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Mercury contamination in a changing climate: One health approaches to human, wildlife, and ecosystem protection

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HIGHLIGHTS

- One Health approach links mercury, human, wildlife, and ecosystem health.
- Climate change amplifies mercury cycling and exposure pathways.
- Cross-species toxicity reveals shared neurotoxic and immune effects.
- Integrated One Health strategies guide mercury risk mitigation.

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ABSTRACT

Mercury contamination exemplifies one of the most pressing global environmental health challenges of the 21st century, requiring integrated solutions that transcend disciplinary boundaries. This review provides the first comprehensive synthesis of mercury science through an explicit One Health lens, linking human, wildlife, and ecosystem health with the accelerating impacts of climate change. Mercury's global cycling and long-range transport generate interconnected exposure pathways, which simultaneously endanger human populations, biodiversity, and ecosystem integrity. The neurotoxic effects of methylmercury display remarkable cross-species consistency, driven by conserved mechanisms such as oxidative stress, mitochondrial dysfunction, and impaired neurodevelopment. Vulnerable groups, including pregnant women, lactating women, infants, children, populations with chronic comorbidities, indigenous communities, and apex predators, face disproportionate risks due to biomagnification and cumulative exposures. Despite regulatory advances, notably the Minamata Convention and its 2023 amendments, ongoing emissions and climate-driven perturbations continue to undermine management efforts. The present review identifies critical gaps in mixture toxicology, ecosystem-level consequences, and climate-mercury feedback. It proposes integrated interventions that simultaneously safeguard human health, wildlife populations, and environmental quality. By situating mercury contamination within the One Health framework, the review highlights the urgent need for coordinated transdisciplinary action to mitigate risks and advance planetary health.

1. Introduction

1.1. Mercury in the Anthropocene: A global environmental health Crisis

Mercury contamination represents one of the most pervasive and persistent environmental health challenges of the Anthropocene epoch, characterized by complex biogeochemical cycling, global atmospheric transport, and severe health consequences across multiple species and ecosystems (Basu et al., 2023; Sonke et al., 2023). As a naturally occurring toxic metal that has been extensively mobilized through

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anthropogenic activities, mercury possesses unique physicochemical properties, enabling it to exhibit worldwide distribution patterns and create health risks that transcend geographic, political, and species boundaries (Driscoll et al., 2013; Giang and Selin, 2016; Streets et al., 2011). The historical case of Minamata disease provides a paradigmatic example of how mercury contamination crystallized the need for integrated perspectives that consider human, ecological, and societal health simultaneously (Basu et al., 2023). The global mercury cycle is largely governed by emissions of elemental mercury (Hg^0) vapor, which has an atmospheric lifetime of about 6–12 months. This relatively long residence time allows mercury to travel long distances and deposit in remote regions. After deposition, mercury can be chemically reduced and re-emitted into the atmosphere, maintaining a dynamic and persistent global reservoir. Human activities, particularly coal combustion, artisanal and small-scale gold mining, and land-use changes such as deforestation, have substantially intensified this natural cycle. These processes increase mercury deposition and methylation in sensitive ecosystems (Obrist et al., 2018). This anthropogenic disruption has led to measurable ecological damage, including biodiversity declines and reproductive failures in piscivorous wildlife, highlighting the profound influence of the Anthropocene on global mercury contamination (Eagles-Smith et al., 2018; Scheuhammer et al., 2007).

1.2. The One Health imperative for mercury management

The One Health approach recognizes the fundamental interconnectedness of human health, animal health, and environmental health, providing a conceptual framework for addressing complex environmental challenges that affect multiple health domains simultaneously (CDC, 2025; Domingo et al., 2025; Wang et al., 2024). This approach is increasingly seen as essential for planetary health, emphasizing that the health of humans, domestic and wild animals, plants, and the wider environment (including ecosystems) are closely linked and interdependent. Mercury contamination provides a compelling case study for One Health principles due to several key characteristics that demonstrate intrinsic linkages between environmental contamination, wildlife health, and human health outcomes (Basu et al., 2023; Eagles-Smith et al., 2018). First, mercury exposure results in remarkably similar adverse health outcomes across phylogenetically diverse species, reflecting shared toxicological mechanisms and conserved biological targets (Peterson et al., 2023; Scheuhammer et al., 2007). Neurological dysfunction, including impaired cognitive function, altered behavior, and developmental abnormalities, is consistently observed across taxa ranging from laboratory rodents to marine mammals and human populations exposed to methylmercury (Clarkson and Magos, 2006; Ha et al., 2017; Liu et al., 2024). These convergent effects support the relevance of wildlife and laboratory animal data for human health risk assessment, while also highlighting the importance of ecosystem-based management strategies that safeguard both human and wildlife health (Al-Sulaiti et al., 2023; Eagles-Smith et al., 2018). Second, aquatic food webs serve as the primary pathway for mercury exposure in both humans and wildlife, creating direct mechanistic linkages between ecosystem health and both human and wildlife health outcomes (Al-Sulaiti et al., 2022; Díez, 2009; Lavoie et al., 2013; Liu et al., 2020). It should be considered that fish consumption is the major source of methylmercury exposure for most human populations (Bridges et al., 2020; González et al., 2019; Jacobs et al., 2017; Li et al., 2024), while piscivorous wildlife species consistently exhibit the highest tissue mercury concentrations within their respective ecosystems (Karimi et al., 2012; Yu et al., 2020). Third, the global nature of atmospheric mercury transport creates shared vulnerabilities that affect both remote ecosystems and human populations far from emission sources (Streets et al., 2011; Yuan et al., 2023). Arctic environments provide a particularly striking example, where global mercury transport combines with unique polar environmental conditions to create elevated exposures for both Arctic wildlife and indigenous populations that rely on traditional marine foods (AMAP, 2021; Chételat et al., 2024). Climate change is altering these transport patterns and environmental conditions, potentially exacerbating mercury risks for both human and wildlife populations in vulnerable regions (Giang and Selin, 2016; Wilkening et al., 2025; Zhou et al., 2024). Fourth, environmental justice considerations highlight the disproportionate impacts of mercury contamination on vulnerable human populations and sensitive ecosystems, creating parallel patterns of vulnerability across health domains (Dai et al., 2023; Gibb and O'Leary, 2014; Spiegel, 2009). Accordingly, mercury represents not only a toxicological challenge but also a governance issue that requires cooperation across human medicine, veterinary science, and environmental policy. While One Health approaches have been applied to mercury research in isolated contexts (e.g., wildlife monitoring (Scheuhammer et al., 2007)) or indigenous health (AMAP, 2021), no prior review has systematically linked these principles with climate change interactions, a critical oversight given the accelerating impacts of global warming on mercury biogeochemistry (Giang and Selin, 2016; Zhou et al., 2024). The interconnections linking mercury contamination, climate change, and health outcomes across the One Health spectrum are shown in Fig. 1. The present review addresses this gap by: (1) mapping climate-mercury feedback loops across ecosystems, (2) evaluating their disproportionate effects on vulnerable populations, and (3) proposing adaptive One Health interventions to mitigate emerging risks.

1.3. Recent scientific advances and emerging challenges

The period extending from 2020 to the present has witnessed significant advances in the comprehension of mercury environmental fate, health effects, and management challenges, driven by technological innovations in analytical chemistry, exposure assessment, and health effects research (Basu et al., 2023; Davies, 2023; Kung et al., 2024; Wu et al., 2024). Simultaneous advances in biomarker development and exposure assessment have provided new insights into mercury health effects at environmentally relevant exposure levels, revealing subtle but significant impacts on immune function, endocrine regulation, and neurodevelopmental processes that occur at exposures previously considered safe (Charkiewicz et al., 2025; Peterson et al., 2023). These findings have important implications for risk assessment and regulatory decision-making, particularly for vulnerable populations including pregnant women, infants, and children (Al-Sulaiti et al., 2023; Grandjean and Landrigan, 2014). The emergence of climate change as a significant modifying factor in mercury environmental fate and health risks represents another major development in contemporary mercury science (Giang and Selin, 2016; Zhou et al., 2024). Climate change influences mercury cycling through multiple pathways, including warming-enhanced microbial methylation in soils and sediments, altered precipitation patterns that affect runoff and mercury transport, and extreme events like wildfires and droughts, which can remobilize legacy mercury. Furthermore, thawing permafrost in Arctic regions is releasing historically stored mercury, creating new sources of contamination (Obrist et al., 2018; Schuster et al., 2018). Recent studies have also shown that gut microbiota may influence mercury absorption and methylation, suggesting a novel pathway for interindividual variability in toxicity (Coe et al., 2023; Pinto et al., 2020). Furthermore, the role of the gut microbiome in modulating mercury toxicity is an emerging One Health issue, as microbial communities in wildlife and humans can affect the bioavailability and demethylation of mercury, influencing susceptibility across species (Coe et al., 2023; Zhang et al., 2022).

Recent advances in exposome science have revolutionized the understanding of how multiple environmental factors interact across the lifespan to influence health outcomes (Bocato et al., 2019). Within this framework, mercury can be conceptualized as a sentinel contaminant that exemplifies how chemical exposures intersect with nutrition, genetics, microbiome composition, and social determinants of health. Systems toxicology approaches integrating high-dimensional omics data (transcriptomics, proteomics, metabolomics, and epigenomics) with

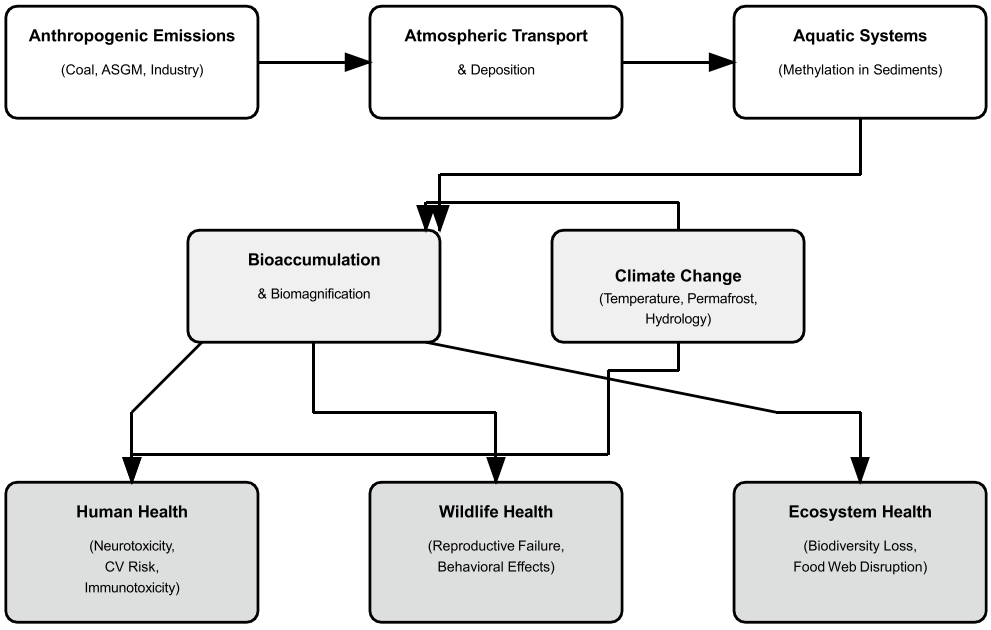


Fig. 1. Conceptual framework of mercury contamination pathways and climate-mercury-health feedback loops within the One Health paradigm.

exposure metrics now enable mechanistic mapping of mercury's impact on biological networks rather than isolated pathways. Applying exposomic methodologies to mercury research can elucidate synergistic effects between mercury and co-occurring stressors, including climate change, malnutrition, and infectious diseases. This systems-level perspective aligns closely with the One Health concept, providing a framework for translating molecular insights into holistic prevention and intervention strategies (Gao, 2021)

1.4. Objectives

The specific objectives of this review are to: (1) characterize shared sources and environmental fate processes that create interconnected contamination patterns across health domains, (2) evaluate convergent health impacts and toxicological mechanisms across human and wildlife populations, (3) identify critical interconnections and feedback loops linking environmental, wildlife, and human health, with particular emphasis on climate change, (4) analyze environmental justice considerations and vulnerability patterns in mercury exposure, (5) assess the impacts of climate change on mercury cycling and health risks, (6) propose integrated intervention strategies based on One Health principles, and (7) highlight critical knowledge gaps that require transdisciplinary research collaboration.

2. Methods of data collection and extraction

This thematic review adopts a One Health-climate framework to synthesize recent advances in mercury contamination, focusing on three key themes: shared exposure pathways, convergent health effects, and climate interactions. Rather than a systematic approach, we employed purposive sampling to select studies that bridged two or more One Health domains (human/wildlife/ecosystem health), ensuring relevance to emerging challenges such as climate-driven methylation and environmental justice.

2.1. Database selection and search strategy

The databases PubMed/MEDLINE (https://pubmed.ncbi.nlm.nih.gov), Web of Science (https://www.webofscience.com), Scopus (https://www.scopus.com), and Google Scholar (https://scholar.google.com.br) were searched to ensure comprehensive coverage of relevant scientific literature published until August 2025. The search strategy employed Boolean operators and controlled vocabulary terms to maximize the retrieval of relevant studies while maintaining specificity. Keyword combinations related to mercury, One Health, exposure pathways, toxicological mechanisms, and environmental justice were searched.

2.2. Inclusion and exclusion criteria

Studies were included if they met the following criteria: (1) focused primarily on mercury, methylmercury, or specific mercury compounds as the main subject of investigation, (2) demonstrated clear relevance to at least two domains of the One Health triad (human health, animal/wildlife health, or environmental health), (3) reported original empirical data, systematic reviews, meta-analyses, or significant conceptual advances that advance understanding of interconnections between health domains, (4) published in peer-reviewed journals with impact factors or recognized international organization reports. Exclusion criteria included: (1) studies focused exclusively on analytical method development without health or environmental implications, (2) case reports or small case series without broader implications for population health or environmental management, and (3) duplicate publications or studies that presented identical data without novel analysis or interpretation.

2.3. Data extraction and quality assessment

Scientific literature from authoritative international organizations, including the United Nations (UN) Environment Program (UNEP), World Health Organization (WHO), Arctic Monitoring and Assessment Program (AMAP), and National Environmental Protection Agencies, was reviewed to supplement peer-reviewed sources and ensure policy relevance. The selected literature was analyzed using a structured One Health framework that considered: (a) shared contamination sources and environmental fate processes that create interconnected exposure pathways, (b) convergent toxicological mechanisms and health effects documented across human and wildlife populations, (c) feedback loops and bidirectional interactions between environmental contamination, ecosystem health, and human health, (d) vulnerable population

characteristics and environmental justice considerations that create disproportionate risks, (e) integrated intervention strategies that generate co-benefits across multiple health domains, and (f) critical knowledge gaps that require transdisciplinary research approaches to address effectively.

3. Results

3.1. Mercury sources, environmental fate, and interconnected exposure pathways

Mercury contamination arises from complex interactions between natural and anthropogenic sources, creating interconnected exposure pathways that simultaneously affect human health, wildlife populations, and ecosystem integrity across multiple spatial and temporal scales (Chen and Evers, 2023; Davies, 2023; Driscoll et al., 2013; Streets et al., 2011). Understanding these shared sources and fate processes is fundamental to developing effective One Health management strategies that address root causes while protecting vulnerable populations and ecosystems (UNEP, 2019; Wang et al., 2024; WHO, 2025).

