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Invisible Harm: The Neurological and Developmental Impact of Environmental Pollution on Children

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Abstract: ***Environmental pollution poses a profound threat to child health, with neurological development being particularly vulnerable. This review synthesises evidence linking exposure to key pollutants-including particulate matter, heavy metals, pesticides, and organic compounds-with adverse outcomes such as cognitive impairment, attention-deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), anxiety, and psychotic symptoms. Mechanistic insights reveal that oxidative stress, neuroinflammation, neurotransmitter disruption, and endocrine interference underpin these effects, especially during critical developmental windows such as the prenatal period and the first two years of life. Beyond the brain, pollution also compromises physical growth, contributing to stunting, low birth weight, and impaired growth trajectories. Disproportionate exposure among socioeconomically disadvantaged and marginalized populations highlights the environmental justice dimension of this issue. Together, the evidence underscores the urgent need for strengthened environmental policies, equitable public health interventions, and clinical awareness to safeguard children's neurological and developmental potential.***

Keywords: child health, environmental pollution, neurodevelopment, developmental disorders, environmental justice

1. Introduction

Environmental pollution has emerged as one of the most pressing public health challenges of the 21st century. Among its wide-ranging consequences, the impact on children's neurological development is especially concerning. Children are uniquely susceptible to pollutants due to their higher intake of air, food, and water relative to body weight, immature detoxification systems, and underdeveloped blood–brain barriers (1). These vulnerabilities, combined with critical periods of rapid brain growth, amplify the risk of long-lasting harm.

A growing body of evidence links exposure to criteria ambient air pollutants such as particulate matter ($PM_{2.5}$ and PM_{10}), oxides of nitrogen (NO_x), and ozone with a spectrum of neurodevelopmental and behavioural disorders, including attention-deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), anxiety, depression, and psychotic symptoms (1,3). Additionally, heavy metals like lead and mercury, as well as pesticides and industrial chemicals, have been implicated in disrupting normal neurodevelopment through mechanisms involving oxidative stress, neuroinflammation, and interference with neurotransmitter systems (2,33–35). Mechanistic studies consistently provide biological plausibility, highlighting the roles of oxidative damage, inflammation, and endocrine disruption in mediating these effects (40).

Importantly, the consequences of pollution are not limited to the nervous system. Substantial evidence also demonstrates adverse effects on physical growth, including low birth weight, stunting, and impaired growth velocity (41–45). These findings underscore pollution's dual impact on both brain and body development, reinforcing its importance as a determinant of lifelong health.

Despite the expanding literature, the developmental consequences of pollution remain under-recognized in clinical and policy settings. While there are several individual research studies on these aspects, comprehensive reviews and aggregated conclusions derived out of these studies are limited reported. This review addresses this gap by comprehensively examining the evidence on pollution and child neurodevelopment, with particular focus on:

1) The vulnerabilities of the developing brain,
2) Behavioural and neuropsychiatric outcomes,
3) Pollutant-specific mechanisms of neurotoxicity,
4) Impacts on physical growth,
5) The role of disparities and environmental justice, and
6) Public health implications and strategies for intervention.

By integrating clinical, mechanistic, and epidemiological findings, this review aims to highlight pollution not merely as an environmental issue, but as a central determinant of child health and development.

2. Methodology

This review was conducted using a structured narrative approach. Relevant literature was identified through searches of PubMed, Scopus, and Web of Science databases up to August 2025, using combinations of keywords including *"child health," "environmental pollution," "neurodevelopment," "developmental disorders," "environmental justice."* Reference lists of key articles and meta-analyses were also hand-searched to identify additional studies.

Priority was given to:
1) Systematic reviews and meta-analyses that provided pooled evidence (4,6,8,24,25,37,41,42,43).

2) Large cohort and longitudinal studies examining pollutant exposure and developmental outcomes (5,11,12,26,36,45).
3) Mechanistic and experimental studies offering biological plausibility for observed associations (7,10,13,15,17,27,33,40).

Studies were included if they focused on children or prenatal exposures, assessed either neurological/behavioural outcomes or physical growth parameters, and were published in peer-reviewed journals. Evidence was synthesized thematically under four domains: (1) vulnerability of the developing child, (2) neurodevelopmental and behavioural effects, (3) pollutant-specific mechanisms of neurotoxicity, and (4) impact on physical growth and disparities.

