

***In The Beginning Lies the Risk:
Fetal Toxic Exposure and The Origins of Adult Disease***

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To Cite This Chapter:

Kirsever, E., Şişman, G., & Şişman, H. (2025). In the beginning lies the risk: Fetal toxic exposure and the origins of adult disease. In S. Demir, F. N. Takı, & Y. Uzun (Eds.), *Current studies for sustainable health* (pp. 151–182). ISRES Publishing.

Introduction

In the late twentieth century, the rapid increase in chronic diseases worldwide demonstrated that these diseases could not be explained solely by genetic predisposition or lifestyle factors during adulthood. During this period, groundbreaking research conducted by British epidemiologist David Barker and his colleagues gave rise to a new paradigm in medical history: the “Fetal Origins of Adult Disease” (FOAD) hypothesis (Barker, 2004). Barker’s cross-sectional analyses of United Kingdom birth registrations with adult mortality rates concluded that low birth weight (LBW) had a strong association with coronary artery disease (CAD), type 2 diabetes (T2DM), osteoporosis, and certain psychiatric illnesses in adult life (Cooper et al., 1997; Lahti et al., 2010; Michels et al., 1996). The findings indicate intrauterine life to be an important biological window that predetermines adult health.

Throughout history, many cultures have recognized that a woman’s pre-pregnancy health plays a decisive role for both mother and child and have placed great importance on maintaining health during pregnancy (Korenbroet et al., 2002). Ancient medical traditions have adopted this awareness centuries earlier and developed a holistic approach to maternal and infant health. For example, Traditional Chinese Medicine (TCM), Mexican Traditional Medicine (MTM), Unani Medicine, and various African medical practices emphasize the use of balanced and holistic health-oriented therapies to prepare the woman’s body for the birth process. According to TCM, a woman’s health before pregnancy depends on the balance of “qi” and “blood,” and the mother undergoes a pre-pregnancy preparation process to address imbalances in these energy and blood circulations (Wu et al., 2020; Zhang et al., 2022). Similarly, MTM utilizes a combination of local herbs, massage techniques, and spiritual practices to protect women’s reproductive

health (Cabada-Aguirre et al., 2023). The concept of “Tadabir-i-Haamla” (*Precautions for Pregnancy*) in Unani Medicine is devoted to pregnancy care and includes detailed information on pregnancy symptoms, nutrition, lifestyle recommendations, and management of common ailments (Khan & Itrat, 2015). This concept aims to maintain the mother’s physical, emotional, and spiritual balance during the pre- and post-natal period. In the 17th century, Şaban Şifaî’s work, named “Tedbîrü’l-Mevlûd” (*Precautions for Newborn*) and Gevrekzâde Hâfız Hasan Efendi’s work, named “Netîcetü’l-Fikriyye fi Tedbîri Velâdeti’l-Bikriyye” (*Outcome Of Healthy Birth Management*), demonstrate the profound and systematic understanding of Ottoman medicine regarding maternal and child health (Bahadır, 2017). These holistic approaches aimed not only at healthy births but also at ensuring the health of future generations.

The FOAD hypothesis, which confirms the approach of ancient medical traditions, is based on the concept of “developmental plasticity,” which is the adaptation capacity of the fetus to intrauterine environmental conditions. Reprogramming of fetal metabolism occurs for its adaptation to nutritional deficiencies, toxins, infection, or stress (Calkins & Devaskar, 2011).

One of the biggest challenges in addressing health problems today is the rapid increase and transformation of disease-causing agents. The cause of most of our ailments is no longer viruses, bacteria, or parasites alone, as current medical literature would lead us to believe, but rather toxic synthetic chemicals such as environmental pollutants, industrial chemicals, genetically modified unnatural foodstuffs, and agricultural chemicals. Alongside its comforts, modern life has brought a chemical burden with unseen but significant effects on human health. Everyday products such as detergents, polishes, plastics, cosmetics, and synthetic textiles represent some of the most hazardous environmental pollutants (Wang et al., 2025).

Toxic exposure has reached such an extent that even the maternal womb—once considered the safest and most protected environment—can no longer fully shield the developing fetus. Recent studies have demonstrated that the placenta is permeable to numerous chemical compounds, allowing toxic substances to pass into the fetal circulation. The detection of hundreds of chemicals in maternal blood, placenta, and umbilical cord blood reveals that the fetus develops under a far greater environmental burden than previously thought (Morello-Frosch et al., 2016; Wang et al., 2021). These findings have added a new dimension to the concept of FOAD. High levels of toxins transmitted from mother to baby through the umbilical cord accumulate in the fetus, causing permanent effects on brain and organ development. Such exposures have been associated with congenital anomalies and diseases such as hormonal disorders, infertility, diabetes, asthma, and cancer later in life.

In conclusion, the toxic load of modern lifestyles is a severe threat to fetal and maternal health, endangering future generations from the very beginning of life. Therefore, the first step toward addressing this crisis should be the elimination of major sources of toxicity and a societal shift toward a more natural and sustainable environment. From a modern medical standpoint, a holistic health approach should be aimed which protecting the body against toxic exposures and enhancing the body's natural detoxification mechanisms.

FOAD Hypothesis

The rise in chronic diseases during the late twentieth century prompted public health researchers to seek new etiological explanations, as it became evident that genetic predisposition and adult lifestyle factors alone could not account for the increasing disease burden. Within this framework, the pioneering epidemiological work of David Barker and his colleagues in England led to the formulation of the FOAD hypothesis, which profoundly transformed perspectives in medicine and public health (Calkins & Devaskar, 2011).

In the 1980s, by linking historical birth records with adult mortality data from Lancashire and Hertfordshire, Barker's research group identified a striking correlation: individuals with lower birth weights had a higher risk of death from CAD in adulthood (Barker, 2004). This observation revealed that LBW is not only a passive indicator of fetal health but an active predictor of adult disease risk. This insight marked a paradigm shift in understanding the lifelong implications of the intrauterine environment and became known as the Barker Hypothesis (Calkins & Devaskar, 2011). LBW is associated with numerous chronic diseases, including CAD, T2DM, cancer (Michels et al., 1996), osteoporosis (Cooper et al., 1997), and various psychiatric disorders (Lahti et al., 2010).

