

## Effects Heavy Metal and Organic Contaminants During Pregnancy and Lactation on Child Health

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### Introduction

This study aimed to review the scientific evidence on the influence of heavy metals and organic contaminants in foods consumed by pregnant and lactating women on child health. Heavy metal toxicity in humans is the accumulation of inorganic and organic forms of heavy metals, in toxic amounts, in cell, tissues, organs, and bodily fluids [1-3]. Symptoms and physiological outcomes are directly associated the different heavy metals, the location of the accumulation, the amount of accumulation and chronicity of the accumulation [4-5]. The poisoning caused by each heavy metal will vary depending on its inorganic or organic variant form. The most common essential heavy metals in the human body includes but are not limited to zinc, copper, chromium, iron and manganese; most of these are vital to body function in very small amounts but may become toxic if they accumulate and concentrations sufficiently [6]. The heavy metals most commonly associated with toxicity and poisoning in humans include arsenic, cadmium, lead, and mercury. Most heavy metal toxicity is associated with and the result of industrial exposure, air or water contamination, food adulteration, medicines, improperly coated food containers, tooth amalgam or the ingestion of chips or flakes of lead-based paints [7-9].

**Keywords:** Arsenic, Bioaccumulation, Breastfeeding, Breastmilk, Cadmium, Fish, Lead, Methyl Mercury, Mercury Toxicity, Minamata Mercury Convention, Rice, Vulnerable populations.

Since breastmilk is universally considered the best natural source of essential nutrients for all infants, any exposure of breastmilk containing toxic heavy metals could potential be harmful to the developing infant [10-11]. The World Health Organization (WHO) considers breastfeeding as the preferred source of nutrition for all infants worldwide, yet in much of the developing world breastmilk is contaminated with hazardous environmental pollutants such as heavy metals, pesticides, fungal toxins, prescribed and illegal drugs and endocrine and nervous system disrupting chemicals [12-13]. Many of these toxic substances are found in women of childbearing age and can be transferred via maternal and/or placental cord blood to the developing fetus or to the newborn or breastfeeding infant through contaminated breastmilk [14-16].

Toxic levels of these contaminants in infants are associated with low birth weight, neurobehavioral development and dysfunction, fetal growth restrictions and retardations, cognitive dysfunctions, and low APGAR and IQ scores [17-19]. These factors may be compounded by

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many sociodemographic factors such as mother's age and educational levels, smoking, alcohol, drug use and abuse, and dietary habits such as eating fish and rice containing heavy metals especially mercury and the associated methylated organic compounds it can form [19-21].

Once in the environment, inorganic mercury can be methylated to methylmercury by bacteria [22-24]. Methylmercury can bio-accumulate in many upper trophic level animals, especially humans such that they contain higher concentrations of methylmercury [25-28]. According to Bernhoft (2012) toxicity in human varies depending on the form, dose and rate of exposure of mercury. The target organ for inhaled mercury vapor is primarily the brain, while mercurous and mercuric salts chiefly damage the gut lining and kidney [29]. While methylmercury is widely distributed throughout human organs and tissues, methylmercury is not the only organic form that mercury can be converted to in the human body [30-32].

Mercury can readily attach to carbon-based molecules in the methyl, ethyl, phenyl or related organic molecules [33-34]. Methyl mercury reacts with sulfhydryl groups throughout the body, interfering with cellular or subcellular structure [35-36]. Dimethylmercury is a neurodegenerative toxic agent that once in the human body can be metabolized after several days to methylmercury [29,37-39]. Ethyl mercury another toxic organic form of mercury, has its origin from thimerosal-containing vaccines [40-41]. All of these toxic organic forms of mercury can bio-accumulate through the ingestion of foods such as fish, rice and grains, each containing various forms of mercury [29,42-44]. Methylmercury is lipid-friendly and can cross the cell membranes easily. The placenta cross rate of methylmercury is 10 times higher than for other mercury compounds, thereby affecting the developing fetus. Organic forms of mercury is excreted through breastmilk readily; the half-life period of methylmercury in breastfeeding women is shorter than in non-lactating women, putting breastfeeding infants at greater risk for mercury toxicity [45-47]. In the human body, both infant and adult, methylmercury is associated with impairment of the nervous and immune system, disruption of DNA repair, destruction of mitochondrial membranes, chronic fatigue syndrome, neurodegenerative diseases resulting in loss of IQ, cardiovascular and respiratory diseases, and diseases of the kidney, skin and eyes [29,34,43]. Exposure to high levels of methylmercury is known as Minamata disease [48,49]. Developing fetuses, breastfeeding infants and young children are at greatest risk to acute methylmercury exposure, where even low levels can have devastating permanent health consequences [50-51]. The effects of long-term low-dose exposure to methylmercury in this age group remains unclear [7,52].

