Arsenic Effects in Infants and Children: A Comprehensive Review of Developmental Toxicity, Mismetallation, Microbiome Disruption, and Microbial Metallomics

This comprehensive review examines the profound and multifaceted impacts of arsenic exposure on infants and children, exploring developmental toxicity, molecular mechanisms of mismetallation, microbiome disruption, and the emerging field of microbial metallomics.

Arsenic Exposure Pathways and Early-Life Vulnerability

Sources and Routes of Prenatal and Early Childhood Exposure

Arsenic contamination represents one of the most significant global public health threats, with **over 200 million people exposed to elevated arsenic concentrations**, primarily through contaminated drinking water [1]. Infants and young children face particular vulnerability due to their early developmental windows and enhanced toxicological sensitivity. Maternal-fetal arsenic transfer occurs readily across the placenta, with cord blood arsenic concentrations reaching levels nearly as high as maternal exposure [2]. The median placental arsenic concentration in exposed populations can reach $34 \mu g/kg$ compared to $7 \mu g/kg$ in non-exposed individuals, indicating substantial placental accumulation [2].

Geographic disparities in exposure are pronounced, with infants in **Bangladesh, Pakistan, and India** exposed to dietary arsenic levels significantly exceeding health reference values of $0.3\text{-}8 \mu g/kg/day$ [1]. Early childhood exposure through breast milk presents an additional route of transmission, with breast milk arsenic levels varying considerably based on maternal dietary intake and environmental exposure [3]. The metabolic handling of arsenic differs markedly in early development compared to adults, with methylation processes showing enhanced activity during pregnancy and early infancy, converting inorganic arsenic to less immediately toxic dimethylarsinic acid (DMA) [2].

Critical Developmental Windows

The critical developmental windows for arsenic toxicity in infants and children extend from fetal life through early childhood. Prenatal exposure during late gestation facilitates efficient transplacental transfer, and the newborn period represents an especially vulnerable stage for neurological and immune system development [4]. Studies demonstrate that arsenic exposure affects infants from birth through 12 years of age, with **cognition being the most frequently evaluated neuropsychological domain (94.8% of studies)**, followed by psychomotor function (40.3%) and social-emotional function (29.9%) [4].

01

In-Utero Exposure

Mid-to-late pregnancy exposure influences brain growth during critical neurodevelopmental periods

02

Newborn Period

Especially vulnerable stage for neurological and immune system development

03

Early Childhood

Continued vulnerability through age 12, with persistent cognitive effects

In-utero exposure to arsenic, particularly during mid-to-late pregnancy, influences brain growth during critical neurodevelopmental periods. Arsenic exposure is associated with **reduced head circumference in infants at 1-3 months of age**, suggesting diminished brain size development [5]. The effect size demonstrates dose-dependent relationships, with a one standard deviation increase in in-utero arsenic exposure corresponding to decrements in developmental indices [6]. Sex-specific vulnerabilities have also been identified, with arsenic exposure showing adverse effects on IQ in girls but not boys at 5 years of age, suggesting differential developmental susceptibilities [7].