A central principle underlying the FOAD hypothesis is the concept of '*developmental plasticity*', which refers to the fetus's capacity to undergo structural and functional adaptations in response to environmental conditions. A single genotype can produce multiple phenotypes depending on intrauterine events, reflecting the existence of critical developmental windows during which the organism is particularly sensitive or "responsive" to environmental influences. Phenotypic variation is maximized to ensure adaptation to environmental conditions. For example, under circumstances such as maternal malnutrition, the fetus undergoes developmental remodeling, altering the structure and function of non-vital organs (e.g., kidney, pancreas, skeletal muscle) to protect neurodevelopment and promote survival. These adaptive responses also prepare the fetus for additional stressors it may encounter in life outside the womb (Calkins & Devaskar, 2011).

Epidemiological Evidence

The FOAD hypothesis is primarily supported by findings from large-scale birth registries and longitudinal cohort studies—particularly those examining populations exposed to famine—where pregnant women experienced severe malnutrition. These registries allowed researchers to trace individuals from birth to adulthood, linking early-life indicators such as birth weight and childhood growth patterns with the later development of chronic diseases.

One of the most prominent examples is the Dutch Famine Study (1944–1945), during which pregnant women endured six months of severe caloric restriction. Infants exposed to famine in mid- or late gestation were born with significantly lower birth weights, while those exposed during early gestation generally had normal birth weights. However, adults whose mothers experienced famine during mid- or late pregnancy showed impaired glucose tolerance, whereas those exposed during early gestation exhibited a more atherogenic lipid profile and higher body mass index (BMI) (Ravelli et al., 1976). These findings were later confirmed and expanded upon, demonstrating that the timing of nutritional deprivation during pregnancy plays a critical role in determining both fetal growth and adult disease risk (Roseboom et al., 2001). Collectively, these studies indicate that while birth weight serves as a useful surrogate marker, it is ultimately the intrauterine environment that shapes the developmental trajectory of adult health. Early gestational programming, which occurs during organogenesis, tends to result in broad metabolic and neurodevelopmental consequences, whereas late gestational programming, which corresponds to rapid growth phases, induces permanent structural and functional changes in key metabolic organs (Calkins & Devaskar, 2011).

A complementary perspective emerged from the Siege of Leningrad (1941–1944), during which individuals experienced prolonged and extreme malnutrition for over 800 days. Interestingly, unlike the Dutch cohort, those exposed to famine in utero during the Leningrad siege did not exhibit increased rates of insulin resistance, dyslipidemia, hypertension, or CAD in adulthood (Stanner et al., 1997). This striking difference underscores the complexity of developmental programming. In the Netherlands, the abrupt transition from famine to postwar food abundance likely created a mismatch between intrauterine deprivation and postnatal nutritional excess. In contrast, the continued nutritional scarcity in post-famine Leningrad may have provided an environmental match, mitigating the adverse long-term effects.

These contrasting outcomes provided invaluable insights to Barker and his contemporaries, highlighting the intricate interplay between maternal and fetal genotypes and their adaptation to environmental conditions. This concept resonates with the Thrifty Genotype Hypothesis proposed by Neel et al. (1998), which suggests that genetic adaptations favoring efficient energy storage during periods of scarcity become maladaptive in

modern contexts characterized by caloric abundance, predisposing individuals to obesity and metabolic disease.

Building upon this, Hales and Barker later introduced the Thrifty Phenotype Hypothesis (CN, 2001), which emphasizes developmental plasticity rather than genetics. According to this model, nutrient restriction during fetal development induces metabolic adaptations—such as reduced insulin sensitivity and increased energy conservation—that enhance survival in nutrient-poor environments. However, in contemporary societies where nutrient availability is high, these same adaptations predispose individuals to metabolic syndrome, T2DM, and CAD.

Biological Mechanisms Underlying FOAD

The biological process underlying the FOAD hypothesis is the concept of fetal programming, which is defined as the way environmental conditions during the intrauterine period have lasting effects on a fetus's organ development, metabolism, and gene expression, determining lifelong health and disease risk (Calkins & Devaskar, 2011). A range of maternal and environmental factors can influence this programming process, including nutritional status, inflammation, infection (Dalman et al., 2008), glucocorticoid exposure (Welberg et al., 2000), hypoxia, psychological stress (Fride et al., 1986), toxin exposure (Ho et al., 2006), and elevated maternal homocysteine levels (Yajnik et al., 2005). The intrauterine environment signals the conditions expected after birth in childhood and adulthood, prompting adaptive metabolic changes that promote survival.

Epigenetic Regulation

Epigenetics is one of the fundamental mechanisms underlying the persistence of fetal programming. Environmental factors, such as nutrition, stress, and toxins, lead to permanent alterations in gene expression without modifying the DNA sequence.

Epigenetic mechanisms form the bridge between genetics and environmental factors in the FOAD hypothesis. Through processes such as DNA methylation (DNAm), histone modification, and non-coding RNA regulation, epigenetic mechanisms can stably alter gene expression patterns without changing the nucleotide sequence (Rodger & Chatterjee, 2017). The epigenome is highly sensitive to environmental factors, especially in early pregnancy. Therefore, factors such as maternal nutrition, toxin exposure, or stress can induce permanent phenotypic changes during critical windows of development (Safi-Stibler & Gabory, 2020). DNAm profiles in umbilical cord blood serve as biomarkers of intrauterine exposures and can predict an individual's lifelong risk of disease (Dutta & Ruden, 2024).

Inflammation and Oxidative Stress

The common thread of the aforementioned intrauterine pathological factors is the triggering of chronic, low-grade inflammation and oxidative stress in the fetus and placenta. Metabolites of toxic elements increase ROS production, which in turn leads to DNA damage, lipid peroxidation, and cellular senescence (Jomova et al., 2023). Such damage is particularly detrimental to rapidly dividing fetal cells, thereby disrupting organ development and programming. Moreover, prenatal exposure to toxins has been shown to elevate the levels of pro-inflammatory cytokines, including TNF- κ B, IL-6, and IL-8, in both maternal and cord blood (Wang et al., 2025).

Placenta and Umbilical Cord

The placenta, a unique and transient organ formed through the union of maternal and fetal tissues, develops during pregnancy to mediate the exchange of nutrients, gases, and waste products between the mother and fetus (Maltepe et al., 2010). Functioning as a selective gatekeeper, the placenta regulates maternal–fetal interactions and can effectively filter or limit the transfer of many toxic substances. However, this barrier function is not always fully protective; in certain cases, it may be inadequate, or a placental disorder may cause excessive transmission of maternal exposure to the fetus (Rager et al., 2020).

