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Prenatal Exposure to Ambient Fine Particulate Matter ($PM_{2.5}$) and Adverse Perinatal Outcomes: A Literature Review

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ABSTRACT

Background. Prenatal exposure to fine particulate matter ($PM_{2.5}$), composed of airborne particles smaller than 2.5 micrometers, is a significant environmental risk that can adversely affect pregnancy. Exposure to $PM_{2.5}$ during pregnancy has been associated with a number of adverse birth outcomes, including small for gestational age (SGA), low birth weight (LBW), and preterm birth (PTB). This literature review summarizes evidence published between 2020 and 2025 regarding the relationship between prenatal $PM_{2.5}$ exposure during different pregnancy trimesters and the risk of PTB, LBW, and SGA.

Aim of the review. This review gathers and evaluates epidemiological studies from 2020 to 2025 examining the connection between prenatal exposure to $PM_{2.5}$ and adverse perinatal outcomes such as SGA, LBW, and PTB

Methods. In our review, we included case-control and cohort primary epidemiological studies that were published between 2020 and 2025. Maternal exposure to ambient $PM_{2.5}$ in connection with PTB, LBW, or SGA was evaluated in eligible studies.

Results. Across the reviewed studies, prenatal exposure to $PM_{2.5}$ was linked to an increased risk of LBW and SGA, with heightened susceptibility in the first and second trimesters. Associations with PTB were also noted but more variable. Many large population-based studies found small but statistically significant effects, especially those using thorough exposure assessment methods.

Conclusions. The current evidence suggests that maternal exposure to ambient $PM_{2.5}$ during pregnancy is associated with an increased risk of adverse birth outcomes, especially indicators of restricted fetal growth. These findings highlight the need for further study to clarify critical exposure windows, underlying biological processes, and effective strategies to reduce exposure during pregnancy. They also support the importance of air quality as a controllable environmental factor for maternal and offspring health.
Keywords: $PM_{2.5}$, prenatal exposure, small for gestational age, low birth weight, preterm birth, air pollution

1. Introduction

Ambient air is a major global health concern, with fine particulate matter ($PM_{2.5}$; aerodynamic diameter ≤2.5 μm) has been recognized as a major environmental risk factor for a range of adverse health outcomes, including those during pregnancy. $PM_{2.5}$ can penetrate deeply into the lungs and bloodstream, leading to systemic inflammation and oxidative stress, which have been proposed as key mechanisms in its effects on pregnancy[1–3]. Pregnant women and their fetuses represent particularly vulnerable populations. In recent years, increasing attention has been directed toward the possible consequences of $PM_{2.5}$ exposure during pregnancy[1,4,5]. Prenatal exposure to ambient $PM_{2.5}$ is associated with adverse birth outcomes including preterm birth (PTB), low birth weight (LBW), and small for gestational age (SGA), according to a growing body of epidemiological studies published between 2020 and 2025[3–19]. Associations between maternal $PM_{2.5}$ exposure and congenital heart defects have been reported in large surveillance-based studies, but these outcomes were not addressed in this review[20]. Large population-based cohort studies carried out in a variety of geographic regions, including North America, Europe, Asia, and South America, have found statistically significant, modest and generally consistent relationships between maternal $PM_{2.5}$ exposure and impaired fetal growth indicators[4–7,9,11,12,18,19,21,22]. Among the assessed outcomes, measures of fetal growth restriction, such as LBW and SGA, seem to have the strongest correlations with prenatal $PM_{2.5}$ exposure[1,6,7,9,11,12]. Higher $PM_{2.5}$ concentrations during pregnancy are associated with shifts toward lower birth weight percentiles or reduction in mean birth weight even among term births[5–7,12,15]. These results have been noted in both city and country populations, using different ways to assess exposure, such as fixed-location monitoring, satellite data-based models, and hybrid methods[8,11,17,18,21]. The evidence about the connection between prenatal exposure to $PM_{2.5}$ and

