

Cadmium Effects in Infants and Children: A Comprehensive Review of Health Impacts, Microbiome Shifts, and Microbial Metallomics

This comprehensive review synthesizes current evidence on cadmium toxicity in infants and children, with particular emphasis on microbiome-mediated mechanisms and the analytical approaches used to characterize metal-microbe interactions through metallomics.



Introduction and Public Health Significance

Cadmium (Cd) exposure represents a significant and increasingly recognized threat to child health and development worldwide. As a non-essential heavy metal with no physiological role in human biology, cadmium poses particular dangers to infants and young children due to their elevated food consumption relative to body weight, immature gastrointestinal systems, and developing organ systems [1]. The vulnerability of the pediatric population extends across multiple organ systems, with documented adverse effects on neurodevelopment, renal function, bone health, and immune competence. Research from the past five years has illuminated previously underappreciated mechanisms through which cadmium compromises child health, particularly through alterations of the gut microbiota and disruption of microbial-derived beneficial metabolites. Understanding these complex interactions is essential for developing effective mitigation strategies and establishing protective public health policies.

The epidemiological landscape reveals that cadmium contamination of the food supply is ubiquitous, with exposure occurring through multiple dietary pathways that vary by geography, food production practices, and cultural dietary patterns. Recent meta-analyses demonstrate that prenatal cadmium exposure leads to measurable reductions in offspring growth parameters, while longitudinal studies reveal persistent effects on cognitive function throughout childhood [2]. The emerging recognition of gut microbiota as a critical mediator between environmental exposures and systemic health outcomes has prompted investigation into how cadmium disrupts these essential microbial communities and their metabolic functions [3]. This comprehensive review synthesizes current evidence on cadmium toxicity in infants and children, with particular emphasis on microbiome-mediated mechanisms and the analytical approaches used to characterize metal-microbe interactions through metallomics.

Sources and Dietary Exposure Pathways in Infants and Young Children

Cadmium exposure in infants and children occurs primarily through dietary consumption, with infant formula and processed baby foods representing the primary contributors for the youngest age groups [1]. Contaminated soils used in agriculture accumulate cadmium from natural geological sources and anthropogenic inputs, leading to uptake by staple crops including rice, wheat, leafy vegetables, and root vegetables. The bioavailability of cadmium in different food matrices varies substantially, influencing the extent to which consumers absorb ingested cadmium across the gastrointestinal barrier.

Infant formula presents a particularly significant exposure route for non-breastfed infants aged 0-12 months, as formula represents a large proportion of total dietary intake during this critical developmental period [4].

Complementary foods introduced around 6 months of age, particularly cereal-based products and vegetable purees, further contribute to cumulative cadmium exposure. Research from the U.S. Food and Drug Administration's Total Diet Study (2018-2020) found that processed baby food and infant formula food groups were major contributors to lead and cadmium exposure in infants 0-11 months, driven primarily by intake volume.

Geographic variation in cadmium exposure reflects differences in soil geochemistry and agricultural practices. Asian countries, particularly Thailand and China, demonstrate significantly higher cadmium intake levels among children compared to Western countries, with some regions showing dietary exposures exceeding international guidance values [1]. The critical role of staple foods means that children consuming rice-based diets face cumulative exposures orders of magnitude higher than those in non-rice-consuming populations.

Breast Milk as an Exposure Route

Although breast milk provides substantial nutritional and immunological benefits, it can serve as a vector for maternal cadmium transmission to nursing infants. Detection rates of cadmium in breast milk are remarkably high, with 99% of samples from Korean mothers containing measurable cadmium concentrations [5]. Maternal dietary patterns significantly influence milk cadmium concentrations, with positive associations observed between cadmium levels and maternal consumption of vegetables and seaweeds, foods known to accumulate cadmium from contaminated soils.

Compared to infants fed formula, breastfed infants may experience exposure up to 12 times higher than formula-fed infants when consuming weaning foods, though this relationship depends on both maternal body burden and the cadmium content of complementary foods [6]. This paradox reflects the bioaccumulative nature of cadmium in maternal tissues; women with accumulated cadmium burden will transfer this reservoir to nursing infants regardless of recent dietary intake. Interventions to reduce maternal dietary cadmium exposure, particularly during pregnancy and lactation, represent an important strategy for reducing early-life exposure.