The chorionic plate, the fetal-facing side of the placenta, is connected to the fetus via the umbilical cord. The umbilical cord contains two arteries and one vein; the umbilical vein carries oxygen-rich blood and nutrients from the placenta to the fetus, while the umbilical arteries carry deoxygenated blood and waste from the fetus back to the placenta (Maltepe et al., 2010). Maternal and fetal exposure to toxic substances can be directly assessed through umbilical cord blood analysis. The detection of hundreds of industrial chemicals—including lead (Pb), mercury (Hg), polychlorinated biphenyls (PCBs), per- and polyfluoroalkyl substances (PFAS), and bisphenol A (BPA)—in cord blood underscores both the complexity of prenatal exposure and the extent of the fetal chemical burden. Moreover, DNA methylation profiles in umbilical cord blood serve as biomarkers of persistent epigenetic alterations resulting from these exposures (Dutta & Ruden, 2024; Gundacker et al., 2021). Although the placenta functions as a selective barrier between mother and fetus, it cannot completely prevent the transfer of toxic substances such as arsenic, lead, and mercury. Placental dysfunction or deficiency (e.g., due to gestational hypertension or chronic maternal inflammation) can lead to excessive transmission of maternal toxic exposure to the fetus. Furthermore, individual genetic variants (detoxification genes such as GSTT1 and GSTM1) can increase the transplacental transfer of toxic substances and their accumulation in the fetus, making genetically predisposed individuals more vulnerable (Gundacker et al., 2021).

Toxin Exposure of Fetus

Recent studies have shown that hundreds of toxic substances accumulate in the blood and umbilical cord blood of pregnant women. Prenatal exposure to environmental toxins and heavy metals poses serious risks to fetal development and postnatal health. These exposures predispose to both short-term neonatal complications and long-term chronic diseases through oxidative stress, inflammation, endocrine disruption, and epigenetic changes (Dutta & Ruden, 2024).

Potentially Toxic Elements (PTEs) are elements released into the environment through both natural geological processes and anthropogenic activities such as mining, smelting, manufacturing, fossil fuel combustion, and pesticide use (Nieder & Benbi, 2024). Among these, arsenic (As), cadmium (Cd), chromium (Cr), lead (Pb), and mercury (Hg) are of primary public health concern due to their high toxicity and persistence in the environment (Tchounwou et al., 2012). Exposure to these elements occurs through various routes, including contaminated drinking water, seafood, old paints, and industrial emissions (Dutta & Ruden, 2024).

Over two million individuals around the world are exposed to arsenic, primarily through contaminated drinking water. Arsenic acts as a potent endocrine disruptor and carcinogen (Ullah et al., 2023). Industrial exposure to arsenic occurs in various sectors, including glass manufacturing, textile production, ammunition and paper processing, as well as in the use of wood preservatives and related industries. Furthermore, in the cosmetics industry, color pigments used in eyeshadow formulations frequently contain toxic metals, including arsenic. Arsenic present in cosmetics can be absorbed into the bloodstream through percutaneous penetration; therefore, the permissible limit for heavy metal impurities in cosmetic products is set below 5 ppm. Major dietary sources of organically bound arsenic include seafood, mushrooms, rice, and poultry (Jones, 2007).

Mercury (Hg) concentrations in the oceans have tripled since the Industrial Revolution. Mercury is released into the environment from both natural and anthropogenic sources, including coal-fired power plants (Dutta & Ruden, 2024). In aquatic ecosystems, Hg is converted to methylmercury (MeHg) by anaerobic microorganisms, leading to bioaccumulation at higher trophic levels. Humans are primarily exposed to MeHg through the consumption of contaminated fish and seafood. Another important route of exposure is occupational, particularly in industries such as electrical equipment and automotive manufacturing, chemical processing, metalworking, construction, and healthcare professions, where mercury is used in instruments (Kim et al., 2016).

Dental practices represent one of the major sources of inorganic mercury use. Extensive evidence indicates that dentists and dental staff handling amalgam are chronically exposed to mercury vapor, leading to body burdens substantially higher than those found in the

general population. Elemental mercury, a major component of dental amalgam used as a restorative material, poses a notable occupational risk. The mean Hg concentration in dentists' breathing zones, measured using personal dosimeters, was reported as 29.2 $\mu\text{g}/\text{m}^3$ —exceeding the Occupational Exposure Standard of 25 $\mu\text{g}/\text{m}^3$ (Ritchie et al., 2004). Correspondingly, dentists exhibited urinary mercury concentrations three to four times higher than the control group (Karahalil et al., 2005). In dental personnel, mercury levels in hair, urine, toenails, and blood were on average at least twice as high as those of controls (Kim et al., 2016).

Cadmium (Cd) is primarily encountered in industrial settings such as battery manufacturing, pigment and coating production, and plastic processing. It is also emitted into the environment as a result of mining, metal smelting, and the combustion of fossil fuels. Due to its persistence and long biological half-life, cadmium accumulates in soil and can contaminate food sources, particularly crops grown in polluted areas. Major sources of human exposure include industrial byproducts, contaminated water or soil, and tobacco products. Smoking represents a significant exposure route, as tobacco plants efficiently absorb cadmium from the soil (Dutta & Ruden, 2024).

Lead (Pb), another toxic heavy metal, is widely distributed throughout the environment as a consequence of its widespread industrial use. Many household and consumer products—such as paints, plumbing materials, pipes, eyeglasses, batteries, cosmetics, and ammunition—contain lead or lead compounds. Additionally, air pollution in densely populated urban areas contributes to ongoing lead exposure and environmental accumulation (Jomova et al., 2025).

Studies have identified various toxic substances in the urine, peripheral blood, amniotic fluid, and umbilical cord blood of pregnant women, demonstrating that pregnant women and their fetuses are exposed to numerous environmental pollutants originating from industrial chemicals and consumer products.

Dutta and Ruden (2024) conducted a systematic analysis and showed that exposure to various heavy metals, such as arsenic (As), cadmium (Cd), chromium (Cr), lead (Pb), and mercury (Hg), causes DNA methylation (DNAm) changes in umbilical cord blood, and these DNAm changes lead to serious short- and long-term health problems.

