

Cadmium

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Abstract

This page is the first cross-source synthesis on cadmium for Heavy Metal Index. It draws on the canonical Handbook on the Toxicology of Metals chapter 32 (Nordberg, Nogawa, Nordberg 2015, Nordberg 2015), the Casarett & Doull's Essentials of Toxicology chapter 23 (Ufelle and Barchowsky 2021, Ufelle & Barchowsky 2021), Patty's Toxicology chapter 7 (Jakubowski 2012, Jakubowski 2012), the EFSA 2009 scientific opinion (EFSA Cd 2009), the paired California OEHHA Prop 65 documents (OEHHA 1996, OEHHA 2001), the JECFA 91st meeting 2022 dietary exposure assessment (JECFA 91st 2022), the ATSDR 2012 Toxicological Profile (ATSDR 2012), and the Codex CCCF17 session report (Codex CCCF17 2024). Codex CXS 193-1995 (the General Standard for Contaminants and Toxins in Food and Feed, matrix-level Cd MLs) and Codex CXC 81-2022 (Code of Practice for Cadmium in Cocoa Beans, the load-bearing source for cocoa-cadmium mitigation specifics) are also ingested. Operative 1985 EPA IRIS record, JECFA 73rd meeting derivation documents, and the EFSA 2011 reaffirming statement remain flagged as pending for later ingest waves. Open questions are tracked on synthesis.

1 | Overview

Cadmium is a non-essential heavy metal and an environmental contaminant of the human food system. It enters the food supply through three pathways that compound over time: natural occurrence in soils at background concentrations of 0.1 to 1 mg/kg, accumulating anthropogenic loading from phosphate-fertilizer use and atmospheric deposition from non-ferrous metal refining, and biological concentration in filter-feeding marine invertebrates and in certain plant commodities (cereals, leafy vegetables, root vegetables, cocoa, tobacco) (Nordberg 2015, Jakubowski 2012). For non-smoking members of the general population, food is the dominant source of cadmium exposure; for smokers, tobacco contributes an internal exposure comparable in magnitude to diet (Nordberg 2015).

The metal is absorbed at low rates from the gastrointestinal tract (5 to 10 percent on average, with a 1 to 10 percent range that widens upward under iron deficiency and in pregnancy) and at substantially higher rates

from the lung (10 to 50 percent of inhaled cadmium), and is then efficiently retained in the kidney and liver with a biological half-life measured in decades (Nordberg 2015, Jakubowski 2012). Cadmium body burden therefore accumulates across the human lifespan; the renal cortex concentration reaches its peak near age 55 under conditions of constant lifetime intake (ATSDR 2012). The primary toxic endpoint is renal tubular dysfunction, with skeletal demineralization a secondary endpoint that combines direct bone effects with calcium-handling consequences of the renal damage (Nordberg 2015, EFSA 2009). Cadmium is classified by the International Agency for Research on Cancer as a Group 1 human carcinogen on the basis of occupational inhalation studies, and dietary-exposure general-population epidemiology has produced statistical associations with cancers of the lung, endometrium, bladder, and breast (EFSA 2009, Nordberg 2015). No effective clinical treatment for cadmium intoxication exists; population-level dietary exposure reduction is the appropriate public-health response (Ufelle and Barchowsky 2021).

Four major regulatory and scientific bodies have issued dietary cadmium reference values. In order from tightest to most permissive when expressed on a daily per-body-weight basis: the US Agency for Toxic Substances and Disease Registry 2012 chronic oral Minimal Risk Level of 0.1 µg Cd/kg/day; the European Food Safety Authority 2009 tolerable weekly intake of 2.5 µg/kg b.w./week (daily equivalent approximately 0.36); the Joint FAO/WHO Expert Committee on Food Additives 2010 provisional tolerable monthly intake of 25 µg/kg b.w./month (daily equivalent approximately 0.83); and the US EPA Integrated Risk Information System oral reference dose, operative from 1985 and understood from secondary citations as approximately 1×10^{-3} mg/kg/day for food (pending ingest of the primary 1985 IRIS record). All four values rest on overlapping primary literature but arrive at different numbers through different methodological choices. The factor of approximately 8 between the tightest and most permissive of the four is a live fact that any threshold-setting exercise must acknowledge rather than paper over; averaging across the four reference values without naming which one is being calibrated to obscures a methodological divergence the literature itself has not resolved.

2 | At a glance

The three facts that matter most for a consumer trying to interpret cadmium in the food supply.

First, cadmium accumulates in the body over decades and is never meaningfully excreted; the kidney half-life is estimated at 20 years or more, and kidney-cadmium burden at middle age reflects dietary intake across essentially the whole of adult life, not recent consumption (Nordberg 2015, Jakubowski 2012). There is no clinical treatment that removes cadmium once it is accumulated (Ufelle and Barchowsky 2021). This is the medical basis for treating cadmium as a prevention problem rather than a treatment problem.

Second, the foods that contribute the most to population-level cadmium exposure do so because they are eaten in large volume, not because they are the most cadmium-contaminated foods. The population-level contributors are cereals (rice and wheat), potatoes and other starchy roots, leafy vegetables, nuts, oilseeds, pulses, and meat (EFSA 2009, JECFA 91st). The high-concentration foods, which matter most to consumers who eat them regularly, are bivalve molluscs (other than oysters), wild mushrooms, cocoa and chocolate products, seaweed, fungi, oilseeds, and edible organ meats (kidney and liver) (EFSA 2009). Regular consumers of bivalve molluscs show mean cadmium exposures above the EFSA tolerable weekly intake at 4.6 µg/kg body weight per week; regular consumers of wild mushrooms reach 4.3 µg/kg body weight per week (EFSA 2009).

