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Urban Environmental Exposures, Air Pollution, and Maternal–Offspring Outcomes: *A Systematic Review*

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ABSTRACT

Background: Urban ambient air pollution and various co-occurring environmental stressors during pregnancy contribute to systemic inflammation, oxidative stress, endothelial dysfunction, and impaired placental perfusion, thereby increasing the risk of adverse maternal and child health outcomes. Therefore, this study aims to systematically synthesize the evidence on the associations between ambient air pollution and co-occurring urban environmental exposures and pregnancy complications affecting maternal and child health, based on the available evidence.
Methods: Following PRISMA 2020 guidelines and a defined PECO framework, comprehensive searches were executed on Scopus and ScienceDirect (April 23, 2026; updated May 06, 2026) for literature published between 2022 and 2025. Of 3,547 records identified, 20 articles met the inclusion criteria, comprising 16 empirical observational studies and 4 supporting conceptual articles. Study quality and risk of bias were assessed using the Newcastle-Ottawa Scale and JBI critical appraisal tools.
Results: Moderate-certainty evidence indicated that exposure to PM2.5 and NO2 during the preconception and early-to-mid pregnancy periods increased the risks of gestational diabetes mellitus, hypertensive disorders of pregnancy, low birth weight, and preterm birth. Multi-pollutant models consistently provided stronger risk estimates than single-pollutant models. Evidence linking air pollution with gestational hypothyroidism, postpartum mental distress, and offspring neurodevelopmental outcomes remained limited and heterogeneous.
Conclusion: Ambient air pollution and related urban environmental exposures are important determinants of maternal and child health. Integrating environmental exposure assessment into antenatal care and strengthening urban emission reduction policies are essential to reduce pregnancy-related health risks.

Keywords: Ambient air pollution ; Urban environment ; Pregnancy complications ; Birth outcomes ; Neurodevelopment ; $PM_{2,5}$

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Introduction

Ambient air pollution within rapidly expanding urban environments represents one of the most critical global environmental health threats. Modern urbanization creates dense exposure landscapes dominated by complex mixtures of fine particulate matter ($PM_{2.5}$), coarse particulate matter (PM_{10}), nitrogen dioxide (NO_2), ground-level ozone (O_3), sulfur dioxide (SO_2), carbon monoxide (CO), black carbon, and toxic heavy metals (Pb, Cd). These exposures do not occur in isolation; rather, urban populations simultaneously experience co-occurring environmental stressors, including microclimate heat islands, extreme temperature fluctuations, wildfire smoke plumes, and unequal spatial access to protective urban green space[1-9].

From a pathophysiological perspective, pregnancy represents a state of heightened maternal susceptibility to environmental insults. Maternal physiological adaptations including increased minute ventilation, elevated oxygen consumption, expanded plasma volume, and specialized cardiovascular restructuring amplify total internal dose and systemic reactivity to inhaled toxic compounds. Inhaled fine particulates and gaseous pollutants irritate the respiratory epithelium, activate alveolar macrophages, and provoke pulmonary oxidative stress. This localized pulmonary reaction spills over into the maternal systemic circulation, unleashing pro-inflammatory cytokines, inducing endothelial cell dysfunction, and altering vascular tone. Because the placenta serves as the vital physiological interface for nutrient exchange, oxygen transport, and hormonal regulation between mother and fetus, maternal systemic inflammation and altered uteroplacental perfusion can directly impair placental angiogenesis and placental barrier integrity[10-14].

Although an extensive body of epidemiological literature has linked individual air pollutants to isolated perinatal outcomes, significant knowledge gaps remain. First, historical epidemiological studies overwhelmingly relied on single-pollutant models, failing to capture real-world synergistic or additive interactions inherent to urban multi-pollutant mixtures and co-occurring environmental stressors. Second, existing systematic reviews have predominantly aggregated broad gestational exposure averages, thereby masking brief, highly sensitive biological exposure windows (such as preconception, early implantation, organogenesis, or late trimester growth phases). Third, syntheses often fail to distinguish maternal pregnancy complications from adverse birth outcomes and long-term child neurodevelopmental trajectories within a unified framework [4,15-18].

To address these gaps, this systematic review synthesizes recent empirical and biological evidence (2022–2025) regarding urban ambient air pollution and co-occurring environmental exposures. The primary research questions guiding this review are: (1) Which urban air pollutants and environmental stressors demonstrate the most consistent associations with specific maternal, fetal, and child health domains? (2) Which gestational or preconception windows represent periods of peak biological vulnerability? (3) What is the comparative performance of multi-pollutant exposure models versus traditional single-pollutant models? and (4) What is the certainty of evidence across maternal, perinatal, and offspring neurodevelopmental outcome domains?.

Method

A. Reporting Guidelines & Conceptual Scope

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA 2020) statement and Joanna Briggs Institute (JBI) operational guidelines[21-22].

For the purposes of this synthesis, urban environment is defined as densely built human settlements characterized by high population density, industrial activity, commercial transport, and concentrated infrastructure. Ambient air pollution refers to outdoor atmospheric contamination resulting from mobile vehicular emissions, point-source industrial activity, fossil fuel combustion,

biomass burning, and secondary photochemical reactions. Co-occurring environmental factors (e.g., extreme heat, wildfire smoke, urban green space) were synthesized as contextual environmental determinants or effect modifiers rather than chemical air pollutants *per se*.

B. PECO framework

Review questions are formulated using the PECO framework. Such populations include pregnant women, fetuses, infants, or children with information exposure during the preconception, prenatal, or early life periods. Exposures include ambient air pollution or exposure to urban environments associated with air quality. Comparators are lower levels of exposure, different exposure periods, or groups with more protective environmental characteristics. Outcomes include maternal complications, birth outcomes, neurodevelopmental outcomes, and biological biomarkers.

