

Arsenic

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Abstract

This page draws on the ATSDR 2007 Toxicological Profile for Arsenic (ATSDR 2007), the EPA IRIS January 2025 inorganic arsenic toxicological review (EPA IRIS iAs 2025), the EFSA CONTAM 2009 Scientific Opinion on Arsenic in Food (EFSA As 2009), the JECFA 82nd meeting arsenic monograph (JECFA 82nd), the OEHHA Proposition 65 inorganic arsenic listing (OEHHA Prop 65 As), the EPA drinking water arsenic MCL rule (EPA As MCL), the FDA inorganic arsenic infant rice cereal action level (FDA iAs 2020), the joint FDA/EPA fish consumption advice (FDA/EPA Fish Advice), and the Thomas 2021 review of arsenic methylation (Thomas 2021).

Chapter-level cross-metal toxicology context for arsenic toxicokinetics, methylation/excretion, acute and chronic toxicity, carcinogenicity, and treatment is connected from Ufelle & Barchowsky 2021.

1 | Overview

Arsenic is a metalloid that occurs in food and water as multiple chemical species with substantially different toxicity profiles. Inorganic arsenic (iAs) species, principally arsenite (As(III)) and arsenate (As(V)), are the toxicologically dominant forms and are classified as Group 1 human carcinogens by IARC, with documented dose-response associations for lung, bladder, and skin cancer at chronic exposures occurring in groundwater-exposed populations worldwide (ATSDR 2007, EFSA 2009). The wiki's arsenic content is organized primarily around inorganic arsenic because that is the species relevant to dietary risk and to all major regulatory reference values.

The dominant dietary exposure route for inorganic arsenic in non-groundwater-affected populations is rice and rice-based foods; rice efficiently accumulates iAs from flooded-paddy soils because flooded conditions reduce arsenic to the As(III) species which is more bioavailable to plants (EFSA 2009, Su et al. 2023). Major regulatory bodies have concluded that the prior threshold-based reference values for iAs (notably the JECFA PTWI of 15 µg/kg b.w./week) are no longer appropriate because cancer occurs at exposures below those thresholds; modern regulatory framing operates against benchmark dose lower confidence limits and matrix-specific action levels rather than a single international PTWI (EFSA 2009, JECFA 82nd). EPA finalized a major IRIS reassessment

of inorganic arsenic in January 2025, the most consequential US arsenic regulatory update in decades (EPA IRIS 2025).

2 | At a glance

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

First, the form of arsenic matters more than the amount. Inorganic arsenic in rice, rice-based foods, drinking water, and certain juices is the carcinogen of regulatory concern (EFSA 2009). Total-arsenic measurements that do not distinguish between inorganic and organic species are not informative for risk; the wiki's content focuses on inorganic arsenic specifically. When you see a "total arsenic" number on a product test or in a news article, the actionable question is what fraction is inorganic, which often requires speciation analysis (HPLC-ICP-MS) rather than total-arsenic ICP-MS (Navaretnam et al. 2025).

Second, rice is the dominant single dietary source of inorganic arsenic for non-groundwater-affected US and European consumers (EFSA 2009). Brown rice carries more inorganic arsenic than white rice because the bran layer concentrates the metal (Su et al. 2023). Rice cereal is often the first solid food introduced to infants, which is why FDA established a 100 ppb action level for infant rice cereal in 2020 (FDA 2020). Frequent rice consumers (gluten-free diets, certain regional cuisines) and parents of infants on rice cereal should treat rice as their primary inorganic-arsenic exposure variable.

Third, private well water is not regulated under EPA's 10 ppb drinking-water Maximum Contaminant Level; private-well users in regions with documented elevated groundwater arsenic should test their water and treat or substitute water sources where concentrations exceed 10 µg/L (EPA Arsenic MCL).

3 | Toxicology

Inorganic arsenic is a Group 1 human carcinogen (IARC) with dose-response associations for lung, bladder, and skin cancer at chronic exposures including via drinking water and food. EFSA 2009 reports BMDL01 values for cancer endpoints ranging 0.3 to 8 µg iAs/kg b.w./day depending on endpoint and study selection, and concludes that the prior JECFA PTWI of 15 µg iAs/kg b.w./week is no longer appropriate because cancer occurs at exposures below the PTWI. ATSDR 2007 derives a chronic oral MRL of 0.3 µg As/kg/day anchored on dermal effects (hyperpigmentation, palmar/plantar keratosis) observed in chronically exposed populations, with cancer dose-response derived through EPA IRIS rather than ATSDR.

