Journal of Trace Elements in Medicine and Biology 93 (2026) 127810

Contents lists available at ScienceDirect

Journal of Trace Elements in Medicine and Biology

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

Aluminium adjuvants and childhood health: a call for science

Guillemette Crépeaux [a,b,*], Jeremy R. Hammond [c], Jonathan B. Handley [d], Brian Hooker [e], Karl Jablonowski [e], Lluís Luján [f,g], James Lyons-Weiler [h,i], Marika Nosten-Bertrand [j], Christopher A. Shaw [k], Yehuda Shoenfeld [l], Lucija Tomljenovic [c], Christopher Exley [m]

[a] Univ Paris Est Créteil, INSERM, IMRB, Créteil F-94010, France
[b] Ecole Nationale Vétérinaire d'Alfort, EnvA, IMRB, Maisons-Alfort F-94700, France
[c] Independent researcher
[d] Independent author
[e] Children's Health Defense, Franklin Lakes, NJ, USA
[f] Departamento de Patología Animal, Universidad de Zaragoza, Zaragoza, Spain
[g] Instituto Universitario de Investigación Mixto Agroalimentario de Aragón (IA2), Universidad de Zaragoza, Zaragoza 50013, Spain
[h] The Institute for Pure and Applied Knowledge, Pittsburgh, PA 15101, USA
[i] MAHA Institute, USA
[j] NeuroSU, Site du Fer à Moulin, UMR Inserm 1341, CNRS, Paris 8265, France
[k] Dept. of Ophthalmology and Visual Sciences, Program in Experimental Medicine and Program in Neuroscience, Univ. of British Columbia, Canada
[l] Zabludowicz Centre for Autoimmune Diseases, Sheba Medical Center, Tel Aviv University, Tel-Hashomer, Israel
[m] University of the Highlands and Islands, UHI House, Old Perth Rd, Inverness IV2 3JH, UK

ARTICLE INFO

Keywords:
Aluminium-Based Adjuvants
Vaccine safety
Neurodevelopmental outcomes
Autoimmune diseases
Epidemiology
Methodological flaws

ABSTRACT

In July 2025, Andersson et al. reported in Annals of Internal Medicine that early-life exposure to Aluminium (Al)-adjuvanted vaccines was not associated with increased risk of 50 chronic diseases, based on a Danish cohort of 1.2 million children. While widely cited as reassuring evidence of Al-Based Adjuvant (ABA) safety, closer scrutiny reveals major methodological and conceptual flaws. Specifically, the study demonstrates limited understanding of Al toxicology, weaknesses in cohort design and statistical analysis, and insufficient transparency regarding potential conflicts of interest. We argue that these shortcomings prevent meaningful conclusions about ABA safety, particularly in relation to neurodevelopmental and autoimmune outcomes, and highlight the need for more rigorous, transparent, and scientifically grounded investigations.

1. Introduction

In July 2025, Andersson et al. published a large-scale cohort study in *Annals of Internal Medicine* that concluded that exposure to Aluminium (Al)-adjuvanted vaccines in early childhood was not associated with an increased risk of 50 chronic diseases [1]. Two days later a correction was published with updated supplementary material [1, erratum]. While the Danish analysis performed on 1.2 million children is already cited as reassuring evidence of the safety of vaccines that include Al-Based Adjuvants (ABAs), a closer examination of the authors' published data reveals fundamental flaws that compromise the study's ability to detect harm. In addition, it raises concern about the role of ABAs in several chronic diseases, such as those with neurodevelopmental outcomes.

As experts in the fields of Al and ABAs' toxicology, epidemiology, neurodevelopmental disorders, autoimmunity, and vaccine-related injuries, with individual experience ranging from one to four decades, we are in a position to assess critically the scientific and methodological soundness of this study.

In the following sections, we briefly outline the study's major concerns that critically undermine its conclusions, including: i) the striking lack of knowledge regarding Al and ABAs; ii) critical shortcomings in cohort design and statistical analysis; iii) insufficient transparency and potential conflicts of interest.

In our view, all these biases prevent this work from reliably evaluating the safety of early-life exposure to ABAs. A study that fails - or refuses - to ask the right questions cannot yield meaningful answers, regardless of cohort size or indeed media coverage.

* Corresponding author at: Univ Paris Est Créteil, INSERM, IMRB, Créteil F-94010, France.
E-mail address: guillemette.crepeaux@vet-alfort.fr (G. Crépeaux).

https://doi.org/10.1016/j.jtemb.2025.127810
Received 15 September 2025; Received in revised form 19 December 2025; Accepted 19 December 2025
Available online 25 December 2025

2. Major concerns

2.1. Al and ABAs: the published science

2.1.1. Dismissal of prior evidence on ABA toxicity

Al salt as potassium alum has been added in vaccines since the 1920s [2]. Vaccines that include ABAs (Al oxyhydroxide, Al hydroxyphosphate and amorphous Al hydroxyphosphate sulfate) remain approved by regulatory agencies without any scientifically established safe limit for their Al content [3]. This is despite burgeoning experimental evidence in animals and humans concerning the biopersistence and neurotoxicity of ABAs and their links to chronic inflammatory conditions such as neurodevelopmental disorders and Auto Immune/inflammatory Syndrome Induced by Adjuvants (ASIA) [4–7]. Furthermore, ABAs' mechanisms of action are still not fully understood, and misconceptions about their pharmacokinetics persist [8], while cerebral translocation of ABAs in animal models and Al accumulation in human brain tissue have been repeatedly reported [9,10].

