Jurnal Epidemiologi Kesehatan Komunitas
11 (3), 2026, 305-316

JEKK

# Urban Pm2.5 Exposure And Placental Dysfunction And Its Impact On Fetal Development

Murni Lestari[1*] ,Muhammad Fachri[2], Nurmalia Lusida[3]

[1]Doctoral Program in Public Health, Faculty of Public Health, Universitas Muhammadiyah Jakarta
[2]Faculty of Public Health, Universitas Muhammadiyah Jakarta
[3]Faculty of Medicine and Health, Universitas Muhammadiyah Jakarta

*Article Information : Received 01 June 2026 ; Last Revised 24 August 2026 ; Accepted 24 August 2026 ; Available Online 27 August 2026 ; Published 27 August 2026*

## ABSTRACT

**Background :** Exposure to particulate matter (PM2.5) measuring ≤2.5 micrometers in Indonesia continues to increase. Particularly in urban areas, severe air pollution is a significant issue in maternal and perinatal health due to its potential for adverse effects during pregnancy. Exposure to PM2.5 is known to be associated with impaired placental function through inflammatory mechanisms, increased oxidative stress, and vascular dysfunction, which can reduce uteroplacental perfusion and inhibit optimal fetal growth and development Objective This systematic review aims to evaluates the correlation between PM2.5 exposure and placental dysfunction and the impact of fetal development based on a systematic review based on the latest literature review.
**Methods :** A systematic literature review based on PRISMA 2020 guidelines was conducted across PubMed and Scopus, ScienceDirect databases 2021-2026, using the PECO framework.
**Result :** Of the 2,845 articles, 21 met the inclusion criteria. The most consistent research evidence indicates that exposure to PM2.5 in the first to mid-trimester of pregnancy is associated with placental inflammation, increased oxidative stress, and impaired uteroplacental perfusion. These conditions can inhibit fetal growth and development, increasing the risk of low birth weight (LBW), intrauterine growth restriction (IUGR), and preterm birth.
**Conclusion :** Exposure to PM2.5 during pregnancy is associated with placental dysfunction through inflammatory and oxidative stress mechanisms which ultimately have a negative impact on fetal growth and developmen.

**Keywords:** PM2.5 ; Pregnancy ; Placental dysfunction ; Fetal development.


DOI : https://doi.org/10.14710/jekk.v11i3.32541

*Corresponding author, murnilestari0892@gmail.com

## Introduction

Exposure to air pollution, especially PM2.5, is a global health problem, particularly in urban areas. According to the 2019 Global Burden of Disease (GBD) report, the average concentration of PM2.5 in urban areas reached 35 μg/m³ and contributed to more than 1.8 million deaths globally. More than 85% of the urban population is exposed to PM2.5 particles that exceed the annual threshold recommended by the WHO of 10 μg/m³.[1,2]

PM2.5 particles are ≤2.5 μm in size, allowing them to penetrate the lung alveoli and the blood circulation of pregnant women. This can affect the placenta's function in the exchange of oxygen, nutrients, and immunological protection between mother and fetus. Impaired placental function due to PM2.5 exposure carries the risk of various pregnancy complications. Furthermore, this condition can hinder fetal development.[3,4,5,6]

Exposure to PM2.5 is associated with an increased risk of low birth weight (LBW), preterm birth, neurodevelopmental disorders, and fetal growth restriction. LBW is known to be associated with increased neonatal mortality, impaired growth and development, and long-term health problems. Underlying mechanisms include oxidative stress, inflammation, placental dysfunction, endocrine disruption, epigenetic changes, and impaired fetal mitochondrial function, all of which impact fetal growth and development.[5,7,8,9,10]

Placental development requires an oxidative balance to support angiogenesis and trophoblast growth. Exposure to PM2.5 can increase the formation of reactive oxygen species (ROS), which triggers excessive oxidative stress. This condition causes inflammation, impaired vascularization, and placental dysfunction. Dysfunction is associated with preeclampsia, premature birth, fetal growth restriction (FGR), and intrauterine fetal death.[11, 12, 13]

When exposed to PM2.5 during pregnancy, changes in placental blood vessels can impair the supply of nutrients and oxygent to reach the fetus. This condition triggers inflammation, increased pro-inflammatory cytokines, hormonal disruption, and decreased uteroplacental perfusion. These effects can lead to fetal growth restriction and low birth weight (LBW).[14,15,16]

Furthermore, experimental studies have shown that exposure to PM2.5 particles can cross the placenta and trigger oxidative stress and inflammation, which impact fetal growth and development, alter neurodevelopment, and increase the incidence of low birth weight (LBW) in areas with high pollution exposure. These impacts are associated with oxidative stress, systemic inflammation, endocrine disruption, epigenetic changes, and mitochondrial dysfunction, all of which affect fetal growth and development.[4,14,13]