3.1.1. Anthropogenic sources and global distribution patterns

Coal-fired power plants continue to represent the largest single category of anthropogenic mercury emissions globally, accounting for approximately 35–40 % of total anthropogenic releases despite ongoing emission reduction efforts in many developed countries (Streets et al., 2011; Yuan et al., 2023). Table 1 summarizes the major mercury source categories, their environmental fate processes, and the interconnected exposure pathways, which simultaneously affect human and wildlife populations across different spatial scales. Artisanal and small-scale gold mining (ASGM) activities represent the largest global source of anthropogenic mercury emissions, releasing an estimated 800-1000 metric tons annually and creating severe localized contamination hotspots that directly affect mining communities while contributing to global atmospheric mercury burdens (Davies, 2023; Gibb and O'Leary, 2014; UNEP, 2019). Various studies have reported blood mercury concentrations in ASGM communities that exceed WHO guidelines by factors of 10–100, highlighting the urgent need for integrated health and environmental interventions (Cezarette et al., 2025; Cruz et al., 2024; Davies, 2023; Lemire et al., 2011; Gibb and O'Leary, 2014; Vergara-Murillo et al., 2022)

3.1.2. Environmental fate and biogeochemical cycling processes

The environmental fate of mercury involves extraordinarily complex biogeochemical processes that determine the spatial and temporal distribution of contamination across ecosystems and ultimately influence exposure risks for both human and wildlife populations (Gilmour et al., 2013; Mason et al., 2012; McClelland et al., 2024). Atmospheric elemental mercury (Hg^0) vapor, emitted from both natural and anthropogenic sources, is highly persistent and capable of long-range transport. This persistence allows mercury released in one region to be distributed globally, influencing ecosystems and human populations thousands of kilometers away. Through atmospheric oxidation, Hg^0 is converted into more reactive and water-soluble forms that deposit on land and water surfaces, where mercury becomes integrated into complex biogeochemical cycles (AuYang et al., 2022; Driscoll et al., 2013; Lindberg et al., 2007; Wang et al., 2004). Once deposited in environmental systems, mercury undergoes various chemical transformations that profoundly influence its bioavailability and toxicity to both wildlife and humans (Davies, 2023; Gilmour et al., 2013). In aquatic environments, particularly those characterized by anoxic sediments and elevated organic matter content, sulfate-reducing bacteria and other anaerobic microorganisms methylate inorganic mercury to form methylmercury (MeHg), the most toxic and bioaccumulative form of mercury (Gallorini and Loizeau, 2021; McClelland et al., 2024). This biotransformation process is critical because methylmercury readily crosses biological membranes, accumulates in organisms, and biomagnifies through food webs, creating the primary exposure pathway for both humans and wildlife (Lavoie et al., 2013; Sinclair et al., 2024). Wetland environments consistently exhibit the highest methylation rates due to optimal conditions for anaerobic bacterial activity, making them critical sources of methylmercury to downstream aquatic systems and adjacent terrestrial ecosystems (Davies, 2023; Marvin-DiPasquale et al., 2014).

3.1.3. Bioaccumulation and biomagnification in food web networks

Mercury bioaccumulation in aquatic food webs is the principal pathway linking environmental contamination to adverse health outcomes in both humans and wildlife, thereby establishing direct mechanistic connections between ecosystem integrity and health effects across multiple taxa (Lavoie et al., 2013; Sinclair et al., 2024). Methylmercury demonstrates exceptional efficiency in crossing biological membranes and binding to sulfur-containing amino acids and proteins, leading to rapid uptake and prolonged retention in organisms across all trophic levels (Clarkson and Magos, 2006; Toyama et al., 2023; Wu et al., 2024). Unlike many organic environmental pollutants that preferentially accumulate in lipid-rich tissues, mercury concentrates primarily in protein-rich tissues, including skeletal muscle, liver, kidney, and brain tissue, making it particularly problematic for both wildlife health and human dietary exposure (Peterson et al., 2023; Scheuhammer et al., 2007). Biomagnification processes result in methylmercury concentrations that increase exponentially at successive trophic levels, with apex predators often exhibiting tissue concentrations that are 10^6-10^7 times

Table 1
Major mercury sources, environmental fate, and interconnected exposure pathways.

Source Category	Primary Emission Type	Global Contribution	Environmental Fate	Key Exposure Pathways	Affected Populations	References
Coal-fired Power Plants	Elemental mercury vapor	35–40 % of anthropogenic emissions	Long-range atmospheric transport (6–12 months residence time)	Atmospheric deposition → aquatic methylation → bioaccumulation	Global populations via fish consumption	Streets et al. (2011); Yuan et al. (2023)
Artisanal Small-scale Gold Mining (ASGM)	Metallic mercury vapor, liquid mercury	Largest global source	Local contamination hotspots + atmospheric transport	Inhalation, dermal contact, contaminated fish consumption	Mining communities, local populations	Davies (2023); 2019; Gibb and O'Leary (2014); UNEP (2019)
Natural Sources	Elemental mercury from geological processes	Background atmospheric burden	Global atmospheric cycling	Atmospheric deposition → environmental methylation	All populations (baseline exposure)	AuYang et al. (2022); Driscoll et al., (2013)
Industrial Processes	Various mercury compounds	Variable by region/ industry	Local to regional contamination	Occupational exposure, local environmental contamination	Workers, nearby communities	Streets et al. (2011)
Wetlands and Sediments	Methylmercury production	Critical transformation sites	Methylation by sulfate-reducing bacteria	Primary source of bioavailable mercury for food webs	Consumers of aquatic organisms	Gilmour et al. (2013); McClelland et al. (2024)

higher than ambient water concentrations (Chételat et al., 2024; Lavoie et al., 2013). This biomagnification creates the highest exposures in long-lived piscivorous species, including both commercially important fish species consumed by humans and wildlife species. which serve as ecosystem sentinels and conservation priorities (Eagles-Smith et al., 2018; Liu et al., 2024). Marine ecosystems demonstrate particularly pronounced mercury biomagnification due to complex food web structures and the prevalence of long-lived apex predators (Karimi et al., 2012; Sisma-Ventura et al., 2024). In turn, freshwater ecosystems show high variability in mercury bioaccumulation patterns based on factors including lake morphometry, watershed characteristics, pH, dissolved organic carbon, and food web structure (Eagles-Smith et al., 2018; Lescord et al., 2024). While aquatic food webs are the primary focus, mercury also bioaccumulates in terrestrial ecosystems. A significant pathway is through rice paddies, where anaerobic conditions promote methylation, leading to high methylmercury accumulation in rice grain. This represents a major exposure route for populations in rice-dependent regions, particularly in Asia (Zhao et al., 2020). Furthermore, mercury is transferred from aquatic to terrestrial systems via emergent insects, which are consumed by spiders, birds, and bats, leading to biomagnification in terrestrial food webs (Howie et al., 2018). Forest ecosystems can also accumulate atmospheric mercury, which enters food chains through soil invertebrates and their predators (Cristol et al., 2008; Roffler et al., 2025).

3.1.4. Biodiversity loss, ecosystem resilience, and mercury cycling

Biodiversity loss has profound implications for mercury dynamics and ecosystem resilience. Declines in key species, such as top predators, detritivores, or microbial communities, can alter food web structures and methylation processes, thereby modifying mercury bioavailability and biomagnification rates. In aquatic environments, rivers and oceans act as major conduits and sinks for mercury, where complex hydrological and biogeochemical processes regulate its transformation and redistribution. In riverine systems, seasonal hydrology, sediment transport, and organic matter decomposition promote the conversion of inorganic mercury into methylmercury by anaerobic microorganisms, particularly within flooded soils and anoxic sediments Downstream transport delivers both particulate and dissolved mercury to estuaries and marine ecosystems, where methylmercury can enter pelagic and benthic food webs, bioaccumulating through trophic levels and affecting fish, marine mammals, and human consumers. (Sonke et al., 2023). Floodplain dynamics, deforestation, hydroelectric reservoirs, and coastal eutrophication can all intensify these processes by altering nutrient cycling, redox conditions, and biological productivity, thereby amplifying mercury methylation and accumulation in aquatic organisms (Farella et al., 2006; Guimaraes, 2020; Obrist et al., 2018; Pestana et al., 2019). This cross-ecosystem connectivity highlights the global integration of mercury cycling, linking terrestrial, freshwater, and marine environments through hydrological and biological pathways.

3.2. Human health impacts: contemporary understanding of pathways and effects

Human exposure to mercury occurs through multiple interconnected pathways that reflect the global distribution of environmental contamination, creating exposure scenarios parallel to those experienced by wildlife populations (Eagles-Smith et al., 2018; Ha et al., 2017; Sunderland et al., 2018). Recent advances in exposure assessment and biomarker development have revealed previously unrecognized aspects of mercury toxicity while confirming the persistence of high-exposure scenarios in vulnerable populations worldwide (Al-Sulaiti et al., 2023; Pacyna, 2020).

3.2.1. Exposure pathways and population variability

Fish and seafood consumption continues to represent the dominant source of methylmercury exposure for most human populations globally, contributing 80–95 % of total intake in populations with regular fish consumption (González et al., 2019; Sunderland et al., 2018; Yu et al., 2020; Zhou et al., 2025). This primary exposure pathway creates direct linkages between human health and aquatic ecosystem health. Environmental factors affecting mercury bioaccumulation in fish simultaneously influence human exposure levels across diverse geographic and cultural contexts (Al-Sulaiti et al., 2023; Liu et al., 2024). In many regions, rice consumption is a significant and sometimes dominant dietary source of methylmercury exposure (Zhao et al., 2020). Rice paddies, with their flooded, anoxic conditions, are efficient environments for mercury methylation, leading to bioaccumulation in the grain. Populations that rely on rice as a primary food source can experience significant methylmercury exposure, even in the absence of fish consumption, a critical consideration for risk assessment and public health advisories (Li et al., 2010; Rothenberg et al., 2014). Occupational exposures in ASGM communities continue to represent some of the highest documented human mercury exposures globally, with workers and their families experiencing exposures that can exceed background population levels by factors of 100–1000 (Gibb and O'Leary, 2014; Basu et al., 2023). These extreme exposures occur through multiple simultaneous pathways, including inhalation of metallic mercury vapor during gold processing, dermal contact with mercury-contaminated materials, and consumption of locally caught fish from mercury-contaminated waters (Davies, 2023; Waack et al., 2022). Women and children in ASGM communities face vulnerabilities due to developmental susceptibility and involvement in mercury-handling activities, creating intergenerational health impacts that persist long after direct occupational exposure ceases (Ha et al., 2017; Reuben et al., 2020). Experimental studies also point to interactions between mercury and the gut microbiome, which may influence both detoxification processes and systemic toxicity (Duan et al., 2020; Pinto et al., 2020; Tian et al., 2023). This convergence of exposure pathways, host physiology, and microbial ecology reveals a complex dimension of mercury risk that reflects the integrative principles of One Health (Duan et al., 2020).

3.2.2. Human health effects: from molecular mechanisms to population health

Mercury exerts its toxic effects through fundamental molecular mechanisms that disrupt cellular function. Methylmercury has a high affinity for sulfhydryl groups, binding to cysteine residues in proteins and enzymes, which can alter their structure and function. This binding facilitates its transport across the blood-brain barrier and placental barrier, leading to its accumulation in the central nervous system and fetus. At the cellular level, mercury induces oxidative stress by depleting antioxidants like glutathione and generating reactive oxygen species, leading to lipid peroxidation, protein oxidation, and DNA damage. It also compromises mitochondrial function, leading to impaired energy production and disrupted calcium homeostasis, which may culminate in apoptotic cell death (Clarkson and Magos, 2006; Farina and Aschner, 2017; Wu et al., 2024). These molecular perturbations translate into measurable health effects at both individual and population levels. Mercury toxicity in humans manifests primarily as neurological dysfunction, with the developing nervous system demonstrating particular vulnerability to methylmercury exposure across all stages of development from prenatal through adolescence (Farina and Aschner, 2017; Grandjean and Landrigan, 2014; Leal-Nazaré et al., 2024). It has been suggested that the cardiovascular effects of mercury may be mediated by pathways previously described (see Section 3.4), including oxidative stress and endothelial dysfunction, with additional contributions from altered lipid metabolism and nitric oxide signaling.

3.2.3. Developmental neurotoxicity and intergenerational effects

Prenatal mercury exposure represents the most significant human health concern due to the remarkable efficiency with which methylmercury crosses the placental barrier and accumulates preferentially in fetal brain tissue, where concentrations can exceed those in maternal

levels (Dack et al., 2022; Grandjean and Landrigan, 2014). Large prospective epidemiological studies, particularly the seminal research conducted in the Faroe Islands, Seychelles, and New Zealand, have reported robust associations between prenatal methylmercury exposure and deficits in multiple neurodevelopmental domains, including cognitive function, memory, attention, language development, and fine motor coordination (Debes et al., 2006; Grandjean et al., 2014). The developing nervous system demonstrates exceptional vulnerability to mercury toxicity, due to its immature blood–brain barrier, limited repair capacity, and the mechanistic pathways discussed in Section 3.4 (oxidative stress, mitochondrial dysfunction, and disruption of neurodevelopmental processes) (Abbott and Nigussie, 2021; Grandjean and Landrigan, 2014; Maraslis et al., 2025; Wu et al., 2024).

3.2.4. Adult neurotoxicity and cognitive effects

Adult mercury exposure produces a characteristic pattern of neurological symptoms (Clarkson and Magos, 2006; Bernhoft, 2012). High-level occupational exposures documented in ASGM communities can result in severe neurological manifestations, which may persist for years following exposure reduction (Gibb and O'Leary, 2014; Basu et al., 2023).

3.2.5. Cardiovascular and metabolic effects

Mercury exposure may contribute to the development of cardiovascular disease through multiple interconnected mechanisms, including oxidative stress, endothelial dysfunction, altered lipid metabolism, and inflammatory processes (Guallar et al., 2002; Ha et al., 2017). Epidemiological studies have found associations between mercury exposure and increased risk of myocardial infarction, hypertension, and carotid atherosclerosis, although these associations remain somewhat controversial due to potential confounding by the beneficial effects of fish consumption (Genchi et al., 2017; Roman et al., 2011). Associations between mercury exposure and metabolic disorders, including insulin resistance, metabolic syndrome, and type 2 diabetes, have also been suggested, highlighting a broader spectrum of health concerns (He et al., 2013; Hu et al., 2025; Roy et al., 2017).

3.2.6. Immunotoxicity and infectious disease susceptibility

Mercury exposure consistently impairs immune system function across diverse populations and exposure scenarios, potentially increasing susceptibility to infectious diseases and reducing vaccine efficacy (Bjørklund et al., 2020; Nyland et al., 2011). Recent immunological research has revealed that mercury exposure affects multiple aspects of immune function, including antibody production, T-cell proliferation, natural killer cell activity, and cytokine production (Peterson et al., 2023). The immunotoxic effects can have particular significance for vulnerable populations, including children, infants, the elderly, and immunocompromised persons who can be at increased risk for severe infectious disease outcomes (Wyatt et al., 2019).

3.2.7. Nutritional modulation and mercury–selenium interactions

Dietary composition profoundly influences mercury toxicity. Selenium plays a pivotal protective role by forming biologically inert mercury–selenium complexes and supporting antioxidant selenoenzymes such as glutathione peroxidases and thioredoxin reductases. The molar Hg:Se ratio in blood and tissues has emerged as a key determinant of susceptibility, with evidence from distinct exposed populations, showing that adequate selenium intake may mitigate certain neurological outcomes (de Moraes et al., 2025; Hu et al., 2017; Lemire et al., 2010, 2011; Wesolowska et al., 2024). In the Brazilian Amazon, the coexistence of high selenium intake and significant mercury exposure offers a natural experiment that has advanced understanding of the biochemical balance between toxicity and nutritional protection (de Moraes et al., 2025). Conversely, deficiencies in essential nutrients such as omega-3 fatty acids, zinc, and protein can exacerbate mercury's effects by impairing detoxification and repair mechanisms (Dufault et al., 2009). Thus, considering the nutritional context of mercury exposed populations, especially those dependent on fish for protein and micronutrients, is essential for designing balanced risk-benefit communication strategies consistent with One Health principles.