The authors of Andersson et al. seemingly disregard a significant body of these published data demonstrating cellular fate, biopersistence, cerebral translocation and multiple adverse effects (allergy, autoimmunity, neurological and neuroinflammatory events, behavioral alterations) in humans, animal and cellular models [4–7,10–16].

2.1.2. Weak studies' referencing

This glaring omission of numerous relevant peer-reviewed studies, while referencing outdated and largely irrelevant studies performed on different Al compounds and ways of exposure other than intramuscular and subcutaneous injection, undermines the authors' purported interest in the safety of ABAs and raises questions about their level of expertise in the subject. For instance, rather than engaging with more recent published toxicological research [17,18], Andersson et al. refer to outdated discredited U.S. Food and Drug Administration (FDA) models (e.g., [19]), which further weakens the study's foundation and indicates inadequate and superficial understanding of the toxicological issues related to ABA exposure by the authors of the study.

Similarly, the authors' reliance on methodologically limited studies that assess children's exposure to ABAs by measuring Al levels in blood or hair is misguided and reflects a fundamental misunderstanding of the known mechanisms governing the cellular capture and biopersistence of vaccine-derived insoluble particulate Al salts [20–23].

2.1.3. Erroneous exposure quantification

In a study claiming to investigate a potential link between exposure to certain compounds and the occurrence of adverse effects, accurate characterization of exposure is the cornerstone. However, it is evident that, in the present case, infant exposure to Al from vaccination is not properly estimated, rendering any attempt to interpret the data and draw conclusions invalid.

Al content and adjuvant types from manufacturers are not sufficient. Indeed, ABA exposure was inferred solely from vaccine records and manufacturer-reported Al content of each vaccine given to an infant. However, it has already been published that significant variability between vaccine batches exists, meaning that data given by manufacturers of vaccines on their Al content is at best random and in any case does not allow an accurate evaluation of ABA exposure [24]. Unpublished data also indicated that the Al content in the pediatric Neisvac vaccine was consistently four times lower than expected, with comparable unpublished variability observed in veterinary vaccines (personal data, GC & LL). Thus, infants receiving the same Al-adjuvanted vaccines may in fact receive vastly different amounts of Al, and in some cases, a single dose may contain more Al than several combined doses. This entirely random nature of the data on vaccine Al content described in a peer-reviewed paper published in 2021 has not been refuted either by vaccine manufacturers or by regulatory bodies such as the European Medicines Agency (EMA) or FDA. Al-content variability is compounded by the fact such agencies neither measure nor hold independent data on vaccine Al content.

The mere uncertainty surrounding the actual Al content of the vaccines recorded in the registry is, on its own, enough to nullify any reliable estimate of real exposure. This fundamental flaw renders all subsequent analyses and conclusions invalid, particularly given the study's narrow exposure range (0–4.5 mg, assessed per 1 mg increment), where even small misclassifications will critically distort risk assessment.

Furthermore, no distinction was made in the methodology between the different types of injected ABAs, despite significant differences in their physicochemical properties, which affect their pharmacokinetics and pharmacodynamics [14]. Ignoring these differences and treating all ABAs as a single Al exposure fails to capture the true variability and impact of each distinct compound. In the same time, the authors should have been more cautious also regarding information provided by manufacturers and regulatory product characteristics, as discrepancies in the reported chemical nature of ABAs (e.g., "*All formulations contain approximately 0.5 mg of aluminum (provided as amorphous aluminum hydroxyphosphate sulfate, previously referred to as aluminum hydroxide) per mL of vaccine*") can be read in FDA package insert of several vaccines such as Recombivax HB® [25], highlighting uncertainties that further weaken the accurate characterization of children's exposure. Such limitations warrant explicit discussion.

Body weight and age of exposure should be considered. In addition, the authors made no dose adjustment relative to recipient body weight, a critical parameter in neurodevelopmental toxicology [26].

In the same way, the exact exposure timing was ignored as the cumulative Al dose over 24 months was treated uniformly, masking potentially critical windows of susceptibility in early life, especially considering the role of the immature blood-brain barrier. This is also in opposition with previous studies showing that delaying or avoiding Al-containing vaccines appears associated with lower risk of atopic dermatitis and other allergic outcomes, with incidence highest before age 2 [27,28]. The question of timing is still neglected as exposure of premature babies was not detailed, whereas prematurity is a major risk factor of neurodevelopmental disorders [29]. This is wholly inconsistent with toxicological principles, which emphasize timing over cumulative quantity, especially concerning early exposures and neurodevelopmental risk assessment [26].

Other vaccination and other sources of Al exposure should be studied. No consideration was given to potential maternal vaccination during pregnancy, which can modulate fetal immune and neurological development as well as Al body burden of the baby [7]. Similarly, no consideration for a potential Al-booster injection at 5 years of age was given, including for children followed up to age 8. No consideration was given for potential co-administered vaccines such as Measles Mumps Rubella (MMR), which though not Al-adjuvanted, remain immunologically relevant. Live vaccines like measles can induce MCP-1 production [30], a chemokine shown to facilitate cerebral translocation of previously administered ABA in mice [31]. Given its administration after multiple Al-containing vaccines, MMR warrants consideration in the Danish study despite lacking Al, as it may modulate ABA-related neurotoxicity.

