PERSISTENT ORGANIC POLLUTANTS AND CHILDREN'S HEALTH

unicef for every child

Executive summary

Persistent organic pollutants (POPs) are a class of toxic chemicals that persist in the environment for long periods and can remain present over extended time frames. Some examples include dichloro-diphenyl-trichloroethane (DDT), polychlorinated biphenyls (PCBs) and dioxins. Ingestion is the principal route by which children are exposed to POPs. These carbon-based chemicals become increasingly concentrated as they move up the food chain, thus polluting food sources and harming wildlife and humans. Because these chemicals are resistant to degradation and can be transported through air and water, they can travel long distances and become widely distributed across the globe, including in regions where they have never been used. POPs may also be transported through migratory species, further contributing to their global spread. These chemicals are associated with serious adverse health effects in humans and other living organisms.

POPs are found in a variety of industrial and household sources. They may also be produced unintentionally during manufacturing processes and waste incineration. Humans and animals around the world are exposed to POPs at low levels over extended periods. Children are especially vulnerable to POPs on account of their unique physiological characteristics, rapid development and distinct exposure patterns.

Children have been shown to suffer adverse effects from higher levels of exposure. Even at low levels, POPs can disrupt endocrine and immune systems, impair neurodevelopment, increase cancer risk and affect reproductive health. There also is increasing concern that long-term exposure to low levels of POPs may contribute to the overall burden of disease, including an increased incidence of breast and other cancers, learning and behavioural disabilities, other neurodevelopmental problems and reproductive effects such as decreased sperm quality and count.

Although some POPs have been banned or are regulated under the Stockholm Convention on Persistent Organic Pollutants, they continue to affect communities worldwide, particularly in developing countries, where capacities for monitoring, regulatory enforcement, remediation and public awareness may be limited.

Because the effects of early-life exposure often manifest later in life, they may go unnoticed for many years. Minimizing children's exposure to POPs is therefore essential for promoting lifelong health and well-being. Reducing POP levels in the environments where children live, learn and play is not only a public health imperative but also a matter of environmental justice and child rights.

Healthy Environments for Healthy Children

Introduction

Persistent organic pollutants (POPs) are a class of toxic chemicals that persist in the environment for long periods and can remain present over extended time frames. POPs bioaccumulate and biomagnify, meaning that they become increasingly concentrated as they move up the food chain, thus polluting and exposing wildlife and humans. Because these carbon-based chemicals are resistant to degradation and can be transported through air and water, they can travel long distances and become widely distributed across the globe, including in regions where they have never been used. POPs may also be transported through migratory species, further contributing to their global spread. These chemicals are associated with serious adverse health effects in humans and other living organisms.

POPs are found in a variety of industrial and household sources. A complete list of POPs listed under the Stockholm Convention is available on the Convention's website and POPs are classified under Annex A ('Elimination'), Annex B ('Restriction') and Annex C ('Unintentional production') (Secretariat of the Stockholm Convention, 2026a). Although POPs have been banned or are regulated under the Stockholm Convention, they continue to affect communities worldwide, particularly in developing countries, where capacities for monitoring, regulatory enforcement, remediation and public awareness may be limited (United Nations Environment Programme, 2024).

Prevalence of exposure

Globally, children are exposed to POPs through air, water, food, household dust and consumer products. In many regions, especially those with weak chemicals regulation, exposure occurs through multiple pathways, including legacy contamination, industrial emissions, agricultural pesticide residues and informal waste processing. The open burning of plastics, electronic waste (e-waste) and other household waste leads to the release of dioxins, furans and other toxic substances into the atmosphere, where they subsequently contaminate soil, water and food sources.

Children living near informal e-waste recycling sites, industrial zones or agricultural areas where banned pesticides are still used face a disproportionately high level of exposure (World Health Organization, 2021). Yet, POPs are not only a local problem: Given their persistence and ability to travel long distances across borders, they are detected in remote regions and among populations that are not in close proximity to emission sources. Studies have documented the presence of POPs in maternal blood, umbilical cord blood, breast milk and infant tissues – indicating that exposure can begin at the earliest stages of life.

Initially, 12 POPs were listed under the Stockholm Convention and recognized as causing adverse effects on human health and the environment (Table 1).

The Stockholm Convention on Persistent Organic Pollutants later extended the list to include many other chemicals (Secretariat of the Stockholm Convention, 2026a).