3.2.8. Vulnerable populations and health equity considerations

Certain human populations face disproportionately high mercury exposures or demonstrate enhanced susceptibility to mercury-induced health effects, creating important environmental justice concerns that must be addressed through targeted intervention strategies (Ha et al., 2017; Sexton and Linder, 2010).

3.2.9. Pregnant women, infants, and children

Pregnant women, infants, and children represent the populations at greatest risk from mercury exposure due to enhanced developmental susceptibility during critical periods of organ system maturation (Grandjean and Landrigan, 2014). Biomonitoring data indicate that approximately 6–8 % of women of reproductive age in developed countries have blood mercury concentrations exceeding current health guidelines, while this proportion may exceed 20–30 % in populations with high fish consumption or proximity to emission sources (Sunderland et al., 2018; WHO, 2021).

3.2.10. Indigenous and subsistence communities

Indigenous communities worldwide face unique vulnerabilities to mercury exposure due to their traditional reliance on fish, marine mammals, and other wildlife that bioaccumulate high mercury concentrations (Chételat et al., 2024). Arctic indigenous populations exhibit some of the highest documented dietary mercury exposures globally, largely due to their reliance on marine mammals, including seals, whales, and polar bears, which occupy apex positions in marine food webs (AMAP, 2021; McClelland et al., 2024). These communities face the culturally devastating choice between maintaining traditional food systems that are central to their identity and reducing mercury exposure, creating complex tradeoffs between nutritional, cultural, and health considerations.

3.2.11. ASGM communities and occupational populations

Communities involved in artisanal gold mining experience some of the highest reported mercury exposures in contemporary human populations, with exposure levels that often exceed those found in historical mercury poisoning incidents (Gibb and O'Leary, 2014; Davies, 2023). These populations often lack adequate healthcare infrastructure and are simultaneously exposed to multiple environmental and social stressors that exacerbate the health risks associated with mercury exposure, including poverty, malnutrition, limited education, and exposure to other environmental contaminants (Basu et al., 2023). Women and children in ASGM communities face vulnerabilities due to their involvement in mercury-handling activities, including gold amalgamation and mercury recovery processes (Ha et al., 2017).

3.3. Wildlife health impacts: sentinels, indicators, and conservation concerns

Wildlife populations play multiple interconnected roles in mercury contamination. They act as environmental sentinels providing early warnings of ecosystem health problems, serve as conservation endpoints deserving protection in their own right, and function as components of food webs that shape human mercury exposure through consumption pathways (Scheuhammer et al., 2007; Eagles-Smith et al., 2018). Studies on wildlife mercury have revealed an increasingly sophisticated understanding of species-specific vulnerabilities while highlighting the population and ecosystem-level consequences of mercury contamination (Chételat et al., 2024; Peterson et al., 2023).

3.3.1. Exposure pathways and bioaccumulation patterns in wildlife

Wildlife mercury exposure primarily occurs through dietary intake, with bioaccumulation patterns demonstrating remarkable consistency across diverse taxa while simultaneously revealing species-specific vulnerabilities related to feeding ecology, habitat use, and physiological characteristics (Scheuhammer et al., 2007; Sinclair et al., 2024). Piscivorous species consistently exhibit the highest tissue mercury concentrations due to efficient biomagnification through aquatic food webs, while species feeding at lower trophic levels generally demonstrate correspondingly lower but still toxicologically significant exposures (Eagles-Smith et al., 2018; Lescord et al., 2024).

3.3.2. Aquatic ecosystems and food web dynamics

Fish species exhibit a wide variation in mercury bioaccumulation, influenced by factors such as trophic position, longevity, habitat use, and species-specific physiological characteristics (Lavoie et al., 2013; Liu et al., 2024). Piscivorous birds, including loons, eagles, osprey, and cormorants, serve as particularly valuable sentinels for aquatic mercury contamination due to their position at the apex of aquatic food webs and their integration of mercury exposure across large geographic areas (Chételat et al., 2024; Evers et al., 2008).

3.3.3. Marine ecosystems and apex predator vulnerabilities

Marine mammals represent some of the most mercury-contaminated wildlife species globally due to their apex position in marine food webs, their longevity, and the prevalence of methylmercury in marine environments (Dietz et al., 2013; Peterson et al., 2023). Arctic marine mammals, including polar bears, beluga whales, narwhals, and various seal species, show elevated mercury concentrations due to the combination of global mercury transport to polar regions and unique Arctic environmental conditions that enhance methylation (AMAP, 2021; Chételat et al., 2024). Studies on northern elephant seals have provided unprecedented insights into the interactive effects of mercury exposure and physiological stress in marine mammals, revealing complex relationships between mercury bioaccumulation, endocrine function, and immune system response that can influence population health and reproductive success (Peterson et al., 2015, 2023).

3.3.4. Terrestrial ecosystems and cross-system transfers

While mercury concentrations are generally lower in terrestrial compared to aquatic systems, certain terrestrial species can accumulate significant mercury burdens through consumption of aquatic prey or foraging in mercury-contaminated environments (Rimmer et al., 2005; Roffler et al., 2025). Insectivorous birds that consume aquatic insects during their emergence from contaminated waters can exhibit elevated mercury concentrations, reflecting contamination in the aquatic system, while simultaneously transferring mercury to terrestrial food webs (Brasso et al., 2020; Cristol et al., 2008; Poblete et al., 2023). Spiders and other terrestrial arthropods that consume emergent aquatic insects can bioaccumulate mercury and subsequently transfer it to terrestrial predators, including birds, bats, and small mammals (Azevedo et al., 2018; Ouédraogo et al., 2015). This cross-system transfer creates exposure pathways for terrestrial wildlife species that may appear removed from aquatic mercury sources but face significant exposure risks through consumption of mercury-contaminated arthropod prey.

3.3.5. Species-specific health effects and population implications

The health effects of mercury in wildlife arise from the same core toxicological mechanisms outlined in this section, notably protein binding, oxidative stress, and mitochondrial dysfunction. These conserved mechanisms produce comparable patterns of physiological impairment across diverse species. Mercury toxicity in wildlife demonstrates remarkable consistency across taxonomic groups, with neurological dysfunction, reproductive impairment, and immunosuppression representing the most reported effects across diverse species (Eagles-Smith et al., 2018; Scheuhammer et al., 2007). These convergent effects reflect shared toxicological mechanisms while revealing species-specific vulnerabilities that influence population-level outcomes.

3.3.6. Neurological and behavioral effects

In wildlife, mercury exposure manifests primarily as behavioral alterations, including impaired foraging, predator avoidance, mating, and parental care, which compromise survival and reproductive success. These outcomes reflect the shared toxicological mechanisms outlined in Section 3.4 (Eagles-Smith et al., 2018; Chételat et al., 2024).

3.3.7. Reproductive and developmental toxicity

Mercury exposure impairs wildlife reproduction, leading to reduced fertility, embryonic mortality, developmental abnormalities, and altered parental behavior, outcomes that are consistent with the mechanistic pathways discussed in Section 3.4 (Evers et al., 2008; Scheuhammer et al., 2007). Piscivorous birds demonstrate particular sensitivity to mercury-induced reproductive effects, with exposure-response relationships found for reduced egg hatchability, increased embryonic deformities, and decreased chick survival (Eagles-Smith et al., 2018; Peterson et al., 2023).

3.3.8. Immunotoxicity and disease susceptibility

Mercury-induced immunosuppression in wildlife can increase susceptibility to infectious diseases, parasites, and other environmental stressors that may contribute to population-level health effects (Nyland et al., 2011; Peterson et al., 2023). Associations between mercury exposure and increased parasite loads, altered inflammatory responses, and reduced antibody production have been found in various wildlife species (Chételat et al., 2024; Eagles-Smith et al., 2018; Peterson et al., 2023).

3.3.9. Population dynamics and conservation implications

Common loon populations in mercury-contaminated lakes of the northeastern United States and southeastern Canada have shown evidence of population-level effects, including reduced breeding success and altered population age structure that may contribute to long-term population declines (Evers et al., 2008; Kenow et al., 2003, 2011). These population effects occur at mercury concentrations that are widespread across much of the species' range, suggesting that mercury contamination can be a significant factor limiting population recovery in some regions (Dykstra et al., 2019; Eagles-Smith et al., 2018).

3.4. Convergent toxicological mechanisms: molecular basis for one health applications

The remarkable consistency of mercury-induced health effects across phylogenetically diverse species reflects fundamental, shared toxicological mechanisms and conserved biological targets, providing the scientific bases for One Health approaches to mercury risk assessment and management (Clarkson and Magos, 2006; Pan et al., 2025). Understanding these convergent mechanisms allows wildlife and laboratory animal data to be applied to human health protection, while also guiding management strategies that safeguard both human and ecosystem health (Peterson et al., 2023; Scheuhammer et al., 2007). Table 2 shows a comparison of convergent health effects observed in humans and wildlife, highlighting the shared toxicological mechanisms that justify integrated One Health approaches to mercury risk assessment, and the parallel vulnerability patterns across taxa.

3.4.1. Cellular and molecular mechanisms of mercury toxicity

3.4.1.1. Protein binding and cellular uptake mechanisms. Methylmercury demonstrates exceptionally high affinity for sulfur-containing amino acids, particularly cysteine and methionine, enabling formation of stable

Table 2
Convergent health effects across human and wildlife populations.

Health Domain	Human Effects	Wildlife Effects	Shared Mechanisms	Vulnerable Populations/ Species	Critical Exposure Levels	References
Neurological/ Behavioral	Cognitive deficits, memory impairment, motor dysfunction, developmental delays	Altered foraging behavior, impaired predator avoidance, reduced territorial defense, abnormal mating behavior	Oxidative stress, mitochondrial dysfunction, disruption of neurotransmitter systems	Pregnant women, children, developing animals	Prenatal exposure most critical	Chen and Evers (2023); Grandjean and Landrigan (2014); Scheuhammer et al. (2007)
Reproductive	Reduced fertility, increased miscarriage risk, developmental abnormalities	Decreased egg hatchability, increased embryonic mortality, impaired parental care	Disruption of hormone signaling, cellular damage in reproductive tissues	Women of reproductive age, breeding wildlife populations	Effects at relatively low chronic exposures	Eagles-Smith et al. (2018); Ha et al. (2017)
Immunological	Reduced antibody production, increased infection susceptibility, altered vaccine responses	Increased parasite loads, reduced disease resistance, altered inflammatory responses	Impaired T-cell function, disrupted cytokine production, oxidative damage to immune cells	Children, elderly, immunocompromised individuals, stressed wildlife	Immunosuppression at environmentally relevant levels	Nyland et al. (2012); Peterson et al. (2023)
Cardiovascular	Increased risk of myocardial infarction, hypertension, atherosclerosis	Cardiovascular stress, altered heart rate variability	Endothelial dysfunction, disrupted nitric oxide signaling, increased oxidative stress	Adults with existing cardiovascular risk factors	Effects reported at moderate exposure levels	Al-Sulaiti et al. (2023); Ha et al. (2017)
Developmental	Prenatal brain damage, reduced IQ, learning disabilities	Developmental abnormalities, reduced survival to independence	Enhanced vulnerability during rapid cell division, immature detoxification systems	Fetuses, infants, juvenile animals	Critical windows during organogenesis	Chen et al. (2023); Evers et al. (2008)

mercury-amino acid complexes that facilitate cellular uptake through amino acid transport systems (Clarkson and Magos, 2006; Farina and Aschner, 2017). This molecular mechanism allows methylmercury to cross biological membranes efficiently via large amino acid transporters, explaining its rapid distribution throughout body tissues and its preferential accumulation in protein-rich organs, including brain, kidney, and skeletal muscle (Al-Sulaiti et al., 2023). The blood-brain barrier, which effectively excludes most toxic substances from neural tissue, is readily crossed by methylmercury through the L-type amino acid transporter, which recognizes the methylmercury-cysteine complex as a methionine analog (Aschner and Clarkson, 1988; Farina and Aschner, 2017). This mechanism explains the preferential neurotoxicity of methylmercury compared to inorganic mercury forms and underlies the consistent patterns of neurological dysfunction observed across species (Aaseth et al., 2020; Peterson et al., 2023).

3.4.1.2. Oxidative stress and cellular damage pathways. Mercury exposure induces cellular oxidative stress through multiple interconnected mechanisms, including depletion of cellular antioxidants, inhibition of antioxidant enzyme systems, and direct generation of reactive oxygen species (Kang et al., 2024; Maraslis et al., 2025). Glutathione, the primary cellular antioxidant, is rapidly depleted by mercury exposure as glutathione sulfhydryl groups bind mercury with high affinity, reducing cellular antioxidant capacity while simultaneously facilitating mercury retention (Al-Sulaiti et al., 2023; Clarkson and Magos, 2006). Oxidative stress contributes to cellular damage through lipid peroxidation, protein oxidation, and DNA damage that can result in cell dysfunction and death (Peterson et al., 2023).

3.4.1.3. Mitochondrial dysfunction and energetic impairment. Mercury disrupts mitochondrial function through multiple mechanisms, including inhibition of respiratory chain complexes, alteration of mitochondrial membrane permeability, and disruption of calcium homeostasis (Sun et al., 2022; Yin et al., 2007). The mitochondrial effects provide mechanistic explanations for the vulnerability of neural tissue to mercury toxicity, while highlighting the shared cellular targets that create consistent effects across species.

3.4.1.4. Genetic and epigenetic determinants of individual susceptibility. Recent research highlights that interindividual and interspecies variability in mercury toxicity may arise from genetic and epigenetic factors that modulate mercury absorption, distribution, metabolism, and excretion. Polymorphisms in genes encoding glutathione-related enzymes (e.g., GSTM1, GSTT1, GCLM), selenoproteins (SEPP1, GPX1), and metal transporters (SLC7A11, ABCC2) have been associated with differential mercury body burden and neurotoxic outcomes (Barcelos et al., 2013; Maraslis et al., 2025). Moreover, epigenetic modifications, including DNA methylation, histone acetylation, and non-coding RNA expression, have been linked to long-term neurobehavioral and immunological alterations, even at low exposure levels (Cediel-Ulloa et al., 2023; Khan et al., 2019). Such mechanisms may contribute to transgenerational effects observed in wildlife and human populations. Integrating genetic and epigenetic biomarkers into One Health mercury studies could significantly enhance risk stratification and enable precision prevention approaches tailored to vulnerable populations.

3.5. Environmental justice and vulnerable populations

Indigenous communities worldwide experience disproportionate mercury exposure through their traditional reliance on fish, marine mammals, and other wildlife that bioaccumulate high mercury concentrations (Basta et al., 2021; Chételat et al., 2024). These exposures create profound cultural conflicts as communities must choose between maintaining traditional food systems that are central to their cultural identity and reducing health risks through dietary modification (AMAP, 2021; McClelland et al., 2024). These communities often lack the political power to influence mercury emission policies while bearing the health consequences of industrial activities occurring thousands of kilometers away.