3. Vulnerability of the Developing Child

Children are uniquely vulnerable to the harmful effects of environmental pollutants due to a combination of physiological, developmental, and behavioural factors. These vulnerabilities significantly amplify the neurotoxic and developmental consequences of exposure, particularly during early life stages.

1) Physiological Susceptibility

Several physiological characteristics render children more susceptible to pollution-related harm:

- **Immature Blood–Brain Barrier**: In early life, the blood–brain barrier (BBB) is underdeveloped, allowing easier penetration of neurotoxic substances into the central nervous system. This increases the risk of brain damage from pollutants such as particulate matter and heavy metals (1).
- **Higher Intake per Body Weight**: Children consume more air, food, and water relative to their body weight compared to adults. This results in proportionally greater exposure to airborne and dietary pollutants for the same environmental concentrations(1).
- **Immature Detoxification Systems**: The enzymatic and renal systems responsible for detoxifying and excreting harmful chemicals are not fully developed in young children. This leads to slower clearance of toxins and prolonged systemic exposure (1).
- **Behavioural Risk Factors**: In addition to biological susceptibility, children often engage in behaviours that elevate exposure risk, such as crawling on the ground, hand-to-mouth activity, and playing outdoors for extended periods in potentially contaminated environments(1).

2) Critical Developmental Windows

Timing of exposure plays a crucial role in determining the severity of adverse outcomes:

- **Prenatal Period**: The intrauterine environment is highly sensitive to toxic exposures. Many pollutants, including fine particulate matter, heavy metals, and endocrine disruptors, can cross the placenta and interfere with neurogenesis and organ development. The prenatal period is thus considered the most critical window of vulnerability (3).
- **First 1,000 Days of Life**: This window, encompassing pregnancy and the first two years after birth, is marked by rapid brain and body growth. Disruptions during this phase can have irreversible effects on cognition, behaviour, and physical development. Immature detoxification systems and higher metabolic rates further compound susceptibility during this period (3).

4. Neurodevelopmental Effects of Pollution

1) Behavioural and Neuropsychiatric Outcomes

Exposure to environmental pollutants has been consistently associated with a range of adverse behavioural and neuropsychiatric outcomes in children. These include attention-deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), mood disorders, and psychotic experiences. These disorders often emerge during critical developmental windows and may persist into adolescence and adulthood.

a) **Attention-Deficit Hyperactivity Disorder (ADHD):** Postnatal exposure to particulate matter ($PM_{2.5}$ and PM_{10}) and nitrogen dioxide (NO_2) is strongly associated with increased risk of ADHD. A systematic review and meta-analysis of 25 studies demonstrated that each 10 µg/m³ increase in $PM_{2.5}$ correlates with odds ratios ranging from 1.4 to 1.87 for ADHD. The most critical period of exposure appears to be between ages one and three, with a dose-response effect observed—$PM_{2.5}$ levels exceeding 50 µg/m³ significantly escalate ADHD risk (4). In the ECO PATHWAYS multicohort study involving 1,967 children, a 2-unit increase in NO_2 exposure at ages 2–4 years was associated with a 3.59-point increase in Child Behaviour Checklist (CBCL) total problem scores and a 2.63-point reduction in IQ scores (5).
b) **Autism Spectrum Disorder (ASD):** Prenatal exposure to $PM_{2.5}$ and ozone (O_3) has been linked to elevated ASD risk. Fine particulate matter can cross the placental barrier and disrupt foetal neurodevelopment in genetically predisposed individuals (6). Animal studies in mice have demonstrated that exposure to ultrafine ambient particles during critical neurodevelopmental windows leads to structural brain changes characteristic of ASD. These include corpus callosum reduction, white matter abnormalities, and sex-specific vulnerabilities—particularly among males (7).
c) **Anxiety, Depression, and Psychotic Symptoms:** Children exposed to high levels of air pollution exhibit a 73% higher likelihood of experiencing anxiety and depression. Structural and functional changes have been documented in brain regions such as the hippocampus, amygdala, and prefrontal cortex (8). Furthermore, exposure to the high NO_2 levels is associated with a 1.7-fold increased risk of developing psychotic symptoms, even after adjusting for confounders such as socioeconomic status and urbanicity. This risk is notably higher in children living in urban compared to rural environments (9).