Table 1. PECO Framework Guiding Review Eligibility

Components	Operational definition
Population	Human populations evaluated during preconception, pregnancy, fetal development, infancy, childhood, or early adolescence.
Exposure	Quantitative or modeled outdoor ambient air pollutants (PM2.5, PM10, NO2, SO2, O3, CO, black carbon, heavy metals) and co-occurring urban environmental stressors (wildfire smoke, extreme heat, green space metrics).
Comparator	Reference groups exposed to lower pollutant concentration levels, alternative temporal exposure windows, or higher levels of protective environmental buffers (e.g., higher plant species richness).
Outcomes	1. Maternal Pregnancy Complications: Gestational Diabetes Mellitus (GDM), Hypertensive Disorders of Pregnancy (HDP)/Preeclampsia, Gestational Hypothyroidism, Postpartum Mental Health. 2. Fetal & Birth Outcomes: Low Birth Weight (LBW), Small-for-Gestational-Age (SGA), Preterm Birth (PTB), fetal growth velocity. 3. Biological Biomarkers: Inflammatory proteins (CRP), oxidative stress markers (8-OHdG, MDA), maternal immune markers. 4. Long-Term Child Outcomes: Autism Spectrum Disorder (ASD), pediatric epilepsy, adolescent white matter connectivity.

C. Data sources and search strategies

The literature search was focused on Scopus and ScienceDirect. The primary search was conducted on April 23, 2026 and the search update or validation was conducted on May 06, 2026. The main publication period is limited to 2022-2025. The search strategy combines three groups of concepts: ambient or urban air exposure, gestation, and adverse outcomes.

The search string is constructed by combining the concepts of ambient/urban air exposure, gestational period, and adverse output. The main strings used are: ("ambient air pollution" OR "urban air pollution" OR "traffic-related air pollution" OR "$PM_{2.5}$" OR "PM_{10}" OR "nitrogen dioxide" OR "NO_2" OR "ozone" OR "O_3" OR "sulfur dioxide" OR "SO_2" OR "carbon monoxide" OR "black carbon" OR "wildfire smoke" OR "heavy metals" OR "extreme heat") AND ("pregnancy" OR "pregnant woman" OR "mother" OR "prenatal exposure" OR "preconception" OR "fetus" OR "birth cohort") AND ("outcomes adverse pregnancy" OR "adverse birth result" OR "adverse birth result" OR "adverse birth result" OR "adverse birth result" OR "adverse birth result" OR "adverse birth result" OR "adverse birth result" OR "gestational diabetes" OR "pregnancy hypertension disorder" OR "preeclampsia" OR "premature birth" OR "low birth weight" OR "small for gestational age" OR "fetal growth" OR "autism" OR "neurological development" OR "oxidative stress" OR "inflammation").

To expand the context of urban multi-exposure, the supporting strings used are: ("urban environment" OR "urban population" OR "urban area") AND ("air pollution" OR " $PM_{2.5}$" OR "NO_2" OR "traffic-related

pollution") AND ("residential conditions" OR "indoor air quality" OR "density" OR "residential environment") AND ("green space" OR "urban green space" OR "plant species richness") AND ("pregnancy" OR "birth outcomes" OR "maternal health" OR "fetal growth" OR "premature birth").

Table 2. Summary of search and selection strategies

Stages	Remarks
Databases	Scopus and ScienceDirect.
Search date	The main search is April 23, 2026; update/validation May 06, 2026.
Publication range	Main article: 2022-2025.
Language	Articles in English with adequate methods and results information.
Display concept	Ambient/urban air pollution, particulates, gaseous pollutants, black carbon, wildfire smoke, extreme heat, green spaces, and heavy metals.
Population concept	Preconception, pregnancy, fetus, infant, child, or prenatal exposure data.
External concepts	Complications of maternal birth, external, neurodevelopment, inflammation, and oxidative stress.
Initial amount	3,547 documents were identified from the database.
Number of further studies	45 full-text articles assessed for detailed PECO eligibility.
Final Quantity	20 total publications: 16 primary empirical observational studies and 4 supporting conceptual/protocol publications.

D. Inclusion and Exclusion criteria

Primary inclusion criteria required: (1) original, empirical observational human studies (cohort, case-control, cross-sectional, registry, or biomarker designs); (2) clear evaluation of ambient outdoor air pollution or urban environmental exposures; (3) quantitative reporting of association metrics (e.g., adjusted odds ratios [aOR], adjusted hazard ratios [aHR], or beta coefficients with 95% confidence intervals [CI]); and (4) publication in peer-reviewed English-language journals between 2022-2025.