3.5.1. Urban environmental justice and cumulative impacts

Low-income urban communities often experience elevated mercury exposures through proximity to industrial sources, contaminated sites, and waste management facilities while simultaneously facing multiple other environmental health stressors (Basu et al., 2023). These cumulative exposures create health risks that exceed those associated with mercury alone, affecting communities with limited healthcare access and reduced political influence (Ha et al., 2017).

3.5.2. Socioeconomic factors and health access

Limited access to healthcare services prevents many vulnerable populations from receiving appropriate monitoring, treatment, and prevention services related to mercury exposure (Davies, 2023). In the Brazilian Amazon, riverine communities living along the Tapajós and Madeira Rivers are among the most chronically exposed populations worldwide, largely due to high fish consumption and the remobilization of mercury from natural and anthropogenic sources (Berzas Nevado et al., 2010; Canela et al., 2024; Cruz et al., 2024; Grotto et al., 2010; Guimarães et al., 2024). Despite decades of documented contamination, these populations often remain outside formal healthcare systems, with limited laboratory capacity for mercury biomonitoring and no systematic screening for neurological or developmental outcomes. Geographical isolation, scarcity of transportation, and socioeconomic inequalities further restrict access to medical care, education, and risk communication, exacerbating health vulnerabilities.

ASGM communities often lack basic healthcare infrastructure, experiencing some of the highest mercury exposures globally, creating situations where severe health effects can be undiagnosed and untreated for extended periods (Basu et al., 2023; Gibb and O'Leary, 2014). In Amazonian contexts, overlapping challenges, including malnutrition, infectious diseases, and exposure to other environmental contaminants such as lead and polycyclic aromatic hydrocarbons (PAHs), contribute to a complex exposome that amplifies health disparities (Barbosa et al., 2009; González et al., 2024; Orellana et al., 2025).

Community-based participatory approaches have shown promise for addressing these information barriers by involving affected communities in research design and implementation, with the building of local capacity for environmental health protection (Ha et al., 2017). These approaches not only enhance data collection and interpretation but also strengthen environmental citizenship and local capacity for sustainable health protection (Ha et al., 2017).

3.5.3. Psychosocial impacts and community well-being

Beyond measurable biomedical effects, mercury exposure can deeply affect psychosocial well-being in exposed communities. Anxiety, stigma, and loss of trust in traditional food systems could be psychological factors influenced by the knowledge of their staple food is contaminated and after non-appropriate risk communication campaigns, stimulating the reduction of fish consumption. In the Brazilian Amazon, these psychosocial dimensions are intensified by historical marginalization and the cultural centrality of fish consumption in riverine and Indigenous communities. Moreover, climate change is exacerbating these vulnerabilities by disrupting fisheries, altering food availability, and increasing migration pressure on riverine populations. One Health frameworks must therefore incorporate psychosocial indicators of community health and emphasize participatory communication strategies that respect cultural contexts while empowering affected populations to co-design solutions.

3.6. Climate change interactions and future risk scenarios

Climate change is increasingly significant in modifying the environmental fate, biogeochemical cycling, and health risks of mercury, creating complex interactions that may fundamentally alter global mercury contamination patterns over the coming decades (Giang and Selin, 2016; Zhou et al., 2024). Climate change affects mercury cycling through multiple interconnected pathways, including alterations in atmospheric transport patterns, precipitation regimes, temperature-dependent reaction rates, and ecosystem structure and function (Giang and Selin, 2016; Zhou et al., 2024). Rising global temperatures can accelerate microbial methylation processes in soils and sediments, potentially increasing the formation of bioavailable methylmercury. Alterations in hydrological regimes, such as more intense rainfall events and extended droughts, modify both mercury runoff from terrestrial to aquatic systems and the residence time of water within ecosystems, thereby influencing methylation dynamics and bioaccumulation potential. The growing frequency and severity of wildfires further release substantial quantities of mercury stored in soils and biomass back into the atmosphere, enhancing its long-range transport and redeposition. In addition, the thawing of permafrost in Arctic and sub-Arctic regions is mobilizing large historical mercury reservoirs, reintegrating them into modern biogeochemical cycles (Obrist et al., 2018; Schuster et al., 2018; Zhou et al., 2024). The complex interplay of anthropogenic emissions, global transport, aquatic methylation, and climate-driven modifiers is shown in Fig. 2, which illustrates the feedback loops linking mercury contamination to human, wildlife, and ecosystem health within the One Health framework.

3.6.1. Arctic amplification and polar vulnerabilities

Arctic environments face particularly severe vulnerabilities from climate-mercury interactions due to rapid warming rates, changing ice dynamics, and unique polar environmental conditions that enhance both mercury transport and bioavailability (AMAP, 2021; Zhou et al., 2024). Thawing permafrost may release stored mercury that has accumulated over millennia, potentially creating new sources of environmental contamination as Arctic temperatures continue to rise (Schuster et al., 2018; McClelland et al., 2024).

3.7. Integrated One Health intervention strategies

Effective mercury management requires comprehensive intervention strategies that address contamination sources, simultaneously protecting human health, wildlife populations, and ecosystem integrity (UNEP, 2019; Wang et al., 2024). The One Health approach provides a conceptual framework for developing these integrated interventions by recognizing the interconnected nature of health risks and seeking solutions that generate co-benefits across multiple health domains (Basu et al., 2023; Davies, 2023). This approach requires moving beyond isolated interventions toward integrated strategies that explicitly address the interconnections among human, animal, and environmental health. For example, controlling emissions at their source not only decreases atmospheric deposition and improves ecosystem integrity, but also decreases mercury concentrations in fish, protecting both human and wildlife health. Likewise, wetland restoration can simultaneously sequester carbon, enhance biodiversity, and mitigate methylmercury formation, providing multiple co-benefits across environmental and health dimensions. Table 3 shows key One Health intervention strategies and their anticipated benefits across human health, wildlife health, and ecosystem health domains, along with implementation challenges and measurable success indicators for each approach.

3.7.1. Global source control and regulatory frameworks

3.7.1.1. International cooperation and the Minamata Convention. The Minamata Convention on Mercury, which entered into force in 2017 and was amended in 2023, represents the primary international framework for coordinating global mercury reduction efforts (UN, 2025; UNEP, 2019). The Convention requires parties to reduce anthropogenic mercury emissions and releases, phase out certain mercury-containing products, including dental amalgam, and address contaminated sites through coordinated international action (Selin and Selin, 2022; UNEP, 2019). Successful implementation requires sustained international cooperation, technology transfer, capacity building, and financial support for countries that face the greatest challenges in meeting Convention obligations (Basu et al., 2023).

3.7.1.2. Sectoral emission reduction strategies. Coal-fired power plants represent the most controllable major source of mercury emissions, and regulations requiring the installation of emission control technologies can achieve an 85–95 % reduction in mercury releases, simultaneously

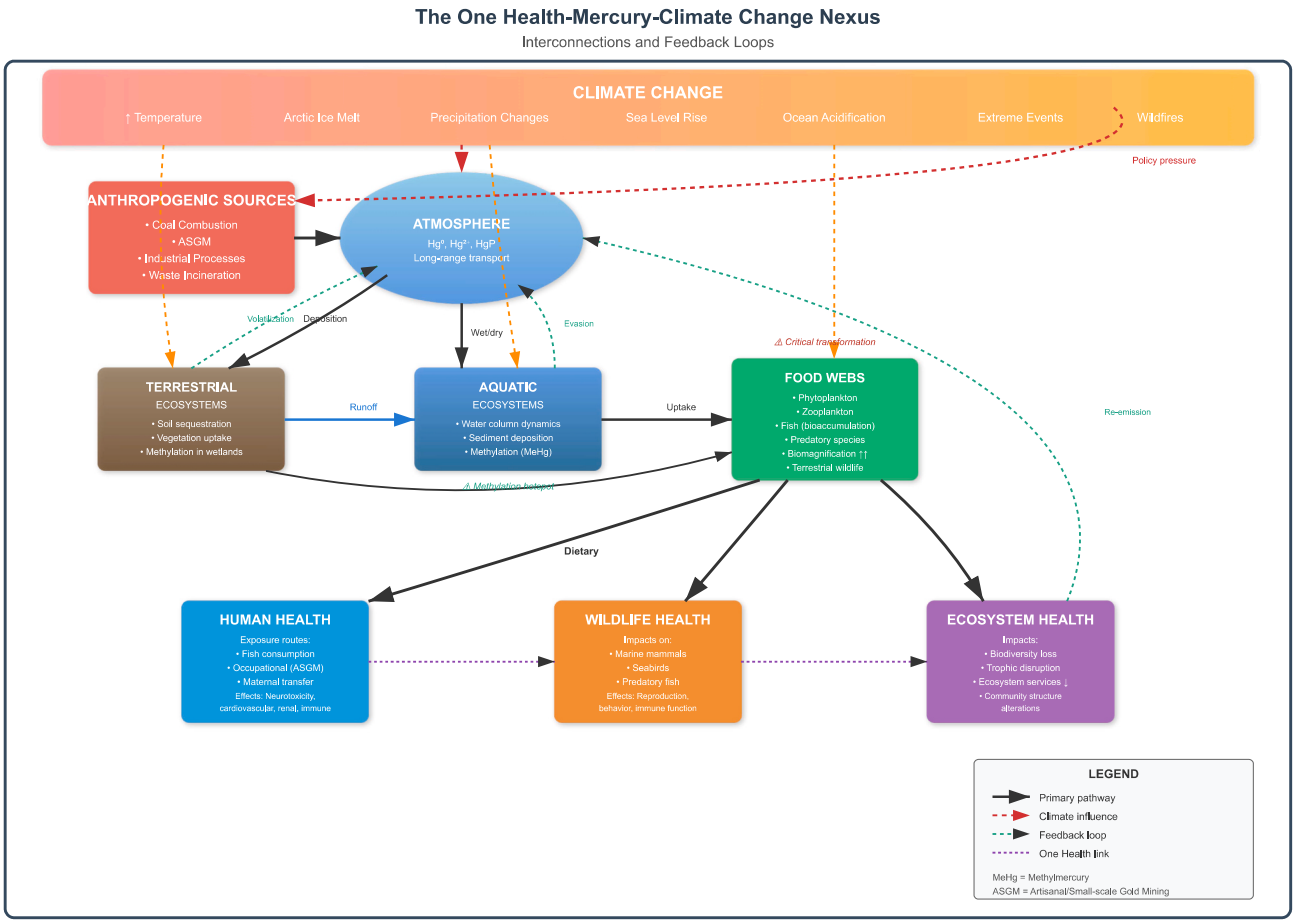


Fig. 2. The One Health-mercury-climate change nexus.

reducing other pollutants, including particulate matter, sulfur dioxide, and nitrogen oxides (Streets et al., 2011; Yuan et al., 2023). Addressing mercury use in ASGM requires complex integrated interventions that consider environmental, health, and socioeconomic factors affecting mining communities (Basu et al., 2023; Gibb and O'Leary, 2014).

3.7.2. *Environmental remediation and ecosystem restoration*

3.7.2.1. Contaminated site management and restoration. Historical mercury contamination sites require long-term integrated management approaches that protect human health and support ecosystem recovery (Davies, 2023; Mason et al., 2012).

3.7.2.2. Ecosystem-based management approaches. Wetland restoration and management can influence mercury methylation and bioaccumulation in downstream ecosystems, providing multiple ecosystem services including flood control, water quality improvement, and habitat provision (Gilmour et al., 2013; McClelland et al., 2024). For example, re-establishing native vegetation in riparian zones can reduce erosion and organic matter loading to water bodies, limiting the substrates and conditions that favor methylation. Such nature-based solutions exemplify the One Health approach by simultaneously improving ecosystem function, reducing a key exposure pathway for wildlife and humans, and enhancing climate resilience.

3.7.3. *Public health interventions and community-based approaches*

3.7.3.1. Fish consumption advisories and dietary guidance. Fish consumption advisories represent the primary tool for reducing human dietary mercury exposure, but they must carefully balance mercury risks against the substantial nutritional benefits of fish consumption, including high-quality protein, omega-3 fatty acids, and essential micronutrients (Al-Sulaiti et al., 2023; Domingo et al., 2007; Mozaffarian and Rimm, 2006). One Health approaches can inform advisory development by considering ecosystem health implications, alternative protein sources, and the differential impacts of advisories on various population groups (Danasekaran, 2024; Liu et al., 2024; Yu et al., 2020). These approaches recognize that blanket consumption advisories may create unintended consequences, including reduced consumption of beneficial nutrients and disproportionate impacts on communities that depend on fish for subsistence (Chételat et al., 2024). A One Health–informed advisory would employ an integrative, evidence-based framework to encourage the consumption of low-mercury fish species derived from sustainably managed stocks, protecting human health, while preserving nutritional adequacy and preventing additional anthropogenic pressures on ecologically sensitive wildlife populations.

3.7.3.2. Biomonitoring and health surveillance systems. Integrated biomonitoring programs that include both human and wildlife components can provide a comprehensive assessment of mercury contamination trends, enabling early detection of emerging health risks (Basu et al., 2018; Peterson et al., 2023). Wildlife monitoring can serve as an early warning system for human health risks, while also documenting ecosystem recovery following the implementation of interventions (Chételat et al., 2024; Eagles-Smith et al., 2018). For instance, monitoring mercury levels in sentinel species like loons or otters can provide cost-effective and ecologically relevant data on the effectiveness of emission controls long before changes are detectable in human populations or water samples.

Table 3
One health intervention strategies and Expected outcomes.

Intervention Category	Specific Actions	Human Health Benefits	Wildlife Health Benefits	Ecosystem Benefits	Implementation Challenges	Success Indicators	References
Global Source Control	Minamata Convention implementation, coal plant emission controls, ASGM mercury reduction	Reduced global atmospheric mercury burden, decreased dietary exposure	Lower tissue mercury concentrations across species	Reduced environmental mercury loading	International coordination, technology transfer, enforcement	Declining atmospheric mercury trends, reduced bioaccumulation	UNEP (2019); WHO (2025); Yuan et al. (2023)
Ecosystem-based Management	Wetland restoration, watershed management, habitat protection	Improved water quality, reduced methylmercury production	Enhanced habitat quality, reduced mercury bioavailability	Restored ecosystem function, improved biogeochemical cycling	Complex ecosystem interactions, long-term commitment required	Reduced methylmercury production rates, improved food web health	Gilmour et al. (2013); McClelland et al. (2024)
Community-based Interventions	Health education, exposure reduction training, alternative livelihoods (ASGM)	Direct exposure reduction, improved health outcomes, enhanced health literacy	Reduced local contamination sources	Decreased point source pollution	Cultural sensitivity, resource constraints, capacity building needs	Reduced biomarker levels, improved health outcomes, community engagement	Basu et al. (2023); Davies (2023); Gibb and O'Leary (2014)
Integrated Monitoring	Human biomonitoring, wildlife health surveillance, environmental monitoring	Early detection of health risks, exposure trend assessment	Population health assessment, early warning of ecosystem problems	Comprehensive contamination assessment	Resource intensive, technical capacity requirements	Coordinated data on exposure trends across health domains	Eagles-Smith et al. (2018); Peterson et al. (2023)
Risk Communication	Fish consumption advisories, targeted health education, community engagement	Informed decision-making, reduced high-risk exposures	Indirect benefits through reduced human fishing pressure	Reduced pressure on contaminated food sources	Balancing risks and benefits, cultural considerations	Appropriate behavioral changes, maintained nutritional benefits	Al-Sulaiti et al. (2023); Chételat et al. (2024)
Climate Adaptation	Adaptive management frameworks, climate-mercury interaction research	Protection under changing exposure scenarios	Resilience to climate-altered contamination patterns	Ecosystem adaptation to changing environmental conditions	Scientific uncertainty, complex modeling requirements	Maintained protection effectiveness under climate change	McClelland et al. (2024); Zhou et al. (2024)

3.7.3.3. Community-based interventions and capacity building. Community-based interventions in high-exposure populations can address multiple dimensions of mercury risk, building local capacity for environmental health protection and advocacy (Davies, 2023; Gibb and O'Leary, 2014). These interventions integrate health education, exposure reduction strategies, healthcare access improvement, and community empowerment approaches that address both immediate health risks and underlying structural inequities (Basu et al., 2023). In ASGM communities, this might involve training on mercury-free gold extraction techniques, providing personal protective equipment, establishing community health worker programs for basic diagnosis and referral, and supporting alternative livelihoods. The effectiveness of such interventions depends on meaningful community engagement and the systematic integration of local and traditional knowledge into decision-making processes.

