

Lead

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Abstract

This synthesis draws on ATSDR, EFSA, JECFA, EPA IRIS, CDC, OEHHA, FDA Closer to Zero, mechanism reviews, and chapter-level textbook context from Ufelle & Barchowsky 2021. The complete citation list remains at the bottom of the page so first-time readers can get the main interpretation before the source trail.

1 | Overview

Lead is a non-essential heavy metal and a pervasive environmental contaminant. It enters the human food system through atmospheric deposition from historical leaded gasoline use, soil contamination from leaded paint and industrial emissions, contact with legacy lead-containing food processing equipment and water distribution materials, and uptake by crops from contaminated soil (ATSDR 2020). The metal is absorbed at higher rates by children than by adults (5 to 15 percent in adults, with children retaining more of what they absorb), distributes to soft tissues in the short term and to bone over decades, and is excreted slowly through urine and faeces (ATSDR 2020). Blood lead has a half-life of approximately 30 days; bone lead has a half-life of 10 to 30 years and serves as an internal reservoir that releases lead during pregnancy, lactation, and bone resorption (ATSDR 2020).

The defining feature of lead's regulatory framing is the absence of a demonstrated effect threshold for the most sensitive endpoint, developmental neurotoxicity in young children (EFSA 2010). JECFA withdrew its previous provisional tolerable weekly intake of 25 µg/kg b.w./week in 2010 (JECFA 73rd), EFSA concluded the same year that no PTWI is health-protective (EFSA 2010), EPA IRIS does not derive an oral reference dose (EPA IRIS Lead), and ATSDR does not derive Minimal Risk Levels for lead (ATSDR 2020). Operational US public-health action runs through CDC's blood lead reference value of 3.5 µg/dL (CDC BLRV) and FDA's Closer to Zero Interim Reference Levels (2.2 µg/day for children, 8.8 µg/day for women of childbearing age, derived from the BLRV with a 10x safety factor) (FDA CTZ 2025). California Proposition 65 lists lead under both cancer and reproductive-toxicity categories with separate quantitative implementation thresholds (OEHHA Prop 65).

2 | At a glance

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

First, no major regulatory body has identified a safe blood lead level for children. Lead's developmental neurotoxicity occurs at the lowest measurable blood concentrations, and effects on cognition, behavior, and brain development have been documented at blood lead levels well below the population threshold the CDC uses for screening (3.5 µg/dL) (EFSA 2010, CDC BLRV). The framing for lead is "minimize exposure as far as practicable," not "stay below a specific number." Unlike most environmental hazards, lead does not have a "safe daily dose" that consumers can target.

Second, children are at materially higher risk than adults. Children absorb a larger fraction of ingested lead, retain more of what they absorb, and are exposed during developmental windows when the brain is most susceptible to lead's effects (ATSDR 2020). EFSA 2010 estimates that European children's average dietary lead exposure (0.80 to 3.10 µg/kg body weight per day) already exceeds the developmental-neurotoxicity benchmark dose lower confidence limit of 0.50 µg/kg b.w./day across most of the country range; high-consumer European children exceed the BMDL by approximately 10-fold. Pregnancy is also a high-risk period because bone lead accumulated over a lifetime can mobilize during pregnancy and lactation, transferring to the fetus or nursing infant (ATSDR 2020).

Third, lead reaches the food supply through specific identifiable pathways that consumers can prioritize. Cereals are the dominant population-level dietary contributor (per EFSA 2010); root vegetables grown in contaminated soil are an FDA Closer to Zero target category (FDA CTZ 2025). Outside of food, dust and soil from older homes with deteriorating leaded paint, drinking water from lead service lines or lead-soldered plumbing, and traditional or imported cosmetics, lead-glazed ceramics, and certain herbal medicines are the meaningful non-dietary pathways for most consumers in jurisdictions where leaded gasoline has been phased out (ATSDR 2020).