2) Pollutant-Specific Neurotoxicity

A wide range of pollutants have been identified as neurotoxic, each with distinct mechanisms and clinical outcomes. These include heavy metals, air pollutants, organic compounds, and pesticides, all of which interfere with brain development through oxidative stress, neurotransmitter disruption, and direct structural damage.

Heavy Metals

a) **Lead:** Lead is one of the most extensively studied environmental neurotoxins. Even low-level exposure, below the traditional threshold of 10 µg/dL, has been linked to adverse behavioural and cognitive outcomes. Lead crosses the immature blood–brain barrier by mimicking calcium and accumulates in the brain, where it interferes with neurotransmitters such as dopamine, gamma-aminobutyric acid (GABA), and acetylcholine. It induces oxidative stress and impairs calcium-dependent signalling, particularly in the prefrontal cortex, hippocampus, and cerebellum (10). Clinically, lead exposure results in reduced IQ, attention deficits, emotional reactivity, impulsivity, and antisocial behaviour. In a cohort of 1,341 preschool children in China, each 1 µg/dL increase in blood lead was associated with significant increases in emotional reactivity and anxiety scores (11). Another study found that blood lead levels of just 1–5 µg/dL were linked to substantial behavioural disturbances (12).

b) **Mercury:** Methylmercury is the most neurotoxic form and poses the greatest risk during the foetal period. It readily crosses the placenta and accumulates in foetal tissues, including the brain, where it persists and causes long-term neurotoxicity. Mercury disrupts motor development, coordination, attention, and memory (13,14). It is more strongly linked to ASD than ADHD. Populations with high fish consumption report cognitive impairments in approximately 1.5–1.7 per 1,000 children due to mercury exposure.

c) **Cadmium:** Cadmium crosses both the placenta and the blood–brain barrier, inducing early neurodevelopmental damage. Its mechanisms include oxidative stress, mitochondrial dysfunction, and disruption of neurotransmitter systems (15–18). Clinically, cadmium exposure is associated with learning and memory deficits and behavioural disorders such as ADHD, particularly in boys (15).

d) **Manganese:** Manganese affects the basal ganglia, especially the dopaminergic pathways linked to executive function. Its toxicity follows a U-shaped curve, where both deficiency and excess are harmful. Children are more vulnerable due to higher absorption and immature excretion mechanisms. Exposure leads to reduced IQ, impaired motor coordination, and decreased visual attention. Hair manganese levels serve as a reliable biomarker in exposed children aged 6 to 12 years (19).

e) **Arsenic:** Arsenic targets the pituitary gland and interferes with brain development by inducing oxidative stress and altering neurotransmitter systems, including monoaminergic and glucocorticoid pathways (20). It disrupts neurogenesis and is associated with deficits in intelligence and memory. Systematic reviews covering over 20 studies confirm significant cognitive decline in arsenic-exposed children (21,22).

Criteria Air Pollutants

Criteria air pollutants, particularly fine and ultrafine particulate matter and nitrogen-based gases, have demonstrated potent neurotoxic effects in children. These pollutants are capable of crossing the immature blood–brain barrier or triggering systemic inflammation that affects the central nervous system.

a) **$PM_{2.5}$ (Particulate Matter ≤2.5 µm):** PM2.5 is small enough to penetrate deep into the lungs, enter the bloodstream, and cross the blood–brain barrier. Exposure during early childhood and prenatal periods has been linked to a spectrum of neurodevelopmental disorders, including ADHD and ASD (4,6,23). A meta-analysis involving 4,860 children showed that each 1 µg/m³ increase in $PM_{2.5}$ exposure was associated with a 0.27-point reduction in full-scale IQ, with performance IQ being disproportionately affected (0.39-point reduction) (24). $PM_{2.5}$ exposure is also linked to structural brain changes on neuroimaging, including white matter loss and connectivity deficits (25).

