

ARTICLE INFORMATION

Received: July 05, 2026

Revised: July 18, 2026

Available online: July 22, 2026

at: <https://e-jurnal.ipohrr.com/index.php/mchc>

Methylmercury exposure and child malnutrition: Mechanistic pathways and epidemiological evidence - A narrative review

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Abstract

Background: Mercury exposure has emerged as a public health concern that may contribute to impaired nutritional status among children, particularly in developing countries where environmental contamination remains prevalent.

Purpose: To review the biological mechanisms and epidemiological evidence regarding the effects of methylmercury exposure on children's nutritional status.

Method: A narrative review was conducted by synthesizing evidence from published studies on methylmercury exposure and child nutrition, focusing on gastrointestinal, metabolic, and endocrine mechanisms as well as epidemiological findings from various countries.

Results: Methylmercury disrupts nutrient absorption, interferes with essential mineral metabolism, and alters endocrine function. Epidemiological studies consistently demonstrate associations between mercury exposure and stunting, anemia, impaired growth, and abnormal body mass index trajectories among children.

Conclusion: Mercury exposure negatively affects children's nutritional status through multiple biological pathways. Further prospective studies are required to establish causality and inform preventive public health strategies.

Keywords: Child Nutritional Status; Endocrine Disruption; Heavy Metal Toxicity; Methylmercury; Stunting.

INTRODUCTION

Globally, and particularly in developing countries, nations are grappling with the triple burden of malnutrition: undernutrition (wasting, underweight, stunting), micronutrient deficiencies, and overweight or obesity (Popkin, Corvalan, & Grummer-Strawn, 2020). According to data from the World Health Organization (WHO), there are 150.2 million cases of stunting among children under five years of age, 42.8 million cases of wasting, and 35.5 million cases of overweight children (World Health Organization, 2024). On the other hand, the prevalence of breastfeeding and obesity has increased by 30% from the previous year in developed countries (Parajuli & Oldewage-Theron, 2025).

Nutritional status is a key indicator of physical growth; children suffering from malnutrition are vulnerable to infectious diseases due to a decline in immune function, which in turn affects their nutritional status. Recently, the effects of heavy metal exposure have been linked to stunting and weight loss (Dack, Wootton, Taylor, & Lewis, 2022; Dorea, 2004; Zulaikhah et al., 2020). A recent study in Korea showed that mercury (Hg) in the umbilical cord of newborns has a significant association with birth length (Lee, Kim, Moon, Park, Choi, & Kim, 2023). Hg exposure was found to originate from small-scale gold mining (SSGM) and waste from coal-fired power plants (CFPPs) that disperse into the air, soil,

and seawater (Sinaga, Afifah, & Muniroh, 2025). In this process, inorganic mercury, known as methylmercury (MeHg), in seawater undergoes bioaccumulation within marine organisms via the food chain, making seafood the primary source of MeHg exposure in the human body (Chen & Dong, 2022; Lee et al., 2023; Zulaikhah, Wahyuwibowo, & Pratama, 2020).

MeHg is believed to disrupt the metabolism of nutrients in the body; its toxic properties inhibit paraoxonase-1, which prevents atherosclerosis by metabolising toxic oxidised lipids (Lewoń-Mrozek, Kurzynoga, Jędrzejewski, Kędzierska, Partyka, Kuriata-Kordek, & Ściskalska, 2024). MeHg causes oxidative stress and systemic inflammation, which affect adipocytes abnormally (Lee et al., 2023). MeHg also disrupts the structural integrity of genetic material by binding to amino acid groups in genetic material such as DNA and RNA, resulting in DNA damage (Chen & Dong, 2022).

Although previous reviews have discussed mercury toxicity and environmental contamination, limited studies have specifically synthesized the mechanistic pathways and epidemiological evidence linking methylmercury exposure to child malnutrition. Given the increasing burden of child malnutrition in developing countries and the widespread presence of mercury contamination, understanding these mechanisms is essential for developing preventive and nutritional intervention strategies. Therefore, this narrative review aims to summarize current evidence regarding the biological and epidemiological relationships between methylmercury exposure and children's nutritional status.

