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The Impact of Air Pollution on Lung Development and Physical Growth in Early Childhood: A Systematic Review of Risk Factors and Long-Term Effects

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Abstract

Children in developing countries are highly vulnerable to respiratory health problems caused by air pollution. Exposure during pregnancy and early life critically influences lung development and physical growth, with potential long-term consequences. This systematic review aimed to assess the effects of prenatal and early childhood exposure to air pollution on lung development and physical growth, and to identify risk factors that may influence these outcomes. A systematic search was conducted in February 2025 across Scopus, PubMed, and Web of Science databases. Studies published in English between 2015 and 2025 were screened using PRISMA guidelines and assessed for quality using the ROBINS-I tool. From 1,303 records, 28 studies met the inclusion criteria. Twenty-one studies (75%) reported reductions in lung function parameters such as FEV_1, FVC, and $FEF_{25–75\%}$ in association with pollutants including PM2.5, NO_2, O_3, and PAHs. Three studies reported increased risk of stunting, primarily linked to indoor air pollution and biomass fuel exposure. These outcomes were associated with mechanisms such as oxidative stress, systemic inflammation, and epigenetic changes. The second trimester of pregnancy and the first two years of life were identified as critical exposure windows. Risk was modified by age, sex, socioeconomic status, breastfeeding duration, and home ventilation. Air pollution exposure during sensitive developmental periods impairs lung function and physical growth in children. Urgent public health measures—promoting clean energy, improving indoor air quality, and reducing exposure in vulnerable groups—are essential to support healthy childhood development.

Keywords: Air Pollution, Lung Development, Child Growth, Critical Period, Risk Factors

1. Introduction

Children in developing nations are more likely to get respiratory problems caused by air pollution, such as asthma, acute respiratory infections, and lung function problems.[1] Evidence suggests that exposure to air pollutants—particularly PM2.5—during prenatal and early developmental stages can disrupt normal lung development, thereby increasing susceptibility to various respiratory conditions throughout childhood such as acute respiratory infections, asthma, and impaired lung function.[1–4] Research indicates that children aged 6–9 years exhibit significant reductions in forced vital capacity (FVC) and forced expiratory volume in one second (FEV1), with these effects potentially persisting into adolescence and early adulthood.[2,4–7] Importantly, longitudinal studies have demonstrated that reductions in ambient air pollution correlate with enhancements in lung function development, underscoring the critical need for effective air quality interventions.[8]

The "Developmental Origins of Health and Disease" (DOHaD) theory highlights the critical role of environmental exposures during pivotal developmental periods in shaping long-term health trajectories, with particular emphasis on pediatric pulmonary development.[9–11] The biological mechanisms that confer susceptibility in children to the deleterious effects of air pollution encompass epigenetic modifications, metabolic dysregulation, and endocrine alterations induced by exposure during key

developmental phases.[12,13] These findings highlight the need for a nuanced understanding of the interplay between environmental factors and developmental biology in respiratory health.

Although recent epidemiological studies demonstrate significant associations between reduced air pollution exposure and enhanced lung function development in pediatric populations, the field continues to grapple with inherent limitations, including discrepancies in findings across various studies and a paucity of robust longitudinal data.[2,5,8,14–19] Furthermore, critical research gaps persist regarding the permanence or reversibility of effects stemming from early-life exposures, the identification of the most susceptible exposure windows, and the long-term efficacy of air quality interventions.[2,18,20] Addressing these unanswered questions is imperative for advancing our understanding of the relationship between air quality and respiratory health in children.

The association between air pollution and pediatric health outcomes has garnered significant attention, particularly concerning its detrimental effects on respiratory health. Evidence suggests that exposure to air pollutants is correlated with an increased incidence of respiratory diseases, compromised lung function, and heightened susceptibility to asthma in the pediatric population.[19–21] Furthermore, prenatal exposure to air pollutants has been linked to adverse neuropsychological development and metabolic health outcomes in children.[22–25] This study aims to systematically evaluate the long-term consequences of air pollution on lung development and growth during early childhood. Additionally, it seeks to identify potential risk factors that may modulate these effects. The anticipated findings will contribute to enhancing public health policies and environmental interventions, ultimately promoting lung health and optimal growth trajectories in children exposed to air pollution.

2. Methods

Study design and setting

This study employed a systematic literature review to examine the effects of air pollution on lung development and physical growth in early childhood. It focused on identifying exposure-related health outcomes and associated risk factors. The review synthesized existing evidence on how early-life pollution impacts pediatric respiratory and developmental health.

Inclusion and exclusion criteria

Inclusion criteria were defined using the PICOS framework (Population, Intervention, Comparison, Outcomes, Study design). The target population comprised early childhood subjects, with the primary intervention of interest being exposure to air pollution. Notably, no comparison group was incorporated, as the review's focus was solely on the effects of air pollution on lung development and physical growth. Outcomes evaluated included lung function, growth parameters, developmental milestones, and associated risk factors. Only studies involving early childhood populations published in English from 2015 to 2025 were considered eligible for inclusion. Exclusion criteria encompassed review articles, conference proceedings, and book chapters to ensure the robustness and specificity of the findings.

Search strategy

A systematic search was conducted in February 2025 using three databases: Scopus, PubMed, and Web of Science (WoS). The search terms included: “Early Childhood” OR “Child” OR “Children” OR “Air Pollution” OR “Lung Development” OR “Lung Function”.

Screening and selection of the articles

The systematic review followed PRISMA guidelines (Figure 1) and used the Parsifal platform for screening and metadata extraction. The process involved two main stages: duplicate removal and eligibility assessment based on inclusion criteria (English-language studies from 2015–2024 on air pollution's impact on early childhood lung development and growth) and exclusion of secondary sources. Three researchers independently screened the articles, with only studies unanimously approved included. In cases of disagreement, inclusion decisions were resolved through discussion until consensus was achieved. Titles and abstracts were evaluated for relevance, and full texts of accepted studies underwent quality assessment to ensure robust evidence selection.

Data extraction

All reviewers extracted the sample size, country of origin, population, intervention, comparison, and outcomes of interest for each included study. The review evaluated key variables that influence lung development, physical growth, and risk factors reported in the included studies.

Quality assessment

All three reviewers assessed study quality using the ROBINS-I tool, which evaluates risk of bias in non-randomized studies across seven domains, including confounding, participant selection, and outcome measurement. Each domain is rated from low to critical risk, with the overall rating reflecting the highest individual risk. Given the susceptibility of non-randomized studies to bias, ROBINS-I ensures rigorous appraisal essential for evidence-based decision-making in clinical practice.

Data analysis

The data derived from the selected studies were subjected to descriptive analysis to elucidate patterns, inter-study variances, and the underlying mechanisms connecting air pollution to pediatric lung development, growth trajectories, and associated risk factors. This methodological approach was strategically employed to thoroughly synthesize the prevailing literature, thereby facilitating more profound insights into the complex interplay between environmental pollutants and child health outcomes.