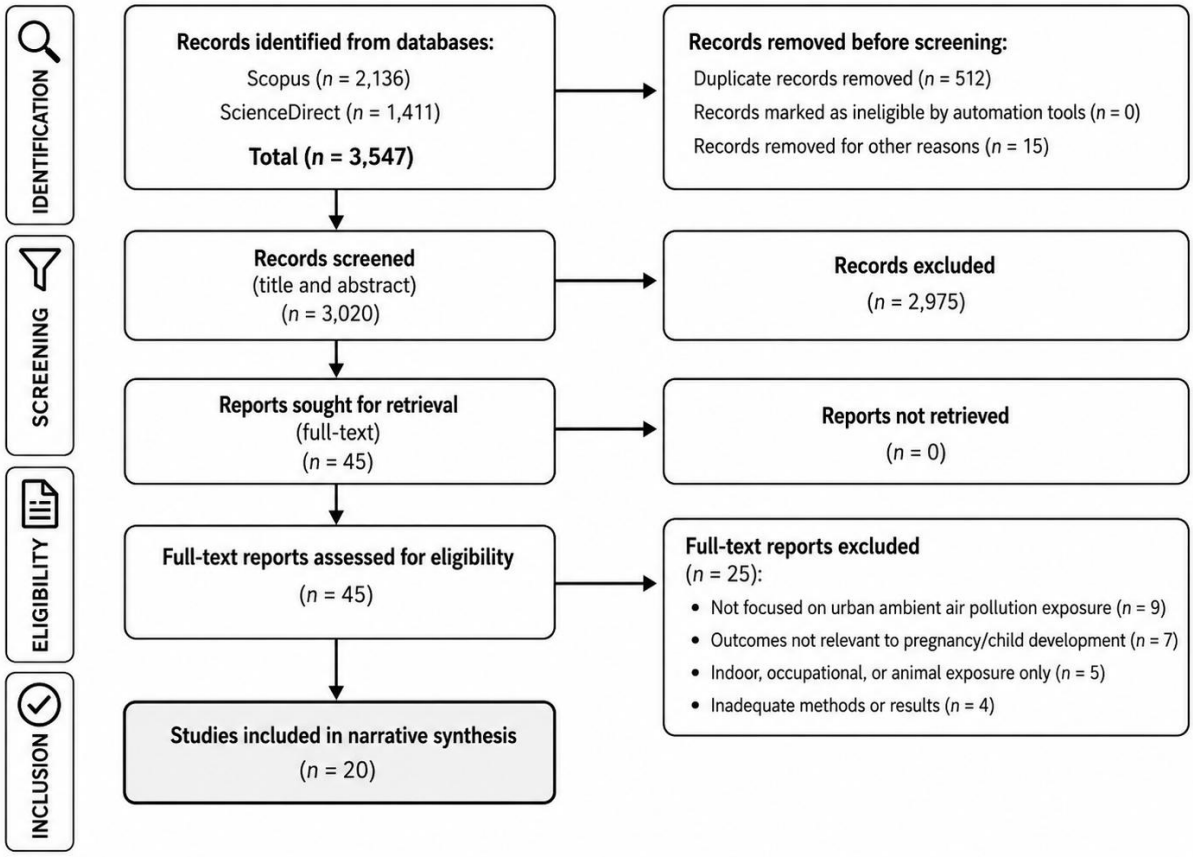
Exclusion criteria were strictly applied: (1) studies evaluating exclusively indoor air pollution (e.g., unvented indoor cookstoves without ambient data) or occupational exposures; (2) non-human animal or in vitro laboratory studies; (3) non-urban populations lacking transport/industrial air pollution characterization; (4) duplicate cohort publications lacking novel endpoints; and (5) non-empirical conceptual papers, narrative reviews, protocols, or systematic reviews (these were cataloged separately as supporting context).

E. Data extraction and quality assessment

Data extraction was executed independently by two reviewers using a pre-tested, standardized extraction form. Extracted variables included author(s), publication year, geographic location, study design, cohort sample size, specific exposure assessment methods, evaluated exposure windows, primary maternal/child endpoints, statistical modeling approaches (including multi-pollutant and machine learning structures), fully adjusted effect estimates (aOR/aHR/β with 95% CIs), unit exposure increments (e.g., per IQR or specific μg/m3), key controlled covariates, and reported null findings. Discrepancies were resolved through consensus or adjudication by a senior author. Multiple reports derived from identical underlying birth cohorts were cross-checked to avoid duplicate participant counts.

Methodological quality and risk of bias (ROB) were evaluated using validated, domain-specific instruments. Cohort and case-control studies were assessed using the Newcastle-Ottawa Scale (NOS), evaluating selection, comparability, and exposure/outcome assessment (maximum score: 9 stars). Cross-sectional, biomarker, and health claims studies were evaluated using corresponding JBI Critical Appraisal Checklists. Non-primary publications (reviews, frameworks, protocols) were not assigned quantitative observational risk-of-bias scores but were qualitative-contextual papers.

Figure 1. PRISMA flowchart



Results

A. Study selection and risk of bias evaluation

The systematic literature search identified 3,547 total records. Following automated deduplication (n=512) and screening, 45 full-text articles were retrieved. Twenty-five full-text articles were excluded with documented reasons: non-urban/non-ambient exposure (n=9), irrelevant health outcomes (n=7), purely occupational/indoor/animal exposures (n=5), and inadequate statistical reporting (n=4).

A final total of 20 publications met synthesis criteria, comprised of 16 primary empirical observational studies and 4 supporting conceptual articles (reviews, exposome frameworks, protocols). The 16 primary studies represented geographic regions across North America (USA, Canada), East Asia (China, South Korea), and Western Europe (Italy, Belgium, UK).