Molecular and Cellular Mechanisms of Cadmium Toxicity

Cadmium exerts toxicity through multiple overlapping mechanisms, all of which are particularly pronounced in pediatric populations with developing physiological systems. Unlike essential metals that require homeostatic regulation, cadmium has no known biological function and accumulates in tissues with a biological half-life exceeding 15-20 years [7]. At the cellular level, cadmium generates reactive oxygen species, leading to oxidative stress that overwhelms antioxidant defenses; this mechanism appears particularly devastating in developing tissues with immature antioxidant systems. Cadmium directly inhibits critical enzymes by competing with zinc and calcium at enzyme active sites, disrupting energy metabolism, protein synthesis, and DNA damage repair mechanisms.

Epigenetic alterations represent an increasingly recognized mechanism of cadmium-induced developmental toxicity. Prenatal cadmium exposure triggers hypermethylation of the glucocorticoid receptor gene (NR3C1), disrupting development of the hypothalamic-pituitary-adrenal axis and resulting in abnormal cognitive behavior in neonates [8]. Cadmium also upregulates placental expression of miR-509-3p and miR-193-5p, microRNAs implicated in impaired development of the central nervous system. These epigenetic modifications can persist beyond the exposure period, establishing a lasting impact on neurodevelopmental trajectory. The ability of cadmium to cross the blood-brain barrier and placental barriers—due to its physicochemical similarity to divalent cations like zinc and calcium—means that even moderate maternal exposures result in substantial fetal and neonatal exposures.

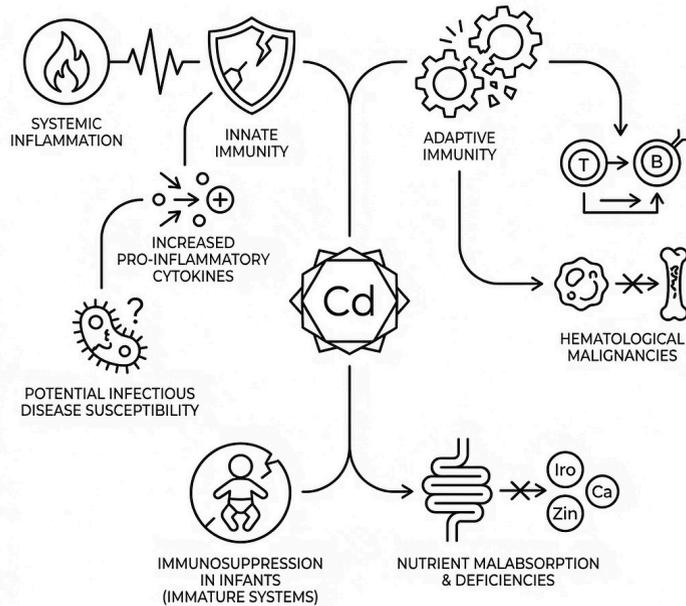
Renal and Metabolic Effects

The kidneys represent the primary target organ for cadmium toxicity, with chronic exposure leading to progressive glomerular and tubular dysfunction that in severe cases progresses to chronic kidney disease (CKD). In children, cadmium exposure induces proteinuria and reduced glomerular filtration rate at exposures below currently accepted thresholds, with emerging evidence suggesting that children may be more susceptible than adults to cadmium-induced renal injury. Cadmium competes with calcium for absorption in the proximal tubule, impairing calcium reabsorption and contributing to hypercalciuria and bone demineralization.

Research on vitamin D metabolism reveals that cadmium exposure negatively correlates with circulating 25-OH vitamin D3 concentrations, with children exposed to high blood cadmium levels showing **23% reductions in vitamin D levels** [7]. This disruption of vitamin D synthesis represents a particularly concerning mechanism in pediatric populations, as vitamin D plays essential roles in calcium homeostasis, immune function, and neurological development. The combination of cadmium-induced renal dysfunction and vitamin D deficiency creates a synergistic threat to bone health and immune competence in exposed children.

Immune Dysfunction and Inflammatory Responses

Cadmium exposure dysregulates both innate and adaptive immune responses through multiple mechanisms. Exposure to elevated cadmium concentrations induces systemic inflammation, increasing blood concentrations of pro-inflammatory cytokines. This inflammatory microenvironment may predispose to infectious disease susceptibility and potentially increase risk of hematological malignancies. The immunosuppressive effects of cadmium are particularly concerning in infants with immature immune systems still establishing tolerance and developing protective responses to environmental antigens. Additionally, cadmium-induced disruption of nutrient absorption may exacerbate immune deficiencies through secondary zinc, selenium, or vitamin D depletion.