Third, three groups are at materially higher risk than the general population and should pay more attention to cadmium than reference-value-for-adults summaries suggest. Children in the 0.5 to 12 year range have mean dietary cadmium exposure approximately 60 percent higher than adults because they eat more food relative to body weight (EFSA 2009); high-percentile child exposures in Australia and the United States reach 82 to 88

percent of the JECFA provisional tolerable monthly intake, and when cocoa-product contributions are added, the total for children aged 0.5 to 12 can reach 96 percent of that reference value (JECFA 91st). Women of reproductive age with low iron stores absorb cadmium more efficiently across multiple pregnancies (Nordberg 2015, Ufelle and Barchowsky 2021). Smokers receive an internal cadmium exposure from tobacco comparable in magnitude to their dietary exposure, which roughly doubles their body burden (Nordberg 2015).

3 | Toxicology

The primary target organ of cadmium toxicity is the kidney, specifically the proximal tubular cells of the renal cortex, where cadmium accumulates bound to metallothionein and gradually induces tubular dysfunction (Nordberg 2015, EFSA 2009). The kidney cortex concentration is approximately 1.25 times the whole-kidney concentration, reflecting the renal cortex's role as the site of active reabsorption and therefore the tissue to which circulating cadmium-metallothionein complexes are preferentially delivered through glomerular filtration and tubular uptake (Nordberg 2015). The earliest detectable biological response is an increase in the urinary excretion of low molecular weight proteins, particularly beta-2-microglobulin, retinol binding protein, and protein HC, reflecting the loss of tubular reabsorption capacity (EFSA 2009, Nordberg 2015). Prolonged or high exposure progresses from tubular damage to decreased glomerular filtration rate and eventually to renal failure; in itai-itai disease patients, mean creatinine clearance of 42 mL/min and tubular reabsorption of phosphate of 51 percent have been observed alongside characteristic histopathology (tubular atrophy and dilatation with marked epithelial degeneration, in the absence of glomerular abnormalities at the light-microscopic level) (Nordberg 2015).

Skeletal toxicity is the second canonical endpoint. Cadmium causes bone demineralization through two mechanisms that operate in parallel: a direct effect on bone cells and mineralization, likely related to interference with calcium metabolism and with the enzyme lysyl oxidase (and therefore with bone collagen cross-linking), and an indirect effect secondary to renal dysfunction, through altered calcium and phosphate handling by the damaged kidney (Nordberg 2015). The bone effects manifest clinically as osteomalacia, osteoporosis, and increased fracture risk, with the heaviest burden falling on postmenopausal women and on populations with inadequate calcium, vitamin D, iron, zinc, or protein intake (Nordberg 2015). The combination of renal and skeletal damage in cadmium-polluted populations is what produces itai-itai disease in its severe form (see the itai-itai section below). At lower exposures, epidemiological studies have found associations between decreased bone mineral density, increased fracture risk, and urinary cadmium levels around 1 µg/g creatinine and higher (Nordberg 2015).

Cardiovascular effects are described differently by the two most current textbook sources. The Handbook on the Toxicology of Metals chapter 32 (Nordberg, Nogawa, Nordberg 2015) treats cardiovascular endpoints as contested in the literature: some human biomonitoring studies report associations between cadmium exposure and blood pressure, atherosclerosis, peripheral arterial disease, and cardiovascular mortality, while other studies do not reproduce these associations, with the chapter concluding that the evidence continues to be variable and that residual confounding, particularly by tobacco smoke, cannot be excluded in much of the positive literature. The Casarett & Doull's Essentials of Toxicology chapter 23 (Ufelle and Barchowsky 2021), published six years later, uses notably less hedged language: it describes a "strong association" between cadmium exposure and peripheral vascular disease, and raises the possibility that cadmium partially mediates the negative effect of smoking on peripheral artery disease. Both positions are recorded here without resolution; the first synthesis pass has not completed a search of the 2015-to-present cardiovascular literature that would support reconciling them. See synthesis for tracking.

Cadmium does not interact directly with DNA. Its genotoxicity operates through induction of oxidative stress and inhibition of DNA repair mechanisms (Nordberg 2015). Cadmium causes lung cancer in rats after inhalation exposure (Nordberg 2015). In humans, IARC's Group 1 classification rests on occupational inhalation studies; dietary cadmium's carcinogenic potential at general-population exposure levels remains an area where the dose-response evidence is suggestive but not yet quantitative, with general-population epidemiology producing statistical associations for cancers of the lung, endometrium, bladder, and breast that the major regulatory bodies have not yet translated into quantitative dose-response reference values (EFSA 2009, Nordberg 2015).

Developmental and male reproductive toxicity are separate endpoints for which cadmium has been judged a sufficient hazard to warrant listing under California's Proposition 65 (OEHHA 1996). The 1996 OEHHA hazard identification document compiled the human and animal evidence: reduced birthweight and pre-term labor correlated with maternal blood or infant hair cadmium in human epidemiology (with the acknowledgment that confounding by lead and tobacco smoking makes isolated cadmium effects difficult to establish), decreased pup birthweight and altered postnatal development in rodents exposed in utero via oral or inhalation routes, and effects on sperm counts, testes weight, and testicular histopathology in male experimental animals (OEHHA 1996). The California DART Identification Committee's determination on the basis of this evidence produced the Prop 65 reproductive-toxicity listing effective May 1997; the 2001 MADL of 4.1 µg/day oral implements that listing (OEHHA 2001). The developmental endpoint (Ali et al. 1986 LOEL of 0.706 mg/kg/day in rats) was controlling because it was more sensitive than the male reproductive endpoint (Laskey et al. 1980 NOEL 1 ppm) (OEHHA 2001). Casarett & Doull's Essentials 2021 adds the observation that pregnant women appear to accumulate more cadmium than nonpregnant women, a population-level exposure-biology fact that reinforces the reproductive-toxicity endpoint's weight (Ufelle and Barchowsky 2021).