preterm birth is more heterogeneous across studies. Increased prenatal $PM_{2.5}$ exposure has been linked to a higher risk of PTB in several studies[3,4,10,13–16], others have found comparatively weaker or context-dependent effects that are influenced by geographic region, exposure levels, and analytical strategies[18,21,23]. Several recent studies have also explored the timing of exposure during pregnancy, suggesting that susceptibility to $PM_{2.5}$ may differ across gestational windows. Specifically, it has been suggested that exposure during the first and second trimesters may be crucial for fetal growth and the early stages of parturition, although findings are not totally consistent across all studies[5,8,15,17,21,22]. Nationwide cohort data further suggest that $PM_{2.5}$ alone may not fully capture risk, as combined exposure with ozone has been linked to fetal growth restriction[24]. These observations highlight how important it is to look at exposure patterns unique to each trimester when assessing perinatal risks. Emerging evidence suggests that certain $PM_{2.5}$ sources and components, especially those related to wildfire smoke, may present additional risks during pregnancy in addition to ambient $PM_{2.5}$ from general urban pollution sources[10,13,23]. It is important to account for both source-specific and compositional aspects of particulate matter exposure by studies on wildfire-attributable $PM_{2.5}$ which have demonstrated higher risks of preterm birth and, in some populations, lower birth weight outcomes[3,10,13]. Despite the increasing number of high-quality epidemiological studies, the current literature is characterized by methodological heterogeneity in exposure assessment, outcome definitions, and statistical modeling approaches[4–9,12,13,15,21,22]. Systematic reviews published during the study period highlight these methodological limitations while confirming an overall correlation between prenatal $PM_{2.5}$ exposure and unfavorable perinatal outcomes[1,3]. An organized synthesis of recent evidence is required to clarify the consistency of reported associations, identify important sources of heterogeneity, and provide a summary of current knowledge about vulnerable exposure windows and outcome-specific risks[1,3,5,8,15,21,22].

2. **Methods of review**

2.1. Review Design

In this review, we focused on studies published between 2020 and 2025, including case-control and cohort studies. We performed a systematic search of available sources. Due to the heterogeneity in study designs, exposure assessment methods, and outcome definitions across the available literature, a formal meta-analysis was not feasible. Therefore, we chose a qualitative narrative synthesis to summarize the findings in a structured, descriptive manner.

2.2. PECO Framework

The PECO framework was specifically chosen for its ability to systematically define the key elements of our review. This structured approach ensures a focused review process, aligning study selection with our research question and facilitating a consistent analysis of study findings.

Population (P):

The group under consideration includes pregnant women and their offspring from the general population. We included both singleton and multiple pregnancies, as reported in population-based cohort and case–control studies. No restrictions were applied with respect to geographic region.

Exposure (E):

The exposure of interest was prenatal exposure to ambient fine particulate matter with an aerodynamic diameter ≤2.5 μm ($PM_{2.5}$). These studies used fixed-site air quality monitoring stations, satellite-based models, land-use regression models, or hybrid exposure assessment approaches. Exposure was evaluated across the entire pregnancy as well as within specific gestational windows or trimesters when available.

Comparator (C):

The comparison group consists of people with same study population and health characteristics who have been exposed to lower levels of $PM_{2.5}$, as defined by the exposure contrasts used in the original studies.

Outcomes (O):

The primary outcomes of interest were adverse perinatal outcomes, including preterm birth (PTB), low birth weight (LBW), and small for gestational age (SGA), as defined by the original studies. We included only studies that used clinical or registry data.

2.3. Inclusion Criteria

Studies were included if they met all of the following criteria:

• **Study design:** Original epidemiological studies, including prospective or retrospective cohort studies and case–control studies, examining the link between prenatal exposure to ambient $PM_{2.5}$ and perinatal outcomes.

• **Population:** Studies that include human populations of pregnant women and their offspring, both singleton and multiple pregnancies.