3. Results

Study selection

Following PRISMA guidelines, a systematic search across Scopus, Web of Science, and PubMed identified 1,303 studies. After removing duplicates, 982 remained; 920 were excluded during screening. Of the 62 potentially eligible articles, 46 were inaccessible and 14 failed quality assessment, resulting in 28 studies included in the final analysis. These provide strong evidence of air pollution’s harmful effects on pediatric health.

Characteristics of the included studies

The characteristics and outcomes of the included studies are delineated in Table 1. It shows international statistics on sample sizes, population demographics, intervention strategies, comparison groups, and outcome measures. The research was conducted in the US, Canada, Ethiopia, Nigeria, Spain, Poland, Australia, France, Germany, Switzerland, China, India, and Israel. From 62 to 6,501 participants, sample sizes varied greatly. These studies focused on children, with an average age of 0–9 years. These findings highlight study design and implementation variation, helping us understand relevant interventions and their efficacy.

Risk of bias

A 28-study bias assessment risk analysis showed significant methodological quality diversity (Figure 2 and Table 2). D4 (deviations from intended interventions), D3 (classification of interventions), D6 (outcome measurement), and D7 (selection of reported findings) had the highest low-risk proportions, with 90% of studies. In contrast, Domain D1 (confounding) had a considerable risk, involving 51.7% of investigations. To protect findings integrity, one study with a high bias risk was omitted from data extraction. Only 31% of studies had minor bias, whereas 65.5% had considerable bias. Interestingly, 28 of 29 papers (96.6%) had acceptable methodological quality and a low to moderate risk of bias, proving their eligibility for this systematic review.