Morello-Frosch et al. (2016) analyzed 59 different chemicals in maternal and umbilical cord blood samples from 78 mother-baby pairs. Of the chemicals analyzed, 89% (52 of 59) were detected in maternal blood, and 70% (41 of 59) in umbilical cord blood, indicating that pregnant women, and therefore their babies, are exposed to a wide variety of environmental toxins. The study reported particularly high levels of endocrine-disrupting chemicals (EDCs) such as phthalates and perfluoroalkyl substances (PFAS), with substantial placental transfer efficiency. Moreover, chemicals commonly used in

personal care products, including bisphenol A (BPA) and parabens, were frequently detected in both maternal and fetal circulation, suggesting that daily exposure to these substances poses a potential risk to fetal development.

BPA, a well-known endocrine disruptor, has been linked to various adverse health outcomes, including cancer, infertility, diabetes, and obesity. Today, this compound is widely used to manufacture optical materials, electronic equipment, reusable plastic bottles, plates, bowls, cups, microwave-safe utensils, and food containers. It is also used in epoxy resin paints, vinyl chloride-based medical materials, thermal paper, flame retardants, printing inks, sports equipment, dental materials, and compact discs (Susana Almeida et al., 2018).

Wang et al. (2021) used a “suspect screening” method to investigate the presence of industrial chemicals in maternal and newborn blood, identifying 557 unique chemical formulas in serum samples taken from 30 mother-newborn pairs. The study revealed that numerous industrial compounds are present in both maternal and fetal circulation, demonstrating that many of these substances are capable of crossing the placental barrier. Among the detected chemicals were PFAS, plasticizers, and PTEs, 55 of which had never previously been reported in human blood. Because some of the chemicals have never been reported before, their sources of exposure and potential health effects remain unknown.

Similarly, a two-year study commissioned by the Environmental Working Group (EWG) and Rachel’s Network (2009), conducted at five independent laboratories, analyzed the umbilical cord blood of 10 babies of different races and identified 287 toxic industrial chemicals and contaminants. Among the 287 chemicals detected in umbilical cord blood, 180 are recognized as carcinogenic in humans or animals, 217 are neurotoxic, and 208 have been demonstrated to induce birth defects or abnormal development in animal studies. The full impact of pre- and postnatal exposure to these complex mixtures of carcinogens, developmental toxins, and neurotoxins remains incompletely understood. Collectively, these findings clearly demonstrate that fetuses are exposed in utero to a broad spectrum of environmental contaminants, including hundreds of chemicals known or suspected to disrupt normal development, impair neurocognitive function, or contribute to carcinogenesis.

Short- and Long-Term Health Effects of Fetal Exposure to Heavy Metals and Toxins

Prenatal exposure to PTEs and heavy metals poses serious risks to fetal development and postnatal health. These exposures predispose to both short-term neonatal complications and long-term chronic diseases through oxidative stress, inflammation, endocrine disruption, and epigenetic changes (Niede & Benbi, 2022).

Heavy Metals

Prenatal exposure to heavy metals poses critical risks to the fetus. Al-Saleh et al. (2017) and Wang et al. (2025) have shown that metals such as cadmium, lead, arsenic, mercury, chromium, and nickel are detected in maternal and umbilical cord blood, and that this exposure leads to increases in inflammation and oxidative stress markers (TNF α , IL-6, IL-8, TGF β , PAB). These findings demonstrate that maternal exposure directly affects the fetus and initiates biochemical stress responses in the intrauterine period.

Heavy metal toxicity has been proven to cause dysfunction in numerous physiological processes, leading to various pathological outcomes. The heavy metals with the most significant health effects are listed below:

Lead (Pb) exposure causes cognitive developmental disorders, learning disabilities, and permanent damage to nervous system functions in children. Lead poisoning usually develops as a result of cumulative exposure over months or years. Children under 6 years of age are especially susceptible to lasting adverse health effects due to their higher absorption rates and ongoing brain development. In addition to its neurotoxicity, Pb exposure has also been associated with a broad range of systemic impacts, including reproductive and hormonal dysfunctions, anemia and leukopenia, non-alcoholic fatty liver disease, coronary artery and cardiovascular disorders, and various cancers such as lung cancer. Moreover, Pb is associated with behavioral abnormalities, memory and attention deficits, depression, and anxiety (Adriana et al., 2025).

Mercury (Hg) exhibits significant toxic effects on the neurological system and renal structures. Even low-dose Hg exposure in humans can cause serious health problems, particularly during prenatal development, infancy, childhood, and adolescence (Kim et al., 2016). Hg toxicity is multisystemic, affecting the lungs, skin, eyes, and the nervous, cardiovascular, and renal systems. Intrauterine mercury exposure is known to impair fetal brain development. Additionally, inhalation of mercury vapor may result in necrotizing bronchitis and pneumonitis, which can progress to respiratory failure. Hg exposure has been associated with arrhythmias, cardiomyopathy, and nephrotoxicity. The wide-ranging systemic and developmental toxicity of mercury underscores the critical need for minimizing maternal and early-life exposure (Tchounwou et al., 2012).

Cadmium (Cd) has been strongly associated with bone demineralization and renal dysfunction (Åkesson et al., 2006). Chronic low-dose Cd exposure is believed to contribute to the development of several chronic lung diseases, including emphysema, asthma, and bronchitis, as well as hypertension. Prolonged Cd exposure contributes to the onset of various pathological conditions, including cancer, leukemia, and genetic toxicity. Epidemiological evidence further suggests a potential association between Cd exposure and certain cancers, such as prostate, bladder, pancreatic, kidney, and breast cancers.

Beyond its carcinogenicity, Cd may play an etiological role in central nervous system pathologies such as Alzheimer's disease, Parkinson's disease, Huntington's disease, amyotrophic lateral sclerosis, and multiple sclerosis. It is also associated with cognitive and behavioral impairments and with chronic skeletal disorders such as osteoporosis and osteomalacia, particularly affecting the pelvic bones, femurs, vertebral bodies, and scapulae. Importantly, Cd can cross the placental barrier, exerting teratogenic effects and posing serious risks to fetal growth and development (Charkiewicz et al., 2023).

Arsenic (As) toxicity is strongly correlated with the development of dermal, pulmonary, and bladder carcinomas. According to the International Agency for Research on Cancer (IARC) (2012) and the U.S. Food and Drug Administration (FDA), arsenic is classified as a Group 1 human carcinogen and increases the risk of bladder, lung, kidney, and liver tumors. Arsenic can affect multiple organ systems, leading to various cardiovascular disorders, neurological effects, respiratory problems, and kidney diseases. Chronic exposure has been linked to vascular pathologies, including stroke, ischemic heart disease, and peripheral artery disease. Furthermore, arsenic acts as a potent endocrine disruptor and carcinogen, with prenatal and early-life exposures linked to elevated cancer risk and mortality later in life (Palma-Lara et al., 2020).