4. Knowledge gaps and research priorities

Despite extensive research progress in recent decades, significant knowledge gaps remain that limit the ability to fully implement One Health approaches to mercury management and protection of vulnerable populations and ecosystems (Basu et al., 2023; Davies, 2023).

4.1. Mechanistic understanding and low-dose effects

4.1.1. Molecular mechanisms of chronic low-dose exposure

While high-dose mercury effects are well-characterized across species, the molecular mechanisms underlying subtle effects at environmentally relevant exposure levels require additional investigation to support protective risk assessment and regulatory decision-making (Al-Sulaiti et al., 2023; Branco et al., 2017). Advances in omics technologies, including genomics, proteomics, and metabolomics, offer opportunities for identifying sensitive biomarkers of early effect that could improve exposure assessment and health protection (Peterson et al., 2023). Epigenetic mechanisms underlying mercury toxicity represent a particularly important research priority, as emerging evidence suggests that mercury exposure may cause DNA methylation changes and histone modifications, which persist long after exposure cessation and may be transmitted to subsequent generations (Ha et al., 2017; Cediel-Ulloa et al., 2023; Ke et al., 2023).

4.1.2. Mixture effects and cumulative risk assessment

Real-world mercury exposure occurs within the context of complex chemical mixtures that may include other toxic metals, persistent organic pollutants (POPs), and various industrial chemicals, but mixture toxicology remains poorly understood (Basu et al., 2023; Budnik and Casteleyn, 2019; Wu et al., 2024). Interactive effects between mercury and other environmental stressors, including nutritional deficiencies, infectious diseases, and social stressors, may significantly modify toxicity in ways that are not captured by single-chemical risk assessment approaches (Ha et al., 2017).

4.2. Ecosystem-level effects and food web dynamics

4.2.1. Population and community-level effects assessment

While individual-level effects of mercury have been extensively reported, population and ecosystem-level consequences require additional long-term research to understand how mercury contamination affects wildlife population dynamics, community structure, and ecosystem function (Eagles-Smith et al., 2018; Scheuhammer et al., 2007). Understanding these broader ecological effects is essential for comprehensive ecosystem management approaches that consider mercury impacts within the context of multiple environmental stressors.

4.2.2. Food web modeling and predictive capabilities

Advanced food web models that integrate biogeochemical processes

with ecological dynamics are needed to predict mercury bioaccumulation under changing environmental conditions and assess the effectiveness of different management strategies (Lavoie et al., 2013; Sinclair et al., 2024). Machine learning and artificial intelligence approaches offer opportunities for analyzing complex ecological datasets and identifying patterns in mercury bioaccumulation that may not be apparent through traditional statistical methods (Davies, 2023).

4.3. Climate change interactions and adaptive management

4.3.1. Predictive modeling of climate-mercury interactions

Sophisticated Earth system models that couple climate, biogeochemical, and health risk components are needed to predict how climate change will affect mercury cycling, bioaccumulation, and health risks over the coming decades (Giang and Selin, 2016; Zhou et al., 2024). These models should integrate atmospheric transport, terrestrial and aquatic biogeochemistry, food web dynamics, and human exposure pathways to provide comprehensive assessments of future mercury risks. The use of artificial intelligence (AI) and machine learning (ML) into mercury research would offer powerful new tools for predictive risk assessment and decision support. These methods can synthesize complex datasets, ranging from remote-sensing imagery and climate models to human biomonitoring and wildlife surveillance, to predict emerging hotspots and exposure trends (Olawade et al., 2024)

4.3.2. Adaptive management framework development

Research is needed to develop adaptive management frameworks that can respond effectively to changing mercury risks under climate change scenarios while maintaining protection for both human health and ecosystem integrity (McClelland et al., 2024; Zhou et al., 2024). These systems should integrate environmental monitoring, wildlife health surveillance, and human biomonitoring data to provide comprehensive assessments of changing risk patterns.

4.4. Intervention effectiveness and community engagement

4.4.1. Comparative effectiveness research

Limited research exists on the comparative effectiveness of different mercury reduction strategies when evaluated from a comprehensive One Health perspective (UNEP, 2019; Wang et al., 2024). Studies are needed that evaluate the human health, wildlife, and ecosystem benefits of different intervention approaches while considering implementation costs, feasibility, and sustainability (Basu et al., 2023; Davies, 2023).

4.4.2. Technology transfer and capacity building

Research is needed to identify effective approaches for transferring mercury reduction technologies to developing countries and communities with limited technical and financial resources (Davies, 2023).

5. Discussion and conclusions

The current comprehensive analysis demonstrates that mercury contamination represents perhaps the most compelling contemporary example of a global environmental health challenge that demands One Health approaches integrating human health protection, wildlife conservation, and ecosystem management (Basu et al., 2023; Wang et al., 2024). The evidence synthesized through this One Health lens reveals fundamental conclusions that reshape the understanding of mercury risks and the institutional changes needed for effective management (Wilkening et al., 2025).

5.1. Key scientific findings and their implications

Mercury contamination exemplifies how industrial activities create cascading effects that propagate across environmental, wildlife, and human health domains through shared exposure pathways and toxicological mechanisms (Budnik and Casteleyn, 2019; Davies, 2023). The global nature of atmospheric mercury transport ensures that emission sources in one region affect human and ecosystem health thousands of kilometers away, highlighting the inadequacy of purely local or national approaches to mercury management (Streets et al., 2011; Yuan et al., 2023). The remarkable consistency of mercury-induced health effects across phylogenetically diverse species provides compelling evidence for shared toxicological mechanisms that validate integrated risk assessment approaches (Peterson et al., 2023; Scheuhammer et al., 2007). Neurological dysfunction, reproductive impairment, and immunosuppression occur across taxa with similar exposure-response relationships, supporting the use of wildlife and laboratory animal data to inform human health protection while simultaneously justifying ecosystem-based management approaches that protect multiple species simultaneously (Chételat et al., 2024). Aquatic food webs serve as the critical linkage connecting environmental contamination to both human and wildlife health through efficient bioaccumulation and biomagnification processes (Lavoie et al., 2013; Sinclair et al., 2024).

5.2. Implications for science, policy, and planetary health

The findings of the current review have significant implications across scientific, policy, and management domains. Scientifically, they emphasize the urgent need for transdisciplinary research that integrates toxicology, ecology, climatology, and social sciences to comprehensively characterize and mitigate mercury risks. From a policy perspective, the evidence supports enhancing and expanding the Minamata Convention's mandate by explicitly incorporating One Health principles to foster integrative, cross-sectoral strategies. This includes developing regulatory frameworks that address the climate-mercury nexus and prioritize environmental justice. In terms of management, the present review advocates shifting from reactive contamination control toward proactive prevention approaches that sustain ecosystem integrity as the foundation of human and wildlife health. Prioritizing interventions such as wetland restoration and emissions reduction can yield synergistic environmental and health co-benefits. Ultimately, framing mercury risk within a One Health context represents a critical model for advancing planetary health by illustrating the inseparable links between ecosystem protection and the well-being of all species, including humans.

5.3. Environmental justice and equity imperatives

Mercury contamination reveals stark patterns of environmental injustice that demand immediate attention and systemic intervention (Davies, 2023; Lucchini et al., 2025). ASGM communities, indigenous populations, and low-income communities near emission sources experience mercury exposures that can exceed background levels by considerable factors, while simultaneously lacking access to healthcare, environmental protection, and political representation (Basu et al., 2023; Gibb and O'Leary, 2014). These environmental justice dimensions create moral imperatives for mercury management that extend beyond technical risk reduction to address underlying structural inequities that create and perpetuate disproportionate exposure burdens.

5.4. Regulatory evolution and implementation challenges

The Minamata Convention represented a significant advance in international environmental governance, providing the first comprehensive global framework for mercury management. It incorporated important innovations, including technology transfer provisions, financial mechanisms, and stakeholder engagement requirements (UN, 2025; UNEP, 2019). Current regulatory approaches often treat human health protection, wildlife conservation, and environmental management as separate objectives, limiting their effectiveness in addressing the interconnected nature of mercury risks (Mason et al., 2012; Wang et al., 2024). Future regulatory frameworks need fundamental evolution

toward integrated approaches that explicitly recognize One Health principles, seeking management strategies that generate co-benefits across multiple health domains simultaneously. The 2023 amendments to the Minamata Convention have represented significant progress toward more comprehensive mercury management, particularly in addressing mercury-added products and emissions monitoring requirements (UN, 2025).

5.5. Scientific and technological frontiers

Significant advances in analytical chemistry, exposure assessment, biomarker development, and risk evaluation are needed to support effective One Health mercury management (Basu et al., 2023; Charkiewicz et al., 2025; Peterson et al., 2023). Omics technologies, including genomics, proteomics, and metabolomics, provide new opportunities for understanding mercury toxicity mechanisms, identifying sensitive biomarkers that could improve both individual clinical assessment and population health surveillance (Oliveira et al., 2020; Zhang et al., 2022).

5.6. Climate change adaptation and future scenarios

The emerging recognition of climate change as a significant modifier of mercury environmental fate and health risks represents a fundamental challenge that requires adaptive management approaches capable of responding to changing environmental conditions (Giang and Selin, 2016; Zhou et al., 2024). Climate change may also alter the geographic distribution of vulnerable populations and sensitive ecosystems, creating new environmental justice challenges, such as sea-level rise, extreme weather events, and changing precipitation patterns, which can lead to population movements and ecosystem shifts (Zhou et al., 2024). Mercury management frameworks must anticipate these changes and develop flexible approaches that can protect health under evolving demographic and environmental conditions.

5.7. Toward integrated mercury management: A path forward

Our thematic review, focusing on interdisciplinary connections rather than exhaustive quantification, highlights critical but understudied gaps in climate-mercury interactions and environmental equity dimensions, which are often missed by traditional systematic reviews. Addressing these challenges demands collaborative action among scientists, policymakers, industry, affected communities, and international bodies to develop adaptive solutions that tackle the root causes of mercury contamination while safeguarding vulnerable populations and ecosystems (UNEP, 2019; Wang et al., 2024).

5.7.1. Institutional transformation and capacity building

Effective One Health mercury management requires fundamental transformation of institutions to break down traditional disciplinary silos, creating new forms of collaboration across human health, veterinary medicine, environmental science, and policy sectors (Basu et al., 2023; CDC, 2025; Davies, 2023). This includes fostering "transdisciplinary" teams that not only work alongside each other but integrate knowledge and methodologies to create novel solutions (WHO, 2023).

5.7.2. Research and innovation priorities

Priority research areas include mechanistic understanding of low-dose chronic effects, mixture toxicology and cumulative risk assessment, ecosystem-level effects assessment, climate-mercury interactions, intervention effectiveness evaluation, and community-based participatory research approaches (Basu et al., 2023; Davies, 2023).

5.7.3. Measuring success and adaptive management

Success in One Health mercury management must be measured across multiple dimensions including environmental mercury concentrations, human and wildlife health outcomes, health equity indicators, community capacity measures, and ecosystem health metrics (Eagles-Smith et al., 2018; Peterson et al., 2023). The One Health approach to mercury management has both the potential for more effective environmental health protection and the institutional, scientific, and social transformations needed to realize this potential (Basu et al., 2023; Wang et al., 2024). Mercury contamination provides important lessons for managing other global environmental health challenges, highlighting the fundamental interconnections between human health, wildlife conservation, and environmental protection that demand integrated solutions. Addressing mercury contamination through a One Health lens is a critical step toward achieving the broader goals of planetary health, ensuring the well-being of both human civilizations and the natural systems on which they depend (Whitmee et al., 2015).

CRediT authorship contribution statement

Marília Cristina Oliveira Souza: Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Formal analysis. **Fernando Barbosa:** Jr, Writing – review & editing, Writing – original draft, Validation, Supervision, Formal analysis, Conceptualization. **Jose L. Domingo:** Writing – review & editing, Writing – original draft, Validation, Supervision, Methodology, Investigation, Conceptualization.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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No data was used for the research described in the article.

References

Aaseth, J., Wallace, D.R., Vejrup, K., Alexander, J., 2020. Methylmercury and developmental neurotoxicity: a global concern. Curr. Opin. Toxicol. 19, 80–87. https://doi.org/10.1016/j.cotox.2020.01.005.

Abbott, L.C., Nigussie, F., 2021. Mercury toxicity and neurogenesis in the Mammalian brain. Int. J. Mol. Sci. 22 (14), 7520. https://doi.org/10.3390/ijms22147520.

Al-Sulaiti, M.M., Soubra, L., Al-Ghouti, M.A., 2022. The causes and effects of mercury and methylmercury contamination in the marine environment: a review. Curr. Pollut. Rep. 8 (3), 249–272. https://doi.org/10.1007/s40726-022-00226-7.

Al-Sulaiti, M.M., Al-Ghouti, M.A., Ramadan, G.A., Soubra, L., 2023. Health risk assessment of methyl mercury from fish consumption in a sample of adult Qatari residents. Environ. Monit. Assess. 195 (5), 617. https://doi.org/10.1007/s10661-023-11194-w.

AMAP (Arctic Monitoring and Assessment Programme), 2021. AMAP assessment 2021: Mercury in the Arctic. Tromsø, Norway: arctic monitoring and assessment Programme. Available at: https://www.amap.no/documents/doc/amap-assessment-2021-mercury-in-the-arctic/3581. (Accessed 10 September 2025).

Aschner, M., Clarkson, T.W., 1988. Uptake of methylmercury in the rat brain: effects of amino acids. Brain Res. 462 (1), 31–39. https://doi.org/10.1016/0006-8993(88)90581-1.

AuYang, D., Chen, J., Zheng, W., Lang, Y., Wang, Y., Wang, Z., Zhang, Y., Liu, Y., Zhang, K., Cai, H., Yuan, W., Widory, D., 2022. Seasonal variations of the mercury multiple isotopic compositions of subrural and urban aerosols highlight an additional atmospheric Hg0 oxidation pathway. Frontiers Environ. Sci. 9, 773327. https://doi.org/10.3389/fenvs.2021.773327.

Azevedo, L.S., Pestana, I.A., Rocha, A.R.M., Meneguelli-Souza, A.C., Lima, C.A.I., Almeida, M.G., Bastos, W.R., Souza, C.M.M., 2018. Drought promotes increases in total mercury and methylmercury concentrations in fish from the lower Paraíba do Sul river, southeastern Brazil. Chemosphere 202, 483–490. https://doi.org/10.1016/j.chemosphere.2018.03.059.