Cognitive Impairment and Neuropsychological Deficits

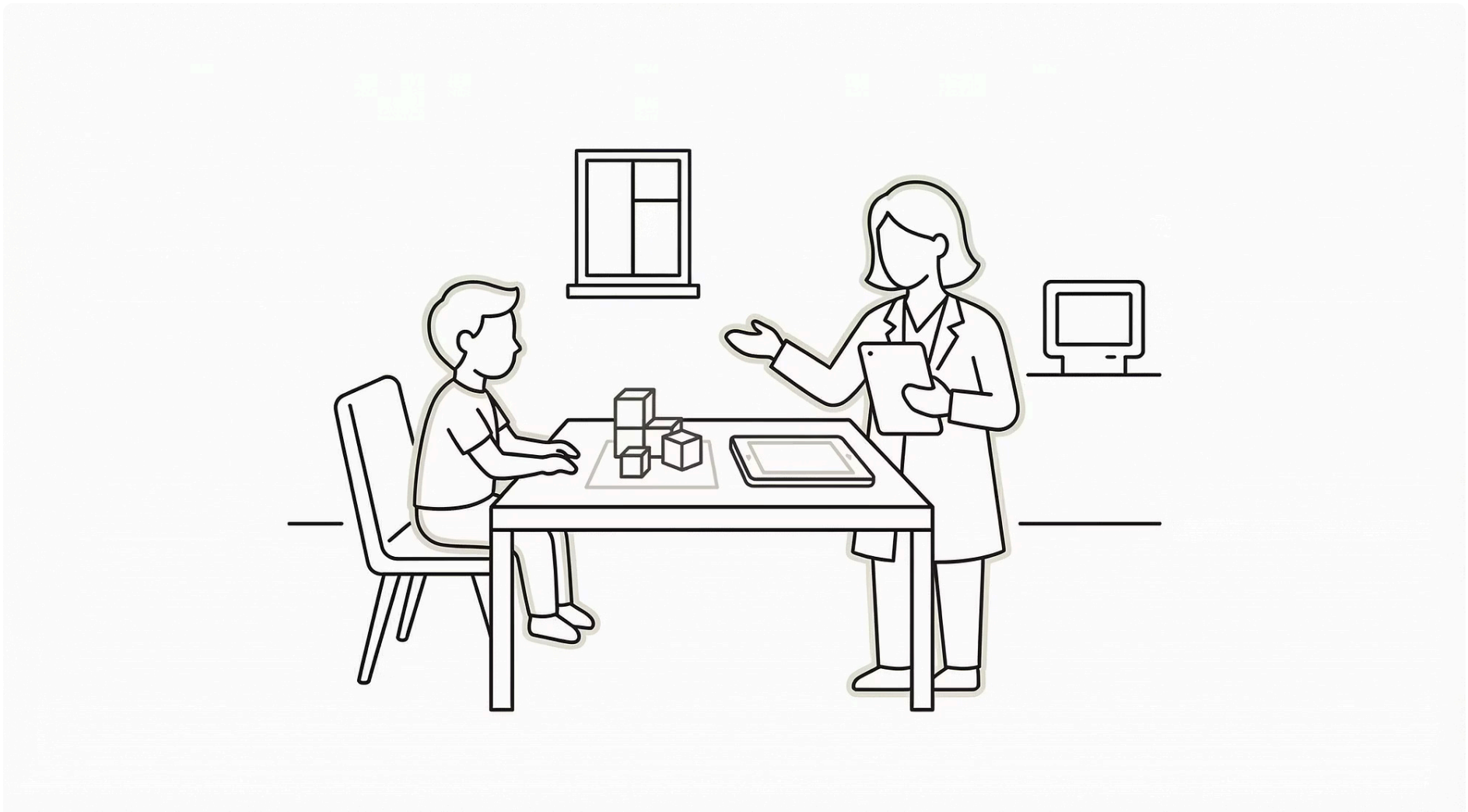
The most extensively documented consequence of childhood arsenic exposure is **cognitive impairment**. A systematic review analyzing 24 studies demonstrated a consistent inverse relationship between arsenic exposure and cognitive performance in children, with higher arsenic levels associated with lower IQ scores, slower processing speeds, and impaired memory and language skills [8]. These cognitive deficits were evident across diverse geographical regions and persisted even after adjusting for sociodemographic factors.

Mental Development Index (MDI)

Meta-analysis of 17 observational studies involving 6,907 participants found that a **50% increase in prenatal arsenic exposure** was associated with reductions of 0.51 points in the Mental Development Index (MDI) and 0.15 points in the Psychomotor Development Index (PDI), though these effects did not achieve statistical significance across all populations [6].

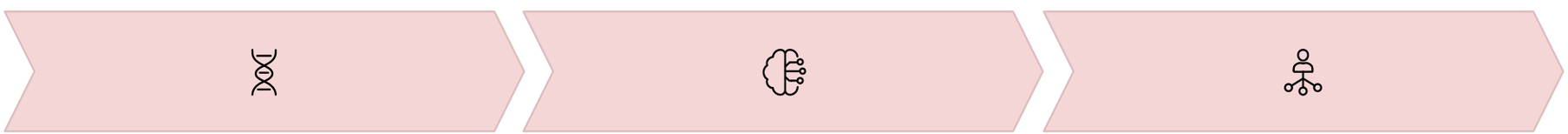
Measurement Challenges

This apparent paradox may reflect heterogeneity in exposure measurement methods, with studies measuring total urinary arsenic without species differentiation potentially underestimating inorganic arsenic toxicity.



Neurodevelopmental Mechanisms and Molecular Pathways

Developmental arsenic exposure triggers molecular pathways that fundamentally alter brain maturation. In a maternal arsenic exposure model in mice, time-series RNA sequencing revealed significant temporal correlation between arsenic neurodevelopmental toxicity and altered hippocampal mRNA expression profiles during critical postnatal developmental windows [9]. **Downregulation of genes associated with neurogenesis and telomere maintenance** was observed, with critically shortened telomeres inhibiting neural stem cell proliferation, impairing neuroblast maturation, and reducing neurosphere numbers and sizes.



Gene Downregulation

Neurogenesis and telomere maintenance genes affected

Structural Changes

Impaired dendritic complexity and altered spine morphology

Synaptic Disruption

Altered synaptic ultrastructure leading to cognitive impairment

Arsenic exposure impaired dendritic complexity in the hippocampus, altered dendritic spine morphology, and disrupted synaptic ultrastructure, ultimately leading to cognitive impairment [9]. These structural alterations in neural tissue represent **permanent modifications of brain architecture** during critical windows of vulnerability. The neural stem cell population exhibited decreased stemness markers (BrdU+ and MAP2+ cells were reduced while GFAP+ cells were increased), indicating a fundamental shift in neural progenitor cell behavior that would compromise long-term cognitive capacity.

