

Aluminum (Al) — HMTc analyte

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THE ANALYTE

13 · Al · Total elemental aluminum

Aluminium is limited because chronic dietary intake accumulates against a low tolerable weekly intake derived from animal neurotoxic, reproductive, and developmental endpoints — corroborated in humans by dialysis encephalopathy and osteomalacia — with renally impaired people, frequent antacid users, and formula-fed infants the most exposed; the resulting body burden is controllable through water treatment and process controls.

Why it is limited

The government and expert-body assessments that underpin limiting aluminum in food, and what each concluded. The maximum levels in force apply these findings; this page is the rationale, not the certification threshold.

BODY	YEAR	ASSESSMENT	WHAT IT CONCLUDED
EFSA	2008	Scientific Opinion, Safety of Aluminium from Dietary Intake (EFSA AFC Panel), The EFSA Journal 2008;754:1-34	Established a tolerable weekly intake (TWI) of 1 mg Al/kg b.w./week, anchored on neurotoxic, reproductive, and developmental effects in animal studies. The Panel derived no single critical study and reference point; the TWI integrates evidence across multiple animal endpoints with safety factors for interspecies extrapolation and human variability. EFSA concluded the TWI is likely to be exceeded in a significant part of the European population (population mean dietary intake 0.2 to 1.5 mg/kg b.w./week). EFSA's TWI is the assessment of a separate body from JECFA and is a distinct value from the JECFA PTWI; it remains current and is the more conservative of the two, sitting below JECFA's current PTWI of 2 mg Al/kg b.w./week (74th meeting, 2011). The two bodies genuinely differ.
JECFA (Joint FAO/WHO Expert Committee on Food Additives)	2011	Evaluation of certain food additives — 74th JECFA meeting (2011); WHO Technical Report Series 966 / WHO Food Additives Series 65	JECFA established a group provisional tolerable weekly intake (PTWI) for aluminium of 2 mg Al/kg b.w./week at its 74th meeting (2011), derived from a no-observed-adverse-effect level (NOAEL) of 30 mg/kg b.w./day in animal studies with a 100-fold safety factor. This 2 mg/kg b.w./week value is the current JECFA PTWI; it superseded the provisional 1 mg/kg b.w./week set at the 67th meeting (2006), which had itself replaced the 7 mg/kg b.w./week value in force since the 33rd meeting (1988). JECFA's current PTWI (2 mg/kg) is a distinct value from EFSA's TWI (1 mg/kg, 2008); the two bodies genuinely differ, with JECFA the less conservative. Like the EFSA TWI, the PTWI governs cumulative weekly intake rather than the concentration in any single food.

BODY	YEAR	ASSESSMENT	WHAT IT CONCLUDED
US ATSDR	2008	Toxicological Profile for Aluminum (U.S. Dept. of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry)	Set a chronic oral minimal risk level (MRL) of 1 mg Al/kg/day and an intermediate oral MRL of 1 mg Al/kg/day. Documented dialysis encephalopathy and dialysis osteomalacia as the clearest human aluminium disease entities; established that GI absorption is low (0.07 to 0.39 percent for drinking water, approximately 0.1 percent for diet, less than 1 percent overall), that excretion is primarily renal, and that renal failure is the major risk factor for aluminium body-burden accumulation.
Clinical literature (documented via ATSDR 2008)	1970s-1980s	Dialysis encephalopathy and dialysis osteomalacia case series, 1970s-1980s hemodialysis cohorts	Dialysis encephalopathy (progressive dementia with dysarthria, myoclonus, seizures) and dialysis osteomalacia (bone pain, fractures, characteristic radiographic and histological findings) were traced to aluminium-contaminated dialysate water entering the circulation directly via the dialyzer, bypassing the gastrointestinal barrier. These two entities are the strongest human evidence for aluminium's neurotoxicity and bone effects and the empirical anchor for regulatory neurotoxicity concern; new cases were essentially eliminated once dialysate water was treated (reverse osmosis) to remove aluminium. This demonstrates that renal function is the critical variable controlling body burden and that routes bypassing GI absorption produce disease at body burdens oral dietary exposure does not reach.

Toxicological basis

Why food-safety authorities regulate dietary aluminium, and why the anchor is the nervous system and bone rather than cancer.