Barbosa, F Jr, Fillion, M., Lemire, M., Passos, C.J., Rodrigues, J.L., Philibert, A., Guimarães, J.R., Mergler, D., 2009. Elevated blood lead levels in a riverside population in the Brazilian Amazon. Environ. Res. 109 (5), 594–599. https://doi.org/10.1016/j.envres.2009.03.005.

Barcelos, G.R., Grotto, D., de Marco, K.C., Valentini, J., Av, Lengert, de Oliveira, A.Á., Garcia, S.C., Braga, G.Ú., Schläwicke Engström, K., Cólus, I.M., Broberg, K., Barbosa Jr., F., 2013. Polymorphisms in glutathione-related genes modify mercury concentrations and antioxidant status in subjects environmentally exposed to methylmercury. Sci. Total Environ. 463–464, 319–325. https://doi.org/10.1016/j.scitotenv.2013.06.029.

Basta, P.C., Viana, P.V.S., Vasconcellos, A.C.S., Périssé, A.R.S., Hofer, C.B., Paiva, N.S., Kempton, J.W., Ciampi de Andrade, D., Oliveira, R.A.A., Achatz, R.W., Perini, J.A., Meneses, H.D.N.M., Hallwass, G., Lima, M.O., Jesus, I.M., Santos, C.C.R.D., Hacon, S. S., 2021. Mercury exposure in Munduruku Indigenous communities from Brazilian Amazon: methodological background and an overview of the principal results. Int J Environ Res Public Health 18 (17), 9222. https://doi.org/10.3390/ijerph18179222.

Basu, N., Horvat, M., Evers, D.C., Zastenskaya, I., Weihe, P., Tempowski, J., 2018. A State-of-the-Science review of mercury biomarkers in human populations worldwide between 2000 and 2018. Environ. Health Perspect. 126 (10), 106001. https://doi.org/10.1289/EHP3904.

Basu, N., Bastiansz, A., Dórea, J.G., Fujimura, M., Horvat, M., Shroff, E., Weihe, P., Zastenskaya, I., 2023. Our evolved understanding of the human health risks of mercury. Ambio 52 (5), 877–896. https://doi.org/10.1007/s13280-023-01831-6.

Bernhoft, R.A., 2012. Mercury toxicity and treatment: a review of the literature. J. Environ. Public Health 2012, 460508. https://doi.org/10.1155/2012/460508.

Berzas Nevado, J.J., Rodríguez Martín-Doimeadios, R.C., Guzmán Bernardo, F.J., Jiménez Moreno, M., Herculano, A.M., do Nascimento, J.L., Crespo-López, M.E., 2010. Mercury in the Tapajós River basin, Brazilian Amazon: a review. Environ. Int. 36 (6), 593–608. https://doi.org/10.1016/j.envint.2010.03.011.

Bjørklund, G., Peana, M., Dadar, M., Chirumbolo, S., Aaseth, J., Martins, N., 2020. Mercury-induced autoimmunity: drifting from micro to macro concerns on autoimmune disorders. Clin Immunol 213, 108352. https://doi.org/10.1016/j.clim.2020.108352.

Bocato, M.Z., Bianchi Ximenez, J.P., Hoffmann, C., Barbosa, F., 2019. An overview of the current progress, challenges, and prospects of human biomonitoring and exposome studies. J. Toxicol. Environ. Health B Crit. Rev. 22 (5–6), 131–156. https://doi.org/10.1080/10937404.2019.1661588.

Branco, V., Caito, S., Farina, M., Teixeira da Rocha, J., Aschner, M., Carvalho, C., 2017. Biomarkers of mercury toxicity: past, present, and future trends. J. Toxicol. Environ. Health B Crit. Rev. 20 (3), 119–154. https://doi.org/10.1080/10937404.2017.1289834.

Brasso, R., Rittenhouse, K.A., Winder, V.L., 2020. Do songbirds in wetlands show higher mercury bioaccumulation relative to conspecifics in non-wetland habitats? Ecotoxicology 29 (8), 1183–1194. https://doi.org/10.1007/s10646-020-02160-0.

Bridges, K.N., Furin, C.G., Gerlach, R.F., 2020. Subsistence fish consumption in rural Alaska: using regional monitoring data to evaluate risk and bioavailability of dietary methylmercury. Sci. Total Environ. 736, 139676. https://doi.org/10.1016/j.scitotenv.2020.139676.

Budnik, L.T., Casteleyn, L., 2019. Mercury pollution in modern times and its socio-medical consequences. Sci. Total Environ. 654, 720–734. https://doi.org/10.1016/j.scitotenv.2018.10.408.

Canela, T.A., Monteiro, L.C., Cabral, C.D.S., Ximenes, F.D.S., Oliveira, I.A.D.S., Bernardi, J.V.E., Almeida, R., Bastos, W.R., 2024. Mercury in fish and human hair and estimated dietary intake in a Riverside community of the Madeira River Basin in the Brazilian Amazon. Toxics 12 (3), 208. https://doi.org/10.3390/toxics12030208.

CDC (Centers for Disease Control and Prevention), 2025. About One Health. CDC, Atlanta. Available at: https://www.cdc.gov/one-health/about/index.html. (Accessed 15 September 2025).

Cediel-Ulloa, A., Lindner, S., Rüegg, J., Broberg, K., 2023. Epigenetics of methylmercury. Neurotoxicology 97, 34–46. https://doi.org/10.1016/j.neuro.2023.05.004.

Cezarette, G.N., Esplugas, J., Cruz, J.C., Rocha, B.A., Cassulatti Dos Santos, L., Bueno, M., Zayas, Z.P., Barbosa Jr., F., 2025. Exploring mercury and selenium dynamics in Amazonian human populations: insights from urine, blood, and plasma analyses. Chemosphere 375, 144258. https://doi.org/10.1016/j.chemosphere.2025.144258.

Charkiewicz, A.E., Omeljaniuk, W.J., Garley, M., Nikliński, J., 2025. Mercury exposure and health effects: what do we really know? Int. J. Mol. Sci. 26 (5), 2326. https://doi.org/10.3390/ijms26052326.

Chen, C.Y., Evers, D.C., 2023. Global mercury impact synthesis: processes in the Southern Hemisphere. Ambio 52 (5), 827–832. https://doi.org/10.1007/s13280-023-01842-3.

Chételat, J., Heath, J.P., Arragutainaq, L., Lameboy, J., McClelland, C., Mickpegak, R., 2024. River influence on mercury bioaccumulation in the coastal food web of Eeyou Istchee, James Bay, Canada. FACETS 9, 1–13. https://doi.org/10.1139/facets-2023-0121.

Clarkson, T.W., Magos, L., 2006. The toxicology of mercury and its chemical compounds. Crit. Rev. Toxicol. 36 (8), 609–662. https://doi.org/10.1080/10408440600845619.

Coe, G.L., Krout, I.N., Munro-Ehrlich, M., Beamish, C.R., Vorojeikina, D., Colman, D.R., Boyd, E.J., Walk, S.T., Rand, M.D., 2023. Assessing the role of the gut microbiome in methylmercury demethylation and elimination in humans and gnotobiotic mice. Arch. Toxicol. 97 (9), 2399–2418. https://doi.org/10.1007/s00204-023-03548-7.

Cristol, D.A., Brasso, R.L., Condon, A.M., Fovargue, R.E., Friedman, S.L., Hallinger, K.K., Monroe, A.P., White, A.E., 2008. The movement of aquatic mercury through terrestrial food webs. Science 320 (5874), 335. https://doi.org/10.1126/science.1154082.

Cruz, J.C., Cassulatti Dos Santos, L., Devoz, P.P., Gallimberti, M., Cerazette, G.N., de Assis Aguilar Duarte, N., Eloísa de Lima, L., Nunes, E.A., de Medeiros Soares, J., Laise Dos Santos Pinto, M., da Silva Soares, G., Santos de Souza, S., Paradell, N.G., Bueno, M., Rocha, B.A., Barcelos, G.R.M., Meneses, H.D.N.M., Domingo, J.L., Zayas, Z.P., Barbosa Jr., F., 2024. Blood levels of 21 metals and metalloids in riverside villagers of the Brazilian Amazon: a human biomonitoring study with associations with sociodemographic, dietary, and lifestyle factors. Environ. Res. 261, 119767. https://doi.org/10.1016/j.envres.2024.119767.

Dack, K., Fell, M., Taylor, C.M., Havdahl, A., Lewis, S.J., 2022. Prenatal mercury exposure and neurodevelopment up to the Age of 5 years: a systematic review. Int J Environ Res Public Health 19 (4), 1976. https://doi.org/10.3390/ijerph19041976.

Dai, M.Q., Geyman, B.M., Hu, X.C., Thackray, C.P., Sunderland, E.M., 2023. Sociodemographic disparities in mercury exposure from United States coal-fired power plants. Environ. Sci. Technol. Lett. 10 (7), 589–595. https://doi.org/10.1021/acs.estlett.3c00216.

Danasekaran, R., 2024. One health: a holistic approach to tackling global health issues. Indian J. Community Med. 49 (2), 260–263. https://doi.org/10.4103/ijcm.ijcm_521_23.

Davies, T.C., 2023. An updated review of the salient geomedical aspects of mercury for enhancement of data quality in simulation modelling and other prognostic applications: africa case descriptions. Frontiers Anal Sci 3, 1069678. https://doi.org/10.3389/frans.2023.1069678.

de Moraes, Pontes JG., Rocha, B.A., Cruz, J.C., Cezarette, G.N., Omage, F.B., Tasic, L., Barbosa Jr., F., 2025. Unraveling mercury-selenium interactions through metabolomics: impacts on riverside communities in the Amazon. Environ Pollut 376, 126398. https://doi.org/10.1016/j.envpol.2025.126398.

Debes, F., Budtz-Jørgensen, E., Weihe, P., White, R.F., Grandjean, P., 2006. Impact of prenatal methylmercury exposure on neurobehavioral function at age 14 years. Neurotoxicol. Teratol. 28 (5), 536–547. https://doi.org/10.1016/j.ntt.2006.02.005.

Díez, S., 2009. Human health effects of methylmercury exposure. Rev. Environ. Contam. Toxicol. 198, 111–132. https://doi.org/10.1007/978-0-387-09647-6_3.

Dietz, R., Sonne, C., Basu, N., Braune, B., O'Hara, T., Letcher, R.J., Scheuhammer, T., Andersen, M., Andreasen, C., Andriashek, D., Asmund, G., Aubail, A., Baagøe, H., Born, E.W., Chan, H.M., Derocher, A.E., Grandjean, P., Knott, K., Kirkegaard, M., Krey, A., Lunn, N., Messier, F., Obbard, M., Olsen, M.T., Ostertag, S., Peacock, E., Renzoni, A., Rigét, F.F., Skaare, J.U., Stern, G., Stirling, I., Taylor, M., Wiig, Ø., Wilson, S., Aars, J., 2013. What are the toxicological effects of mercury in Arctic biota? Sci. Total Environ. 443, 775–790. https://doi.org/10.1016/j.scitotenv.2012.11.046.

Domingo, J.L., Bocio, A., Falcó, G., Llobet, J.M., 2007. Benefits and risks of fish consumption Part I. A quantitative analysis of the intake of omega-3 fatty acids and chemical contaminants. Toxicology 230 (2–3), 219–226. https://doi.org/10.1016/j.tox.2006.

Domingo, J.L., Souza, M.C.O., Nadal, M., Barbosa, F., 2025. A one Health approach to polybrominated diphenyl ethers (PBDEs): integrating human, animal, and environmental health perspectives. Chemosphere 386, 144644. https://doi.org/10.1016/j.chemosphere.2025.144644.

Driscoll, C.T., Mason, R.P., Chan, H.M., Jacob, D.J., Pirrone, N., 2013. Mercury as a global pollutant: sources, pathways, and effects. Environ. Sci. Technol. 47 (10), 4967–4983. https://doi.org/10.1021/es305071v.

Duan, H., Yu, L., Tian, F., Zhai, Q., Fan, L., Chen, W., 2020. Gut microbiota: a target for heavy metal toxicity and a probiotic protective strategy. Sci. Total Environ. 742, 140429. https://doi.org/10.1016/j.scitotenv.2020.140429.

Dufault, R., Schnoll, R., Lukiw, W.J., Leblanc, B., Cornett, C., Patrick, L., Wallinga, D., Gilbert, S.G., Crider, R., 2009 Oct 27. Mercury exposure, nutritional deficiencies and metabolic disruptions may affect learning in children. Behav Brain Funct 5, 44. https://doi.org/10.1186/1744-9081-5-44.

Dykstra, C.R., Route, W.T., Williams, K.A., Meyer, M.W., Key, R.L., 2019. Trends and patterns of PCB, DDE, and mercury contamination in bald eagle nestlings in the upper Midwest. J Great Lakes Res 45 (2), 252–262. https://doi.org/10.1016/j.jglr.2019.01.010.

Eagles-Smith, C.A., Silbergeld, E.K., Basu, N., Bustamante, P., Diaz-Barriga, F., Hopkins, W.A., Kidd, K.A., Nyland, J.F., 2018. Modulators of mercury risk to wildlife and humans in the context of rapid global change. Ambio 47 (2), 170–197. https://doi.org/10.1007/s13280-017-1011-x.

Evers, D.C., Savoy, L.J., DeSorbo, C.R., Yates, D.E., Hanson, W., Taylor, K.M., Siegel, L.S., Cooley Jr., J.H., Bank, M.S., Major, A., Munney, K., Mower, B.F., Vogel, H.S., Schoch, N., Pokras, M., Goodale, M.W., Fair, J., 2008. Adverse effects from environmental mercury loads on breeding common loons. Ecotoxicology 17 (2), 69–81. https://doi.org/10.1007/s10646-007-0168-7.

Farella, N., Lucotte, M., Davidson, R., Daigle, S., 2006. Mercury release from deforested soils triggered by base cation enrichment. Sci. Total Environ. 368 (1), 19–29. https://doi.org/10.1016/j.scitotenv.2006.04.025.

Farina, M., Aschner, M., 2017. Methylmercury-induced neurotoxicity: focus on pro-oxidative events and related consequences. Adv Neurobiol 18, 267–286. https://doi.org/10.1007/978-3-319-60189-2_13.

Gallorini, A., Loizeau, J.L., 2021. Mercury methylation in oxic aquatic macro-environments: a review. J Limnology 2080 (2). https://doi.org/10.4081/jlimnol.2021.2007.

Gao, P., 2021. The exposome in the era of one health. Environ. Sci. Technol. 55 (5), 2790–2799. https://doi.org/10.1021/acs.est.0c07033.

Genchi, G., Sinicropi, M.S., Carocci, A., Lauria, G., Catalano, A., 2017 Jan 12. Mercury Exposure and Heart Diseases. Int J Environ Res Public Health 14 (1), 74. https://doi.org/10.3390/ijerph14010074.

Giang, A., Selin, N.E., 2016. Benefits of mercury controls for the United States. Proc. Natl. Acad. Sci. U. S. A. 113 (2), 286–291. https://doi.org/10.1073/pnas.1514395113.