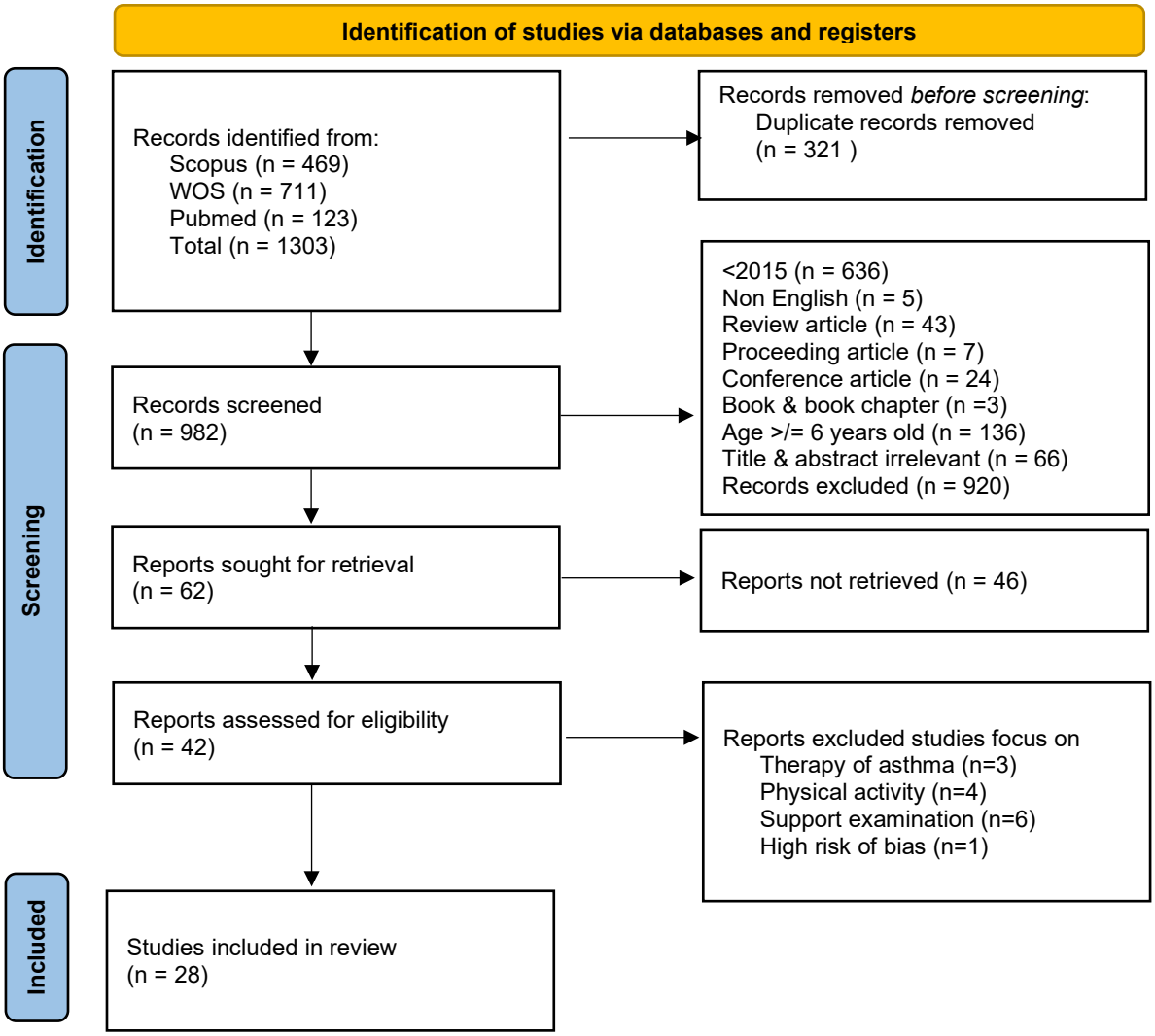

Figure 1. PRISMA flowchart illustrating the literature selection process

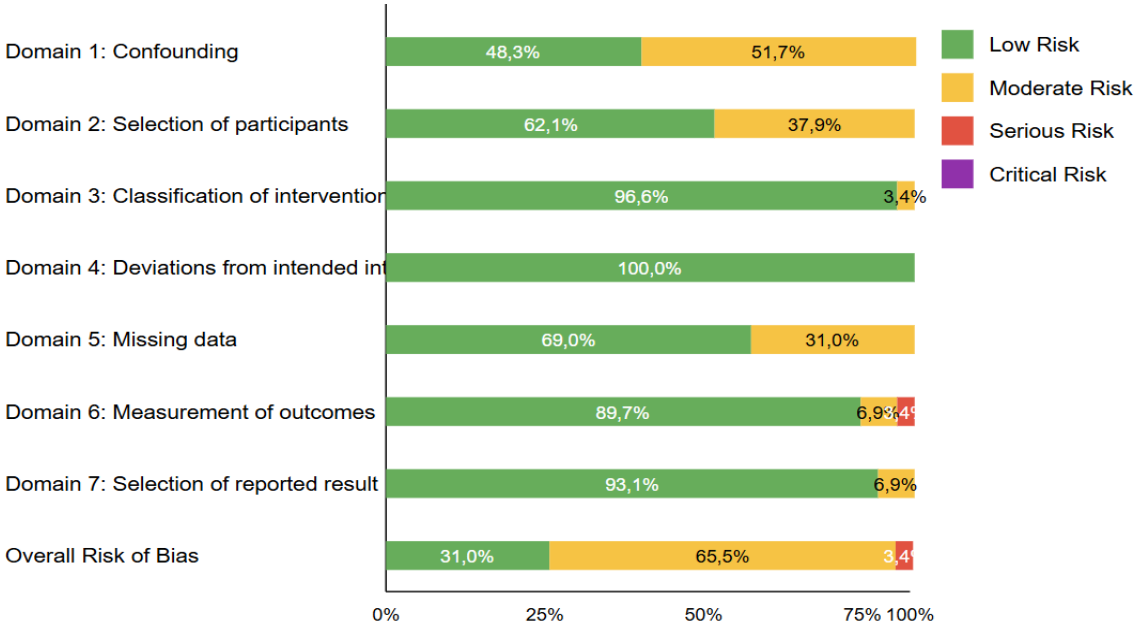

Figure 2. Quality assessment result using ROBINS-I

3.1. Respiratory impact

Scientific evidence consistently demonstrates the significant impact of air pollution on lung development in pediatric populations through a series of complex biological mechanisms (Table 3). Exposure to ozone (O_3) is associated with airway dysfunction and inflammatory processes (Bai et al., 2023), while particulate matter (PM2.5) correlates with impairments in lung capacity, notably reduced forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC).[26,27] Prenatal exposure to airborne pollutants, particularly PM2.5 and polycyclic aromatic hydrocarbons (PAHs), has been linked with compromised pulmonary function in early childhood.[28,29]

Volatile organic compounds (VOCs), particularly benzene, present indoor air quality challenges by significantly elevating asthma risk, estimated at two- to threefold.[30] Children are especially vulnerable due to their underdeveloped pulmonary and immune systems and higher minute ventilation rates.[31] Even low to moderate air pollution levels have been associated with reduced lung function and increased respiratory symptoms.[32] Long-term consequences of early-life exposure include persistent deficits in lung function and increased risk for chronic respiratory diseases.[33–35]

Moreover, air pollution has been linked to epigenetic alterations, including DNA methylation and gene regulation, which contribute to increased asthma susceptibility and reduced lung volumes.[36,37]

Children's susceptibility to the respiratory effects of air pollution is modulated by a complex interplay of risk factors. These include behavioral patterns (e.g., frequency of window opening, which increases O_3 exposure), nutritional status (e.g., short breastfeeding duration), demographic factors (e.g., age, sex), and environmental conditions (e.g., indoor ventilation, secondhand smoke exposure, outdoor physical activity in polluted environments).[38,39] Biological predispositions, such as prematurity or a maternal history of asthma, also amplify vulnerability.[29,32,36,40]

Table 1. Characteristics and findings of the included studies

Author, Year	Study Design	Country	Sample Size	Population	Exposure/Intervention	Comparison	Outcomes
Bai et al., 2023[38]	Prospective birth cohort	China (Jinan)	6,501 children	Children (0-2 years)	O_3 exposure during pregnancy and first 2 years of life	Low vs high O_3 exposure levels	1) Cumulative incidence of asthma and wheezing (AW was 1.4% at age 2). 2) High O_3 exposure after birth (not before) was associated with AW (OR: 1.82, 95% CI: 1.08-3.12) 3) Individual effect of high O_3 exposure: HR 2.44 (95% CI: 1.53-3.89) 4) Two sensitive windows identified: 31-37 weeks before birth and 1-105 weeks after birth
Zhao et al., 2021[27]	Prospective birth cohort	Germany (Munich and Wesel)	915 children	Children (6-15 years) with spirometric measurements	Air pollution exposure (NO_2, $PM_{2.5}$, PM_{10}, PMcoarse, $PM_{2.5}$ absorbance) during first year of life	Comparison across pollution levels (using IQR increase)	1) ↓ FEV_1 z-scores from -0.012 to -0.023 per IQR increase in air pollutants 2) ↓ in FVC z-scores from -0.006 to -0.011 per IQR increase 3) Effects are more pronounced on FEV_1 than FVC 4) ↑ effect estimates for children with asthma, older maternal age, and breastfeeding <12 weeks
Usemann et al., 2024[29]	Cross-sectional population-based study	Switzerland	2,182	Children (6-17 years)	Fine-scale spatiotemporal model estimates of PM2.5 and NO2 linked with residential address histories	Different time windows: pregnancy (whole, trimesters), first year of life, preschool age	1) PM2.5 exposure during pregnancy is associated with ↓ lung function at school age 2) Per 10 µg/m³ increase in PM2.5 during pregnancy: FEV1 was 55 mL lower and FVC 62 mL lower in 12-year-olds 3) Associations were age-dependent (stronger in younger children) 4) PM2.5 exposure after birth is not associated with ↓ lung function 5) No association between NO2 exposure and lung function
Neophytou et al., 2023[5]	Cross-sectional study with distributed lag non-linear models	USA (Fresno-Clovis area, California)	222	Children ages 6-9 years	Monthly average exposure to PM2.5 and O3 interpolated using inverse distance-squared weighting based on residential history	Exposure during prenatal months and first three years of life	1) PM2.5 exposure during the prenatal period and first 3 years associated with lower FVC and FEV1 2) ↑ from 7.55 µg/m³ to 12.69 µg/m³ associated with 0.42 L lower FVC and 0.38 L lower FEV1 3) Second half of pregnancy identified as particularly influential window 4) Associations for ozone were weaker with confidence intervals including null
Wu et al., 2021[26]	Longitudinal panel study	China (Shanghai)	62 children	Children with repeated health measurements	Short-term exposure to PM2.5 and its constituents	Comparison across different levels of exposure to PM2.5 and constituents	1) PM2.5 exposure associated with ↑ TNF-α, FeNO levels (inflammation markers) and ↓ lung function 2) OC, EC, NO3-, and NH4+ had strongest associations with airway inflammation and lung function impairment 3) PM2.5 exposure associated with ↓ diversity in buccal mucosa bacterial community 4) Two bacterial phyla (Fusobacteria and Proteobacteria) were identified as differential microbes with PM2.5 exposure