PFAS Exposure

In recent years, per- and polyfluoroalkyl substances (PFAS) have become a serious global concern. These substances are persistent in the environment, accumulate in biological tissues, and exhibit endocrine-disrupting effects. Common PFAS compounds include perfluorononanoic acid (PFNA), perfluorooctanoate (PFOA), and perfluorooctane sulfonate (PFOS). These synthetic chemicals are resistant to degradation and can accumulate in maternal and fetal tissues, leading to adverse developmental outcomes. Several cohort studies have demonstrated the detrimental effects of prenatal and early-life PFAS exposure. High levels of PFOA and PFOS in cord blood have been associated with reduced language and cognitive abilities in children (Oh et al., 2022). Similarly, PFOS and PFNA exposures have been linked to impairments in memory and reasoning skills in childhood (Spratlen et al., 2020). PFAS mixtures have also been shown to disrupt thyroid hormone regulation, increasing total and free thyroxine (TT4, FT4) levels while decreasing thyroid-stimulating hormone (TSH) concentrations in newborns, indicating impaired thyroid function (Guo et al., 2021). PFOS and PFNA exposures have also been associated with alterations in triglyceride and lipid profiles in umbilical cord blood (Sinisalu et al., 2021). Furthermore, PFAS and organochlorine pesticides have been shown to disrupt the gut microbiome in infants, leading to a decline in beneficial species such as *Bifidobacterium* (Naspolini et al., 2022). In addition, PFOS and PFOA exposures have been associated with lower body mass index (BMI) in childhood (Horikoshi et al., 2021) and decreased lung function (FVC, FEV1) in children (Kung et al., 2021).

Microplastics

Epidemiological and experimental evidence suggest that microplastics (MP) can accumulate in key physiological systems such as the digestive, reproductive, and cardiovascular systems and disrupt biological function. Defined as plastic particles smaller than 5 mm, MP occur in various forms such as fragments, fibers, spheres, or granules. The most common types of MP identified in biological and environmental samples are polyethylene (PE), polypropylene (PP), polycarbonate (PC), polystyrene (PS), polyvinyl chloride (PVC), and polyethylene terephthalate (PET) (Sun & Wang, 2023). Prenatal exposure to MP has been associated with adverse pregnancy outcomes, including restricted fetal growth, low birth weight, and pregnancy-induced hypertension (PIH) (Wang et al., 2024). Negative correlations have also been reported between PE and PC derived MP and neonatal Apgar scores, while PVC exposure has been linked to increased maternal complications and reduced neonatal length and weight (Zhang et al., 2025).

Microplastics exhibit multifaceted effects on the immune system. Experimental studies have shown that PE exposure increases TNF- α production in macrophages by 2.3-fold and IL-6 by 1.8-fold, while in animal models it elevates the proportion of CD3⁺CD4⁺ T cells and reduces uterine NK cell counts by approximately 42%, resulting in an immune imbalance at the maternal–fetal interface (Qiao et al., 2025; Zhang et al., 2025). MP also compromises intestinal integrity by disrupting tight junction proteins, leading to increased intestinal permeability and a reversal of the Firmicutes/Bacteroidetes ratio within the gut microbiota (Fackelmann et al., 2023; Qiao et al., 2025). Reproductive health appears particularly vulnerable to MP exposure. Bisphenol A (BPA) released from MP may disrupt hormonal balance by binding to the estrogen receptor- α and lead to changes associated with polycystic ovary syndrome (PCOS) (Adhikari et al., 2024). Furthermore, nanosized PS-MP can cross the blood-testis barrier, causing Sertoli cell apoptosis and decreased sperm motility (Liu et al., 2024; Zhao et al., 2024). In women, microplastic exposure has been associated with endometrial thinning, collagen accumulation, granulosa cell apoptosis, and a decrease in follicle count (Qiao et al., 2025; Zhang et al., 2025).

A particularly striking recent finding links maternal MP burden to mental health outcomes. Elevated MP concentrations in maternal blood have been correlated with a higher risk of postpartum depression, reflected by increased Edinburgh Postnatal Depression Scale (EPDS) scores (Saraluck et al., 2024). These data highlight the broad systemic and psychological implications of maternal MP exposure and underscore the critical need for further mechanistic and epidemiological research.

Table 1

Most common toxicants, exposure routes, and health effects.

No	Substance	Source of Exposure	Health Effects	References
1	Arsenic (As)	Contaminated drinking water; gold, silver, and other metal mining; pesticide production and use; processing of glass, textiles, ammunition, paper, and wood preservatives; cosmetics industry (eyeshadow).	A potent endocrine disruptor and carcinogen.	(Palma-Lara et al., 2020; Ullah et al., 2023)
2	Cadmium (Cd)	Industrial batteries, paint, coating, plastic production; mining; casting and fossil fuel combustion; widespread exposure through contaminated water/soil and tobacco products; air pollution.	Bone demineralization and renal function disorders; neurodegenerative diseases; neurological, learning, and/or behavioral difficulties in children, neuropathy, and impairment in memory, attention, and psychomotor function; cardiovascular diseases; respiratory diseases; reproductive system disorders.	(Atsdr, 1997; Charkiewicz et al., 2023)
3	Chromium (Cr)	Stainless steel production; leather tanning; pigment production; painting; metal plating; wood processing.	Gastrointestinal problems; male reproductive system function disorders; neurological, cardiovascular, hepatological, and neuronal toxicity; allergic reactions; anemia.	(Hossini et al., 2022; Shin et al., 2023)
4	Lead (Pb)	Paint; plumbing materials; pipes; glasses; batteries; cosmetics and ammunition; fossil fuels; mine sites; foundry sites.	Cognitive development disorders, learning difficulties, and permanent damage to nervous system functions in children; associated with reproductive system and hormonal disorders; lung cancer; anemia/leukopenia; non-alcoholic fatty liver disease; coronary artery diseases; some neurological diseases.	(Adriana et al., 2025)
5	Mercury (Hg)	Coal-fired power plants, contaminated water, fish consumption, and dental materials.	Toxic effect on the neurological system and kidneys; cardiomyopathy; also a negative effect on the respiratory and reproductive systems.	(Kim et al., 2016; Tchounwou et al., 2012)
6	Per- and polyfluoroalkyl substances (PFAS)	Consumption of Ultra-Processed Foods (UPF); food packaging; synthetic textile products (raincoats, umbrellas, carpets, and upholstery fabrics); house dust agar; non-stick pots and pans; cosmetics.	Impairment of thyroid function; disruption of the gut microbiome.	(Guo et al., 2021; Napolini et al., 2022)
7	Perfluorononanoic acid (PFNA)	Consumption of Ultra-Processed Foods (UPF); food packaging; synthetic textile products; house dust agar; non-stick pots and pans; cosmetics.	Impairments in memory and reasoning skills in childhood; impairment in triglyceride and lipid profiles.	(Sinisalu et al., 2021; Spratlen et al., 2020)