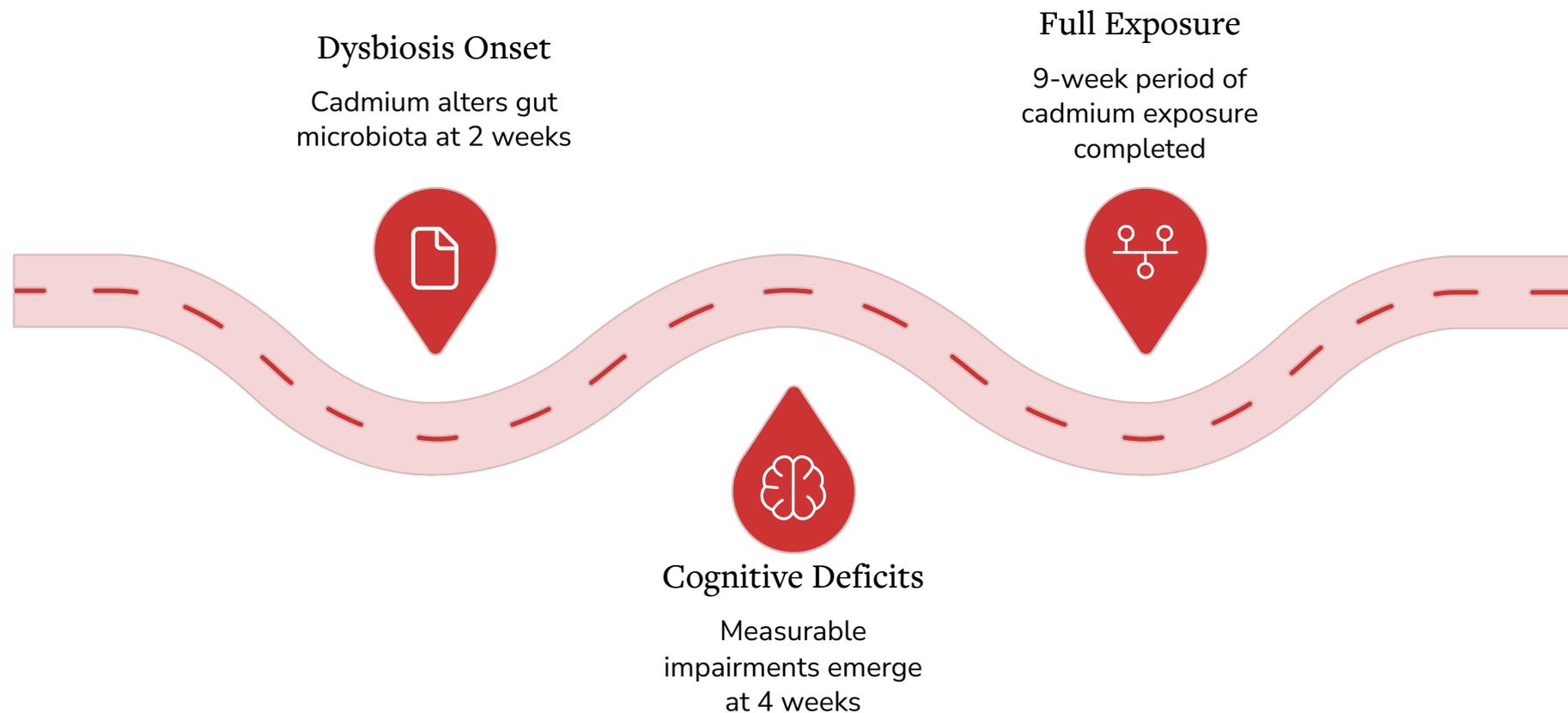


Neurodevelopmental and Cognitive Effects

Among the most significant health impacts of cadmium exposure in children is impaired neurodevelopment and cognitive dysfunction. A meta-analysis of prenatal and early childhood cadmium exposure on neurodevelopment in 17 observational studies involving 6,907 participants found that a 50% increase in cadmium exposure resulted in a 0.44-point drop in Full-Scale Intelligence Quotient (FSIQ) in children aged 5-9 years, with these effects showing remarkable consistency across studies [9]. This reduction in cognitive performance has profound implications for educational achievement, socioeconomic outcomes, and lifetime productivity in affected children.

