

Aluminum

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Abstract

This page draws on the EFSA AFC Panel 2008 Scientific Opinion on the Safety of Aluminium from Dietary Intake (EFSA 2008), the ATSDR 2008 Toxicological Profile for Aluminum (ATSDR 2008), and the JECFA Combined Compendium of Food Additive Specifications (JECFA FA Compendium).

Chapter-level cross-metal toxicology context for aluminum exposure, poor oral absorption, transferrin binding, renal excretion, dialysis dementia, bone effects, neurotoxicity, and chelation treatment is connected from Ufelle & Barchowsky 2021.

1 | Overview

Aluminum is the third most abundant element in Earth's crust and a non-essential metal that enters the human food system through aluminum-containing food additives, aluminum-amended drinking water (used as a flocculant in water treatment), aluminum cookware and food packaging in contact with acidic foods, antacid medications, and natural occurrence in some plant-based foods. Aluminum is poorly absorbed from the gastrointestinal tract (typically less than 1 percent), but cumulative exposure across the food supply produces dietary intakes that the EFSA AFC Panel 2008 concluded routinely exceed the established tolerable weekly intake of 1 mg Al/kg body weight per week in significant portions of the European population.

The toxicological concerns associated with aluminum focus on neurotoxicity (most clearly documented in dialysis-related encephalopathy from aluminum-contaminated dialysate fluid in the 1970s and 1980s, and in chronic occupational exposure), reproductive and developmental effects, and bone effects (aluminum-induced osteomalacia in dialysis patients was a significant clinical entity before water treatment for dialysis was redesigned). The EFSA TWI of 1 mg/kg b.w./week (2008) followed JECFA's own lowering of its provisional tolerable weekly intake from 7 mg/kg b.w./week to 1 mg/kg b.w./week at its 67th meeting (2006); JECFA subsequently revised its PTWI upward to 2 mg/kg b.w./week at its 74th meeting (2011, WHO Technical Report Series 966), where it currently stands. EFSA's TWI (1 mg/kg) and JECFA's current PTWI (2 mg/kg) are set by separate expert bodies and genuinely differ, with EFSA the more conservative.

Aluminum's health profile is qualitatively different from the other metals on this wiki. The clearest documented adverse health effects (dialysis encephalopathy, dialysis osteomalacia) occur in patient populations with impaired aluminum excretion (renal failure) and intravenous-route exposure that bypasses GI absorption barriers; for the general population with normal renal function and oral exposure, the dose-response evidence for aluminum-related disease is weaker than for cadmium, lead, mercury, or arsenic, and the regulatory TWI rests largely on extrapolation from animal neurotoxicity studies rather than on robust human cohort data at typical dietary exposure levels.

Ufelle & Barchowsky 2021 is attached here as textbook-level toxicology context only. It does not supply aluminum occurrence values for foods or product categories.

2 | At a glance

Three facts that matter most for a consumer trying to interpret aluminum exposure.

First, aluminum exposure for most consumers is dominated by aluminum-containing food additives (sodium aluminium phosphate as a leavening agent in baked goods, sodium aluminium silicate as an anti-caking agent in salt and powdered foods, aluminium-containing colorants), antacid medications, and aluminum-cookware contact with acidic foods (tomato sauce, pickles, citrus). Naturally occurring aluminum in plants is generally a smaller contributor. EFSA 2008 concluded that the population-level TWI of 1 mg/kg b.w./week is exceeded in significant fractions of the European population, particularly among consumers of foods containing aluminium-based additives.

Second, the clearest documented adverse health effects of aluminum (dialysis encephalopathy, dialysis osteomalacia) occur in patient populations with impaired renal excretion of aluminum and via routes (intravenous dialysate) that bypass the protective GI absorption barrier of typical dietary exposure. For the general consumer with normal renal function and oral exposure only, the regulatory TWI is the primary benchmark; documented neurological or musculoskeletal disease from typical dietary aluminum is uncommon and difficult to attribute to aluminum specifically rather than to confounding factors.