The placenta plays a crucial role as an organ for oxygen exchange, nutrition, and immunological protection between the mother and fetus. Placental development and function require an optimal oxidative balance to support angiogenesis, trophoblast differentiation, and uteroplacental vascularization. However, exposure to PM2.5 is known to disrupt homeostasis by increasing the formation of reactive oxygen species (ROS), which triggers excessive oxidative stress.[3,11,12]

PM2.5 exposure has also been reported to penetrate placental tissue and trigger local inflammation, leading to impaired vascularization, reduced uteroplacental perfusion, and placental dysfunction. These conditions contribute to various pregnancy complications, such as preeclampsia, premature delivery, fetal growth restriction (FGR), and even intrauterine fetal death.[4,13,19]

Molecularly, PM2.5 exposure has been reported to increase the production of reactive oxygen species (ROS) and the secretion of pro-inflammatory cytokines such as IL-1β and TNF-α (Lee et al., 2019; Pu et al., 2023). This inflammatory response leads to impaired trophoblast invasion, inhibited angiogenesis, and decreased placental nutrient transport. Experimental studies have also shown that urban dust particles can impair trophoblast invasion, decrease Matrix Metalloproteinase-2 (MMP-2) expression, and affect epigenetic regulation and placental cellular function. Changes in gene expression, including Insulin-like Growth Factor Binding Protein-3 (IGFBP3), indicate the involvement of

inflammatory mechanisms and oxidative stress in impaired fetal growth caused by air pollution exposure.[5,12,15,20]

Although numerous studies have linked PM2.5 exposure to placental dysfunction and impaired fetal development, the molecular mechanisms linking oxidative stress, inflammation, and placental damage remain incompletely understood. Recent evidence shows that PM2.5 and polycyclic aromatic hydrocarbons (PAH) compounds are able to induce the endoplasmic reticulum (ER) as a major mediator between oxidative stress and inflammatory responses.[13,17,20]

Endoplasmic reticulum (ER) activation is known to activate the unfolded protein response (UPR) pathway, increase chronic inflammation, inhibit trophoblast invasion, and disrupt placental angiogenesis. However, the integration of ER stress mechanisms into placental dysfunction and its impact on fetal development due to PM2.5 exposure still requires a comprehensive scientific synthesis.[12,15,19]

Although the association between PM2.5 exposure and adverse pregnancy outcomes has been widely reported, most research has focused on epidemiological associations and partial biomarkers of inflammation. To date, there has been no comprehensive synthesis integrating the relationships between oxidative stress, inflammation, endoplasmic reticulum stress (ER stress), trophoblast dysfunction, and placental insufficiency as a single, coherent pathophysiological pathway contributing to impaired fetal development. Furthermore, recent research indicates variations in molecular mechanisms based on exposure duration, particulate matter composition, and gestational period, so the dominant biological mechanism remains a matter of scientific debate.[2,3,16]

The inclusion criteria for this systematic review encompass relevant scientific syntheses and human studies that evaluate urban PM2.5 exposure during the procenception and gestational periods in relation to fetal placental dysfunction, oxidative stress, inflammation, and endoplasmic reticulum stress, inflammation, and endoplasmic reticulum stress. Additionally, eligible studies must investigate the impacts of such exposure on trophoblast function, angiogenesis, and uteroplacental vascularization, while reporting consistent fetal developmental outcomes including low birth weight, premature birth, and fetal growth restriction.

## Methods

This study was a systematic review with narrative synthesis. Narrative synthesis was chosen because articles that met the criteria showed heterogeneity in study design, population characteristics, exposure estimation methods, outcome definitions, effect sizes, and analytic approaches. Reporting was structured according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 principles, while the quality of evidence was assessed descriptively and qualitatively, referring to the principles of appraisal of observational studies and the JBI guidelines.[31]

The review questions were formulated using the PECO framework. The population included preconception, pregnant women, placentas, and fetuses. Exposures included PM2.5 from air pollution during pregnancy. Comparisons included lower exposure levels or different exposure periods. Outcomes included placental dysfunction and fetal developmental disorders such as low birth weight (LBW) and prematurity.

Systematic literature search was conducted using PubMed and Science Direct databases. The literature search was conducted using combinations of search terms and medical subject headings including urban PM2.5, exposure, fine particulate matter, air pollution, pregnancy, gestational period, preconception, placental dysfunction, oxidative stress, inflammation, endoplasmic reticulum stress, trophoblast function, fetal development, low birth weight, premature birth, and fetal growth restriction.