Table 1. The first 12 POPs listed under the Stockholm Convention

Category	POP type
Pesticides	Aldrin, chlordane, dichloro-diphenyl-trichloroethane (DDT), dieldrin, endrin, heptachlor, hexachlorobenzene, mirex, toxaphene
Industrial chemicals	Hexachlorobenzene, polychlorinated biphenyls (PCBs)
Unintentionally produced POPs	Hexachlorobenzene, polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDDs/PCDFs), PCBs

Source: Adapted from Secretariat of the Stockholm Convention (2026b).

Sources and routes of exposure

Children encounter POPs through multiple environmental and behavioural pathways:

- **Ingestion or dietary intake** is the primary route of exposure, especially through fatty animal products, dairy and fish/seafood from contaminated waterbodies. In many rural or subsistence communities, food chains remain contaminated by long-banned chemicals such as dichloro-diphenyl-trichloroethane (DDT) or PCBs. POPs can be deposited in waterways, be taken up by invertebrates and accumulate in fish and animals eaten by people. Food could be contaminated with some pesticide residues containing POPs.
- **Prenatal exposure** occurs when POPs cross the placenta and accumulate in fetal tissues during critical windows of development (Needham et al., 2011). Even low levels of prenatal exposure can interfere with hormone signalling and organ formation.
- **Breastfeeding**, while critical for infant health and development, can be a route of POP transmission in settings with high maternal body burdens, as many POPs are lipophilic and concentrate in breast-milk fat. The presence of dioxins and polychlorinated biphenyls (PCBs) in human breast milk is not an indication for avoiding breastfeeding.
- **Direct dermal contact** with persistent soil residues of banned pesticides such as DDT or dieldrin can lead to absorption through skin.
- **Indoor environments** can expose children to flame retardants (e.g., polybrominated diphenyl ethers (PBDEs)) from ageing furniture, electronics or insulation materials. These compounds accumulate in household dust, and young children are particularly at risk of exposure owing to their frequent hand-to-mouth behaviour. Building materials, furniture, textiles, carpets and curtains, packing materials, electric and electronic appliances, and toys could contain PBDEs or other POPs, and any of these items could be mouthed by children.
- **Inhalation** of fumes from the heating or burning of items containing PCBs and/or PBDEs, such as the heating of transformers and/or open waste burning, is another route of exposure.
- **Community-level sources** include waste incineration, informal e-waste recycling, pesticide spraying and industrial emissions. These processes release POPs into surrounding environments where children live, learn and play.

Children's unique vulnerability

Children are especially vulnerable to POPs on account of their unique physiological characteristics, rapid development, and distinct exposure patterns (Etzel, 2020) and behaviours. Their metabolic systems are still maturing, and their brains and endocrine and immune systems are undergoing rapid development. Their higher respiratory and metabolic rates relative to body size mean that they take in more pollutants per kilogram of body weight than adults. Moreover, children's behaviours such as crawling, mouthing objects and playing on the ground result in greater contact with contaminated surfaces and materials (Etzel, 2020).

Even at low levels, POPs can disrupt endocrine and immune systems, impair neurodevelopment, increase cancer risk and affect reproductive health (World Health Organization, 2010). Exposure to POPs during early life can interfere with normal developmental programming, leading to adverse health outcomes that may not become apparent until adolescence or adulthood. Because these impacts often emerge gradually, early prevention and exposure reduction are key public health priorities.

Case studies:
Contamination of cooking oil in Asia

Exposure to high levels of POPs can result in serious adverse health effects. Mass poisonings have occurred in Asia when cooking oil was inadvertently mixed with heat-degraded PCBs, resulting in heavy contamination with polychlorinated dibenzofurans (PCDFs), which are partially oxidized PCBs.

In 1968, about 1,200 people in the Kyushu province of **Japan** ingested contaminated oil over 20 to 90 days. These people eventually developed Yusho (oil disease). Exposed people developed reproductive dysfunction, severe chloracne, hyperpigmentation, eye discharge, headaches, vomiting, fever, visual disturbances and respiratory problems. Thirteen of the women exposed were pregnant at the time; one of their children was stillborn and pigmented ('cola coloured'). Some of the live-born children were small, hyperbilirubinemic and pigmented, and had conjunctival swelling and dilatation of the sebaceous glands of the eyelid. Up to nine years later, the children still showed apathy, lethargy and soft neurological signs.