• **Exposure:** Assessment of maternal exposure to ambient fine particulate matter ($PM_{2.5}$) during pregnancy, estimated using validated methods such as fixed-site air monitoring, satellite-based models, land-use regression models, or hybrid approaches.

• **Outcomes:** Reporting at least one of the predefined adverse perinatal outcomes- preterm birth (PTB), low birth weight (LBW), or small for gestational age (SGA), with outcomes clearly defined

according to clinical, registry-based, or standardized epidemiological criteria.

• **Publication period:** Studies published between January 2020 and December 2025.

• **Language:** Articles published in English.

• **Availability:** Full-text articles available in open-access format.

These inclusion criteria were chosen to ensure that only studies directly addressing the relationship between prenatal $PM_{2.5}$ exposure and adverse perinatal outcomes were included, thereby providing the most relevant and reliable evidence for this review.

2.4. Exclusion Criteria

Studies were excluded if they:

• were conducted in animals or in vitro;

• did not provide quantitative estimates of prenatal $PM_{2.5}$ exposure;

• focused on air pollution exposure outside the prenatal period;

• did not report data for at least one of the key outcomes (PTB, LBW, or SGA);

• represented duplicate analyses of the same study population without additional or updated information, in which case the most comprehensive or recent publication was retained.

• lacked sufficient data on prenatal exposure to $PM_{2.5}$ or the key perinatal outcomes;

• used unreliable or non-validated methods for assessing prenatal exposure to $PM_{2.5}$, such as self-reported exposure data or non-standardized models

2.5. Information Sources and Search Strategy

A literature search was conducted using major biomedical databases and open-access scientific platform, including **PubMed/MEDLINE and selected environmental health journals.** The search covered publications released between **January 2020 and December 2025.** The search strategy combined controlled vocabulary terms and free-text keywords related to prenatal exposure to fine particulate matter and adverse perinatal outcomes. Key search terms included *"$PM_{2.5}$"*,"air pollution*", "prenatal exposure", "pregnancy", "preterm birth", "low birth weight",* and *"small for gestational age"*.Reference lists of relevant articles and reviews were also manually screened to identify additional eligible studies. Only peer-reviewed, full-text, open-access publications meeting the predefined inclusion criteria were considered for further evaluation.

2.6. Study Selection Process

Study selection was performed based on predefined eligibility criteria. In order to eliminate studies that were obviously unrelated to prenatal $PM_{2.5}$ all records found through the database search were first screened by title and abstract. After that, full-text articles of potentially pertinent studies on

exposure or adverse perinatal outcomes were retrieved and their eligibility were evaluated. During full-text screening, studies were evaluated with regard to study design, assessment of prenatal $PM_{2.5}$ exposure, outcome definitions (preterm birth, low birth weight, or small for gestational age), and availability of extractable data. To prevent duplication, only the most comprehensive or most recent article was included when several publications came from the same study population. Systematic reviews and meta-analyses identified during the search were not included as primary studies but were used to support interpretation of findings. Only studies meeting all inclusion criteria were included in the final qualitative synthesis.

2.7. Data Extraction and Synthesis

Data were extracted in a standardized manner from all included studies. Extracted information included study design, population characteristics, geographic setting, methods of prenatal $PM_{2.5}$ exposure assessment, timing of exposure during pregnancy, and definitions of adverse perinatal outcomes, namely preterm birth, low birth weight, and small for gestational age. Key effect estimates and the direction of reported associations were recorded, with particular attention to results stratified by gestational window when available. Due to heterogeneity in exposure assessment, outcome definitions, and analytical approaches, a quantitative meta-analysis was not undertaken. The study results were presented in a narrative synthesis. Patterns and trends were examined across different situations and stages of pregnancy, and possible explanations for differences in pregnancy outcomes.