Gibb, H., O'Leary, K.G., 2014. Mercury exposure and health impacts among individuals in the artisanal and small-scale gold mining community: a comprehensive review. Environ. Health Perspect. 122 (7), 667–672. https://doi.org/10.1289/ehp.1307864.
Gilmour, C.C., Podar, M., Bullock, A.L., Graham, A.M., Brown, S.D., Somenahally, A.C., Johs, A., Hurt Jr., R.A., Bailey, K.L., Elias, D.A., 2013. Mercury methylation by novel microorganisms from new environments. Environ. Sci. Technol. 47 (20), 11810–11820. https://doi.org/10.1021/es403075t.
González, N., Calderón, J., Rúbies, A., Timoner, I., Castell, V., Domingo, J.L., Nadal, M., 2019. Dietary intake of arsenic, cadmium, mercury and lead by the population of Catalonia, Spain: analysis of the temporal trend. Food Chem. Toxicol. 132, 110721. https://doi.org/10.1016/j.fct.2019.110721.
González, N., Souza, M.C.O., Cezarette, G.N., Rocha, B.A., Devoz, P.P., Dos Santos, L.C., Barcelos, G.R.M., Nadal, M., Domingo, J.L., Barbosa Jr., F., 2024. Evaluation of exposure to multiple organic pollutants in riparian communities of the Brazilian Amazon: screening levels and potential health risks. Sci. Total Environ. 908, 168294. https://doi.org/10.1016/j.scitotenv.2023.168294.
Grandjean, P., Landrigan, P.J., 2014. Neurobehavioural effects of developmental toxicity. Lancet Neurol. 13 (3), 330–338. https://doi.org/10.1016/S1474-4422(13)70278-3.
Grandjean, P., Weihe, P., Debes, F., Choi, A.L., Budtz-Jørgensen, E., 2014. Neurotoxicity from prenatal and postnatal exposure to methylmercury. Neurotoxicol. Teratol. 43, 39–44. https://doi.org/10.1016/j.ntt.2014.03.004.
Grotto, D., Valentini, J., Fillion, M., Passos, C.J., Garcia, S.C., Mergler, D., Barbosa Jr., F., 2010. Mercury exposure and oxidative stress in communities of the Brazilian Amazon. Sci. Total Environ. 408 (4), 806–811. https://doi.org/10.1016/j.scitotenv.2009.10.053.
Guallar, E., Sanz-Gallardo, M.I., van't Veer, P., Bode, P., Aro, A., Gómez-Aracena, J., Kark, J.D., Riemersma, R.A., Martín-Moreno, J.M., Kok, F.J., 2002. Heavy metals and myocardial infarction study group. mercury, fish oils, and the risk of myocardial infarction. N. Engl. J. Med. 347 (22), 1747–1754. https://doi.org/10.1056/NEJMoa020157.
Guimaraes, J.R., 2020. Mercury in the Amazon: problem or opportunity? A commentary on 30 years of research on the subject. Elementa: Science of the Anthropocene 8 (1), 32. https://doi.org/10.1525/elementa.032.
Guimarães, K.L.A., do Nascimento Andrade, S.J., Liscano-Carreño, A.A., de Oliveira, R. B., Rodrigues, L.R.R., 2024. Systematic review and spatiotemporal assessment of Mercury concentration in fish from the Tapajós River Basin: implications for environmental and human health. ACS Environ Au 5 (1), 86–100. https://doi.org/10.1021/acsenvironau.4c00053.
Ha, E., Basu, N., Bose-O'Reilly, S., Dórea, J.G., McSorley, E., Sakamoto, M., Chan, H.M., 2017. Current progress on understanding the impact of mercury on human health. Environ. Res. 152, 419–433. https://doi.org/10.1016/j.envres.2016.06.042.
He, K., Xun, P., Liu, K., Morris, S., Reis, J., Guallar, E., 2013. Mercury exposure in young adulthood and incidence of diabetes later in life: the CARDIA Trace Element Study. Diabetes Care 36 (6), 1584–1589. https://doi.org/10.2337/dc12-1842.
Howie, M.G., Jackson, A.K., Cristol, D.A., 2018. Spatial extent of mercury contamination in birds and their prey on the floodplain of a contaminated river. Sci. Total Environ. 630, 1446–1452. https://doi.org/10.1016/j.scitotenv.2018.02.272.
Hu, X.F., Eccles, K.M., Chan, H.M., 2017. High selenium exposure lowers the odds ratios for hypertension, stroke, and myocardial infarction associated with mercury exposure among Inuit in Canada. Environ. Int. 102, 200–206. https://doi.org/10.1016/j.envint.2017.03.002.
Hu, X.F., Loan, A., Chan, H.M., 2025. Re-thinking the link between exposure to mercury and blood pressure. Arch. Toxicol. 99 (2), 481–512. https://doi.org/10.1007/s00204-024-03919-8.
Jacobs, S., Sioen, I., Jacxsens, L., Domingo, J.L., Sloth, J.J., Marques, A., Verbeke, W., 2017. Risk assessment of methylmercury in five European countries considering the national seafood consumption patterns. Food Chem. Toxicol. 104, 26–34. https://doi.org/10.1016/j.fct.2016.10.026.
Kang, B., Wang, J., Guo, S., Yang, L., 2024. Mercury-induced toxicity: mechanisms, molecular pathways, and gene regulation. Sci. Total Environ. 943, 173577. https://doi.org/10.1016/j.scitotenv.2024.173577.
Karimi, R., Fitzgerald, T.P., Fisher, N.S., 2012. A quantitative synthesis of mercury in commercial seafood and implications for exposure in the United States. Environ. Health Perspect. 120 (11), 1512–1519. https://doi.org/10.1289/ehp.1205122.
Ke, T., Tinkov, A.A., Skalny, A.V., Santamaria, A., Rocha, J.B.T., Bowman, A.B., Chen, W., Aschner, M., 2023. Epigenetics and methylmercury-induced neurotoxicity, evidence from experimental studies. Toxics 11 (1), 72. https://doi.org/10.3390/toxics11010072.
Kenow, K.P., Gutreuter, S., Hines, R.K., Meyer, M.W., Fournier, F., Karasov, W.H., 2003. Effects of methyl mercury exposure on the growth of juvenile common loons. Ecotoxicology 12 (1–4), 171–182. https://doi.org/10.1023/a:1022598525891.
Kenow, K.P., Meyer, M.W., Rossmann, R., Gendron-Fitzpatrick, A., Gray, B.R., 2011. Effects of injected methylmercury on the hatching of common loon (Gavia immer) eggs. Ecotoxicology 20 (7), 1684–1693. https://doi.org/10.1007/s10646-011-0743-9.
Khan, F., Momtaz, S., Abdollahi, M., 2019. The relationship between mercury exposure and epigenetic alterations regarding human health, risk assessment and diagnostic strategies. J. Trace Elem. Med. Biol. 52, 37–47. https://doi.org/10.1016/j.jtemb.2018.11.006.
Kung, H.C., Wu, C.H., Huang, B.W., Chang-Chien, G.P., Mutuku, J.K., Lin, W.C., 2024. Mercury abatement in the environment: insights from industrial emissions and fates in the environment. Heliyon 10 (7), e28253. https://doi.org/10.1016/j.heliyon.2024.e28253.
Lavoie, R.A., Jardine, T.D., Chumchal, M.M., Kidd, K.A., Campbell, L.M., 2013. Biomagnification of mercury in aquatic food webs: a worldwide meta-analysis. Environ. Sci. Technol. 47 (23), 13385–13394. https://doi.org/10.1021/es403103t.
Leal-Nazaré, C.G., Arrifano, G.P., Lopes-Araújo, A., Santos-Sacramento, L., Barthelemy, J.L., Soares-Silva, I., Crespo-Lopez, M.E., Augusto-Oliveira, M., 2024. Methylmercury neurotoxicity: beyond the neurocentric view. Sci. Total Environ. 920, 170939. https://doi.org/10.1016/j.scitotenv.2024.170939.
Lemire, M., Fillion, M., Frenette, B., Mayer, A., Philibert, A., Passos, C.J., Guimarães, J. R., Barbosa, F.J., Mergler, D., 2010. Selenium and mercury in the Brazilian Amazon: opposing influences on age-related cataracts. Environ. Health Perspect. 118 (11), 1584–1589. https://doi.org/10.1289/ehp.0901284.
Lemire, M., Fillion, M., Frenette, B., Passos, C.J., Guimarães, J.R., Barbosa Jr., F., Mergler, D., 2011. Selenium from dietary sources and motor functions in the Brazilian Amazon. Neurotoxicology 32 (6), 944–953. https://doi.org/10.1016/j.neuro.2011.04.005.
Lescord, G.L., Simard, J., Johnston, T.A., Seguin, J., Farrell, C.E., O'Driscoll, N.J., O'Connor, C.M., 2024. The long-term effects of water resource development on blood mercury levels and the trophic ecology of Namew (lake sturgeon, Acipenser fulvescens). FACETS 9 (1), 1–13. https://doi.org/10.1139/facets-2023-0151.
Li, P., Feng, X., Qiu, G., 2010. Methylmercury exposure and health effects from rice and fish consumption: a review. Int J Environ Res Public Health 7 (6), 2666–2691. https://doi.org/10.3390/ijerph7062666.
Li, M.L., Thackray, C.P., Lam, V.W.Y., Cheung, W.W.L., Sunderland, E.M., 2024. Global fishing patterns amplify human exposures to methylmercury. Proc. Natl. Acad. Sci. U. S. A. 121 (40), e2405898121. https://doi.org/10.1073/pnas.2405898121.
Lindberg, S., Bullock, R., Ebinghaus, R., Engstrom, D., Feng, X., Fitzgerald, W., Pirrone, N., Prestbo, E., Seigneur, C., 2007. Panel on source attribution of atmospheric mercury. A synthesis of progress and uncertainties in attributing the sources of mercury in deposition. Ambio 36 (1), 19–32. https://doi.org/10.1579/0044-7447(2007)36[19:asopau]2.0.co;2.
Liu, M., Xiao, W., Zhang, Q., Shi, L., Wang, X., Xu, Y., 2020. Methylmercury bioaccumulation in deepest ocean fauna: implications for ocean mercury biotransport through food webs. Environ Sci Technol Letters. 7 (7), 469–476. https://doi.org/10.1021/acs.estlett.0c00299.
Liu, T., An, M., Chen, J., Liu, Y., Chao, L., Liu, J., Zhang, M., 2024. Variations in methylmercury contamination levels and associated health risks in different fish species across three coastal bays in China. Frontiers Environ Sci. 12, 1376882. https://doi.org/10.3389/fenvs.2024.1376882.
Lucchini, R.G., Basta, P.C., Crespo-Lopez, M.E., Gastañaga, M.D.C., O'Callaghan-Gordo, C., Olivero-Verbel, J., Vega, C., Moncada Barbosa, S.M., Espinal, C., Felty, Q., Deoraj, A., 2025. Mercury contamination and Co-exposures in the Amazon Basin: at the Center of the planetary environmental crisis. Ann. Glob. Health 91 (1), 42. https://doi.org/10.5334/aogh.4817.
Maraslis, F.T., Duarte, N.A.A., Nunes, E.A., de Lima, L.E., Serpeloni, J.M., Horvat, M., Domingo, J.L., Barbosa Jr., F., Barcelos, G.R.M., 2025. Polymorphisms in Hg-related genes and their impact on mercury burden and oxidative stress biomarkers among riverside Amazonian populations. Chemosphere 387, 144646. https://doi.org/10.1016/j.chemosphere.2025.144646.
Marvin-DiPasquale, M., Windham-Myers, L., Agee, J.L., Kakouros, E., Kieu le, H., Fleck, J.A., Alpers, C.N., Stricker, C.A., 2014. Methylmercury production in sediment from agricultural and non-agricultural wetlands in the Yolo Bypass, California, USA. Sci. Total Environ. 484, 288–299. https://doi.org/10.1016/j.scitotenv.2013.09.098.
Mason, R.P., Choi, A.L., Fitzgerald, W.F., Hammerschmidt, C.R., Lamborg, C.H., Soerensen, A.L., Sunderland, E.M., 2012. Mercury biogeochemical cycling in the ocean and policy implications. Environ. Res. 119, 101–117. https://doi.org/10.1016/j.envres.2012.03.013.
McClelland, C., Chételat, J., Conlan, K., Aitken, A., Forbes, M.R., Majewski, A., 2024. Methylmercury dietary pathways and bioaccumulation in Arctic benthic invertebrates of the Beaufort Sea. Arctic Science 10 (2), 305–320. https://doi.org/10.1139/as-2023-0021.
Mozaffarian, D., Rimm, E.B., 2006. Fish intake, contaminants, and human health: evaluating the risks and the benefits. JAMA 296 (15), 1885–1899. https://doi.org/10.1001/jama.296.15.1885.
Nyland, J.F., Fillion, M., Barbosa, F Jr, Shirley, D.L., Chine, C., Lemire, M., Mergler, D., Silbergeld, E.K., 2011. Biomarkers of methylmercury exposure immunotoxicity among fish consumers in Amazonian Brazil. Environ. Health Perspect. 119 (12), 1733–1738. https://doi.org/10.1289/ehp.1103741.
Obrist, D., Kirk, J.L., Zhang, L., Sunderland, E.M., Jiskra, M., Selin, N.E., 2018. A review of global environmental mercury processes in response to human and natural perturbations: changes of emissions, climate, and land use. Ambio 47 (2), 116–140. https://doi.org/10.1007/s13280-017-1004-9.
Olawade, D.B., Wada, O., Ige, A., Egbewole, B., Olojo, A., Oladapo, B., 2024. Artificial intelligence in environmental monitoring: advancements, challenges, and future directions. Hyg. Environ. Heal. Adv 12, 100114. https://doi.org/10.1016/j.heha.2024.100114.
Oliveira, C.S., Segatto, A.L.A., Nogara, P.A., Piccoli, B.C., Loreto, É.L.S., Aschner, M., Rocha, J.B.T., 2020. Transcriptomic and proteomic tools in the Study of Hg toxicity: what is missing? Front. Genet. 11, 425. https://doi.org/10.3389/fgene.2020.00425.
Orellana, J.D.Y., Parry, L., Santos, F.S.D., Moreira, L.R., Carignano Torres, P., Balieiro, A. A.D.S., Fonseca, F.R., Moraga, P., Chacón-Montalván, E.A., 2025. Estimating double burden of malnutrition among rural and urban children in Amazonia using Bayesian latent models. Front. Public Health 13, 1481397. https://doi.org/10.3389/fpubh.2025.1481397.
Ouédraogo, O., Chételat, J., Amyot, M., 2015. Bioaccumulation and trophic transfer of mercury and selenium in African sub-tropical fluvial reservoirs food webs (Burkina Faso). PLoS One 10 (4), e0123048. https://doi.org/10.1371/journal.pone.0123048.