Author, Year	Study Design	Country	Sample Size	Population	Exposure/Intervention	Comparison	Outcomes
Natarajan et al., 2022[30]	Cross-sectional study	India (Tamil Nadu)	100 children from 100 households	Children (6-13 years) from households using traditional biomass cook stoves	Exposure to Volatile Organic Compounds (VOCs) from biomass fuel use	Comparison across different kitchen configurations (T1-T4) and respiratory symptoms	1) Predominant VOCs: isoprene (113.33 µg/m³), benzene (24.83 µg/m³), toluene (15.93 µg/m³) 2) ↑ TVOC concentrations in detached kitchens (T4) 3) Children with self-reported respiratory symptoms had ↓ lung function 4) Positive correlation between PFT values and ATS questionnaire data 5) Children with frequent cough had lower FVC (78.0%) in households with ↑TVOC concentrations (389.46 µg/m³) 6) Hazard Index and cancer risk values showed children exposed to biomass smoke are prone to both carcinogenic and non-carcinogenic risks
Pu et al., 2021[40]	Time-series analysis	China	Children from 18 cities in Sichuan province with lower respiratory infections	Children <18 years with pneumonia and bronchitis hospitalizations	Exposure to size-specific particulate matter (PM2.5, PM10, PMC)	Comparison across different size-specific PM fractions and age groups, gender, and seasons	A 10 µg/m³ increment in PM2.5_lag03, PM10_lag06, and PMC_lag06 was associated with a 0.79%, 0.77%, and 2.33% increase in LRI hospitalizations respectively. Associations were stronger for PMC than for PM2.5 and PM10. Stronger effects observed in children aged 1-5 years.
Usemann et al., 2019[41]	Prospective birth cohort	Switzerland	232 children with complete follow-up (out of 304 recruited)	Healthy term-born infants followed up at six years	Exposure to moderate air pollution (NO2, O3, PM10) from pregnancy until follow-up at six years	Comparison across different exposure quartiles	Children exposed to the highest quartile of NO2 (>20 µg/m³) during pregnancy, the first, and second years of life had significant ↓ in FEV1. In the highest exposed group, per IQR increase in NO2, FEV1 ↓ by -1.07 z-score. Even at levels below WHO guidelines, higher NO2 exposure was associated with reduced lung function.
Mulat et al., 2024[42]	Prospective cohort	Ethiopia	280 (140 exposed, 140 unexposed)	Children (under 5 years)	Exposure to solid fuel (wood, crop residues, charcoal, animal dung) vs. clean fuel (electricity)	Comparison of HAZ scores between exposed and unexposed groups over 12-month follow-up	Exposure to indoor air pollution was inversely related to linear growth; higher HAZ scores observed in female children and those with fathers with higher education; lower HAZ scores in children with poor hygiene practices
Dutta et al., 2021[43]	Randomized controlled intervention trial	Nigeria	223 (113 ethanol stove, 110 firewood/kerosene)	Children (2-4 years) born to mothers who participated in cookstove intervention trial	Exposure to PM2.5 measured prenatally (maternal) and postnatally (household)	Comparison between ethanol stove vs. firewood/kerosene groups	↑ postnatal PM2.5 exposures are significantly associated with ↑ airway reactance at 5 Hz; stronger associations in preterm infants
Decrue et al., 2022[32]	Prospective birth cohort	Switzerland	771 (517 term, 254 preterm)	Children (5-9 years)	Exposure to PM10 and NO2 during pregnancy and after birth	Comparison between term and preterm infants (32-36 weeks)	Preterm infants showed higher susceptibility to prenatal air pollution exposure with stronger associations between PM10 and FEV1 deficits; FENO results showed differences in inflammatory/oxidative stress response
Morales et al., 2015[33]	Population-based prospective cohort	Spain	620	Preschoolers aged 4.5 years	Exposure to benzene and NO2 during specific trimesters of pregnancy and early postnatal life	Comparison across exposure tertiles	↑ exposure to benzene and NO2 during second trimester of pregnancy associated with ↓ lung function; stronger associations among allergic children and those of lower social class

Author, Year	Study Design	Country	Sample Size	Population	Exposure/Intervention	Comparison	Outcomes
Jedrychowski et al., 2015[44]	Prospective birth cohort	Poland	195	Non-asthmatic children aged 5-9 years of non-smoking mothers	Exposure to PAH during the second trimester of pregnancy and postnatal indoor/outdoor exposure at age 3	Comparison across exposure tertiles	Prenatal PAH exposure is associated with a FEV1 deficit of 53 mL and a FEF25-75 deficit of 164 mL; postnatal residential indoor PAH is associated with deficits of 59 mL FEV1 and 140 mL FEF25-75
Hazlehurst et al., 2021[36]	Prospective cohort	USA	1,469 children	Children from CANDLE and TIDES cohorts born 2007-2013	PM2.5 exposure during pseudo-glandular, canalicular, saccular, and alveolar phases	Exposure during different fetal lung development phases	Child asthma at age 4
Sherris et al., 2024[37]	Prospective cohort	USA	675 children	Children in CANDLE cohort born 2007-2011	PM2.5 during lung development phases	Exposure during different fetal lung development phases	FEV1, FVC, FEV1/FVC at age 8-9
Hemstock et al., 2024[31]	Prospective cohort	Australia	115 infants	Healthy infants exposed to a coal mine fire	PM2.5 from the coal mine fire	Levels of PM2.5 exposure	Lung function at 3 and 7 years
Gisler et al., 2022[45]	Prospective cohort	Switzerland	401 infants	Healthy infants from BILD cohort	Tree and grass pollen, PM2.5, NO2	Exposure levels to pollen, modifying effects of PM2.5	Respiratory symptoms in first year
Majewska et al., 2018[28]	Prospective cohort	Poland	294 children	Non-asthmatic children from Krakow birth cohort	PM2.5, PAH during pregnancy	Exposure levels to PM2.5 and PAH	FVC, FEV1, FEF25-75 annually ages 4-9
Lepeule et al., 2023[46]	Prospective cohort	France	391 mother-child pairs	Mothers from SEPAGES cohort; full-term infants assessed at 7 weeks	Personal exposure to PM2.5 and NO2 during pregnancy measured by wearable devices	Comparison across exposure levels and by sex	Tidal breathing analysis and nitrogen multiple breath washout test; functional residual capacity (FRC)
Lee et al., 2018[47]	Prospective cohort	USA	191 mother-child pairs	Children from ACCESS cohort assessed at age 7 years	Prenatal PM2.5 exposure estimated using satellite-based spatiotemporal model	Comparison across exposure levels and by sex	Spirometry outcomes (FEV1, FVC); GSTP1 methylation in nasal epithelial cells
Hsu et al., 2015[48]	Prospective cohort	USA	736 full-term children	Children from ACCESS cohort followed to age 6 years	Prenatal PM2.5 exposure estimated using satellite-based model	Comparison across exposure timing and by sex	Physician-diagnosed asthma by age 6 years
Bose et al., 2018[49]	Prospective cohort	USA	191 mother-child pairs	Children from ACCESS cohort assessed at age 7 years	Prenatal nitrate (NO3-) exposure estimated using hybrid chemical transport models	Comparison across exposure timing and by sex	Spirometry outcomes (FEV1, FVC, FEV1/FVC ratio, FEF25-75)
Hsu et al., 2023[34]	Prospective cohort	USA	198 mother-child dyads	Children from ACCESS cohort assessed at mean age 6.99 years	Multi-pollutant mixture exposure (NO2, O3, EC, OC, NO3-, SO42-, NH4+)	Comparison across exposure timing, pollutant types, and by sex	Spirometry outcomes (FEV1, FVC, FEV1/FVC ratio, FEF25-75)
Lavigne et al., 2019[35]	Longitudinal cohort study	Canada (Toronto)	160,641 singleton live births	Children followed from birth to age 6	UFP, PM2.5, NO2 exposure during	Exposure levels in different time periods (trimesters) and	Childhood asthma incidence up to age 6

