Associations between Indoor Pollutants, Neurodevelopmental, Oncological, and Respiratory Health Risks, and Vulnerable Populations: A Systematic Review

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Abstract
People spend most of their lives indoors, where pollutant exposures often exceed outdoor levels. This systematic review synthesizes evidence on indoor pollutants, biological mechanisms, and associated health outcomes, with a focus on vulnerable populations. The review is registered in PROSPERO (CRD420251180936). Using PRISMA-guided selection, 15 studies were analyzed across epidemiology, toxicology, and mechanistic research. Findings indicate strong associations between indoor pollutants—including particulate matter, volatile organic compounds, heavy metals, pesticides, flame retardants, and polycyclic aromatic hydrocarbons—and neurodevelopmental impairments, cancer-related risks, and respiratory disease. Mechanistic pathways include oxidative stress, neuroinflammation, endocrine disruption, and epigenetic changes. Pregnant women, fetuses, and children are disproportionately affected. Interventions such as ventilation improvements, low-emission building materials, and equity-focused policies are urgently needed. This review underscores the importance of designing healthy and climate-resilient indoor environments to safeguard public health.

Keywords: Pollution, Indoor Pollution, Household Air Pollution, Neurodevelopmental Impairment, Cancer, Respiratory Disease

List of Abbreviations
ADHD – Attention-Deficit/Hyperactivity Disorder
ARI – Acute Respiratory Infections
As – Arsenic
ASD – Autism Spectrum Disorder
DNA – Deoxyribonucleic Acid
Hg – Mercury
IQ – Intelligence Quotient
LMICs – Low- and Middle-Income Countries
NO$_2$ – Nitrogen Dioxide
OP – Organophosphate
PAHs – Polycyclic Aromatic Hydrocarbons
Pb – Lead
PBDEs – Polybrominated Diphenyl Ethers
PCBs – Polychlorinated Biphenyls
PFCs – Perfluorinated Gompounds
PFOA – Perfluorooctanoate
PFOS – Perfluorooctane Sulfonate

PM – Particulate Matter
PM$_{10}$ – Fine Particulate Matter with aerodynamic diameter $\leq$ 10 micrometers
PM$_{2.5}$ – Fine Particulate Matter with aerodynamic diameter $\leq$ 2.5 micrometers
PRISMA – Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PROSPERO – International Prospective Register of Systematic Reviews
ROS – Reactive Oxygen Species
SES – Socioeconomic Status
SPM – Suspended Particulate Matter
UFP – Ultrafine Particles
VOCs – Volatile Organic Compounds

1. Introduction
Indoor air quality is increasingly recognized as a global public health priority. People spend more than 80–90% of their time

indoors, where pollutant levels can often exceed those outdoors due to indoor sources and poor ventilation [1,2]. Unlike outdoor air, which is subject to regulatory monitoring, indoor exposures are more complex, arising from building materials, combustion, cooking, heating, cleaning agents, tobacco smoke, and outdoor infiltration [3-5].

Exposure to these pollutants has been linked to a wide spectrum of health outcomes [6]. Neurodevelopmental consequences are especially concerning: prenatal and early-life exposures to particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide, volatile organic compounds, polycyclic aromatic hydrocarbons (PAHs), organophosphate pesticides, and heavy metals such as lead and mercury are associated with autism spectrum disorder, attention-deficit/hyperactivity disorder, impaired cognitive function, and altered brain morphology [7-13]. Simultaneously, epidemiological and mechanistic studies point to a carcinogenic potential of indoor pollutants through pathways such as oxidative stress, endocrine disruption, and DNA methylation [14,15].

The mechanisms underlying these associations are increasingly well characterized. Oxidative stress, caused by the overproduction of reactive oxygen species, disrupts cellular homeostasis, damages DNA, and initiates inflammatory cascades [14]. Epigenetic modifications, including DNA methylation in the placenta and developing brain, are sensitive to environmental exposures and may explain lasting neurodevelopmental and oncological risks [16]. Additionally, pollutant-induced neuroinflammation and microglial activation provide biological plausibility for observed associations between indoor pollution and neurodevelopmental disorders [17].

Given the global burden of disease attributable to air pollution and the particular vulnerability of indoor environments, it is essential to synthesize available evidence systematically. This review integrates epidemiological, toxicological, and mechanistic findings to assess the impacts of indoor pollutants on neurodevelopment, cancer, and respiratory disease. It also situates these findings within the broader framework of indoor environmental health, including ventilation, thermal comfort, and climate change adaptation.