b) **PM_{10} (Particulate Matter ≤10 µm):** Though less deeply penetrating than $PM_{2.5}$, PM_{10} still poses significant risks during gestation and early life. PM_{10} exposure during pregnancy has been associated with low birth weight and subsequent cognitive impairments (26). It also contributes to ADHD risk, especially with chronic postnatal exposure above 50 µg/m³ (4).

c) **Nitrogen Dioxide (NO_2):** NO_2 is a key traffic-related pollutant that has been linked to behavioural and cognitive disturbances. In the study (ECO PATHWAYS), a 2-unit increase in NO_2 exposure at ages 2–4 resulted in higher behavioural problem scores and a measurable decline in IQ (5). High NO_2 exposure is also associated with a 1.7-fold increase in the risk of psychotic symptoms in adolescents, even after controlling for confounding factors (9). Neuroimaging findings correlate NO_2 exposure with structural changes in the hippocampus and prefrontal cortex (8).

d) **Ozone (O_3):** Prenatal exposure to ozone has been implicated in autism spectrum disorders. O_3 is capable of generating oxidative stress that disrupts foetal brain development, especially when exposure coincides with critical neurodevelopmental windows (6). Although the evidence base is smaller compared to PM and NO_2, the association with ASD is consistent across several cohorts.

Organic Pollutants

Organic pollutants, including polycyclic aromatic hydrocarbons (PAHs), benzene, and toluene, are widely distributed environmental toxins. Due to their lipophilicity and small molecular size, they readily cross the immature blood–brain barrier in children and disrupt neurodevelopment through oxidative damage, neurotransmitter imbalance, and epigenetic alterations.

a) **Polycyclic Aromatic Hydrocarbons (PAHs):** PAHs are produced by incomplete combustion of organic material and are commonly found in polluted urban air. These compounds cross the blood–brain barrier and interfere with brain development by generating oxidative stress, reducing brain-derived neurotrophic factor (BDNF), and forming DNA adducts that impair gene regulation (27). Clinical consequences include attention-deficit hyperactivity disorder, cognitive impairments, and delayed memory development (27–29). Studies have also demonstrated associations with altered DNA methylation and disrupted cholinergic neurotransmission, which may contribute to long-term behavioural abnormalities.

b) **Benzene:** Benzene exposure disproportionately affects children due to their immature metabolic detoxification systems and greater pollutant intake per body weight. Benzene induces oxidative stress and alters neurotransmitter activity, particularly in the central nervous system(30). Clinically, benzene exposure has been associated with symptoms such as unsteady gait, headaches, and memory problems in children, as observed in a study involving 157 exposed individuals (30).

c) **Toluene:** Toluene is a volatile organic solvent found in paints, adhesives, and fuels. Its lipophilic nature allows it to cross the blood–brain barrier, where it accumulates in myelin-rich areas. Toluene induces white matter degeneration and oxidative injury, particularly affecting the frontal and parietal lobes (31,32). The most severe clinical outcome is dementia, although affected children may also present with cognitive deficits, motor impairments, and behavioural changes. Neuroimaging studies reveal reduced brain volumes in chronically exposed individuals (31).

Pesticides

Pesticides are among the most potent environmental neurotoxins, with widespread use in agriculture and household settings. Children are particularly susceptible to pesticide-related neurotoxicity due to their developing brains, immature detoxification pathways, and higher intake-to-body-weight ratios. The most implicated pesticide classes in neurodevelopmental disorders are organophosphates, neonicotinoids, and organochlorines.

a) **Organophosphates (OPs):** Organophosphates exert neurotoxicity primarily by inhibiting acetylcholinesterase, leading to excessive accumulation of acetylcholine and cholinergic overstimulation. This is followed by receptor downregulation and disruption of normal neurotransmission (33–35). Children exposed to OPs exhibit memory deficits (especially noticeable by age 7), behavioural problems such as hyperactivity and inattention (noted in toddlers), and motor impairments (present from the neonatal stage). Chlorpyrifos, the most studied OP, has been associated with cortical surface thinning and altered brain morphology (36). A systematic review of 27 studies found that 26 reported significant adverse neurodevelopmental outcomes associated with OP exposure (37).