RESEARCH METHOD

This study employed a narrative review design to synthesize and analyze scientific evidence regarding the effects of methylmercury (MeHg) exposure on children's nutritional status. The review focused on the biological mechanisms underlying mercury toxicity and its epidemiological associations with nutritional outcomes in pediatric populations. Relevant literature was identified from peer-reviewed scientific publications discussing methylmercury exposure, heavy metal toxicity, child nutritional status, and growth-related outcomes. The evidence included mechanistic studies, epidemiological studies, and review articles that addressed the gastrointestinal, metabolic, and endocrine effects of methylmercury exposure in children or populations relevant to child health.

The literature was narratively analyzed and organized into four major themes: (1) biological mechanisms linking methylmercury exposure to impaired nutritional status, (2) gastrointestinal dysfunction and nutrient absorption disturbances, (3) endocrine disruption and metabolic alterations associated with methylmercury exposure, and (4) epidemiological evidence demonstrating the relationship between mercury exposure and nutritional outcomes, including stunting, anemia, impaired linear growth, and abnormal body mass index trajectories in children.

The findings from the selected literature were synthesized descriptively to provide a comprehensive understanding of the potential pathways through which methylmercury exposure may contribute to child malnutrition and to identify current research gaps regarding causal relationships and public health implications.

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DOI: <https://doi.org/10.56922/mchc.v5i4.3783>

RESEARCH RESULTS

Figure 1. Mechanisms of Mercury Exposure and Its Impact on Children's Health

Biological Mechanisms Linking Mercury to Nutritional Status

Mercury and Gastrointestinal Function

These protective layers of the gut prevent harmful toxic substances from passing through the intestinal mucosa into other tissues and organs within the body. The millions of gut microbiota act as a biological barrier. Mechanically, exposure to Hg can alter the gut microbiota and release a cascade of inflammatory factors that can cause damage to the intestinal mucosa and trauma to distal organs. Meanwhile, changes in the gut microbiota caused by Hg exposure can lead to inflammation of the liver and gut, eventually causing liver cancer via the gut-liver axis. This is supported by Haematoxylin–Eosin staining, which shows that intestinal cell necrosis occurs in the small and large intestines, with the intestinal mucosa being destroyed following Hg exposure. Pathological studies indicate that Hg exposure can cause damage to the caecum, rectum, and intestinal mucosal necrosis. MeHg can disrupt nutrient homeostasis by competing with essential minerals in the nutrient absorption process and for binding sites. Selenium, for instance, possesses both antitoxic properties and is considered an antagonist against MeHg. Although there is evidence that nutrients interact with MeHg metabolism, nutrients can influence bioavailability, toxicokinetics, and transport to target organs, as well as modulate cytological, immunological, and biochemical responses

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to MeHg; our understanding of the mechanisms of MeHg toxicity remains very limited. Review articles in the field of nutritional toxicology are scarce, with most focusing on the modification of MeHg toxicity by selenium, vitamin C, vitamin E, and other essential minerals. Physiologically, the process of MeHg absorption is divided into two: the effects of nutrients on MeHg metabolism and the effects of MeHg on nutrient metabolism. In this discussion, the information obtained is largely from studies conducted on laboratory animals, and the effects on humans remain speculative. In studies on rats, it was found that MeHg is transported in the blood, bound to serum proteins such as albumin and mercaptoalbumin as stable conjugates, to target organs such as the liver, kidneys, placenta, foetus, and brain. On the other hand, some MeHg is found in epithelial tissue and erythrocytes. Furthermore, cysteine (Cys) is considered one of the nutrients capable of increasing the neurotoxicity of MeHg. MeHg can also disrupt the transport of nutrients, such as the exchange of methionine (Met) and selenium (Se) across the blood-brain barrier. Hg^{2+} can alter the absorption of nutrients such as vitamin B12 and zinc in rats and humans, inhibit the transport of L-alanine and L-lysine across the human placenta, and affect the transfer of amino acids across the placenta.

