



Environmental determinants of undernutrition and micronutrient deficiencies in children

Executive summary

Nutrition, particularly during the first 1,000 days of life – from conception to the child's second birthday – is a core determinant of child survival, physical growth and development, present and future health and well-being, and learning and earning potential, with positive impacts into the next generation. Multiple factors determine children's diets and nutrition globally. Growing evidence reinforces the role of the environment as a fundamental determinant of nutrition and other health-related outcomes. **Environmental hazards** – agents or conditions that exist in a person's environment with the potential to cause harm – can be physical (e.g., heat, radiation, noise, climate and weather), chemical pollutants/toxicants, and biological (bacteria, viruses, parasites, vectors). Water, sanitation and hygiene (WASH) is a well-known determinant of poor nutritional outcomes,

linked to 10 per cent of cases of undernutrition. Beyond WASH, the effects of exposure to chemicals and other biological hazards on nutrition outcomes are under-recognized. This document highlights the influence of environmental hazards on childhood undernutrition and micronutrient deficiencies, with a focus on chemical and biological exposures. It supports integrating **environmental health** through a multisectoral approach to reduce the environmental determinants of undernutrition and micronutrient deficiencies. Health care workers can help advance this integration by screening for environmental exposures and providing guidance during child health visits, educating families on reducing exposure to environmental hazards during nutritional counselling, and championing healthier environments for all children.

Introduction

Under international norms, [malnutrition](#) is a violation of children's rights (UNICEF, 2026a). The United Nations second Sustainable Development Goal ([SDG 2](#)) aims to "end hunger, achieve food security and improved nutrition, and promote sustainable agriculture by 2030." However, despite global efforts and progress, compounding drivers continue to impact the systems needed to end childhood hunger and meet the global nutrition targets. Inequities, globalization, [relentless urbanization](#) and recent and evolving crises – including climate hazards, public health crises such as the COVID-19 pandemic, and armed conflicts – undermine the quality of children's diets and care,

disproportionately affecting children in low- and middle-income countries (LMICs). Increasingly, children face a triple burden of malnutrition, with the coexistence of undernutrition (stunting, wasting, and underweight), micronutrient deficiencies ('hidden hunger'), and overweight and obesity, resulting in negative long-term outcomes for children that affect whole societies and nations. UNICEF's nutrition strategy prioritizes the prevention of all forms of child malnutrition across the life cycle and supports the early detection and treatment of life-threatening malnutrition (UNICEF, 2020). This document focuses on undernutrition and micronutrient deficiencies.





Overview of childhood undernutrition and micronutrient deficiencies

Childhood malnutrition is an imbalance (deficiency or excess) between the body's nutrient requirements and the intake of energy, macronutrients, and/or micronutrients (World Health Organization, 2026). The clinical manifestations of malnutrition include undernutrition (wasting, stunting and underweight), micronutrient deficiencies and overnutrition (overweight and obesity).

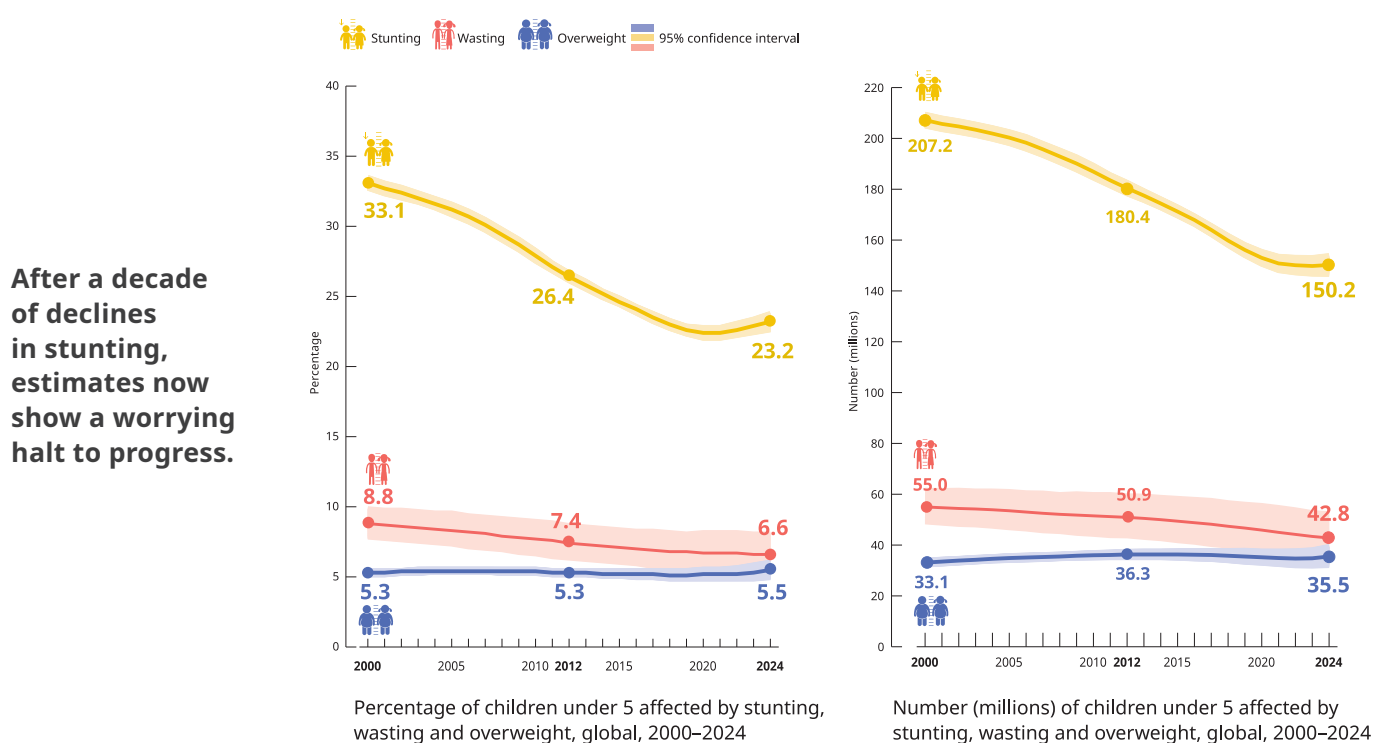
Table 1. World Health Organization (WHO) definitions for childhood undernutrition and micronutrient deficiencies