Central and peripheral nervous system effects are plausible but less well characterized. Animal data suggest that cadmium can alter brain development when exposure occurs before the blood-brain barrier is fully established, likely through interference with zinc metabolism and with metallothionein function in neural tissue (metallothionein-3 is a brain-expressed form rich in zinc and relevant to neurodegenerative disease; cadmium can displace zinc from metallothionein-1 and metallothionein-2 but does not induce metallothionein-3) (Nordberg 2015).

Clinical treatment

There is no effective clinical treatment for cadmium intoxication (Ufelle and Barchowsky 2021). Chelation therapy for cadmium generally produces significant adverse effects and is not an established standard of care. This is a durable clinical statement and it grounds the appropriate public-health response: population-level dietary exposure reduction is the intervention available to regulators, certification programs, and consumers, not pharmacological treatment after exposure.

4 | Typical exposure routes

Dietary intake is the dominant route for the non-smoking general population (Nordberg 2015). Tobacco smoking contributes an internal exposure comparable in magnitude to dietary intake; uptake from smoking 20 cigarettes per day is approximately 2 µg of cadmium, and direct measurement in tissue shows that body burden in smokers is roughly double that of non-smokers (Nordberg 2015). House dust can be an important source of exposure for children (Nordberg 2015). Inhalation exposure in the non-smoking general population contributes only marginally to total body burden (Nordberg 2015). Dermal absorption of cadmium compounds is negligible (Jakubowski 2012).

Gastrointestinal absorption of ingested cadmium is 5 to 10 percent in humans on average, varying across a 1 to 10 percent range depending on individual factors, with low body iron stores common in women of reproductive age as the single largest upward variability factor; multiple pregnancies, preexisting health conditions, and nutritional deficiencies in zinc or calcium also raise absorption (Nordberg 2015). Pulmonary absorption of inhaled cadmium is higher, in the range of 10 to 50 percent (Nordberg 2015) or 10 to 60 percent (Ufelle and Barchowsky 2021), depending on particle size and solubility. These individual-level and route-level variability factors are incorporated into the EFSA TWI derivation through the toxicokinetic model, which is why the Panel applied no additional uncertainty factor for individual susceptibility; they are accounted for in ATSDR's MRL derivation through the empirical environmental-exposure study base (EFSA 2009, ATSDR 2012).

Biological half-life and body burden accumulation

The biological half-life of cadmium in humans is long and has been estimated through multiple approaches. One-compartment models fit to empirical data on whole-body and renal cortex accumulation produce estimates of at least 20 years (Kjellström 1971, Tsuchiya and Sugita 1971, WHO/IPCS 1992), and this is the value most regulatory documents cite. Biopsy-based work (Akerstrom et al. 2013) has found that the half-life in the kidney depends on the cadmium burden itself: approximately 21 years at lower renal cortex concentrations (around 8 mg/kg) and approximately 43 years at higher concentrations (around 23 mg/kg). In urinary cadmium measurements from a Japanese cadmium-polluted region, half-lives of approximately 14 years and 24 years have been estimated for men and women respectively (Suwasono et al. 2009). Jakubowski 2012 gives the summary range as 10 to 30 years, consistent with the above.

Eight-compartment physiologically based toxicokinetic models (Kjellström and Nordberg 1978, Nordberg and Kjellström 1979) are used by ATSDR and by Thun et al. 1991 for quantitative risk assessment, with age-dependent amendments (Choudhury et al. 2001) that ATSDR explicitly adopted in the 2012 Toxicological Profile. These models take transfers between muscle, liver, and kidney into account, with short component half-lives (8 to 14 years for extra-renal tissues) and the dominant long-half-life component in the kidney. The practical consequence for wiki content is that cadmium body burden in a 50-year-old reflects dietary exposure over essentially the whole of adult life, not recent intake; population-level interventions to reduce exposure operate on timescales of decades, not months.

The renal cortex concentration typically reaches a peak near age 55 under conditions of lifetime constant intake. This is the anchor age for the ATSDR chronic oral MRL derivation and for the EFSA TWI's kinetic back-translation, both of which calibrate their dietary-intake reference values against the kidney burden at this peak.

5 | Food sources

Major dietary contributors to population cadmium exposure, identified by EFSA's analysis of approximately 140,000 European occurrence samples collected 2003 to 2007 and consumption data from EFSA's Concise European Food Consumption database, are cereals and cereal products, vegetables, nuts and pulses, starchy roots and potatoes, and meat and meat products. These categories dominate because of high consumption volumes rather than because they carry the highest cadmium concentrations. The JECFA 91st meeting 2022 reanalysis using the GEMS/Food database (277,292 cleaned records covering 10 of 17 GEMS/Food cluster diets) confirms the same commodity list across the international regional diets, with rice, wheat, root vegetables, tuber vegetables, leafy vegetables, other vegetables, and molluscs collectively accounting for 40 to 85 percent of total mean dietary cadmium exposure depending on regional diet.

The foods with the highest cadmium concentrations, rather than the highest contribution to exposure, are seaweed, fish and seafood (particularly bivalve molluscs other than oysters), chocolate and cocoa, foods for special dietary uses, fungi, oilseeds, and edible offal (especially kidney and liver). These food categories produce substantial per-serving exposure even at moderate consumption levels; regular consumers of bivalve molluscs showed a mean cadmium exposure of 4.6 µg/kg b.w./week and regular consumers of wild mushrooms 4.3 µg/kg b.w./week, both well above the EFSA mean of 2.3.