Allowing for the fact that a direct comparison between injected and ingested Al salts is not correct, no analysis was discussed regarding other Al environmental sources that may confound the estimated vaccine-specific burden. In particular, formula-fed infants are exposed to substantial amounts of Al through their diet [32]. This issue is further compounded by additional sources of Al, such as Al-containing medications including antacids and infant pain relievers.

Reduced exposure of injured babies. Because children who experienced early adverse reactions to vaccines may have had their vaccination schedules modified or interrupted, they may have been misclassified as "low exposure," introducing misdirection in the dose-response modeling. This risk of reverse causality is not addressed. These biases, previously

discussed by one of the authors in earlier work [33,34], are not compatible with drawing firm conclusions that precisely rely on the absence of such biases, as acknowledged by the authors themselves in the discussion of the study.

2.2. Cohort design, statistical analyses & data interpretation

Improper statistical adjustments made in Andersson's study have already been explained elsewhere [23,35]. The main concerns are discussed here in brief.

2.2.1. Systemic data bias evidenced by implausible protective effects of ABA exposure

By their own analysis, Andersson et al. are 95 % confident in the protective effects of 12 categorical diseases. Indeed, an increase in exposure of 1 mg Al via ABAs administered in the first two years of life was shown to reduce disease outcomes between 2 and 5 years old for: ulcerative colitis by 38.9 %; erythema nodosum by 35.1 %; asthma by 4.2 %; angioedema and urticaria by 11.1 %; allergy unspecified by 16.3 %; anaphylaxis or epinephrine autoinjector by 19.0 %; food allergy by 19.0 %; neurodevelopmental outcomes by 7.5 %; autistic disorder by 6.4 %; autism spectrum disorder composite by 7.5 %; attention deficit-hyperactivity disorder by 11.1 %; other pervasive developmental disorders by 12.4 %. To our knowledge and understanding, this is the first time in medical literature where exposure to Al has been shown to be beneficial. Bearing in mind what is known about human exposure to Al [36] these unlikely benefits seriously challenge the validity of the whole study and its conclusions, and this fails to meet Hill's criterion of biological plausibility.

2.2.2. Major selection biases in the cohort design

Absence of a control group. By omitting a truly unexposed control group (i.e., children with no ABA exposure), the study is unable to assess baseline toxicity or threshold effects. As previously explained in the section dedicated to erroneous exposure quantification, the only verifiable comparison authors could have made is between infants never receiving an Al-adjuvanted vaccine and infants receiving at least one Al-adjuvanted vaccine.

Exclusion of highly exposed children. Moreover, key exclusions may have strongly removed subpopulations at greatest risk for ABA-related outcomes. Indeed, the study excluded children with unusually high vaccination counts, discussed as potentially due to medical or registry errors (see above the section on the reliability of the national registry). It is worth noting that ABA exposure levels deemed "implausible" in the Danish schedule would actually be consistent with other schedules such as that published by the CDC [37]. It should also be noted that in response to online comments on this point, authors performed a reanalysis "*restricted to those fully vaccinated, and without excluding children with an implausible number of vaccinations*" and stated that "*similar overall estimates*" were observed [35]. Surprisingly, the results for atopic/allergic disorders are actually inconsistent: the initial analysis showed no meaningful association (HR 0.99, 95 % CI 0.98–1.01), whereas the reanalysis reported a marginally elevated risk (HR 1.03, 95 % CI 1.00–1.07), highlighting the sensitivity of estimates to methodological choices and rounding.

Omitting death outcome. The study excluded children who died before reaching two years of age. Mortality was not considered as a health endpoint, despite non-live vaccines, which generally are those containing ABAs, have been associated with an increased rate of childhood mortality [38,39]. Omitting death as an outcome limits the study's ability to capture the full spectrum of potential ABA-related adverse effects.

Exclusion of children under two with severe health conditions. The exclusion of patients with severe health issues prior to 24 months was applied without a clear rationale or justification. By removing this subgroup, potentially more susceptible to ABAs, the authors narrowed the scope of their analysis. Nonetheless, they concluded that ABA exposure is "safe for all", a statement that represents an overgeneralization not supported by the available data.

Focusing only on children from 2 to 5 years old. The study also excluded children diagnosed before age two, which is misleading, as for instance the incidence of atopic dermatitis is highest before this age for children followed by the age of 14 months (supplement figure 6 in [1]). The follow-up window, which ends for most children at age 5, is insufficient to capture many chronic conditions that manifest earlier or later, and even the secondary analysis extending follow-up to age 8 remains inadequate for detecting numerous relevant outcomes.

Exclusion of foreign population. The study excluded children whose mothers had not lived in Denmark for at least 2 years before childbirth, which raises the issue of excluding foreign populations, despite evidence that some may be at higher risk of neurodevelopmental disorders [40].