The mechanisms underlying cadmium-induced neurotoxicity involve multiple pathways. Prenatal cadmium exposure alters gene expression in the hippocampus and other brain regions critical for learning and memory formation [10]. Importantly, recent research demonstrates that cadmium-induced cognitive deficits are preceded by alterations in gut microbiota composition and function, suggesting that microbiota disruption represents an early mechanistic step in the pathway to neurocognitive impairment. Cadmium exposure induces hippocampus-dependent learning deficits in animal models, with these cognitive changes following changes in fecal microbiota composition identified through metagenomic analysis.

Temporal Relationship Between Dysbiosis and Cognitive Decline



Longitudinal studies in rodent models demonstrate that gut dysbiosis precedes the onset of measurable cognitive deficits by approximately 4 weeks of exposure, suggesting that microbiota alterations represent an initiating event rather than a consequence of neural dysfunction [10]. Exposure of adult male mice to 3 mg/L cadmium via drinking water for nine weeks induced progressive gut dysbiosis within the first 2 weeks, followed by cognitive impairment detected at 4 weeks into exposure. This temporal sequence supports a microbiota-mediated mechanism, with specific bacterial species identified that correlate with cognitive deficits.

The disruption of short-chain fatty acid (SCFA)-producing bacteria represents a key mechanistic link between cadmium exposure and cognitive impairment. Reduced butyrate production following cadmium exposure compromises intestinal barrier integrity, leading to increased intestinal permeability ("leaky gut"), bacterial lipopolysaccharide (LPS) translocation, and systemic inflammation. Elevated circulating LPS and pro-inflammatory cytokines cross the blood-brain barrier, inducing neuroinflammation that impairs synaptic plasticity and cognitive function. These findings underscore the importance of the gut-brain axis as a target organ system for cadmium toxicity.

Growth and Anthropometric Effects

Meta-analytic evidence demonstrates that cadmium exposure during pregnancy and early childhood is associated with reduced growth parameters, including decrements in height, weight, and body mass index z-scores [2]. Among 6,181 participants across 15 studies, cadmium exposure was inversely linked to child height and weight-for-age z-scores. These associations persisted in prospective cohort studies and were specifically supported by evidence from urinary cadmium assessments, suggesting dose-response relationships.

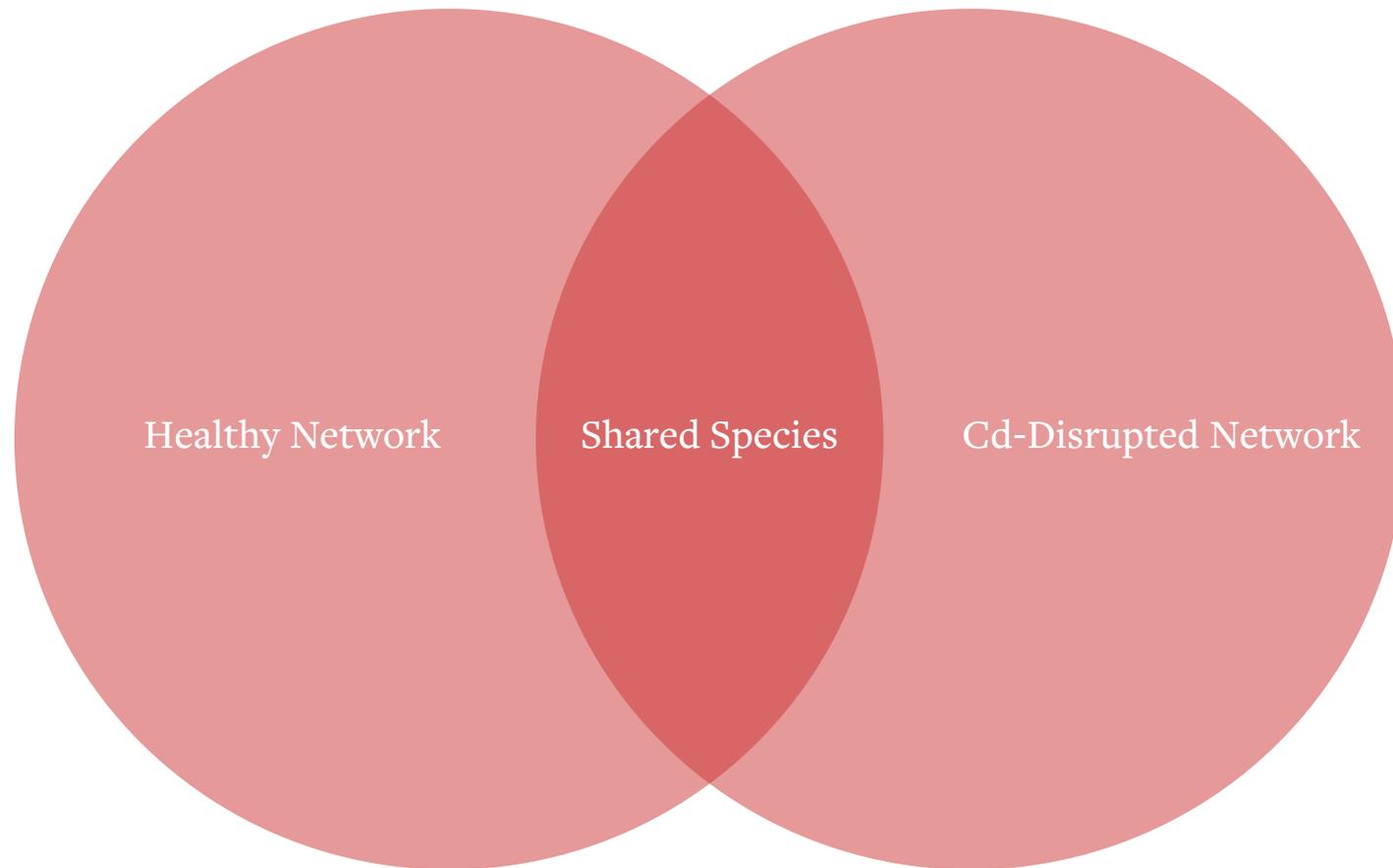
Prenatal cadmium exposure demonstrates sex-specific effects on birth outcomes, with female infants showing greater vulnerability to growth restriction [11]. A Korean study of 5,215 women found that maternal cadmium concentrations during early and late pregnancy were significantly associated with low birth weight, with the strongest associations observed for female infants. The mechanisms underlying cadmium-induced growth restriction involve multiple pathways: direct effects on placental development and function, interference with nutrient absorption and metabolism, and disruption of endocrine systems regulating growth. Placental cadmium accumulation may impair placental nutrient transport capacity, while simultaneous disruption of zinc and calcium absorption exacerbates nutritional deficiencies critical for growth.