Wasting	Acute malnutrition indicated by low weight for height. It is often indicative of recent, severe weight loss, but can also persist for a long time. Worldwide, 42.8 million children under 5 have wasting.
Stunting	Chronic malnutrition is indicated by low height for age (linear growth retardation). Child's height for age exceeds two standard deviations below WHO child growth standards . Worldwide, 150.2 million children are stunted.
Underweight	Low weight for age. The underweight child may be wasted, stunted or both.
Micronutrient deficiencies (also known as 'hidden hunger')	Lack of vitamins and minerals that are essential for bodily functions, growth and development. Depending on the micronutrient, these deficiencies in children are associated with serious adverse health outcomes, including impaired physical and cognitive development, blindness, anaemia, immune deficiencies, increased susceptibility to infections and mortality, among others.

Burden of undernutrition and micronutrient deficiencies in children

Global child stunting and wasting rates have declined during the last decade, with an estimated prevalence of 23.2 per cent and 6.6 per cent respectively. Despite these advancements, according to the [Joint Malnutrition Estimates](#), only 28 per cent of the countries assessed in 2025 are on track to halve the number of children affected by stunting by 2030.

Figure 1. Global overview, prevalence and number (millions) affected



Source: UNICEF, WHO, World Bank Group Joint Malnutrition Estimates, 2025 edition.

Children under 5 years are the most vulnerable to undernutrition, particularly those aged under 2 living in LMICs (Mao et al., 2024). Undernutrition is associated with up to 45 per cent of deaths in children under 5, mainly through synergistic effects between childhood wasting and infectious diseases such as diarrhoea and pneumonia (Carvalho et al., 2025; World Health Organization, 2026). Children with concurrent wasting and stunting have the highest risk of death; and severe underweight and combined moderate wasting/stunting are associated with a mortality relative risk of 4.8 by age 24 months (Yue et al., 2022). Child growth failure is the second leading risk factor associated with health burden in children under 5 years, and it is associated with 880,000 deaths (18.8 per cent of all under 5 deaths) and an estimate of 79.4 million under 5 disability-adjusted life years (DALYs) (GBD 2023 Child Growth Failure Collaborators, 2026).

Globally, approximately 40 per cent (269 million) of children aged 6 months to 59 months have anaemia, and 372 million are deficient in at least one of three core micronutrients (iron, zinc and vitamin A). Approximately 37 per cent of pregnant women have anaemia (World Health Organization, 2025a). The most prevalent micronutrient deficiencies in children are iron, iodine, vitamin A, zinc and folate (Bailey et al., 2015). True population-based national data on micronutrient deficiencies are available from very few countries; therefore, global burden of disease estimates rely heavily on modelled data. According to estimates, in 2019, there were 435 million prevalent cases of childhood nutritional deficiencies globally, with 52 million new incident cases, 105,000 deaths, and 26 million DALYs. The burden remains disproportionately concentrated in low-sociodemographic index (SDI) and low-middle-SDI countries (Liu et al., 2024).

“Child malnutrition and climate change are two of the greatest threats facing nations today. They converge geographically, share common systemic and societal drivers, and exacerbate one another. Millions of families are struggling to meet children’s nutritional needs under the strain of climate change, while greenhouse gas emissions from food systems remain one of the main drivers of the climate crisis.”

– UNICEF (n.d.)