Third, aluminum cookware does contribute to dietary aluminum, particularly when cooking acidic foods (tomato sauce, citrus dishes, vinegar-based preparations) for prolonged periods. Anodized aluminum cookware substantially reduces this leaching; stainless steel and cast iron alternatives eliminate it. The leaching contribution is generally modest relative to additive and antacid contributions for the average consumer.

3 | Toxicology

The EFSA AFC Panel 2008 TWI of 1 mg Al/kg b.w./week is anchored on neurotoxic, reproductive, and developmental effects in animal studies. The Panel did not derive a single critical study and reference point; the TWI integrates evidence across multiple animal endpoints with safety factors for interspecies extrapolation and human variability. The replacement of the prior JECFA PTWI of 7 mg/kg b.w./week with the EFSA value of 1 mg/kg b.w./week reflects accumulated evidence at lower exposures and a more conservative evaluation.

The clearest documented human aluminum disease entities are dialysis encephalopathy and dialysis osteomalacia, both observed in chronic hemodialysis patients exposed to aluminum-contaminated dialysate fluid in the 1970s and 1980s before water treatment for dialysis was redesigned (ATSDR 2008).

Dialysis encephalopathy presented with progressive dementia, dysarthria, myoclonus, and seizures; dialysis osteomalacia produced bone pain, fractures, and characteristic radiographic and histological findings. Both

entities are essentially eliminated in modern dialysis practice because dialysate water is now routinely treated to remove aluminum.

4 | Typical exposure routes

Dietary intake is the dominant route. EFSA 2008 estimated typical adult European dietary aluminum exposure at 0.2 to 1.5 mg/kg b.w./week (potentially higher in some sub-populations), with substantial variation across Member States driven by aluminum-additive use patterns. Antacid medications can contribute substantially to aluminum intake in users (typical aluminum-containing antacid daily aluminum delivery 100 to 1000 mg, dwarfing typical dietary intake of 5 to 15 mg/day) (ATSDR 2008). Drinking water aluminum (from aluminum-based water-treatment flocculants in some municipal systems) is typically below 0.2 mg/L and a smaller contributor than dietary additives (ATSDR 2008).

GI absorption of aluminum is typically less than 1 percent (ATSDR 2008: 0.07-0.39 percent for drinking water, ~0.1 percent for diet).

Excretion is primarily renal. Renal failure is the major risk factor for aluminum body-burden accumulation; this is why dialysis-related encephalopathy and osteomalacia were the canonical aluminum disease entities before dialysate-water treatment was implemented (ATSDR 2008).

5 | Food sources

MATRIX	ALUMINUM CONCERN
Foods with aluminium-based additives	Sodium aluminium phosphate (leavening), sodium aluminium silicate (anti-caking), aluminium-containing colorants
Aluminum-containing antacids	Single largest aluminum exposure source for users (100 to 1000 mg/day)
Acidic foods cooked or stored in aluminum	Tomato sauce, citrus, vinegar-based preparations cooked for prolonged periods in non-anodized aluminum cookware
Drinking water (post-aluminum-flocculation treatment)	Typically <0.2 mg/L; varies by treatment-plant practice
Tea	Naturally elevated aluminum content; brewed tea can deliver 1 to 5 mg/L
Processed cheese (aluminium-containing emulsifying salts)	Sodium aluminium phosphate as emulsifier in some products
Baked goods with aluminium-based leavening	Cakes, biscuits, pancakes using aluminum baking powder
Infant formula	Variable by product; some soy-based formulas higher than milk-based; prescription formulas span 41.4 to 1956 µg/L (Redgrove 2019)

EFSA 2008 estimated dietary exposure at 0.3 to 0.9 mg/kg b.w./week for milk-based infant formulas and 1.1 mg/kg b.w./week for soya-based formulas; breast-fed infants were estimated at less than 0.07 mg/kg b.w./week.