Compositional and Functional Alterations in the Gut Microbiota

The discovery that cadmium toxicity is mediated through alterations of the gut microbiota represents a paradigm shift in understanding heavy metal health effects. Recent *in vitro* studies of human fecal microbiota exposed to cadmium reveal marked compositional and functional changes, with dramatic shifts in short-chain fatty acid production [3]. Importantly, the magnitude and direction of these changes depend on the baseline microbiota composition, with some communities classified as "sensitive" (showing dramatic dysbiosis and SCFA reduction) and others as "resilient" (maintaining relatively stable function).

In sensitive microbiota communities exposed to cadmium, butyrate production decreased significantly while acetate and lactate production increased, indicating an alteration in fermentation pathways and the loss of metabolically specialized anaerobic bacteria. The genus *Anaerostipes*, a primary butyrate producer, showed the most striking compositional changes between sensitive and resilient communities, with a 7.15 centered-log-ratio difference between resistant and susceptible individuals. Resilient communities maintained beneficial short-chain fatty acid producers, or alternatively shifted to *Anaerostipes*-dominated communities that maintained butyrate production capacity.

Bacterial Network Disruption and Species-Specific Vulnerabilities



Network analysis of microbial communities exposed to cadmium reveals substantial disruption of inter-bacterial interactions and cooperative metabolic relationships [3]. Sensitive microbiota exhibited significantly greater disruption of microbial network structure compared to resilient communities, with loss of cooperative interactions that normally support the growth of fastidious anaerobes and preserve community stability. The loss of these positive interactions may explain why dysbiotic communities show reduced resilience and increased susceptibility to secondary perturbations.

Different bacterial taxa show remarkable variation in their susceptibility to cadmium toxicity, reflecting differences in metal tolerance mechanisms and metabolic strategies. Taxa such as Bacteroidetes and Firmicutes (particularly the family Lachnospiraceae) are depleted in cadmium-exposed microbiota, while opportunistic pathogens such as Proteobacteria may be enriched [10]. The loss of Firmicutes is particularly consequential because this phylum includes many of the SCFA producers essential for intestinal barrier health and immune homeostasis.

Cadmium exposure reduces gene expression for nutrient uptake and metabolism in the intestinal epithelium and resident microbiota. Transcriptomic analysis of mouse intestines following cadmium exposure reveals reduced expression of genes involved in intestinal barrier integrity, including tight junction proteins and mucus-layer components. Simultaneously, cadmium exposure increases the expression of pro-inflammatory cytokines and reduces the expression of antimicrobial peptides that normally shape the microbiota composition.