Multiplex Metal Exposure Effects and Synergism

Most environmental exposures involve multiple heavy metals simultaneously, with arsenic co-occurring with lead, mercury, cadmium, and other metalloids. In an artisanal gold mining region of Tanzania, prenatal multi-chemical exposure to lead, mercury, cadmium, and arsenic showed **synergistic effects on developmental outcomes** [10].

17.78%

Gross Motor Skills

Decrease from joint metal exposure

55.36%

Language Ability

Reduction in language development

13.36%

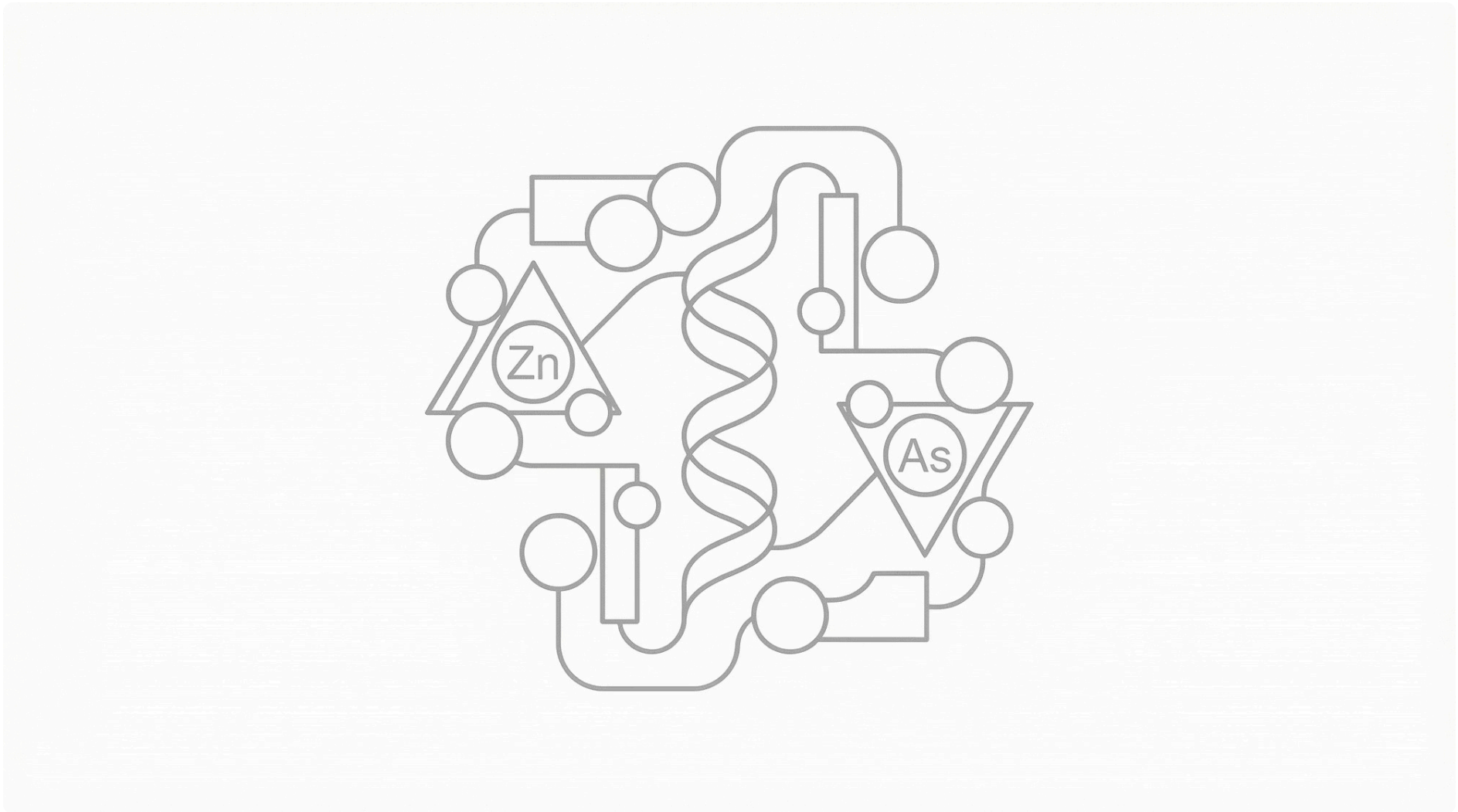
General Development

Decrease in developmental milestones

Joint metal exposure decreased gross motor skills by 17.78%, language ability by 55.36%, and general developmental milestones by 13.36%, with the combined effect greater than would be expected from additive exposure models. **Lead amplified the developmental toxicity of cadmium and arsenic**, suggesting that co-occurring toxic metals potentiate their individual developmental effects beyond simple additive toxicity.