8	Perfluorooctanoate (PFOA)	Consumption of Ultra-Processed Foods (UPF); food packaging; synthetic textile products; house dust agar; non-stick pots and pans; cosmetics.	Associated with decreased language and perceptual abilities in children; associated with lower BMI in childhood; decreased lung function (FVC) in children.	(Horikoshi et al., 2021; Kung et al., 2021; Oh et al., 2022)
9	Perfluorooctane sulfonate (PFOS)	Consumption of Ultra-Processed Foods (UPF); food packaging; synthetic textile products; house dust agar; non-stick pots and pans; cosmetics.	Associated with decreased language and perceptual abilities in children; impairments in memory and reasoning skills in childhood; impairment in triglyceride and lipid profiles; associated with lower BMI in childhood; decreased lung function (FVC) in children.	(Horikoshi et al., 2021; Kung et al., 2021; Oh et al., 2022; Sinisalu et al., 2021; Spratlen et al., 2020)
10	Organochlorine pesticides (OCs)	DDT; chlordane; other pesticides.	Disrupts the gut microbiome; cancer and reproductive system disorders.	(Naspolini et al., 2022)
11	Microplastics (MP)	Consumption of Ultra-Processed Foods (UPF); long-term use of plastic bottles, plastic cutlery, take-out consumption; textiles in homes and clothing; marine foods; seafood; table salt; drinking water; beverages; drug capsules; cosmetic products; packaged foods.	Associated with restricted fetal growth, LBW, PIH; multilayered effects on the immune system; increases gut permeability.	(Fackelmann et al., 2023; Le et al., 2024; Qiao et al., 2025; Sangkham et al., 2022; Wang et al., 2024; Yun et al., 2024)
12	Nano-sized polystyrene microplastics (PS-MP)	Table salt, fish; drinking water, bottled water.	Sertoli cell apoptosis and reduced sperm motility.	(Liu et al., 2024; Zhao et al., 2024)
13	Bisphenol A (BPA)	Widely used in PC plastics for optical materials and electronic equipment; bottles; reusable plastic bottles; plates; bowls; cups; microwaveable kitchenware and food containers. Also in epoxy resin paints, medical vinyl chloride, thermal paper, flame retardants, printing inks, sports equipment, dental materials, CDs, and DVDs.	Hormonal balance disorder, PCOS, cancer, and infertility; associated with diabetes and obesity.	(Adhikari et al., 2024; S. Almeida et al., 2018)
14	Polyaromatic hydrocarbons (PAHs)	Pollutants from gasoline and garbage incineration.	Associated with cancer.	(EWG, 2009)
15	Polybrominated dibenzodioxins and furans (PBDD/F)	Contaminants in brominated flame retardants. Pollutants and byproducts from plastic production and incineration.	Toxic to the developing endocrine system.	(EWG, 2009)

16	Perfluorinated chemicals (PFCs)	Active ingredients or breakdown products of Teflon, Scotchgard, fabric and carpet protectors, and food packaging coatings.	Linked to cancer and birth defects.	(EWG, 2009)
17	Polychlorinated dibenzodioxins and furans (PCDD/F)	Contaminants: byproducts of PVC production; industrial bleaching and incineration.	Cancer, toxic to the endocrine system.	(EWG, 2009)
18	Polybrominated diphenyl ethers (PBDEs)	Furniture foam, computers, televisions.	Negatively affects brain development and the thyroid.	(EWG, 2009)
19	Polychlorinated Naphthalenes (PCNs)	Wood preservatives; varnishes; machine lubricants; waste incineration.	Causes liver and kidney damage.	(EWG, 2009)
20	Polychlorinated biphenyls (PCBs)	Industrial insulators and lubricants.	Causes cancer and nervous system problems.	(EWG, 2009)

Current evidence strongly indicates that both heavy metals and persistent environmental contaminants—such as PFAS and MP—adversely affect fetal health. These effects manifest in the short term as oxidative stress, inflammation, hormonal imbalance, and metabolic disruptions, while in the long term, they can lead to serious health consequences such as cognitive developmental problems, immune disorders, cardiovascular diseases, respiratory problems, and increased cancer risk.

Given these multifactorial risks, preventing toxic exposures during the prenatal period is essential not only for individual health but also for public health and the well-being of future generations. Environmental toxin control must therefore be prioritized within public health strategies. Developing and enforcing stricter regulations, especially for vulnerable populations such as pregnant women and infants, is imperative to mitigate transgenerational health consequences.

An Important Agent in Toxicity: Detergent

Detergents are chemical agents containing anionic surfactants such as sodium dodecyl sulfate (SDS) and sodium lauryl sulfate (SLS), which are widely used in numerous household and industrial applications. These compounds are recognized as toxic agents with the potential to penetrate cellular barriers, reach systemic circulation, and interact with intracellular structures.

In daily life, humans are continuously exposed to detergents through common products such as soap, shampoo, body wash, laundry detergent, and dishwashing liquids. Detergent residues tend to accumulate on fabrics and in the environment, often requiring multiple rinsing cycles for complete removal (Bernal-Jácome et al., 2024; Matrosenko et al., 2025).

A study by Wang et al. (2019) reported that even detergent residue diluted 1:2500 caused

toxic and barrier-disrupting effects. It was stated that laundry detergent exposure, barely at a dilution of 1:50,000, had negligible effects. Based on these findings, approximately 50 tons of water would be required to neutralize the effects of a single liter of detergent. High concentrations of detergent residue accumulated on clothing can enter the body through inhalation, as well as direct contact with the skin, and can reach internal organs such as the lungs (Wang et al., 2019). Similarly, dishwashing detergent residues can be ingested with food and disrupt epithelial integrity in the gastrointestinal tract.