Primary occurrence: complementary infant foods (Brazilian market)

de Paiva et al. 2020 surveyed 95 infant food samples across 9 commercial brands in Campinas, Brazil, covering salty purees, fruit purees, infant drinks (including soy-based), and petit-suisse. Total Al by ICP-OES after microwave digestion; bioaccessibility by optimized in vitro gastrointestinal digestion. The highest total Al concentrations: petit-suisse 4170 µg/kg, soy-based drink 2860 µg/kg, mixed-vegetable salty purees 2310-2760 µg/kg, dark chocolate milk drink 2175 µg/kg, fruit purees 1900-2500 µg/kg. The bioaccessibility findings substantially modify the exposure picture: percent of total Al released to gut absorption ranges from 0.5 percent (apple-and-plum puree) to 48 percent (cereal milk drink), a 96-fold spread driven by matrix composition. High-cellulose plant purees and high-protein dairy reduce bioaccessibility through Al-cellulose and Al-protein binding; high-polyphenol or low-fiber matrices increase it. Consumption of three portions per day of soy-based drink delivers a substantial fraction of the EFSA TWI; handmade-gourmet brands carried 2-3x higher Al than industrial counterparts at similar compositions, reflecting variable contamination control across production scales. The bioaccessibility distinction is methodologically important and currently absent from EU and US regulatory frameworks for Al in infant food.

Primary occurrence: prescription infant formulas

Redgrove et al. 2019 surveyed 24 UK prescription infant formulas (ready-to-drink and powdered) by transversely heated graphite furnace atomic absorption spectrometry following microwave-assisted acid/peroxide digestion. Ready-to-drink concentrations ranged from 49.9 µg/L (Cow & Gate Nutriprem 1 for preterm infants) to 1956.3 µg/L (Abbott PediaSure Plus Juice Apple, a weight-gain supplement), a roughly 40-fold range across products fed to medically vulnerable infants. Abbott PediaSure Plus Juice Apple delivers 391 µg Al per 200 mL serving. The fortified-weight-gain product subcategory (Danone Nutricia Fortini range, Abbott PediaSure Plus, Nutrinovo ProSource) consistently exceeded 500 µg/L. The amino-acid-based and peptide-based powdered subcategory (SMA Alfamino, Neocate LCP, Aptamil Pepti 1) showed the lowest concentrations: SMA Alfamino at 41.4 µg/L was the lowest-Al formula measured in the Keele group's accumulated literature. The roughly 50-fold range from cleanest to most contaminated demonstrates that infant formula Al contamination is not inevitable; selected ingredients can produce dramatically lower Al concentrations even within prescription-grade products. The likely sources of contamination identified by Redgrove include whey protein hydrolysate ingredients (the Keele group separately measured 4.1 to 8.1 µg/g Al in whey hydrolysates supplied to a major formula manufacturer), fruit and fruit-flavoring components, packaging, and aluminum-based processing equipment.

What this means for food choice

For consumers with normal renal function: the EFSA TWI exceedance documented at the population level does not translate to a clear individual action threshold for most consumers. The leverage points in approximate order of impact:

For frequent antacid users: aluminum-containing antacids deliver 100 to 1000 mg of aluminum per daily dose, dwarfing dietary intake. Switching to non-aluminum antacids (calcium carbonate, magnesium hydroxide, proton pump inhibitors) eliminates this exposure. Patients with chronic acid-related symptoms should discuss antacid choice with their physician, particularly if renal function is impaired.

For consumers concerned about additive intake: reading ingredient labels for sodium aluminium phosphate (SALP), sodium aluminium silicate, aluminium ammonium sulfate, and aluminum-containing colorants (including some FD&C lake colors) identifies the additive sources. Choosing baked goods with non-aluminum leavening (cream of tartar plus baking soda, or "aluminum-free" baking powder), and choosing salt and powdered products without anti-caking-agent aluminum, are the operative substitutions. Many "aluminum-free" baking powders are now available in mainstream markets.

