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Research Article

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Prenatal Exposure to Airborne and Indoor Pollutants and the Risk of Childhood Neurodevelopmental Disorders

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Abstract

A substantial proportion of Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) cases remain without clear etiology, with environmental factors increasingly implicated alongside genetic predispositions. This review synthesizes evidence linking prenatal exposure to outdoor air pollutants and indoor environmental contaminants with childhood behavioral and cognitive impairments. We evaluated peer-reviewed studies examining prenatal exposure to major neurotoxicants—including traffic-related air pollution (TRAP), ultrafine particles, polycyclic aromatic hydrocarbons (PAHs), persistent organic pollutants (POPs), heavy metals, pesticides, bisphenol-A, and phthalates—and their associations with ASD, ADHD, depression, and cognitive deficits. Evidence was integrated by pollutant type, emphasizing mechanistic pathways. Across diverse geographical settings, consistent associations emerged between prenatal exposure to TRAP, PAHs, polychlorinated biphenyls (PCBs), pesticides, and mercury with adverse neurodevelopmental outcomes. Mechanistic studies highlight oxidative stress, neuroinflammation, endocrine disruption, and epigenetic modifications of the placenta as central mediators. Notably, risks were observed even at pollutant concentrations below current World Health Organization (WHO) guidelines, underscoring the heightened vulnerability of the developing brain. Prenatal exposure to a spectrum of environmental pollutants is a plausible and preventable contributor to childhood neurodevelopmental disorders. Enhanced public health measures, stricter environmental regulations, and targeted maternal protection strategies are critical to mitigate these risks.

Keywords: Prenatal Exposure, Air Pollution, Indoor Pollutants, Environmental Pollutants, Neurodevelopmental Disorders, Behavioral Disorders, Child Development

1. Introduction

Air pollution shapes human health before life even begins [1-5]. Few environmental threats rival the global health burden of air pollution, a complex mixture of particulate matter, gases, trace metals, and adsorbed organic contaminants that is especially concentrated in urban environments [1-3]. The World Health Organization (WHO) lists it among the most serious health hazards of the modern era [4]. Exposure accumulates across the lifespan, starting in utero, making it a critical public health concern [5-7]. During pregnancy, both chemical and physical pollutants can alter placental function through epigenomic modifications, with lasting effects on fetal programming and development [8,9]. The placenta, essential for nutrient transfer, oxygen exchange, and immune regulation, is highly sensitive to such environmental insults [8,10]. Prenatal exposure is linked to impaired conceptus development and outcomes including embryonic mortality, fetal loss, intrauterine growth restriction, preterm birth, congenital anomalies, childhood diseases, neuropsychological deficits, altered

timing of sexual maturation, and some adult cancers [8,9,11-14]. While genetics account for only 30–40% of neurodevelopmental disorders, environmental exposures—often interacting with inherited susceptibilities and social influences—play a substantial causal role [15,16].

The health risks of ambient air pollution are now recognized in both developed and developing countries [17]. Traffic-derived pollutants such as carbon monoxide (CO), nitrogen dioxide (NO_2), and lead are established neurotoxicants, acting through pathways involving oxidative stress and neuroinflammation [1,5,18,19]. Prenatal exposure has been associated with impaired cognitive abilities, attention deficits, lower mental development indices and intelligence quotient (IQ) scores, heightened anxiety and depression symptoms, reduced nonverbal reasoning, and delayed psychomotor milestones [5,20-27]. These effects underscore the susceptibility of the developing central nervous system (CNS) and implicate air pollution as a plausible risk factor for autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD) [1,28,29]. While genetic and environmental determinants jointly contribute to these conditions, environmental factors alone are estimated to account for 10–40% of ADHD risk and approximately 40% of autism risk [31,32]. The biological impacts of air pollution align with hypothesized mechanisms underlying ASD and ADHD, and multiple lines of evidence—from epidemiology, animal models, and experimental studies—link prenatal or postnatal exposure to deficits in cognition, psychomotor ability, and specific behavioral outcomes [3,6,19,33-42].