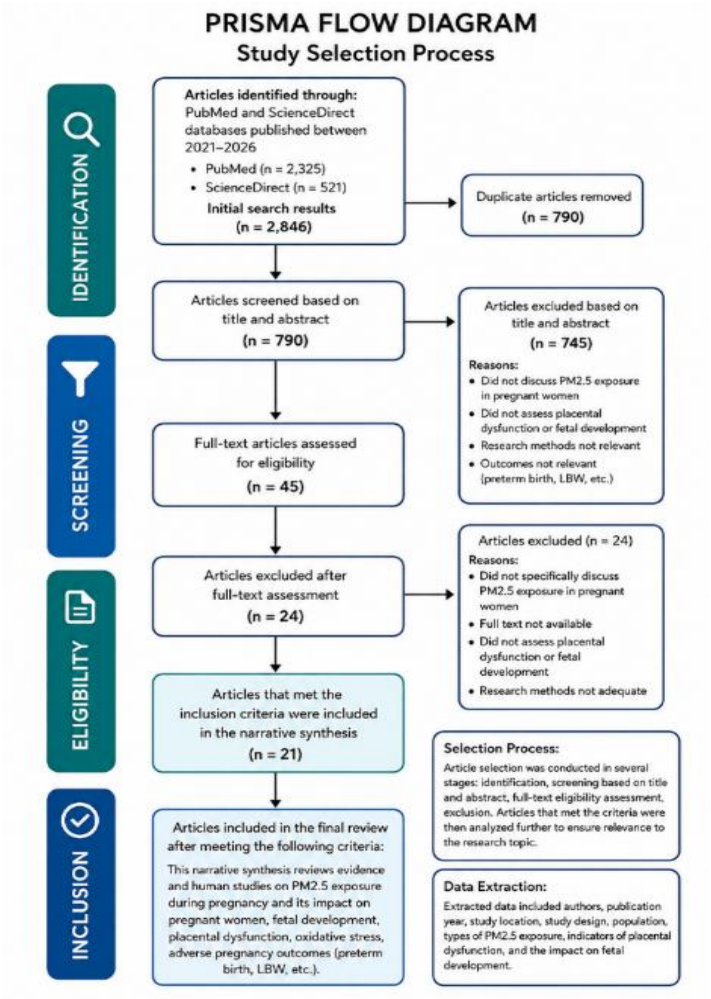
Articles were limited to English-language publications published between 2021 and 2026, with adequate research methods and results. The study focused on urban PM2.5 exposure, placental dysfunction, and its impact on fetal development. The initial search

identified 2,846 articles. After screening titles and absracts, we removed duplicate records from the initial 790 articles, leaving 45 studies that met the criteria for full review. After evaluating the methodology and relevance of the research outcomes, 21 final articles were included in the narrative synthesis.

Article selection was carried out in stages through identification, title and abstract screening, and full-text eligibility assessment based on inclusion and exclusion criteria. Eligible articles were then further analyzed to ensure relevance to the research title. Extracted data included author(s), year of publication, study location, study design, population, type of PM2.5 exposure, indicators of placental dysfunction, and its impact on fetal development. To minimize interpretation bias, the results of each study were analyzed by outcome group and compared based on the consistency of the relationship and methodological quality.

## A. **Colour illustrations**

Full color illustrations ar permitted for online publication,however, hard copy proceedings are resctricted to grayscale unless prior arrangements are made with the event host. Authors must verify the printing specifications directly with the organizers. If physical copies are printed in black and white, all color references within captions and body text must be excised. Furthermore, graphics should be designed to ensure data clarity is maintained when desaturated to monochrome.



## Result

A total of 21 articles met the inclusion criteria and were synthesized. Regarding Urban PM2.5 Exposure and Placental Dysfunction and Its Impact on Fetal Development, the articles were published between 2021 and 2026. Based on the results of the literature, it shows that research on urban PM2.5 exposure in pregnancy is dominated by cohort studies, trophoblast experiments, animal models, and systematic reviews and meta-analyses.

Most studies report that PM2.5 is associated with oxidative stress, placental inflammation, DNA damage, trophoblast apoptosis, and changes in placental microarchitecture. Research by Chu et al. 2024, Gong et al. 2022, and Erlandsson 2026 Studies have shown that PM2.5 triggers inflammation and cellular damage in the placenta. Much of the literature also indicates that placental dysfunction caused by PM2.5 exposure directly impacts fetal development and growth. Studies by Deyssenroth et al. (2021) and Kaur et al. (2022) found changes in placental gene expression associated with reduced birth weight and fetal metabolic disorders. Studies by Tao et al. (2022) and Wang et al. (2026) showed that PM2.5 can cause fetal growth restriction (FGR) through impaired spiral artery remodeling and uteroplacental inflammation. Furthermore, meta-analyses by Parasin et al. (2024) and Wang et al. (2023) strengthen the evidence that prenatal PM2.5 exposure increases the risk of low birth weight, preterm delivery, and adverse birth outcomes.