The 91st meeting of JECFA, convened in November 2020 at the request of the Codex Committee on Contaminants in Foods, substantially updated the evidence base for cocoa and cocoa-derived products. Occurrence data submitted to the Committee in 2019 showed higher mean cadmium concentrations in cocoa products than had been recognized in the JECFA 77th meeting 2013 cocoa-specific assessment. The updated analysis estimated that cocoa-product contributions push total dietary cadmium exposure in children aged 0.5 to 12 years to as high as 96 percent of the JECFA provisional tolerable monthly intake, essentially saturating the international reference value from dietary sources alone. Cocoa powder specifically drove a high-percentile (P97.5) exposure of 12 µg/kg b.w./month in European children aged 7 to 11. Cocoa, cocoa powder, and chocolate are therefore priority ingredient-page targets when the ingredient wave begins.

Cadmium accumulates in plants through root uptake from soil. The use of phosphate fertilizers, which contain cadmium as a trace contaminant of the phosphate rock, has progressively elevated agricultural soil cadmium levels across much of Europe (Nordberg 2015, Jakubowski 2012). Local contamination from mining, smelting, and sewage-sludge application can produce substantially higher soil concentrations in hotspot areas, with corresponding elevation in locally produced food (Nordberg 2015).

For rice specifically, irrigation regime is an emerging cadmium-risk driver that interacts with the established arsenic story. Soil redox conditions controlled by water management drive opposite accumulation dynamics: flooded paddies suppress Cd and favor iAs; aerobic or alternate-wetting-and-drying (AWD) paddies suppress iAs and favor Cd. Two companion field studies from the University of Delaware RICE facility quantify the trade-off. Limmer and Seyfferth 2024 reports Cd ranges of approximately 10 to 150 µg/kg dw in polished grain across six irrigation treatments, with continuously flooded plots at the low end and aerobic management at the high end. Seyfferth et al. 2025 found that the driest paddy conditions exceeded the Codex Cd maximum level of 0.4 mg/kg in polished grain in both years of the study. As AWD adoption scales globally in response to climate adaptation pressure, the cadmium dimension of rice contamination may grow relative to the arsenic dimension that has historically defined regulatory attention. See climate-metals-tradeoffs for the full synthesis and rice (irrigation-management cadmium risk section).

Industrial cadmium production and use is itself a major upstream driver of environmental cadmium loading. Global cadmium production declined from approximately 22,000 metric tons per year at the early-2000s peak to approximately 16,000 metric tons per year by the 2012 period (Jakubowski 2012). The dominant uses are electrode material in nickel-cadmium batteries (77 percent), pigments (11 percent), protective plating on steel (8 percent), and alloys (4 percent). Ambient air cadmium concentrations reflect this industrial footprint: remote areas at 0.1 to 1 ng/m³, rural areas at 0.1 to 5, urban areas at 1 to 20, with higher concentrations in proximity to cadmium-emitting industrial sources.

What this means for food choice

The population-level dietary contributors to cadmium exposure are foods most people cannot and should not avoid. Rice, wheat, potatoes, leafy vegetables, nuts, and meat are nutritional staples, and the cadmium they deliver to an average adult diet is at or slightly above the European tolerable weekly intake of 2.5 µg/kg

body weight per week (EFSA 2009). The appropriate consumer response to this baseline is not avoidance of staples but attention to the high-concentration foods and high-exposure behaviors that push a person above the population average.

The foods and behaviors that materially raise an individual's cadmium exposure above the population average:

- Regular consumption of bivalve molluscs other than oysters (clams, mussels, scallops, cockles) at mean levels that place regular consumers at approximately twice the EFSA mean dietary exposure (EFSA 2009).
- Regular consumption of wild mushrooms, which can concentrate cadmium from their substrate at levels that place regular consumers similarly elevated (EFSA 2009).
- Regular consumption of organ meats (kidney and liver of cattle, pigs, horses), which are the tissues where cadmium accumulates most in food animals (EFSA 2009, Nordberg 2015).
- Cocoa powder and chocolate products consumed frequently, particularly by children in the 0.5 to 12 year range, for whom cocoa alone can drive a 97.5th-percentile exposure of 12 µg/kg body weight per month on top of dietary intake from other sources (JECFA 91st).
- Tobacco smoking, which contributes an internal cadmium exposure comparable in magnitude to dietary intake (approximately 2 µg of cadmium per 20 cigarettes), roughly doubling body burden in smokers (Nordberg 2015).
- Locally-produced food from areas with historic mining, smelting, or sewage-sludge-amended agriculture (Nordberg 2015).

The framing for consumer choice is not "safe versus dangerous" but "your total weekly exposure, summed across diet, environment, and smoking, relative to the reference values you find most credible." Consumers who would like to benchmark their own exposure to the tighter reference values (ATSDR chronic oral MRL of 0.1 µg/kg/day, which is approximately 0.7 µg/kg b.w. per week) should treat the population-level dietary staples as their baseline exposure and actively manage the high-concentration items above (ATSDR 2012).