In this manuscript we will discuss and review organic and inorganic dietary contaminants, especially heavy metals, in women of childbearing age in developing countries and the toxic health effects of these contaminants have on developing embryos, the quality of breastmilk and in the development of breastfeeding infants. We will also discuss the role of fish and rice in the bio-accumulation and bio-magnification of methylmercury in the above vulnerable populations.

Our review of the literature aimed at looking at the organic and inorganic dietary contaminants in women of childbearing age in developing countries and at the toxic health effects of these contaminants have on nascent embryos and breastfeeding infants. We specifically focused on heavy metals especially methylmercury and its passage into breast milk, negatively affecting the developing child. A literature search was done using MEDLINE database of references and abstracts on life sciences and biomedical topics, and Google Scholar with the keywords: Arsenic, bioaccumulation, breastmilk, cadmium, fish, lead, methyl mercury, mercury toxicity, Minamata Mercury Convention, rice, and vulnerable populations, used in the search. Publications were selected that observed the geographic locations with high fish and rice consumption containing organic and inorganic contaminants with focus on heavy metals especially methylmercury and the transport of methylmercury in breast milk to the developing child. Articles used were from 2006 onward, and a 1994 paper used due to its explanation of the negative effects of methylmercury on early development.

### **Minamata Convention on Mercury**

In January 2013, the Minamata Convention on Mercury was agreed upon by 128 nations in Geneva, Switzerland to address the ongoing issue of methyl-mercury pollution [53]. It was later ratified in October of the same year in Kumamoto, Japan for implementation [54]. Then in May 2017 the documents achieved 50 ratifications allowing the legally binding mercury reduction measures to go into effect in August of that same year [54-56]. Since that time, the number of nations to ratify the legally-binding documents continue to grow, with 113 participants currently [56]. The convention was named after Minamata, Japan; considered to be the location of one of the worst mercury contamination events to date [57]. Under further investigation, this poisoning was believed to be caused by wastewater from a local chemical factory contaminating one of the city's main food sources, fish; bringing to light just one of the many sources increasing pollution on a daily basis [58]. The Convention addressed many aspects of this issue including mercury availability, and its use in products such as thermometers [59-60]. It also stressed reducing emissions from artisanal and small-scale gold mining, considered one of the largest culprits for the increased mercury emissions over the years [61]. It focused on reducing emissions from power plants and metal producing facilities as well as encouraged governments to have an action plan in place to manage the health effects of already occurring mercury exposure [59-60].

The first benefit of the Minamata Convention was bringing greater attention to the severity of the mercury issue and the necessity to remedy that issue [56,60,62]. Participating members of the Convention were tested for hair mercury levels resulting in a more motivated discussion. Mercury was found on every single person and, alarmingly, one third of participants were over the safe reference dose, set by the US National Research Council [62]. With a motivated populace the second benefit was realized, putting into place a foundational start to reducing the participating countries contributions to the mercury issue and the adverse health

outcomes it causes [54]. So far, the foundation set by the Minamata Convention has not been completely appreciated. However, early studies have shown a decrease in the atmospheric mercury and the projected global emission rates [63,64]. A third benefit of the convention was that it brought countries with the means to make change into the fight against mercury pollution, including the first country to join, the United States [59]. This issue cannot be solved globally if participating countries do not help each other in the process [65]. The convention outlined what assistance developing countries should receive, hopefully ensuring that every participant is able to meet the standards [60]. Having key countries like the United States is considered crucial in ensuring the goal of the convention is achieved [59].