**Table 1. Characteristics and Main Findings of Non-Primary Studies**

| No | Reference | Study Design | Research Focus | Key Findings |
|---|---|---|---|---|
| 1 | Holme et al., 2024 | Mechanistic Review | PM2.5 and Neurodevelopment | Prenatal PM2.5 exposure is associated with impaired neurodevelopment. |
| 2 | Johnson et al., 2021 | Narrative Review | Prenatal Particulate Matter Exposure | PM2.5 exposure during pregnancy increases the risk of developmental disorders in children. |
| 3 | Jakia et al., 2023 | Review | Placental Dysfunction | Oxidative stress induced by PM2.5 contributes to placental dysfunction and adverse fetal development. |
| 4 | Parasin et al., 2024 | Systematic Review and Meta-analysis | Prenatal PM2.5 Exposure | Prenatal PM2.5 exposure increases the risk of low birth weight (LBW). |
| 5 | Sun et al., 2022 | Review | PM2.5 and Metabolic Diseases | Prenatal PM2.5 exposure may program metabolic diseases in offspring later in life. |
| 6 | Wang et al., 2023 | Cohort Meta-analysis | Prenatal PM2.5 Exposure | PM2.5 exposure during pregnancy is associated with an increased risk of adverse pregnancy outcomes. |
| 7 | BMJ PRISMA, 2021 | Guideline | Reporting of Systematic Reviews | Provides standardized guidance for reporting systematic reviews. |

**Tabel 1** Summarizes the key studies included in this literature review regarding the effect of prenatal PM2.5 exposure on prenatal function and fetal development. Overall, the evidence consistently demonstrates that maternal exposure to PM2.5 during pregnancy is associated with adverse pregnancy and neonatal outcomes through biological mechanisms such as oxidative stress, inflammation, vascular dysfunction, and impaired placental function, These mechanisms contribute to fetal growthrestriction, low birth weight, neurodevelopmental imparment, and an

increased risk of metabolic disorders later in life. The inclusion of the PRISMA 2021 guideline also ensures that this review follows a transparent and standardized reporting process, thereby improving the quality and reproducibility of the literatur review.

**Table 2.** Primary Study.

| No. | Reference | Study Design | Exposure | Fetal Outcome | Key Findings |
|---|---|---|---|---|---|
| 1 | Chen et al., 2025 | Cohort Study | Prenatal PM2.5 Exposure | Impaired Fetal Growth | PM2.5 exposure alters fetal growth. |
| 2 | Deyssenroth et al., 2021 | Molecular Cohort Study | Early Pregnancy PM2.5 Exposure | Low Birth Weight | PM2.5 exposure alters placental gene networks associated with reduced birth weight. |
| 3 | Gong et al., 2022 | Human Experimental Study | PM2.5 Exposure | Placental Inflammation | PM2.5 triggers early placental inflammation. |
| 4 | Kaur et al., 2022 | Birth Cohort Study | PM2.5 Exposure During Pregnancy | Placental Metabolic Dysfunction | PM2.5 alters placental lipid metabolism genes. |
| 5 | Lin et al., 2023 | Repeated-Measures Study | Oxidative PM2.5 Exposure | Low Fetal Weight | PM2.5 increases fetoplacental vascular resistance. |
| 6 | Li et al., 2023 | Cellular Experimental Study | PM2.5 Exposure | Adverse Pregnancy Outcomes | PM2.5 induces trophoblast apoptosis. |
| 7 | Li & Zhang, 2024 | Causal Analysis Study | Fetal PM2.5 Exposure | Adverse Birth Outcomes | PM2.5 exposure worsens birth outcomes. |
| 8 | Wang et al., 2024 | Experimental Study | Gestational PM2.5 Exposure | Impaired Fetal Development | PM2.5 inhibits trophoblast syncytialization. |
| 9 | Wang et al., 2026 | Animal Experimental Study | Maternal PM2.5 Exposure | Fetal Growth Restriction (FGR) and Placental Alterations | PM2.5 causes uterine inflammation. |
| 10 | Yu et al., 2025 | Biomarker Cohort Study | PM2.5 Exposure | Adverse Pregnancy Outcomes | PlGF mediates the effects of PM2.5 on adverse outcomes. |
| 11 | Zhou et al., 2024 | Observational Study | Early Pregnancy PM2.5 Exposure | Maternal Hormonal Disturbances | PM2.5 affects maternal thyroid hormones. |

**Table 2.** Primary Study. Summarizes evidence from 11 studies examining the effects of prenatal PM2.5 exposure on fetal outcomes. Across various study designs, the findings consistently show that PM2.5 impairs placental function through inflammaetion, oxidative stress, trophoblast apoptosis, vascular dysfunction, and altered gene expression. These mechanisms are associated with adverse outcomes, including impaired fetal growth, low birth weight (LBW), fetal growth restriction (FGR), and other pregnancy complications. Resent evidence also suggests that Placental growth factor (PIGF) and maternal and maternal thyroid hormone alterations these effects.