b) **Neonicotinoids:** Neonicotinoids act as agonists at acetylcholine receptors, mimicking nicotine and disrupting synaptic signalling. They have been detected in cerebrospinal fluid, amniotic fluid, and breast milk, indicating significant perinatal exposure(38). Animal studies reveal that exposure to neonicotinoids such as acetamiprid and imidacloprid causes brain tissue shrinkage, reduced auditory startle reflex, diminished motor activity, and learning deficits. Neuroanatomical changes are prominent in regions like the corpus callosum and caudate-putamen—areas implicated in ADHD. The European Food Safety Authority has recommended setting exposure limits for these compounds (38).

c) **Organochlorines:** Despite being banned in many countries, organochlorines like DDT (dichloro diphenyl trichloroethane), and DDE (dichloro diphenyl dichloroethylene) persist in the environment due to their high bio accumulative potential. These compounds interfere with brain development through poorly understood mechanisms but have been linked to significant neurodevelopmental disturbances (35,39). Organochlorine exposure is associated with reduced psychomotor function, particularly in girls, and increased risk of autism spectrum disorders. These effects may persist long after exposure has ceased.

5. Mechanisms of Neurotoxicity

Environmental pollutants disrupt neurodevelopment through multiple interrelated mechanisms, many of which target the immature and rapidly evolving nervous system in children. These mechanisms vary by pollutant class but converge on key pathological pathways that impair brain structure, function, and connectivity.

a) **Oxidative Stress:** Oxidative stress is a central mechanism by which pollutants such as heavy metals, particulate matter, PAHs, and pesticides cause neuronal injury. Reactive oxygen species (ROS) generated by these toxins damage cell membranes, proteins, and DNA, impairing neuronal function and development. For example, $PM_{2.5}$, lead, and mercury increase oxidative stress in the brain, leading to neurodegeneration and impaired cognition (40).

b) **Neuroinflammation:** Exposure to pollutants like nitrogen oxides and ultrafine particles activates microglia and astrocytes, initiating inflammatory cascades within the central nervous system. These immune responses release cytokines that interfere with synaptogenesis and myelination. Both direct infiltration of particles across the blood–brain barrier and systemic inflammation contribute to this process(40).

c) **Neurotransmitter Disruption**: Many pollutants interfere with neurotransmitter pathways. Lead affects dopamine, GABA, and acetylcholine signalling (10); organophosphates disrupt acetylcholine metabolism by inhibiting acetylcholinesterase (33,34,37); and cadmium interferes with calcium signalling and synaptic transmission (17,18). These disruptions are implicated in behavioural abnormalities, motor dysfunction, and cognitive impairment.

d) **Epigenetic Modifications:** Pollutants can induce long-lasting changes in gene expression without altering the DNA sequence. PAHs, for example, alter DNA methylation patterns and reduce brain-derived neurotrophic factor (BDNF) levels, both critical for neurodevelopment and synaptic plasticity (27). Epigenetic changes may underlie delayed or progressive neurological effects even after exposure has ceased.

e) **Hormonal Dysregulation:** Endocrine-disrupting pollutants such as BPA (Bisphenol A), PFAS (Per- and polyfluoroalkyl substances), and certain pesticides alter hormone synthesis, metabolism, and receptor function. These disruptions affect not only physical growth but also neurological development, especially thyroid hormone pathways, which are essential for brain maturation (35).

6. Impact on Physical Growth and Development

While neurological outcomes are a major focus of pollution-related health effects, substantial evidence also links environmental pollutants to impaired physical growth in children. Growth disruptions include stunting, wasting, low birth weight, and reduced growth velocity, especially when exposure occurs during prenatal and early postnatal periods.

Growth Parameters Affected

a) **Stunting and Wasting:** Exposure to ambient and household air pollution is strongly associated with linear growth restriction. Meta-analyses of 45 studies across 29 countries report a 13% increased risk of stunting from ambient air pollution and up to 90% increased risk from household air pollution (41). In a Chinese cohort of 2,759 children, worsening air quality was significantly linked to stunting, underweight, and wasting, with effects persisting up to six years [23](42). A study from India involving over 218,000 children found that a 100 $\mu g/m^3$ increase in $PM_{2.5}$ during the month of birth corresponded to a 0.05 standard deviation height reduction, equating to a 0.24 cm height deficit at age five (43).