Detergents disrupt the integrity of tight junction proteins in the skin, lung, and gastrointestinal epithelium, increasing barrier permeability and causing structural damage (Doyle et al., 2023; Rinaldi et al., 2024). This barrier defect increases the risk of tissue penetration of allergens, microbial enzymes, toxins, and environmental pollutants, paving the way for the development of inflammatory responses (Wang et al., 2019). Consequently, detergents represent a dual toxic threat—not only due to their direct harmful effects but also by enhancing the absorption and toxicity of other xenobiotics.

At the cellular level, detergents exert cytotoxic effects through lipid dissolution and protein denaturation within cell membranes (Fan et al., 2024). SLS, in particular, can induce cell death by interacting with lipids and proteins in cell membranes (Birant et al., 2022). Epidemiological data indicate that chronic or repeated exposure to detergents increases the prevalence of asthma, atopic dermatitis, and eosinophilic esophagitis in genetically predisposed individuals (Sözener et al., 2020; Yazici et al., 2023). It has also been reported that detergents induce dose-dependent cytotoxicity in human lymphocytes, causing significant cell death at high concentrations (Cooley-Andrade et al., 2016). Moreover, some detergent ingredients may weaken the immune response by suppressing immunoglobulin (IgG, IgM) synthesis (Jahnová et al., 1993).

Studies in animal models have revealed that exposure to powdered detergents causes significant histopathological changes in liver tissue. The severity of damage increases proportionally with exposure duration and concentration (Tayfur et al., 2020). These findings demonstrate that detergents have significant hepatotoxic potential.

Furthermore, it has been reported that some detergent ingredients, such as nonylphenol ethoxylates, exhibit endocrine-disrupting effects and may contribute to the development of metabolic disorders by antagonizing thyroid hormone signaling (Bernal-Jácome et al., 2024).

In conclusion, detergents, which are frequently used in daily life and difficult to rinse off, are among the most common and longest-lasting toxins. Far from being harmless cleaning agents, detergents are significant contributors to environmental and systemic toxicity, disrupting cellular barrier integrity, leading to systemic toxicity, activation of inflammatory responses, and structural damage at the organ level. By disrupting cellular

barrier integrity, detergents not only induce direct cytotoxic and inflammatory responses but also facilitate the penetration and bioaccumulation of other environmental toxins.

Fetal Effects of Maternal Diet

The mother's nutrition during pregnancy is one of the most important factors affecting fetal development and health. Malnutrition during this period can have lasting effects by impairing placental nutrient transfer, leading to intrauterine growth restriction (IUGR). Insufficient nutrient supply also alters fetal metabolic programming, increasing the risk of insulin resistance, type 2 diabetes, and obesity in later life through mechanisms such as reduced pancreatic β -cell mass and dysregulated adipose tissue development (Calkins & Devaskar, 2011).

The quality of the diet is as important as inadequate nutrition itself. In a cohort conducted between 2007 and 2015 (Kim et al., 2019), it was shown that consuming high amounts of trans fatty acids (TFA) containing flour and sugar products during pregnancy negatively impacts fetal programming by triggering a proinflammatory response and causing dysbiosis in the gut microbiota. TFA lipotoxicity disrupts the intestinal barrier, increasing permeability and allowing allergens to enter the circulation, which increases the risk of food allergy (FA) in newborns. Moreover, continuation of a TFA-rich diet during the first six months of breastfeeding has been shown to exacerbate these adverse effects on the offspring. This effect is particularly pronounced in infants with certain genetic variations in the CD14, GSTM1, and GSTT1 genes, demonstrating the complex interaction between environmental triggers and genetic predisposition (Kim et al., 2019).

Maternal diet can also elicit exposure to environmental pollutants. For individuals not occupationally exposed to the toxicants, food is the main source of exposure. Although these substances are present in relatively low concentrations in food products, they pose a serious threat, especially when chronic exposure is considered (Koch et al., 2022). Recent evidence indicates a strong association between the consumption of ultra-processed foods (UPFs) during pregnancy and increased newborn exposure to PFAS. UPFs often contain PFAS originating from packaging materials. In a cohort study (Naspolini et al., 2022), infants of mothers who consumed three or more UPF subgroups per week exhibited significantly higher PFAS concentrations compared to infants of mothers who consumed none (mean 2.47 ng/mL vs. 1.86 ng/mL; $p < 0.05$). This finding suggests that maternal dietary habits, particularly UPF consumption, may increase fetal PFAS exposure and program early-life exposure to environmental toxins with potential long-term health effects.

Similarly, a recent study by Zhang et al. (2025) examined the relationship between maternal lifestyle factors and MP exposure. PE intake was found to be significantly correlated with prolonged use of plastic bottles and cutlery, as well as with the

frequency of takeaway food consumption, both in the year preceding pregnancy and during pregnancy. Accordingly, PC intake was strongly correlated with the frequency of takeaway food consumption one year before and during pregnancy. Total MP burden was linked to the frequency of plastic utensil use and takeaway consumption, indicating that common plastic-related lifestyle practices may meaningfully contribute to maternal and fetal MP exposure.

Collectively, these findings highlight that maternal everyday dietary behaviors can serve as important determinants of the toxic exposure of the fetus. Common plastic polymers such as PE and PC, and persistent chemicals like PFAS, may increase the risk of pregnancy complications and interfere with fetal development. Maternal nutrition is one of the most critical determinants of fetal development. Mother's nutritional intake and toxic exposure influence not only intrauterine growth but also the fetus's metabolic and epigenetic programming in later life, thus affecting lifelong health.

Toxic Exposure Prevention and Detoxification Approaches

The first and most effective step in preventing toxic accumulation is to minimize exposure at its source. In modern daily life, individuals are frequently and often unknowingly exposed to various environmental chemicals through common products such as plastic bottles, disposable kitchenware, food storage containers, packaged and processed foods, cosmetics, and household cleaning agents. It has been shown that these products frequently contain phthalates, bisphenols (especially BPA and BPS), parabens, and heavy metal residues (EWG, 2009). Beyond household and consumer products, additional exposure sources include agricultural produce containing pesticide residues, seafood with elevated concentrations of heavy metals—especially mercury in large predatory fish—and ambient air pollution. Promoting clean and sustainable agricultural practices, reducing the use of single-use plastics, and preferring recyclable and glass-based packaging materials represent practical and effective strategies to mitigate both individual and population-level toxic exposure. These measures not only decrease environmental contamination but also play a critical role in protecting maternal and fetal health across generations.