**Tabel 3. Synthesis based on output**

| No | References | Fetal/Pregnancy utcomes | Main Mechanisms |
|---|---|---|---|
| 1 | Tao et al., 2022; Wang et al., 2026 | Fetal Growth Restriction (FGR) | Impaired spiral artery remodeling and uterine inflammation |
| 2 | Deyssenroth et al., 2021; Parasin et al., 2024 | Low Birth Weight (LBW) | Placental gene dysfunction and oxidative stress |
| 3 | Wang et al., 2024; Singh et al., 2026 | Impaired Fetal Development | Trophoblast dysfunction and placental inflammation |
| 4 | Holme et al., 2024 | Neurodevelopmental Impairment | Oxidative stress and polycyclic aromatic hydrocarbons (PAHs) exposure |
| 5 | Gong et al., 2022; Erlandsson et al., 2026 | Placental Dysfunction | Inflammation, DNA damage, and alterations in placental microarchitecture |

**Tabel 3.** Synthesis based on output. Summarizes the major fetal and gestational effects linked to prenatal exposure to PM2.5. Across the reviewed articles PM2.5 consistently contributes to fetal groth restriction, low birth weight, impaired fetal development, neurodevelopmental impairment, and placental dysfunction. These outcomes are primarily driven by placental inflammation, oxidative stress, vascular dysfunction, oxidative stress, vascular dysfunction, trophoblast impairment, and altered placental gene expression.

## A. PM2.5 Exposure During Pregnancy

A literature review indicates that inhalation of urban PM2.5 during pregnancy plays a significant role in placental dysfunction through mechanisms such as oxidative stress, inflammation, oxidative DNA damage, trophoblast apoptosis, uteroplacental vascular disorders, and altered placental gene expression. Various cohort studies, trophoblast cell experiments, animal models, and combined studie have revealed that PM2.5 exposure before birth is associated with fetal growth restriction (FGR), low birth weight, preterm delivery, impaired fetal development, and offspring neurodevelopment. PM2.5-induced placental dysfunction has also been reported to involve impaired spiral artery remodeling, fetoplacental vascular resistance, and activation of prooxidative inflammatory pathways, which impair uteroplacental perfusion and increase the risk of adverse birth outcomes.[3,6,8,20]

## B. Placental Dysfunction

Placental dysfunction has consistently been linked to impaired placental structure and function during pregnancy. Several cohort studies over the past five years have reported that PM2.5 exposure in the first and second trimesters is associated with increased placental inflammation, oxidative stress, impaired angiogenesis, and reduced uteroplacental perfusion. Studies in China and Europe have shown that PM2.5 exposure correlates with changes in placental biomarkers, such as increased proinflammatory cytokines, trophoblast dysfunction, and placental vascular damage, which contribute to placental insufficiency and impaired fetal growth.[3,8]

These results indicate that the early to mid-pregnancy period is the most vulnerable period to the effects of air pollution on the placenta. This phase involves implantation, vascular remodeling, and the establishment of the uteroplacental circulation, which is crucial for fetal growth. PM2.5 exposure can trigger the formation of reactive oxygen species (ROS), systemic inflammation, and endothelial dysfunction, which impede the transfer of oxygen and nutrients from the mother to the fetus. Biologically, these mechanisms support the link between air pollution and fetal growth restriction, low birth weight, and prematurity.[1,12,13]

Furthermore, several recent studies have also demonstrated epigenetic changes and impaired placental mitochondrial function due to chronic PM2.5 exposure. These alterations

can impact gene function related to fetal development and vascular perfomance in the placental. Although results vary between studies due to differences in exposure measurement methods, population characteristics, and local pollutant levels, the overwhelming evidence continues to indicate that PM2.5 is a significant risk factor for placental dysfunction and impaired fetal development.[9,11]

### C. Fetal Development

Fetal development has been consistently linked to exposure to urban PM2.5 during pregnancy, particularly in the first and second trimesters. Several cohort studies have reported that PM2.5 exposure is associated with an increased risk of fetal growth restriction, small for gestational age (SGA), low birth weight, prematurity, and impaired fetal organ development. These risks are increased in pregnant women living in urban areas with high levels of air pollution and chronic exposure to fine particulate matter[7,8]

PM2.5 is a very small particle that can carry toxic compounds such as heavy metals, polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds, and free radicals. This combination can trigger systemic inflammation, oxidative stress, and placental vascular disruption, which can reduce the supply of oxygen and nutrients to the fetus. As a result, fetal cell growth and differentiation can be disrupted, increasing the risk of intrauterine growth restriction and preterm birth.[9,11,12,21]

Fetal developmental disorders caused by PM2.5 have also been linked to epigenetic changes and impaired neurocognitive development. Several studies have found that exposure to air pollution during pregnancy can affect the expression of genes involved in nervous system development, metabolic function, and fetal immune responses. In addition to impacts in the neonatal period, these effects are thought to persist into childhood, leading to an increased risk of respiratory disorders, developmental delays, and metabolic diseases later in life.[9,11]

