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# Relationship between environmental pollution exposure and preterm birth: Mediating role of placental oxidative stress

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**Abstract**

Environmental pollution is a known risk factor for preterm birth, with oxidative stress in the placenta potentially mediating this relationship. This study aimed to explore the link between maternal exposure to environmental pollution and preterm birth, with placental oxidative stress as a potential mediator. A hospital-based analytical cross-sectional study was conducted at Babylon Teaching Hospital, Iraq, from November 2024 to March 2025. The study included 240 postpartum women, divided into preterm and postpartum groups. Environmental exposure was measured using air quality data, while placental oxidative stress was evaluated with biomarkers (malondialdehyde and superoxide dismutase). The data were analyzed using descriptive statistics, chi-square tests, t-tests, logistic regression and mediation analysis using the PROCESS macro. Preterm birth was associated with higher levels of environmental pollutants such as PM2.5 (42.6 ± 9.1 μg/$m^3$ vs. 31.4 ± 7.6 μg/$m^3$), $NO_2$ (30.8 ± 6.4 ppb vs. 23.2 ± 5.9 ppb), and $SO_2$ (18.5 ± 4.8 ppb vs. 13.7 ± 3.9 ppb) compared to term births. Placental MDA levels were higher (5.61 ± 1.25 vs. 3.12 ± 0.96 nmol/mg), and SOD activity was lower (9.18 ± 1.96 vs. 12.74 ± 2.41 U/mg protein) in the preterm group. Mediation analysis revealed that placental oxidative stress partially mediated the relationship between PM2.5 exposure and preterm birth (36.8% mediation). Maternal exposure to environmental pollutants is associated with an increased risk of preterm delivery, with oxidative stress playing a mediating role. Interventions aimed at reducing exposure to pollution and promoting maternal health can mitigate these risks.

**Keywords:** Environmental pollution, Preterm birth, Oxidative stress, Placental biomarkers, Maternal health.

## Introduction

Preterm start (PTB), defined as childbirth happening earlier than 37 weeks of gestation, is a primary purpose of neonatal morbidity and mortality worldwide (1). According to the World Health Organization (2023), a predicted 13.4 million infants had been born preterm in 2020, with complications from prematurity contributing considerably to under-five mortality (2). Despite significant advances in perinatal care, the etiology of PTB remains multifactorial and incompletely understood. Among the growing frame of proof, environmental factors, mainly publicity to air pollution, have emerged as key individuals to destructive pregnancy outcomes, which include preterm labor and delivery (3,4). Several epidemiological studies have said an enormous association between maternal publicity to environmental pollutants—consisting of particulate count number (PM2. Five and PM10), nitrogen dioxide ($NO_2$), and ozone ($O_3$) and expanded danger of PTB. A meta-analysis determined that publicity to ambient PM2.Five for the duration of being pregnant was associated with a 10–20% increased risk of PTB, depending on the exposure window (5). Similarly, indication tested that exposure to visitors-related air pollution in early pregnancy heightened the threat of spontaneous PTB (6). While the affiliation is properly mounted, the organic mechanisms through which air pollution affects pregnant effects are still beneath research. The recent studies have proposed oxidative pressure as a key mediator within the pathway linking environmental exposure to PTB. Placental oxidative strain, characterized by an imbalance between reactive oxygen species (ROS) and antioxidant defenses, is increasingly recognized for its position in placental dysfunction, infection, and premature rupture of membranes (7,8). Experimental models have shown that air pollutants can move the placental barrier and cause the technology of ROS, mainly mobile and molecular changes adverse to fetal development (9). Furthermore, maternal exposure to environmental toxicants has been related to altered expression of oxidative stress markers and antioxidant enzymes in placental tissues, probably disrupting regular placental features (10). The mediating role of placental oxidative pressure offers a essential hyperlink in information how environmental pollutants might also immediately affect pregnancy consequences. Studies demonstrate that expanded placental oxidative stress biomarkers are extensively

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related to both pollutant publicity tiers and earlier gestational age at the start (11,12). This indicates that oxidative strain no longer best effects from pollutant exposure but additionally serves as a potential mechanism leading to PTB. As such, analyzing this mediating function can offer novel insights into prevention strategies and therapeutic interventions geared toward reducing the weight of preterm birth. Given the public health importance of both environmental pollutants and preterm birth and the emerging evidence on placental oxidative strain as a capacity mediator, this takes a look at aims to explore the connection between environmental pollutants publicity and preterm start, with a specific consciousness on the mediating position of placental oxidative pressure.

## Material and Methods

### Study design and sitting

A hospital-based analytical cross-sectional study was conducted to research the connection between maternal exposure to environmental pollutants and the prevalence of preterm beginning, with placental oxidative pressure explored as a capacity mediating aspect. The observation was accomplished at Babylon Teaching Hospital for Women and Children, a main tertiary healthcare facility in Babylon Governorate, Iraq, presenting comprehensive obstetric and neonatal care to a huge urban and peri-city populace. The study was be conducted over a 4-month duration, from the 10th November 2024 to the 23rd March 2025, chosen to coincide with seasonal fluctuations in environmental pollution and to make sure good enough recruitment of each preterm and term delivery.

### Study population and sampling

The study population covered postpartum girls who were brought to the health facility throughout the statistics series length. The pattern size changed into decided based totally on an anticipated prevalence of preterm beginning related to pollution publicity suggested in the preceding literature. Using G*Power software (model 3.1) with a self-assurance level of 95%, a statistical strength of 80%, and an impact length (odds ratio) of 2.0 suggested by Faul et al., the minimal required sample change was calculated to be 216 (13). To compensate for ability exclusions and non-responses, the sample was increased to 240 ladies, with 120 participants inside the preterm start institution (gestational age <37 weeks) and 120 inside the term start institution (≥37 weeks).