Source: Introduction to UNICEF’s Child Nutrition and Climate Change framework, published on their technical knowledge portal, Knowledge @ UNICEF.



Clinical consequences of childhood undernutrition and micronutrient deficiencies

Undernutrition impairs both innate and adaptive immunity, increasing vulnerability to infections and the severity of illness (Ibrahim et al., 2017; Bourke et al., 2019). Protein and micronutrient deficiencies disrupt haematopoietic and lymphoid organs and are associated with malnutrition-related changes in the gut microbiome that can further compromise immune defence mechanisms (Ibrahim et al., 2017).

Stunting and wasting are linked to impaired cognitive and motor development (Ali et al., 2025). Severe acute malnutrition is associated with significantly lower cognitive and motor scores than those of well-nourished peers. Stunting before age 2 is a significant predictor of impaired cognitive development and reduced educational attainment in later childhood and adolescence and is consistently correlated with long-term consequences for human capital, workforce productivity and wage potential, particularly in LMICs (Akseer et al., 2022; McGovern et al., 2017).

Early-life undernutrition, especially during the critical periods of in utero and early childhood growth, is associated with an increased risk of non-communicable diseases (NCDs) in adulthood, including cardiovascular disease, type 2 diabetes, and obesity (Grey et al., 2021). This risk for NCDs is magnified when children, who are chronically underweight and/or stunted early in life, later experience rapid weight gain through transition

to nutritional excess through energy-dense and/or obesogenic diets that result in central adiposity and metabolic syndrome, as nutritional deficiencies constrain metabolically critical tissues early in development (Wells et al., 2020).

The effects of childhood micronutrient deficiencies on health depend on the type of micronutrient (Table 2) and may persist into adulthood and across generations when maternal deficiencies affect fetal programming (Victora et al., 2021).



Table 2. Clinical consequences of the most common prenatal and childhood micronutrient deficiencies

Micronutrient	Clinical consequences linked to deficiency
Iron	Developmental and cognitive deficits, attention, learning and academic problems; poor pregnancy outcomes (including low birth weight, pre-eclampsia, preterm delivery and maternal mortality), and immune dysfunction
Vitamin A	Xerophthalmia (a leading cause of preventable blindness in children); poor growth and development; increased risk of death from infectious diseases, particularly diarrhoeal, respiratory and measles (Song et al., 2023)
Zinc	Impaired growth and weight gain; neuronal abnormalities; iron deficiency anaemia; compromised immunity; increased risk of pollutant hazards through altered response to oxidative stress; risk for cardiovascular diseases
Iodine	Pregnancy loss and stillbirth; congenital anomalies; congenital hypothyroidism; perinatal infant mortality; impaired mental function and cognitive disability; delayed physical development; reduced somatic growth; thyroid gland enlargement (goitre) (Zimmermann, 2012; Taylor et al., 2014; Walker et al., 2007)
Folate (vitamin B₉)	Periconceptional and prenatal folate deficiency are associated with increased risk of neural tube defects, impaired fetal growth and premature birth

The determinants of undernutrition and micronutrient deficiencies in children

The aetiology of malnutrition is multifactorial, arising from the complex interplay among individual characteristics and environmental and socioeconomic determinants, operating through diverse pathways. These determinants co-occur and interact, amplifying effects across generations. To achieve SDG 2, multiple system actions are needed to promote and support the determinants of good nutrition across the life cycle, from preconception through adolescence. The [UNICEF Conceptual Framework on Maternal and Child Nutrition \(2020\)](#) frames childhood nutrition as resulting from the interaction of preconceptual, prenatal and postnatal determinants, including diets and care as the immediate determinants. Good diets are driven by adequate food and care (feeding and dietary practices). The underlying determinants include the food, practices and services available in households and communities to support good nutrition, while enabling determinants include the resources, norms and governance that facilitate it.



The environment as a key determinant of good nutrition

Pollution is conceptualized as the “presence or introduction into the environment of substances or energy that cause adverse effects on human health, the environment or living organisms; or that exceed the quality or quantity criteria established for certain environmental media” (United Nations Children’s Fund, n.d.). Environmental degradation drives human degradation. Literature describes a ‘triple planetary crisis’ (climate change, biodiversity loss and pollution) that is affecting human health and well-being, threatening ecosystems, altering socioeconomic stability and undermining food and water safety and security. Evidence on human populations, and animal and cellular models show that pollution can drive childhood undernutrition and micronutrient deficiencies through multiple pathways, including repeated diarrhoea, helminth infection, and environmental enteric dysfunction in the case of inadequate WASH, and direct toxicity and impaired nutrient absorption and utilization, in the case of chemical and other biological hazards. Physical environmental determinants also operate upstream on the agriculture/food system itself – reducing food quantity, degrading food quality/nutrient density, and disrupting access and affordability – which increases food insecurity and, in turn, undernutrition (Figure 1).