The biological plausibility of this association is strengthened by the discovery of black carbon particles and PM2.5 components in placental tissue, umbilical cord blood, and maternal-fetal circulation. These findings indicate that air pollution particles can cross the placental barrier and directly affect the fetus's biological environment. With increasing urbanization and global air pollution, PM2.5 exposure has become an important environmental factor to consider in efforts to protect maternal health and fetal development.[9,5]

## Discussion

The finding of this systematic review indicate that maternal exposure to urban PM2.5 is consistently associated with placental dysfunction and adverse fetal outcomes, including fetal growth restriction (FGR). Low birth weight (LBW), preterem birth, and impaired fetal development. Although the strength of these associations varied across studies, there was substantion agreement that oxidative stress, inflammation, trophoblast dysfunction, and impaired placental vascular function are central mechanisms linking PM2.5 exposure with disrupted fetal growth.[5,6,8] The consistency of findings across cohort studies, experimental models, and systematic reviews strengthens the biological plausibility of this relationship and suggests that placental dysfunction is a key mediator between maternal environmental exposure and adverse pregnancy outcomes.

Inter-study variabelity likely reflects methological and biological differences rether that conflicting evidence. Differences in exposure assessment techniques, regional PM2.5 composition, population characteristics, and critical exposure periods may explain the variation in reported effect magnitudes. PM2.5 exposure in early pregnancy alters placental gene networks associated with reduced bith weight, whereas Tao et al. (2022) and Wang et al. (2026) identified impaired spiral artery remodeling and uterine inflammation as key factors driving fetal growth restriction. Similarly, Lin et al. (2023) reported increased fetoplacental vascular resistance, while Wang et al. (2024) found that PM2.5 disrupts trophoblast syncytialization via the progranulin/mTOR signaling pathway. Overall, these findings

indicate that PM2.5 acts through multiple interconnected biological pathways, with the first and second trimesters appearing to be the most vulnerable periods, as placental implantation, angiogenesis, and uteroplacental vascular remodeling are actively occurring during these stages.[3,11.]

The biological mechanisms underlying these associations are supported by accumulating experimental evidence. Fine particulate matter and its toxic constituents, including polycyclic aromatic hydrocarbons (PAHs) and transition metals, can penetrate maternal circulation and accumulate in placental tissue, leading to excessive production of reactive oxygen species (ROS). Increased oxidative stress subsequently activates inflammatory pathways, including NF-κB signaling, elevates pro-inflammatory cytokines such as TNF-α and IL-6, disrupts mitochondrial function, and impairs trophoblast proliferation and angiogenesis [ 5,11,20]. Emerging evidence further indicates that PM2.5 induces endoplasmic reticulum stress, epigenetic alterations, repression of IGFBP3, changes in maternal thyroid hormones, and altered placental growth factor (PlGF), suggesting that placental dysfunction results from the interaction of several molecular pathways rather than a single pathogenic mechanism.[17,29,31] These mechanisms provide a biologically coherent explanation for the observed associations between PM2.5 exposure and impaired fetal growth.

The results of this literature review indicate that the association between PM2.5 exposure during pregnancy and impaired fetal development is supported by consistent biological mechanisms. PM2.5 exposure increases the formation of reactive oxygen species (ROS), triggers oxidative stress and inflammation, and disrupts trophoblast function and uteroplacental perfusion. These condition lead to reduced oxygen and nutrient tranfer to the fetus,thereby increasing the risk of fetal growth restriction (FGR), low birt weight (LBW), preterm birth, and impaired fetal development.[5,6,13,21]

This literature review is supported by consistent findings from cohort studies, experimental research, and meta-analyses. However, most human studies remain observational; thus, they do not fully establish a causal relationship and are potentially subject to confounding factors—such as smoking habits, socioeconomic status, pollutant exposure, and variations in PM2.5 exposure measurement methods, population characteristics, and biomarkers used. Therefore, prospective studies employing more accurate exposure measurements and biomolecular approaches are still needed to strengthen the evidence for a causal link between PM2.5 exposure, placental dysfunction, and impaired fetal development.[8,18,26]

## Conclusions

This systematic review demonstrates that prenatal exposure to urban PM2.5 is consistently associated with placental dysfunction and adverse vetal development. The evidence indicates thet the firdt and second trimesters represent the most vulnerable periods of pregnancy, as they coincide with placental implantation, trophoblast invasion, angiogenesis, and uteroplasental vascular remodeling. PM2.5 exposure during these critical windows promotes oxidative stress, inflammation, trophoblast dysfunction, vascular impairment, and altered plasental gene expression, resulting in reduced oxygen and nutrient transport to the fetus. These biological mechanisms contribute to fetal growth restriction (FGR), low birth weight (LBW), preterm birth, and impaired neurodevelopment. The findings highlight the imporance of reducing maternal exposure to PM25 throgh strengthened air quality policies, environmental monitoring, and targeted public health interventions for pregnant women, particularly in highly polluted urban areas. Future prospective studies integrating molecular biomarkers and standardized exposure assessment are needed to further clarify the causal pathways and support evidence based stategies for protecting maternal and fetal health

**Acknowledgement**

The author expresses his gratitude to the supervisor who has provided direction and constructive input so that this literature review study can be completed well.