6 | Regulatory limits

Reference values and maximum levels established to date, with more to populate as ingests complete:

JURISDICTION / BODY	TYPE	VALUE	PAGE
US ATSDR	Chronic oral MRL	0.1 µg Cd/kg/day	atsdr-cadmium-mrls
US ATSDR	Intermediate oral MRL	0.5 µg Cd/kg/day	atsdr-cadmium-mrls
US ATSDR	Chronic inhalation MRL	0.01 µg Cd/m³	atsdr-cadmium-mrls
US ATSDR	Acute inhalation MRL	0.03 µg Cd/m³	atsdr-cadmium-mrls
EFSA (EU)	TWI	2.5 µg/kg b.w./week	efsa-cadmium-twi

EU	Binding maximum levels in food commodities	Regulation-specific by matrix: infant formula 5-20 ug/kg depending format/protein source; baby food and processed cereal-based infant/young-child food 40 ug/kg; rice/quinoa/wheat bran/wheat gluten 150 ug/kg; cereals general 100 ug/kg; spinach/herbs 200 ug/kg; sunflower/linseed 500 ug/kg; cocoa powder 600 ug/kg; chocolate 100-800 ug/kg by cocoa solids; bivalves 1000 ug/kg; liver 500 ug/kg; kidney 1000 ug/kg	eu-2023-915-cadmium
JECFA (international)	PTMI	25 µg/kg b.w./month (≈ 5.83/week equivalent)	jecfa-cadmium-ptmi
US EPA	IRIS oral RfD (food)	1 × 10 ⁻³ mg/kg/day (1.0 µg/kg/day)	epa-iris-cadmium-rfd
US EPA	IRIS oral RfD (water)	5 × 10 ⁻⁴ mg/kg/day (0.5 µg/kg/day)	epa-iris-cadmium-rfd
US California	Prop 65 MADL (oral, reproductive toxicity)	4.1 µg/day	oehha-cadmium-prop65
Codex Alimentarius	ML for cadmium in quinoa (CCCF17 2024, CAC47 adoption)	0.15 mg/kg	codex-cadmium-mls
Codex Alimentarius	Matrix-level MLs (CXS 193-1995)	Polished rice 0.4 mg/kg; wheat 0.2; leafy vegetables 0.2; root and tuber 0.1; pulses 0.1; bivalve molluscs 2; cocoa powder 2.0; chocolate 0.3-0.9 by cocoa-solid percentage; full table in source page	Codex CXS 193-1995
US FDA	CTZ action levels	Pending ingest	fda-closer-to-zero (Pb rules ingested; Cd document pending)

The four dietary-reference values on the daily per-body-weight scale that are comparable to one another: ATSDR chronic oral MRL 0.1 µg/kg/day (tightest, anchored on a UCDL10 of 0.5 µg/g creatinine with a toxicokinetic translation to 0.33 µg/kg/day in females at age 55 and an uncertainty factor of 3 for human variability); EFSA TWI 2.5 µg/kg b.w./week (daily equivalent 0.36, anchored on a BMDL5 of 4 µg U-Cd/g creatinine with a chemical-specific adjustment factor of 3.9 and a kinetic back-translation); JECFA PTMI 25 µg/kg b.w./month (daily equivalent 0.83, established at the 73rd meeting in 2010 with a monthly averaging window reflecting

cadmium's long half-life); EPA IRIS oral RfD 1×10^{-3} mg/kg/day for food (1.0 µg/kg/day, from the 1989 IRIS record verified directly from the EPA IRIS database). The factor-of-10 range across the four values reflects different methodological choices on overlapping primary literature, not disagreements on underlying toxicology; the ordering tightest-to-most-permissive is ATSDR → EFSA → JECFA → EPA IRIS.

ATSDR itself notes (p. 43 of the 2012 profile) that its chronic oral MRL is below the estimated age-weighted US dietary cadmium intake of approximately 0.3 µg/kg/day, and that the UCDL10 point of departure is approximately twofold above the CDC 2011 US adult geometric mean urinary cadmium of 0.247 µg/g creatinine. The tightest regulatory reference value is below typical US exposure. This is a load-bearing fact for interpreting the reference values: at least one major regulatory body has set a chronic oral limit beneath the level at which the general US population is already exposed through diet.

What the reference values mean in practice

A consumer reading the table above and wondering which number to use as a benchmark for personal decision-making is asking the right question, and it does not have a single answer. Each of the four major dietary cadmium reference values is defensible within its own methodological frame, and a consumer should choose among them based on the level of precaution that matches their own situation.

The tightest reference, ATSDR's chronic oral MRL at 0.1 µg Cd per kilogram body weight per day, is the appropriate benchmark for a consumer who prioritizes margin of safety, who is a member of a vulnerable subpopulation (pregnancy, childhood, smokers, high-seafood or high-cocoa consumption), or who is accumulating cadmium body burden that will continue to compound over decades (ATSDR 2012). For a 70 kilogram adult, this reference corresponds to approximately 7 µg of cadmium per day. Typical US dietary cadmium intake is estimated at approximately 0.3 µg per kilogram body weight per day, or 21 µg per day for the same adult (ATSDR 2012). The ATSDR reference value is approximately one-third of typical US intake.

The EFSA tolerable weekly intake at 2.5 µg/kg body weight per week, which is the operative reference value across the European Union, corresponds to approximately 175 µg per week for the 70 kg adult, or approximately 25 µg per day. Typical European dietary cadmium intake sits close to or slightly above this reference value at the population mean (2.3 µg/kg b.w./week); the European Panel's risk characterization states that population-level cadmium exposure should be reduced (EFSA 2009).

The JECFA provisional tolerable monthly intake of 25 µg/kg b.w./month, which is the international reference value Codex standards are aligned to, corresponds to approximately 1,750 µg per month for the 70 kg adult, or approximately 58 µg per day (JECFA 91st). This value is the most permissive of the four daily-equivalent dietary references on the wiki.

A consumer's practical choice: if your dietary pattern is high in seafood (particularly bivalve molluscs), wild mushrooms, organ meats, or cocoa, or if you are pregnant or a frequent smoker, calibrate your attention to the ATSDR value and consider the EFSA value as a secondary reference. If your dietary pattern is European and typical of a Member State average, the EFSA TWI is the operative regulatory reference in your jurisdiction and the benchmark that informs national-level exposure-reduction policy. The JECFA value is more appropriate as a benchmark for regulators considering trade-standard harmonization than for individual dietary decisions.