Evidence from animal and human studies suggests that these neurodevelopmental effects often begin in utero with animal models showing that high prenatal exposure can induce neurotoxicity [5,11,43-45]. Despite growing recognition of these risks, no investigation has examined the combined influence of prenatal air pollution, meteorological variation, and genome-wide placental DNA methylation [8]. Addressing this knowledge gap is essential for elucidating how environmental exposures shape neurodevelopment and contribute to long-term behavioral and cognitive trajectories [46]. This study links prenatal exposure to outdoor and indoor air pollution with childhood behavioral and cognitive deficits, and explores potential mechanistic pathways (**Figure 1 and Figure 2**).

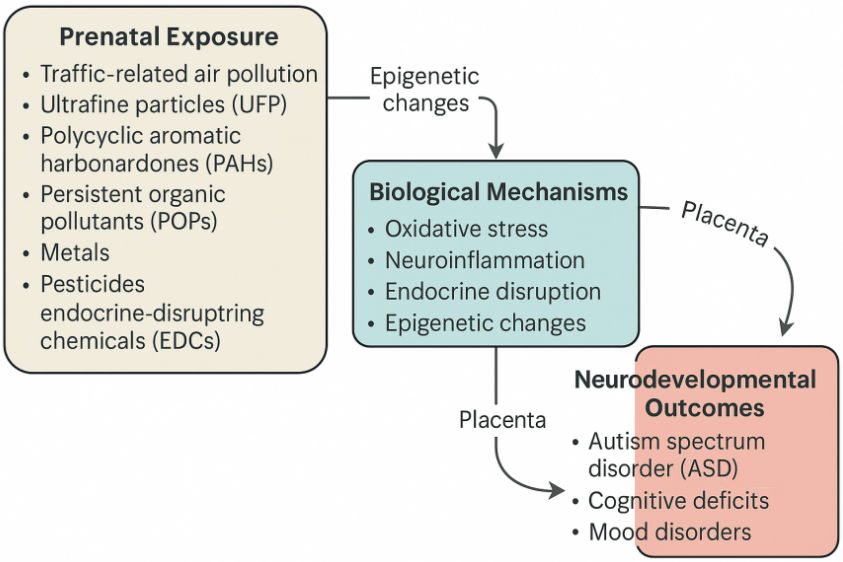

Figure 1: Conceptual Framework of Prenatal Pollutant Exposure and Neurodevelopment

Maternal exposure to airborne and indoor environmental pollutants during pregnancy can trigger multiple biological pathways—oxidative stress, inflammation, endocrine disruption, and epigenetic modification—that interfere with fetal brain development, increasing the risk of neurodevelopmental disorders such as ASD and ADHD. *Created by authors using original artwork.*