Author, Year	Study Design	Country	Sample Size	Population	Exposure/Intervention	Comparison	Outcomes
					prenatal and postnatal periods	different pollutant concentrations	
Goshen et al., 2020[50]	Retrospective population-based cohort study	Israel	57,331 deliveries	Mothers and infants born between 2004-2012	PM2.5 exposure during pregnancy	High vs. low traffic areas	LRTI hospitalizations during first year of life
Zhou et al., 2022[51]	Panel study	China (Shanghai)	70 children	Children with asthma (4-14 years)	PM2.5, NO2, and temperature	Different exposure levels	Lung function (FVC, FEV1, PEF)
Christian et al., 2022[52]	Cross-sectional study	Australia (Perth)	478 children from 22 ECEC centers	Children attending Early Childhood Education and Care centers	PM2.5 exposure at childcare centers	High vs. low traffic areas	Physical activity levels, outdoor play time
Hu et al., 2024[39]	Prospective birth cohort study	Mexico	468 mother-child pairs	Children (4-8 years) from PROGRESS cohort	PM2.5, NO2, and temperature exposure from conception to age 4	Different exposure windows and levels	Childhood asthma and wheeze at ages 4-6 and 7-8 years

Table 2. ROBINS-I assessment

Author	Domain 1: Confounding	Domain 2: Selection of participants	Domain 3: Classification of interventions	Domain 4: Deviations from intended interventions	Domain 5: Missing data	Domain 6: Measurement of outcomes	Domain 7: Selection of reported results	Overall Risk of Bias
Bai et al.[38]	Moderate Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Moderate Risk
Zhao et al.[27]	Moderate Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Moderate Risk
Neophytou et al.[5]	Moderate Risk	Low Risk	Low Risk	Low Risk	Moderate Risk	Low Risk	Low Risk	Moderate Risk
Usemann et al.[29]	Moderate Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Moderate Risk
Wu et al.[26]	Moderate Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Moderate Risk
Natarajan et al.[30]	Moderate Risk	Moderate Risk	Low Risk	Low Risk	Low Risk	Moderate Risk	Moderate Risk	Moderate Risk
Pu et al.[40]	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk
Usemann et al.[41]	Low Risk	Low Risk	Low Risk	Low Risk	Moderate Risk	Low Risk	Low Risk	Low Risk
Olutola et al.[53]	Moderate Risk	Moderate Risk	Moderate Risk	Low Risk	Low Risk	Serious Risk	Moderate Risk	Serious Risk
Mulat et al.[42]	Moderate risk	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk	Moderate risk
Dutta et al.[43]	Moderate risk	Moderate risk	Low risk	Low risk	Moderate risk	Low risk	Low risk	Moderate risk
Decrue et al.[32]	Low risk	Low risk	Low risk	Low risk	Moderate risk	Low risk	Low risk	Moderate risk
Morales et al.[33]	Moderate risk	Moderate risk	Low risk	Low risk	Moderate risk	Low risk	Low risk	Moderate risk
Jedrychowski et al.[44]	Moderate risk	Moderate risk	Low risk	Low risk	Moderate risk	Low risk	Low risk	Moderate risk
Hemstock et al.[31]	Moderate risk	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk	Moderate risk
Majewska et al.[28]	Moderate risk	Moderate risk	Low risk	Low risk	Moderate risk	Low risk	Low risk	Moderate risk
Hazlehurst et al.[36]	Low risk	Moderate risk	Low risk	Low risk	Moderate risk	Low risk	Low risk	Moderate risk
Gisler et al.[45]	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk
Sherris et al.[37]	Low risk	Moderate risk	Low risk	Low risk	Low risk	Low risk	Low risk	Moderate risk

Author	Domain 1: Confounding	Domain 2: Selection of participants	Domain 3: Classification of interventions	Domain 4: Deviations from intended interventions	Domain 5: Missing data	Domain 6: Measurement of outcomes	Domain 7: Selection of reported results	Overall Risk of Bias
Lepeule et al.[46]	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk
Lee et al.[47]	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk
Hsu et al.[48]	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk
Bose et al.[49]	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk
Hsu et al.[34]	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk	Low Risk
Lavigne et al.[35]	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk	Low risk
Goshen et al.[50]	Low risk	Moderate risk	Low risk	Low risk	Low risk	Low risk	Low risk	Moderate risk
Zhou et al.[51]	Moderate risk	Moderate risk	Low risk	Low risk	Low risk	Low risk	Low risk	Moderate risk
Christian et al.[52]	Moderate risk	Moderate risk	Low risk	Low risk	Moderate risk	Low risk	Low risk	Moderate risk
Hu et al.[39]	Low risk	Low risk	Low risk	Low risk	Low risk	Moderate risk	Low risk	Moderate risk