3 | Toxicology

Lead is primarily a developmental neurotoxicant. The 2010 EFSA opinion identified developmental neurotoxicity in young children as the most sensitive critical endpoint, with a Benchmark Dose Lower Confidence Limit (BMDL01) of 12 µg/L blood lead, corresponding to a dietary intake of 0.50 µg/kg body weight per day. Cognitive deficits, behavioral abnormalities, and reduced IQ have been documented in cohort studies at blood lead levels below the BMDL, supporting the regulatory position that no safe level exists. The molecular mechanism of lead neurotoxicity operates through multiple pathways: substitution for divalent cations (Ca²⁺ and Zn²⁺) in zinc-finger transcription factors essential for brain development (Ordemann and Austin 2016), disruption of glutamatergic signaling through interference with NMDA, AMPA, and metabotropic glutamate receptors and the EAAT, VGLUT, and SNAT transporter families (Tamagno and Freeman 2025), induction of oxidative stress and excitotoxicity, and inhibition of DNA repair.

Cardiovascular effects are the second canonical adult endpoint. EFSA 2010 derived a BMDL01 of 36 µg/L blood lead for systolic blood pressure effects (1.50 µg/kg b.w./day dietary equivalent). Renal effects are the third canonical adult endpoint, with a BMDL10 of 15 µg/L for chronic kidney disease prevalence (0.63 µg/kg b.w./day dietary equivalent), comparable in dietary-equivalent terms to the developmental BMDL01 (EFSA 2010).

Hematological effects are mechanistically central to lead toxicity. Lead inhibits δ-aminolevulinic acid dehydratase (ALAD), an essential enzyme in heme biosynthesis; Huang et al. 2020 used Generalized Additive Models to detect the dose-response threshold for ALAD activity reduction in occupationally exposed workers,

providing biomarker-level evidence of mechanistic effects at exposures below those producing clinical anemia. Additional documented endpoints include immunological effects, peripheral neuropathy in adults, and reproductive effects (the basis of California Prop 65 reproductive listing) (ATSDR 2020, OEHHA Prop 65).

Lead is classified by EPA as B2 (probable human carcinogen, animal evidence with inadequate human evidence) (EPA IRIS Lead) and by California Prop 65 as a chemical known to cause cancer (OEHHA Prop 65); quantitative cancer dose-response is not derived in the IRIS file because the developmental neurotoxicity endpoint is more sensitive and dominates regulatory action (EPA IRIS Lead).

4 | Typical exposure routes

Dietary intake is one of several meaningful routes for the general population. Adult gastrointestinal absorption of ingested lead is approximately 5 to 15 percent, with adults retaining typically less than 5 percent of an ingested dose; children absorb a substantially higher fraction (often cited as 30 to 50 percent of the ingested dose) and retain more of what they absorb because of incomplete bone-deposition equilibrium (ATSDR 2020). Inhalation of airborne lead is operationally minor since the phase-out of leaded gasoline but remains relevant in occupational settings, in proximity to active smelting or battery manufacture, and from re-entrainment of historically deposited soil and dust (ATSDR 2020). Dust and soil ingestion is the dominant non-dietary route for young children (hand-to-mouth behavior, contaminated indoor dust from deteriorating leaded paint) (EFSA 2010, ATSDR 2020).

Bone lead accumulated over a lifetime is itself an internal exposure source: bone lead mobilizes during pregnancy, lactation, postmenopausal bone resorption, and any other period of accelerated bone turnover, providing endogenous lead exposure to the bloodstream and to the fetus or nursing infant (ATSDR 2020). This bone-mobilization effect is the reason that pre-pregnancy lead exposure remains relevant to fetal exposure decades after the original intake (ATSDR 2020).

Biological half-life and body burden accumulation

Lead distributes between three kinetic compartments with sharply different half-lives. Blood lead has a half-life of approximately 30 days; soft-tissue lead (liver, kidney, brain) has a half-life on the order of weeks to months; and bone lead has a half-life of 10 to 30 years (cortical bone slower than trabecular) (ATSDR 2020). Approximately 90 percent of total body lead burden in adults resides in bone (ATSDR 2020). Bone lead concentrations measured by K-shell X-ray fluorescence (KXRF) reflect cumulative lifetime exposure rather than recent intake, and are increasingly used in epidemiological studies of chronic lead effects (ATSDR 2020).