Microbiota Alterations in Agricultural Soil Ecosystems

Emerging evidence suggests that cadmium-driven microbiota alterations extend beyond the human microbiome to affect agricultural and environmental microbiota. Climate-driven changes in soil pH and temperature interact with cadmium contamination to alter soil microbiota structure [12]. In soils with pH below 7, climate change combined with cadmium contamination results in increased porewater cadmium availability and altered microbiota composition, with reduced microbial activity and nutrient cycling. These soil-level changes have implications for food crop contamination, as altered soil microbiota may affect cadmium uptake by plants and the nutrient status of crops affecting trace element bioavailability to consumers.

The ability of soil microbiota to tolerate or remediate cadmium contamination depends on microbial metal tolerance mechanisms and the presence of genetically distinct metal-tolerant lineages [13]. Cadmium-tolerant bacteria can be isolated from contaminated sites and possess high metal-binding capacity, offering potential for bioremediation approaches. However, the selective pressure imposed by chronic cadmium exposure may reduce overall microbial diversity and metabolic potential in heavily contaminated soils, potentially affecting soil ecosystem functions and food crop production.

Metallomics as a Systems-Level Approach

Metallomics—the comprehensive analysis of all metals and their metal-dependent molecules in biological systems—provides a powerful framework for understanding how cadmium disrupts microbiota function and contributes to health impairment [14]. Unlike traditional approaches that measure only bulk cadmium concentrations, metallomics reveals the speciation, localization, and functional consequences of metal interactions with microbial proteins, metabolites, and structural components. This systems-level perspective has illuminated previously unknown mechanisms through which cadmium toxicity propagates from the cellular to the systemic level.

In human infants and children, metallomics analysis reveals substantial differences in the accumulation of toxic metals. Zinc, a structural component of many metalloenzymes and a critical regulator of immune function, competes with cadmium for absorption and transport, potentially limiting cadmium bioavailability. Studies demonstrating inverse relationships between zinc status and cadmium body burden support zinc supplementation as a potential mitigation strategy.

Metal Speciation and Bacterial Tolerance Mechanisms

Metal Speciation

Understanding the chemical speciation of cadmium and other metals in the intestinal microbiota is essential for predicting bioavailability and absorption. The same total metal concentration can exist in multiple chemical forms—including free ions, protein-metal complexes, polysaccharide-bound forms, and mineral phases—each with different bioavailability profiles.

Bacterial Efflux Pumps

Bacteria have evolved multiple mechanisms for surviving in metal-contaminated environments, including active efflux pumps that use ATP hydrolysis to pump metals out of cells, protecting sensitive cellular targets. The Cad operon in some bacteria encodes cadmium-specific efflux systems [15].

Intracellular Sequestration

Cadmium-tolerant bacteria accumulate metal into precipitated deposits or bound forms within intracellular organelles, preventing interaction with sensitive proteins and DNA. Biofilm formation offers population-level protection through extracellular matrix components.

In fecal samples from humans, cadmium speciation analysis demonstrates that the majority is bound to organic or mineral phases. The composition of the microbiota influences speciation patterns; dysbiotic communities with reduced EPS-producing bacteria may show greater proportions of bioavailable cadmium, explaining why dysbiosis-prone individuals show higher absorption and body burdens. This mechanism represents a key link between microbiota composition and cadmium toxicity: dysbiosis increases cadmium bioavailability through altered metal speciation.