An outbreak of disease resulting from contaminated rice oil occurred in **Taiwan** in 1979. In total, 117 children born during or after this incident were exposed to PCBs and PCDFs through their mothers' body burdens. They were examined in 1985 (and subsequently) and found to have ectodermal defects such as excess pigmentation, dental caries, poor nail formation and short stature. They continued to have persistent behavioral abnormalities and cognitive impairment. The developmental delays were as severe in children born up to six years after exposure as in those born within a year or two of exposure in 1979. Older children (who were not exposed) resembled control children.

Source: Adapted from World Health Organization (2010).

Chemical plant explosion in Italy

A chemical plant explosion in Seveso, **Italy**, in 1976 resulted in the release of large quantities of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), a congener of dioxin. The highest recorded serum levels of TCDD in humans occurred in children in the most heavily exposed areas. Children living in the area near the explosion developed chloracne, most prominent on areas of skin that were not protected by clothing; tests showed that some also developed abnormal liver function.

Source: Adapted from World Health Organization (2010).

Chronic low-dose exposure

POPs are toxic at high levels, as illustrated by mass poisoning incidents. Exposure to high levels of POPs may cause adverse health effects including death, disease and birth defects among humans and animals. What remains less clear is whether significant adverse health effects can occur from background levels or chronic low-dose exposure. Of most concern are the possible effects of low levels of exposure on the developing fetus, infants and children.

There is also increasing concern that chronic exposure to low levels of POPs may contribute to public health trends including increased cancer incidence (breast cancer and others), learning disabilities and other neurodevelopmental problems, and reproductive problems such as decreased sperm count and quality, male genital anomalies and endocrine and immune diseases.

Health impacts

Neurodevelopment, cognitive function and behaviour

Numerous studies have linked early-life exposure to PCBs (Fábelová et al., 2025), PBDEs (Herbstman & Mall, 2014) and dioxins (Yim et al., 2022) with deficits in cognitive, behavioural and motor development. Prenatal exposure to POPs can disrupt brain development, leading to lower IQ scores, language delays, attention deficits and poor executive functioning. For example, cohort studies in the Faroe Islands and the Kingdom of the Netherlands have shown that the maternal consumption of PCB-contaminated fish during pregnancy correlates with impaired executive functioning, processing speed, visual recognition and memory in children (Boucher et al., 2009).

Endocrine disruption and growth

POPs interfere with hormone signalling pathways that regulate growth, development and metabolism. The disruption of thyroid hormones, which are critical for brain development, has been observed in infants with prenatal exposure to flame retardants (Yeshoua et al., 2024). Several studies have also documented altered pubertal timing and abnormal levels of sex hormones in children exposed to certain POPs (Windham et al., 2015). These disruptions can affect stature, bone maturation and reproductive system development.

Immune system suppression

POP exposure has been shown to impair the immune system, reducing the ability of children to respond to common pathogens and vaccines. For instance, children exposed to high levels of perfluorinated compounds (often called “forever chemicals”) have been shown to have reduced antibody responses to diphtheria and tetanus vaccines (Grandjean et al., 2012). This immune suppression may leave children more vulnerable to infections and diminish the effectiveness of public health immunization efforts.

Cancer risk

Several POPs are classified by the International Agency for Research on Cancer as Group 1 carcinogens (meaning that they are known to cause cancer in humans), including dioxins and PCBs, and Group 2 carcinogens (classified as probable carcinogens), such as DDT (International Agency for Research on Cancer, 1997, 2015, 2016). Early-life exposure may increase lifetime risk of testicular cancer, leukaemia, lymphomas and breast cancer. The association of POPs with these cancers is of particular concern given the long latency period of many environmentally driven cancers.

Reproductive and birth outcomes

Prenatal exposure to POPs has been linked to adverse birth outcomes including low birth weight, preterm delivery and congenital abnormalities such as undescended testes (cryptorchidism) and hypospadias. These outcomes are believed to be mediated by the disruption of reproductive hormones during fetal development and may have implications for fertility later in life. Exposure to low levels of POPs during pregnancy also may impair fetal growth (Oudir et al., 2020) and be a precursor to obesity in later childhood (Stratakis et al., 2022).