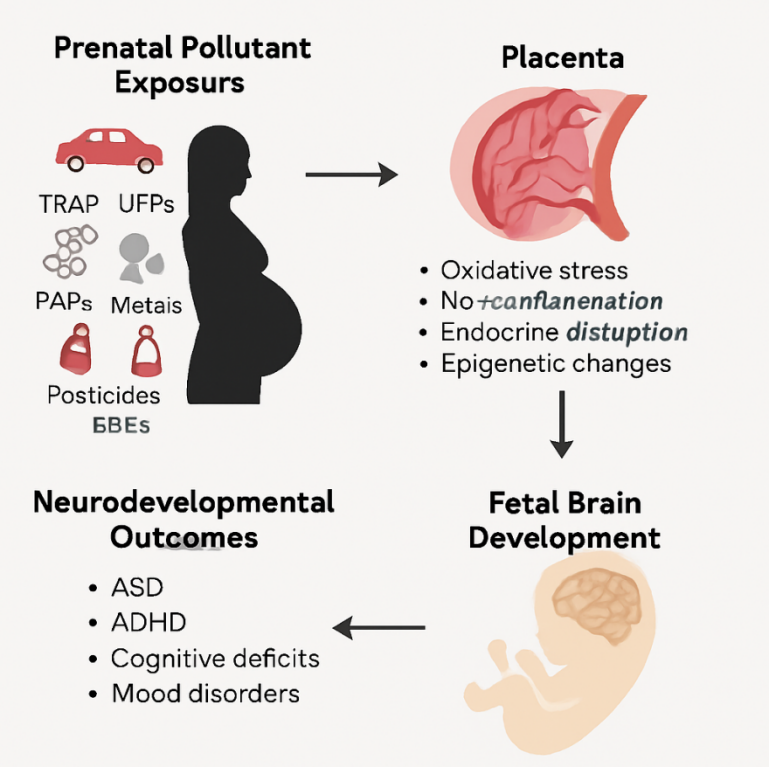

Figure 2: Mechanistic Pathways Linking Prenatal Pollutant Exposure to Neurodevelopmental Outcomes

Overview of cellular and molecular mechanisms, including oxidative stress, neuroinflammation, endocrine disruption, and epigenetic changes, illustrating how pollutant-induced perturbations affect neuronal development and synaptic connectivity, leading to adverse neurobehavioral outcomes. *Created by authors using original artwork.*

2. Autism Spectrum Disorder (ASD)

Epidemiological research has linked high levels of air pollution exposure to adverse neurobehavioral outcomes **(Table 1)**. Air pollution has emerged as a potential environmental risk factor for ASD [1]. ASD is a complex neurodevelopmental condition characterized by deficits in social interaction, abnormalities in verbal and nonverbal communication, and restricted or repetitive patterns of behavior, which are believed to result from insults to the developing fetal and infant brain [5,34]. The prevalence of autistic disorder has increased markedly over recent decades prompting investigation into modifiable environmental contributors [34].

Experimental and epidemiological evidence suggests that inflammatory processes triggered by air pollution during neurodevelopment **(Table 2)** may play a key role in ASD pathogenesis [47-51]. Meta-analyses of observational studies support an association between ambient air pollution exposure and increased autism risk [52]. Multiple large-scale epidemiological studies across diverse populations have identified positive associations between prenatal exposure to air pollutants and elevated ASD risk [30]. A Danish population-based study confirmed a relationship between air pollution exposure and clinically diagnosed ASD [53].

Table 1: Summary of Key Pollutants and Associated Neurodevelopmental Outcomes

Pollutant Type	Example Compounds	Major Sources	Strength of Evidence	Associated Outcomes	Key References
Traffic-Related Air Pollution (TRAP) & Ultrafine Particles (UFPs)	$PM_{2.5}$, PM_{10}, NO_2, CO, benzene	Vehicle emissions, industrial combustion	Strong (multiple large cohort studies)	ASD, ADHD, reduced memory, impaired cognition	[1,11,18,29,34,64, 70,72–76]
Polycyclic Aromatic Hydrocarbons (PAHs)	Benzo(a)pyrene	Fossil fuel combustion, biomass burning, tobacco smoke, charred food	Strong	Reduced neurodevelopmental index, cognitive deficits, behavioral issues	[1,85–94]

Bisphenol-A (BPA)	BPA	Plastic containers, can linings	Moderate (mixed epidemiological results)	Hyperactivity, behavioral changes	[77–81]
Phthalates	MnBP, high/low MW phthalates	Food packaging, cosmetics, medical equipment	Moderate	Psychomotor delay, behavioral problems	[77,82–84]
Organophosphates	Chlorpyrifos (CPF), dialkyl phosphates	Agriculture, pest control	Strong	Reduced IQ, working memory deficits	[95–105]
Perfluorinated Compounds (PFCs)	PFOA, PFOS	Coatings, packaging, textiles	Weak–Moderate	ADHD, impulsivity (limited evidence)	[106–109]
PCBs, Dioxins, HCB	PCB 153, PCDD, PCDF	Electrical equipment, pesticides	Moderate–Strong	Cognitive delay, ADHD traits	[110–121]
PBDEs (POPs)	PBDE-47, PBDE-99	Flame retardants	Moderate	Cognitive deficits, reduced development indices	[122–129]
Heavy Metals	Hg, Pb, As	Industry, contaminated water/ food	Strong	Cognitive and psychomotor delay	[16,110,130–133]

Longitudinal cohort data further demonstrate that prenatal exposure to nitrogen oxides (NOx) is associated with increased ASD risk, even at concentrations below current WHO air quality thresholds, underscoring the sensitivity of the developing brain [30]. In addition, Windham et al. Reported that residential proximity to elevated ambient concentrations of chlorinated solvents and heavy metals near the time of birth was linked to higher autism prevalence [54].