The endpoint that anchors regulation is chronic, cumulative, and neurological. The EFSA AFC Panel (2008) set a tolerable weekly intake of 1 mg Al/kg body weight, derived not from one pivotal study but by integrating neurotoxic, reproductive, and developmental effects seen across multiple animal studies, with safety factors for interspecies extrapolation and human variability. Aluminium is poorly absorbed from the gut (typically under 1 percent, roughly 0.1 percent from a mixed diet) and is cleared renally, so body burden is governed far more by kidney function than by any single meal. The limit nonetheless bites because exposure is cumulative: aluminium enters the food supply through additives and processing at a scale that, EFSA concluded, causes the tolerable weekly intake to be exceeded in a significant part of the population. It is a weekly-intake problem, not a single-dose one.

The metal reaches three populations that the reference values are built to protect. People with impaired renal function accumulate aluminium because reduced excretion is the rate-limiting step on body burden. Frequent users of aluminium-containing antacids take in far more than diet delivers, with typical antacid aluminium of 100 to 1000 mg per day dwarfing dietary intake of roughly 5 to 15 mg per day. And formula-fed infants are exposed above the breastfed baseline, soy-based formula more than milk-based. The human proof-of-concept sits in this group: dialysis encephalopathy, presenting as progressive dementia with dysarthria, myoclonus, and seizures, and dialysis osteomalacia, presenting as bone pain and fractures, appeared in 1970s and 1980s hemodialysis patients exposed to aluminium-contaminated dialysate and resolved once dialysate water was treated. That episode is the empirical anchor that aluminium can produce neurological and bone disease at sufficient body burden.

Route precision is the honest core of the aluminium story. The strongest human disease arose through a parenteral route, contaminated dialysate entering the circulation directly, in patients who could not excrete the metal, not through ordinary oral diet in people with normal kidneys. Those routes reach body burdens that oral dietary exposure does not achieve at any reasonable intake. That distinction bounds the claim: for the general oral-exposure population the dose-response evidence for aluminium disease is weaker than for [lead](#), [cadmium](#), [mercury](#), or [arsenic](#), and the tolerable weekly intake rests largely on animal extrapolation. Aluminium is not regulated as a dietary carcinogen; the decades-long investigation of a link to Alzheimer's disease has not produced consensus, and the dominant view is that aluminium is not a primary causal factor. The defensible position is a real but bounded neuro-and-bone signal, anchored by the dialysis evidence, with the food-safety concern resting on cumulative intake rather than on any single toxic dose.

The agreement across bodies is what makes the case durable. EFSA's tolerable weekly intake of 1 mg/kg (2008), JECFA's provisional tolerable weekly intake of 2 mg/kg (74th meeting, 2011, WHO TRS 966), and the US ATSDR chronic oral minimal risk level of 1 mg/kg/day are three independent derivations from the same evidence base of animal endpoints plus the dialysis disease record. They genuinely differ, with EFSA the more conservative, which is itself a signal of independent evaluation rather than one body copying another. Aluminium is also unusual in that almost no government sets a per-food maximum concentration; control is exercised through these intake-based reference values, with Australia and New Zealand's [FSANZ](#) infant-formula limit the lone food maximum level in force. The science came first and the intake limits followed; where per-food limits do not exist, the gap is stated plainly rather than papered over, which is exactly the posture a certification standard has to be able to defend.

Occurrence & exposure

How much aluminium food actually carries, and how close ordinary intake runs to the tolerable weekly limit.

Reported total aluminium in Brazilian commercial infant foods (de Paiva et al. 2020; total Al by ICP-OES on as-sold product, µg/kg). Values are measured occurrence in a category survey, not certification thresholds.

FOOD GROUP (BRAZILIAN INFANT-FOOD SURVEY)	TOTAL AL (MG/KG)	NOTE
Petit-suisse (fresh cheese)	4170	highest single category measured
Soy-based infant drink	2860	soy protein base
Mixed-vegetable salty puree	2310-2760	high-fibre plant matrix
Chocolate milk drink	2175	cocoa and dairy matrix
Fruit puree	1900-2500	bioaccessible fraction as low as 0.5%

Estimated dietary aluminium intake versus the EFSA tolerable weekly intake of 1 mg Al/kg bw/week (EFSA AFC Panel 2008). Figures are EFSA range and point estimates, not measured percentiles, and describe intake rather than any per-food limit.