b) **Low Birth Weight (LBW):** Prenatal exposure to particulate matter increases the risk of LBW. A meta-analysis of 3 million births found that PM_{10} and $PM_{2.5}$ exposure were associated with odds ratios of 1.03 and 1.10 for LBW, respectively (26). Studies in China and Sweden have also identified sulphur dioxide (SO_2) and carbon monoxide (CO) as contributors to reduced birth weight (44,45).

c) **Reduced Growth Velocity:** Delayed growth velocity has been observed in areas with high pollution levels. For example, exposure to SO_2 in Hong Kong led to reduced height at age 13, although catch-up growth occurred by age 15—suggesting that while growth trajectories may be altered, final height may normalize in some cases (46). However, pollutants generally hinder catch-up growth, unlike nutritional deficits, due to the chronic and persistent nature of exposure (47).

Specific Pollutants and Effects

a) **Criteria Air Pollutants:** $PM_{2.5}$, PM_{10}, and SO_2 have all been implicated in reduced fetal growth, low birth weight, and long-term deficits in linear growth (41). These pollutants exert their effects via placental dysfunction, systemic inflammation, and repeated respiratory infections that impair nutrition and metabolism.

b) **Heavy Metals:** Lead disrupts bone formation and mineral metabolism, leading to reduced height and delayed skeletal maturation (48,49). Mercury exposure during development is also linked to physical growth impairments, especially depending on timing and dose (50). Manganese has shown a consistent negative association with postnatal growth in all reviewed studies (51). Cadmium impairs kidney and intestinal function and causes bone demineralization, particularly in children, increasing fracture risk (49).

c) **Endocrine-Disrupting Chemicals:** PFAS, phthalates, and bisphenol A (BPA) interfere with hormonal signalling, particularly thyroid hormones, insulin, and sex steroids. Early-life PFAS exposure has been linked to lower birth weight, altered BMI, and long-term growth abnormalities (52,53). BPA disrupts reproductive and thyroid hormones, with more severe effects observed in girls, whereas phthalates have been more detrimental to boys' reproductive development.

d) **Pesticides:** Pesticides impair nutrient absorption and disrupt gut microbiota composition. They can induce environmental enteric dysfunction—a condition of increased intestinal permeability, impaired nutrient uptake, and chronic inflammation. This leads to growth retardation, particularly in children exposed in early life (35,54). Household pesticide exposure also contributes to repeated infections, which increase metabolic demands and further hinder growth (55).

7. Disparities and Environmental Justice

Environmental pollution does not affect all children equally. Disparities in exposure and outcomes are strongly influenced by socioeconomic status, race, geography, and systemic inequities. Children from low-income families and minority communities are disproportionately exposed to environmental hazards and are less likely to benefit from protective public health infrastructure.

1) **Higher Exposure Among Marginalized Populations:** More than 300 million children live in areas where air pollution exceeds World Health Organization (WHO) limits by at least six times. In regions like East Asia and the Pacific, over 100 children under the age of five die each day due to pollution-related causes

2) **Proximity to Toxic Environments:** Schools and residential areas serving low-income and minority populations are more likely to be located near industrial zones, highways, and toxic waste sites. This spatial clustering is often a consequence of discriminatory urban planning, zoning laws, and underinvestment in infrastructure.

3) **Differential Susceptibility and Outcomes:** Beyond exposure, certain populations exhibit greater biological vulnerability. For instance, PFAS exposure has been associated with elevated blood pressure and growth issues in adolescents, with more pronounced effects seen in boys and underprivileged. This intersection of systemic racism and environmental exposure amplifies the risk and severity of health outcomes.

4) **Compounded Effects of Poverty and Pollution:** Children living in poverty are more likely to suffer from poor nutrition, limited access to healthcare, and overcrowded living conditions, which can intensify the biological effects of pollution. The pollution-infection-malnutrition cycle, particularly in regions relying on solid fuels, exemplifies how environmental and social determinants interact to hinder growth and development(55).

Addressing these disparities requires not just environmental regulation but also policies that promote environmental justice—ensuring that no population bears a disproportionate burden of pollution and that all children have access to clean, safe environments.