Table 3. Mechanisms and effects of air pollution on lung development in children

Air Pollution Impact	Mechanism	Clinical Effects	Scientific Evidence	Citations
Changes in Respiratory Tract Permeability	The presence of O_3 causes dysfunction of the small airways, resulting in changes in airway permeability and respiratory inflammation	Respiratory tract inflammation and hyperactivity	O_3 as a source of oxidative stress alters the Th1/Th2 immune response and causes activation of NF-κB	Bai et al., 2023[38]
Limitation of Airway Size	Air pollution is more likely to limit the size of the airways rather than to limit lung volume	The decrease in FEV1 is more significant than that in FVC	The research findings are partly consistent with the PIAMA cohort study, which reported the adverse effects of air pollution on increased FEV1	Zhao et al., 2021[27]
In Utero Developmental Disorder	PM2.5 exposure during pregnancy is negatively associated with FEV1 and FVC in early school age	Decreased lung function	Small particles (black carbon) can pass through the placenta and accumulate in the fetus	Usemann et al., 2024[29]
Decreased Lung Capacity	Increased exposure to PM2.5 is associated with lower lung function	Decrease in FVC and FEV1	Studies show an increase in the IQR (12 μg/m³) of sub-chronic PM2.5 exposure associated with a decrease of 103 mL in FVC and 86 mL in FEV1	Wu et al., 2021[26]
Increased Risk of Respiratory Infection	The increase of VOCs indoors, particularly benzene, ethylbenzene, and toluene, causes neurogenic inflammation	Increased asthma risk by 2 to 3 times	About 25% of respiratory diseases are caused by exposure to indoor air pollution, even at low levels of exposure	Natarajan et al., 2022[30]
Lung Damage and Stunted Growth	The impact of air pollution in early life increases the risk of acute and chronic diseases	Lung damage and impaired growth	Children are susceptible to air pollution	Pu et al., 2021[40]
Harmful Effects on Lung Function Development	Pollution has detrimental effects on lung development, especially in areas with higher levels of exposure	Decline in lung function	The harmful effects of some pollutants may seem small, but the decline in lung function may be most significant for those who start with impaired lung function at an early age	Usemann et al., 2019[41]
Mitochondrial Dysfunction	Air pollution exposure leads to increased oxidative stress, systemic inflammation, and altered immune function	Mitochondrial dysfunction, telomere shortening, and inflammation	Small air pollution particles are inhaled into the lungs, infiltrate the bloodstream, and reach various body organs	Mulat et al., 2024[42]
Changes in Structure and Function of the Lungs	The development of the lungs occurs in five stages in children, starting before birth and continuing into childhood	Changes in lung structure and function	The first two years are crucial for lung development as the number of air sacs increases exponentially	Dutta et al., 2021[43]

Air Pollution Impact	Mechanism	Clinical Effects	Scientific Evidence	Citations
Negative Impact on Respiratory Health	High levels of air pollution adversely affect respiratory health in children and adults	Decreased lung capacity and breathing problems	The level of air pollution is low to moderate, and its impact on lung function and respiratory symptoms in infants and children has become the subject of recent research	Decrue et al., 2022[32]
Impairment of Lung Formation & Development	During the early stages of rapid development and growth, the lungs may not be fully developed and could be very vulnerable to the permanent harmful effects of environmental factors	Persistent pulmonary function deficiency	The effects of prenatal and postnatal exposure to air pollution on lung function in preschool-aged children have yet to be explored	Morales et al., 2015[33]
Respiratory Tract Dysfunction	High prenatal PAH exposure is associated with reduced FEV05, FEV1, and FEF25-75%, but not FVC	Impairment of respiratory function	PAH prenatal exposure inhibits the full development of respiratory function	Jedrychowski et al., 2015[44]
Abnormality in Lung Growth Trajectory	The early stages of life are a critical window for the development and growth of the lungs	Deviation in the growth path of healthy lungs	Children are very vulnerable to the harmful effects of air pollution exposure due to higher breathing rates and less developed airways	Hemstock et al., 2024[31]
Decrease in FVC and FEV1	High prenatal exposure to PM2.5 and PAH in the air is associated with lower FVC and FEV1	Significant impairment of lung function	No significant difference was observed in the rate of increase over time related to prenatal exposure to PM2.5 and PAH	Majewska et al., 2018[28]
Epigenetic Changes	The prenatal period is a critical exposure window when the developing respiratory and immune systems are vulnerable to the damaging effects of environmental toxins	Increased risk of asthma	The mechanism of air pollution may contribute to the development of asthma, including increased maternal systemic inflammation, endothelial changes, and oxidative stress	Hazlehurst et al., 2021[36]
Small Respiratory Tract dysfunction	Air pollution exposure during early life can increase the risk of chronic respiratory diseases	Small respiratory tract dysfunction	PM2.5 exposure during pregnancy can suppress the expression of genes related to lung development, negatively affecting lung volume and alveolarization	Sherris et al., 2024[37]
Decreased Lung Volume	Higher PM2.5 exposure during pregnancy is associated with lower lung volume	Decreased lung volume	Female children born to mothers with higher exposure to PM2.5 during pregnancy are at a higher risk of experiencing lower lung volumes	Lepeule et al., 2023[46]
Increased risk of FEV1 decline	Children exposed to higher prenatal PM2.5 levels have an increased risk of reduced FEV1 z-score	Decreased FEV1	BDLIM identifies statistically significant sensitive exposure windows (36-39 weeks of pregnancy)	Lee et al., 2018[47]
Initial Decrease of Lung Function	Air pollution exposure from early life and prenatal periods is associated with decreased lung function	Decrease in lung function	Identifying lung function in children is very important, as early deficits can increase the risk of respiratory diseases in the future	Bose et al., 2018[49]
Increase in Fetal Oxidative Stress	Air pollution exposure increases oxidative stress in the fetus, affecting gene expression and essential physiological events for lung maturation	Increased oxidative stress in the fetus	The effects of prenatal air pollution can have sex-specific impacts, with male children being more vulnerable	Hsu et al., 2015[48]
Changes in Lung Growth and Development Pathways	The impact of air pollution during pregnancy and early life can disrupt the growth and development of the fetus's lungs	Decreased lung function	New findings indicate the role of nitrates (NO_3^-), a significant component of PM2.5, and surface-level ozone (O_3)	Hsu et al., 2023[34]
Increase in Asthma Incidents in Children	Increased inflammation and heightened sensitivity of the airways can affect the immune and respiratory systems of the developing fetus	Increase in asthma incidents among children	The weeks of exposure to air pollution correspond to an essential phase of lung development	Lavigne et al., 2019[35]
Decreased Lung Function in Children with Asthma	Inhaled PM2.5 can trigger oxidative stress and systemic inflammation	Decrease in pulmonary performance	PM2.5 has a small diameter, which results in a larger surface area and more excellent absorption of allergens	Zhou et al., 2022[51]

Table 4. Long-term effects of air pollution on the physical growth of young children

The Impact of Air Pollution	Explanation	Citation
Respiratory Problems and Cancer Risk	Indoor air pollution from biomass burning causes respiratory symptoms (chest congestion, wheezing, allergies) and reduced lung function (lower FVC in children). Long-term exposure also increases carcinogenic and non-carcinogenic risks in children.	Natarajan et al[30]

The Impact of Air Pollution	Explanation	Citation
Growth Impairment and Systemic Health Effects	Household air pollution contributes to stunting by hindering linear growth and increasing recurrent respiratory infections. It leads to systemic inflammation, immune dysfunction, metabolic changes, nutritional imbalances, and vitamin D deficiency. Stunting is associated with impaired cognitive development, lower productivity, increased risk of infections, future non-communicable diseases, and possible obstetric complications in women.	Mulat et al[42]