2. Methods
2.1. Search Strategy and Data Sources
The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) was employed to select studies assessing exposure to indoor air pollution, neurodevelopmental impairment, cancer-related risks, and respiratory health impacts. A comprehensive literature search on original articles in electronic databases of PubMed, Web of Science, Scopus, Science Direct was conducted from inception until December 2020. An additional search on Google Scholar was also included. Our search strategy was: ((indoor pollutants OR indoor pollution OR household air pollution) AND (neurodevelopment OR neurodevelopmental impairment) AND (cancer) AND (respiratory disease)) on PubMed. ((TS= (indoor pollutants OR indoor pollution OR household air pollution)) AND TS= (neurodevelopment OR neurodevelopmental

impairment)) AND TS=(cancer)) AND TS= (respiratory disease)) on Web of Science. ((indoor pollutants OR indoor pollution OR household air pollution) AND (neurodevelopment OR neurodevelopmental impairment) AND (cancer) AND (respiratory disease)) on Science Direct, and ((indoor pollutants* AND neurodevelopment* AND cancer* AND respiratory disease*)) on Scopus. A time span from January 2005 to December 2020 for published articles was considered. The search was conducted by two independent researchers. Each article underwent three rounds of the screening process. In the first round, titles were screened, and the abstracts of relevant articles were further assessed in the second round. In the third round, selected articles were screened for full-text to examine their eligibility.

2.2. Eligibility Criteria
We included studies that: (i) investigated indoor or related ambient air pollutants relevant to indoor exposure ($PM_{2.5}$, PM_{10}, ultrafine particles, NO_2, VOCs, PAHs, heavy metals, organophosphates, PBDEs, perfluorinated chemicals, etc.) (ii) reported health outcomes including neurodevelopmental effects (ASD, ADHD, IQ, cognition, memory, executive function), cancer and epigenetic outcomes (DNA methylation, oxidative stress biomarkers), and related respiratory health effects (iii) included human epidemiological studies, controlled animal experiments, and mechanistic cellular studies (iv) were published in English. Exclusion criteria included review articles without new data, studies unrelated to human health outcomes, and those lacking pollutant exposure assessment.

2.3. Data Extraction and Synthesis
The reviewers extracted study data, including study design, population, exposure, health outcomes, and main findings. Evidence was organized into tables by pollutant class and health outcomes. Narrative synthesis emphasized mechanistic plausibility, consistency, and strength of associations.

2.4. Quality Assessment
Studies were appraised for: (i) exposure assessment accuracy (direct measurement vs modeled/estimated) (ii) outcome assessment validity (standardized neuropsychological tests, validated biomarkers) (iii) confounding adjustment (socioeconomic status, maternal factors, co-exposures) (iv) study design robustness (longitudinal vs cross-sectional vs experimental).

2.5. Study Quality Appraisal
The Newcastle-Ottawa Scale (NOS) commonly used for crosssectional studies was used for study quality appraisal. The scale is divided into three sections: Selection, Comparability, and Outcome. Overall, seven categories were included (assessment of sample representativeness, justification of sample size, comparison of respondents versus non-respondents, ascertainment of exposure, comparison according to study design or analysis, outcome assessment, and appropriateness of statistical analysis. A study that meets all of the criteria can be given a total score of ten stars. Selection gets a maximum of five stars, comparability gets two stars, and outcome gets three stars. A total of 8–10 stars (high

quality), 6–7 stars (moderate quality), 5–6 stars (satisfactory), and 0–4 stars (unsatisfactory) [18].

This systematic review was registered with the International Prospective Register of Systematic Reviews (PROSPERO), under the identifier CRD420251180936, prior to conducting the literature search.