**References**

1. Jakia. Yixue,Q et al. "Involvement of oxidative stress in placental dysfunction, the pathophysiology of fetal death and pregnancy disorders". Jul. 5, 2023. Doi: https://doi.org/10.1530/rep-22-0278
2. Sun J, Liu H, Zhang C, Liu X,Sun X, et al. Predisposed obesity and long-term metabolic diseases from maternal exposure to fine particulate matter ($PM_{2.5}$) — A review of its effect and potential mechanisms. Research.2022;310. Sciences.doi https://doi.org/10.1016/j.lfs.2022.121054
3. Chu M, Yang J, Gong C, et al. Effects of fine particulate matter mass and chemical components on oxidative DNA damage in human early placenta**.** *Environmental Research*. 2024;263(Pt 2):120136 doi https://doi.org/10.1016/j.envres.2024.120136
4. Johnson N, M. Hoffmann A.R, Behlen J.C, Lau C, Pendleton D et al, " Air pollution and children's health—a review of adverse effects associated with prenatal exposure from fine to ultrafine particulate matter" 2021 Jul 12;26(1):72. Environmental Health and Preventive Medicine. Doi: https://doi.org/10.1186/s12199-021-00995-5
5. Gong C, Chu M, Yang J, et al. Ambient fine particulate matter exposures and human early placental inflammation.*Environmental Pollution*. 2022;315:120446.PMID:36265729. doi https://doi.org/10.1016/j.envpol.2022.120446
6. Wang Y, Chen Z, Jien Li, Wan T, et al. Gestational exposure to PM2.5 disrupts fetal development by P. Nichapa Parasin. A, Teerachai et al. Prenatal PM2.5 Exposure and Its Association with Low Birth Weight: A Systematic Review and meta.Analysis. June. 21,2024 doi: https://doi.org/10.3390/toxics12070446
7. Chen X, Lin Y, YiLu, et al. Associations of prenatal exposure to air pollution with fetal growth trajectories phenotype in a cohort study: Implications for child neurodevelopmen. 27 October 2025. https://doi.org/10.1016/j.jes.2025.10.043
8. Deyssenroth MA, Rosa MJ, Eliot MN, et al. Placental gene networks at the interface between maternal PM2.5 exposure early in gestation and reduced infant birthweight. *Environmental Research*. 2021;199:111342. PMID: 34015297 doi https://doi.org/10.1016/j.envres.2021.111342
9. Erlandsson L, Hirschmugl B, Hansson E, et al. Urban air pollution disrupts placental microarchitecture and shifts hofbauer cells towards a pro-inflammatory state. February 2026 doi; https://doi.org/10.1016/j.jes.2025.03.043
10. L.youngjie et al. "Maternal urban particulate matter (SRM 1648a) exposure disrupted the cellular immune homeostasis during early life: The potential attribution of altered placental transcriptome profile".*ScienceDirect*. Feb. 20, 2024. doi: https://doi.org/10.1016/j.scitotenv.2023.169432
11. Kaur K, Lesseur C, Deyssenroth MA, et al. PM2.5 exposure during pregnancy is associated with altered placental expression of lipid metabolic genes in a US birth cohort**.** *Environmental Research*. 2022;211:113066.PMID: 35248564. https://doi.org/10.1016/j.envres.2022.113066
12. Lin W, Lai Y, Zhuang S, et al. The effects of prenatal PM2.5 oxidative potential exposure on feto-placental vascular resistance and fetal weight: A repeated-measures study**.** *Environmental Research*. 2023;234:116543.PMID: 37406720 doi https://doi.org/10.1016/j.envres.2023.116543
13. Li L, Zhang X. The causal impact of fetal exposure to PM2.5 on birth outcomes: Evidence from rural China. April 2024, 101380.