Pacyna, J.M., 2020. Recent advances in mercury research. Sci. Total Environ. 738, 139955. https://doi.org/10.1016/j.scitotenv.2020.139955.
Pan, K., Xu, J., Li, F., Yu, H., Yu, J., 2025. The association between mercury exposure during pregnancy and adverse birth outcomes: a systematic review and meta-analysis. Environ. Res. 264 (Pt 1), 120357. https://doi.org/10.1016/j.envres.2024.120357.
Pestana, I., Azevedo, L.S., Bastos, W.R., de Souza, C., 2019. The impact of hydroelectric dams on mercury dynamics in South America: a review. Chemosphere 219, 546–556. https://doi.org/10.1016/j.chemosphere.2018.12.035.
Peterson, S.H., Ackerman, J.T., Costa, D.P., 2015. Marine foraging ecology influences mercury bioaccumulation in deep-diving northern elephant seals. Proc. Biol. Sci. 282 (1810), 20150710. https://doi.org/10.1098/rspb.2015.0710.
Peterson, S.H., Ackerman, J.T., Holser, R.R., McDonald, B.I., Costa, D.P., Crocker, D.E., 2023. Mercury bioaccumulation and cortisol interact to influence endocrine and immune biomarkers in a free-ranging marine mammal. Environ. Sci. Technol. 57 (14), 5678–5692. https://doi.org/10.1021/acs.est.2c08974.
Pinto, D.V., Raposo, R.S., Matos, G.A., Alvarez-Leite, J.I., Malva, J.O., Oriá, R.B., 2020. Methylmercury interactions with Gut Microbiota and potential modulation of neurogenic niches in the brain. Front. Neurosci. 14, 576543. https://doi.org/10.3389/fnins.2020.576543.
Poblete, Y., Contreras, C., Fernández, C., Flores, C.R., Vega, P., Ávila, M., Sabat, P., 2023. Geographic variation in the altitudinal migration patterns, body size, oxidative status and exploratory behavior in a neotropical bird. Ecol. Evol. 13 (3), e9941. https://doi.org/10.1002/ece3.9941.
Reuben, A., Frischtak, H., Berky, A., Ortiz, E.J., Morales, A.M., Hsu-Kim, H., Pendergast, L.L., Pan, W.K., 2020. Elevated hair mercury levels are associated with neurodevelopmental deficits in children living near artisanal and small-scale gold mining in Peru. GeoHealth 4 (5). https://doi.org/10.1029/2019GH000222 e2019GH000222.
Rimmer, C.C., Mcfarland, K.P., Evers, D.C., Miller, E.K., Aubry, Y., Busby, D., Taylor, R. J., 2005. Mercury concentrations in Bicknell's thrush and other insectivorous passerines in Montane forests of northeastern North America. Ecotoxicology 14 (1–2), 223–240. https://doi.org/10.1007/s10646-004-6270-1.
Roffler, G.H., Gastaldi, A., Lieske, C., Beckmen, K., Castellini, J.M., Barst, B.D., 2025. Switching to marine prey leads to unprecedented mercury concentrations in a population of coastal Alaska wolves. Sci. Total Environ. 980, 179542. https://doi.org/10.1016/j.scitotenv.2025.179542.
Roman, H.A., Walsh, T.L., Coull, B.A., Dewailly, É., Guallar, E., Hattis, D., Mariën, K., Schwartz, J., Stern, A.H., Virtanen, J.K., Rice, G., 2011. Evaluation of the cardiovascular effects of methylmercury exposures: current evidence supports development of a dose-response function for regulatory benefits analysis. Environ. Health Perspect. 119 (5), 607–614. https://doi.org/10.1289/ehp.1003012.
Rothenberg, S.E., Windham-Myers, L., Creswell, J.E., 2014. Rice methylmercury exposure and mitigation: a comprehensive review. Environ. Res. 133, 407–423. https://doi.org/10.1016/j.envres.2014.03.001.
Roy, C., Tremblay, P.Y., Ayotte, P., 2017. Is mercury exposure causing diabetes, metabolic syndrome and insulin resistance? A systematic review of the literature. Environ. Res. 156, 747–760. https://doi.org/10.1016/j.envres.2017.04.038.
Scheuhammer, A.M., Meyer, M.W., Sandheinrich, M.B., Murray, M.W., 2007. Effects of environmental methylmercury on the health of wild birds, mammals, and fish. Ambio 36 (1), 12–18. https://doi.org/10.1579/0044-7447(2007)36[12:eoemot]2.0.co;2.
Schuster, P.F., Schaefer, K.M., Aiken, G.R., Antweiler, R.C., Dewild, J.F., Gryziec, J.D., Gusmeroli, A., Hugelius, G., Jafarov, E., Krabbenhoft, D.P., Liu, L., Herman-Mercer, N., Mu, C., Roth, D.A., Schaefer, T., Striegl, R.G., Wickland, K.P., Zhang, T., 2018. Permafrost stores a globally significant amount of mercury. Geophys. Res. Lett. 45 (3), 1463–1471. https://doi.org/10.1002/2017GL075571.
Selin, H., Selin, N.E., 2022. From Stockholm to Minamata and beyond: governing mercury pollution for a more sustainable future. One Earth 5 (10), 1109–1125. https://doi.org/10.1016/j.oneear.2022.09.001.
Sexton, K., Linder, S.H., 2010. The role of cumulative risk assessment in decisions about environmental justice. Int J Environ Res Public Health 7 (11), 4037–4049. https://doi.org/10.3390/ijerph7114037.
Sinclair, C.A., Garcia, T.S., Eagles-Smith, C.A., 2024. A meta-analysis of Mercury biomagnification in freshwater Predatory invertebrates: community diversity and dietary exposure drive variability. Environ. Sci. Technol. 58 (43), 19429–19439. https://doi.org/10.1021/acs.est.4c05920.
Sisma-Ventura, G., Silverman, J., Guy-Haim, T., Stern, N., Shachnai, A., Mar Mori, M., Abu Khadra, M., Jacobson, Y., Segal, Y., Katz, T., Herut, B., 2024. Accumulation of total mercury in deep-sea sediments and biota across a bathymetric gradient in the Southeastern Mediterranean Sea. Chemosphere 351, 141201. https://doi.org/10.1016/j.chemosphere.2024.141201.
Sonke, J.E., Angot, H., Zhang, Y., Poulain, A., Björn, E., Schartup, A., 2023. Global change effects on biogeochemical mercury cycling. Ambio 52 (5), 853–876. https://doi.org/10.1007/s13280-023-01855-y.
Spiegel, S.J., 2009. Occupational health, mercury exposure, and environmental justice: learning from experiences in Tanzania. Am J Public Health 99 (Suppl. 3), S550–S558. https://doi.org/10.2105/AJPH.2008.148940. Suppl 3.
Streets, D.G., Devane, M.K., Lu, Z., Bond, T.C., Sunderland, E.M., Jacob, D.J., 2011. All-time releases of mercury to the atmosphere from human activities. Environ. Sci. Technol. 45 (24), 10485–10491. https://doi.org/10.1021/es202765m.
Sun, Q., Li, Y., Shi, L., Hussain, R., Mehmood, K., Tang, Z., Zhang, H., 2022. Heavy metals induced mitochondrial dysfunction in animals: molecular mechanism of toxicity. Toxicology 469, 153136. https://doi.org/10.1016/j.tox.2022.153136.
Sunderland, E.M., Li, M., Bullard, K., 2018. Decadal changes in the edible supply of seafood and methylmercury exposure in the United States. Environ. Health Perspect. 126 (1), 017006. https://doi.org/10.1289/EHP2644.
Tian, X., Lin, X., Zhao, J., Cui, L., Gao, Y., Yu, Y.L., Li, B., Li, Y.F., 2023. Gut as the target tissue of mercury and the extraintestinal effects. Toxicology 484, 153396. https://doi.org/10.1016/j.tox.2022.153396.
Toyama, T., Xu, S., Kanemitsu, Y., Hasegawa, T., Noguchi, T., Lee, J.Y., Matsuzawa, A., Naganuma, A., Hwang, G.W., 2023. Methylmercury directly modifies the 105th cysteine residue in oncostatin M to promote binding to tumor necrosis factor receptor 3 and inhibit cell growth. Arch. Toxicol. 97 (7), 1887–1897. https://doi.org/10.1007/s00204-023-03520-5.
UN, United Nations. The Minamata Convention on Mercury: 8 years of global progress, August 2025. Available at: https://www.un.org/en/delegate/minamata-convention-mercury-8-years-global-progress. (Accessed 5 September 2025).
United Nations Environment Program, 2019. Global Mercury Assessment 2018. UN Environment Program, Chemicals and Health Branch, Geneva, Switzerland. Available at: https://www.unep.org/resources/publication/global-mercury-assessment-2018. (Accessed 10 September 2025).
Vergara-Murillo, F., González-Ospino, S., Cepeda-Ortega, N., Pomares-Herrera, F., Johnson-Restrepo, B., 2022. Adverse health effects and Mercury exposure in a Colombian artisanal and small-scale gold mining community. Toxics 10 (12), 723. https://doi.org/10.3390/toxics10120723.
Waack, A., Ranabothu, M., Vattipally, V., 2022. Mercury poisoning from artisanal gold mining equipment. Radiol. Case Rep. 18 (2), 607–609. https://doi.org/10.1016/j.radcr.2022.10.099.
Wang, Q., Kim, D., Dionysiou, D.D., Sorial, G.A., Timberlake, D., 2004. Sources and remediation for mercury contamination in aquatic systems–a literature review. Environ Pollut 131 (2), 323–336. https://doi.org/10.1016/j.envpol.2004.01.010.
Wang, F., Xiang, L., Sze-Yin, Leung K., Elsner, M., Zhang, Y., Guo, Y., Pan, B., Sun, H., An, T., Ying, G., Brooks, B.W., Hou, D., Helbling, D.E., Sun, J., Qiu, H., Vogel, T.M., Zhang, W., Gao, Y., Simpson, M.J., Luo, Y., Chang, S.X., Su, G., Wong, B.M., Fu, T.M., Zhu, D., Jobst, K.J., Ge, C., Coulon, F., Harindintwali, J.D., Zeng, X., Wang, H., Fu, Y., Wei, Z., Lohmann, R., Chen, C., Song, Y., Sanchez-Cid, C., Wang, Y., El-Naggar, A., Yao, Y., Huang, Y., Cheuk-Fung Law, J., Gu, C., Shen, H., Gao, Y., Qin, C., Li, H., Zhang, T., Corcoll, N., Liu, M., Alessi, D.S., Li, H., Brandt, K.K., Pico, Y., Gu, C., Guo, J., Su, J., Corvini, P., Ye, M., Rocha-Santos, T., He, H., Yang, Y., Tong, M., Zhang, W., Suanon, F., Brahushi, F., Wang, Z., Hashsham, S.A., Virta, M., Yuan, Q., Jiang, G., Tremblay, L.A., Bu, Q., Wu, J., Peijnenburg, W., Topp, E., Cao, X., Jiang, X., Zheng, M., Zhang, T., Luo, Y., Zhu, L., Li, X., Barceló, D., Chen, J., Xing, B., Amelung, W., Cai, Z., Naidu, R., Shen, Q., Pawliszyn, J., Zhu, Y.G., Schaeffer, A., Rillig, M.C., Wu, F., Yu, G., Tiedje, J.M., 2024. Emerging contaminants: a one Health perspective. Innovation 5 (4), 100612. https://doi.org/10.1016/j.xinn.2024.100612.
Wesolowska, M., Yeates, A.J., McSorley, E.M., Watson, G.E., van Wijngaarden, E., Bodin, N., Govinden, R., Jean-Baptiste, J., Desnousse, S., Shamlaye, C.F., Myers, G.J., Strain, J.J., Mulhern, M.S., 2024. Dietary selenium and mercury intakes from fish consumption during pregnancy: Seychelles Child Development Study Nutrition Cohort 2. Neurotoxicology 101, 1–5. https://doi.org/10.1016/j.neuro.2023.12.012.
Whitmee, S., Haines, A., Beyrer, C., Boltz, F., Capon, A.G., de Souza Dias, B.F., Ezeh, A., Frumkin, H., Gong, P., Head, P., Horton, R., Mace, G.M., Marten, R., Myers, S.S., Nishtar, S., Osofsky, S.A., Pattanayak, S.K., Pongsiri, M.J., Romanelli, C., Soucat, A., Vega, J., Yach, D., 2015. Safeguarding human health in the Anthropocene epoch: report of the Rockefeller Foundation-Lancet Commission on planetary health. Lancet 386 (10007), 1973–2028. https://doi.org/10.1016/S0140-6736(15)60901-1.
WHO, World Health Organization, 2021. Minamata Convention on Mercury: annotated bibliography of WHO information. Available at: https://www.who.int/publications/i/item/9789240022638. (Accessed 16 September 2025).
WHO, World Health Organization, 2023. One health high-level expert Panel annual report 2022. Available at: https://www.who.int/publications/m/item/one-health-high-level-expert-panel-annual-report-2022. (Accessed 10 September 2025).
WHO, 2025. One Health. World Health Organization. https://www.who.int/health-topics/one-health#tab=tab_1. (Accessed 10 September 2025).
Wilkening, J.L., Kurthen, A.L., Guilbeau, K., Libera, D.A., Nelson, S.J., Ming, J., 2025. Climate-Driven alterations in the Mercury cycle: implications for wildlife managers through a one health lens. Land 14 (4), 856. https://doi.org/10.3390/land14040856.
Wu, Y.S., Osman, A.I., Hosny, M., Elgarahy, A.M., Eltaweil, A.S., Rooney, D.W., Chen, Z., Rahim, N.S., Sekar, M., Gopinath, S.C.B., Mat Rani, N.N.I., Batumalaie, K., Yap, P.S., 2024. The toxicity of Mercury and its chemical compounds: molecular mechanisms and environmental and human health implications: a comprehensive review. ACS Omega 9 (5), 5100–5126. https://doi.org/10.1021/acsomega.3c07047.
Wyatt, L., Permar, S.R., Ortiz, E., Berky, A., Woods, C.W., Amouou, G.F., Itell, H., Hsu-Kim, H., Pan, W., 2019. Mercury exposure and poor nutritional status reduce Response to Six Expanded Program on immunization vaccines in children: an observational cohort Study of communities affected by gold mining in the Peruvian Amazon. Int J Environ Res Public Health 16 (4), 638. https://doi.org/10.3390/ijerph16040638.
Yin, Z., Milatovic, D., Aschner, J.L., Syversen, T., Rocha, J.B., Souza, D.O., Sidoryk, M., Albrecht, J., Aschner, M., 2007. Methylmercury induces oxidative injury, alterations in permeability and glutamine transport in cultured astrocytes. Brain Res. 1131 (1), 1–10. https://doi.org/10.1016/j.brainres.2006.10.070.
Yu, X., Khan, S., Khan, A., Tang, Y., Nunes, L.M., Yan, J., Ye, X., Li, G., 2020. Methyl mercury concentrations in seafood collected from Zhoushan Islands, Zhejiang, China, and their potential health risk for the fishing community: capsule: Methyl mercury in seafood causes potential health risk. Environ. Int. 137, 105420. https://doi.org/10.1016/j.envint.2019.105420.

Yuan, C.S., Chiang, K.C., Yen, P.H., Ceng, J.H., Lee, C.E., Du, I.C., Soong, K.Y., Jeng, M.S., 2023. Long-range transport of atmospheric speciated mercury from the eastern waters of Taiwan Island to northern South China Sea. Environ Pollut 318, 120899. https://doi.org/10.1016/j.envpol.2022.120899.

Zhang, L., Zhou, Y., Song, Z., Liang, H., Zhong, S., Yu, Y., Liu, T., Sha, H., He, L., Gan, J., 2022. Mercury induced tissue damage, redox metabolism, ion transport, apoptosis, and intestinal microbiota change in red swamp crayfish (*Procambarus clarkii*): application of multi-omics analysis in risk assessment of Hg. Antioxidants 11 (10), 1944. https://doi.org/10.3390/antiox11101944.

Zhao, L., Meng, B., Feng, X., 2020. Mercury methylation in rice paddy and accumulation in rice plant: a review. Ecotoxicol. Environ. Saf. 195, 110462. https://doi.org/10.1016/j.ecoenv.2020.110462.

Zhou, Y., Wang, X., Wang, C., Ji, Z., Niu, X., Dong, H., 2024. Fate of 'forever chemicals' in the global cryosphere. Earth Sci. Rev. 259, 104973. https://doi.org/10.1016/j.earscirev.2024.104973.

Zhou, H., Li, Y., Zhong, Q., Wu, X., Liang, S., 2025. Global mercury dataset with predicted methylmercury concentrations in seafoods during 1995-2022. Sci. Data 12 (1), 241. https://doi.org/10.1038/s41597-025-04570-3.