For aluminum cookware users: anodized aluminum cookware substantially reduces aluminum leaching into food. Stainless steel, cast iron, glass, and ceramic cookware eliminate aluminum leaching. The cookware contribution is generally modest relative to additive and antacid contributions, but for consumers cooking acidic foods (tomato sauce, citrus, vinegar) regularly in non-anodized aluminum, switching cookware is a reasonable precaution.

For pregnant women and parents of infants: soy-based infant formulas were documented by EFSA 2008 to deliver higher aluminum than milk-based formulas, which deliver higher aluminum than breastfeeding. For non-breastfeeding situations, milk-based formula is the lower-aluminum choice unless other clinical factors (allergy, intolerance) require soy.

6 | Regulatory limits

JURISDICTION / BODY	TYPE	VALUE	PAGE
EFSA (EU)	Dietary TWI	1 mg Al/kg b.w./week	efsa-aluminium-twi
JECFA (international)	PTWI	2 mg Al/kg b.w./week (74th meeting, 2011, WHO TRS 966)	Source ingest pending
Prior JECFA PTWI (superseded)	1 mg/kg (67th meeting, 2006); 7 mg/kg before then	superseded by the 2011 revision	
US ATSDR	Chronic oral MRL	1 mg Al/kg/day	atsdr-aluminum-mrls
US ATSDR	Intermediate oral MRL	1 mg Al/kg/day	atsdr-aluminum-mrls
EU	Maximum levels for aluminium in food contact materials	Regulation (EU) 10/2011 amendments	Pending separate ingest

What the reference values mean in practice

The EFSA TWI of 1 mg Al/kg b.w./week corresponds to 70 mg Al/week or 10 mg Al/day for a 70-kilogram adult. EFSA's estimate of typical European adult dietary intake is 0.2 to 1.5 mg/kg b.w./week (14 to 105 mg/week for the same adult), placing the population mean at the upper end of the TWI range and the high consumers above. This is the regulatory finding that "the TWI is likely to be exceeded in a significant part of the European population."

For an individual consumer: a daily diet without aluminum-additive-heavy processed foods, without antacid use, and using non-aluminum cookware would typically deliver well under the TWI. Adding aluminum-containing antacids to the dietary baseline can rapidly push intake to 10 to 100 times the TWI; this is why antacid users are the most consequential subpopulation for aluminum exposure regulatory concern.

7 | Testing

Biomonitoring for aluminum exposure typically measures serum or plasma aluminum (operative for dialysis-patient monitoring) or urinary aluminum (recent integrated exposure).

8 | Microbiome effects

Pending dedicated microbiome ingests. Aluminum-microbiome interactions are not a major area of established evidence. The wikibiome-crosswalk anchors are not yet established.

9 | Historical context: dialysis encephalopathy

Dialysis encephalopathy (also called dialysis dementia) was the canonical clinical entity that established aluminum's neurotoxicity in humans. Beginning in the 1970s, hemodialysis patients in some centers developed progressive dementia with dysarthria, myoclonus, and seizures. The cause was traced to aluminum contamination of dialysate water, which entered patients' circulation directly via the dialyzer. After the etiology was identified, treatment of dialysate water to remove aluminum (typically with reverse osmosis) eliminated new cases. Concurrent dialysis osteomalacia, with similar dialysate-aluminum etiology, also resolved with the dialysate-water-treatment intervention. These two clinical entities together constitute the strongest human evidence for aluminum's adverse health effects and are the empirical anchor for aluminum-related neurotoxicity and bone-effect concerns at the regulatory level.

The lessons from dialysis encephalopathy are: (a) aluminum is a potential neurotoxicant and bone-affecting agent in humans at sufficient body-burden levels; (b) renal function is the critical variable controlling aluminum body-burden accumulation; (c) intravenous and dialysis routes bypass the protective GI absorption barrier and produce body-burden far above what oral dietary exposure achieves at any reasonable intake. The TWI is set as a population-protective threshold for normal-renal-function oral-exposure consumers, with the dialysis evidence providing the proof-of-concept that aluminum can produce disease at sufficient body burden.