Non-cancer endpoints documented in the inorganic arsenic literature include cardiovascular effects (atherosclerosis, blackfoot disease in highly exposed Taiwanese populations), neurodevelopmental effects in children, dermal effects (the basis of the ATSDR chronic oral MRL), peripheral neuropathy in adults, reproductive effects, immunological effects, and emerging diabetes-mellitus associations (ATSDR 2007). Arsenic mechanism includes binding to vicinal sulfhydryl groups in proteins and enzymes, oxidative stress generation, methylation interference (biomethylation of inorganic arsenic to MMA and DMA species; see below), and direct genotoxicity through indirect pathways (arsenic does not produce direct DNA adducts but operates through oxidative stress and DNA repair inhibition) (Thomas 2021).

Inorganic arsenic biomethylation, reviewed by Thomas 2021 across three decades of research, is the major in vivo metabolic transformation: AS3MT methyltransferase catalyzes sequential methylation of inorganic arsenic to monomethylated (MMA) and dimethylated (DMA) species. The toxicokinetic and toxicodynamic implications were long debated as detoxification (dimethylated species are excreted faster) versus toxification (some

intermediates may be more reactive than the inorganic precursors). Inter-individual variation in methylation efficiency, driven by AS3MT polymorphisms and dietary cofactors (folate, B12), influences susceptibility to chronic arsenic effects. Methylation patterns are themselves biomarkers used in cancer-risk studies of arsenic-exposed populations.

The January 2025 EPA IRIS Toxicological Review of Inorganic Arsenic is the current US federal scientific anchor for arsenic dose-response and supersedes prior IRIS values. The full quantitative outputs (oral RfD, oral cancer slope factor) require detailed extraction from the review document and are pending consolidation on this wiki.

4 | Arsenic speciation and the organoarsenical inertness assumption

The regulatory architecture for arsenic rests on a species distinction. Inorganic arsenic is the carcinogen of concern and is regulated, characterized with dose-response relationships, and the subject of the FDA Closer to Zero initiative and EU maximum levels, whereas organic arsenic species in seafood, principally arsenobetaine, have historically been excluded from risk characterization on the assumption that they are metabolically inert and pass through the body unchanged. That assumption is what makes the inorganic-versus-total-arsenic distinction meaningful in practice: a product reporting total arsenic at 200 ppb that is 97 percent arsenobetaine has been treated as fundamentally different from one at 200 ppb that is 97 percent inorganic arsenic.

The organoarsenical inertness assumption is now under active challenge on three independent evidentiary fronts, drawn together in the Heavy Metal Index synthesis. The organoarsenical inertness assumption is increasingly contested. On the biomonitoring front, Davydiuk et al. 2023 documents that arsenosugars and arsenolipids from seafood are metabolized to dimethylarsinic acid (DMA) by a route that does not pass through the inorganic arsenic methylation sequence, so studies that use urinary percent-DMA as a methylation-efficiency index in seafood-eating populations can systematically underestimate those populations' inorganic arsenic exposure. On the metabolism front, Liu et al. 2023 found that organic arsenicals in crab and scallop underwent a two- to threefold increase in the inorganic As(V) fraction during a standardized in vitro gastrointestinal digestion, indicating that the speciation measured in a food at the moment of consumption is not necessarily the speciation present at the point of intestinal absorption. On the epidemiology front, the Kagawa et al. 2023 J-MICC Daiko prospective cohort of 2,709 Japanese adults reported that serum total arsenic, estimated at 98 percent arsenobetaine, was associated with dose-dependent hypertension risk after adjustment for fish-consumption frequency and cardiovascular risk factors, an association the inertness assumption would not predict.

The concern is not confined to marine fish. Raab et al. 2024 found that 57 percent of the total arsenic in the green microalga *Chlamydomonas reinhardtii* exists as arsenolipids, which bears on spirulina, chlorella, and algae-protein ingredients whose total-arsenic measurement may not quantitatively recover the arsenolipid fraction. Walenta et al. 2024 identified a previously unreported arsenobetaine amide in four mushroom species, extending the diversity of dietary organoarsenicals beyond the well-characterized marine suite. Sim et al. 2024 found that *Laminaria digitata* carries more than half of its total arsenic as inorganic arsenic, showing that the default assumption that seaweed arsenic is organic and therefore low-risk is wrong for this commercially important kelp genus, a matrix for which EU Regulation 2023/915 now sets specific inorganic arsenic limits.