The kinetic implication for risk assessment is that blood lead at any given moment reflects a weighted combination of recent intake and bone-released endogenous exposure, with the bone fraction dominating in older adults and during physiological states of accelerated bone turnover (ATSDR 2020). This is mechanistically why the developmental window for fetal exposure includes the pre-conception lead body burden of the mother, not just her gestational dietary intake.

5 | Food sources

EFSA 2010 identified cereal products as the dominant population-level dietary contributor to lead exposure across European Member States. Vegetables (root vegetables in particular for children) contribute substantially (EFSA 2010).

Adult dietary lead exposure across European populations ranges from 0.36 to 1.24 µg/kg b.w./day average, up to 2.43 in high consumers. Child dietary lead exposure ranges from 0.80 to 3.10 µg/kg b.w./day average, up to 5.51 in high consumers. Infant exposure ranges from 0.21 to 0.94 µg/kg b.w./day. The EFSA developmental--neurotoxicity BMDL01 of 0.50 µg/kg b.w./day is therefore exceeded by mean adult European exposure on the upper end of the country-specific range, by mean European child exposure across most of the range, and by high-consumer European child exposure by approximately 10-fold (EFSA 2010).

MATRIX	LEAD CONCERN
Cereal products	Dominant population-level contributor in EFSA 2010 European data
Root vegetables (carrots, sweet potatoes)	Soil-uptake driven; FDA CTZ baby-food 20 ppb

What this means for food choice

The framing for lead is different from the framing for most other dietary contaminants because there is no demonstrated safe blood lead level for children. Consumer guidance is therefore not "stay below a specific number" but rather "minimize exposure across all sources." Cereals dominate population-level dietary lead because they are eaten in large volume; an adult who consumes a varied diet within EFSA's average-consumer exposure range (0.36 to 1.24 µg/kg b.w./day) is already at 0.7 to 2.5 times the developmental-neurotoxicity dietary BMDL, and high-consumer children are at approximately 10-fold the BMDL. There is no consumer dietary choice that brings exposure below the BMDL while maintaining a normal diet.

The leverage points for individual consumers, in approximate order of impact:

For children specifically, the FDA Closer to Zero matrix-specific action levels (10 ppb in fruits, non-root vegetables, mixtures, yogurts, custards, and single-ingredient meats; 20 ppb in single-ingredient root vegetables; 20 ppb in dry infant cereals) define an enforcement-relevant federal guidance context for what FDA may regard as adulterated (FDA CTZ 2025; fda2025-lead-processed-baby-foods). They are nonbinding recommendations, not statutory maximum levels. Choosing baby-food brands and products with documented testing programs that operate below these thresholds reduces exposure relative to the population mean.

For non-dietary lead, the dominant exposure pathways for most US consumers are deteriorating leaded paint and contaminated indoor dust in pre-1978 housing, drinking water from lead service lines or lead-soldered plumbing, and (where applicable) imported spices, traditional cosmetics, and lead-glazed ceramics (ATSDR 2020).

For pregnancy and pre-pregnancy planning, the bone-lead reservoir matters: lead body burden accumulated over decades mobilizes during pregnancy, lactation, and postmenopausal bone resorption (ATSDR 2020). Consumers planning pregnancy can reduce future fetal exposure by minimizing current lead intake but cannot meaningfully reduce existing bone burden in the short term (ATSDR 2020).