Arsenic-Induced Zinc Finger Protein Mismetallation

A central molecular mechanism of arsenic toxicity involves **displacement of zinc ions from zinc finger proteins** and other metalloproteins, causing protein misfolding, loss of function, and cellular dysfunction. Arsenite (As3+) selectively binds to cysteine-rich zinc finger motifs with C3H1 and C4 configurations, displacing the coordinated zinc ion [11]. This binding is not a simple replacement but rather induces conformational changes in the zinc finger domain structure.



Arsenite exposure in human keratinocytes impaired the splicing function of ZRANB2, an alternative splicing regulator protein containing two C4 zinc finger motifs essential to its structure [11]. Within 3-24 hours of arsenic exposure, **ZRANB2-dependent splicing of target mRNA was impaired**, demonstrating functional consequences of zinc displacement.

The mismetallation mechanism extends beyond simple zinc loss. Chemoproteomic approaches using biotin-As(III) probes identified multiple nuclear arsenite-binding proteins involved in mRNA splicing, DNA repair, and replication [12]. As3+ binding to splicing factor 1 (SF1) perturbed mRNA splicing in human cells, indicating that mismetallation of splicing machinery disrupts this essential RNA processing function. The selectivity of arsenite for C3H1 and C4 zinc fingers versus C2H2 zinc fingers suggests that not all zinc proteins are equally affected; rather, arsenite targets a specific subset of zinc finger configurations based on cysteine spacing and coordination geometry.

Arsenic-Induced Protein Aggregation and Proteotoxic Stress

Beyond direct zinc displacement, arsenic exposure induces **protein aggregation through mismetallation-dependent mechanisms**. In bacterial systems, soft metalloids including arsenic promote protein aggregation both during translation and post-translationally, disrupting multiple essential biological processes [13]. The aggregated proteins are involved in amino acid biosynthesis, energy metabolism, and other critical functions, with accumulation of aggregates directly linked to cell death.

Protein Aggregation

Arsenic coordinates with cysteine residues, causing proteins to misfold and aggregate

Proteotoxic Stress

Upregulation of chaperones and proteasomal degradation creates metabolic burden

Cell Death

Overwhelmed proteostatic capacity leads to cellular dysfunction and death

In human systems, arsenic's interaction with cysteine and histidine-containing proteins initiates aggregation cascades. The aggregation occurs preferentially in proteins with multiple cysteine residues that are susceptible to arsenic coordination. This proteotoxic stress activates cellular quality control mechanisms, including upregulation of molecular chaperones and proteasomal degradation pathways, representing a metabolic burden on developing tissues already stressed by arsenic exposure [14]. In cells lacking adequate chaperone systems, **arsenic-induced protein aggregation overwhelms proteostatic capacity**, leading to cell death.

Epigenetic Modifications and Gene Regulation Through Mismetallation

Arsenic exposure modulates gene expression through **epigenetic mechanisms involving metal-responsive transcription factors**. The metal-activated transcription factor 1 (MTF1) mediates induction of metallothionein genes in response to arsenic binding [15]. Arsenic binds to a C-terminal cysteine cluster of MTF1, activating its transcriptional activity and upregulating genes involved in metal homeostasis and detoxification. However, this compensatory response has a metabolic cost, diverting cellular resources from normal developmental processes.

Histone Modification

Arsenic exposure reduces global histone H4 acetylation at lysine 16 through direct binding to histone acetyltransferase hMOF, inhibiting its catalytic activity [16]. This epigenetic modification alters chromatin structure and gene expression patterns across multiple genes, potentially affecting genes essential for neurodevelopment and immune function.