These findings do not refute the inertness assumption, and they do not demonstrate that arsenobetaine is toxic. Arsenobetaine remains the dominant arsenic species in marine fish, and high fish consumption remains a net nutritional benefit for most populations. What the evidence establishes is narrower and load-bearing for how this wiki characterizes arsenic in seafood-containing products: total arsenic in seafood is not equivalent to inorganic arsenic in seafood, and it is also not equivalent to harmless organic arsenic. The unqualified statement that organic arsenic from seafood is not a health concern can no longer be reproduced without qualification,

because it is now inconsistent with the published literature. The missing evidence that would resolve the question is a controlled human in vivo feeding study measuring inorganic arsenic absorption from a seafood meal; until it is conducted, seafood and microalgae-derived matrices tested for total arsenic only are incompletely characterized with respect to their inorganic arsenic burden. See organoarsenical-inertness-assumption for the full synthesis and its eight anchor sources.

5 | Typical exposure routes

Drinking water is the dominant inorganic arsenic exposure route in groundwater-affected regions worldwide. The EPA Maximum Contaminant Level for arsenic in drinking water is 10 ppb (10 µg/L), established in 2001 to replace the prior 50 ppb standard, applicable to community water systems and non-transient non-community water systems but not to private wells (EPA Arsenic MCL). The 50-to-10 ppb reduction was driven by accumulating cancer-epidemiology evidence in moderately exposed populations (EPA Arsenic MCL).

Dietary intake is the dominant route in non-groundwater-affected populations (EFSA 2009). Rice and rice-based foods carry the highest inorganic arsenic concentrations among common dietary commodities; brown rice is higher than white rice because the bran concentrates the metal (Su et al. 2023); rice-based infant cereal is a particular concern because rice is often the first solid food (FDA 2020).

6 | Food sources

MATRIX	INORGANIC ARSENIC CONCERN
Rice and rice-based foods	Dominant single dietary contributor; brown rice > white rice; 100 ppb FDA action level for infant rice cereal
Drinking water (private wells, groundwater-affected)	Dominant in groundwater-affected populations; EPA 10 ppb MCL applies to public systems
Seaweed (especially hijiki)	Can carry elevated inorganic As; species variation substantial
Apple and grape juice	Documented elevated inorganic As in some products; FDA has issued guidance
Certain leafy vegetables grown on As-contaminated soil	Geography-dependent
Finfish	Predominantly arsenobetaine; historically treated as low-risk for As specifically, though the organoarsenical inertness assumption is now under active challenge (see the arsenic speciation section)
Bivalves and crustaceans	Mixed organic and inorganic species; total-As tests can mislead
Rice protein powder and rice syrup	Concentrate iAs from source rice; per-product characterization needed

EFSA 2009 reports inorganic arsenic exposure across 19 European countries: 0.13 to 0.56 µg/kg b.w./day for average consumers, 0.37 to 1.22 µg/kg b.w./day for 95th-percentile consumers. Children under three years are exposed at 2 to 3 times adult per-kg rates due to greater per-kg food intake.

Primary occurrence: infant formula and first foods

Jackson et al. 2012 from the Dartmouth Trace Element Analysis Laboratory provides the foundational U.S. dataset for As in infant formulas and complementary foods, with speciation by IC-ICP-MS. Total As in 15 main-brand infant formulas ranged 2.2 to 12.6 ng/g; non-dairy formulas (including soy) were significantly higher than dairy-based ($P = 0.003$); formulas containing rice starch were higher again. Speciation showed essentially 100 percent inorganic arsenic in formulas with total As > 6 ng/g, with As(V) dominant. The exposure consequence: a 3-month-old 6.2 kg infant consuming six 120 mL formula bottles daily is exposed to 0.036 to 0.21 $\mu\text{g As/kg/day}$ from formula alone; the upper end exceeds the 0.17 $\mu\text{g/kg/day}$ reference derived from an adult drinking 1 L water at the WHO/EPA 10 $\mu\text{g/L}$ MCL, indicating vulnerable-population overrun before any complementary food enters the diet. In 18 stage 2/3 mixed-meal foods, a significant meat $\times$ rice interaction emerged ($P = 0.0005$): meals containing both meat and rice averaged 18.45 ng/g As (least-squares mean), rice-only meals 11.90 ng/g, and meals without rice 3.75 ng/g. Speciation in these mixed meals was predominantly inorganic As (>70 percent), unexpected for U.S. rice (where DMA usually dominates) and suggesting rice-bran inclusion. In 41 fruit and vegetable purees, most samples ranged 0.32 to 7.81 ng/g, but Brand E pear-containing products averaged 16.6 ng/g, driven by source-specific contamination rather than a general property of pears.