POPULATION	TYPICAL INTAKE (MG/KG BW/WK)	HIGH INTAKE (MG/KG BW/WK)	RELATIVE TO EFSA TWI (1 MG/KG BW/WK)
Adult (European diet)	0.2	1.5	high consumers ~150% of TWI
Infant, milk-based formula	0.3	0.9	up to ~90% of TWI
Infant, soy-based formula	1.1	—	~110%, exceeds TWI
Infant, breast-fed	<0.07	—	under ~7% of TWI

Aluminium in food is dominated by deliberate additives and by processing, not by the raw commodity. Aluminium-based leavening agents, anti-caking agents, and colorants are the largest food-borne contributors, supplemented by aluminium leached from cookware and packaging during prolonged contact with acidic foods and by aluminium carried in specific ingredients such as whey-protein hydrolysates and fruit components used in infant products. Concentrations therefore track formulation and manufacturing practice far more than geography, and within a single product category the cleanest and most contaminated items can differ by more than an order of magnitude.

Two features make aluminium worth monitoring even though a single serving is rarely acutely hazardous. First, the toxicological concern is cumulative: intake accrues against a low tolerable weekly value that expert bodies already judge to be exceeded across a meaningful share of the population, so category-level occurrence, not any one lot, is the operative signal. Second, total aluminium overstates the absorbed dose to a degree that varies enormously with the food matrix, because plant fibre and dairy protein bind aluminium and lower its bioaccessibility while low-fibre and high-polyphenol matrices raise it; the populations that combine high occurrence with high intake and low excretion capacity, formula-fed infants and people with impaired renal function, are the ones the reference values are set to protect.

Occurrence anchored on de Paiva et al. 2020 (Journal of Food Composition and Analysis, A-tier), a 95-sample survey of Brazilian commercial infant foods; intake-versus-TWI figures from the EFSA AFC Panel 2008 Scientific Opinion (A-tier). Both are routed to the Index aluminium page (/metals/aluminum). The full occurrence corpus is on the [Heavy Metal Index](#).

How it is regulated

Aluminium is unusual among the HMTc analytes in that almost no government sets a per-food concentration maximum level: the derivation tables on the cert standard pages (see [/standards/teething-and-snacks](#) and [/standards/infant-formula-powder](#)) show blank Al cells for the EU ([/regulations/eu-2023-915](#)), UK ([/regulations/uk-1881-2006](#)), Codex ([/regulations/codex-cxs-193](#)), Canada, China ([/regulations/china-gb-2762](#)), India ([/regulations/india-fssai](#)), Brazil, and the US FDA ([/regulations/us-fda-c2z](#)). The only government aluminium food ML in any jurisdiction is Australia/New Zealand FSANZ Food Standards Code Schedule 19 ([/regulations/au-nz-fsanx](#)): 500 µg/kg for infant formula, follow-on formula, and special-medical-purpose products for infants other than preterm products, and 1000 µg/kg for soy-based infant formula, both on a portion-ordinarily-consumed basis. Everywhere else, control is exercised through intake-based reference values rather than a food ML — EFSA's TWI (1 mg Al/kg b.w./week, 2008), JECFA's current PTWI (2 mg Al/kg b.w./week, 74th JECFA

meeting 2011), and ATSDR's oral MRL (1 mg Al/kg/day) — which govern total weekly intake, not the concentration in a single food. EFSA and JECFA are separate bodies and their reference values genuinely differ (EFSA 1, JECFA 2). Because no government regulates most product-analyte cells for aluminium, HMTc governs by read-across from the FSANZ value; see the /regulations/ pages for the maximum-level derivations and the Index page (/metals/aluminum) for the intake-based reference values.

How the HMTc program treats it

Aluminium (certification species: total elemental aluminium; no speciation trigger, unlike As→iAs, Hg→MeHg, or Cr→Cr-VI) is a claim analyte on the eight-total panel, equal at the gate with every other published limit. Because only FSANZ Schedule 19 sets any government aluminium ML, HMTc limits are set by disclosed read-across from that value: the teething-and-snacks standard applies 500 µg/kg at face value on an as-sold basis (the 8:1 reconstitution factor is formula-specific and is not applied to a non-formula product), while the infant-formula-powder standard converts the FSANZ portion-consumed values at the 8:1 powder-to-liquid ratio to 4000 µg/kg (non-soy) and 8000 µg/kg (soy-based). The four-status lifecycle, the regulatory hard-stop, basis conversion, and lot testing operate downstream of the limit regardless of how the number is set; a product is certified only at or below its published limit, and a product above it enters Remediation unmarked.

Literature baseline

The complete peer-reviewed and regulatory literature for aluminum is maintained independently at the [Heavy Metal Index](#)^[7]. That reference reports what the literature says; this certification site applies it. The two are kept editorially separate by design.