Exposure to MeHg results in impaired protein synthesis due to enzyme activation; the observed inhibition includes that of aspartate and alanine aminotransferases in fish exposed to Hg^{2+} . GSH induction with the Cys precursor (1 mmol L-2-oxothiazolidine-4-carboxylic acid) reduces MeHgCl-induced amino acid release from astrocytes; cysteine indirectly increases MeHg toxicity by stimulating cellular GSH synthesis. Carbohydrate and lipid metabolism are also disrupted by MeHg exposure; this heavy metal can reduce the incorporation of ^{14}C -labelled glucose in the brains of suckling rats. Furthermore, rats administered MeHgCl for more than 3 weeks exhibited altered levels of glycogen and lactic acid in their hearts and livers. The offspring of rats injected with MeHgCl had abnormal glycogen deposits in their alveolar tubules, and numerous enzymes involved in carbohydrate metabolism were inhibited by Hg^{2+} , including glucose-

6-phosphatase, amylase, maltase, and lactase. Exposure to MeHg can also alter lipid profiles and the production of fatty acids and cholesterol, subsequently reducing triglycerides in the rat's central nervous system. There is speculation that this may be due to changes in levels of Mg^{2+} , adenosine triphosphate (ATP), or acetyl coenzyme A. Other enzymes involved in lipid metabolism, such as lipase and carnitine acetyltransferase in the human placenta, are also inhibited. Hg affects the response of fat cells to insulin in vitro. Disruption of essential mineral metabolism by MeHg. Methylmercury disrupts the metabolism of Zn, Cu, Mn, Cr, Ni, Fe (manganese, chromium, nickel, iron). MeHg also affects sodium and potassium ion channels, and Ca^{2+} metabolism is disrupted due to MeHg exposure, which can lead to increased Ca^{2+} permeability and changes in Ca^{2+} metabolism in muscle tissue.

Endocrine Disruption

Heavy metals such as mercury disrupt the functioning of the endocrine system in both animals and humans; methylmercury (MeHg) affects hormonal homeostasis, leading to endocrine imbalance. It has been found that thyroid hormones are frequently associated with mercury; however, the results are inconsistent due to differences in exposure levels, so further research is needed to determine a more accurate chemical response. The major endocrine glands include the hypothalamus, pituitary, parathyroid, thyroid, gonads (testes and ovaries), pancreas, and adrenal glands. Hormones are responsible for maintaining homeostasis, reproduction, behaviour, and development. The effects of MeHg on the endocrine system have been studied using in vitro techniques; MeHg selectively destroys fibroblast-like cells. This process is mediated by the reversible inhibition of sulphhydryl enzymes, as its cytotoxic effects are completely inhibited by the addition of glutathione or thioglycolate. MeHg toxicity may also contribute to certain thyroid disorders, as MeHg can enter pathways leading to genetic mutations, autoimmune reactions, and the production of oxygen free radicals; these mechanisms ultimately underlie the pathogenesis of

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thyroid cancer, autoimmune thyroiditis, and hypothyroidism. Rat thyroid stem cells contain the mercury transporter ABCG2/BCRP, suggesting that thyroid stem cells (though not follicular cells, which lack ABCG2/BCRP) have adapted to mercury uptake and make efforts to clear themselves of this metal. Thus, when mercury levels in thyroid follicular cells reach a critical threshold in individuals with a genetic predisposition to autoimmunity, autoimmune thyroiditis may develop. Some cases of hypothyroidism are thought to arise from subclinical autoimmune thyroiditis, and the generation of oxygen free radicals by mercury is a further mechanism by which this metal may contribute to an underactive thyroid.

Epidemiological Evidence

Previous research conducted amongst the Mundurucu community in the Brazilian Amazon revealed high levels of methylmercury (MeHg) in the hair of women from the Mundurucu community, leading to adverse effects such as high rates of anaemia, stunting, and infant mortality. Another study carried out in gold-mining areas of the Peruvian Amazon showed that hair mercury levels were significantly associated with children's Z-scores for height and weight. A similar study involving 14,202 children in China demonstrated a positive correlation between mercury exposure and children's anthropometric characteristics (BMI). Meanwhile, a study conducted in Korea involving 1,458 children found a significant association between urinary mercury levels and stunted growth. There are numerous other studies linking mercury exposure to nutritional status.