What this means for food choice

For consumers without elevated drinking-water arsenic, rice is the dominant single dietary lever. The leverage points in approximate order of impact:

For infants: rice cereal as a first food is the highest-leverage exposure variable. Alternatives include oat cereal, multi-grain cereal that is not rice-dominant, and barley cereal, all of which carry substantially less inorganic arsenic per serving than rice cereal. The FDA 100 ppb infant rice cereal action level is a non-binding industry guidance, not a consumer-product label commitment; choosing brands that publicly test for and report iAs concentration in their infant rice cereal is the consumer-facing version of this regulation.

For older children and adults: white rice carries less inorganic arsenic than brown rice from the same source, on the order of 50 percent less by mass (Su et al. 2023).

7 | Regulatory limits

JURISDICTION / BODY	TYPE	VALUE	PAGE
EPA (US)	Drinking water MCL (total As)	10 ppb	epa-arsenic-mcl
EPA (US)	IRIS oral RfD and cancer slope (iAs)	January 2025 finalized; values pending detailed extraction	epa-iris-inorganic--arsenic-rfd
FDA (US)	CTZ infant rice cereal action level (iAs)	100 ppb	fda-iAs-rice-cereal--100ppb
ATSDR (US)	Chronic oral MRL (iAs)	0.3 $\mu\text{g As/kg/day}$	ATSDR 2007
OEHHA (US-CA)	Prop 65 cancer listing (iAs)	NSRL pending separate ingest	oehha-arsenic-prop65

EFSA (EU)	BMDL01 anchors (no PTWI)	Cancer BMDL01 0.3 to 8 µg iAs/kg b.w./day depending on endpoint	efsa-arsenic-contam--2009
JECFA (international)	PTWI	Withdrawn (15 µg iAs/kg b.w./week deemed inappropriate at JECFA 72nd, 2010)	JECFA 82nd

What the reference values mean in practice

Arsenic is structurally similar to lead in that the major regulatory bodies have explicitly declined to set a single tolerable intake level for inorganic arsenic. JECFA withdrew its PTWI in 2010 (the same meeting that withdrew the Pb PTWI); EFSA reports BMDLs without setting a TWI; EPA IRIS (January 2025) provides quantitative dose-response that the wiki has not yet extracted. The operational anchors are the EPA drinking water MCL (10 ppb), the FDA infant rice cereal action level (100 ppb), and the ATSDR chronic oral MRL (0.3 µg As/kg/day, anchored on dermal effects rather than cancer).

For a 70-kilogram adult, the ATSDR MRL corresponds to approximately 21 µg iAs/day. EFSA's average European adult iAs exposure of 0.13 to 0.56 µg/kg b.w./day corresponds to approximately 9 to 39 µg/day for the same adult, placing typical exposure at roughly half the ATSDR MRL on the low end and approximately twofold above on the high end. EFSA's 95th percentile (0.37 to 1.22 µg/kg b.w./day, or 26 to 85 µg/day for the 70 kg adult) is consistently above the ATSDR MRL. Average European children are exposed at 2 to 3 times adult per-kg rates.

The practical consumer position: there is no internationally harmonized "safe daily inorganic arsenic intake," but the ATSDR MRL of 0.3 µg/kg/day (dermal-effects anchor) and the EPA drinking water MCL of 10 ppb (cancer-driven derivation) are useful benchmarks. Total dietary iAs above 21 µg/day for a 70 kg adult, or above approximately 6 µg/day for a 20 kg child, is approaching or exceeding the most-conservative regulatory threshold currently in force.

8 | Testing

Inorganic arsenic measurement requires speciation analysis to distinguish iAs from organic arsenic species (arsenobetaine, arsenosugars, MMA, DMA). The standard analytical method is HPLC-ICP-MS, in which liquid chromatography separates the species before ICP-MS detection. Detection limits in the 0.001 to 0.1 µg/L range are achievable (Navaretnam et al. 2025).