2.8. Assessment of Methodological Heterogeneity and Limitations

Methodological heterogeneity was assessed qualitatively across the included studies. The reviewed studies used various sources and methods to quantify ambient $PM_{2.5}$ exposure including variations in exposure metrics, spatial resolution, and the definition of exposure windows during pregnancy. We also observed differences in how studies defined and measured conditions such as small for gestational age (SGA), which is an important indicator for understanding perinatal outcomes, particularly in relation to $PM_{2.5}$ exposure. While birth weight and low birth weight are generally defined using standardized clinical criteria, definitions of SGA differed across studies depending on reference growth standards and analytical approaches. These differences complicated direct comparisons between study results. Additional variability was observed in analytical approaches. Researchers may consider various factors, such as maternal health and social circumstances, which may influence pregnancy outcomes. Such differences likely contributed to variability in reported effect estimates, especially for preterm birth. Only studies published in English and available in open-access format were included, which may have resulted in the exclusion of some relevant

research. Additionally, the differences among the studies posed challenges for those conducting the review. Because of this heterogeneity, quantitative pooling of results was not feasible, and findings were synthesized narratively. Despite these limitations, the review provides a clear summary of recent epidemiological evidence and supports the conclusion that prenatal exposure to ambient $PM_{2.5}$ is associated with adverse perinatal outcomes, particularly concerning fetal growth.

3. Current knowledge

3.1. $PM_{2.5}$ exposure and preterm birth

Preterm birth (PTB) has been a frequent outcome of interest in studies investigating prenatal exposure to ambient $PM_{2.5}$, although the reported associations are generally less consistent than those observed for indicators of fetal growth. Several large population-based cohort studies from North America, Europe, and Asia have shown that higher prenatal exposure to $PM_{2.5}$ is associated with an increased risk of PTB[4,10,12,15]. In most cases, the observed effect sizes were modest, but statistical significance was achieved in studies with large sample sizes and more detailed exposure assessment. Some studies reported stronger associations in settings characterized by higher exposure contrasts or when analyses focused on specific $PM_{2.5}$ sources. In particular, wildfire-related particulate matter has been linked to a higher risk of PTB, suggesting that both exposure intensity and particle composition may play a role[3,10,14]. At the same time, other cohort studies reported weaker or non-significant associations, with findings varying depending on geographic location, background pollution levels, and methodological approaches to exposure assessment[8,21,23]. Systematic reviews published during the study period generally support an association between prenatal $PM_{2.5}$ exposure and PTB, but also emphasize substantial heterogeneity across studies and less consistent evidence compared with outcomes related to impaired fetal growth[1,3]. Taken together, the available evidence suggests that prenatal exposure to ambient $PM_{2.5}$ may contribute to an increased risk of preterm birth, particularly in settings with higher exposure levels or specific pollution sources. However, the variability of findings indicates that this relationship is influenced by both methodological and contextual factors, highlighting the need for cautious interpretation and further research[1,8,21].

3.2. $PM_{2.5}$ exposure and fetal growth outcomes (LBW, SGA)

Outcomes related to impaired fetal growth, particularly low birth weight (LBW) and small for gestational age (SGA), show the most consistent associations with prenatal exposure to ambient $PM_{2.5}$. Across multiple large population-based cohort studies conducted in diverse geographic settings, higher maternal $PM_{2.5}$ exposure during pregnancy has been associated with reduced birth

weight and increased risks of LBW or SGA[4,5,7,11,19,24]. Associations between $PM_{2.5}$ exposure and fetal growth restriction have been observed not only for preterm births but also among term infants, indicating that prenatal air pollution exposure may affect intrauterine growth independently of gestational duration[5–7]. Several studies also reported exposure–response relationships or shifts toward lower birth weight percentiles with increasing $PM_{2.5}$ concentrations[5,6,9]. Systematic reviews and meta-analyses published during the study period consistently identify LBW and SGA as outcomes with stronger and more stable associations with prenatal $PM_{2.5}$ exposure compared with preterm birth[1,3]. Although effect sizes were generally modest, the consistency of findings across populations, exposure assessment methods, and study designs supports the plausibility of a causal relationship between prenatal $PM_{2.5}$ exposure and impaired fetal growth[4,5,7,11].