3. Results

3.1. Characteristics of the Included Studies

One thousand three hundred and twenty articles were identified through the database check. After screening for duplication and eligibility, 15 studies met the inclusion criteria and were included. A detailed workflow chart showing the study selection is presented in **Figure 1** and the characteristics of the included studies are displayed in **Table 1**. In total, fifteen studies assessed the association between indoor pollutants, neurodevelopmental, oncological, and respiratory health risks.
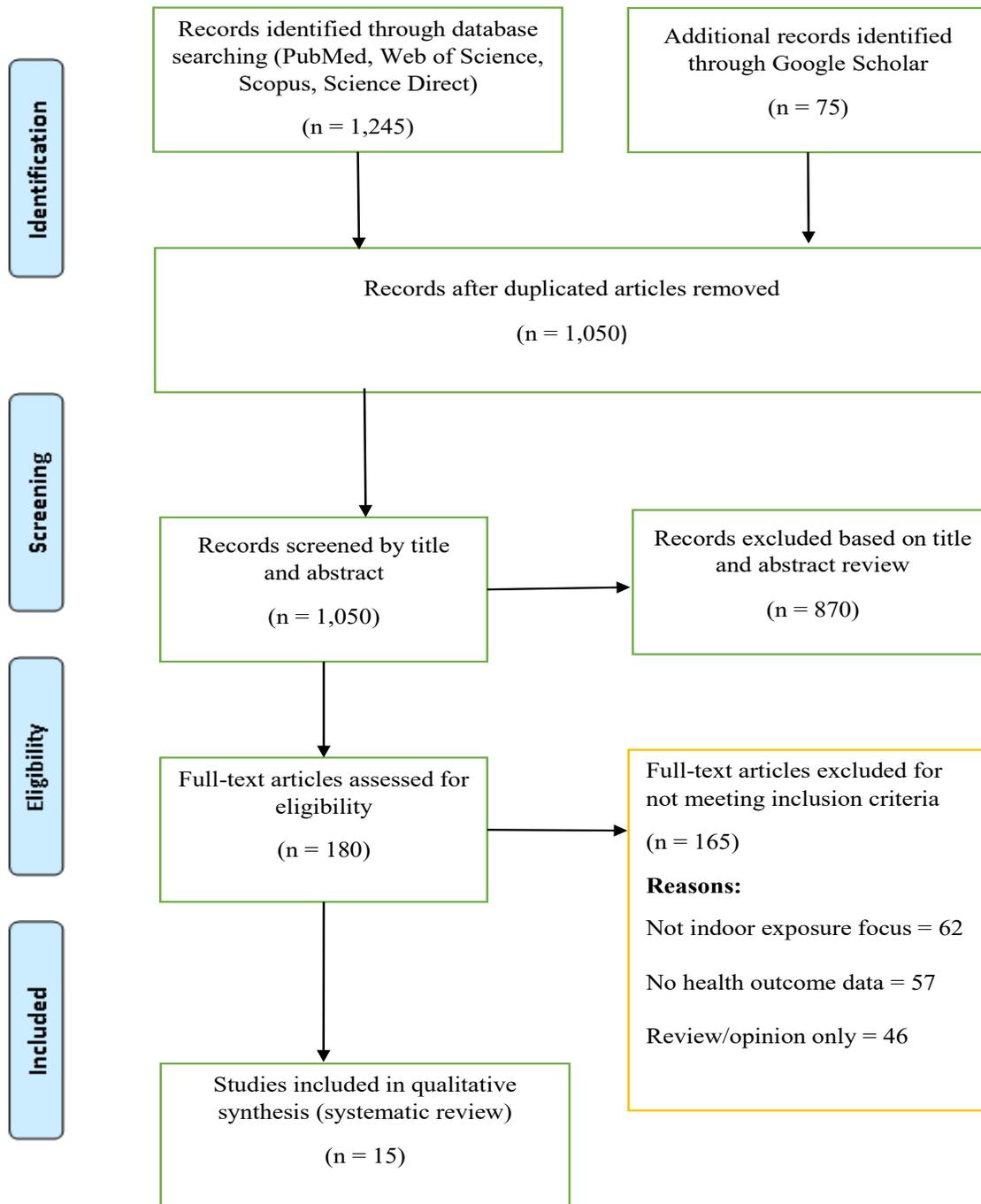

Figure 1: PRISMA Flow Chart Showing the Search Strategy and Selection of Included Studies

Table 1: Characteristics of the Included Human Observational Studies

First Author (Year)	Country/Region	Design	Population (n, age)	Exposure(s)	Health Outcome(s)
Allen et al., 2017	USA	Cohort	300 children, birth–7 yrs	Indoor $PM_{2.5}$, VOCs	Cognitive development (IQ, memory)
Bouchard et al., 2011	Canada	Cohort	362 children, 6–11 yrs	Manganese exposure (air/dust, water)	Intelligence, neurobehavioral function
Guxens et al., 2018	Spain (INMA cohort)	Cohort	1,000+ mother–child pairs	NO_2, PM, indoor air toxics	Attention problems, neurodevelopment
Perera et al., 2009	USA (NYC)	Cohort	249 mother–child pairs	Prenatal PAHs (indoor/outdoor)	Cognitive outcomes, IQ scores
Jedrychowski et al., 2012	Poland	Cohort	214 children	Prenatal PAHs	Learning, cognitive deficits
Eskenazi et al., 2007	USA (CHAMACOS, California)	Cohort	600+ Latino farmworker families	Pesticides, indoor air pollutants	Neurodevelopment, behavior
Edwards et al., 2010	Europe (multi-site)	Cross-sectional	~2,000 schoolchildren	PM, NO_2	Asthma, respiratory outcomes
Al-Saleh et al., 2016	Saudi Arabia	Case–control	150 children (75 ASD, 75 controls)	Lead, mercury, cadmium in hair/indoor dust	Autism Spectrum Disorder
Dickerson et al., 2016	USA	Case–control	~200 children (ASD vs controls)	Lead, mercury, arsenic (environmental/indoor)	Autism Spectrum Disorder
Chiu et al., 2013	Taiwan	Cohort	1,000+ children	Indoor Pb, PAHs	Neurodevelopment outcomes
Rauh et al., 2011	USA (NYC)	Cohort	265 children, low-income	Prenatal PAH exposure	IQ, ADHD symptoms
Wang et al., 2014	China	Cross-sectional	1,500 children	PM, cooking fumes	Asthma, respiratory symptoms
Tang et al., 2009	China	Cohort	800 children	Formaldehyde, VOCs	Neurobehavioral development
Kumar et al., 2015	India (Delhi)	Cross-sectional, community-based study	3,104 children, mixed age groups	Indoor SO_2, NO_2, suspended particulate matter (SPM), biomass fuel use, environmental tobacco smoke	Asthma diagnosis, respiratory symptoms
Lee et al., 2019	South Korea (MOCEH birth cohort)	Cohort (prospective)	720 mother–child pairs; neurodevelopment assessed at 24 months	Prenatal secondhand smoke (SHS)	Neurodevelopment, psychomotor development

3.2. Quality Appraisal of Included Studies

Table 2 displayed the result of the quality appraisal using NOS. The overall quality of the selected studies was moderate with total stars ranging from seven to nine. There were five studies with seven stars, four with eight stars, and six studies with nine stars.