7 | Testing

Analytical methods in use across the occurrence and biomonitoring literature span flame atomic absorption spectrometry (FAAS, detection limits 0.8 to 12.5 µg/L), inductively coupled plasma optical emission spectrometry

(ICP-OES, detection limits 0.1 to 1 µg/L), graphite furnace atomic absorption spectrometry (GFAAS, detection limits 0.002 to 0.02 µg/L), and inductively coupled plasma mass spectrometry (ICP-MS, detection limits 0.00001 to 0.001 µg/L). The four-order-of-magnitude range in detection limits across methods is material for what wiki claims about cadmium occurrence can actually be supported by individual data points: FAAS data at 20 µg/L and ICP-MS data at 0.1 µg/L both encode "non-detect" by different thresholds, and pooled datasets must be handled carefully to avoid false precision. FDA's Toxic Elements Program uses an ICP-MS method; the ATSDR 2012 profile uses an ICP-MS protocol for biomonitoring; the NIOSH Method 7048 for occupational air cadmium uses flame AAS at 228.8 nm with deuterium background correction, producing a working range of 0.1 to 2 mg/m³ at 25 L air-sample volume.

Biomonitoring for exposure assessment typically measures cadmium in whole blood (reflecting recent exposure with a few-month memory) or urine (reflecting body burden and cumulative exposure over years, normalized to urinary creatinine). The EFSA TWI derivation, the ATSDR chronic oral MRL derivation, and the JECFA PTMI all use urinary cadmium normalized to creatinine as the exposure metric. Reference intervals and clinical action levels for urinary β₂-microglobulin as a tubular-dysfunction biomarker use an upper limit of 300 µg/g creatinine (Jakubowski 2012). See Jakubowski 2012 for the occupational sampling-procedure detail (NIOSH Method 7048, urine sample handling requirements, matrix-modifier protocols for GFAAS urinary cadmium).

A dedicated wiki/testing/ section with detailed method-specific pages will be stood up in a later wave.

8 | Microbiome effects

_Pending. The metal-microbiome literature for cadmium will enter through dedicated microbiome ingests and the Handbook chapter; the wikibiome_crosswalk anchors are tentatively cadmium-gut-axis and cadmium-dysbiosis._

9 | Historical context: itai-itai disease

Itai-itai disease is the most severe clinical presentation of chronic dietary cadmium poisoning documented in the human record, and the historical event that established cadmium's renal and skeletal toxicity as a paired public-health concern rather than two separate endpoints (Nordberg 2015). It occurred in the Jinzu River basin of Toyama Prefecture, Japan, from the 1950s onward, in a population exposed to cadmium primarily through the consumption of rice and other produce grown in paddies and fields irrigated with water contaminated by effluent from a zinc mine upstream (Nordberg 2015). The clinical picture combines renal injury (both tubular and glomerular dysfunction, with urinary excretion of low molecular weight proteins elevated several-thousand-fold above normal, together with glucosuria and general aminoaciduria) and skeletal injury (osteomalacia and osteoporosis severe enough to produce spontaneous bone fractures, with the associated moderate-to-severe anemia and elevated serum alkaline phosphatase characteristic of active bone disease) (Nordberg 2015).

Through 2011, 196 cadmium-exposure cases had been officially recognized as itai-itai disease (3 men and 193 women), with an additional 255 subjects (46 men and 209 women) requiring observation for the condition (Nordberg 2015). The overwhelming sex-skew toward postmenopausal women reflects the combined vulnerability of calcium-depleted skeletons and iron-deficiency-enhanced cadmium absorption across multiple pregnancies and lactations, against a dietary pattern with limited zinc and calcium (Nordberg 2015). Renal tubular injury is highly prevalent across the broader Jinzu basin population beyond the diagnosed cases: urinary B2M concentrations exceeded 4 mg/L in 71 to 74 percent of residents aged 50 to 59 in the most heavily polluted district, and in 91 to 100 percent of residents 70 years of age or older (Nordberg 2015). At least 16 itai-itai-like cases have been reported from other cadmium-polluted regions outside the Jinzu basin (Nordberg 2015).

The disease is the historical anchor for every regulatory cadmium reference value derived since the 1970s. The Kjellström-Nordberg toxicokinetic model was developed in the same period in direct response to the Jinzu epidemic (Nordberg 2015). The EFSA, JECFA, ATSDR, and EPA IRIS derivations all operate against the Jinzu cohort and the subsequent environmental-exposure epidemiology that extended the observations to less-polluted populations (EFSA 2009, ATSDR 2012, JECFA 91st). The modern wiki content on cadmium's renal and skeletal endpoints descends, through those regulatory derivations, from this clinical cohort.

10 | Vulnerable populations

Populations for which cadmium exposure warrants particular attention, synthesized across the ingested sources:

POPULATION	BASIS
Children aged 0.5 to 12	Mean dietary exposure approximately 60 percent higher than adults due to greater food intake relative to body weight; UK toddlers aged 1.5 to 4.5 had exposure 165 percent higher than adults (FSA 2009 TDS); high-percentile cocoa-inclusive total dietary exposure reaches 96 percent of the JECFA PTMI (JECFA 91st meeting 2022)
Women of reproductive age with low iron stores	Gastrointestinal absorption of cadmium is substantially elevated with low iron status; pregnant women appear to accumulate more cadmium than nonpregnant women (Ufelle and Barchowsky 2021)
Postmenopausal women	Overwhelming sex-skew in itai-itai disease (193 of 196 recognized patients); skeletal vulnerability to cadmium-induced bone demineralization peaks after menopause
Vegetarians	Mean dietary exposure up to 5.4 µg/kg b.w./week, from high cereal, nut, oilseed, and pulse consumption (EFSA 2009)
Regular consumers of bivalve molluscs	Mean exposure 4.6 µg/kg b.w./week (EFSA 2009), approximately twofold above the EFSA TWI of 2.5
Regular consumers of wild mushrooms	Mean exposure 4.3 µg/kg b.w./week (EFSA 2009)
Frequent cocoa and chocolate consumers, particularly children	JECFA 91st meeting 2022 finding: cocoa powder alone drives a P97.5 exposure of 12 µg/kg b.w./month in European children aged 7 to 11; cocoa-inclusive total dietary exposure reaches 96 percent of PTMI for children 0.5 to 12