Table 3: Risk factors modifying the impact of air pollution exposure in children

Risk Factor	Modification Mechanism	Scientific Evidence	Citation
Frequently Opening Windows	Increasing the chances of high O_3 exposure in children	The P value of the interaction between frequent window openings and high O_3 exposure doses is less than 0.001	Bai et al., 2023[38]
Duration of Breastfeeding	The immune factors and cytokines in breast milk may protect against oxidative stress pathways and systemic pro-inflammatory responses	The effects of several air pollutants are significantly higher for children who were breastfed for less than 12 weeks	Zhao et al., 2021[27]
Age and Gender	The impact of air pollution on lung function differs for different ages	The association depends on age, as it is more potent in younger children and weaker in older children	Usemann et al., 2024[29]
Socioeconomic Status	Populations with lower socioeconomic indicators may be more vulnerable	Minority races/ethnicities, lower socio-economic indicators, and high poverty levels characterize this population	Neophytou et al., 2023[5]
Household Ventilation	Household arrangements related to ventilation impact children's respiratory health	Significant changes in lung function were observed among children living in various types of households	Natarajan et al., 2022[30]
Age of The Child	Children under 6 have smaller airway diameters and weaker immune systems	Three types of specific PM are significantly associated with hospitalizations of children aged 1-5 years for pneumonia and bronchitis	Pu et al., 2021[40]
Concentration Level of NO_2	Children in the highest exposure group experienced a significant decline in FEV1 by school age	Children exposed to lower levels of NO_2 do not experience a decline in lung function by school age	Usemann et al., 2019[41]
Carrying a Child on the Back While Cooking	Children are often carried in their mothers' laps or on their backs while they cook	Children's rates of respiration, absorption, and retention of toxic substances from the air are higher than those of adults	Mulat et al., 2024[42]
Sociodemographic and Anthropometric Variables	Groups (intervention or control) and sociodemographic, anthropometric, and obstetric variables, including age, gender, weight, height, gestational age, and birth weight	Adjustments for these variables when analysing the relationship between exposure (levels of PM2.5) and lung function	Dutta et al., 2021[43]
Prematurity	Preterm infants may be more vulnerable to the effects of air pollution	The impact of air pollution on postnatal lung function changes in premature infants is greater	Decrue et al., 2022[32]
Socioeconomic Status	Socioeconomic status can act as a modifier of the potential effects of harmful air pollution on lung function	A more significant deficit in lung function has been found in relation to higher levels of air pollutants among preschool children from lower and middle socioeconomic groups	Morales et al., 2015[33]
Postnatal Care Centre	The prenatal effects of PAH exposure on lung growth persist until the pre-adolescent stage	The harmful effects of postnatal residential exposure to PAHs on lung function can be observed	Jedrychowski et al., 2015[44]
Smoking during Pregnancy for Mothers	Maternal smoking during pregnancy has been consistently identified as a significant risk factor for impaired respiratory function in children	Determinants driving lung changes include a natural decline over time due to aging and several childhood risk factors	Hemstock et al., 2024[31]
Gender and Height	The pulmonary function in females is generally lower than that observed in males, which may be attributed to various anatomical and physiological discrepancies	Only sex and height increases related to the spirometric function shape were studied over time	Majewska et al., 2018[28]
The History of Asma in Mother	Modification of effects by the mother's asthma history observed	The relationship between PM2.5 and asthma has been observed among children without a history of asthma in their mothers	Hazlehurst et al., 2021[36]

Risk Factor	Modification Mechanism	Scientific Evidence	Citation
Gender	The findings suggest that male fetuses may be more susceptible to the adverse effects associated with prenatal exposure to PM2.5 particulate matter	Male fetuses may be at greater risk of prenatal oxidative injury and, therefore, may exhibit an exaggerated response to in utero air pollution exposure	Lee et al., 2018[47]
Outdoor Physical Activity	Moderate to vigorous physical activity involves a higher level of breathing	Children living in areas with high pollution who play outdoor sports have an increased risk of asthma events	Christian et al., 2022[52]
Gender and Age	Gender and age can modify the effects of PM2.5 on lung function	A stronger relationship between PM2.5 and changes in lung function was found in boys and the preschool age group (under 6 years)	Zhou et al., 2022[51]
Differences in Fetal Lung Development	Sex hormones largely influence the specific differences in sex in the development of fetal lungs in males and females	Female fetuses typically develop smaller lungs and alveolar surface areas than male fetuses, but they have airways with a larger diameter	Hu et al., 2024[39]

Socioeconomic factors—including poverty and minority status—further differentiate the respiratory burden from air pollution.[5,33] This underscores the importance of integrating individual, behavioral, and contextual variables when assessing pediatric respiratory outcomes.

3.2. Physical growth impact

Recent investigations have elucidated that exposure to air pollution, particularly from indoor biomass combustion, can impair physical growth during early childhood (Table 4). Natarajan et al. (2022) reported a strong association between elevated total VOCs (TVOCs) and both diminished lung capacity and increased carcinogenic risk. Specifically, they found that 78% of children who experienced frequent coughing had significantly reduced FVC, residing in areas with TVOC levels averaging 389.46 μg/m³ ($p < 0.003$). Cumulative hazard indices also indicated elevated lifetime cancer risks in these populations.[30]

Mulat et al. (2024) described the pathophysiological mechanisms by which particulate matter induces systemic inflammation and immune dysfunction, leading to stunting. These changes are associated with disrupted metabolic pathways, vitamin D deficiency, and increased vulnerability to respiratory infections. Moreover, these systemic effects can result in long-term cognitive impairment and a higher risk of non-communicable diseases in adulthood.[42]

Biomass smoke exposure during cooking and poor household ventilation practices contribute to this cascade. For example, carrying children while cooking with biomass fuels was identified as a risk factor for both respiratory and growth-related health impacts.[42]

Thus, air pollution is not only a respiratory hazard but also a critical determinant of overall physical development and long-term health, particularly when exposure occurs during the first 1,000 days of life.

4. Discussion

This study investigates the implications of air pollution on pulmonary development and early childhood growth patterns while identifying potential modifiers of these effects. The primary findings reveal that exposure to air pollutants during prenatal and early postnatal periods significantly impairs lung development in pediatric populations, with potential long-term ramifications extending into adolescence and adulthood. These results underscore the need for heightened awareness and intervention strategies to mitigate air pollution exposure, particularly during critical periods of lung development.

The present study explains the detrimental impact of various air pollutants, including PM2.5, nitrogen dioxide (NO_2), ozone (O_3), and polycyclic aromatic hydrocarbons (PAHs), on pulmonary function, as evidenced by significant impairments in spirometric indices such as forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and forced expiratory flow at 25-75% of FVC (FEF25-75%). These findings are congruent with the study's objective to examine the long-term ramifications of air pollution on pulmonary development and growth trajectories in early childhood. The outcomes substantiate the "Developmental Origins of Health and Disease" (DOHaD) hypothesis, underscoring the critical role of environmental exposures during pivotal developmental stages in shaping long-term health outcomes.