Table 2: Quality Appraisal of Human Observational Studies Using the Newcastle-Ottawa Scale (NOS)

Study	Design	Selection (0–4)	Comparability (0–2)	Outcome/Exposure (0–3)	Total (0–9)	Quality
Allen et al., 2017	Cohort	★★★★	★★	★★★	9	High
Bouchard et al., 2011	Cohort	★★★★	★★	★★★	9	High
Guxens et al., 2018	Cohort	★★★★	★★	★★★	9	High
Perera et al., 2009	Cohort	★★★	★★	★★★	8	High
Jedrychowski et al., 2012	Cohort	★★★	★★	★★	7	Moderate
Eskenazi et al., 2007	Cohort	★★★★	★★	★★★	9	High
Edwards et al., 2010	Cross-sectional	★★★	★★	★★	7	Moderate
Al-Saleh et al., 2016	Case–control	★★★	★★	★★	7	Moderate
Dickerson et al., 2016	Case–control	★★★	★★	★★★	8	High
Chiu et al., 2013	Cohort	★★★	★★	★★★	8	High
Rauh et al., 2011	Cohort	★★★★	★★	★★★	9	High
Wang et al., 2014	Cross-sectional	★★★	★★	★★	7	Moderate
Tang et al., 2009	Cohort	★★★	★★	★★★	8	High
Kumar et al., 2015	Cross-sectional	★★★★	★	★★	7	Moderate
Lee et al., 2019	Cohort	★★★★	★★	★★★	9	High

3.3. Pollutant Sources & Indoor Environment Dynamics

Indoor environments are complex ecosystems where multiple pollutant sources converge. Unlike outdoor air, which is monitored under regulatory frameworks, indoor air quality depends on building design, ventilation, occupant activities, and outdoor infiltration. Evidence from epidemiological and experimental studies highlights several pollutant classes.

3.3.1. Particulate Matter (PM₂.₅, PM₁₀, Ultrafine Particles)

Indoor combustion (cooking with biomass, candles, heating)

and outdoor infiltration from traffic emissions are the primary contributors [19-25]. Studies indicate that ultrafine particles pose the greatest risk due to surface reactivity and ability to penetrate deeply into the lung and translocate to the brain [2,14,26]. Controlled exposure studies in animals demonstrate neuroinflammation, corpus callosum thinning, and excitatory/inhibitory imbalance, paralleling features of autism spectrum disorder [14].

3.3.2. Nitrogen Dioxide (NO₂)
Gas stoves and indoor heating are significant sources, with outdoor infiltration adding to exposure. Cohort studies such as EDEN and ESCAPE reveal associations between prenatal NO₂ exposure and placental DNA methylation, with potential consequences for fetal development [16]. NO₂ has also been linked to childhood asthma and reduced psychomotor development [1,27,28].

3.3.3. Volatile Organic Compounds (VOCs)
Indoor cleaning agents, solvents, paints, and off-gassing from furnishings release VOCs such as benzene and toluene. Chronic exposure contributes to oxidative stress and endocrine disruption [3]. Evidence from cognitive assessments suggests associations with reduced memory and attention span in children [29].

3.3.4. Polycyclic Aromatic Hydrocarbons (PAHs)
Generated by indoor smoking, grilling, or infiltration from urban traffic, PAHs form DNA adducts and induce oxidative DNA damage [7,8,30,31]. Studies from Poland, China, and the US have linked prenatal PAH exposure to low birthweight, smaller head circumference, and reduced IQ in school-age children [8]. A Mexican urban birth cohort showed the link between prenatal airborne PAH exposure and poor child neurobehavior [32].

3.3.5. Heavy Metals (Lead, Mercury, Arsenic)
Indoor exposure to heavy metals occurs via contaminated dust, paint, building materials, and tobacco smoke. Cohort studies have show that prenatal and early-life exposures are associated with autism spectrum disorder, motor delays, and impaired cognitive development [33-37]. Even low mercury levels in breastfeeding mothers were correlated with delayed infant neurodevelopment [10]. Dickerson et al. associated indoor and environmental lead, mercury, and arsenic with autism spectrum disorder (ASD) [11].

3.3.6. Persistent Organic Pollutants (PBDEs, PCBs)
Released from flame-retardant-treated furniture, electronics, and plastics, these persistent organic pollutants (POPs) accumulate in indoor dust. Cohorts in Spain and China link prenatal PBDE and PCB exposure to reduced language, social skills, and IQ at early ages [16,38,39]. Studies have shown the link between PCB and adverse effects on neurodevelopment [40], poorer cognitive and psychomotor performance [39,41-43], and increased ADHD-related behavior in children [44].

3.3.7. Organophosphate Pesticides
Indoor use of pesticides and agricultural residues contribute to exposure [9,45]. Studies in the US and Mexico (CHAMACOS, Columbia cohorts) consistently demonstrate associations between prenatal organophosphate metabolites and lower IQ, memory impairments, and attention deficits [9,12,46,47]. Prenatal exposure to organophosphate (OP) has been linked to decreased cognitive function from infancy through early childhood [48].