Exclusion of vulnerability groups. In addition, no subgroup analyses were performed for known biologically vulnerable groups such as preterm infants, children with low birth weight, or those with familial or genetic susceptibility to neurodevelopmental or autoimmune disorders. Finally, no stratified analysis by sex beyond statistical adjustments was provided, despite well-known sex differences in immune responses and neurodevelopmental outcomes [40,41].

Taken together, all these exclusions almost certainly mask or underestimate specific and potentially serious risks, rendering the study far too weak to support any reliable conclusions.

2.2.3. Biased statistical modeling and interpretation

Dose-response relationships. Statistical models assume linear dose–response relationships, yet Al exhibits non-linear effects. Biphasic, non-monotonic neurotoxic responses have indeed been observed both for dietary Al-*in vitro* [42–44] and *in vivo* [45–48], and also for ABAs [49]. The assumption that a certain amount of Al administered as a vaccine is needed to result in any infant health outcome is thus unsubstantiated and indeed contradicted by published studies.

Collider bias and petitio principii. Adjusting for covariates such as general practitioner visits before age 2 may introduce bias, as these visits can reflect both higher ABA exposure and early symptoms or adverse reactions, creating a "collider bias". For instance, children with asthma tend to have more medical visits, so adjusting for visit frequency could reduce any true association between asthma and Al exposure. In response to this online comment, Andersson et al. reanalyzed the data and reported "*similar results*." However, for atopic/allergic disorders, first analysis showed no meaningful association (HR 0.99, 95 % CI 0.98–1.01), while the second found a modest but statistically significant increase (HR 1.05, 95 % CI 1.04–1.07), indicating inconsistencies in the authors' own results and conclusions [35].

Additionally, treating birth year as a covariate may have artificially suppressed genuine associations by neutralizing concurrent increases over time in both Al exposure and disorder diagnoses, an example of *petitio principii*.

2.2.4. Overly confident conclusions unsupported by the data

Increased risks. More surprisingly, and beyond all that has just been presented, a review of the article and the supplemental material updated two days after publication highlights an increased risk of certain disorders with the highest ABA doses [23]. Andersson et al. did not directly analyze a zero-exposure group. However, by inferring the unexposed cohort from the supplemental data, it can be found that children who did not receive any Al-containing vaccines in their first two years were 25.7 % less likely to develop atopic dermatitis and 49.6 % less likely to develop allergic rhinoconjunctivitis between ages 2 and 5, compared with exposed children (respective unadjusted odds ratios of 0.796 (95 % CI: 0.698–0.907, $p = 0.0006$) and 0.668 (95 % CI: 0.579–0.771, $p < 0.0001$). Andersson et al. included 11 figures examining Al exposure and Asperger's syndrome, which is now classified under Level 1 Autism Spectrum Disorder [50,51]. All figures show a positive association with

Al exposure, with statistical significance emerging in Supplementary Figures 4 and 11 for children born after 2006 and those exposed to 0–1.5 mg compared with 3–4.5 mg cumulative Al dose. Supplementary Figure 11, the only analysis reporting risk differences and Al exposure as a categorical variable, found significantly higher risks for multiple neurodevelopmental outcomes in 464,378 children receiving > 3–4.5 mg aluminum versus 701,571 receiving > 1.5–3 mg, including + 9.73 overall neurodevelopmental cases per 10,000. In response to online comments noting these results contradicted their reassuring conclusions, the authors excluded children born before 2002 - removing 38 % of the moderate-dose group - citing a "positivity" violation, which erased statistical significance. Though possible, this would require early-birth moderate-dose children to have much lower risk than later-birth peers, a pattern not supported by Supplementary Figure 4.

Finally, the authors' analysis demonstrates evidence of harm induced by ABAs, and the reanalysis is insufficient to demonstrate otherwise. In any case, all these observations must be interpreted in light of the previously described mischaracterization of Al exposure in the dataset.

2.3. Transparency and conflicts of interest

2.3.1. Registry validity and access to raw data

Previous publications from the same group have faced important criticisms for registry misclassification (children recorded as unvaccinated who were in fact vaccinated and coding errors for type of vaccines) or underreporting adverse effects [35,52–55]. In the present study, registry data used for both exposure and outcomes were not independently validated, as access for external audit remains restricted. Indeed, no assessment of data quality, diagnostic accuracy, or registry misclassification was provided. Given the magnitude and inconsistency of the findings described, access to the underlying deidentified raw data would be valuable for independent verification and to advance the scientific debate. This lack of transparency limits reproducibility and public trust.

2.3.2. Potential conflicts of interest

The importance of the topic, combined with the range of criticisms highlighted here and elsewhere, raises legitimate questions regarding potential conflicts of interest that warrant transparent disclosure. The potential conflicts of interest appear to be the same as those in a previous study, which have already been discussed [33,54]. Briefly, several authors are affiliated with the Statens Serum Institut, a national entity involved in vaccine production. One of the authors reported affiliations to VAC4EU, a European vaccine surveillance consortium, and funding from Novo Nordisk Fonden and Lundbeckfonden, both closely linked to Danish health policy and biomedical interests. The Novo Nordisk Foundation, through its wholly owned subsidiary Novo Holdings A/S, maintains a controlling interest in Novo Nordisk A/S, the largest pharmaceutical company in Denmark. Consequently, the foundation providing research funding is also the controlling shareholder of a major pharmaceutical manufacturer. Although declared as "outside the scope" of the study, these affiliations raise legitimate concerns about impartiality, particularly given his consistent track record of publishing findings supportive of industry and public health agendas [56–59].