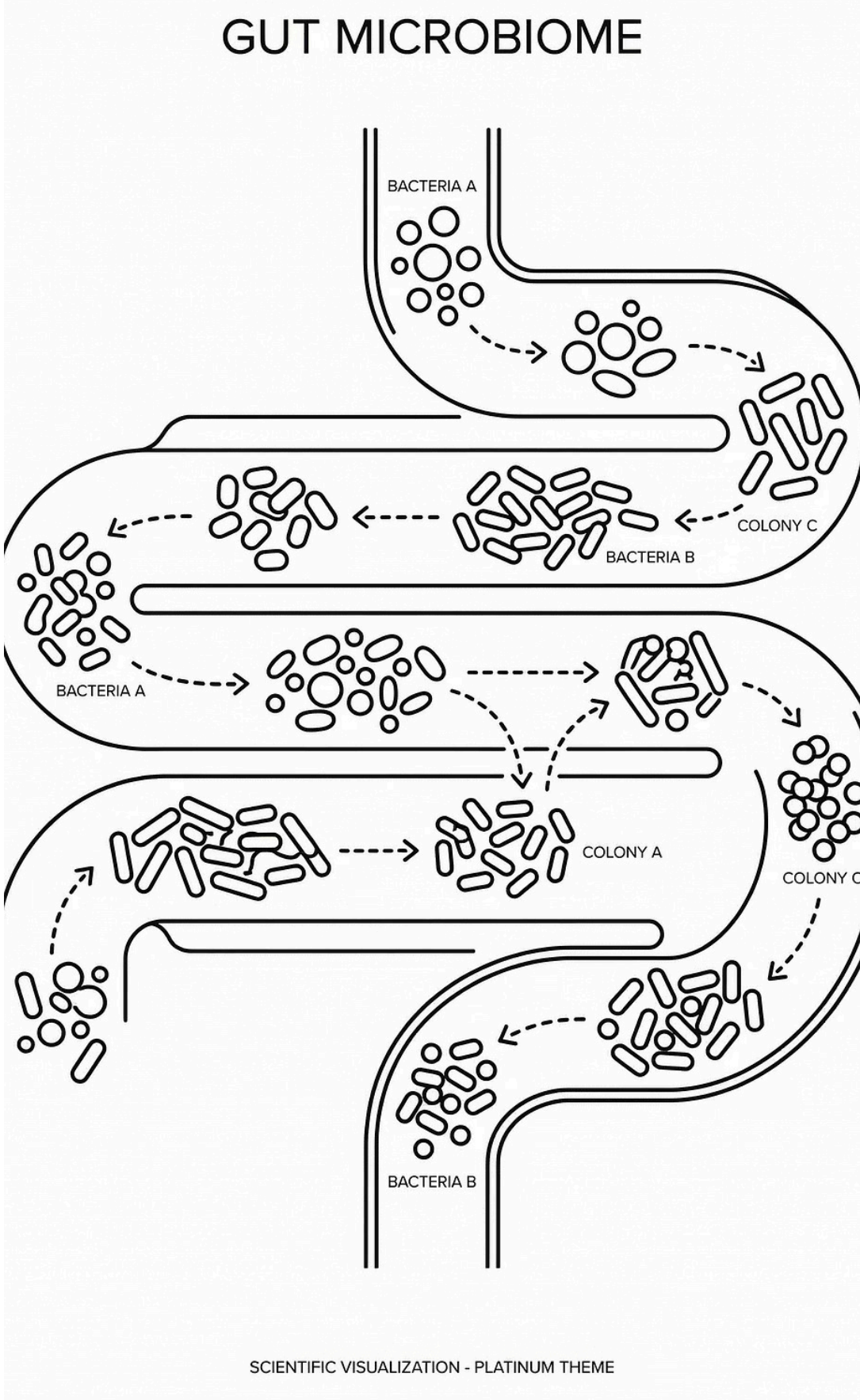
Long-Term Effects

The altered methylation patterns persist beyond the exposure period, suggesting **long-term epigenetic dysregulation** from developmental arsenic exposure [17].

Microbiome Composition Shifts in Response to Arsenic Exposure

Arsenic exposure profoundly alters microbial community composition in infant and child gut microbiota. Prenatal arsenic exposure in murine models altered the fecal microbiome composition, with **significant decreases in Firmicutes abundance** in arsenic-exposed offspring [18]. Functional analysis revealed that arsenic exposure shifted genes involved in crucial metabolic pathways such as insulin signaling and non-alcoholic fatty liver disease pathways, suggesting that arsenic-induced microbiome dysbiosis could predispose to metabolic disease.

In human populations exposed to arsenic through contaminated drinking water, the microbiota exhibits characteristic dysbiotic shifts. Food chain microbiome studies examining responses to arsenic across multiple ecosystem components revealed that chemical stressors, including arsenic, decreased microbiome diversity in soil but caused compositionally distinctive shifts in water, sediment, plant, and animal microbiomes [19]. The dysbiotic communities became compositionally more similar to each other in response to stress, suggesting convergence toward a stress-selected community composition. Importantly, different bacterial taxa responded specifically to arsenic exposure, with stochastic effects particularly notable in host-associated communities.



Functional Consequences of Microbiome Dysbiosis

Beyond compositional changes, arsenic-induced microbiota dysbiosis impairs critical bacterial metabolic functions. In arsenic-contaminated groundwater systems, distinct microbial communities enriched in response to varying electron acceptor regimes included arsenite-oxidizing bacteria (*Deinococcus*), nitrate-reducing bacteria (*Denitratisoma*), sulfate-reducing bacteria (*Macellibacteroides*), and other functional groups [20]. However, the metabolic plasticity of these communities was constrained by arsenic stress, leading to **functional narrowing**.

Xenobiotic Metabolism

Heavy metal exposure alters functional genes related to xenobiotic metabolism and amino acid biosynthesis, reducing the microbiota's capacity to synthesize essential metabolites [21].