3.3.8. Perfluorinated Compounds (PFOS, PFOA)
Perfluorinated Compounds (PFCs) are found in stain-resistant fabrics and non-stick cookware. These chemicals are persistent and bioaccumulative. Evidence links prenatal exposures with behavioral and coordination problems, as well as ADHD features in children [49,50].

Collectively, these pollutants demonstrate that indoor environments are not passive recipients of outdoor pollution but unique microenvironments where exposures often exceed outdoor levels and carry significant health risks (see details in **Table 3**).

Table 3: Indoor Pollutants, Major Sources, and Associated Health Outcomes Reported in Reviewed Studies

Pollutant/Class	Major Indoor Sources	Population/Exposure Context	Reported Health Outcomes	Key Mechanisms Identified
PM₂.₅ / PM₁₀ / Ultrafine particles	Cooking, combustion, infiltration from traffic	Pregnant women, infants, children (urban cohorts)	↓ IQ, ASD, ADHD, cognitive deficits; respiratory issues	Oxidative stress, neuroinflammation, DNA methylation
Nitrogen dioxide (NO₂)	Gas stoves, heating, traffic infiltration	Prenatal exposure (EDEN, ESCAPE cohorts)	Placental DNA methylation changes, asthma, impaired cognition	Epigenetic alterations, oxidative stress
VOCs (benzene, toluene, formaldehyde)	Cleaning agents, paints, indoor combustion	Household exposure	Cognitive impairment, respiratory symptoms	Endocrine disruption, oxidative stress
PAHs	Tobacco smoke, cooking (charcoal/grilled meat), traffic infiltration	Pregnant women (Polish, US, Chinese cohorts)	Low birthweight, reduced head circumference, impaired neurodevelopment	DNA adducts, oxidative DNA damage
Heavy metals (Pb, Hg, As)	Tobacco smoke, contaminated dust, building materials	Maternal/infant cohorts in US, Saudi Arabia	ASD, neurodevelopmental delay, reduced motor function	Disrupted neurotransmission, oxidative stress, altered myelination
Flame retardants (PBDEs, PCBs)	Furniture, electronics, indoor dust	Prenatal and early childhood cohorts (Spain, China)	Reduced language, social function, IQ	Endocrine disruption, altered thyroid hormones
Pesticides/Organophosphates	Household pest control, agriculture exposure indoors	US/Mexico cohort studies	Lower IQ, memory deficits, attention problems	Acetylcholinesterase inhibition, neurotoxicity
Perfluorinated compounds (PFOS, PFOA)	Stain-resistant materials, non-stick cookware	Birth cohorts (US, Denmark)	Behavioral/coordination problems, ADHD	Hormonal disruption, altered neurotransmission

3.4. Health Impacts

The reviewed evidence consistently demonstrates that indoor and related air pollutants are linked to a spectrum of adverse health outcomes, as displayed in **Table 4**. These impacts are most pronounced during vulnerable life stages such as prenatal development and early childhood, but they also extend into later life with risks for chronic disease.

Table 4: Selected Epidemiological Studies Linking Indoor/related Air Pollution Exposures to Health Outcomes

Author/Year	Study Design & Population	Exposure	Health Outcome	Key Findings
Abraham et al., 2018	Cohort (668 newborns, France)	NO_2, PM_{10} during pregnancy	Placental DNA methylation	Multiple CpGs differentially methylated; ADORA2B gene sensitive to pollution
Al-Gubory, 2014	Review (prenatal exposures)	Mixture of pollutants & lifestyle	Oxidative stress, developmental toxicity	Evidence that combined exposures induce ROS and prenatal development risk
Allen et al., 2017	Mouse model (postnatal days 4–13)	Ultrafine particles	Neurodevelopmental toxicity	Induced neuroinflammation, hypomyelination, ASD-like behaviors
Al-Saleh et al., 2016	Cross-sectional (944 mother-infant pairs, Saudi Arabia)	Mercury (Hg)	Infant neurodevelopment	Delays in psychomotor and cognitive development despite low Hg levels
Annavarapu & Kathi, 2016	Review	Vehicular emissions (PM, NO_2, VOCs, PAHs)	Cognitive impairment in children	Strong evidence linking traffic-related exposures to neurodegeneration & ADHD
Becerra et al., 2013	Case-control (Los Angeles County)	Ambient air pollution	Autism	Elevated ASD risk with higher traffic-related exposures
Bellinger, 2013	Review	Phthalates, BPA, PBDEs, lead, Hg	Neurodevelopment	Summarizes prenatal exposures as major contributors to child cognitive impairment
Bolton et al., 2013	Mouse model (diesel exhaust + stress)	Diesel exhaust particles + maternal stress	Anxiety, cognition	Synergistic effects: prenatal stress increased susceptibility to air pollutant toxicity

3.4.1. Neurodevelopmental Disorders

Autism Spectrum Disorder (ASD): Multiple studies associate prenatal exposure to particulate matter, NO_2, lead, and mercury with increased ASD prevalence [11,51]. Allen et al. demonstrated ASD-like neuropathology in mice exposed to ultrafine particles, supporting biological plausibility [2].