https://doi.org/10.1016/j.ehb.2024.101380
14. Wang Y, Chen Z,Jie Lie, et al. Gestational exposure to $PM_{2.5}$ disrupts fetal development by suppressing placental trophoblast syncytialization via progranulin/mTOR signaling 2024;921:171101 doi https://doi.org/10.1016/j.scitotenv.2024.171101
15. Southerland V, Braoer M, Mohegh A, et al. Global urban temporal trends in fine particulate matter ($PM_{2·5}$) and attributable health burdens: estimates from global datasets. February 2022, Pages e139-e146.doi; https://doi.org/10.1016/S2542-5196(21)00350-8
16. Wang S, Chen H, Singh A, et al. Maternal $PM_{2.5}$ exposure causes fetal growth restriction and alters placental morphology: Role of uterine inflammation. March 2026, 109177.doi; https://doi.org/10.1016/j.reprotox.2026.109177
17. Sing S, Goel I, Rana A, Gul A et al. "IGFBP3 repression driven by inflammation links air pollution to placental and developmental defects" March.24. 2026 doi: https://doi.org/10.1038/s44321-026-00403-x
18. Wang Y, Chen Z,Jie Lie, et al. Gestational exposure to $PM_{2.5}$ disrupts fetal development by suppressing placental trophoblast syncytialization via progranulin/mTOR signaling 2024;921:171101 doi https://doi.org/10.1016/j.scitotenv.2024.171101
19. Requia W, Kill E F, Papatheodorou, et al. Critical exposure windows for wildfire $PM_{2.5}$ during pregnancy and preterm birth in Brazil. 1 June 2026, 128048.doi; https://doi.org/10.1016/j.envpol.2026.128048
20. Holme J A, Myhre O, ovrevik J,et al. Adverse neurodevelopment in children associated with prenatal exposure to fine particulate matter ($PM_{2.5}$) – Possible roles of polycyclic aromatic hydrocarbons (PAHs) and mechanisms involved. December 2024, 108718.doi; https://doi.org/10.1016/j.reprotox.2024.108718
21. Tao S, Zhang X, Tian F, et al. Maternal exposure to ambient PM2.5 causes fetal growth restriction via the inhibition of spiral artery remodeling in mice**.** *Ecotoxicology and Environmental Safety*. 2022;237:113512.PMID: 35429798.doi https://doi.org/10.1016/j.ecoenv.2022.113512
22. Li S, Li L, Zhang C, et al. PM2.5 leads to adverse pregnancy outcomes by inducing trophoblast oxidative stress and mitochondrial apoptosis via KLF9/CYP1A1 transcriptional **axis.** *eLife*. 2023;12:e85944.PMID: 37737576 doi https://doi.org/10.7554/eLife.85944
23. Lan Q, Henderson S, Coker E, et al. Heatwaves and $PM_{2.5}$ from wildfire smoke, non-wildfire sources, and all sources in relation to preterm birth: a nationwide cohort study in Canada. March 2026.doi; https://doi.org/10.1016/j.envint.2026.110185
24. Ling Liu R, Lei xu J, Ling hu Y,et al. Association between $PM_{2.5}$ components and poor ovarian response in assisted reproductive technology patients: A retrospective cohort study identifying sensitive exposure windows in China. February 2025.doi; https://doi.org/10.1016/j.envint.2025.109321
25. Naav A, Erlandsson L, Isaxon C, et al. Urban PM2.5 Induces Cellular Toxicity, Hormone Dysregulation, Oxidative Damage, Inflammation, and Mitochondrial Interference in the HRT8 Trophoblast Cell Line. 2020 Mar 12:11:75.doi; https://doi.org/10.3389/fendo.2020.00075
26. Parasin N; Amnuaylojaroen teerachai, saokaew S Prenatal PM2.5 Exposure and Its Association with Low Birth Weight: A Systematic Review and Meta-Analysis Jun 21;12(7):446. https://doi.org/10.3390/toxics12070446
27. Parru S, Heso A.C, Llop S, et al. Association between the external urban

exposome and systemic inflammation in pregnant women. 15 June 2026, 124267 doi; https://doi.org/10.1016/j.envres.2026.124267

28. Wang W, Mu S, Yan W, et al. Prenatal PM2.5 exposure increases the risk of adverse pregnancy outcomes: evidence from meta-analysis of cohort studies. *Environmental Science and Pollution Research*. 2023;30(48):106145–106197. PMID: 3772339 doi https://doi.org/10.1007/s11356-023-29700-5
29. Yu Y, Fan W, Ni J, et al. Maternal placental growth factor mediates the association between $PM_{2.5}$ and its constituents with adverse pregnancy outcomes. 5 October 2025, 139598.doi; https://doi.org/10.1016/j.jhazmat.2025.139598
30. Yin L, Liu M, Jiang H, et al. Prenatal exposure to microplastics and biomarkers of renal dysfunction in umbilical cord blood: Evidence from a birth cohort in China. 15 November 2025. 119318.doi; https://doi.org/10.1016/j.ecoenv.2025.119318
31. Zhou J, Yu S, Wang C, et al. Relationship between environmental $PM_{2.5}$ exposure in early pregnancy and thyroid hormone levels in pregnant women. 1 November 2024, 117182 doi; https://doi.org/10.1016/j.ecoenv.2024.117182
32. BMJ,Mathew. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. 2021; 372.doi; https://doi.org/10.1136/bmj.n71