3.3. Trimester-specific and critical exposure windows

Several studies indicate that the impact of prenatal $PM_{2.5}$ exposure may differ depending on the stage of pregnancy, suggesting the presence of sensitive periods during gestation. In many analyses, stronger associations have been observed for exposure occurring in the first and second trimesters, particularly in relation to fetal growth outcomes such as low birth weight and small for gestational age[4,8,21]. Exposure during early pregnancy has been associated with lower birth weight and a higher likelihood of LBW or SGA, which may reflect interference with placentation and early fetal development[15,21]. Associations with $PM_{2.5}$ exposure later in pregnancy have also been reported, although these findings are less consistent and appear to depend on both the outcome considered and the analytical approach used[8,21]. With respect to preterm birth, trimester-specific results are notably heterogeneous. Some studies have reported elevated risks linked to exposure during mid- or late pregnancy, whereas others have found weak or no clear associations[3,4,13,15]. Overall, the available evidence suggests that early and mid-pregnancy may represent periods of increased vulnerability to $PM_{2.5}$ exposure. However, differences in study design, exposure assessment, and outcome definitions limit the ability to define a single, clearly delineated critical exposure window[3,4,21,24].

3.4. Methodological considerations and sources of heterogeneity

The interpretation of associations between prenatal exposure to ambient $PM_{2.5}$ and adverse perinatal outcomes is influenced by several methodological aspects that vary across studies. One of the main sources of heterogeneity relates to differences in exposure assessment. The reviewed studies relied on a range of approaches, including fixed-site air monitoring, satellite-based exposure models, land-use regression, and hybrid methods, which differ in spatial resolution and their ability to reflect individual-level exposure during pregnancy[8,9,17,18]. Further variability arises from differences in how

exposure windows across pregnancy were defined and analyzed. These inconsistencies make direct comparisons between studies more difficult and may partly explain the mixed findings reported for trimester-specific effects[8,15,17,21]. Although definitions of preterm birth, low birth weight, and small for gestational age were broadly comparable, the use of different growth references, covariate selection strategies, and statistical models likely contributed to variation in reported effect estimates[1,3,21]. As with most observational research, residual confounding cannot be fully excluded. Factors such as socioeconomic conditions, maternal health characteristics, and co-exposure to other air pollutants may influence the observed associations[1,3,4,8]. Nevertheless, the repeated observation of similar patterns—particularly for outcomes related to fetal growth—across different populations and study designs suggests that the overall findings are robust. At the same time, the substantial methodological heterogeneity across studies should be taken into account when interpreting results and comparing effect estimates[1,3].

4. Discussion

4.1. Summary of main findings

This review brings together recent epidemiological evidence showing that prenatal exposure to ambient $PM_{2.5}$ is linked to adverse perinatal outcomes. The clearest and most consistent findings concern impaired fetal growth, particularly low birth weight and small for gestational age. Such associations have been reported in several large cohort studies conducted in different parts of the world and across a range of exposure assessment methods[4,7,9,18,19]. Results for preterm birth are more variable. While some studies identified an increased risk associated with higher prenatal $PM_{2.5}$ exposure, others reported weaker or less consistent effects, with differences observed between populations and analytical approaches[1,3,8,13]. This variability suggests that the relationship between $PM_{2.5}$ exposure and preterm birth is likely influenced by both contextual factors and methodological differences across studies. Studies examining the timing of exposure during pregnancy indicate that early and mid-pregnancy may be periods of greater vulnerability, particularly in relation to fetal growth outcomes. However, inconsistencies in study design and exposure window definitions make it difficult to point to a single critical period with certainty[8,17,21]. Taken together, the available evidence highlights ambient $PM_{2.5}$ as an important environmental factor in perinatal health, while also emphasizing the need for cautious interpretation given the heterogeneity of the underlying studies[1,3].