Table 2: Mechanistic Pathways Linking Pollutants to Neurodevelopmental Disorders

Mechanism	Supporting Evidence	Associated Pollutants	References
Oxidative stress	Increased ROS, neuronal damage	TRAP, UFP, PAHs, metals	[5,18,29,44,72–76]
Neuroinflammation	Elevated cytokines in CNS tissue	TRAP, PAHs, metals	[5,18,47–51]
Endocrine disruption	Estrogenic/anti-androgenic effects	BPA, phthalates, PBDEs, PCBs	[77–84,110–121]
Epigenetic alterations	DNA methylation changes in placenta	Multiple pollutant classes	[8,10,122–129]
Neurotransmitter system disruption	Altered dopamine/serotonin regulation	OPs, CPF, metals	[95–105,132,133]

3. Attention-Deficit/Hyperactivity Disorder (ADHD) and Depression

ADHD is one of the most prevalent neurodevelopmental conditions in children, defined by age-inappropriate levels of inattention, impulsivity, and hyperactivity [55,56]. Individuals with ADHD frequently exhibit deficits in attention regulation, working memory, and temporal processing, alongside broader impairments in cerebellar-associated cognitive functions [57,58]. Although distinct from ASD, ADHD can also involve challenges in communication, social interaction, and patterns of restricted or repetitive behavior [1,42].

ADHD is a heterogeneous disorder; while its symptoms are treatable, untreated cases often lead to significant long-term consequences, including depression, substance dependence, and poor psychosocial outcomes—emphasizing the importance of early diagnosis and intervention [24,25,59,60]. Environmental exposures, including dietary additives, lead, toxins, and prenatal exposure to tobacco smoke and alcohol, have been implicated in ADHD pathogenesis [61,62].

Epidemiological research has linked specific associations between prenatal and early-life exposure to traffic-related air pollutants and increased ADHD prevalence [63]. Other studies report relationships between ambient air pollution and lower cognitive functioning, including reduced IQ and impairments in learning, executive function, and memory [26,64,65].

However, recent findings from eight European population-based birth cohorts suggest that neither prenatal nor postnatal exposure to air pollution is significantly associated with depressive, anxiety, or aggressive symptoms in children aged 7 to 11 years, indicating potential variability in outcomes depending on the pollutant type, exposure window, and developmental stage assessed [37].

4. Common Air and Environmental Pollutants

4.1. Ultrafine Particles (UFPs) and Traffic-Related Air Pollution (TRAP)

Ultrafine particles (UFPs), defined as particulate matter with a diameter below 0.1 μm (100 nm), are among the most reactive and biologically potent components of air pollution [66,67]. In the United States, UFPs are especially concentrated in high-traffic urban centers, with motor vehicle emissions representing their primary source [5,68]. Exposure to traffic-related air pollution (TRAP) has been linked to a range of neurobehavioral problems in children [69].

Evidence suggests that gestational timing influences vulnerability to the effects of environmental pollutants **(Figure 3)** (see details in **Table 3**). Higher $PM_{2.5}$ exposure at 18–26 weeks of gestation is associated with reduced visual memory, while exposure at 12–20 weeks correlates with diminished general memory [11,70]. Studies also indicate that prenatal or early postnatal exposure to TRAP components such as NO_2, particulate matter $PM_{2.5}$, and PM_{10} is associated with elevated autism risk [1,34]. Conversely, Harris et al. found no significant association between prenatal or early-life TRAP exposure and deficits in executive function or behavior, even during the third trimester [69].