The relationship between the environment and nutrition in children is bidirectional; healthy environments drive good nutrition, and children who are well nourished have a decreased risk of adverse outcomes from pollution. Research suggests that diets rich in anti-inflammatory bioactive food components (phytochemicals, polyphenols, and antioxidant nutrients) may modulate and reduce the toxicity and disease risks associated with certain environmental pollutants, with the most consistent evidence from studies in adults supporting whole dietary patterns rather than isolated antioxidant supplements (Hoffman & Hennig, 2017). Conversely, nutritional deficiencies can amplify vulnerability to toxicant-related health effects with data suggesting mechanisms that include enhanced absorption via shared transport pathways, impaired detoxification capacity, altered pharmacokinetics (Ngcobo, 2025), and increased susceptibility to oxidative stress and inflammation (Palmer et al., 2024). Furthermore, nutrient deficiencies and chemical pollutants can synergize to amplify adverse health outcomes (e.g., anaemia/micronutrient deficiencies and lead absorption).

Chemical exposures, undernutrition, and micronutrient deficiencies

Over 80,000 chemicals (3,000 produced in high volumes) are found in the water, food, air, soil and built environments. The same food that should nourish and provide essential nutrients for optimal health and development can also expose people to contaminants linked to undernutrition. Chemical toxicants can enter children’s diets through the food chain by three main pathways:

- 1) Contaminating water, livestock, fish and other seafood
- 2) Agricultural practices (e.g., the use of pesticides and fertilizers, polluted soil and irrigation water, and inadequate harvesting practices)
- 3) Food processing and packaging.

Quantifying the burden of childhood undernutrition and micronutrient deficiencies attributable to environmental exposures is challenging. The potential effects of most chemicals already introduced in our environment on childhood nutrition have not been studied and the evidence on combined environmental exposures and child undernutrition is sparse, with much mechanistic work done on animal and in vitro models. Nevertheless, some chemicals and groups of chemicals have been studied and identified as health and nutrition risks, especially for children. Data have shown that some of these exposures can adversely impact several distinct outcomes: growth, nutrition, neurodevelopment and risk for later cardiometabolic disease, among others (Potiris et al., 2025; Romano et al., 2026).

Metals

Prenatal and early-life exposure to metals (e.g., lead, cadmium, mercury, arsenic) has been associated with impaired growth, stunting and anaemia (Bede-Ojimadu et al., 2025). Metals are linked to disruption of nutrient absorption and metabolism; direct toxicity to organ systems involved in growth and haematopoiesis; synergism with undernutrition (e.g., dietary deficiencies of calcium, zinc and iron enhance neurodevelopmental impacts of lead); and increased metabolic demands. Cadmium, lead and mercury are common air pollutants (World Health Organization, n.d.). The coexistence of high blood lead or cadmium levels and nutrient deficiencies has been linked to subclinical damage to haematopoietic, renal and nervous tissues (Bharatraj et al., 2018). On the other hand, nutritional deficiencies can alter the absorption, distribution and retention of various metals. For example, iron deficiency, with or without anaemia, enhances lead absorption

through a common intestinal transporter. This is concerning because lead is responsible for a significant global disease burden and, even at low exposure levels, can cause lifelong and irreversible adverse neurodevelopmental outcomes (Disalvo et al., 2025). Lead remains a global public health threat, with one third (800 million) of children having blood lead levels at or above 5 µg/dL (UNICEF and Pure Earth, 2020).



Air pollutants

Air pollutants – specifically particulate matter with a diameter of 2.5 micrometres or less (PM_{2.5}), and to a lesser degree, nitrogen dioxide (NO₂), sulfur dioxide (SO₂), and carbon monoxide (CO) – are associated with impaired fetal and early childhood growth, stunting and anaemia (Brumberg et al., 2021; Khezri et al., 2025; Pun et al., 2021; Pambianchi et al., 2022; Jin et al., 2025). Proposed mechanisms include systemic inflammation supported by human meta-analytic data showing elevated CRP¹ and TNF-α² with gaseous pollutant exposure (Xu et al., 2022), and alterations in nutrient metabolism, based largely on in vitro and animal evidence (Sangphech et al., 2026). **Household air pollution (HAP)** is associated with several adverse health outcomes. In 2020, approximately one third of the global population used solid fuels (e.g., firewood and charcoal) for cooking (Woolley et al., 2022; GBD, 2025; Stoner et al., 2021). The observational association between HAP from incomplete combustion and childhood stunting is well documented and consistent across settings. However, the causal pathway appears complex, and clean fuel interventions alone have not yet demonstrated reductions in stunting in randomized controlled trials (Checkley et al., 2024), suggesting that HAP reduction must be part of broader integrated multisectoral approaches that invest in community infrastructure. Children whose parents/caretakers smoke **tobacco** face higher dose-dependent risks of growth stunting (World

Health Organization, 2025b). Maternal smoking and exposure to second-hand smoke are strongly linked to preterm birth, congenital anomalies, restricted fetal growth, low birth weight and an increased risk for chronic diseases later in life, especially if the child has growth stunted by age two (Al-Mughathwai et al., 2025). Quitting smoking during pregnancy improves child growth outcomes. Second-hand tobacco smoke exposure after birth is associated with neurodevelopmental and behavioural problems and increases the risk and severity of respiratory infections and developmental problems; and has also been associated with reduced height for age and stunting in some populations (World Health Organization, 2025b).