4.2. Biological plausibility and potential mechanisms

Prenatal exposure to ambient $PM_{2.5}$ has been linked to various adverse perinatal outcomes, and several biological mechanisms have been proposed to explain these associations. Fine particulate matter can penetrate the respiratory system, entering the bloodstream and promoting oxidative stress and inflammation, both of which contribute to pregnancy complications[2]. This process generates reactive oxygen species (ROS), which damage cellular structures and trigger an inflammatory response. This exacerbates pregnancy-related issues [2]. Inflammation is considered a central factor in the development of complications, particularly those affecting fetal growth[6]. A primary mechanism through which $PM_{2.5}$ influences pregnancy is oxidative stress. ROS produced by the inhalation of fine particulate matter can initiate an inflammatory process that causes tissue damage, particularly in the placenta [5]. Increased levels of pro-inflammatory cytokines such as TNF-α, IL-6, and IL-8, which are elevated after $PM_{2.5}$ exposure, have been shown to impair placental function[3]. This impairment leads to reduced placental perfusion and disrupted nutrient and oxygen transfer to the fetus, contributing to restricted fetal growth[3].

Placental dysfunction caused by $PM_{2.5}$ exposure has been identified as a key pathway for fetal growth restriction[1]. Exposure to fine particulate matter has been associated with changes in trophoblast function, including increased apoptosis and impaired trophoblast invasion, which are essential for normal placental development [2]. These disruptions compromise the placenta's ability to support fetal growth, which is reflected in the association between $PM_{2.5}$ exposure and low birth weight (LBW) or small for gestational age (SGA)[6].

Additionally, prenatal exposure to $PM_{2.5}$ has been linked to metabolic changes in pregnant women, which can further impact pregnancy outcomes[5]. Notably, $PM_{2.5}$ exposure is associated with disturbances in glucose metabolism, which may lead to gestational diabetes mellitus (GDM), a condition that increases the risk of fetal growth restriction[15]. Furthermore, mitochondrial dysfunction induced by $PM_{2.5}$ exposure may disrupt metabolic pathways, further exacerbating the adverse effects on intrauterine growth[2]. The timing of exposure during pregnancy is also crucial in determining its impact on fetal development[5]. Several studies have suggested that the first trimester, a critical period for placental development and vascular adaptation, is particularly vulnerable to the effects of $PM_{2.5}$ exposure[8]. Disruption during this early stage can have long-lasting consequences on fetal growth, leading to a higher likelihood of LBW or SGA[3]. While these biological mechanisms provide a plausible explanation for the associations between $PM_{2.5}$ exposure and adverse pregnancy outcomes, direct causal pathways remain unclear[1]. Much of the current evidence is based on indirect findings, underscoring the necessity for future research that integrates more precise exposure assessment methods with biological markers of placental and fetal health. This approach would help clarify the specific mechanisms by which $PM_{2.5}$ influences pregnancy outcomes, which is essential

for formulating targeted strategies to reduce these risks[15]. Such studies will allow for a better understanding of the precise mechanisms by which $PM_{2.5}$ affects pregnancy outcomes, which is critical for developing effective strategies to mitigate these risks[3].