10 | Vulnerable populations

POPULATION	BASIS
Hemodialysis patients (legacy concern)	Dialysate-water aluminum was the cause of dialysis encephalopathy and osteomalacia; modern dialysis water treatment essentially eliminates this exposure
Patients with chronic renal impairment	Reduced aluminum excretion produces body-burden accumulation; aluminum-containing antacids and phosphate binders are particular concerns
Frequent users of aluminum-containing antacids	Daily aluminum delivery 100 to 1000 mg dwarfs dietary intake
Consumers of aluminum-additive-heavy diets	Population-level TWI exceedance documented in EFSA 2008
Infants on soy-based formula	Higher aluminum than milk-based formula or breastfeeding (EFSA 2008)
Aluminum-industry workers	Inhalation exposure to aluminum dust in smelter and finishing operations

If you are in one of these groups

For patients with chronic kidney disease or on dialysis: aluminum is a clinical concern your nephrology team monitors. Aluminum-containing antacids and aluminum-containing phosphate binders are typically avoided in

this population; non-aluminum alternatives are standard. Discuss any over-the-counter antacid use with your nephrology team.

For frequent antacid users with normal renal function: consider switching from aluminum-containing antacids (Maalox, Mylanta, Amphojel) to non-aluminum alternatives (calcium carbonate antacids like Tums, magnesium hydroxide preparations, proton pump inhibitors for chronic acid-related conditions). The decision depends on your clinical pattern; discuss with a physician if you use antacids more than occasionally.

For parents of infants: breastfeeding is the lowest-aluminum infant feeding option. When formula feeding is required, milk-based formula carries less aluminum than soy-based formula. Specialized formulas for medical conditions should be chosen on clinical rather than aluminum grounds; aluminum is not a primary consideration for most formula-feeding decisions in non-renal-impaired infants.

For consumers concerned about additive intake: read labels for "sodium aluminium phosphate," "sodium aluminium silicate," "aluminium ammonium sulfate," and aluminum-containing colorants. Choose non--aluminum-leavened baked goods (look for "aluminum-free baking powder" on labels). Anodized aluminum cookware reduces leaching relative to non-anodized; stainless steel, cast iron, and glass eliminate it.

11 | App-layer integration

Machine-readable takeaways from this synthesis for the Heavy Metal Index consumer app pipeline.

The aluminum reference values differ by body: EFSA's TWI is 1 mg/kg b.w./week (the more conservative) and JECFA's current PTWI is 2 mg/kg b.w./week (74th meeting, 2011). The app can present a "percent of TWI" benchmark using the more conservative EFSA value as the primary aluminum signal. Default reference: 1 mg Al/kg b.w./week (EFSA).

Critical app handling: aluminum-containing antacid use is the dominant single exposure source for users and dwarfs dietary intake. The app should explicitly query antacid use as a separate input and add antacid-derived aluminum to dietary aluminum for total exposure estimation. Without this query, dietary-only estimates substantially understate total aluminum intake for antacid users.

Pediatric multipliers: EFSA 2008 documented soy-based formula aluminum at approximately 1.1 mg/kg b.w./week vs milk-based at 0.3 to 0.9 vs breast milk at less than 0.07. App pediatric mode should query feeding modality and apply formula-specific exposure defaults.

Structured outputs:

- GI absorption (general): less than 1 percent.
- GI absorption (citrate-complexed): higher, up to several percent.
- Antacid daily aluminum delivery (typical user): 100 to 1000 mg/day.
- High-Al food categories: foods with aluminium-additives, processed cheese with aluminum emulsifiers, baked goods with aluminum baking powder, tea, soy-based infant formula.
- Cookware contribution flag: non-anodized aluminum + acidic food + prolonged cooking = elevated leaching.
- Renal function multiplier: in CKD, body-burden accumulation rate is amplified; this is a clinical population not generally targeted by the consumer app.

12 | Open questions

Two load-bearing open questions for aluminum:

First, the population-level TWI exceedance documented by EFSA 2008 is a regulatory finding that has not produced a clear policy intervention. Reducing dietary aluminum below the TWI through ordinary food choice is difficult given aluminum's broad use as a food additive; whether the TWI is too conservative, or whether population aluminum exposure warrants additive-restriction policy, is an active regulatory debate.