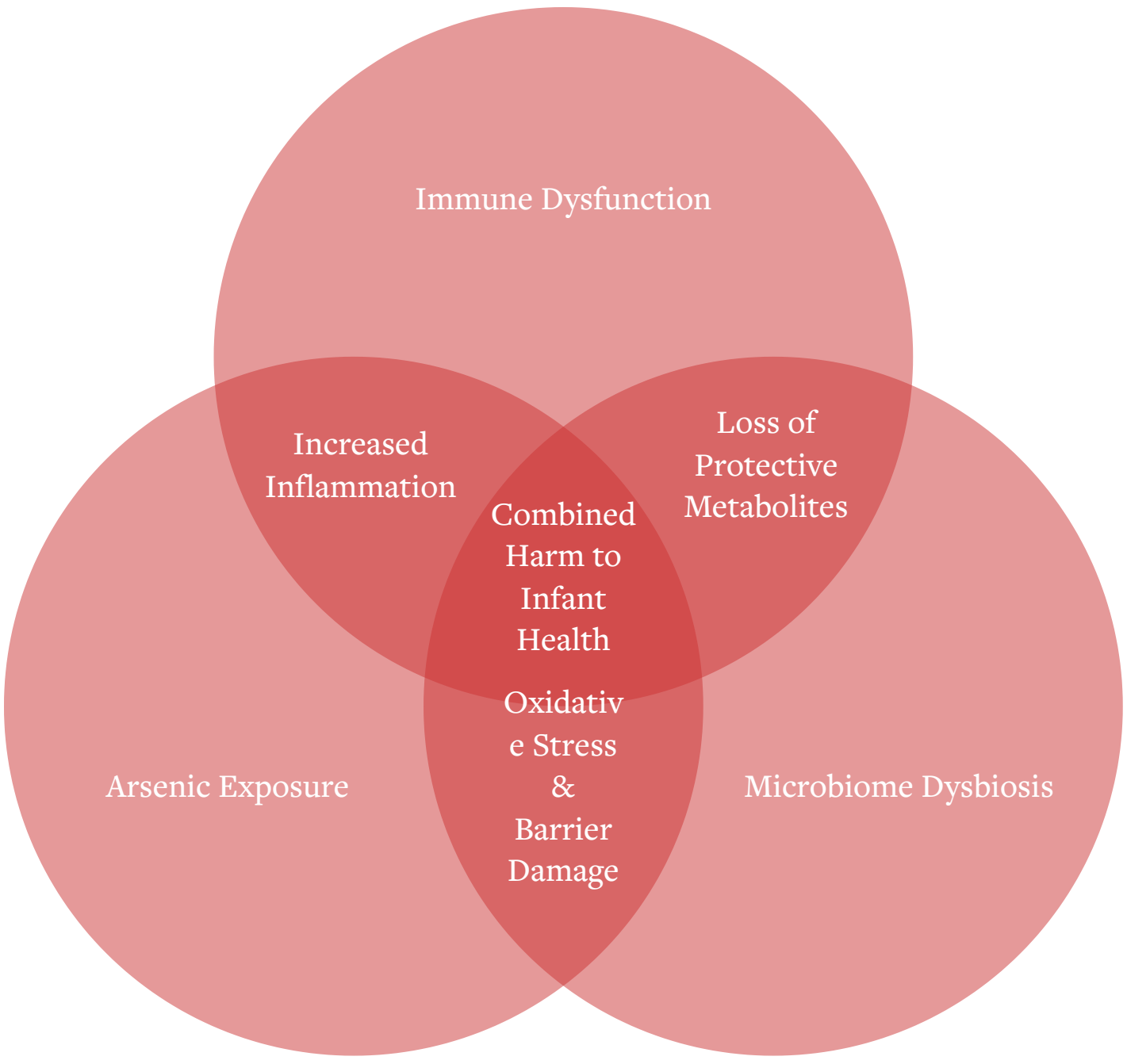
Short-Chain Fatty Acids

Dysbiotic microbiota show reduced abundance of genes involved in short-chain fatty acid production, potentially compromising intestinal barrier integrity and immune development in infants.

The dysbiotic microbiota shows impaired capacity for critical metabolic functions. This functional impairment extends beyond simple reduction in community diversity; specific metabolic pathways are preferentially affected.

Microbiota-Mediated Barrier Dysfunction and Immune Dysregulation

Arsenic-induced microbiota dysbiosis contributes to **intestinal barrier dysfunction and systemic immune dysregulation**. In a preclinical study, arsenic-induced oxidative stress and barrier dysfunction in gut epithelial cells was mitigated by the microbial metabolite urolithin A (UroA), a polyphenol metabolite produced by beneficial bacteria [22]. This finding demonstrates that dysbiosis-related loss of metabolite-producing bacteria exacerbates arsenic toxicity. The dysbiotic microbiota lacks bacteria capable of producing protective metabolites, leaving the epithelium vulnerable to arsenic-induced apoptosis and tight junction protein disruption.