¹ C-reactive protein is released by the liver into the blood in response to an inflammation from an infection, injury, or long-term disease
² Tumour necrosis factor is a chemical messenger produced by the immune system that induces inflammation

Pesticides

Children can be exposed to pesticides prenatally (through the placenta); through contaminated food and water; through chemicals stored in the home or dumped in public areas; if they live in or near agricultural areas; and occupationally. Globally, 84 million children are engaged in agricultural work, with pesticide levels consistently higher than in the general population (ILO & UNICEF, 2026b). A higher risk of exposure is also observed among families living near agricultural land and among those with improper pesticide management. Despite the Codex Alimentarius, which sets maximum pesticide residue limits adopted by most countries, children are exposed to pesticides through toxic residues in the food they eat (UNICEF, 2018). Overreliance on pesticides in agriculture to reduce hunger undermines several SDGs. Pesticides are a critical threat to children's health, affecting their developing immune, endocrine and nervous systems. Exposure to pesticide residues among women and children in farm settings has been linked to low vitamin and mineral levels, acetylcholine inhibition, and altered oxidative stress parameters (Medithi et al., 2022); as well as to oxidative stress, oxidative DNA damage, and inflammation (Aguilar-Bañuelos et al., 2025). Separate studies found lower haemoglobin and lymphocyte counts and elevated inflammation indices in the highest-exposure

community and lower levels of vitamin D. However, most evidence comes from occupational/agricultural exposure settings and cross-sectional studies, and animal models. Direct evidence linking dietary pesticide residue levels or household use at typical consumer exposure to clinically significant micronutrient deficiencies in children remains limited. Direct evidence linking pesticide exposure to stunting or wasting is limited. On the other hand, malnourished children (particularly those with oedematous undernutrition) may be more vulnerable to pesticide toxicity in part due to severe depletion of antioxidant nutrients.



Endocrine-disrupting chemicals

Endocrine-disrupting chemicals (EDCs) are synthetic and natural substances that interfere with any aspect of hormone function (synthesis, transport, metabolism and excretion), leading to a broad spectrum of adverse health effects, particularly on growth, pubertal development and metabolism. Among other sources, children are commonly exposed to mixtures of EDCs through water and food contamination. For example, per- and polyfluoroalkyl substances (PFAS), phthalates and bisphenol A are commonly found in food contact materials (e.g., packaging, storage) and are highly prevalent in ultra-processed foods. Urinary phthalate metabolites have been inversely associated with serum folate concentrations in US children aged 6–18 years, but the evidence is cross-sectional and no link to clinical folate deficiency has been established (He et al., 2024). Even though EDCs have not been a focus of undernutrition literature, exposure to these chemicals during pregnancy has been associated

with reduced birth weight and impaired fetal/infant growth (Svensson et al., 2021). Direct evidence linking exposure to EDCs and undernutrition using WHO-defined stunting and wasting endpoints is limited.



Biological exposures, undernutrition and micronutrient deficiencies

WASH

Inadequate **WASH** results in 1.4 million people dying yearly (395,000 children under the age of 5, in 2019), mostly in LMICs (Wolf et al., 2023).

WASH-related biological exposures can lead to acute and chronic diarrhoeal diseases, enteric fever and parasitic infections. Subclinical enteric pathogens and enteroaggregative *Escherichia coli* infections are more prevalent among children with malnutrition (Lima et al., 2017). Parasitic infections (malaria and helminths) are consistently associated with anaemia and stunting (Hhera et al., 2025).

Environmental enteric dysfunction (EED) – a subclinical inflammatory disorder of the small intestine caused by chronic exposure to faecal contaminated water, food, soil and fomites and recurrent subclinical enteric infections from multiple transmission routes – is characterized by increased intestinal permeability, intestinal and systemic inflammation, malabsorption, alterations in growth-related hormones, and dysbiosis of the intestinal microbiota (Owino et al., 2016; Lauer et al., 2020). EED has been hypothesized as a major contributor to childhood undernutrition, including stunting, and is highly prevalent in environments with heavy faecal contamination and poor sanitation

(Gizaw et al., 2022). Low-cost household-level WASH interventions tested in three landmark trials did not improve child linear growth, but comprehensive or intensive WASH approaches – such as community-level sanitation, sewerage systems and safely managed water services, particularly when integrated with nutrition – have shown benefits by reducing the risk of diarrhoeal disease and child mortality (Gizaw et al., 2022).