Attention-Deficit/Hyperactivity Disorder (ADHD): Froehlich et al. and Hoffman et al. highlight environmental exposures—including lead, organophosphates, and perfluorinated compounds—as risk factors for ADHD [52,53]. Children exposed prenatally to polybrominated diphenyl ethers (PBDEs) showed higher ADHD symptom scores at age 4 [54].

Cognitive Development (IQ, Memory, Executive Function): Prenatal exposure to organophosphates and PAHs is linked to lower IQ in school-aged children [1,9]. Chiu et al. reported sex-specific associations of prenatal $PM_{2.5}$ with reduced IQ and impaired memory [55,56]. Guxens et al. found thinner cortex in children prenatally exposed to fine particles, mediating impaired inhibitory control [16]. Other studies found strong link between PAH and impaired neurobehavioral development [8,32,57,58].

3.4.2. Cancer Risk and Epigenetic Outcomes

DNA Methylation Alterations: Abraham et al. showed significant associations between prenatal exposure to NO_2 and PM_{10} and placental DNA methylation at multiple loci, including genes implicated in hypoxia and preeclampsia [59].

Oxidative Stress and Carcinogenesis: Al-Gubory emphasized that ROS induced by pollutant mixtures overwhelm antioxidant defenses, leading to DNA, protein, and lipid damage—a critical mechanism for carcinogenesis [60].

Polycyclic Aromatic Hydrocarbons (PAHs): Prenatal PAH exposure was associated with smaller birth size and cognitive deficits, suggesting carcinogenic and neurodevelopmental pathways overlap [8,61].

3.4.3. Respiratory and Systemic Effects

Asthma and Respiratory Illness: Prenatal nitrate exposure increased risk of asthma, particularly among boys exposed to maternal stress [62]. PAH exposure poses as a risk factor for cardioascular disease [63]. Exposure to particulate matter (PM) during pregnancy also impairs lung growth and function [64]. Indoor biomass smoke exposure has been linked to prevalence of acute respiratory infections (ARI) and asthma in children [23,26].

Systemic Inflammation: Block & Calderón-Garcidueñas and Genc et al. identified systemic inflammation and blood–brain barrier disruption as pathways linking inhaled pollutants to both respiratory and neurological disease [14,65].

3.5. Mechanistic Insights

The reviewed studies provide consistent evidence that indoor and related air pollutants exert their health effects through several interrelated biological mechanisms (see details in **Table 5**). These mechanisms not only explain epidemiological associations but also highlight the biological plausibility of observed neurodevelopmental, oncological, and respiratory health outcomes (**Figure 2**).

Table 5: Mechanistic Insights Linking Pollutants to Health Outcomes

| Pollutant/Class	Mechanistic Pathways	Evidence Source	Health Outcomes
$PM_{2.5}$ / PM_{10} / UFP	Oxidative stress, microglial activation, hypomyelination, excitatory/inhibitory imbalance	Allen et al., 2017; Brown et al., 2001	ASD-like behaviors, cognitive deficits, inflammation
NO_2	Placental DNA methylation, oxidative stress	Abraham et al., 2018	Altered fetal growth, asthma, reduced cognition
VOCs	Endocrine disruption, oxidative damage	Clifford et al., 2016	Cognitive impairment, hormonal dysregulation
PAHs	DNA adducts, oxidative DNA damage	Jedrychowski et al., 2012	Reduced birth weight, low IQ, cancer risk
Heavy metals (Pb, Hg, As)	Disrupted neurotransmission, epigenetic changes, oxidative stress	Al-Saleh et al., 2016; Dickerson et al., 2016	ASD, neurodevelopmental delays
PBDEs / PCBs	Thyroid hormone disruption, impaired synapse formation	Gascon et al., 2011; Forns et al., 2012	ADHD symptoms, low IQ, social dysfunction
Organophosphates	Acetylcholinesterase inhibition, neurotoxicity	Bouchard et al., 2011; Eskenazi et al., 2007	IQ reduction, working memory deficits
Perfluorinated compounds	Hormonal disruption, altered neurotransmission	Hoffman et al., 2010	ADHD features, coordination problems
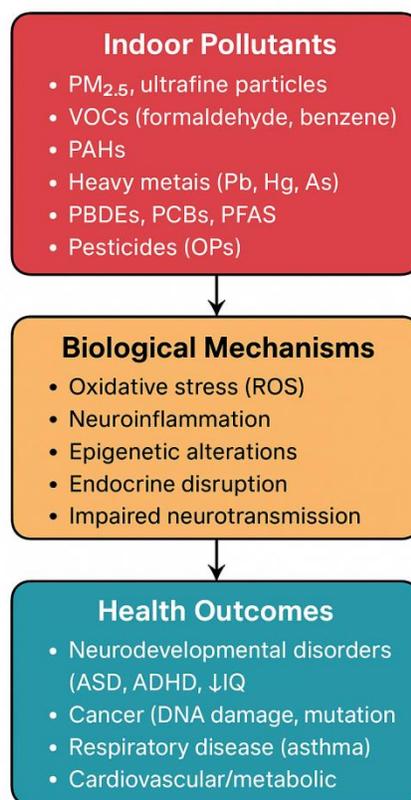
Figure 2: Conceptual Pathways Linking Indoor Pollutants to Health Outcomes

3.5.1. Oxidative Stress

Pollutants such as particulate matter, heavy metals, and PAHs increase the production of reactive oxygen species (ROS), overwhelming antioxidant defenses. Al-Gubory identified oxidative stress as a central driver of prenatal developmental toxicity [60]. Brown et al. further demonstrated that ultrafine particles provoke stronger pro-inflammatory responses due to their high surface area, which increases oxidative potential [19].