3. Conclusion

Despite being published in a leading medical journal, the study has serious methodological limitations, many are acknowledged by the authors themselves, yet its conclusions are presented with unjustified confidence. Key issues include uncertain real exposure, limited follow-up, absence of an unexposed comparison group, no stratification by known risk factors, and structural biases that prevent detection of moderate, delayed, or subgroup-specific harms. Notably, certain analyses in the study indicate statistically significant protective effects of ABA exposure, which is a huge indicator of the weakness of the study, as well as statistically significant increases in neurodevelopmental risks for children presumably receiving higher cumulative Al doses, though these findings are underreported and obscured by *post hoc* reanalysis decisions. The generalization of these findings to other populations, vaccine schedules, or healthcare contexts is methodologically unsound, ignoring critical differences in environmental exposures, genetic susceptibilities, and healthcare disparities.

That such a limited and internally inconsistent study was not only published in a high-impact medical journal, but also presented as reassurance in media coverage, raises uncomfortable questions. In a scientific climate where vaccine hesitancy is often framed as a crisis of misinformation, there may be institutional incentives - whether explicit or implicit - to produce and promote studies that accentuate claims of safety while minimizing uncertainty. However, science built on convenience rather than rigor does not serve public health. We assert that this study does not demonstrate the safety of ABAs. Framing this as definitive evidence of such safety misrepresents the data and undermines scientific integrity and public trust. As previously said "*The role of aluminum adjuvants in vaccines raises issues that deserve independent, rigorous and honest science*" [22].

If Not Now, When? [60].

Author statement

Guillemette Crépeaux: Conceived the study, drafted the initial manuscript, and supervised and validated the work. Jeremy R. Hammond, Jonathan B. Handley, Brian Hooker, Karl Jablonowski, Lluís Luján, James Lyons-Weiler, Marika Nosten-Bertrand, Christopher A. Shaw, Yehuda Shoenfeld, Lucija Tomljenovic, Christopher Exley: Contributed to writing and approved the final manuscript.

CRediT authorship contribution statement

Jeremy R. Hammond: Writing – review & editing, Validation. **Christopher Exley:** Writing – review & editing, Validation. **Guillemette Crépeaux:** Writing – review & editing, Writing – original draft, Supervision, Conceptualization. **Lucija Tomljenovic:** Writing – review & editing. **Yehuda Shoenfeld:** Writing – review & editing. **Christopher A. Shaw:** Writing – review & editing. **Marika Nosten-Bertrand:** Writing – review & editing. **James Lyons-Weiler:** Writing – review & editing. **Lluís Luján:** Writing – review & editing. **Karl Jablonowski:** Writing – review & editing, Formal analysis. **Brian Hooker:** Writing – review & editing, Formal analysis. **Jonathan B. Handley:** Writing – review & editing.

Funding

This study did not receive any specific external funding.

Declaration of Competing Interest

BH declares no financial conflicts of interest and he reports personal experience as the parent of an adult son with a health condition attributed to vaccination. JBH declares being a former board member of Children's Health Defense and the parent of a child with autism. LT has been remunerated as research consultant for the Los Angeles law firm Wisner Baum in an ongoing Gardasil HPV vaccine litigation.

References

[1] N.W. Andersson, I. Bech Svalgaard, S.S. Hoffmann, A. Hviid, Aluminum-adsorbed vaccines and chronic diseases in childhood: A nationwide cohort study (Epub), Ann. Intern Med (2025 Jul 15), https://doi.org/10.7326/ANNALS-25-00997.

[2] A.T. Glenny, C.G. Pope, H. Waddington, U. Wallace, Immunological notes. XVII–XXIV, J. Pathol. Bacteriol. 29 (1926) 31–40, https://doi.org/10.1002/path.1700290105.