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Mycotoxins

Exposure to naturally occurring **mycotoxins** in the food chain is prevalent, and infants are at higher risk (Etzet, 2014). Mycotoxins are chemically stable and can survive food processing (World Health Organization, 2023b). Early-life exposure is associated with low birth weight, stunting, and immune suppression (Watson et al., 2017; Vilcins et al., 2018). A systematic review found that uncertainty remains as to whether dietary mycotoxin exposure directly affects child growth and immunity, grading the overall evidence as very low quality due to inconsistencies across studies, while not ruling out an association with aflatoxin and fumonisin (Hadush et al., 2020); a subsequent meta-analysis of prospective cohorts found aflatoxin B₁ exposure associated with underweight [Odds Ratio (OR) 1.20 (95% CI 1.03–1.40)] and stunting [OR 1.21 (95% CI 1.11–1.33)] (Nejad et al., 2023). Mycotoxins have shown dose-dependent effects on inflammation and immunosuppression (Sun et al., 2022). Aflatoxin interferes with intestinal absorption of vitamins A, C and E, impairs zinc absorption, and contributes to

anaemia via haemolysis, suppressed erythropoiesis and inhibited iron absorption. Data suggest that aflatoxin exposure is associated with anaemia and vitamin A deficiency across human studies (Mshanga et al., 2025). Deoxynivalenol is a foodborne mycotoxin that has been linked to malabsorption of nutrients, appetite suppression, and protein synthesis inhibition (Gonya et al., 2024). Mycotoxin contamination affects both quantity and quality of food, contributing to food insecurity, a determinant of undernutrition and micronutrient deficiencies, particularly anaemia.



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Table 3. Examples of biological and chemical environmental determinants of childhood undernutrition and micronutrient deficiencies

Form of malnutrition	Biological exposures	Chemical exposures
Wasting	Diarrhoeal disease, infections (helminths, tuberculosis, human immunodeficiency virus (HIV), malaria), EED, aflatoxin/ mycotoxin, poor WASH	Metals (lead, cadmium, arsenic, mercury), air pollutants (particulate matter PM _{2.5}), nitrogen dioxide (NO ₂), HAP (biomass, solid fuel), tobacco; linked to decreased weight for age, low birth weight: pesticides (pyrethroids, organophosphates), volatile organic compounds and EDCs
Stunting	Recurrent diarrhoeal disease and infection with specific enteropathogens (cryptosporidium, enteropathogenic and enterotoxigenic <i>E. coli</i>); parasitic infections; chronic faecal-oral pathogen exposure (EED); aflatoxin exposure; inadequate WASH exposures	Metals (lead, cadmium, arsenic); air pollution (PM _{2.5} , HAP, tobacco smoke)
Micronutrient deficiencies		
Iron	Helminth infection (chronic blood loss), malaria, chronic faecal-oral pathogen exposure (EED), bacterial enteropathogens, <i>Helicobacter pylori</i> infection and aflatoxins	Lead, cadmium, arsenic, and ambient air particulate matter (PM)
Vitamin A	Diarrhoeal disease, measles, chronic faecal-oral pathogen exposure (EED), infections (respiratory, helminths, HIV, hepatitis B), aflatoxins	Linked through experimental and wildlife models: persistent organic pollutants [polychlorinated biphenyls (PCBs), organochlorines and pesticides (DDT/DDE)], cadmium, lead
Zinc	Enteric pathogens, contaminated water, and infection with helminths	Increased global CO ₂ atmospheric levels (Myers et al., 2015) decrease the zinc concentration in staple cereal crops (e.g., wheat and rice); cadmium and arsenic, with additional evidence for lead (limited human paediatric data)
Iodine	Chronic faecal-oral pathogen exposure (EED)	Dietary goitrogens (naturally occurring food compounds, such as cyanogenic glucosides, glucosinolates, flavonoids) and chemicals that potentiate existing deficiency by impairing iodine utilization: perchlorate, brominated and chlorinated compounds, thiocyanate, nitrate (e.g., contaminated well water), triclosan, dioxins/organochlorine pesticides, tobacco smoke and industrial exhaust
Folate (vitamin B₉)	Chronic faecal-oral pathogen exposure (EED), infection (helminths, malaria, <i>Giardia lamblia</i>), tropical sprue	Supported by paediatric-specific data: lead and phthalates; supported through adult cohorts, pregnancy cohorts, animal models, or in vitro work: perchlorate, perfluoroalkyl substances, polycyclic aromatic hydrocarbons, phenols, parabens, tobacco smoke (thiocyanate), polybrominated diphenyl ethers, indoor air pollution

Box 1. Climate change: A physical environmental determinant of childhood nutrition

Approximately 85 per cent of the food consumed in Puerto Rico is imported, and food insecurity has been estimated at 30.3 per cent (Dewey et al., 2024).