6 | Regulatory limits

JURISDICTION / BODY	TYPE	VALUE	PAGE
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JECFA (international)	PTWI	Withdrawn 2010; no replacement reference value	jecfa-lead-ptwi--withdrawn
EFSA (EU)	Critical-endpoint BMDLs (no TWI)	Developmental neurotoxicity dietary BMDL01 0.50 µg/kg b.w./day; CV BMDL01 1.50; CKD BMDL10 0.63	efsa-lead-contam-2010
US EPA	IRIS oral RfD	Not derived; qualitative only since 2004	epa-iris-lead-rfd
US ATSDR	MRLs	Not derived (no demonstrated threshold)	ATSDR 2020
US CDC	Blood lead reference value	3.5 µg/dL (97.5th percentile, NHANES 2015-2018)	CDC BLRV
US FDA	Closer to Zero IRL	2.2 µg/day (children); 8.8 µg/day (women of childbearing age)	fda-closer-to-zero
US FDA	CTZ baby food action levels	10 ppb (mixed), 20 ppb (root vegetables), 20 ppb (dry infant cereals); nonbinding final guidance, enforcement--relevant context	fda2025-lead--processed-baby-foods, fda-ctz-Pb-babyfood--10ppb, fda-ctz-Pb-rootveg--20ppb, fda-ctz-Pb-cereal-20ppb
US California	Prop 65 (cancer + reproductive)	Oral NSRL 15 µg/day; reproductive--toxicity MADL 0.5 µg/day	oehha-lead-prop65
Codex Alimentarius	Matrix-level MLs (CXS 193-1995)	Cereal grains 0.2 mg/kg; cereal-based infant foods 0.02; infant formula 0.01 (as consumed); fruits 0.1; leafy vegetables 0.3; fish 0.3; cattle offal 0.2; pig offal 0.15; milk 0.02; full table in source page	Codex CXS 193-1995
EU	Binding maximum levels in food	Infant formula powder 20 ug/kg; infant formula liquid 10 ug/kg; baby food and processed cereal-based infant/young-child food 20 ug/kg; fruit juices 30 or 50 ug/kg by juice type; root/tuber vegetables 100 ug/kg; leaf vegetables 300 ug/kg; fish muscle 300 ug/kg; bivalve molluscs 1500 ug/kg	eu2023-contaminants--maximum-levels

EU	Category 1 infant/young-child lead ppb view	20 ppb for formula powder; 10 ppb for formula liquid; 20 ppb for covered baby foods, processed cereal-based foods, and infant/young-child drinks	eu-2023-915-lead-- infant-young-child-foods
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For the ppb-normalized Category 1 comparison table, see [lead-benchmark-context](#).

What the reference values mean in practice

Lead is unusual among dietary contaminants in that the major regulatory bodies have explicitly declined to set a single tolerable intake level. JECFA withdrew its PTWI of 25 µg/kg b.w./week in 2010 (JECFA 73rd); EFSA reports BMDLs but does not set a TWI (EFSA 2010); EPA IRIS does not derive an oral RfD (EPA IRIS Lead); ATSDR does not derive MRLs (ATSDR 2020). The operational anchor is CDC's blood lead reference value of 3.5 µg/dL, which is itself a population-percentile-based screening threshold, not a health-based safety threshold (CDC BLRV).

For a consumer trying to interpret these values: the CDC value defines "elevated relative to the US child population," not "safe below this threshold." A child below 3.5 µg/dL is not at zero risk; the BLRV simply identifies the top 2.5 percent of measured US children for public-health intervention (CDC BLRV). The FDA Interim Reference Level of 2.2 µg/day for children is the dietary translation of the CDC BLRV, derived with a 10x safety factor for variability in dietary-to-blood-lead conversion (FDA CTZ 2025).

The practical consumer position: there is no "safe daily lead intake" that a regulator has endorsed. Consumer action should focus on minimizing total lead exposure (dietary plus dust plus water plus other sources), with priority weighting toward children and pregnancy.

7 | Testing

Blood lead concentration is the standard exposure biomarker for human health assessment, measured by graphite furnace atomic absorption spectrometry, ICP-MS, or LeadCare anodic stripping voltammetry (point-of-care) (ATSDR 2020). The CDC reference value of 3.5 µg/dL is denominated in whole blood (CDC BLRV). Bone lead measurement by K-shell X-ray fluorescence (KXRF) is used in research settings to assess cumulative lead body burden and is the most direct measurement of long-term exposure (ATSDR 2020). ALAD activity in red blood cells (Huang et al. 2020) is a sensitive biochemical biomarker of lead's interference with heme biosynthesis, with detectable reduction at blood lead levels below clinical anemia thresholds.

Food and water lead concentrations are typically measured by ICP-MS, the method FDA's Toxic Elements Program uses for the Closer to Zero data underlying the action levels (FDA TDS 2018-2020). Detection limits in the 0.001 to 0.1 µg/L range are achievable, sufficient for the 10 ppb level the CTZ guidance specifies as the smallest of the three baby-food action levels (FDA CTZ 2025).