> Several scientists state that data are not yet conclusive about the health effects of background levels of POPs in children: 'Data exist that support harmful effects of POPs. Examples include associations with altered neurodevelopment, thyroid economy, and oestrogen and immune function. Yet for all such associations, human evidence regarding causality at low doses remains equivocal at best, owing to inconsistent results, inadequate replication, and other questions. In the laboratory, the neurotoxic, immunotoxic, and hormonal activity of many of these compounds has been established, and the issue has been whether effects occur at background exposure levels, not whether the compounds are toxic' (Longnecker, 2001a). Other scientists concur that more research is needed before definitive conclusions can be reached (Damstra, 2002a, Kimbrough, 2001). An approach consistent with the precautionary principle, however, acknowledges that exhaustive proof of a substance's toxicity should not be required due to the gravity of the short- and long-term toxic effects of low dose exposure. Lack of scientific certainty does not exclude a precautionary approach to managing chemicals in order to protect health and the environment '...when there are threats of serious or irreversible damage' (UNCED Rio Declaration, 1992).
>
> – World Health Organization (2010)

Intervention and mitigation strategies

Policies and legislation/actions by policymakers

POPs are pervasive in the environment as a result of manufacturing and widespread dispersal over decades. The main way to reduce exposure is through regulation – banning their manufacture, commercial sale and use through laws and enforcement (United Nations Environment Programme, 2020), for example as follows:

- Strengthen national compliance with the Stockholm Convention on Persistent Organic Pollutants, including by phasing out remaining legacy POPs and destroying existing stockpiles.
- Regulate the production, use, import and disposal of products containing flame retardants, plasticizers and other persistent chemicals in household goods.
- Improve formal waste management systems and prevent informal e-waste processing that leads to POP release.
- Expand national monitoring programmes to detect POP residues in food, breast milk, drinking water and ambient air. The United Nations Environment Programme and Global Environment Facility Global Monitoring Plan projects monitor the presence of POPs in the environment and humans (United Nations Environment Programme, 2026).
- Integrate POP risk assessments into housing, zoning, agricultural and land use policies to avoid placing vulnerable populations near contaminated environments.

Actions by caregivers (parents, teachers) and the health sector

- Promote exclusive breastfeeding for the first six months of life as recommended by health authorities, while also taking steps to reduce maternal exposure during pregnancy and breastfeeding.
- Introduce nutritionally adequate and safe complementary (solid) foods at 6 months of age together with continued breastfeeding up to at least 2 years of age.
- Reduce consumption of fatty meats, dairy and contaminated fish, especially from regions with known contamination.
- Avoid the use of older foam furniture, electronics or insulation materials that may contain banned flame retardants in homes, schools and health-care facilities.
- Clean homes and schools regularly with wet mops and damp cloths to reduce indoor dust, which may be contaminated with POPs.

Public engagement and awareness

- Launch culturally tailored communication campaigns to inform families about sources of POP exposure and safe alternatives.
- Engage community leaders, youth groups, health professionals and educators in promoting chemical safety and environmental health literacy.
- Encourage the meaningful participation of children and their families in environmental decision-making and right-to-know initiatives.

Conclusion

POPs represent an invisible but serious threat to children's health. These chemicals can disrupt critical biological systems and compromise the foundations for healthy development. Children around the world continue to be exposed to POPs through air, food, household dust and consumer products – often without their knowledge or the ability to avoid them. Such exposure disproportionately affects low-income and marginalized communities and contributes to global health inequities. Urgent action is needed to reduce POP exposure from the prenatal stage to adolescence, ensure safe environments and uphold every child's right to health and well-being. Coordinated policy, public education and environmental remediation can protect current and future generations from this preventable threat.

Community- and school-level strategies

- Prevent the open burning of rubbish and e-waste near residential or school zones.
- Ensure that new schools and childcare facilities are not sited on contaminated land or near high-risk industrial zones.
- Require the removal of all PCB-containing fluorescent light ballasts.
- Incorporate environmental health education into school curricula and community health programmes to increase awareness of chemical risks.

Monitoring and evaluation

- Develop national biomonitoring frameworks to track levels of POPs in children and high-risk populations over time.
- Invest in longitudinal studies to better understand the long-term health effects of POP exposure and evaluate the effectiveness of mitigation policies.

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