4.3. Interpretation of heterogeneity across studies

The heterogeneity across studies investigating the relationship between prenatal $PM_{2.5}$ exposure and perinatal outcomes is likely due to differences in exposure assessment techniques, study populations, and analytical approaches. Various methods used to estimate $PM_{2.5}$ exposure, such as satellite-based models, hybrid models, and fixed-site monitoring present unique challenges. These techniques vary in their spatial resolution and ability to capture individual exposure, which can influence exposure contrast and potentially lead to misclassification[2,6]. Such discrepancies can affect findings across different settings, adding a layer of complexity to the interpretation of results[3,8]. Moreover, beyond the technical aspects of exposure measurement, other factors such as background pollution levels, source composition of $PM_{2.5}$, and population characteristics, such as maternal health and socioeconomic status also play a significant role in modifying observed associations[1,3]. These factors could help explain why correlations between $PM_{2.5}$ exposure and fetal growth outcomes, particularly low birth weight (LBW) and small for gestational age (SGA) are generally more consistent than associations with preterm birth (PTB)[2,5]. This variability is particularly evident in studies conducted in areas with higher pollution levels or those focused on specific sources of $PM_{2.5}$, such as wildfire-related exposure[3,15]. In these studies, the relationship between $PM_{2.5}$ exposure and fetal growth outcomes tends to be more robust and consistent, in contrast to the more variable associations with PTB[2,5].

Additionally, differences in statistical modeling techniques, the selection of covariates, and how potential confounders are handled may contribute to residual confounding and limit the comparability of effect estimates between studies[1,3]. These variations in methodology highlight the fact that while study heterogeneity may complicate direct comparisons, it does not invalidate the general trends identified in the literature. Rather, it underscores the challenges involved in assessing environmental exposures and their health effects in diverse real-world contexts.

4.4. Public health implications

The importance of ambient $PM_{2.5}$ as a modifiable environmental factor influencing maternal and perinatal health is highlighted by this review. Given the widespread exposure to air pollution, even minor changes in fetal growth caused by pollution may have significant public health implications when taken into account at the population level, despite the generally small reported effect sizes at

the individual level [1,3]. The results highlight the significance of strategies for improving air quality, especially in urban areas and during times of increased exposure like wildfire events [10,13]. From the standpoint of public health, actions to lower ambient $PM_{2.5}$ levels may improve pregnancy outcomes in addition to lowering cardiopulmonary morbidity. Integrating environmental health considerations into maternal health policies, prenatal care guidance, and public health communication may therefore help reduce the burden of preventable adverse perinatal outcomes [1,3].

4.5. Strengths and limitations of the review

This review brings together recent epidemiological evidence published between 2020 and 2025, drawing primarily on large population-based studies conducted across a range of geographic settings.The review's inclusion of studies utilizing various methods for assessing $PM_{2.5}$ exposure and analyses addressing trimester-specific exposure windows, which offer a more comprehensive view of potential times of susceptibility, is a significant strength. It is crucial to acknowledge several limitations. Significant methodological differences between studies made quantitative synthesis impossible and impeded direct comparability, particularly with regard to exposure assessment methods, analytical strategies, and outcome definitions. Additionally, most studies relied on estimates of ambient $PM_{2.5}$ exposure rather than individual-level measurements, which may have resulted in exposure misclassification. As a narrative review based on observational studies, the findings are also subject to the inherent limitations of qualitative synthesis and cannot establish causality[1,3].

5. Conclusions

This review highlights the significant association between prenatal exposure to ambient $PM_{2.5}$ and adverse pregnancy outcomes, particularly fetal growth restriction. The most consistent findings are related to low birth weight (LBW) and small for gestational age (SGA). Associations with preterm birth (PTB) are more variable, likely due to differences in exposure levels and study designs. These results underscore ambient $PM_{2.5}$ as a key environmental risk factor for maternal and perinatal health. Given the widespread nature of air pollution, even small effects at the individual level can have substantial public health implications when considered from a population perspective. Moving forward, it is crucial that future studies refine personal exposure measurement techniques, examine the timing and duration of exposure during pregnancy, and further bridge the gap between epidemiological findings and biological validation. Future research should also focus on identifying critical exposure windows and the underlying biological mechanisms, which will inform public health strategies and policy interventions.

Disclosures

Author's contribution:

Conceptualization: AK, BP, MMT, WP;

Methodology: AK, BP, MMT, JAW;

Formal analysis: AK, BP, MMT;

Investigation: NMK, AG, LO;

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Writing – original draft: AK, BP, MMT;

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