[3] L. Angrand, R.K. Gherardi, G. Crépeaux, Regulatory limits of aluminium content of vaccines have not been set based on toxicological studies, Environ. Toxicol. Pharm. 119 (2025) 104812, https://doi.org/10.1016/j.etap.2025.104812.
[4] Y. Shoenfeld, N. Agmon-Levin, ASIA' – autoimmune/inflammatory syndrome induced by adjuvants, J. Autoimmun. 36 (2011) 4–8, https://doi.org/10.1016/j.jaut.2010.07.003.
[5] L. Luján, M. Perez, E. Salazar, N. Alvarez, M. Gimeno, P. Pinczowski, et al., Autoimmune/autoinflammatory syndrome induced by adjuvants (ASIA syndrome) in commercial sheep, Immunol. Res 56 (2013) 317–324, https://doi.org/10.1007/s12026-013-8400-4.
[6] R.K. Gherardi, G. Crépeaux, F.J. Authier, Myalgia and chronic fatigue syndrome following immunization: Macrophagic myofasciitis and animal studies support linkage to aluminum adjuvant persistency and diffusion in the immune system, Autoimmun. Rev. 18 (2019) 691–705, https://doi.org/10.1016/j.autrev.2019.05.006.
[7] L. Angrand, J.D. Masson, A. Rubio-Casillas, M. Nosten-Bertrand, G. Crépeaux, Inflammation and autophagy: A convergent point between autism spectrum disorder (ASD)-related genetic and environmental factors: Focus on aluminum adjuvants, Toxics 10 (2022) 518, https://doi.org/10.3390/toxics10090518.
[8] J.D. Masson, G. Crépeaux, F.J. Authier, C. Exley, R.K. Gherardi, Critical analysis of reference studies on the toxicokinetics of aluminum-based adjuvants, J. Inorg. Biochem 181 (2018) 87–95, https://doi.org/10.1016/j.jinorgbio.2017.12.015.
[9] C. Exley, M.J. Mold, Aluminium in human brain tissue: How much is too much? J. Biol. Inorg. Chem. 24 (2019) 1279–1282, https://doi.org/10.1007/s00775-019-01710-0.
[10] J.D. Masson, L. Angrand, G. Badran, R. de Miguel, G. Crépeaux, Clearance, biodistribution, and neuromodulatory effects of aluminum-based adjuvants: Systematic review and meta-analysis of animal studies, Crit. Rev. Toxicol. 52 (2022) 403–419, https://doi.org/10.1080/10408444.2022.2105688.
[11] L. Tomljenovic, C. A. Shaw, Aluminum vaccine adjuvants: Are they safe? Curr. Med Chem. 18 (2011) 2630–2637, https://doi.org/10.2174/092986711795933740.
[12] M. Mold, E. Shardlow, C. Exley, Insight into the cellular fate and toxicity of aluminium adjuvants used in clinically approved human vaccinations, Sci. Rep. 6 (2016) 31578, https://doi.org/10.1038/srep31578.
[13] E. Shardlow, M. Mold, C. Exley, From stock bottle to vaccine: Elucidating the particle size distributions of aluminum adjuvants using dynamic light scattering, Front Chem. 4 (2017) 48, https://doi.org/10.3389/fchem.2016.00048.
[14] E. Shardlow, M. Mold, C. Exley, Unraveling the enigma: Elucidating the relationship between the physicochemical properties of aluminium-based adjuvants and their immunological mechanisms of action, Allergy Asthma Clin. Immunol. 14 (2018) 80, https://doi.org/10.1186/s13223-018-0305-2.
[15] E. Shardlow, M. Mold, C. Exley, The interaction of aluminium-based adjuvants with THP-1 macrophages in vitro: Implications for cellular survival and systemic translocation, J. Inorg. Biochem 203 (2020) 110915, https://doi.org/10.1016/j.jinorgbio.2019.110915.
[16] J.D. Masson, G. Badran, R.K. Gherardi, F.J. Authier, G. Crépeaux, Widespread myalgia and chronic fatigue: Phagocytes from macrophagic myofasciitis patients exposed to aluminum oxyhydroxide-adjuvanted vaccine exhibit specific inflammatory, autophagic, and mitochondrial responses, Toxics 12 (2024) 491, https://doi.org/10.3390/toxics12070491.
[17] J. Lyons-Weiler, R. Ricketson, Reconsideration of the immunotherapeutic pediatric safe dose levels of aluminum, J. Trace Elem. Med Biol. 48 (2018) 67–73, https://doi.org/10.1016/j.jtemb.2018.02.025.
[18] G. McFarland, E. La Joie, P. Thomas, J. Lyons-Weiler, Acute exposure and chronic retention of aluminum in three vaccine schedules and effects of genetic and environmental variation, J. Trace Elem. Med Biol. 58 (2020) 126444, https://doi.org/10.1016/j.jtemb.2019.126444.
[19] R.J. Mitkus, D.B. King, M.A. Hess, R.A. Forshee, M.O. Walderhaug, Updated aluminum pharmacokinetics following infant exposures through diet and vaccination, Vaccine 29 (2011) 9538–9543, https://doi.org/10.1016/j.vaccine.2011.09.124.
[20] T.Z. Movsas, N. Paneth, W. Rumbeiha, J. Zyskowski, I.H. Gewolb, Effect of routine vaccination on aluminum and essential element levels in preterm infants, JAMA Pedia 167 (2013) 870–872, https://doi.org/10.1001/jamapediatrics.2013.108.
[21] M.P. Karwowski, C. Stamoulis, L.M. Wenren, G.M. Faboyede, N. Quinn, K.M. Gura, D.C. Bellinger, A.D. Woolf, Blood and hair aluminum levels, vaccine history, and early infant development: A cross-sectional study, Acad. Pedia 18 (2018) 161–165, https://doi.org/10.1016/j.acap.2017.09.003.
[22] G. Crépeaux, F.J. Authier, C. Exley, L. Luján, R.K. Gherardi, The role of aluminum adjuvants in vaccines raises issues that deserve independent, rigorous and honest science, J. Trace Elem. Med Biol. 62 (2020) 126632, https://doi.org/10.1016/j.jtemb.2020.126632.
[23] K. Jablonowski, B. Hooker, Neurodev. Dis. Assoc. Alum. adsorbed Vaccin. A Nationwide cohort Study [Prepr.] Prepr. Org. (2025), https://doi.org/10.20944/preprints202508.0280.v1.
[24] E. Shardlow, C. Linhart, S. Connor, E. Softely, C. Exley, The measurement and full statistical analysis including Bayesian methods of the aluminium content of infant vaccines, J. Trace Elem. Med Biol. 66 (2021) 126762, https://doi.org/10.1016/j.jtemb.2021.126762.
[25] FDA, Recombivax HB package insert, (2025). ⟨https://www.fda.gov/files/vaccines%2C%20blood%20&%20biologics/published/package-insert-recombivax-hb.pdf⟩.
[26] W. Slikker Jr, M.G. Paule, C. Wang, Handbook of developmental neurotoxicology, 2nd ed., Academic Press, 2018, https://doi.org/10.1016/C2015-0-04830-4.