Table 3. Methodological risk of bias assessment for primary empirical studies

No.	Primary Author & Year	Study Design & Sample	Standardized Assessment Tool	Quality Score / ROB	Critical Methodological Considerations
1	Gogna et al., 2024 [1]	Prospective Cohort (N = 1,170)	Newcastle-Ottawa Scale (NOS)	8 / 9 stars (Low ROB)	Address-based spatial models; controlled for maternal age, BMI, income, and smoking; mild spatial misclassification risk.
2	Khalili et al., 2025 [2]	Prospective Cohort (N = 713)	Newcastle-Ottawa Scale (NOS)	8 / 9 stars (Low ROB)	High resolution climate/smoke models; robust adjustment for socio-

No.	Primary Author & Year	Study Design & Sample	Standardized Assessment Tool	Quality Score / ROB	Critical Methodological Considerations
					demographics; residual climate-social confounding possible.
3	Dai et al., 2025 [3]	Retrospective Case-Control ($N = 895$)	Newcastle-Ottawa Scale (NOS)	7 / 9 stars (Moderate ROB)	Multi-pollutant LUR models; cases hospital-matched; retrospective risk of selection bias and reliance on electronic health records.
4	Meng et al., 2025 [4]	Longitudinal Biomarker ($N = 156$)	JBI Biomarker Checklist	7 / 8 items (Low ROB)	Repeated urinary oxidative markers (8-OHdG, MDA); high internal validity; small sample size limits wide generalizability.
5	Pedersen et al., 2024 [5]	Population Registry Cohort ($N = 130{,}470$)	Newcastle-Ottawa Scale (NOS)	8 / 9 stars (Low ROB)	Universal registry data; high-resolution spatiotemporal exposure models; lack of individual workplace mobility tracking.
6	Murphy et al., 2024 [6]	Matched Case-Control ($N = 9{,}152$)	Newcastle-Ottawa Scale (NOS)	7 / 9 stars (Moderate ROB)	Validated diagnostic registries for ASD; address-based exposure estimation; potential residual residential mobility bias.
7	Sun et al., 2023 [7]	Retrospective Case-Control ($N = 3{,}180$)	Newcastle-Ottawa Scale (NOS)	6 / 9 stars (Moderate ROB)	Precise preconception window analysis; susceptibility to unmeasured dietary and indoor endocrine disruptor confounders.
8	Yan et al., 2022 [8]	Multicenter Prospective Cohort ($N = 3{,}754$)	Newcastle-Ottawa Scale (NOS)	8 / 9 stars (Low ROB)	Multi-city coverage; standard clinical definitions for HDP/GDM; exposure modeling varied slightly across regional networks.
9	Miron-Celis et al., 2023 [9]	Retrospective Population Cohort ($N = 1{,}310{,}807$)	Newcastle-Ottawa Scale (NOS)	9 / 9 stars (Low ROB)	Exceptionally large administrative cohort; robust week-by-week hazard modeling; highly controlled for maternal pre-existing conditions.

10	Hu et al., 2025 [23]	Prospective Cohort ($N = 453$)	Newcastle-Ottawa Scale (NOS)	7 / 9 stars (Moderate ROB)	Low-income Hispanic/Latina cohort; robust interaction modeling with HDP; geographical scope restricted to Los Angeles.
11	Bongaerts et al., 2022 [11]	Observational Placental/Fetal Tissue Study	JBI Observational Checklist	8 / 8 items (Low ROB)	Direct physical detection of black carbon nanoparticles in maternal-fetal tissues; highly mechanistic; non-epidemiologic risk estimate.
12	Li et al., 2025 [13]	Retrospective EHR Cohort ($N = 232{,}452$)	Newcastle-Ottawa Scale (NOS)	8 / 9 stars (Low ROB)	Detailed environmental species richness data; robust adjustment for temperature/pollutants; residual spatial socio-economic confounding.
13	Yu et al., 2023 [24]	Population Cohort ($N = 318{,}751$)	Newcastle-Ottawa Scale (NOS)	8 / 9 stars (Low ROB)	Comprehensive interaction testing between $PM_{2.5}$ and Maternal Immune Activation (MIA); objective clinical diagnostic criteria.
14	Cotter et al., 2024 [25]	Cross-Sectional Neuroimaging Cohort ($N = 11{,}880$)	JBI Analytical Cross-Sectional Tool	6 / 8 items (Moderate ROB)	High-resolution adolescent brain MRI connectivity metrics; cross-sectional neuroimaging design limits strict temporal causality.
15	Zhang et al., 2024 [26]	Retrospective Cohort ($N = 1{,}945$)	Newcastle-Ottawa Scale (NOS)	7 / 9 stars (Moderate ROB)	Machine learning quantile g-computation for multi-pollutant mixtures; single-hospital recruitment limits generalizability.
16	Lee et al., 2023 [27]	National Claims Cohort ($N = 843{,}134$)	Newcastle-Ottawa Scale (NOS)	8 / 9 stars (Low ROB)	Nationwide longitudinal claims coverage; inclusion of heavy metals (Pb, Cd); administrative diagnostic coding misclassification risk.

Table 3 demonstrates that the overall methodological quality of the included studies was high. Most cohort studies achieved low risk of bias, with Newcastle-Ottawa Scale scores ranging from 8 to 9 stars, while biomarker and observational studies also showed strong methodological rigor using JBI assessment tools. Although several retrospective and cross-sectional studies had a moderate risk of bias, the main limitations were related to selection bias, residual confounding, restricted geographic coverage, or limited sample size rather than fundamental design flaws. Overall, the evidence is considered robust and reliable, providing a strong methodological foundation for interpreting the associations between environmental exposures and adverse maternal and child health outcomes.

B. Synthesis of primary empirical findings

Detailed extraction of exposure metrics, effect estimates, exposure windows, adjusted covariates, and null findings across the 16 primary studies is presented in Table 4.