8. Public Health Implications

The extensive evidence linking environmental pollution to both neurodevelopmental and physical health outcomes in children underscores the urgent need for coordinated public health action. The developing brain and body are highly sensitive to pollutants, and early exposure can lead to lifelong impairments. As such, a multifaceted public health approach is essential.

1) **Importance of Early Detection and Surveillance:** Given the silent and cumulative nature of pollutant exposure, routine screening for neurodevelopmental delays, behavioural disorders, and growth impairments should be integrated into paediatric healthcare. Special attention is warranted for populations living in high-exposure zones, including urban centres and industrial areas.
2) **Policy Action for Emission Reduction:** Evidence from multiple cohort and meta-analytic studies supports the need for stricter air quality standards, even in regions where pollutant levels currently fall within existing legal limits. Neurodevelopmental harm has been observed at exposure levels below current thresholds, especially for $PM_{2.5}$ and lead. Policy interventions must prioritize the regulation of vehicular emissions, industrial pollutants, and toxic chemicals including pesticides, heavy metals, and endocrine disruptors (35).
3) **Strengthening Paediatric Healthcare and Environmental Literacy:** Paediatricians and primary care providers must be equipped with training to recognize pollution-related illnesses and counsel families on exposure reduction. Public health campaigns should increase environmental literacy among caregivers, especially in high-risk communities. Awareness of critical windows—such as pregnancy and the first two years of life—can empower families to adopt protective behaviours during key developmental phases (3).
4) **Equity-Focused Environmental Interventions:** Addressing environmental injustice requires targeted interventions in under-resourced communities. Investments in clean energy, safer housing, and urban greening can reduce exposure disparities. Furthermore, schools and childcare centres should be prioritized for indoor air filtration upgrades and pollution control policies, particularly in marginalized neighbourhoods.

Pollution's impact on children is not merely a health issue but a determinant of long-term educational, economic, and social outcomes. Protecting children from environmental toxins is an essential public health imperative that must be addressed with urgency and equity at the centre of all interventions.

9. Limitations

This review has several limitations that should be acknowledged. First, although efforts were made to capture a wide range of literature, it is possible that some relevant studies were missed, particularly unpublished or non-English reports, which may introduce selection bias. Second, the included studies were heterogeneous in terms of exposure assessment, outcome measurement, and study design, making direct comparisons challenging. Most of the evidence is derived from observational and epidemiological studies, which can demonstrate associations but not definitive causality. Additionally, many studies rely on proxy measures of exposure, such as ambient monitoring or biomarker levels, which may not fully reflect individual exposure variability. Publication bias may also have influenced the evidence base, as studies showing significant associations are more likely to be published than null findings. Finally, long-term prospective data on the developmental effects of pollution in children remain limited, particularly in low- and middle-income countries, restricting the generalizability of findings.

10. Conclusion

Environmental pollution represents a profound and pervasive threat to child health, with far-reaching consequences on neurological development, behaviour, cognition, and physical growth. Children's unique physiological vulnerability—characterized by immature detoxification systems, underdeveloped protective barriers, and rapid developmental processes—makes them exceptionally sensitive to even low levels of pollutant exposure.

The evidence clearly demonstrates strong associations between various environmental toxins—including particulate matter, heavy metals, organic compounds, and pesticides—and a broad spectrum of neurodevelopmental and growth-related abnormalities. These include ADHD, autism spectrum disorder, anxiety, depression, low birth weight, stunting, and delayed motor and cognitive milestones. Crucially, many of these effects arise during critical developmental windows, such as the prenatal period and the first 1,000 days of life, when damage may be irreversible.

Mechanistic studies reinforce the biological plausibility of these outcomes, implicating oxidative stress, neuroinflammation, hormonal disruption, and epigenetic modifications as key pathways of toxicity. Furthermore, environmental injustice magnifies these risks among socioeconomically disadvantaged and marginalized children, highlighting the intersection of pollution with systemic inequities.

Urgent action is needed at both policy and clinical levels. Regulatory frameworks must be updated to reflect the latest evidence on safe exposure limits, while paediatric care systems should incorporate environmental risk assessment and early intervention strategies. Ultimately, safeguarding children from environmental pollutants is not only essential for individual well-being but also for securing a healthier, more equitable future for all.

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