While the Minamata convention's goal was unquestionably positive, there were still some inadequacies of the final documents. Leading some researchers and reporters to be left with criticisms [66-68]. The Convention did not completely eradicate mercury use, particularly in small scale gold mining and production processes. A compromise was made that prohibited new mercury mining after the treaty went into effect but allowed already existing mining to continue for up to 15 years [68]. In many of the areas where artisanal and small-scale gold mining takes place the use of mercury is essential to the people's livelihood. Banning the metal's use completely with no efficient alternative would cause a strain on those families that have little option but to mine [69-71]. Further complicating the issue, the communities that would be affected by such drastic changes are not required to be a part of the decision-making process. All of these factors could potentially cause difficulty in following through with the regulations set by the Convention [71]. Next, the Convention could not take effect until there were fifty ratifying nations, this did not occur until a little less than three years after the initial agreement, delaying potential mercury reduction progress until 2017 [55]. Another criticized aspect of the Convention was the exemption of Thimerosal vaccines from regulation. This led to some significant controversy about the disparity of where these vaccines are distributed and used, which is mainly in developing nations [72]. Finally, one of the biggest potential downfalls of the Convention is figuring out how to exactly measure its effectiveness. It is thought that some of the guidelines presented for monitoring progress of the Convention are not exactly feasible [73]. There are differing ideas on the best way to measure the global mercury levels. One of the proposed ideas is to measure through the living organisms where the heavy metal becomes trapped [73-74]. Even that has limitations however, receiving differing results depending on where and how mercury levels are tested. This makes it difficult to give an actual evaluation of the effectiveness of the Convention guidelines [74].

Since the Convention, the research on the mercury issue has greatly increased. More locations of mercury pollution are being teased out, new tools and ways to reduce emissions efficiently continue to be discovered, and the cycling process is better understood [75]. It was estimated from 2010 to 2015, the global emission rate per year slowed its ascent compared to previous projections increasing

by only 1.8%. North America and Europe have reduced their mercury use and emissions [64]. However, this has been outweighed by the continued growth in emissions from developing countries, and the largest contributor to atmospheric mercury, China [64,76]. While global emission rates seem to be headed in the right direction the mercury levels found in fish and other food sources have continued to be concerning, causing some researchers to call for a change in diet to reduce exposure [77]. The continued elevated levels of mercury in this reservoir is projected to take longer to improve, highlighting the continued difficulty of effective evaluation of the Convention guidelines [63]. The Convention regulations have also started to cause a strain on the families that require mercury for their livelihood. One example is in Ghana where the ban on mercury has angered the mining population causing the country to find alternate revenue sources for those affected [65]. Even though there has been some criticism, the Minamata Convention built a foundation for reducing current and future exposure to mercury pollution. The Convention addressed the need to revise and improve as more research and feedback is given [60]. This will allow the guidelines to adjust to future issues that arise and hopefully reach the end goal of reduced mercury exposure and improved public health.

## Non-Metal Organic Contaminants

The general consensus on potential risks introduced by other, non-metal environmental pollutants does not override the recommended practice for breastfeeding infants. However, according to the Centers for Disease Control, breastmilk is not routinely assessed for environmental pollutants. Gaps in knowledge leave a void with little to no known data to confirm or refute risk to infants from other potentially hazardous substances in human milk [78]. The American Association of Pediatricians' Committee on Drugs, provided a 2001 report on the transfer of drugs and other chemicals into human milk, including food and environmental contaminants. They stated the need to protect infants from potential adverse effects and protect mothers from unnecessary restrictions of needed medications [79-81].

The potential harmful effects of DDT to humans, wildlife and the environment raised great concern and regulatory action in the United States in the early 1970s, alerting public officials to the risks of exposures to organic substances. To date, there are no signs or symptoms in infants or effects on lactation due to DDT, and other commonly used organic compounds such as benzene, hexachlorides (fungicide), dieldrin and aldrin (pesticides), and heptachlorepoxyde (insecticide) (AAP 2013). More recently, studies of household cleaners containing (quaternary ammonium compounds) are under study to determine exposure effects on growth and development [82]. More surveillance programs and research are needed to continually inform policy makers and the public of the safety and efficacy of an overwhelming number of substances mothers maybe exposed to, or consume, that could pose risks in infants.

Human exposures to environmental contaminants are presumed to be involuntary. Today, it is increasingly more common that concerns arise from overexposure to

prescription, illicit and over-the-counter health products. For example, a common medication used to treat severe acne, Accutane® (isotretinoin, a form of retinoic acid, a form of vitamin A), is a documented teratogen and not prescribed to pregnant women or those planning to become pregnant. Due to the toxicity of this product it can only be marketed under a restricted distribution program, iPLEDGE®, designed specifically to enroll registered prescribers, patients, pharmacies and wholesalers. Nursing mothers should not take Accutane®. It is not known if this drug is excreted in human milk [83-84]. The American Academy of Pediatrics clinical report lists drugs of abuse, once referred to as illicit street drugs, for which adverse effects are reported on the breastfeeding infant. Alcohol, amphetamines, cocaine, heroin, LSD, marijuana are among those listed. Included in the report are effects or reasons for concern and the contraindications during breastfeeding or feeding breastmilk to infant [78].