In 2017, Category 4 Hurricane María struck the territory, barely two weeks after Hurricane Irma, another major hurricane. These compounding climate disasters caused widespread devastation. Food insecurity was one of the most significant and long-term consequences, as 80 per cent of crop value was destroyed, food supply chains were interrupted, and some areas remained without power for 11 months (the longest power outage in US history). This convergence of factors led to prolonged food shortages, diminished food safety and subsequent dependence on nutrient-poor, non-fresh food alternatives (e.g., ultra-processed foods). Data show that climate change is enabling precipitation and temperature extremes that can lead to flooding, droughts, wildfires and more intense and destructive hurricanes, increasing the risk of undernutrition and micronutrient

deficiencies through several pathways, including the impacts on food and water systems, decreased food nutritional density, and the risk of foodborne diseases (Agostoni et al., 2023; Duchenne-Moutien & Neetoo, 2021; McMahon & Gray, 2021; Fanzo et al., 2025; Zajac et al., 2025; Romanello et al., 2024; Watts et al., 2018). Furthermore, climate change is an established modifier of exposure to toxicants (e.g., air pollution, metals, mycotoxins) linked to an increased risk of poor nutrition outcomes by altering the environmental distribution and toxicity of chemical pollutants (Balbus et al., 2013; Noyes et al., 2009). An estimated 850 million children live where at least four climate and environmental shocks overlap (Perera & Nadeau, 2022). Agrifood systems account for about one third of total global greenhouse gas emissions (Food and Agriculture Organization, 2025). The Puerto Rico case is illustrative of the well-documented compounding global climate-nutrition dynamic. Climate adaptation and disaster preparedness are essential for promoting nutrition and safeguarding environmental health.



Addressing environmental hazards as a driver of childhood malnutrition

Success in ending undernutrition and micronutrient deficiencies hinges on simultaneous action across levels, not single-sector fixes. It requires high-level, sustained political commitment, collaborative evidence-based governance, engagement of local governments, private sector, and communities, and monitoring of the progress with accountability features. Addressing environmental hazards is essential to reduce childhood malnutrition, while addressing malnutrition can reduce vulnerability to environmental hazards.

UNICEF provides a comprehensive framework to optimize maternal and child feeding, diet, and nutrition, and detailed [guidance](#) on healthier environments is available. The following is a list of priorities across clinical practice, programmes, research, and policy that can address environmental determinants of undernutrition and micronutrient deficiencies and improve [children's environmental health](#) across all life stages. These recommendations can be adapted to specific contexts and communities.



Box 2. Breastfeeding as a driver of children's nutrition and environmental health

Children in the village of Djingring Kass in Chad are benefiting from a package of community health, sanitation and civil registration services that support exclusive breastfeeding – giving them a better start in life. Denemadji, a 33-year-old mother of five, was a pioneer and is now a leader in exclusive breastfeeding in her village. Her mission began with her decision not to give her youngest child water or any other food until he was six months old, despite the widespread belief that infants need water in their diets from birth. The support for social and behavioural change regarding exclusive breastfeeding was key to Denemadji's success. Exclusive breastfeeding from birth through six

months provides a low-carbon, zero-waste, clean and safe food source that avoids the greenhouse gas emissions, land conversion, and water and biodiversity loss linked with the production of commercial infant formula while preventing exposure to some contaminants and reducing the risk of underweight, wasting and stunting. Breast milk's anti-inflammatory, immunomodulatory, and antioxidant properties may mitigate some of the effects of air pollution on health (Machado et al., 2025).

To learn more about Denemadji's story, visit: <https://www.unicef.org/chad/stories/successful-choice-exclusive-breastfeeding>

Target areas for programme and policy change: A six-pronged approach to environmental determinants of childhood undernutrition and micronutrient deficiencies