Metalloproteomics and Microbial Biosorption

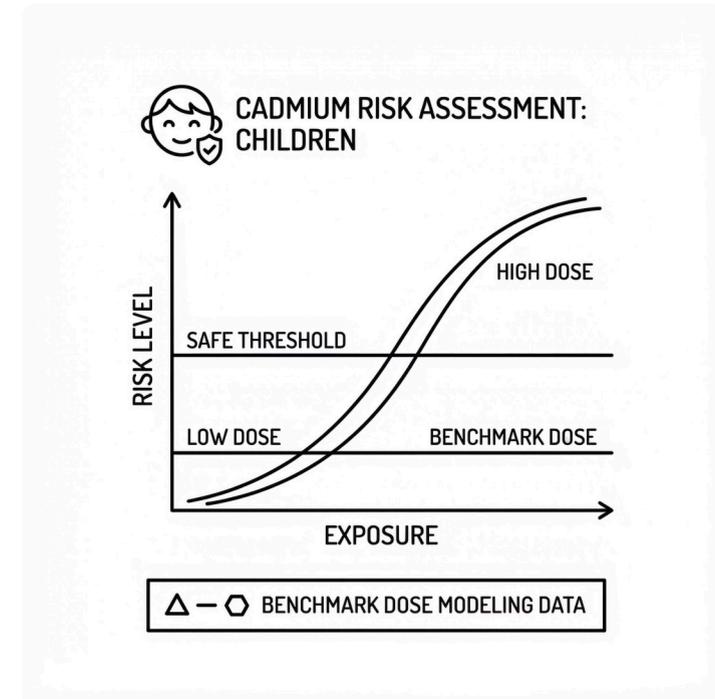
Metalloproteomics—the characterization of all metal-bound proteins in biological systems—reveals how cadmium perturbs the metalloenzyme complement of dysbiotic microbiota. Cadmium can displace essential metals from metalloenzymes or disrupt metalloprotein function through conformational changes or oxidative modifications. For example, cadmium displacement of zinc from zinc-finger domains in transcription factors disrupts gene expression, while cadmium incorporation into metallothionein and phytochelatin molecules redirects these chelation systems away from essential metal homeostasis.

The loss of specific metalloenzymes in cadmium-exposed microbiota has functional consequences for community metabolism. Enzymes containing molybdenum or tungsten at their catalytic centers are particularly sensitive to cadmium toxicity, potentially explaining the selective depletion of certain anaerobic bacteria in dysbiotic communities. Research using coupled analytical techniques including ICP-MS and proteomics reveals that cadmium toxicity in bacteria cannot be predicted from bulk metal accumulation alone but rather depends on the specific interaction of cadmium with catalytic center metals and protein-binding sites.

Microbial biosorption of cadmium—the binding of metal to cell surface functional groups—occurs passively and represents a key mechanism through which microbiota composition influences fecal cadmium levels and absorption. Bacterial cell walls contain carboxyl groups on peptidoglycan, phosphoryl groups on teichoic acids, and amino groups on proteins, all of which can chelate cadmium. The magnitude of biosorption depends on cell wall composition, with Gram-positive bacteria showing greater biosorption capacity than Gram-negative bacteria due to higher teichoic acid content.

Current Risk Assessment Approaches and Limitations

Contemporary risk assessment for dietary cadmium exposure typically employs the Provisional Tolerable Weekly Intake (PTWI) of $7 \mu\text{g}/\text{kg}$ body weight established by the Joint FAO/WHO Expert Committee on Food Additives. However, emerging evidence suggests that this threshold may be insufficiently protective, particularly for developing children. Recent benchmark dose (BMD) modeling studies recommend adoption of exposure guidelines below $0.20 \mu\text{g}/\text{g}$ creatinine in urinary cadmium excretion to protect renal function, substantially lower than current thresholds. For children, the margin of safety may be further reduced, as pediatric populations show greater susceptibility to cadmium effects on growth and cognition at given exposure levels.



Probabilistic risk assessment approaches employing Monte Carlo simulation provide more realistic estimates of population exposure than single-point estimates, revealing that vulnerable subpopulations such as young children and vegetarians often exceed health-based guidance values. These approaches account for natural variation in food consumption patterns, contaminant levels, and inter-individual differences in absorption and metabolism. Meta-analyses of dietary cadmium exposure in multiple countries document that **30% or more of young children exceed the revised health-based guidance value**, indicating a widespread public health concern.

Dietary Interventions and Nutrient-Based Mitigation



Zinc Supplementation

Zinc, a structural component of many metalloenzymes and a critical regulator of immune function, competes with cadmium for absorption and transport, potentially limiting cadmium bioavailability. Studies demonstrating inverse relationships between zinc status and cadmium body burden support zinc supplementation as a potential mitigation strategy.



Selenium Protection

Selenium, an essential element for selenoprotein synthesis including glutathione peroxidase and thioredoxin reductase, acts as an antioxidant opposing cadmium-induced oxidative stress. Animal studies demonstrate that selenium supplementation partially antagonizes microbiota alterations caused by cadmium exposure [16].



Calcium and Fiber

Calcium supplementation may reduce cadmium absorption through competing for intestinal transporters, while dietary fiber, particularly soluble fiber fractions, may bind cadmium in the intestinal lumen and reduce net absorption [1].