Informing breastfeeding mothers and those providing breastmilk to infants is an educational process for all involved, as seen with the unique implementation of the iPLEDGE program [83]. For most prescription drugs providers are expected to explicitly instruct patients regarding the safe use of products potentially harmful to pregnant and lactating women. Additionally, product labels clearly stating warnings as needed for pregnant and lactating mothers [84]. In some circumstances a temporary recommendation to not provide an infant with breastmilk may be warranted dependent on a mother's health condition, i.e., in the case of untreated brucellosis [85].

Other voluntary and intentional exposures to products assumed 'healthy' may actually be harmful to the mother-infant dyad, with extreme cases leading to morbidity and mortality. Misuse of essential nutrients, particularly vitamins and elements (minerals) has prompted establishing recommendations specific for various subpopulations based on age, gender and pregnancy and lactation. Intakes of single nutrients often as dietary supplements, particularly but not exclusively, the elements and the fat-soluble vitamins (Vit A, D, E, and K), can exceed safe and adequate intakes. The national recommendations are established, when scientific data are available, providing guidance on the safe and adequate daily intake levels for all age and gender groups, as well as pregnant and lactating women. If sufficient evidence was available, upper limits are also established to prevent toxicities from more acute, short-term intakes of these essential nutrients [86].

Throughout history, cultures, worldwide, have passed down the ethnic tradition of women taking herbal substances to boost milk production (*galactagogues*) during lactation. The use of herbal galactagogues varies widely and continues to gain popularity among Western cultures is the use of many herbals, i.e., fenugreek (*Trigonella foenum-gracum*), believed to aid lactation [87]. Several have provided reviews appraising the use and evidence for using galactagogues, including fenugreek and other herbs, thought to increase milk production. Lactation support organizations advocate the numerous benefits of herbal galactagogues, including psychologically empowering women struggling with real

and perceived milk production insufficiency [62,63]. The research community emphasizes the lack of data to confirm safety and efficacy of galactagogues, pharmaceutical and herbal, for this purpose citing results from studies with inadequate methodologies [88-89]. Industry wide, herbal product manufacturers have yet to overcome the problem of standards of identity and consistent quantification of active ingredients. The challenges associated with the "pharmacovigilance of herbal products", particularly in breastfeeding women signify the importance of structured studies to determine herbal compound transfer in human placentas and milk [87].

Support for enhancing lactation has also been advancing with the prescription of pharmacological galactagogues. Clinical trials have indicated successful outcomes for domperidone and metoclopramide, prescribed specifically for the purpose of stimulating milk production and research is ongoing to confirm safety and efficacy [88-89].

Universally, breastfeeding remains the recommended practice for feeding infants and is endorsed by leading organizations (e.g., World Health Organization, The Center for Disease Control, The American Academy of Pediatrics, and the Academy of Nutrition and Dietetics). Risk assessment of involuntary and voluntary exposures, particularly during the vulnerable stages of development in unborn and newborn children, is needed to continually inform professionals and the public of risks on an ongoing basis [78,80,90-92].

## Heavy Metal Contaminants

Arsenic, lead, and cadmium, in addition to mercury, are found in various amounts in breastmilk of women from differing countries such as Slovenia, Norway, or Lebanon [93-96]. These metals influence the cellular and humoral response of the immune system of the infant by affecting which cytokines are produced; thus, negatively influencing the ability of the immune system [93]. An increase in the production of cytokines created by lymphocyte Th1 and an inhibition of the cytokines created by Th2 in the presence of heavy metals may occur; however, the opposite may transpire depending on which particular heavy metals are present [93]. Recall lymphocyte Th1 produces interleukin 2 (IL-2), interferon gamma (INF- $\gamma$ ), and tumor necrosis factor-beta (TNF- $\beta$ ). Both Th1 and Th2 produce tumor necrosis factor-alpha (TNF- $\alpha$ ), granulocyte-macrophage colony-stimulating factor (GM-CSF), IL-3, IL-6, and IL-10 [93]. Cadmium and lead inhibit the production of Th1 cytokines [93]. When less of cytokines (IL-2, INF- $\gamma$ , TNF- $\beta$ ) are available, the immune system has a limited ability to sequester and prevent the spread of an infection, indirectly increasing opportunity for a parasitic organism to flourish [93].