Monitoring and reporting	
Environmental monitoring	- Establish and support exposure assessment through environmental sampling, monitoring and reporting infrastructures to accurately measure pollutants in the air, water, food and consumer products, particularly in LMICs. - Monitor food preparation, conservation, packaging and processing methods to guide interventions to decrease exposures to chemicals in the food system. - Monitor household cooking fuel type and facilitate monitoring of PM2.5 and carbon monoxide (CO) levels in homes using biomass fuels.
Disease surveillance	- Increase clinicians' and community health workers' capacity for anthropometric and nutritional surveillance. - Promote evidence-based, accessible and minimally-invasive biomonitoring to assess environmental exposure health risks (e.g., blood lead, urinary cadmium/arsenic, aflatoxin biomarkers). - Incentivize the development of field-deployable diagnostics for environmental enteric dysfunction to facilitate surveillance and management.
Prevention and control	
Regulation of chemicals linked to undernutrition and micronutrient deficiencies	- Establish and maintain an international clearinghouse database of chemicals (Grandjean & Landrigan, 2014). - Set enforceable child-responsive standards for pollutants and contaminants. - Promote the WHO Framework Convention on Tobacco Control measures. - Address and remediate metal contamination of drinking water. - Strengthen regulatory frameworks and enforcement of maximum residue levels for infant and young child foods, consistent with the European Union standards. - Reform the Codex Alimentarius by redesigning its risk assessment framework to account for children's unique vulnerabilities and harms to human health and the environment beyond acute effects. - Scale proven nutrition interventions that also buffer environmental insult.
Primary care and nutrition across the lifespan	- Ensure universal access to primary care and adequate nutrition. - Integrate environmental history-taking (e.g., WHO Green Page) into primary care.
Management, treatment and remediation	
Environmental risk reduction	Design and subsidize nutrition-sensitive programmes that explicitly incorporate environmental hazard reduction – clean cooking, safe water, food safety and mycotoxin control, metal source abatement – alongside nutrition-specific delivery.
Training the health workforce	- Build local community health workers' and clinicians' competence in environmental medicine through training, resources and continued education (e.g., Environment, Climate Change, One Health and Migration training modules for health care workers). - Increase knowledge of UNICEF nutrition strategies among health care workers. - Train nutrition specialists on the environmental determinants of nutrition and health.

Public awareness and behaviour-change

Community education and engagement

- Foster education and behaviour modification programmes through trained community and health professionals and public information campaigns on nutrition, WASH, food safety, preparation and feeding practices, and on [evidence-based strategies](#) to reduce environmental risks.
- Promote partnerships between health care and education sectors with local farmers to source nutritious and diverse foods while promoting the local economy and decreasing the carbon footprint.

Environmental legislation and policy

Enhanced environmental and nutrition policy

- Strengthen and enforce policies to protect water and food sources from pollution.
- Promote policies that adhere to the [WHO Guidance on Ending the Inappropriate Promotion of Foods for Infants and Young Children](#), the [WHO Code](#), and the [WHO Regional Office for Europe Nutrient Profile Model](#) as the recommended benchmarks to improve national food standards.
- Strengthen food additives regulatory frameworks to protect children.
- Implement workplace policies that prohibit child labour.
- Include medical and allied health professional organizations in environmental and nutrition policy decision processes and in the implementation of national nutrition practice standards.

Global and regional actions

Improved global and regional coordination

- Integrate environmental exposure reduction into nutrition programmes, and nutrition into environmental programmes.
- Create and enforce international standards for environmental pollutants.
- Promote safe management of hazardous waste to prevent human exposure and contamination.
- Adopt the [Food and Agriculture Organization of the United Nations](#) agricultural and food system policies to promote 1) co-benefits for nutrition and the environment (e.g., healthy diets from sustainable food systems through [agroecological practices](#), biocontrol and post-harvest practices to reduce aflatoxin contamination, sanitation practices); 2) less toxic food processing (e.g., biosourced plastics and biopolymers); and 3) [climate-smart](#) agricultural technologies and practices for sustainable, shock-resilient, equitable food system models.
- Promote efficient, fair-trade policies and agricultural subsidies, credit and extension services that incentivize the production of nutrient-dense, climate-resistant, biofortified crops rich in essential micronutrients and with low environmental impact; and disincentivize ultra-processed food production.
- Invest in research work that studies effects of mixtures of exposures; measures stunting, wasting, and micronutrient endpoints (as defined by WHO) alongside environmental exposure biomarkers; and assesses which environmental interventions lead to measurable improvement in nutrition outcomes.



Conclusion and call to action

Child undernutrition and micronutrient deficiencies remain a critical global challenge with high disease and mortality burden. Determinants of stunting, wasting and micronutrient deficiencies are complex, and dietary intervention alone cannot prevent or solve poor nutritional outcomes. Environmental determinants are not a peripheral consideration in child nutrition – they operate on the immediate, underlying and enabling determinants simultaneously. Beyond WASH, exposure to chemical and biological environmental hazards is associated with childhood stunting, wasting and micronutrient deficiencies, particularly anaemia. The most consistent evidence exists for metals, air pollutants, aflatoxins and infectious agents. Undernutrition and micronutrient deficiencies increase children's health risks linked to pollutants, while toxicant exposure can lead to poor nutrition and growth outcomes.

Nutrition programming that ignores the environment is likely to underperform, and environmental interventions that overlook nutrition miss vital opportunities. The evidence base is sufficient to act now on exposures with consistent associations, while investing in research on individual chemicals and mixtures, mechanisms, co-exposure effects and environmental interventions on nutrition outcomes. Nutrition programming that ignores the environment is likely to underperform. Health care workers are key to integrating these priorities into child health services.



Collaborative, multilevel action to address nutrition and the environment simultaneously is needed to safeguard children's health.

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