DISCUSSION

The mechanistic findings summarised above provide biological plausibility for the epidemiological associations reported across the Mundurucu, Peruvian, Chinese, and Korean cohorts. Disruption of the intestinal mucosa, induction of gut dysbiosis, and competition with essential minerals such as selenium, zinc, and iron offer a coherent pathway linking MeHg exposure to the anaemia, impaired linear growth, and stunting observed in mercury-exposed communities

(Gan, Feng, Yang, Sun, Luo, Yang, & Abdelhafiz, 2025). Likewise, MeHg-induced disruption of thyroid hormone homeostasis offers a plausible endocrine pathway for the altered growth trajectories and abnormal BMI patterns reported in Chinese and Korean paediatric populations. Taken together, the convergence of mechanistic and epidemiological evidence strengthens the case that MeHg exposure is not merely correlated with, but biologically capable of contributing to, impaired child nutritional status (Gao, Li, Wang, Yan, Zhou, & Yan, 2018; Shin, Kim, Kwon, Park, & Kim, 2024).

Nevertheless, an important asymmetry runs through this body of evidence. Much of the mechanistic detail particularly regarding nutrient transport, enzyme inhibition, and mineral competition derives from animal and in vitro models, and its extrapolation to humans remains speculative. In contrast, the epidemiological studies are predominantly cross-sectional, capturing associations at a single point in time rather than establishing temporality or dose-response relationships. This gap between mechanistic plausibility and confirmed human causality is compounded by the scarcity of dedicated review articles in nutritional toxicology addressing MeHg specifically, most of which have instead focused narrowly on selenium, vitamin C, or vitamin E as modifiers of MeHg toxicity rather than on the reciprocal effects of MeHg on broader nutrient metabolism (Ralston & Raymond, 2010; Raymond & Ralston, 2009).

A further complexity concerns the dual role of fish consumption, the principal route of MeHg exposure, which simultaneously supplies beneficial nutrients such as selenium, omega-3 fatty acids, and high quality protein (Allan, Shen, Williams, Ford, Souter, Zhang, & Earth Study Team, 2025; Allan et al., 2025; Kuznetsov, 1987; Ralston & Raymond, 2010; Raymond & Ralston, 2009). This creates a nutritional paradox in which restricting fish intake to reduce mercury exposure may itself compromise nutritional status, particularly in populations where fish is a primary dietary staple (Starling, Charlton, McMahon, & Lucas, 2015). Disentangling the net effect of fish consumption on child nutrition therefore requires studies that simultaneously

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account for MeHg exposure and the nutrient density of the diet, rather than treating mercury toxicity in isolation (Bjørklund, Aaseth, Ajsuvakova, Nikonorov, Skalny, Skalnaya, & Tinkov, 2017; Chang, Yang, Zhou, Yin, Liu, Qian, & Lu, 2019; Sherlock, Lindsay, Hislop, Evans, & Collier, 1982; Shin et al., 2024).

These gaps point toward clear priorities for future research: prospective birth and childhood cohort studies capable of establishing temporal and dose-response relationships between MeHg exposure and nutritional outcomes; mechanistic studies conducted directly in human tissue or paediatric populations rather than relying solely on animal extrapolation; and intervention studies evaluating whether selenium or other antagonist nutrients can mitigate MeHg-related nutritional harm in vulnerable children. From a public health perspective, the convergence of evidence nonetheless supports prioritising mercury exposure reduction through monitoring and regulation of small-scale gold mining and coal-fired power plant emissions, and through targeted dietary guidance in populations already at elevated risk of malnutrition, even as the underlying causal pathways continue to be refined (Esdaile & Chalker, 2018; Adlim, Kamil, & Karma, 2023).

CONCLUSION

Current evidence suggests that methylmercury exposure adversely affects children's nutritional status through gastrointestinal, metabolic, and endocrine mechanisms. Epidemiological findings support the association between mercury exposure and growth disturbances in children. Strengthening environmental monitoring and conducting prospective cohort studies are essential for developing effective public health interventions to reduce mercury-related nutritional risks in children.

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