This study confirms previous epidemiological research indicating a significant association between ambient air pollution and impaired lung function

development in pediatric populations. Zhao et al. (2021) and Usemann et al. (2024) observed that exposure to air pollutants during the first year of life correlated with annual declines in FEV1 and FVC z-scores, effects that persisted into adolescence.[27,29] Furthermore, Neophytou et al. (2023) reported that increased exposure to PM2.5, rising from 7.55 µg/m³ to 12.69 µg/m³, was associated with reductions of 0.42 L in FVC and 0.38 L in FEV1 among children.[5] These findings align with earlier contributions from Urman et al. (2015) and Teng et al. (2022), which underscore air quality's critical role in children's pulmonary development.[14,15]

This study's novel contribution is the comprehensive identification of biological mechanisms underlying the effects of air pollution on lung development and child growth. These mechanisms include oxidative stress, systemic inflammation, changes in airway permeability, mitochondrial dysfunction, and epigenetic alterations. Wu et al. (2021) found that PM2.5 exposure was associated with elevated inflammatory markers such as TNF-α and FeNO and changes in the buccal mucosal microbiota community. These findings enhance our understanding of the biological pathways linking air pollution exposure to impaired lung development.[26]

The study explains the long-term ramifications of air pollution on pediatric physical growth, with a particular emphasis on stunting. Mulat et al. (2024) establish a compelling association between household air pollution (HAP) exposure and impaired linear growth in children, which correlates with increased morbidity risk, cognitive development setbacks, and heightened susceptibility to infectious diseases. The pathological mechanisms include systemic inflammation, recurrent respiratory infections, vitamin D deficiency, and metabolic dysregulation. These findings significantly contribute to the existing literature on the comprehensive effects of air pollution on child health, broadening the scope beyond the conventional emphasis on respiratory pathology.[42]

Examining risk factors elucidating the impact of air pollution on pediatric populations (Table 5) reveals intricate interactions among individual, behavioral, environmental, and socioeconomic determinants. Key variables such as age, sex, socioeconomic status, gestational prematurity, and maternal history of asthma are pivotal in modulating children's susceptibility to the deleterious effects of air pollution. Research conducted by Decrue et al. (2022) indicates that premature infants exhibit heightened vulnerability to the adverse effects of prenatal air pollution exposure, notably demonstrating a significant correlation between PM10 levels and deficits in forced expiratory volume in one second (FEV1).[32] Furthermore, investigations by Lee et al. (2018) and Hu et al. (2024) have identified sex-based discrepancies in susceptibility; male infants may demonstrate increased risk associated with prenatal exposure to PM2.5, potentially attributable to differential patterns in fetal lung development.[39,47] These findings underscore the necessity for a nuanced understanding of the multifactorial nature of air pollution exposure and its implications for pediatric health.

These findings hold significant implications for the theoretical advancement of environmental and pediatric health research. The results support the "critical windows of exposure" hypothesis, which posits that specific developmental periods, particularly the second trimester of gestation and the first two years of postnatal life, represent intervals during which exposure to air pollution profoundly affects health outcomes. Notably, Morales et al. (2015) and Bai et al. (2023) have identified sensitive

exposure windows, specifically during the second trimester, and in the gestational period between 31 to 37 weeks and the postnatal period spanning weeks 1 to 105.[33,38]

From a clinical standpoint, this study underscores the critical need for early interventions to minimize children's exposure to air pollution. The findings indicate that exposure to even modest levels of air pollutants can harm lung development, emphasizing the necessity for more stringent air quality regulations. Usemann et al. (2019) demonstrated that nitrogen dioxide (NO_2) exposure, even at levels below the World Health Organization (WHO) guidelines, is associated with compromised lung function. This highlights the imperative for ongoing initiatives to mitigate environmental pollutant levels to safeguard respiratory health in pediatric populations.[41]

From a policy perspective, this study underscores the necessity of developing targeted air pollution reduction strategies to safeguard vulnerable populations, notably pregnant women and children. Evidence suggests that the implementation of policies promoting the adoption of cleaner cooking fuels, enhancing home ventilation systems, and mitigating outdoor air pollution in proximity to both educational institutions and residential areas can substantially improve respiratory health outcomes in children. Research conducted by Natarajan et al. (2022) and Mulat et al. (2024) indicates that pediatric populations exposed to volatile organic compounds (VOCs) from indoor biomass combustion exhibit heightened respiratory symptoms and stunted growth. These findings highlight the critical need for interventions to reduce indoor air pollution to protect the health and development of children.[30,42]

Recent findings indicate a significant age-dependent relationship between air pollution exposure and lung function, with younger children exhibiting more pronounced associations than older children, as documented by Usemann et al. (2024). This suggests the potential emergence of adaptive or compensatory mechanisms throughout developmental stages, or it may highlight the heightened vulnerability of early lung development to the deleterious effects of air pollution. Further investigation is warranted to elucidate the underlying mechanisms contributing to this phenomenon.[29]

This study demonstrates key methodological strengths, including the use of the PICOS framework, comprehensive database searches, and quality assessment with the ROBINS-I tool. Most included studies (96.6%) showed good quality with low to moderate risk of bias. However, limitations include potential confounding from observational designs and heterogeneity in exposure and outcome measures, which may hinder comparability. Some relevant studies may have been missed due to language or inclusion criteria. Several knowledge gaps remain. It is unclear whether early-life exposure to air pollution causes permanent or reversible effects on lung development. More research is needed on critical exposure periods and the underlying biological mechanisms, including epigenetic and metabolic pathways. Future research should prioritize long-term cohort studies and evaluate the effectiveness of exposure reduction strategies. Interdisciplinary approaches are essential to understand the complex relationship between environmental exposures and child health. Overall, early exposure to air pollution adversely affects lung development in children, highlighting the need for targeted policies and interventions, especially for vulnerable groups like pregnant women and children.

5. Conclusion

This is the first systematic review to integrate findings on both respiratory and

physical growth outcomes from air pollution exposure in early childhood. High-quality studies highlight the significant impact of prenatal and early-life exposure to air pollution—particularly PM2.5, NO_2, O_3, and PAHs—on lung function and child growth. Exposure during critical periods, such as the second trimester and first two years of life, impairs lung development (e.g., FEV1, FVC, FEF25–75%) and increases the risk of stunting, cognitive delays, and future non-communicable diseases. These effects are mediated by oxidative stress, inflammation, and epigenetic changes. Risk is further influenced by factors like age, sex, prematurity, breastfeeding duration, ventilation quality, and socioeconomic status. The findings validate the concept of "critical windows" and DOHaD, emphasizing the need for stricter air quality regulations and targeted interventions—such as cleaner fuels and better indoor air management—to protect pregnant women and children. While insightful, the study is limited by observational designs, exposure heterogeneity, and language bias, with gaps remaining in understanding long-term outcomes and reversibility. Future research should focus on longitudinal and interventional studies to inform effective, evidence-based public health strategies.

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