Biomonitoring for arsenic exposure typically measures urinary inorganic arsenic plus its methylation metabolites (sum of iAs + MMA + DMA, sometimes denoted U-iAs) (Thomas 2021).

The FDA Toxic Elements Program uses ICP-MS for total arsenic in food matrices, with HPLC-ICP-MS speciation for the regulatory action level enforcement on infant rice cereal (FDA TDS 2018-2020).

9 | Microbiome effects

The gut microbiome operates a substantial parallel arsenal of arsenic-handling enzymes that modify host exposure independently of the AS3MT host pathway. Coryell, Roggenbeck, Walk 2019 from Montana State University is the canonical synthesis. Key pathways:

- **ars operon** (arsR, arsB/acr3, arsC, arsA, arsD, arsH, arsl, arsJ, arsK, arsN, arsP): efflux and detoxification within bacterial cells; protects the microbe but is neutral with respect to host benefit.
- **arsM (arsenic methyltransferase)**: microbial homolog of AS3MT; methylates iAs to MMA and DMA, including the more reactive trivalent intermediates (MMA(III), DMA(III), TMA(III)). The same methylation paradox applies at the microbial level as at the host level: methylation was historically considered detoxification, but trivalent intermediates may be more toxic than the parent iAs.
- **aioAB and arxAB (arsenite oxidases)**: convert As(III) to less-toxic As(V); arxAB functions under the anaerobic conditions of the large intestine, where most of the gut microbiome resides.
- **arrAB (arsenate respiratory reductase)**: certain anaerobic bacteria use As(V) as a terminal electron acceptor for respiration, mobilizing bound As(V).
- **Thiolation**: nonenzymatic sulfide-dependent replacement of As-O bonds with As-S bonds; produces thioarsenicals (MMMTA(V), DMMTA(V)) with cytotoxicity comparable to trivalent oxoarsenicals. Sulfate-reducing bacteria (e.g. desulfovibrio) are implicated as the sulfide source.

The compartment structure of the GIT constrains which pathways operate where. The oral cavity (aerobic, pH 6.2-7.6) supports oxidation. The stomach (microaerobic, pH 1.5-3.5) is hostile to most pathways but tolerated by some. The small intestine (microaerobic to anaerobic, pH 6-7.4) is a mixed-pathway window. The large intestine (anaerobic, pH 5.7-6.7) is where arxAB, arsM, arrAB, and thiolation pathways are most active.

Germ-free and antibiotic-treated mouse data summarized in Coryell 2019 demonstrate that the microbiome causally contributes to arsenic excretion: antibiotic-treated mice excrete less arsenic in stool and accumulate more in organs (liver, spleen, heart, lung) than conventional controls at 25 ppm iAs(V) exposure. AS3MT-knockout mice are hypersensitive to arsenic when germ-free; colonization with human fecal microbiota restores survival, with substantial donor-to-donor variation in protective capacity. Microbiome alpha diversity correlates with arsenic survival in mouse models, suggesting that inter-individual variation in microbiome composition is a partial explanation for the longstanding clinical observation that arsenicosis severity varies dramatically among people with similar exposure.

The Thomas 2021 review summarizes the parallel AS3MT host pathway and the inter-individual genetic variation that drives host methylation efficiency. The wikibiome-crosswalk anchors are arsenic-gut-axis and arsenic-microbial-methylation. Direct human GIT measurement of microbial As biotransformation has not been performed; the wiki tracks the inferential framework while the empirical evidence remains based on environmental, in vitro, and model-organism work.

10 | Historical context: groundwater arsenic and the 10 ppb drinking water rule

The current EPA drinking water MCL of 10 ppb represents a 5-fold reduction from the 50 ppb standard that had been in place since 1942. The reduction was finalized in 2001 and effective for compliance from January 23, 2006 (EPA Arsenic MCL). EPA's 2001 final rule represents the policy translation of the conclusion that arsenic-induced cancer occurs at exposures well below the 1942-era 50 ppb threshold (EPA Arsenic MCL). The driving evidence base for the chronic oral MRL was the dermal-effects literature in chronically exposed populations including the Tseng et al. Taiwanese cohort (ATSDR 2007).