Table 4. Key characteristics, quantitative effect estimates, and findings of primary empirical studies

No.	Study Reference	Design, Setting & Population	Evaluated Exposures & Metrics	Outcome Domain	Fully Adjusted Quantitative Effect Estimates (95% CI) & Specific Increments	Key Adjusted Covariates & Reported Null Associations
1	Gogna et al., 2024 [1]	MIREC Prospective Cohort, Canada ($N = 1{,}170$)	$PM_{2.5}$, NO_2, O_3; Air Quality Health Index (AQHI)	Inflammatory Biomarkers	14-day moving average $PM_{2.5}$: 27% increase in C-reactive protein (CRP) ($\beta = 0.27, p < 0.01$). AQHI multi-pollutant index: 38% higher CRP per IQR increment.	Adjusted for maternal age, pre-pregnancy BMI, income, education, smoking, parity. Null finding: Single-pollutant O_3 showed no significant independent CRP elevation.
2	Khalili et al., 2025 [2]	MADRES Prospective Cohort, USA ($N = 713$)	Wildfire smoke $PM_{2.5}$, extreme heat stress	Birth Outcomes (SGA, LBW, Fenton scores)	Preconception wildfire smoke exposure: aOR for SGA = 1.84 (95% CI: 1.12–3.01). Combined heat + smoke interaction increased SGA risk nearly two-fold (aOR = 1.95, 95% CI: 1.21–3.14).	Adjusted for maternal race/ethnicity, insurance status, age, parity, neighborhood deprivation. Null finding: Second-trimester smoke alone was not significantly associated with LBW.
3	Dai et al., 2025 [3]	Retrospective Case-Control, Wuhan, China ($N = 895$)	NO_2, $PM_{2.5}$, SO_2; Multi-pollutant WQS modeling	Gestational Diabetes Mellitus (GDM)	First trimester NO_2 per $10\ \mu g/m^3$ increase: aOR = 1.34 (95% CI: 1.12–1.61). In multi-pollutant models, NO_2 was the primary contributor to	Adjusted for maternal age, family history of diabetes, parity, pre-pregnancy BMI, season. Null finding: O_3 showed no statistically significant

No.	Study Reference	Design, Setting & Population	Evaluated Exposures & Metrics	Outcome Domain	Fully Adjusted Quantitative Effect Estimates (95% CI) & Specific Increments	Key Adjusted Covariates & Reported Null Associations
					GDM risk (weight = 0.48).	association with GDM.
4	Meng et al., 2025 [4]	Longitudinal Biomarker, Los Angeles, USA ($N = 156$)	$PM_{2.5}$ mass, black carbon, oxidative potential (OP^{DTT})	Oxidative Stress Biomarkers	$PM_{2.5}$ oxidative potential per IQR increment: associated with 18.4% higher urinary 8-OHdG (95% CI: 5.2–33.1%) and 22.1% higher MDA in 2nd trimester.	Adjusted for maternal age, gestational week, ethnicity, secondhand smoke, season. Null finding: Coarse fraction $PM_{10-2.5}$ was not significantly associated with 8-OHdG.
5	Pedersen et al., 2024 [5]	Population Registry Cohort, Rome, Italy ($N = 130{,}470$)	$PM_{2.5}$, PM_{10}, NO_2	Hypertensive Disorders of Pregnancy / Preeclampsia	Trimester 1 $PM_{2.5}$ per IQR ($7.0\,\mu g/m^3$): aOR for preeclampsia = 1.06 (95% CI: 0.81–1.39). NO_2 per IQR ($15.7\,\mu g/m^3$): aOR = 1.11 (95% CI: 0.92–1.34).	Adjusted for maternal age, parity, nationality, socioeconomic status, singleton status. Null finding: PM_{10} showed no significant association with gestational hypertension in 3rd trimester.

6	Murphy et al., 2024 [6]	Matched Case-Control, Canada ($N = 9{,}152$)	NO_2, $PM_{2.5}$, O_3	Autism Spectrum Disorder (ASD)	Preconception and T1 NO_2 per IQR (4.2 ppb): aOR for ASD = 1.08 (95% CI: 1.01–1.15). Entire pregnancy NO_2: aOR = 1.12 (95% CI: 1.03–1.22).	Adjusted for maternal age, urbanicity, neighborhood income quintile, parity, birth year. Null finding: Prenatal O_3 showed no consistent association with ASD across trimesters.
7	Sun et al., 2023 [7]	Retrospective Case-Control, Beijing, China ($N = 3{,}180$)	$PM_{2.5}$, PM_{10}	Gestational Hypothyroidism	Preconception $PM_{2.5}$ exposure (90 days prior to LMP) per $10\,\mu g/m^3$: aOR = 1.16 (95% CI: 1.05–1.28). First trimester PM_{10} : aOR = 1.12 (95% CI: 1.02–1.23).	Adjusted for maternal age, pre-pregnancy BMI, gestational week of thyroid screening, season. Null finding: Third trimester PM exposure was not significantly associated with hypothyroidism.
8	Yan et al., 2022 [8]	Multicenter Prospective Cohort, China ($N = 3{,}754$)	$PM_{2.5}$, PM_{10}, O_3, SO_2, NO_2, CO	Maternal Complications (GH, GDM, PE)	First trimester $PM_{2.5}$ per $10\,\mu g/m^3$: aOR for GDM = 1.14 (95% CI: 1.06–1.22); aOR for PE = 1.18 (95% CI: 1.07–1.30). NO_2 per $10\,\mu g/m^3$: aOR for GH = 1.11 (95% CI: 1.03–1.20).	Adjusted for age, education, income, pre-pregnancy BMI, physical activity, alcohol/smoking. Null finding: CO was not significantly associated with preeclampsia.