[27] N. Kiraly, J.J. Koplin, N.W. Crawford, S. Bannister, R.J. Boyle, R. Loh, M. C. Matheson, M.L.K. Tang, M. Wake, S.C. Dharmage, K.J. Allen, Timing of routine infant vaccinations and risk of food allergy and eczema at one year of age, Allergy 71 (2016) 541–549, https://doi.org/10.1111/all.12830.
[28] L. Gehrt, A. Rieckmann, N. Kiraly, K. Bønnelykke, J. Stokholm, H. Bisgaard, Timeliness of DTaP-IPV-Hib vaccination and development of atopic dermatitis between 4 months and 1 year of age: Register-based cohort study, J. Allergy Clin. Immunol. Pr. 9 (2021) 1520–1528, https://doi.org/10.1016/j.jaip.2020.11.056.
[29] B. Fleiss, P. Gressens, H.B. Stolp, Cortical gray matter injury in encephalopathy of prematurity: Link to neurodevelopmental disorders, Front Neurol. 11 (2020) 575, https://doi.org/10.3389/fneur.2020.00575.
[30] K.J. Jensen, M. Søndergaard, A. Andersen, E. Sartono, C. Martins, M.L. Garly, J. Eugen-Olsen, H. Ullum, M. Yazdanbakhsh, P. Aaby, C.S. Benn, C. Erikstrup, A randomized trial of an early measles vaccine at 4½ months of age in Guinea-Bissau: Sex-differential immunological effects, PLoS One 9 (2014) e97536, https://doi.org/10.1371/journal.pone.0097536.
[31] Z. Khan, C. Combadière, F.J. Authier, V. Itier, F. Lux, C. Exley, M. Mahrouf-Yorgov, X. Decrouy, P. Moretto, O. Tillement, R.K. Gherardi, J. Cadusseau, Slow CCL2-dependent translocation of biopersistent particles from muscle to brain, BMC Med 11 (2013) 99, https://doi.org/10.1186/1741-7015-11-99.
[32] N. Chuchu, B. Patel, B. Sebastian, C. Exley, The aluminium content of infant formulas remains too high, BMC Pedia 13 (2013) 162, https://doi.org/10.1186/1471-2431-13-162.
[33] A. Hviid, J.V. Hansen, M. Frisch, M. Melbye, Measles, mumps, rubella vaccination and autism, Ann. Intern Med 171 (2019) 388, https://doi.org/10.7326/L19-0383.
[34] A. Jain, J. Marshall, A. Buikema, et al., Autism occurrence by MMR vaccine status among US children with older siblings with and without autism, JAMA 313 (2015) 1534–1540, https://doi.org/10.1001/jama.2015.3077.
[35] N.W. Andersson, I. Bech Svalgaard, S.S. Hoffmann, A. Hviid, Online comments, Ann Intern Med. (2025) ⟨https://www.acpjournals.org/doi/10.7326/ANNALS-25-00997⟩.
[36] C. Exley, Human exposure to aluminium, Environ. Sci. Process Impacts 15 (2013) 1807–1816, https://doi.org/10.1039/c3em00374d.
[37] CDC, Child and adolescent immunization schedule by age, (2025). ⟨https://www.cdc.gov/vaccines/hcp/imz-schedules/child-adolescent-age.html⟩.
[38] J.P. Higgins, K. Soares-Weiser, J.A. López-López, A. Kakourou, K. Chaplin, H. Christensen, N.K. Martin, J.A. Sterne, A.L. Reingold, Association of BCG, DTP, and measles containing vaccines with childhood mortality: Systematic review, BMJ 355 (2016) i5170, https://doi.org/10.1136/bmj.i5170.
[39] S.W. Mogensen, A. Andersen, A. Rodrigues, C.S. Benn, P. Aaby, The introduction of diphtheria-tetanus-pertussis and oral polio vaccine among young infants in an urban African community: A natural experiment, EBioMedicine 17 (2017) 192–198, https://doi.org/10.1016/j.ebiom.2017.01.041.
[40] K.A. Shaw, S. Williams, M.E. Patrick, M. Valencia-Prado, M.S. Durkin, E. M. Howerton, C.M. Ladd-Acosta, E.T. Pas, A.V. Bakian, P. Bartholomew, et al., Prevalence and early identification of autism spectrum disorder among children aged 4 and 8 years: Autism and Developmental Disabilities Monitoring Network, 16 sites, United States, 2022, MMWR Surveill. Summ. 74 (2025) 1–22, https://doi.org/10.15585/mmwr.ss7402a1.
[41] S. Sharma, A. Gibbons, E.O. Saphire, Sex differences in tissue-specific immunity and immunology, Science 389 (2025) 599–603, https://doi.org/10.1126/science.adx4381.
[42] C. Exley, J.D. Birchall, An aluminium adjuvant in a vaccine is an acute exposure to aluminium, J. Trace Elem. Med Biol. 57 (1992) 57–59, https://doi.org/10.1016/j.jtemb.2020.126632.
[43] B. Platt, D.O. Carpenter, D. Büsselberg, K.G. Reymann, G. Riedel, Aluminum impairs hippocampal long-term potentiation in rats in vitro and in vivo, Exp. Neurol. 134 (1995) 73–86, https://doi.org/10.1006/exnr.1995.1056.
[44] H. Eidi, M.O. David, G. Crépeaux, L. Henry, V. Joshi, M.H. Berger, M. Sennour, J. Cadusseau, R.K. Gherardi, P.A. Curmi, Fluorescent nanodiamonds as a relevant tag for the assessment of alum adjuvant particle biodisposition, BMC Med 13 (2015) 144, https://doi.org/10.1186/s12916-015-0388-2.
[45] S. Kumar, Biphasic effect of aluminium on cholinergic enzyme of rat brain, Neurosci. Lett. 248 (1998) 121–123, https://doi.org/10.1016/S0304-3940(98)00331-6.
[46] M. Tsunoda, R.P. Sharma, Altered dopamine turnover in murine hypothalamus after low-dose continuous oral administration of aluminum, J. Trace Elem. Med Biol. 13 (1999) 224–231, https://doi.org/10.1016/S0946-672X(99)80005-1.
[47] K. Kim, Perinatal exposure to aluminum alters neuronal nitric oxide synthase expression in the frontal cortex of rat offspring, Brain Res Bull. 61 (2003) 437–441, https://doi.org/10.1016/S0361-9230(03)00150-6.
[48] J.L. Roig, S. Fuentes, M.T. Colomina, P. Vicens, J.L. Domingo, Aluminum, restraint stress and aging: Behavioural effects in rats after 1 and 2 years of aluminum exposure, Toxicology 218 (2006) 112–124, https://doi.org/10.1016/j.tox.2005.10.013.
[49] 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) 48–57, https://doi.org/10.1016/j.tox.2016.11.018.
[50] American Psychiatric Association, Diagnostic and statistical manual of mental disorders, 5th ed., text revision, American Psychiatric Publishing, 2022, https://doi.org/10.1176/appi.books.9780890425787.
[51] S.A. Hosseini, M. Molla, Asperger syndrome, In: StatPearls [Internet], StatPearls Publishing, Treasure Island (FL), 2025 Jan. ⟨https://www.ncbi.nlm.nih.gov/books/NBK556081/⟩.
[52] N. Holt, A. Mygind, F. Bro, Danish MMR vaccination coverage is considerably higher than reported, Dan. Med J. 64 (2017). ⟨https://pubmed.ncbi.nlm.nih.gov/28157059/⟩.