Second, the relationship between dietary aluminum exposure and Alzheimer's disease has been investigated extensively over decades without a definitive consensus emerging. Current dominant view is that aluminum is not a primary causal factor in Alzheimer's, but the literature remains contested in some quarters. The wiki tracks this as a live methodological question without taking a position pending stronger meta-analytic synthesis.

13 | Frequently asked questions

Where does most of the aluminum in my diet actually come from?

For most consumers, aluminum exposure is dominated by aluminum-containing food additives such as sodium aluminium phosphate (a leavening agent in baked goods), sodium aluminium silicate (an anti-caking agent in salt and powdered foods), and aluminium-containing colorants. Aluminum-containing antacid medications and aluminum-cookware contact with acidic foods like tomato sauce and citrus also contribute, while naturally occurring aluminum in plants is generally a smaller source. EFSA 2008 concluded that the tolerable weekly intake is exceeded in significant fractions of the European population, particularly among people eating foods with aluminium-based additives.

Is aluminum in food dangerous, and does it cause Alzheimer's disease?

The clearest documented harms from aluminum (dialysis encephalopathy and dialysis osteomalacia) occurred in kidney-failure patients exposed through contaminated dialysate fluid that bypassed the gut, not through normal diet, and these are essentially eliminated in modern dialysis practice (ATSDR 2008). For people with normal kidney function eating a typical diet, the dose-response evidence for aluminum-related disease is weaker than for cadmium, lead, mercury, or arsenic. On Alzheimer's specifically, the page notes decades of investigation have not produced consensus, with the current dominant view being that aluminum is not a primary causal factor.

What is the regulatory safety limit for aluminum, and is it being exceeded?

EFSA set a tolerable weekly intake (TWI) of 1 mg Al/kg body weight per week, which replaced an earlier JECFA provisional value of 7 mg/kg per week; JECFA later aligned to 1 mg/kg per week (EFSA TWI). For a 70-kilogram adult this corresponds to about 70 mg per week or 10 mg per day. EFSA 2008 estimated typical European adult intake at 0.2 to 1.5 mg/kg per week, placing the population mean at the upper end of the TWI and concluding the limit is likely exceeded in a significant part of the population.

Why are antacid users singled out as the most exposed group?

Aluminum-containing antacids deliver roughly 100 to 1000 mg of aluminum per daily dose, which dwarfs typical dietary intake of about 5 to 15 mg per day (ATSDR 2008). Adding these antacids to a normal diet can rapidly push intake to 10 to 100 times the TWI. Switching to non-aluminum alternatives such as calcium carbonate, magnesium

hydroxide, or proton pump inhibitors eliminates this exposure, and people with impaired kidney function should discuss antacid choice with a physician.

Does cooking in aluminum pots add aluminum to my food?

Yes, aluminum cookware does contribute to dietary aluminum, particularly when cooking acidic foods such as tomato sauce, citrus dishes, and vinegar-based preparations for prolonged periods in non-anodized aluminum. Anodized aluminum cookware substantially reduces this leaching, and stainless steel, cast iron, glass, and ceramic eliminate it. The page notes the cookware contribution is generally modest relative to additive and antacid sources for the average consumer.

Should I worry about aluminum in infant formula?

Aluminum content varies widely by product; EFSA 2008 estimated soy-based formula at about 1.1 mg/kg body weight per week versus 0.3 to 0.9 for milk-based formula and less than 0.07 for breast-fed infants, making breastfeeding the lowest-aluminum option and milk-based formula lower than soy-based. Redgrove et al. 2019 measured UK prescription formulas spanning roughly 41 to 1956 µg/L, a nearly 50-fold range, showing that low aluminum concentrations are achievable and contamination is not inevitable. Specialized formulas for medical conditions should still be chosen on clinical grounds rather than aluminum content for non-renal-impaired infants.

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REVIEW

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