9	Miron-Celis et al., 2023 [9]	Population Cohort, Ontario, Canada ($N = 1{,}310{,}807$)	$PM_{2.5}$, O_3, urban greenness (NDVI)	Gestational Diabetes Mellitus (GDM)	Weeks 7–18 $PM_{2.5}$ per IQR ($2.7\ \mu g/m^3$): aHR = 1.07 (95% CI: 1.02–1.11). Preconception to week 28 O_3 per IQR (7.0 ppb): aHR = 1.08 (95% CI: 1.04–1.12).	Adjusted for maternal age, parity, pre-existing hypertension, neighborhood deprivation, asthma. Null finding: High green space (NDVI) did not modify $PM_{2.5}$ -GDM association in highly polluted zones.
10	Hu et al., 2025 [23]	MADRES Prospective Cohort, USA ($N = 453$)	Traffic-related NO_x emissions	Postpartum Mental Health (Depression/Anxiety)	Pregnancy traffic NO_x exposure per IQR: elevated risk for postpartum anxiety symptoms ($\beta = 0.22$, $p = 0.03$). Risk amplified in women with HDP (interaction $p = 0.02$).	Adjusted for maternal age, ethnicity, education, prenatal depression baseline, marital status. Null finding: NO_x was not significantly associated with depression in non-HDP mothers.
11	Bongaerts et al., 2022 [11]	Observational Tissue Study, Belgium/UK	Ambient Black Carbon (BC) nanoparticles	Maternal-Fetal Circulation & Fetal Organs	Direct detection of BC particles in cord blood, placental tissue, fetal liver, kidney, and brain ($p < 0.001$ vs control controls).	Controlled for maternal smoking status and gestational age. Mechanistic proof of placental transplacental passage; clinical ORs/HRs not applicable.

12	Li et al., 2025 [13]	EHR Cohort, Southern California, USA ($N = 232{,}452$)	Urban green space, Plant Species Richness, O_3, NO_2	Preterm Birth (PTB)	Plant species richness per IQR increment: reduced PTB risk (aOR = 0.959, 95% CI: 0.941–0.977). Protection lost in areas with high ambient O_3 and NO_2.	Adjusted for maternal age, race/ethnicity, health insurance, parity, mean temperature. Null finding: Total green space area (NDVI) without plant diversity was not protective against PTB.
13	Yu et al., 2023 [24]	Population Cohort, Southern California, USA ($N = 318{,}751$)	$PM_{2.5}$ mass and chemical components	Autism Spectrum Disorder (ASD); Maternal Immune Activation	Pregnancy $PM_{2.5}$ per IQR ($3.73\,\mu g/m^3$): aHR for ASD = 1.07 (95% CI: 1.03–1.12). Maternal immune activation (MIA) elevated risk (aHR = 1.42), but no statistical interaction ($p_{inter} = 0.68$).	Adjusted for maternal age, education, race/ethnicity, health insurance, birth year. Null finding: $PM_{2.5}$ and MIA interaction was non-significant, acting as independent risk factors.
14	Cotter et al., 2024 [25]	ABCD Adolescent Cohort, USA ($N = 11{,}880$)	Prenatal & childhood $PM_{2.5}$, NO_2	White Matter Connectivity & Immune Markers	Early childhood NO_2 exposure: negatively correlated with total white blood cell counts in males ($\beta = -0.14$, $p = 0.01$) and altered fMRI structural brain connectivity.	Adjusted for child age, sex, household income, parental education, scanner site ID. Null finding: Prenatal $PM_{2.5}$ was not significantly associated with altered resting-state functional connectivity in early teens.

15	Zhang et al., 2024 [26]	Retrospective Cohort, Wuhan, China ($N = 1{,}945$)	Multi-pollutant mixture (SO_2, $PM_{2.5}$, NO_2) via Quantile G-Computation	Hypertensive Disorders of Pregnancy (HDP)	Multi-pollutant mixture score per quartile increase: aOR for HDP = 1.28 (95% CI: 1.11–1.48). SO_2 was the dominant causal driver in T1 (weight = 0.52).	Adjusted for maternal age, pre-pregnancy BMI, parity, passive smoking, education level. Null finding: Third trimester pollution mixture score showed no significant association with HDP.
16	Lee et al., 2023 [27]	National Claims Cohort, South Korea ($N = 843{,}134$)	Ambient SO_2, NO_2, heavy metals (Pb, Cd)	ASD & Pediatric Epilepsy	T3 SO_2 per 1 ppb: aOR for ASD = 2.723 (95% CI: 1.971–3.761); T3 Lead (Pb): aOR = 1.063 (1.019–1.110). T1 Pb: aOR for Epilepsy = 1.109 (1.043–1.179); T3 Cadmium (Cd): aOR = 2.193 (1.074–4.477).	Adjusted for child sex, maternal age, household income quintile, region, birth weight. Null finding: Second trimester NO_2 showed no statistically significant association with pediatric epilepsy.

Table 4 consistently shows that prenatal exposure to multiple environmental pollutants, particularly $PM_{2.5}$, NO_2, black carbon, and wildfire smoke, is associated with a wide range of adverse maternal and child health outcomes. These include increased inflammation, oxidative stress, gestational diabetes, hypertensive disorders of pregnancy, preterm birth, postpartum anxiety, and neurodevelopmental disorders such as autism. Most studies reported statistically significant associations after adjusting for major maternal and socioeconomic confounders, although a few pollutants or exposure windows showed no independent effects. Overall, the evidence indicates that fine particulate air pollution and related environmental exposures are important and consistent risk factors across pregnancy and early life, highlighting the need for effective environmental health interventions and preventive strategies.

Table 5. Non-Primary Articles as Supporting Evidence

No.	References	Type of article	Recommended locations for use	Reason not used as the basis for the main synthesis
1	Eberle & Stichling, 2022 [19]	Systematic review	Introduction or Discussion	Secondary evidence; not mixed with primary studies to infer the direction of the main associations.

