson, Reconsideration of the immunotherapeutic pediatric safe dose levels of aluminum, J. Trace Elem. Med. Biol. 48 (2018) 67–73.

[5] A.K. Abbas, A.H. Lichtman, Introduction to the immune system. The nomenclature, general properties and components of the immune system, in: A.K. Abbas, A.H. Lichtman (Eds.), Basic Immunology. Functions and Disorders of the Immune System, 2nd updated edition, Sounders, Philadelphia, 2006, pp. 1–20.

[6] I. Ivanovski, A. Ivanovski, D. Nikolić, P. Ivanovski, Aluminium in brain tissue in autism, J. Trace Elem. Med. Biol. 51 (2019) 138–140.

[7] C. Gateff, E.H. Relyveld, G. Le Gonidec, et al., Study of a new pentavalent vaccine combination, Ann. Microbiol. 124 (1973) 387–409.

[8] M. Balwani, R.J. Desnick, K.E. Anderson, The porphyrias - δ-aminolevulinic acid dehydratase porphyria, in: R.M. Kliegman, B.F. Stanton, N.F. Scorn, J.O.W.L. St GemeIII (Eds.), Nelson Textbook of Pediatrics, Elsevier, 1600 John F. Kennedy Blvd. Ste. 1800 Philadelphia, PA 19103-2899, 2016p. 760.

[9] N.Z. Miller, Aluminium in childhood vaccines is unsafe, J. Am. Phys. Surg. 21 (2016) 109–117.

[10] How Common Is Autism? (2019) 17 February https://autismsciencefoundation.org/what-is-autism/how-common-is-autism/.

[11] A. Parker, Testing new hypotheses of neurological and immunological outcomes with aluminum-containing vaccines is warranted, J. Trace Elem. Med. Biol. 51 (2019) 28–30.

[12] N.C. Andrews, Disorders of iron metabolism, N. Engl. J. Med. 341 (26) (1999) 1986–1995.

[13] J.G. Dórea, R.C. Marques, Infants' exposure to aluminum from vaccines and breast milk during the first 6 months, J. Expo. Sci. Environ. Epidemiol. 20 (2010) 598–601.

[14] M.A. Sharpe, A.D. Livingston, D.S. Baskin, Thimerosal-derived ethylmercury is a mitochondrial toxin in human astrocytes: possible role of fenton chemistry in the oxidation and breakage of mtDNA, J. Toxicol. 373678 (2012) 12, https://doi.org/10.1155/2012/373678.

[15] P.N. Alexandrov, A.I. Pogue, W.J. Lukiw, Synergism in aluminum and mercury neurotoxicity, Integr. Food Nutr. Metab. 5 (May (3)) (2018), https://doi.org/10.15761/IFNM.1000214.

Ivan Ivanovski
Clinical Genetics Unit, AUSL-IRCCS Reggio Emilia, Reggio Emilia, 42123, Italy

Megan W. Fletcher
Faculty of Medicine, University of Belgrade, Studies in the English Language, 8 Dr Subotića str., Belgrade, 11000, Serbia

Ana Ivanovski
Faculty of Medicine, University of Belgrade, 8 Dr Subotića str., Belgrade, 11000, Serbia

Dimitrije Nikolić, Petar Ivanovski*
Faculty of Medicine, University of Belgrade, 8 Dr Subotića str., Belgrade, 11000, Serbia
E-mail address: petar.ivanovski@udk.bg.ac.rs (P. Ivanovski).

* Corresponding author.