Vitamin D Sufficiency

Vitamin D sufficiency appears important for optimal immune function and bone health in the context of cadmium exposure, though vitamin D supplementation has not been systematically evaluated as a mitigation strategy in cadmium-exposed children.

Importantly, comprehensive multi-omics analysis including metabolomics, metabolomics, and metataxonomics reveals that selenium-mediated mitigation operates through complex interactions with the microbiota, suggesting that selenium effects are not solely through direct antioxidant mechanisms but rather through promotion of cadmium-tolerant microbial communities [17].

Regulatory Approaches and Microbiota-Targeted Interventions

Regulatory and Policy Approaches

Reducing cadmium contamination of the food supply represents the most direct public health intervention. The European Food Safety Authority and other regulatory agencies have established maximum allowable cadmium concentrations in various foods, with particularly stringent limits for infant formula and baby food products [1]. However, implementation of these limits requires coordination among multiple stakeholders, including farmers, food processors, and regulatory agencies, as well as reduction of cadmium in environmental media (soil and water).

Selective breeding or genetic improvement of crop varieties for reduced cadmium accumulation represents a promising long-term strategy. Some crop varieties naturally accumulate less cadmium from soils, suggesting that agronomic selection for these traits could substantially reduce dietary cadmium exposure. Public health messaging regarding high-risk foods, particularly for vulnerable populations including pregnant women, infants, and young children, could reduce dietary cadmium exposure. Particular attention should be directed toward high-accumulating foods including certain mushroom species, leafy green vegetables, and rice products. For mothers of very young children, reducing cadmium exposure represents both a personal health benefit and a means of reducing lactational transfer of cadmium to nursing infants.

Microbiota-Targeted Interventions

The emerging understanding of cadmium toxicity as mediated through microbiota disruption opens novel therapeutic avenues. Probiotics or specific bacterial strains showing cadmium tolerance and butyrate production capacity could be administered to support recovery of dysbiotic microbiota or prevent dysbiosis in cadmium-exposed individuals [13]. However, this approach requires careful strain selection and evaluation of efficacy in clinical populations, as many proposed probiotics have not been rigorously tested in human subjects.

Prebiotics—dietary components selectively promoting beneficial bacteria—including inulin, fructooligosaccharides, and resistant starch, enhance the growth of butyrate producers and may provide protection against cadmium-induced dysbiosis [3]. These dietary fibers could be incorporated into foods targeted at children, though public health impact would require evaluation through randomized controlled trials. Importantly, the efficacy of microbiota-targeted interventions will likely depend on whether dysbiosis has already induced pathological changes in the intestinal barrier and systemic immune system; early intervention may be substantially more effective than treatment following established dysbiosis.

Monitoring Strategies and Conclusions

Biomonitoring Strategies

Urinary cadmium excretion has emerged as a preferred biomonitoring indicator for assessing population-level cadmium exposure and evaluating the effectiveness of reduction strategies. For children, biomonitoring programs can identify vulnerable populations with elevated cadmium body burdens and direct preventive interventions. However, current biomonitoring thresholds may require downward revision based on recent BMD modeling data demonstrating adverse health effects below current reference values.

Metalomics analysis including comprehensive characterization of trace element and toxic metal tissue levels provides valuable research data for understanding population-level metal exposures and interactions. However, current methods remain expensive and are impractical for routine clinical use. Specialized metalomics research in pediatric populations exposed to cadmium and other heavy metals could identify novel biomarkers of vulnerability or protective phenotypes, potentially informing personalized prevention approaches.



Conclusion

Cadmium exposure in infants and children represents a significant and globally distributed environmental health threat with particular impacts on neurodevelopment, growth, and immune function. The mechanisms underlying these health effects extend beyond direct cadmium toxicity to cells and organs to include profound alterations of the gut microbiota, the intestinal barrier, and systemic inflammation. Recent advances in metallomics and microbiota analysis have illuminated previously unknown interactions between cadmium, bacterial communities, and essential metal homeostasis, establishing the microbiota as a critical mediator of cadmium toxicity.

Public health responses should prioritize reduction of cadmium contamination in the food supply, particularly in infant formula and baby food products, through agronomic, technological, and regulatory approaches. Simultaneously, efforts to understand inter-individual vulnerability to cadmium effects—including genetic, immunological, and microbiota factors—may enable personalized prevention strategies targeting high-risk populations. Future research integrating metallomics, microbiota analysis, and mechanistic toxicology will further elucidate the complex interactions determining cadmium effects in pediatric populations and inform evidence-based prevention and treatment strategies.

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