Classified as a non-threshold carcinogen or group 1 carcinogen according to the International Agency for Research on Cancer, any arsenic exposure constitutes a risk and no tolerable intake level can be established [97-98]. Arsenic is colorless, odorless, tasteless found in rice, carrots, onions, potatoes, apples, poultry, seafood, animal feed, contaminated water and soil, or other grains like wheat or barley [93,97-99]. The inorganic forms called arsenite (III) and arsenate (V) are

both water soluble which leads to soil contamination and the accumulation of arsenic in various foods [93,98].

Rice is the most common food source of arsenic [97]. The type of rice and the soil where the rice was cultivated determine the amount of arsenic present; however brown rice does overall contain more arsenic than white rice [97-98]. Concentration of inorganic arsenic can reach 90% of the total arsenic content [97,99]. Maximum tolerable level of total arsenic for drinking water is defined as 10µg/L according to the WHO; yet, no limit for total or inorganic arsenic in any food exists for the WHO, European Union (EU), or the United States [97]. China regulates the maximum level of inorganic arsenic in rice permitted is 0.15mg/kg [97].

Once ingested, inorganic arsenic is absorbed and then methylated by S-adenosylmethionine (SAM) with the assistance of glutathione (GSH) to yield dimethylarsinous acid (DMA) and monomethylarsonous acid (MMA) which are less toxic [97]. The body usually excretes these acids in the urine [93]. Negative effects associated with exposure to arsenic during pregnancy include weak cognitive functions, lower birth weight, asthma, tachycardia, and extreme toxicity can cause fetal death [93,96-97].

Unlike arsenic, lead enters the body through the skin, digestive tract, or respiratory tract [93]. Ninety percent of lead is accumulated in the bones; but it can be stored in the liver, kidneys, hair, nails or nervous system [93]. Women with blood levels of lead below 40µg/dL can breastfeed without clinical harm to their children since a tiny fraction of lead in blood is bioavailable and human milk is low in protein, the molecular carrier of lead [100]. Thus, lead is unexpected to heavily transfer into human milk; however, this reasoning does not account for the lead released from bones during pregnancy as a result of bone turnover [93,100].

Potatoes, contaminated lead-based cooking utensils, contaminated water, and the liver and kidney of game organisms are sources of lead to consider when preparing food [93,99-101]. It is excreted through urine, feces, sweat, or human milk. Lead has no biological role in the body [102]. Because of bone resorption during pregnancy, lead can be released into the body and into human milk [93].

Depending on the lead blood level in a pediatric patient, the clinical implications vary [103]. Young children absorb lead four to five times more effectively than adults which greatly increases the risk of toxicity for children [103]. Manifestations of toxicity include developmental delays when the blood lead level is 10µg/dL, increased nerve conduction velocity seen at 20µg/dL, and decreased hemoglobin synthesis seen at 40µg/dL [103]. Exposure to lead can also affect intelligence quotient scores and the nervous system [96,103].

Cadmium has no physiological function in the human body and is considered a potent neurotoxin [96]. Cadmium enters the body mainly through inhalation but it can enter by absorption in the digestive tract [96]. It affects the liver and kidneys and is excreted through urine and feces [96]. Food sources include carrots, onions, potatoes, fish, shellfish, and grains [95-96,99].

Cadmium toxicity results in neuronal changes which manifest as reduced IQ levels, learning dysfunctions, olfactory abnormalities, psychiatric problems, attention deficits, or neurodegenerative diseases [96]. Cadmium toxicity can result in hepatotoxicity; however, a combination of vitamin C, tocopherol, and flavonoid has shown to reduce the hepatotoxicity [95-96]. In infants, it is associated with pre-term birth and the development of asthma [96].