8 | Microbiome context

Pending dedicated microbiome ingests. The lead-microbiome literature includes documented gut microbiome perturbations in lead-exposed populations and animal models, with potential implications for lead absorption efficiency itself (microbial activities influence trace metal speciation and bioavailability in the gut). The

wikibiome-crosswalk anchors are tentatively lead-gut-axis and lead-dysbiosis for federation with the WikiBiome project.

9 | Historical context

The current regulatory consensus that no safe blood lead level exists for children rests on a multi-decade public-health record. Population-level blood lead concentrations in US children declined from a NHANES II (1976-1980) geometric mean of approximately 15 µg/dL to a NHANES 2015-2018 geometric mean below 1 µg/dL, with the CDC blood lead reference value declining in parallel from an earlier value of 10 µg/dL (1991-2012), to 5 µg/dL (2012-2021), to the current 3.5 µg/dL (CDC BLRV). Effects are now established at blood lead levels below 5 µg/dL, well within the range where most US children currently fall (CDC BLRV).

The corresponding regulatory conclusion, JECFA's 2010 withdrawal of the PTWI and EFSA's 2010 statement that no PTWI is health-protective (JECFA 73rd, EFSA 2010), is the cumulative position the wiki adopts for current lead content.

10 | Vulnerable populations

POPULATION	BASIS
Children (especially under age 6)	Higher GI absorption, longer retention, exposure during developmental windows for brain development; CDC BLRV of 3.5 µg/dL is the screening threshold (CDC BLRV, ATSDR 2020)
Pregnant and potentially-pregnant women	Bone lead mobilizes during pregnancy and lactation, transferring to fetus and nursing infant; FDA IRL of 8.8 µg/day for women of childbearing age (ATSDR 2020, FDA CTZ 2025)
Residents of older housing (pre-1978 in US)	Deteriorating leaded paint and contaminated indoor dust are major exposure sources; tap water from lead service lines or lead-soldered plumbing (ATSDR 2020)
Communities near smelters, battery operations, or historical mining	Air, soil, and locally produced food carry elevated lead (ATSDR 2020)
Consumers of imported spices, traditional cosmetics, lead-glazed ceramics	Documented adulteration and traditional-use exposure pathways outside standard retail food supply (ATSDR 2020)
Workers in lead-related occupations	Battery manufacture, secondary smelting, radiator repair, painting and abatement, firing-range work (ATSDR 2020)
Postmenopausal women	Bone resorption mobilizes lifetime lead body burden into circulation (ATSDR 2020)

If you are in one of these groups

For parents of children under age 6: pre-1978 housing (the cutoff year for lead-paint phase-out in the US), lead service lines, and lead-soldered plumbing are priority targets for testing and remediation (ATSDR 2020). Pediatric blood lead screening is part of standard well-child care in most US settings; children identified above the BLRV are followed clinically with environmental investigation (CDC BLRV).

For pregnant and potentially-pregnant women: bone lead body burden accumulated over a lifetime is the dominant fetal exposure source, and there is no short-term intervention that meaningfully reduces existing bone lead (ATSDR 2020). Pre-conception minimization of current intake is the operational lever. The California OEHHA Proposition 65 reproductive-toxicity MADL applies to consumer products sold in California and is the directly relevant California regulatory threshold; products carrying a Prop 65 reproductive-toxicity warning for lead are products that, at typical use, would exceed this level (OEHHA Prop 65).

For the immigrant and traditional-medicine consumer: certain imported spices (particularly turmeric and chili powders from some sources), traditional cosmetics (surma, kohl, kajal in some preparations), traditional medicines (ayurvedic, traditional Chinese, some Latin American), and lead-glazed ceramics in contact with acidic foods have produced documented elevated lead exposure cases in the US public-health literature (ATSDR 2020).

11 | App-layer integration

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

The reference-value scale for lead is qualitatively different from cadmium, mercury, or arsenic because no single tolerable-intake reference value exists. The app should not display a "percent of reference" for dietary lead in the way it does for cadmium; instead, the app should reference the FDA Closer to Zero matrix-specific action levels for products in the baby-food categories, the FDA IRL of 2.2 µg/day for children (with 10x safety factor flag noted), and the EFSA dietary BMDL01 of 0.50 µg/kg b.w./day for users wanting an explicit benchmark.