Residents of hotspot areas	Locally produced food near historic or active mining, smelting, or sewage-sludge-amended agriculture can carry substantially elevated cadmium
Smokers	Tobacco smoking produces an internal cadmium exposure comparable in magnitude to dietary intake; body burden in smokers is approximately twofold that of non-smokers
Occupationally exposed workers	Nickel-cadmium battery production, pigment manufacture, electroplating, zinc smelting, non-ferrous metal refining; current occupational air limits 2 to 5 mg/m ³ respirable fraction per Jakubowski 2012; chronic occupational inhalation exposure produces renal tubular dysfunction, obstructive lung disease, and increased lung cancer risk

If you are in one of these groups

The vulnerable-populations framing is not advice to avoid food; it is a framing to help a reader understand whether reference values derived for the general population apply to them or whether their own exposure profile calls for more attention. Three practical implications for consumers who recognize themselves on the list above.

For pregnant and potentially-pregnant women, two facts matter. First, iron deficiency (common in women of reproductive age) raises cadmium absorption from the gut, so correcting iron status through diet or supplementation is itself a cadmium exposure reduction. Second, the California OEHHA Maximum Allowable Daily Level of 4.1 µg per day oral is derived specifically from a developmental-toxicity endpoint and is therefore the most directly relevant regulatory reference for this population; products carrying a California Proposition 65 warning for cadmium are products that, at typical use, would exceed this level.

For parents of children under 12, frequent cocoa-product consumption is the single most consequential dietary variable under parental control. The JECFA 91st meeting 2022 assessment finds that cocoa powder and chocolate products can drive a high-percentile exposure that, combined with whole-diet cadmium, reaches 96 percent of the international tolerable monthly intake in this age group. Moderating cocoa-product frequency is a higher-leverage intervention than modifying any other single food category.

For people who regularly eat bivalve molluscs, wild mushrooms, or organ meats, the population-level mean exposure exceeds the EFSA tolerable weekly intake by approximately twofold, and the ATSDR chronic oral Minimal Risk Level by a larger factor. If these foods are cultural or nutritional staples rather than occasional choices, calibrating attention to the ATSDR reference value rather than to the more permissive EFSA or JECFA values is the more protective posture.

11 | App-layer integration

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

Population-level contamination-profile confidence is high for cereals (rice, wheat), potatoes and other starchy roots, leafy vegetables, nuts, oilseeds, pulses, meat, and offal, on the basis of the 140,000-sample EFSA occurrence dataset (2003 to 2007) and the 277,292-record JECFA GEMS/Food dataset (2011 onward). For these commodities, contamination-profile populated states with confidence: high and n_studies: many are appropriate once ingredient pages are stood up. Cocoa powder, cocoa products, and chocolate carry high-confidence occurrence data from the JECFA 91st meeting 2022 but require specific handling because the 2019 occurrence

data showed higher concentrations than the 77th meeting 2013 values; ingredient pages should cite the 2022 values.

For app exposure estimation, the 90th percentile consumption values from EFSA (adults) and the JECFA GEMS/Food cluster diets provide default per-commodity serving sizes, but pediatric multipliers are required: children aged 0.5 to 12 should use approximately 1.6x adult per-kg intake by default, with the toddler-specific multiplier approximately 2.65x (UK FSA 2009 finding that toddlers 1.5 to 4.5 years had exposure 165 percent higher than adults) (EFSA 2009).

For app risk-scoring against reference values, the four reference values in order of increasing daily-equivalent permissiveness are:

REFERENCE	DAILY EQUIVALENT (µg/KG B.W./DAY)
ATSDR chronic oral MRL	0.1
EFSA TWI daily-equivalent	approximately 0.36
JECFA PTMI daily-equivalent	approximately 0.83
EPA IRIS oral RfD (food, 1985, pending primary ingest)	approximately 1.0

The app should allow a user to choose which reference value they want to benchmark against, default to the ATSDR value for conservative estimation, and never silently average across reference values. For the vulnerable-population flag layer, flag users in pregnancy, childhood (0.5 to 12), frequent-smoker, and high--seafood/high-cocoa categories against the ATSDR value specifically, and render the OEHHA Prop 65 MADL of 4.1 µg/day as a product-labeling cross-reference for California-market products.

Structured outputs the app can consume directly, in addition to the reference-value table:

- Absorption fractions: GI 5 to 10 percent (general population, with 1 to 10 percent individual range), pulmonary 10 to 50 to 60 percent; use midpoint of 7.5 percent GI for default dose estimation, 10 percent for low-iron or pregnancy flags.
- Biological half-life default: 20 years (regulatory convention); use 21 to 43 year range for high-exposure percentiles when kidney-concentration-dependent kinetics matter.
- Peak renal cortex concentration age: 55 years; use this as the reference age for lifetime-cumulative exposure estimates.
- Smoking cadmium contribution: approximately 2 µg per 20 cigarettes absorbed internally; treat as additive to dietary intake for smoker users.