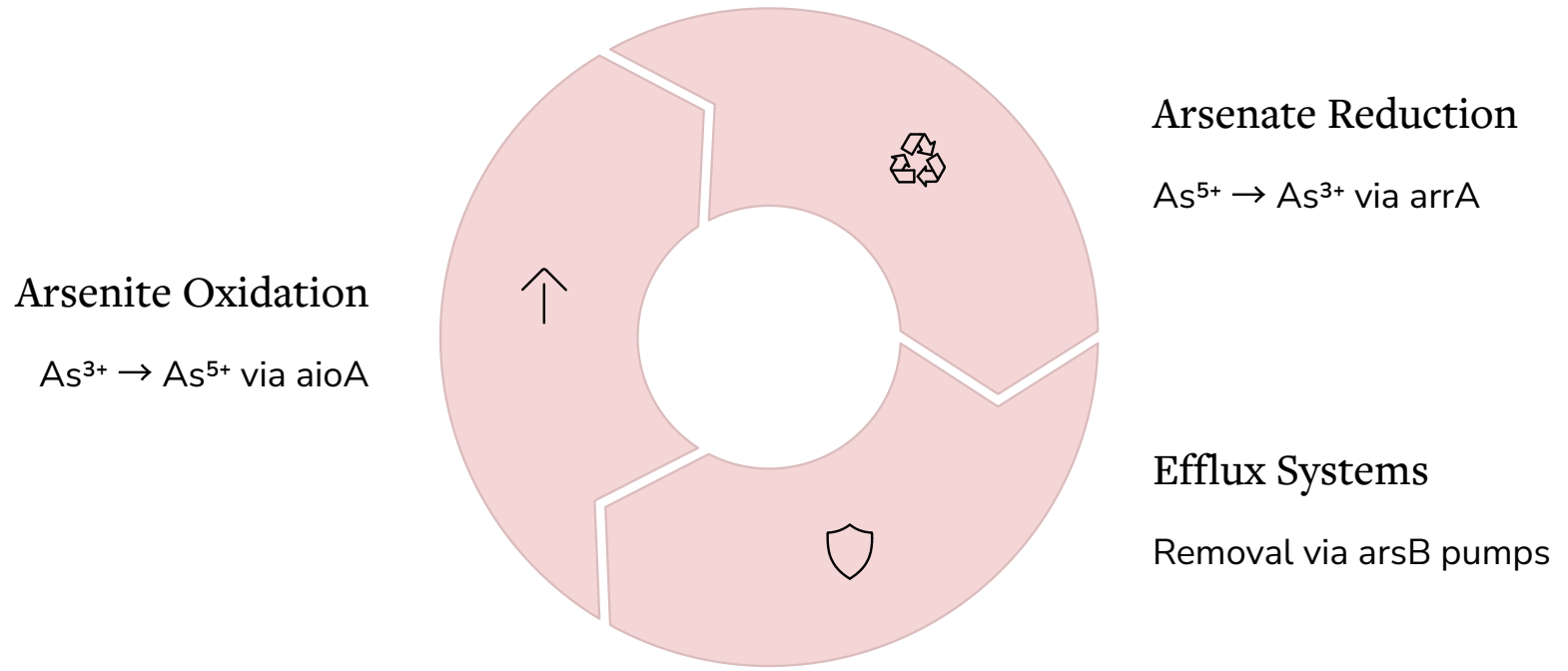


The dysbiotic microbiota exhibits altered production of metabolites that regulate immune development. Short-chain fatty acids produced by fermentative bacteria modulate immune tolerance and promote anti-inflammatory responses, processes critical for proper immune development in infants [23]. Arsenic-induced dysbiosis reduces butyrate-producing bacterial populations, compromising the production of these essential signaling molecules. This metabolic dysfunction contributes to immune dysregulation, potentially explaining the **increased susceptibility to infections** observed in arsenic-exposed children.

Microbial Arsenic Metabolism Pathways and Gene Expression

Microorganisms inhabiting arsenic-contaminated environments express genes encoding arsenic metabolism enzymes that influence arsenic bioavailability and toxicity. Arsenite-oxidizing bacteria oxidize toxic As^{3+} to less mobile As^{5+} through arsenite oxidase (aioA gene products), while arsenate-reducing bacteria convert As^{5+} back to As^{3+} through arsenate reductase (arrA gene products) [24].

In arsenic-contaminated groundwater in Bangladesh, **72 isolated bacterial strains harbored diverse arsenotrophic genes**: 23 isolates possessed the arsenite efflux pump gene (arsB) with high abundance, and 10 isolates harbored the arsenite oxidase gene (aioA). Proteobacteria, Firmicutes, and Acidobacteria dominated the arsenotrophic communities, with genera including *Pseudomonas*, *Acinetobacter*, *Stenotrophomonas*, *Achromobacter*, *Paraburkholderia*, *Comamonas*, and *Klebsiella* identified as potential arsenic detoxifiers.



The metabolic capacity of the microbiota for arsenic transformation creates a dynamic system of arsenic speciation. Rhizosphere microbiota of arsenic-hyperaccumulator plants (*Pteris vittata*) engaged in coordinated arsenic speciation through multiple metabolic processes [25]. The rhizosphere community was dominated by Proteobacteria, Acidobacteriota, and Ascomycota, with 44 bacterial and 10 fungal genera identified as core microorganisms capable of arsenic metabolism. Microbial-mediated arsenic methylation and reduction processes, coupled with carbon fixation, sulfur oxidation, and phosphorus mineralization, contributed to an **"As-multielement cycling" synergy** that enhanced plant arsenic uptake.