[53] T.N. Nissen, N.M. Birk, G. Smits, et al., Bacille Calmette-Guérin (BCG) vaccination at birth and antibody responses to childhood vaccines: A randomised clinical trial, Vaccine 35 (2017) 2084–2091, https://doi.org/10.1016/j.vaccine.2017.03.009.
[54] J.R. Hammond, J. Varia, B. Hooker, Hviid, et al., Vaccine-autism study: Much ado about nothing? J. Biotechnol. Biomed. 8 (2025) (2019) 118–140, https://doi.org/10.26502/jbb.2642-91280185.
[55] K. Jablonowski, B. Hooker, Unadjusted analysis of a population-based study of measles, mumps, and rubella vaccination and autism, Integr Med (Encinitas). 24 (2025) 10–12. ⟨https://pubmed.ncbi.nlm.nih.gov/40703412/⟩.
[56] L. Arnheim-Dahlström, B. Pasternak, H. Svanström, P. Sparén, A. Hviid, Autoimmune, neurological, and venous thromboembolic adverse events after immunisation of adolescent girls with quadrivalent human papillomavirus vaccine in Denmark and Sweden: cohort study, BMJ 347 (2013) f5906, https://doi.org/10.1136/bmj.f5906.
[57] A. Hviid, H. Svanström, N.M. Scheller, O. Grönlund, B. Pasternak, L. Arnheim-Dahlström, Human papillomavirus vaccination of adult women and risk of autoimmune and neurological diseases, J. Intern Med 283 (2018) 154–165, https://doi.org/10.1111/joim.12694.
[58] A. Hviid, N.M. Thorsen, P. Valentiner-Branth, M. Frisch, K. Mølbak, Association between quadrivalent human papillomavirus vaccination and selected syndromes with autonomic dysfunction in Danish females: population based, self-controlled, case series analysis, BMJ 370 (2020) m2930, https://doi.org/10.1136/bmj.m2930.
[59] N.W. Andersson, E.M. Thiesson, J.V. Hansen, A. Hviid, Safety of BA.4-5 or BA.1 bivalent mRNA booster vaccines: nationwide cohort study, BMJ 382 (2023) e075015, https://doi.org/10.1136/bmj-2023-075015.
[60] P. Levi, If not now, when?, London, Abacus, 1982.