Because exposure to toxins cannot be entirely avoided, supporting the body's natural detoxification systems (liver, kidney, lymphatic system, skin) is gaining importance. In this context, dietary supplements used to support detoxification have gained increasing scientific and public interest in recent years. Evidence suggests that various vitamins, minerals, and phytochemicals can support liver function, strengthen antioxidant defenses, and promote metabolic detoxification pathways. For instance, antioxidants such as resveratrol precursors, D-glucaric acid, curcumin, and glutathione, as well as dietary fibers, are among the most researched dietary supplements due to their detoxification-

supporting properties (El-Khodori et al., 2023; Panda et al., 2023). Furthermore, high-chlorophyll foods such as spirulina and chlorella have been reported to exhibit potential detoxification benefits for liver health and heavy metal elimination (Lee et al., 2015; Thitame et al., 2025). Notably, studies on clays such as bentonite and zeolite, which are natural and additive-free substances frequently used in ancient medicine, have shown their effectiveness in detoxification by adsorbing heavy metals and microtoxins (Dhar et al., 2023; José Mendes dos Reis et al., 2024).

An important point to emphasize here is the frequent detection of heavy metals such as lead, cadmium, mercury, and arsenic in food supplements. Recent studies have reported that 68-88% of tested dietary supplements contain at least one heavy metal, with some products exceeding permissible limits, particularly for lead and cadmium (Naz et al., 2024). Moreover, MP contamination was detected in all tested dietary fiber supplements, with powder-based products showing the highest levels of MP (Panneerselvam et al., 2024). These findings highlight the need for caution in the use of industrially produced food supplements. Ironically, these supplements, used for toxin removal, may unknowingly lead to further toxin exposure. Therefore, personalized approaches using holistic medical testing methods are recommended to ensure the optimal and safe use of all these nutritional supplements.

Furthermore, complementary and traditional approaches for detoxification are also being investigated. Cupping therapy is a traditional practice based on the principle of drawing blood using a vacuum method through superficial skin incisions. Recent scientific studies have shown that this method not only improves circulation but also contributes to the removal of toxic elements from the blood. Saeed and Sheikh (2023) reported significantly higher concentrations of heavy metals, including Lead (Pb), Cadmium (Cd), Nickel (Ni), Aluminum (Al), and Copper (Cu), in wet cupping blood compared to venous blood, supporting the detoxification potential of this technique. A pilot study by Umar et al. (2018) observed a significant decrease in Al, Zinc (Zn), and Cd levels 30 days after cupping. The researchers proposed that this effect may be attributed to cupping's potential to enhance circulation, decrease free radical burden, and support the body's detoxification processes.

Acupuncture, a therapeutic modality rooted in TCM, aims to restore homeostatic balance by regulating the flow of vital energy (Qi) throughout the body. Contemporary research indicates that acupuncture not only provides symptomatic relief but also modulates immune responses, attenuates oxidative stress, and supports the physiological functions of key detoxification organs (LaPaglia et al., 2016).

Bioresonance Therapy (BRT) is a non-invasive biophysical method that regulates the body's vibrational communication by influencing the primovascular system and meridian

systems using the body's own electromagnetic frequencies. In BRT applications, environmental toxins, heavy metals, microorganisms, allergens, and other pathogenic factors are detected using vibrational diagnostic methods. Frequency modulation is then employed to neutralize the bioenergetic effects of these factors, thereby restoring the organism's energetic equilibrium. Emerging evidence suggests that BRT may enhance the elimination of metabolic wastes and toxins by re-permeabilizing the disrupted extracellular matrix, thereby improving cellular detoxification capacity (Hennecke, 2012; Karakos et al., 2019; Kirsever et al., 2022).

Protection from toxic exposure is possible through social and individual awareness and behavioral changes, particularly through reestablishing a healthy ecological environment. Scientific data demonstrates that traditional methods such as cupping and acupuncture, and contemporary vibrational methods such as bioresonance and homeopathy, can support the body's natural detoxification system when applied in conjunction with appropriate and natural nutritional supplements.

Conclusion

One of the biggest challenges in dealing with health problems today is the rapid increase and transformation of disease-causing agents. In current medical literature, the etiology of many diseases can no longer be explained solely by the classical pathogens such as viruses, bacteria, or parasites. Instead, toxic synthetic compounds such as environmental toxins, industrial chemicals, genetically modified food, artificial additives, and pesticides used extensively in agriculture also play a significant role in disease development and progression (Landrigan & Fuller, 2015; Grandjean & Landrigan, 2014).

Growing epidemiological, molecular, and epigenetic evidence strongly supports that an individual's lifelong health is shaped within the womb. Factors such as malnutrition, toxic exposures, stress, inflammation, and hypoxia during the fetal period lead to programming at the cellular level, paving the way for metabolic, cardiovascular, neurological, and immunological diseases later in life. This phenomenon, described by the FOAD concept, underscores the importance of the prenatal environment in determining long-term health outcomes.

Today, it is known that toxin exposure caused by modern life no longer threatens adult health alone. Numerous studies have demonstrated that heavy metals, PFAS, MP, endocrine disruptors, and many other environmental contaminants is capable of crossing the placental barrier to reach the fetus via the umbilical cord. This evidence challenges the long-held notion of "intrauterine protection," revealing that even during the prenatal period, the fetus is exposed to the toxic burden of the modern world. The persistence of these effects through epigenetic mechanisms highlights a growing concern

for intergenerational transmission of health risks.

In response to this complex and systemic issue, strategies aimed at reducing toxic exposure and supporting the body's intrinsic detoxification systems are of vital importance. Traditional therapeutic methods such as cupping and acupuncture, alongside contemporary holistic vibrational medicine approaches including BRT and homeopathy, demonstrate potential in enhancing detoxification, restoring homeostatic balance, and improving extracellular matrix permeability. These modalities not only provide symptomatic relief but also promote holistic healing by addressing the underlying causes of disease through mechanisms such as immunomodulation and reduction of oxidative stress.

For the protection of the health of future generations and public health, attention should be refocused on the pre-pregnancy period and the pregnancy process. Based on the principle that “Eliminating harm is more important than providing benefit”, the ultimate goal should be to ensure the health of future generations and to build a clean, toxin-free society for sustainable health. To build a healthy society, preventive medicine should receive at least equal emphasis as curative medicine in medical education and health management, and policies should be developed accordingly.

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