Both indoor and outdoor prenatal exposure to CO can impair fetal brain development [71]. A growing body of research links ambient air pollutants—including TRAP, $PM_{2.5}$, NOx, and polycyclic aromatic hydrocarbons (PAHs)—to adverse neurodevelopmental outcomes [18,29,44,72-76]. For example, a Spanish cohort reported that $PM_{2.5}$ and NO_2 exposures were associated with memory deficits, while NO_2 was also linked to poorer verbal and general cognition in boys [76]. Another study found that prenatal exposure to NO_2 and benzene affected infant mental development, with effects more pronounced among infants of mothers reporting low antioxidant intake [64].

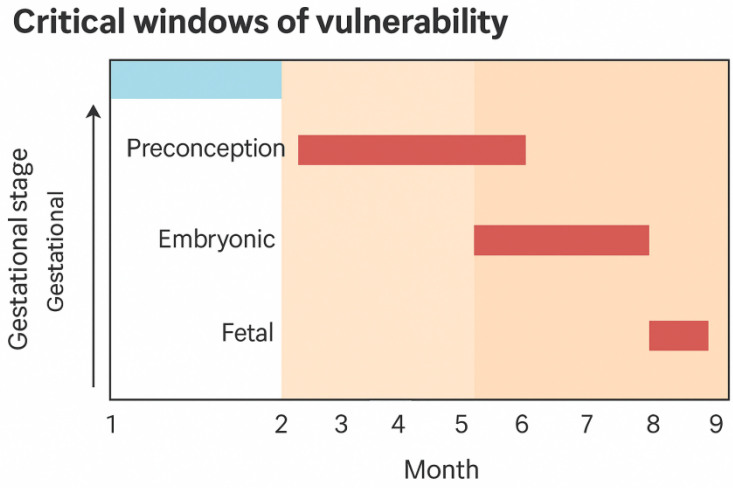

Figure 3: Critical Windows of Vulnerability During Prenatal Development

Timeline of gestational stages showing periods of heightened susceptibility to environmental pollutants, aligned with key neurodevelopmental processes such as neuronal proliferation, migration, synaptogenesis, and myelination. Early- and mid-gestation are depicted as peak vulnerability periods. *Created by authors using original artwork.*

4.2. Bisphenol-A (BPA)

Bisphenol-A (BPA) is a synthetic compound with estrogenic activity widely used in the production of plastics. While relatively few studies have examined its prenatal neurodevelopmental effects, available evidence is mixed. A recent meta-analysis concluded that early-life BPA exposure likely contributes to the development of hyperactivity [77,78]. In a cohort of African-American and Dominican women, prenatal BPA exposure was associated with altered behavioral outcomes in children, with sex-specific patterns [79]. By contrast, other studies have found no significant associations between prenatal urinary BPA concentrations in mid to late gestation and infant neurological status or later social functioning [80,81].

4.3. Phthalates

Phthalates are synthetic plasticizers found in numerous consumer products, including food packaging, cosmetics, and medical equipment. Although critical periods of vulnerability remain unclear, prenatal exposure has been linked to behavioral issues such as anxiety, withdrawal, hyperactivity, and aggression in children [77]. In Korea, prenatal phthalate exposure was inversely associated with mental and psychomotor developmental indices, particularly in boys [82]. In China, mono-n-butyl phthalate (MnBP) exposure was negatively associated with psychomotor development, with possible sex-specific effects of high-molecular-weight phthalates on neurocognition [83]. A recent study found predominantly null associations, with only weak links between low-molecular-weight phthalates and adolescent internalizing or externalizing behaviors [84].

4.4. Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are generated through incomplete combustion of fossil fuels, biomass, and tobaccon [85-87]. Human exposure occurs via inhalation, ingestion of grilled or smoked foods, and dermal contact [88]. PAHs are associated with increased cancer risk, cardiovascular disease, and impaired fetal growth [89-91]. Prenatal PAH exposure has also been identified as a risk factor for impaired neurobehavioral development, with transplacental transfer documented [92-94].

In a Mexican urban birth cohort, prenatal airborne PAH exposure was linked to maternal demoralization and poorer child neurobehavioral outcomes [1]. Dietary PAHs, such as those from grilled meat, have similarly been associated with adverse developmental effects [85].