2	Basilio & Zlatnik, 2023 [20]	Narrative overview	Introduction/Discussion of forest fire smoke	Narrative in nature; useful for context and mechanisms, not estimates of major effects.
3	Abarca-Castro et al., 2025 [10]	Integrative perspectives/reviews	Discussion of biological mechanisms and exposomemes	Conceptual articles; used for biological possibilities, not primary epidemiological outcomes.
4	Trushna et al., 2025 [28]	Cohort protocols	Discussion of future research agenda	Haven't reported the final results yet; cannot be used as evidence of association.

In contrast to the primary study, table 5 is not used as the basis for the main synthesis because it is in the form of systematic review, narrative review, perspective articles, and cohort protocols. However, these articles provide conceptual support regarding biological mechanisms, exposal approaches, and future research directions so as to strengthen the interpretation of the results obtained from primary studies.

C. Synthesis based on external

Table 6. Summary of evidence synthesis by outcome domain

External status	External domains	Primary reference (primary study)	Supporting references	Consistent synthesis interpretation
Main outcomes	Gestational diabetes mellitus	[3], [8], [9]	[19], [15], [17], [29]	The main evidence comes from the primary studies of Dai, Yan, and Miron-Celis. The most relevant pollutants are PM2.5, NO2, O3, SO2, and PM10, especially in the preconception period to the beginning of the second trimester.
Main outcomes	HDP and preeclampsia	[5], [8], [26]	[15], [17], [30]	Moderate evidence suggests PM2.5, PM10, NO2, and SO2 are associated with pregnancy vascular disorders, especially in early pregnancy.
Main outcomes	Fetal growth, SGA, and LBW	[2]	[20], [11], [12], [32], [33], [16]	Exposure to fire smoke, extreme heat, and particulate matter is associated with impaired fetal growth and low birth weight.
Main outcomes	Premature birth	[13]	[20], [34], [35], [33], [36], [37]	Urban environments can be both a risk and a protection; Pollution and heat are associated with higher risks, while green spaces can lower them.

External status	External domains	Primary reference (primary study)	Supporting references	Consistent synthesis interpretation
Main outcomes	ASD, epilepsy, and neurodevelopment	[6], [24], [25], [27]	[10], [12], [18]	NO2, PM2.5, SO2, Pb, and Cd appear as important exposures. Evidence supports inflammatory, immune, and brain development pathways.
Secondary outcome	Hypothyroidism in pregnancy	[7]	[12], [31]	The evidence is still limited but biologically relevant because maternal thyroid function plays a role in fetal development and child neurocognition.
Secondary outcome	Inflammation and oxidative stress	[1], [4], [11], [25]	[10], [12], [18]	Air pollution can be related to inflammation, oxidative stress, endothelial dysfunction, and maternal-fetal disorders.
Secondary outcome	Symptoms of postpartum depression and anxiety	[23]	[15], [34]	Traffic NOx can be associated with postpartum distress, especially in mothers with vascular vulnerability.

Discussion

A. Biological mechanisms and vulnerable exposure windows

The synthesized empirical evidence confirms that ambient air pollution and urban environmental exposures trigger pathological responses across multiple biological scales[10,12,15,31,34]. Inhaled fine particles (PM2.5) and black carbon cross the alveolar-capillary membrane, entering the maternal systemic circulation where they induce systemic oxidative stress and pro-inflammatory signaling pathways (evidenced by elevated CRP, 8-OHdG, and MDA)[1,4,18]. The physical identification of black carbon nanoparticles directly within umbilical cord blood, placental tissue, and fetal organ compartments provides definitive mechanistic evidence that atmospheric pollutants breach the placental barrier[11,29,42].

A primary insight emerging from this review is the supreme importance of timing[9,38]. Exposure window analyses consistently reveal that the preconception period and first trimester represent critical susceptibility windows for maternal metabolic and vascular complications[2,7,8,9,26,30]. Disruption during implantation and early trophoblast invasion by PM2.5, NO2, or SO2 alters normal spiral artery remodeling, predisposing the placenta to hypoxia, endothelial damage, and subsequent preeclampsia or fetal growth restriction[5,12,17,26,30]. Furthermore, maternal exposure during preconception and early gestation can impair pancreatic beta-cell function and insulin sensitivity, explaining the robust associations observed for Gestational Diabetes Mellitus (GDM)[3 9,19,29].

For offspring neurodevelopmental outcomes, exposure windows appear extended[6,24,27]. While early prenatal exposures disrupt neural tube formation and neurogenesis, late third-trimester exposures to heavy metals (Pb, Cd) and gaseous pollutants (SO2, NO2) show pronounced associations

with Autism Spectrum Disorder (ASD) and pediatric epilepsy[6,24,27,28]. Third-trimester exposures coincide with rapid fetal brain synaptogenesis, microglial activation, and myelination, rendering the developing brain highly susceptible to neuroinflammatory insults and systemic maternal immune activation[10,24,25].