11 | Vulnerable populations

POPULATION	BASIS
Infants on rice cereal	First-solid-food exposure; FDA 100 ppb action level addresses this specifically
Children under 3	EFSA 2009: 2 to 3 times adult per-kg inorganic arsenic exposure
Private-well users in arsenic-affected geographies	Outside EPA MCL coverage; concentrations can substantially exceed 10 ppb without public-health monitoring (EPA Arsenic MCL)
Individuals with low folate or B12 status	Reduced methylation efficiency increases retention of inorganic species (Thomas 2021)

If you are in one of these groups

For parents of infants and young children: choose non-rice or rice-minor first cereals where possible (oat, barley, multi-grain). When rice cereal is consumed, choose products with documented testing programs publishing per-batch inorganic arsenic concentrations. Rinse-and-excess-water cooking practice reduces iAs in cooked rice by approximately 40 to 50 percent.

For pregnant women: the OEHHA Proposition 65 California listing for inorganic arsenic compounds (cancer) applies to consumer products sold in California (OEHHA Prop 65).

For private-well users in known arsenic-affected regions: test well water through an EPA-certified laboratory; if levels exceed 10 µg/L, treat or substitute or reduce consumption from that source (EPA Arsenic MCL).

For frequent rice consumers (gluten-free diets, certain regional cuisines, vegetarians and vegans relying on rice as a primary grain): white rice carries less iAs than brown rice from the same source (Su et al. 2023).

12 | App-layer integration

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

The reference-value scale for inorganic arsenic, like lead, is qualitatively complicated by the absence of an internationally harmonized PTWI. The app should reference (a) the EPA drinking water MCL of 10 µg/L for water-source iAs benchmarking, (b) the FDA infant rice cereal action level of 100 ppb for the rice-cereal infant exposure case, (c) the ATSDR chronic oral MRL of 0.3 µg As/kg/day for general dietary benchmarking, and (d) the EFSA cancer BMDL01 range of 0.3 to 8 µg iAs/kg/day depending on endpoint for users wanting an explicit cancer-risk anchor.

Critical app handling: total-arsenic measurements are not interchangeable with inorganic-arsenic measurements. The app must distinguish total-As from iAs in any input data and must not display "percent of MRL" or "percent of MCL" against total-As when the relevant reference is iAs. For seafood especially, total-As measurements substantially overstate iAs because arsenobetaine and other organic species dominate. That a matrix is organic-arsenic-dominant does not, however, make it zero-risk: following the organoarsenical inertness synthesis, ingredients whose arsenic is primarily organic (finfish, shellfish, and microalgae-derived ingredients such as spirulina and chlorella) should not receive a zero-risk modifier on the strength of the organic

classification alone. Where only total arsenic is available for such a matrix, the app should model it as arsenic speciation incompletely characterized and surface an uncertainty flag rather than a dismissal.

Pediatric multipliers for arsenic: 2 to 3x adult per-kg for children under age 3, per EFSA 2009. Combined with the high GI absorption of inorganic arsenic (80 to 90 percent of soluble forms), pediatric exposure-to-blood translation is approximately linear with intake.

Structured outputs:

- Methylation efficiency (population mean): approximately 60 to 80 percent of iAs methylated to MMA + DMA; varies with AS3MT genotype, folate, and B12 status (Thomas 2021).
- Speciation flag: any input must specify whether values are total As or iAs; default treatment of unflagged "arsenic" measurements should warn the user that speciation matters.
- Rice variety multiplier: brown rice is approximately 2x white rice (Su et al. 2023).

Consumer-facing risk communication should never average total-As and iAs into a single "arsenic" number. Acceptable framings: "your estimated weekly inorganic arsenic intake from these products is X µg, which is Y percent of the ATSDR chronic oral MRL for your body weight" or "this product's inorganic arsenic concentration is below the FDA infant rice cereal action level of 100 ppb."

13 | Open questions

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

First, the January 2025 EPA IRIS Inorganic Arsenic Toxicological Review (EPA/635/R-25/005) is finalized but the wiki has not yet extracted the full quantitative outputs (oral RfD, oral cancer slope factor, inhalation unit risk, critical study identification per endpoint). This is a scoped follow-up: the IRIS document is the most consequential US arsenic regulatory update in decades, and the synthesis page will gain substantial precision when those values land on the regulation page.

Second, the dietary contribution to inorganic arsenic cancer risk at general-population exposure levels remains an area where the regulatory bodies have been cautious about quantitative dose-response. Most of the cancer-epidemiology evidence comes from groundwater-arsenic-affected populations at exposures far above typical US dietary iAs levels; extrapolation to the lower dietary range introduces uncertainty that EFSA 2009 explicitly acknowledged with its BMDL01 range of 0.3 to 8 µg iAs/kg/day depending on endpoint. The EPA IRIS 2025 reassessment may resolve some of this uncertainty.