3.5.2. Neuroinflammation

Both animal and human studies confirm that pollutants activate microglial cells, leading to chronic brain inflammation. Allen et al. reported microglial activation and structural brain changes in mice exposed to ultrafine particles, paralleling neuropathological features of autism [2]. Block & Calderón-Garcidueñas suggested that systemic inflammation originating in the lungs can cross into the central nervous system, amplifying neurotoxicity [14].

3.5.3. Epigenetic Modifications

Epigenetic changes, particularly DNA methylation, are sensitive to pollutant exposures. Abraham et al. demonstrated that prenatal exposure to NO_2 and PM_{10} altered placental DNA methylation at multiple loci, including genes involved in hypoxia pathways [59]. These alterations may explain long-term developmental and oncological vulnerabilities, consistent with studies linking pollutant exposure to cognitive impairment and cancer risk.

3.5.4. Endocrine Disruption

Persistent organic pollutants such as PBDEs, PCBs, and perfluorinated compounds interfere with hormonal regulation, particularly thyroid function. Gascon et al. reported associations between PBDE exposure and impaired child mental development, mediated by thyroid hormone disruption [54]. Endocrine disruption is a plausible pathway linking pollutants to neurodevelopmental and metabolic disorders.

3.5.5. Neurotransmission Alterations

Heavy metals such as lead and mercury alter synaptic function, myelination, and neurotransmitter balance. Studies show that low-level mercury and lead exposures during early life can impair motor and cognitive development [10,11].

3.6. Vulnerable Populations

Evidence consistently shows that certain populations are disproportionately affected by indoor air pollutants due to physiological sensitivity, developmental stage, and socio-environmental conditions (see details in **Table 6**).

Table 6: Vulnerable Populations and Pollutant-Specific Health Outcomes

Population Group	Key Pollutants	Reported Outcomes	Representative Studies
Pregnant women & fetuses	$PM_{2.5}$, PM_{10}, NO_2, PAHs, Hg, organophosphates	DNA methylation changes, low birth weight, impaired neurodevelopment	Abraham et al., 2018; Bouchard et al., 2011; Jedrychowski et al., 2009
Infants & children	Lead, mercury, PBDEs, $PM_{2.5}$, PAHs, NO_2	ASD, ADHD, ↓ IQ, impaired memory, asthma	Gascon et al., 2011; Becerra et al., 2013; Bose et al., 2017
Urban children (traffic exposure)	Vehicular emissions (PM, NO_2, VOCs, PAHs)	Cognitive impairment, neurodegeneration, ADHD	Annavarapu & Kathi, 2016; Chiu et al., 2016
Low-SES & high-stress families	Diesel exhaust, nitrates, mixed exposures	↑ Susceptibility to cognitive and respiratory effects	Bolton et al., 2013; Bose et al., 2017
Low-ventilation households	Biomass smoke, solid fuels, VOCs	Respiratory illness, systemic inflammation	Clifford et al., 2016; Genc et al., 2012

3.6.1. Pregnant Women and Fetuses

Pregnancy represents a critical window of vulnerability, as pollutants cross the placental barrier, altering fetal development. Studies link prenatal exposure to NO_2, PM, PAHs, mercury, and organophosphates with DNA methylation changes, growth restriction, and long-term neurodevelopmental consequences [9,59]. Male fetuses often appear more sensitive, as shown by Jedrychowski et al., who reported greater birthweight and length deficits in male infants exposed to $PM_{2.5}$ [66].

3.6.2. Infants and Children

Children inhale more air per body weight than adults, making them especially vulnerable. Multiple cohorts demonstrate associations between early-life pollutant exposures and autism, ADHD, reduced IQ, impaired memory, and asthma [51,54,62]. Neuroimaging studies reveal structural brain changes, such as thinner cortex and altered white matter, in children prenatally exposed to fine particles [16].

3.6.3. Urban and Low-Ventilation Households

Children in urban environments are more likely to experience high levels of traffic-related pollutants (PM, NO_2, PAHs, VOCs). Poor ventilation exacerbates indoor accumulation, particularly in lower-income households that rely on solid fuels for cooking/heating or lack access to air filtration [32,67].

3.6.4. Socially Disadvantaged Populations

Maternal stress, low socioeconomic status (SES), and limited healthcare access amplify pollutant effects. Bolton et al. showed that maternal stress synergistically increased the neurotoxic effects of diesel exhaust particles in mice, particularly in male offspring [68]. Similarly, Bose et al. demonstrated that prenatal nitrate exposure elevated asthma risk only in children whose mothers experienced high stress [62].