12 | Open questions

Five load-bearing open questions tracked on synthesis, surfaced during ingest of the initial cadmium batch:

First, the four major dietary reference values (ATSDR chronic oral MRL, EFSA TWI, JECFA PTMI, EPA IRIS oral RfD) differ by up to approximately a factor of 8 on overlapping primary literature. The synthesis page tracks the methodological choices that drive the differences. Any threshold-setting exercise drawing on these reference values cannot silently average across the four; it must name which reference it calibrates to and why, because

the divergence reflects unresolved methodological choices rather than measurement noise. The EFSA 2011 reaffirming statement, which articulates the EU-side explanation for the divergence from JECFA, is not yet in the corpus and is flagged for future ingest.

Second, dietary cadmium carcinogenicity dose-response remains unresolved. IARC maintains the Group 1 classification on the basis of occupational inhalation studies; general-population epidemiology produces statistical associations for cancers of the lung, endometrium, bladder, and breast; EFSA 2009 judged the data insufficient for quantitative dose-response modeling. The synthesis needs to surface the uncertainty without under- or over-claiming on cancer risk.

Third, the cardiovascular endpoint has shifted in framing between the 2015 Handbook chapter ("variable" evidence) and the 2021 Casarett Essentials chapter ("strong association"). The wiki records both positions without resolution pending a 2015-to-present literature search.

Fourth, the EPA IRIS cadmium assessment provenance gap: the raw PDF labeled "EPA_IRIS_Cadmium-ToxicologicalReview.pdf" is a 1999 external review draft that was never adopted; the operative 1985 IRIS record remains the canonical US EPA reference, but is not in the corpus. The 1999 draft is flagged for a future historical ingest wave.

Fifth, the Codex standards landscape is now substantially covered. Matrix-level Codex cadmium MLs are captured in CXS 193-1995, and the cocoa-cadmium Code of Practice is captured in CXC 81-2022 (the precedent document that anchors the broader CoP work initiated at CCCF17 2024).

13 | Frequently asked questions

Which foods give you the most cadmium?

The foods that contribute most to overall cadmium exposure are everyday staples eaten in large volume rather than the most contaminated ones: cereals like rice and wheat, potatoes and starchy roots, leafy vegetables, nuts, oilseeds, pulses, and meat (EFSA 2009, JECFA 91st). Separately, the highest-concentration foods are bivalve molluscs other than oysters, wild mushrooms, cocoa and chocolate, seaweed, fungi, oilseeds, and organ meats such as kidney and liver (EFSA 2009). Regular consumers of bivalve molluscs average 4.6 µg/kg body weight per week and regular wild-mushroom eaters 4.3, both well above the EFSA population mean of 2.3.

Why is cadmium considered harmful and where does it build up in the body?

Cadmium is a non-essential heavy metal that is retained in the kidney and liver with a biological half-life of decades, so body burden accumulates across a lifetime and is never meaningfully excreted (Nordberg 2015). Its main toxic effect is renal tubular dysfunction, with bone demineralization (osteomalacia, osteoporosis, and fracture risk) as a secondary endpoint (Nordberg 2015, EFSA 2009). It is also classified by IARC as a Group 1 human carcinogen based on occupational inhalation studies. There is no effective clinical treatment once cadmium accumulates, which is why exposure reduction is the appropriate response (Ufelle and Barchowsky 2021).

Who is most at risk from dietary cadmium?

Children aged 0.5 to 12 have mean dietary exposure about 60 percent higher than adults because they eat more food per unit body weight, and with cocoa products their total intake can reach 96 percent of the JECFA reference value (JECFA 91st). Women of reproductive age with low iron stores absorb cadmium more efficiently, and pregnant women appear to accumulate more than nonpregnant women (Nordberg 2015, Ufelle and Barchowsky

2021). Postmenopausal women, vegetarians, regular consumers of bivalve molluscs or wild mushrooms, and smokers also carry elevated exposure; smoking roughly doubles body burden (EFSA 2009, Nordberg 2015).

What are the official limits and safe-intake values for cadmium?

Four major bodies set dietary reference values: ATSDR's chronic oral Minimal Risk Level of 0.1 µg/kg/day (tightest), EFSA's tolerable weekly intake of 2.5 µg/kg body weight per week, JECFA's provisional tolerable monthly intake of 25 µg/kg body weight per month, and the US EPA IRIS oral reference dose of about 1.0 µg/kg/day for food (ATSDR 2012, EFSA TWI, JECFA PTMI). Roughly a tenfold range separates them, reflecting different methodological choices on overlapping data rather than disagreement on the underlying toxicology. There are also binding maximum levels in food, such as EU limits and Codex matrix limits like 0.4 mg/kg for polished rice (Codex CXS 193-1995).

How is cadmium measured in food and in people?

Occurrence and biomonitoring studies use methods spanning flame atomic absorption spectrometry through ICP-MS, whose detection limits differ by four orders of magnitude, so pooled datasets must be handled carefully to avoid false precision (ATSDR 2012). FDA's Toxic Elements Program and ATSDR biomonitoring both use ICP-MS methods. For people, exposure is assessed by measuring cadmium in whole blood (recent exposure) or urine normalized to creatinine (cumulative body burden); the EFSA, ATSDR, and JECFA derivations all use urinary cadmium as the exposure metric (Jakubowski 2012).

What can I actually do to lower my cadmium exposure?

Because the biggest population contributors are nutritional staples like rice, wheat, potatoes, and leafy vegetables, the guidance is not to avoid staples but to manage the high-concentration foods and behaviors that push you above average (EFSA 2009). Moderating frequent cocoa and chocolate consumption is the single highest-leverage step for children under 12 (JECFA 91st). Limiting regular bivalve molluscs, wild mushrooms, and organ meats, not smoking, and correcting iron deficiency (which raises cadmium absorption) all reduce exposure (Nordberg 2015, Ufelle and Barchowsky 2021).

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