4.5. Organophosphates (OP) and Chlorpyrifos (CPF)

Organophosphate (OP) pesticides are extensively used in agricultural, residential, and garden settings [95,96]. Chlorpyrifos (CPF), a commonly applied OP insecticide, has demonstrated neurotoxic potentials [97,98]. Prenatal and early childhood exposure to OPs, including CPF, has been linked to delayed neurodevelopment [99-102]. A New York City cohort reported associations between prenatal OP exposure and reduced cognitive performance, particularly in perceptual reasoning, from infancy through early childhood [103].

Working memory—a key cognitive function—is notably susceptible to CPF-related disruption [104,105]. Horton et al. observed a borderline significant interaction between prenatal CPF exposure and child sex, with males appearing more vulnerable to working memory impairment [97].

4.6. Perfluorinated Compounds (PFCs)

Perfluorinated compounds (PFCs) are synthetic chemicals used in stain-resistant coatings, food packaging, and numerous consumer products. Exposure occurs via ingestion, inhalation of household dust, and bioaccumulation in the food chain. Limited research has addressed prenatal neurotoxicity from PFCs. Some evidence links higher serum PFC levels to increased odds of parent-reported ADHD and impulsivity in children [106,107]. However, large-scale prospective studies—including the Danish National Birth Cohort—have reported no significant associations between prenatal perfluorooctanoate (PFOA) or perfluorooctane sulfonate (PFOS) levels and measures of motor, mental, or behavioral development [108,109].

4.7. Polychlorinated Biphenyls (PCBs), Dioxins, and Related Compounds

Polychlorinated biphenyls (PCBs), polychlorinated dibenzo-dioxins (PCDDs), and dibenzofurans (PCDFs) are persistent organic pollutants with high lipophilicity and bioaccumulation potential [110,111]. Elevated maternal serum or cord blood PCB levels have been associated with poorer cognitive and psychomotor performance in infancy and later childhood, along with increased ADHD-related behaviors [112-116]. However, some studies have found no such associationsns [117,118].

Similar studies have shown that prenatal or postnatal background-level exposure to environmental chemicals, such as PCBs and dioxins, induces adverse effects on children's neurodevelopment [119]. The Rhea mother–child cohort in Crete reported that high maternal serum concentrations of hexachlorobenzene (HCB) or PCBs were linked to lower perceptual performance, general cognition, executive function, and working memory scores in children [120]. No significant relationship was found with behavioral difficulties. Specific PCB congeners, such as PCB 153, have been associated with increased odds of motor delay [121].

4.8. Persistent Organic Pollutants (POPs), Polybrominated Diphenyl Ethers (PBDEs), and Dialkyl Phosphate (DAP)

Persistent organic pollutants (POPs) are resistant to environmental degradation and are known neurotoxicants [122]. Polybrominated diphenyl ethers (PBDEs), widely used as flame retardants, are strongly suspected of developmental neurotoxicity [123]. Prenatal PBDE exposure has been linked to impaired neurodevelopment [124,125], though larger sample sizes are needed to confirm associations with ASD [126].

Maternal dialkyl phosphate (DAP) metabolite concentrations—a biomarker of organophosphate pesticide exposure—have been associated with poorer working memory, processing speed, verbal comprehension, perceptual reasoning, and overall IQ in children [95]. In Spain, higher PBDE concentrations in breast milk were linked to lower mental development index scores in infants [127]. Chinese and Spanish cohorts have also reported inverse associations between prenatal PBDE exposure and developmental quotients or cognitive function, though statistical significance was not always reached [128,129].

Table 3: Selected Epidemiological Studies on Prenatal Pollutant Exposure and Child Neurodevelopment

Study Location	Population	Pollutant(s)	Exposure Window	Main Findings	References
Denmark	Nationwide cohort	NOx	Prenatal	Increased ASD risk, even below WHO thresholds	[30]
USA – California	Birth registry data	TRAP	Near birth	Higher ASD prevalence near industrial/traffic sites	[54]