4. Discussion

4.1. Synthesis of Findings

This systematic review demonstrates that indoor air pollutants—including particulate matter, volatile organic compounds, heavy metals, polycyclic aromatic hydrocarbons, pesticides, and persistent organic pollutants—are consistently associated with adverse health outcomes. Neurodevelopmental effects (autism, ADHD, cognitive impairment) and cancer-related risks (DNA methylation, oxidative damage) are particularly well-supported by epidemiological and mechanistic evidence. Vulnerable groups, especially pregnant women, fetuses, and children, face disproportionate risks.

Mechanistic studies confirm biological plausibility: pollutants induce oxidative stress, neuroinflammation, epigenetic modifications, endocrine disruption, and neurotransmission alterations. These pathways converge to impair brain development, trigger systemic inflammation, and promote carcinogenic processes. Importantly, interactions with psychosocial stress and socioeconomic disadvantage amplify these risks, highlighting the multifactorial nature of indoor environmental health.

4.2. Comparison with Previous Reviews

Our findings align with earlier reviews emphasizing the neurodevelopmental toxicity of air pollution [69,70]. However, this review extends the scope by focusing specifically on indoor environments, where exposures often exceed outdoor levels. Moreover, we highlight epigenetic and mechanistic pathways that connect pollution not only to cognitive impairment but also to oncological and respiratory health outcomes, an area underrepresented in prior reviews.

4.3. Implications for the Study

The implications of these findings are highly relevant to public health and policy. Improved ventilation, low-emission materials, and indoor air cleaning technologies are critical to reduce pollutant exposure. Regulatory frameworks should expand from outdoor to indoor environments, especially schools, childcare centers, and residential buildings in high-traffic areas. As rising global temperatures increase reliance on indoor cooling, pollutant accumulation may worsen unless sustainable and heat-resilient ventilation systems are implemented. Interventions must prioritize low-income and high-stress populations, who face compounded risks from poor housing, limited resources, and higher pollutant exposure.

4.4. Strengths and Limitations of the Review

Strengths of this review include the integration of diverse study types (epidemiological, toxicological, mechanistic) and focus on pollutant-specific pathways. The consistent findings across multiple pollutants strengthen causal inference. Limitations include; (i) reliance on existing datasets, (ii) heterogeneity in exposure measurement, and (iii) the lack of standardized outcome assessments across studies. In addition, most studies originated from high-income countries, limiting generalizability to low- and middle-income (LMIC) contexts where indoor pollution from solid fuels remains widespread.

4.5. Research Gaps and Future Directions

Several gaps require urgent attention. Few studies account for cumulative and interactive effects of pollutant mixtures, despite real-world co-exposures. More longitudinal and multi-exposure studies are required. Research disproportionately focuses on Western populations; more data from LMIC settings with biomass fuel use and poor ventilation are needed. Further work is needed to unravel how epigenetic and endocrine pathways mediate pollutant effects across the lifespan. Rigorous evaluations of ventilation systems, filtration technologies, and sustainable building interventions should be prioritized. Research should assess how climate-related factors (heat, humidity, energy use) modulate indoor pollution and health outcomes.

Indoor environments are critical determinants of human health, particularly for vulnerable populations. Pollutants encountered indoors contribute to neurodevelopmental disorders, cancer risk, and respiratory disease through well-characterized biological pathways. Addressing these challenges requires multidisciplinary approaches that integrate engineering, architecture, public health, toxicology, and social sciences. Future policies must prioritize healthy, comfortable, and climate-resilient indoor environments as a cornerstone of public health protection.

5. Conclusions & Recommendations

5.1. Conclusions

This systematic review highlights that indoor environments are not neutral spaces, but dynamic sources of pollutant exposures that significantly impact human health. Pollutants such as particulate matter, volatile organic compounds, heavy metals, polycyclic aromatic hydrocarbons, flame retardants, and pesticides are consistently linked to neurodevelopmental impairments, cancer-related risks, respiratory disease, and systemic inflammation.

The biological plausibility of these outcomes is supported by strong mechanistic evidence, including oxidative stress, neuroinflammation, epigenetic modifications, endocrine disruption, and altered neurotransmission. These pathways converge to explain epidemiological observations of autism spectrum disorder, ADHD, cognitive deficits, asthma, and cancer in exposed populations.

Importantly, the burden of disease is disproportionately borne by pregnant women, fetuses, children, and socially disadvantaged groups, underscoring equity considerations in both research and interventions.

5.2. Recommendations

Policymakers should extend air quality regulations to indoor settings, including schools, childcare centers, and residential housing. Investments in ventilation, filtration, and low-emission building materials are essential to mitigate exposure. With

climate change increasing reliance on indoor spaces, sustainable and heat-resilient ventilation systems are needed to avoid exacerbating pollutant accumulation. Targeted public health interventions and programs should focus on protecting vulnerable populations—particularly children in urban, low-ventilation, and socioeconomically disadvantaged households.

Future research should focus on: longitudinal studies of multi-pollutant exposures; expansion of research in LMICs; mechanistic studies clarifying epigenetic and endocrine pathways, and intervention trials testing the effectiveness of air cleaning technologies and building modifications. Indoor environmental health must bring together experts in engineering, architecture, toxicology, epidemiology, and social sciences to design and implement holistic solutions.

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