Mitigation Strategies and Bioremediative Approaches

Environmental and Dietary Interventions

Evidence-based strategies for reducing arsenic exposure in infants and children include improved agricultural practices, dietary modifications, and regulatory interventions. The comprehensive review of arsenic exposure in infant and child diets identified critical sources including **rice and rice-based products, infant cereals, and contaminated groundwater** [1]. Geographic disparities in regulatory frameworks were highlighted, with recommendations for stricter regulatory limits on arsenic in infant products and encouragement of low-arsenic dietary alternatives.

Dietary Diversity

Dietary interventions targeting arsenic reduction in infants include promotion of diverse grain sources and modified feeding practices. Rice-based infant cereals present particular concern due to rice's propensity for arsenic accumulation [29].

Thickener Assessment

Heavy metal contamination in commonly used thickeners for infants with reflux or dysphagia was evaluated in 56 infants less than one year of age, with urinary arsenic concentrations assessed across different thickener types.

Cumulative Exposure

Infants with higher servings of alternative arsenic sources via solid foods were more likely to have higher urinary arsenic levels, suggesting that cumulative dietary arsenic from multiple sources represents a significant exposure pathway.

Microbial-Based Bioremediative Approaches

Microbial bioremediation represents a promising strategy for reducing arsenic bioavailability in contaminated environments. Arsenic-oxidizing bacteria (AOB) isolated from highly contaminated mining soils can transform toxic As^{3+} into less mobile As^{5+} [30]. *Acinetobacter* sp. TMKU7 isolated from arsenic-contaminated soil transformed **80% of As^{3-} to As^{5+}** under culture conditions and expressed plant growth-promoting traits including siderophore production and indole-3-acetic acid synthesis. The arsenite oxidase enzyme was constitutively expressed and localized to the periplasmic fraction, with partially purified enzyme showing K_m of 41.43 μM and V_{max} of 0.19 $\mu M\ min^{-1}\ g^{-1}$ protein.

Bioaugmentation with specific arsenic-resistant bacterial strains enhanced arsenic mobility and removal from contaminated soils. In highly arsenic and antimony-contaminated Slovak mining soils, biostimulation and bioaugmentation with *Cupriavidus metallidurans* and *Cupriavidus oxaleticus* resulted in mean bleached arsenic fractions of 37.6% and 41.3%, respectively [31]. Consortium-based approaches combining multiple bacterial strains with complementary metabolic capacities showed enhanced remediation efficiency compared to single-strain inoculants.

Integrated Public Health Strategies and Future Directions

Comprehensive public health approaches addressing arsenic exposure in infants and children require integration of environmental monitoring, dietary regulation, and community awareness. The FDA has received recommendations to re-evaluate permissible limits of arsenic in cereals and juices aimed at children consumption, with proposed revisions emphasizing protection of the most vulnerable populations [32]. Current FDA standards allow up to **100 µg/L of arsenic in apple juice**, a level that may exceed safe exposure for young children given their higher per-kilogram intake relative to body weight.

Groundwater Treatment

Infrastructure development for water purification in high-exposure regions

Agricultural Practices

Modification to reduce arsenic uptake in food crops

Food Fortification

Promotion of low-arsenic food sources and dietary diversity

Community Awareness

Education programs for maternal knowledge and risk reduction

Policy interventions must account for geographic disparities in arsenic exposure and vulnerability. In Bangladesh, Pakistan, and India, where dietary arsenic exposure significantly exceeds health reference values, targeted interventions are most urgently needed [1]. These include groundwater treatment infrastructure development, agricultural practices modification to reduce arsenic uptake, and fortification of foods with low-arsenic sources. Community-based environmental monitoring programs provide essential data for informed decision-making regarding remediation priorities.

Community-awareness programs must address maternal knowledge of arsenic exposure risks during pregnancy and lactation, given the importance of early life windows. Comprehensive information regarding arsenic in drinking water, food sources, and occupational exposures would enable informed maternal decision-making to reduce fetal and early childhood exposure. The integration of microbiome science into public health messaging—emphasizing the protective role of beneficial bacteria and the consequences of dysbiosis from environmental toxin exposure—could support development of microbiota-protective strategies including dietary diversity and reduced antimicrobial exposure.

Conclusion: Arsenic exposure in infants and children represents a complex global public health challenge with profound consequences for neurodevelopment, immune function, and long-term health trajectories. Evidence-based interventions combining environmental remediation through arsenic-resistant microorganisms, dietary modifications reducing arsenic exposure, regulatory limits on arsenic in infant foods, and community-based awareness programs offer pathways to reduce the developmental burden of arsenic exposure. Future research prioritizing longitudinal studies with longitudinal microbiota profiling, mechanistic studies of arsenic-induced dysbiosis and mismetallation, and intervention trials of microbiota-protective strategies could substantially advance prevention of arsenic-related developmental disorders in vulnerable populations.

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