Spain	Birth cohort	$PM_{2.5}$, NO_2	Prenatal	Memory deficits, lower verbal cognition in boys	[76]
Mexico	Urban cohort	PAHs	Prenatal	Maternal demoralization, reduced neurodevelopment	[1]
New York City	Cohort	CPF	Prenatal	Reduced perceptual reasoning, memory	[103]
China	Birth cohort	Phthalates	Prenatal	Psychomotor delay, possible sex-specific effects	[83]
Multiple European countries	8 cohorts	Air pollution	Prenatal & Postnatal	No link with depression/ anxiety in children 7–11 years	[37]

4.9. Mercury (Hg), Lead (Pb), and Arsenic (As)

Mercury (Hg) is a potent neurotoxin capable of crossing both the placental and blood–brain barriers [110,130]. Prenatal Hg exposure has been linked to delayed psychomotor and cognitive development in infancy, with potential persistence into adulthood [16]. Lead (Pb) and arsenic (As) are also well-established developmental toxicants; elevated prenatal exposure to either has been associated with impaired fetal neurodevelopment [131].

Animal models confirm that in utero Pb or As exposure can disrupt neural development [132,133]. Given their persistence and potency, these heavy metals remain high-priority targets for public health intervention.

5. Perspectives

Prenatal exposure to air pollution represents a significant, yet preventable, threat to fetal brain development and is increasingly recognized as a determinant of child health [8,76]. Pregnancy constitutes a critical window of vulnerability during which environmental contaminants can perturb neurodevelopmental trajectories, resulting in measurable postnatal cognitive and behavioral alterations [77]. Converging evidence from epidemiological and experimental studies indicates that the brain is a primary target for the adverse effects of airborne pollutants [5].

Although some inconsistencies exist across studies, multiple investigations have demonstrated that children exposed to air pollution during prenatal and postnatal periods are at heightened risk for cognitive deficits, behavioral disorders, and psychiatric symptoms [20,30,42,134-136]. Importantly, adverse health effects are observed even at pollutant concentrations typical of general population exposure, rather than only at extreme levels [39,137]. Nonetheless, limitations in exposure assessment—such as incomplete maternal exposure data—persist in some studies [39].

Emerging evidence suggests that exposure to air pollutants may disrupt neurodevelopmental processes from the earliest stages of gestation [3,15,74,138,139]. Air pollution affects the developing brain through multiple pathways, including neuroinflammation and oxidative stress [5,18]. Documented outcomes encompass reduced cognitive abilities [1,140], attention deficits, lower mental development index and IQ, anxiety and depression symptoms, reduced nonverbal reasoning, and delayed psychomotor development [1,21-24,26,35,135]. Because exposure begins in utero and continues over the life course, ongoing developmental monitoring is essential, particularly given the high susceptibility of children to environmental insults [16,142].

The rising prevalence of neurodevelopmental disorders globally underscores the need for enhanced health and education resources [143]. The central nervous system is especially vulnerable to pollutants due to its prolonged maturation from early gestation through adolescence [74,144,145]. Even brief exposures to environmental toxicants can disrupt tightly regulated developmental processes—such as cell proliferation, differentiation, and migration—if they occur during critical windows [131]. Current understanding of these interactions remains incomplete, warranting more sophisticated neuroepidemiological research.

6. Conclusion

The cumulative body of evidence supports a strong link between prenatal exposure to a wide range of outdoor and indoor pollutants—including traffic-related air pollution, particulate matter, polycyclic aromatic hydrocarbons, persistent organic pollutants, pesticides, endocrine-disrupting chemicals, and toxic metals—and adverse neurodevelopmental and behavioral outcomes in children. The primary biological mechanisms appear to involve oxidative stress, neuroinflammation, endocrine disruption, and epigenetic alterations in placental function.

Given the documented risks at pollutant concentrations below current regulatory thresholds, urgent public health action is needed [30]. This includes stricter air quality standards, reduction of emissions from key sources, targeted interventions to protect pregnant individuals, and broader community awareness of environmental risks. Longitudinal studies with precise exposure assessment and mechanistic exploration will be essential to further elucidate causal pathways and inform prevention strategies.

Consent for publication

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Credit Authorship Contribution Statement

Rachael Gakii Murithi: Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Validation, Writing - original draft.
Chan Lu: Visualization, Writing - review & editing.
Kun Tang: Supervision, Writing - review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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