Pediatric multipliers for lead are amplified relative to cadmium because of higher absorption and longer retention in children (ATSDR 2020, EFSA 2010). Default app pediatric exposure multipliers: 1.6x adult per-kg for ages 0.5-12 (per the EFSA child:adult ratio finding) (EFSA 2010), and an additional GI absorption multiplier of approximately 3-5x for children to reflect the higher absorption fraction (ATSDR 2020).

Structured outputs for app consumption:

- Adult GI absorption: 5 to 15 percent (use midpoint 10 percent for default) (ATSDR 2020).
- Child GI absorption: 30 to 50 percent (use midpoint 40 percent for default in pediatric mode) (ATSDR 2020).
- Blood lead half-life: 30 days (ATSDR 2020).
- Bone lead half-life: 10 to 30 years (use 20 years for body-burden modeling) (ATSDR 2020).
- Bone fraction of total body lead (adults): approximately 90 percent (ATSDR 2020).
- Non-dietary exposure flags: pre-1978 housing (lead paint, deteriorating dust), lead service lines (drinking water), traditional cosmetics, imported spices, lead-glazed ceramics (ATSDR 2020).

For consumer-facing risk communication, the app should never present "safe lead intake" or "safe blood lead level" framings. Acceptable framings: "your estimated weekly lead intake from these products is X µg, which is Y percent of the FDA Interim Reference Level for your age group" or "no consumer-protective blood lead threshold has been established; minimize total exposure as far as practicable."

12 | Open questions

Three load-bearing open questions for lead, surfaced by the current ingest:

First, the absence of a single international tolerable intake reference value is operationally awkward. JECFA withdrew the PTWI in 2010, EFSA reports BMDLs without setting a TWI, EPA IRIS is qualitative-only, ATSDR does not derive MRLs. Operational US action runs through CDC BLRV → FDA IRL → CTZ matrix-specific action levels, but this chain is not internationally harmonized. A practicability-based framing such as "lead exposure as far below typical as practicable" is methodologically coherent at the framing level but does not resolve to a single numeric anchor, because no agency currently provides one.

Second, the carcinogenicity classification (EPA B2, Prop 65 listed) is settled at the categorical level but not derived to a quantitative cancer slope factor. Whether dietary lead exposures contribute meaningfully to cancer risk at general-population levels remains an area where regulatory bodies have declined to derive quantitative dose-response, citing data limitations.

Third, the bone-lead-mobilization contribution to fetal and infant exposure is mechanistically established but not quantitatively integrated into FDA's IRL framework. The IRL is anchored on dietary intake during the exposure window of interest, but for pregnant women a substantial fraction of fetal blood lead derives from maternal bone reserves accumulated decades earlier. How to incorporate this non-dietary contribution into intake-anchored frameworks for products marketed to pregnant women is an open methodological question.

13 | Frequently asked questions

Is there a safe level of lead in food?

No regulatory body has identified a safe level of lead for children. JECFA withdrew its provisional tolerable weekly intake in 2010 (JECFA 73rd), and EFSA concluded the same year that no intake level is health-protective, because lead's developmental neurotoxicity in young children has no demonstrated threshold (EFSA 2010). The operational framing is to minimize lead exposure as far as practicable rather than to stay below a fixed number.

Which foods contribute the most dietary lead?

In EFSA's European exposure assessment, cereal products are the dominant population-level contributor because they are consumed in large volume; vegetables, and root vegetables in particular for children, also contribute substantially (EFSA 2010). Lead enters these foods mainly through uptake from contaminated soil and atmospheric deposition (ATSDR 2020).

Are children more vulnerable to lead than adults?

Yes. Children absorb a larger fraction of ingested lead, retain more of what they absorb, and are exposed during developmental windows when the brain is most susceptible (ATSDR 2020). EFSA estimates that average European children's dietary lead exposure already exceeds the developmental-neurotoxicity benchmark dose (BMDL01 of 0.50 µg/kg body weight per day) across most of its country range (EFSA 2010).