Cadmium toxicity affects the physiologic functions of zinc, iron, magnesium, calcium, plus vitamins B2, B6, and B12 [96]. For example, it decreases zinc absorption by interfering with binding of zinc to metallothionein, a protein involved with transportation of essential metals [96]. Thus, creating a deficiency of zinc [95-96]. Adequate iron, zinc, selenium, and vitamin E, have illustrated a possible protective mechanism in which absorption of cadmium is diminished [96]. Copper can also aid to reverse cadmium induced alteration of metallothionein [96,104]. In fact, supplementation with a plant-based diet demonstrated protection against cadmium toxicity given that these foods contain micronutrients necessary to combat oxidative stress and induce chelation of cadmium [94,104-105].

## **Fish and Rice as Sources of Mercury Contamination**

Methyl mercury exposure through the consumption of fish and rice, has been documented in developing countries, where these products are a staple in the diet of many people. Rice consumption is highest in regions of China and other East Asian countries [106]. Various factors play a role in the accumulation of methylmercury, such as rice management practices and presence of environmental factors contaminating the rice fields [107-108]. Fish consumption, remains highest in regions of Asian countries, such as Japan, followed by China [109]. Environmental factors, such as the conversion of atmospheric mercury to methylmercury by microorganisms and the type of fish, with longer-lived predatory fish having the highest levels of mercury, all play a role in the levels of methylmercury [110]. The high levels of mercury from the fish and rice pose a considerable risk for pregnant mothers, in whom high levels of mercury pose risks to the future development of their child [107].

Methyl mercury, when ingested combines with glutathione, to make a methylmercury- glutathione compound that is transported throughout the body, crossing the blood brain barrier and placenta with ease [111]. The elimination of methylmercury occurs through the bile, with a fraction of the compound being reabsorbed through the process of enterohepatic circulation, which further slows the elimination of methylmercury. Pregnant women, consuming fish and rice high in methylmercury are at high risk, as the average half-life of methylmercury is 50-70 days [112]. With such a long half-life, concentrations of methylmercury remain high throughout pregnancy and into the breastfeeding, negatively affecting development.

Despite, the fact that the majority of methyl mercury is excreted, the small levels of mercury recycled via enterohepatic circulation are still significant enough to

pose a risk to the developing child [111]. Methylmercury, bound to albumin enters into breastmilk and is then directly consumed by the infant [113]. The effects of consumption of milk with high levels of methylmercury are magnified by factors such as frequency of breastfeeding and the slow elimination of methylmercury during the first year of life [114]. In addition, the efficient gastrointestinal absorption, lack of well-developed homeostasis and detoxification mechanisms present in the growing child further exacerbate the problem [115].

Critical development periods during the growth of a child, make the effects of exposure to mercury even more profound [116]. Methyl mercury is a potent neurotoxin, and high exposures of this through breastmilk, can have negative effects on development leading to mental retardation, cerebral palsy, and seizures [117]. Additional effects of early exposure can manifest as changes in the neurobehavioral function throughout life, with impacts in memory and attention, language, visual- spatial and motor skills [118].

Recent studies have shown that in certain regions of India, China, Vietnam and Thailand where gold and other heavy metals are mined and where coal is burned by power plants, soil mercury levels are high and MeHg accumulates in rich grain. In these regions, median rice MeHg concentrations were up to 10 times lower than those typically measured for fish tissue [119-120]. Daily rice-based meals (without fish) containing MeHg are common to much of the world population. Since MeHg has a strong affinity to bioaccumulate, exposure levels are comparable to a fish meal (Tuna, swordfish or any number of higher food chain fish). Detailed studies of rice containing MeHg are few [121-122]. To our knowledge, no detailed study has been done in the United States where rice exported from India, China, Vietnam and Thailand, has been examined for MeHg. Many ethnic populations eat rice one to three times/day [123]. The potential for MeHg contaminated rice exported from Asia to bioaccumulation in women in the United States, especially those women of childbearing age [124].

## Conclusion

Many toxic organic and inorganic substances found throughout the food chain, end up in solid and liquid foods consumed throughout the world. So many foods we consume come from international sources where toxic materials are found in the soil and water, both fresh and marine. Often these toxic materials can bio-amplify across the various food chains and bio-accumulate in the human body. These toxic materials include drugs, pesticides, heavy metals and food additives, may accumulate and be transferred across the placenta to developing fetuses or accumulate in breastmilk and transferred to breastfeeding infants. The impact of these toxic contaminants on vulnerable infants and children worldwide is high, and may go unmeasured, unnoticed and unrecognized.

## Disclosures

There is no financial disclosure to report. None of the authors are receiving or have received any financial benefit

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