Third, the contribution of gut-microbiome-mediated arsenic methylation to inter-individual susceptibility is an active research area with implications for biomarker interpretation and for the operational meaning of "individual exposure" in chronic-disease risk modeling. The Thomas 2021 review summarizes host-enzyme AS3MT-pathway evidence; microbiome contributions complicate the simple dose-response framing and are flagged for future microbiome-ingest synthesis.

14 | Frequently asked questions

Does the form of arsenic matter more than the total amount?

Yes. Inorganic arsenic (iAs) is the toxicologically dominant form and the carcinogen of regulatory concern, and it is what the wiki's content focuses on (EFSA 2009). Total-arsenic measurements that do not distinguish inorganic

from organic species are not informative for risk, so when you see a "total arsenic" number the actionable question is what fraction is inorganic. Distinguishing them often requires speciation analysis (HPLC-ICP-MS) rather than total-arsenic ICP-MS (Navaretnam et al. 2025).

What foods are the biggest source of inorganic arsenic in the diet?

For people without elevated drinking-water arsenic, rice and rice-based foods are the dominant single dietary source, because rice efficiently accumulates inorganic arsenic from flooded-paddy soils (EFSA 2009). Brown rice carries more than white rice because the bran layer concentrates the metal (Su et al. 2023). Other notable matrices include seaweed (especially hijiki), apple and grape juice, and rice protein powder and rice syrup. Finfish arsenic is predominantly arsenobetaine and has historically been treated as low-risk for arsenic specifically, though the long-standing assumption that seafood organoarsenicals are biologically inert is now under active scientific challenge (Heavy Metal Index synthesis).

Why is inorganic arsenic a health concern, and what are its effects?

Inorganic arsenic is classified as a Group 1 human carcinogen by IARC, with dose-response associations for lung, bladder, and skin cancer at chronic exposures (ATSDR 2007, EFSA 2009). Non-cancer effects documented in the literature include cardiovascular effects, neurodevelopmental effects in children, dermal effects such as hyperpigmentation and keratosis, peripheral neuropathy, and emerging diabetes associations (ATSDR 2007). Mechanistically it binds sulfhydryl groups in proteins, generates oxidative stress, and inhibits DNA repair rather than forming direct DNA adducts (Thomas 2021).

Who is most vulnerable to arsenic exposure?

Infants fed rice cereal as a first solid food are a key vulnerable group, which is why the FDA set a 100 ppb action level for infant rice cereal (FDA 2020). Children under three are exposed at 2 to 3 times adult per-kilogram rates because of greater per-kg food intake (EFSA 2009). Private-well users in arsenic-affected regions fall outside EPA drinking-water monitoring, and people with low folate or B12 status have reduced methylation efficiency, increasing retention of inorganic species (Thomas 2021).

What are the regulatory limits for arsenic in water and food?

The EPA drinking-water Maximum Contaminant Level for total arsenic is 10 ppb (10 µg/L), lowered from the earlier 50 ppb standard, and it applies to public water systems but not private wells (EPA Arsenic MCL). The FDA set a 100 ppb action level for inorganic arsenic in infant rice cereal in 2020 (FDA 2020). Major bodies have declined to set a single tolerable intake: JECFA withdrew its PTWI of 15 µg/kg body weight per week in 2010, and EFSA reports cancer BMDL01 values of 0.3 to 8 µg iAs/kg body weight per day instead (JECFA 82nd, EFSA 2009). ATSDR derives a chronic oral MRL of 0.3 µg As/kg/day anchored on dermal effects (ATSDR 2007).

How can I reduce arsenic exposure from rice and well water?

For infants, choosing non-rice or rice-minor first cereals such as oat, barley, or multi-grain lowers exposure, since these carry substantially less inorganic arsenic per serving than rice cereal. For older children and adults, white rice carries roughly 50 percent less inorganic arsenic than brown rice from the same source, and cooking rice in excess water that is drained off reduces iAs by approximately 40 to 50 percent (Su et al. 2023). Private-well users in arsenic-affected regions should test their water through an EPA-certified lab and treat, substitute, or reduce use if levels exceed 10 µg/L (EPA Arsenic MCL).

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