What are the FDA's lead action levels for baby food?

Under its Closer to Zero program (2025 final guidance), the FDA set action levels of 10 ppb for fruits, non-root vegetables, mixtures, yogurts, custards, and single-ingredient meats; 20 ppb for single-ingredient root vegetables; and 20 ppb for dry infant cereals (FDA CTZ 2025; fda2025-lead-processed-baby-foods). These are nonbinding recommendations that establish an enforcement-relevant context, not statutory maximum levels.

How is lead exposure measured?

Blood lead concentration ($\mu\text{g}/\text{dL}$) is the standard human biomarker; the CDC blood lead reference value is 3.5 $\mu\text{g}/\text{dL}$, a population-screening threshold rather than a health-based safety threshold (CDC BLRV). Bone lead measured by K-shell X-ray fluorescence reflects cumulative lifetime exposure (ATSDR 2020). Lead in food and water is typically measured by ICP-MS (FDA TDS 2018-2020).

References

- [1] Toxic Effects of Metals (Chapter 23), in Casarett & Doull's Essentials of Toxicology, Fourth Edition
Ufelle AC and Barchowsky A · Casarett & Doull's Essentials of Toxicology, Fourth Edition. McGraw Hill Education · 2021 · www.mhprofessional.com
TEXTBOOK
- [2] Toxicological Profile for Lead
Agency for Toxic Substances and Disease Registry · 2020 · doi.org/10.15620/cdc:95222
GOVERNMENT
- [3] Scientific Opinion on Lead in Food
EFSA Panel on Contaminants in the Food Chain (CONTAM) · EFSA Journal 2010;8(4):1570 · 2010 · doi.org/10.2903/j.efsa.2010.1570
GOVERNMENT
- [4] Lead (Addendum), 73rd Meeting of the Joint FAO/WHO Expert Committee on Food Additives — Safety Evaluation of Certain Food Additives and Contaminants
Joint FAO/WHO Expert Committee on Food Additives · WHO Technical Report Series 960 (2011); lead addendum in WHO Food Additives Series No. 64 · 2011 · www.who.int
GOVERNMENT
- [5] Lead and Compounds (Inorganic) — IRIS Chemical Assessment Summary
U.S. Environmental Protection Agency, National Center for Environmental Assessment · U.S. Environmental Protection Agency, Integrated Risk Information System · 2004 · iris.epa.gov
GOVERNMENT
- [6] Blood Lead Reference Value (3.5 $\mu\text{g}/\text{dL}$)
U.S. Centers for Disease Control and Prevention · 2021 · www.cdc.gov
GOVERNMENT
- [7] Action Levels for Lead in Processed Food Intended for Babies and Young Children: Guidance for Industry
U.S. Food and Drug Administration · U.S. Department of Health and Human Services, Food and Drug Administration, Human Foods Program · 2025 · www.fda.gov
GUIDANCE
- [8] Lead and Lead Compounds — Proposition 65 Chemical Listing
California Office of Environmental Health Hazard Assessment · oehha.ca.gov
GOVERNMENT
- [9] Lead neurotoxicity: exploring the potential impact of lead substitution in zinc-finger proteins on mental health
Ordemann JM and Austin RN · Metallomics, Issue 6, 2016 · 2016 · doi.org/10.1039/c5mt00300h
REVIEW
- [10] Glutamate-Mediated Neural Alterations in Lead Exposure: Mechanisms, Pathways, and Phenotypes
Tamagno WA and Freeman JL · Toxics 13(7):519 · 2025 · doi.org/10.3390/toxics13070519
REVIEW
- [11] Use of Generalized Additive Model to Detect the Threshold of δ -Aminolevulinic Acid Dehydratase Activity Reduced by Lead Exposure
Huang CC, Yang CC, Liu TY, Dai CY, Wang CL, and Chuang HY · International Journal of Environmental Research and Public Health · 2020 · doi.org/10.3390/ijerph17165712
REVIEW

[12] Total Diet Study Report: Fiscal Years 2018-2020 Elements Data

U.S. Food and Drug Administration · 2022 · www.fda.gov

GOVERNMENT

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