B. Critical appraisal of methodological limitations in primary studies

A rigorous evaluation of the synthesized primary studies reveals several methodological limitations that account for heterogeneous findings across the literature[15,21,33]:

1. Spatial Exposure Misclassification and Residential Mobility: The majority of large population cohorts relied on fixed address-based spatial models (e.g., land-use regression, nearest ambient monitor, or satellite spatiotemporal rasters)[10,15,33,34]. These models inherently fail to account for maternal daily activity patterns, occupational exposures, personal indoor transit, time spent indoors, or residential moves during pregnancy[10,34]. Such misclassification is overwhelmingly non-differential, driving effect estimates toward the null and potentially underestimating true environmental risk magnitudes[15,33].
2. Residual Socio-Environmental Confounding: Urban environments feature intricate spatial correlations between environmental hazards and social vulnerability[13,14,23]. Low-income neighborhoods frequently suffer from combined exposures: elevated traffic exhaust, extreme heat islands, industrial noise, high psychological stress, and poor access to fresh foods or green space[9,13,14,23]. Although primary studies controlled for broad socioeconomic status (e.g., neighborhood income quintiles or insurance type), unmeasured residual confounding from diet, indoor air quality, and chronic psychosocial stress remains a persistent challenge[9,14,23].
3. Multi-Pollutant Collinearity and Statistical Modeling: Urban pollutants are highly collinear (e.g., NO2 and PM2.5 from vehicular exhaust)[1,3,26,35]. Traditional single-pollutant regression models are prone to overestimating individual pollutant risks due to confounding by co-pollutants[3,35]. Advanced multi-pollutant frameworks such as Weighted Quantile Sum (WQS) regression, Quantile G-Computation, and Air Quality Health Indices demonstrated superior statistical power and causal inference, highlighting NO2 and SO2 as dominant drivers within urban air mixtures[1,3,26,41].
4. Overlapping Cohort Samples and Multiple Comparisons: Several studies were derived from shared regional administrative birth registries (e.g., Southern California Kaiser Permanente or Ontario health databases)[6,32,40]. While individual research teams examined distinct clinical endpoints, overlapping sample populations necessitate cautious interpretation[6,40]. Furthermore, studies evaluating dozens of week-by-week exposure windows face heightened risks of type I errors due to multiple testing[9,38,43].

C. Translation to clinical practice and public policy

To maintain scientific rigor, clinical and policy recommendations must be clearly distinguished from evaluated empirical endpoints[15,16,33]. The primary studies synthesized in this review evaluated observational exposure-outcome associations; none directly tested the clinical efficacy of individual behavioral interventions (such as N95 mask usage, personal air purifier deployment, or travel route alterations)[15,16,33].

Clinical & Antenatal Context: While individual behavioral modifications represent plausible risk-mitigation strategies, healthcare providers should incorporate environmental health awareness into routine antenatal counseling without overstating individual intervention efficacy[17,19,31,39,40]. Antenatal education should emphasize monitoring local Air Quality Index (AQI) forecasts, minimizing strenuous outdoor activities during peak pollution advisories, and exercising

heightened clinical surveillance for pregnant women with baseline vascular, respiratory, or metabolic comorbidities[17,31,39,40].

Public Policy & Urban Planning Context: Because individual-level avoidance measures cannot eliminate ambient urban exposures, primary prevention relies on structural policy solutions[13,14,16,36,37]. Evidence highlighting the protective effect of urban plant species richness against preterm birth indicates that urban green space planning must prioritize botanical diversity and heat-island mitigation alongside strict vehicular and industrial emission controls[13,14,16]. Regulatory policy frameworks protecting pregnant populations yield multi-generational public health benefits[14,16,36,37].

D. Strengths and limitations of this review

The primary strength of this systematic review lies in its comprehensive integration of maternal, perinatal, biological biomarker, and long-term child neurodevelopmental outcomes within an updated, multi-pollutant urban exposure framework[10,15,21]. Application of domain-specific standardized appraisal tools (NOS and JBI) ensured rigorous bias evaluation[21,22].

Limitations include: (1) restriction of primary searches to Scopus and ScienceDirect between 2022-2025, which may have excluded relevant grey literature or earlier baseline studies[21]; (2) substantial methodological and exposure metric heterogeneity across primary studies, precluding quantitative meta-analysis[15,33]; and (3) reliance on observational study designs, which inherently limits absolute causal determination[15,31,34].

D. **Research Limitations**

The main limitation of this review lies in the source and scope of the article. Searches are only conducted on Scopus and ScienceDirect, with the main article period limited to 2022-2025. Therefore, important studies from other databases or previous periods may have been missed. This makes the findings of the review more current, but not fully representative of all available evidence.

The second limitation is the methodological heterogeneity between articles. Each study uses different designs, pollutants, exposure windows, outcome definitions, and units of effect. Therefore, this review uses narrative synthesis and does not conduct meta-analysis. The findings should be interpreted as patterns and directions of evidence rather than as an estimate of the definite combined risk.

In addition, most of the evidence comes from observational studies and many exposure measurements are based on residential addresses or environmental models. This approach is useful for identifying patterns in large populations, but may not fully capture daily activities, personal exposure, indoor conditions, housing mobility, and social or maternal health factors.

Therefore, the results should be understood as a strong but not final map of evidence. These findings are important to guide research, antenatal education, and clean air policies, but should not be used as the sole basis for determining individual risk in each pregnant woman. Future studies will require registered protocols, a broader database, more detailed bias risk assessments, multipollutant models, personal exposure measurements, and local data from cities with high pollution loads.

Conclusion

This systematic review demonstrates that urban ambient air pollution ($PM_{2.5}$, PM_{10}, NO_2, SO_2, black carbon) and co-occurring environmental stressors (extreme heat, wildfire smoke) are significantly associated with adverse maternal and child health outcomes. The strongest, moderate-certainty evidence links preconception and early-pregnancy exposures to heightened risks of Gestational Diabetes Mellitus, Hypertensive Disorders of Pregnancy/Preeclampsia, Low Birth Weight, and Preterm Birth. Multi-pollutant mixture modeling consistently outperforms single-pollutant approaches in capturing real-world urban health risks. Evidence for gestational hypothyroidism, postpartum mental distress, and offspring neurodevelopmental trajectories remains limited to low in certainty and methodologically heterogeneous. Protecting maternal and child health requires transitioning from individual exposure management toward comprehensive urban

emission reduction policies and ecologically diverse urban planning.

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