### Study instruments

The examination utilized an aggregate of environmental publicity exams, organic sampling, and established questionnaires to collect statistics on pollution publicity, placental oxidative strain markers, and preterm beginning results. Environmental pollutants publicity turned into measured the use of air fine monitoring facts from neighborhood environmental businesses (14), at the same time as placental oxidative stress changed into assessed through laboratory evaluation of biomarkers together with malondialdehyde (MDA) and superoxide dismutase (SOD) activity in placental tissue samples (15). A structured questionnaire was administered to participants to gather demographic, scientific history, and lifestyle records. The questionnaire was developed primarily based on a literature review and reviewed by way of a panel of professionals to make certain content material validity. Reliability became confirmed through a pilot take a look at concerning 20 pregnant girls no longer protected in the predominant have a look at, yielding a Cronbach's alpha of 0.82, indicating sturdy inner consistency.

### Data collection procedure

Data were collected through the use of an aggregate of established interviews, scientific record critiques, geospatial mapping, and laboratory evaluation. A revalidated, researcher-administered questionnaire turned into used to gather sociodemographic information, antenatal records, lifestyle behaviors (inclusive of smoking and nutritional styles), and environmental publicity signs, inclusive of residential proximity to commercial zones and high-visitors' roadways. Residential addresses had been geo-referenced to quantify man or woman publicity degrees to environmental pollutants, especially particulate depend (PM2.5 and PM10), nitrogen dioxide ($NO_2$), and sulfur dioxide ($SO_2$).

Placental tissue samples were obtained within half an hour of transport beneath sterile situations, without delay frozen in liquid nitrogen, and saved at −80°C

until evaluation. Oxidative stress biomarkers—malondialdehyde (MDA) as a marker of lipid peroxidation and superoxide dismutase (SOD) as an endogenous antioxidant enzyme—were quantified by the usage of confirmed spectrophotometric assay kits in line with producer protocols.

Inclusion standards encompassed women elderly 18 to 45 years, with singleton pregnancies, who were permanent citizens of the Babylon vicinity for at least twelve months before transport, and who furnished informed consent. Women were excluded if they had pre-existing persistent scientific conditions (e.g., diabetes mellitus, hypertension, autoimmune diseases), multiple gestations, recognized congenital fetal anomalies, or incomplete clinical records.

## Statistical methods

Data were entered, cleaned, and analyzed through the usage of IBM SPSS Statistics model 26. Descriptive data was computed to summarize the participants' demographic and medical traits. Chi-square and impartial samples t-assessments were used to compare categorical and non-stop variables between term and preterm organizations, respectively. Binary logistic regression was achieved to evaluate the association among environmental pollutants publicity and preterm beginning after adjusting for capability confounders. The mediating position of placental oxidative pressure was tested with the use of the PROCESS macro (version four), which allowed for the estimation of direct, oblique, and general outcomes with the use of bootstrapping. A p-fee <0.05 changed into taken into consideration statistically massive.

## Results

The statistical evaluation found that while maternal age and parity have been not significantly exclusive among term and preterm organizations (p >0.05), Table 1. Several other variables confirmed significant associations. Smoking popularity ($\chi^2$= 9.14, p=0.003), low socioeconomic popularity ($\chi^2$ =6.44, p = zero.011), inadequate antenatal visits ($\chi^2$ =5.83, p = 0.016), and residential proximity to business zones ($\chi^2$ =10.21, p = 0.001) were all substantially more typical among girls who experienced preterm birth.

**Table 1. Sociodemographic and Clinical Characteristics of Participants (N =240)**

| Characteristic | Preterm (n=120) | Term (n=120) | p-value |
|---|---|---|---|
| Mean Age (years) | 28.6 ± 5.1 | 27.9 ± 5.3 | 0.308 |
| Primiparity (%) | 46 (38.3%) | 38 (31.7%) | 0.262 |
| Smoker (%) | 28 (23.3%) | 11 (9.2%) | 0.004 |
| Low SES (%) | 65 (54.2%) | 43 (35.8%) | 0.006 |
| Near Industrial Zone (%) | 69 (57.5%) | 34 (28.3%) | <0.001 |
| Antenatal Care <4 Visits (%) | 51 (42.5%) | 24 (20.0%) | <0.001 |

*Chi-square or t-test applied; bolded p-values indicate statistical significance.*

An independent samples t-tests revealed appreciably better mean concentrations of PM2.5 (mean = 47.2 vs. 31.5 μg/m$^3$, p <0.001), $NO_2$ (33.8 vs. 22.6 μg/m$^3$, p =0.002), and $SO_2$ (12.4 vs. 8.1 μg/m$^3$, p = 0.5) inside the preterm institution compared to the term organization, Table 2. Biochemical assays indicated expanded placental MDA stages (4.62 vs. 2.95 nmol/mg, p < 0.001) and decreased SOD (1.85 vs. 3.26 U/mg, p < 0.001) in the preterm organization.

**Table 2. Comparison of Environmental Pollutant Exposure and Oxidative Stress Biomarkers Between Groups.**

| Variable | Preterm (n = 120) | Term (n = 120) | p-value |
|---|---|---|---|
| Mean PM2.5 Exposure (μg/m³) | 42.6 ± 9.1 | 31.4 ± 7.6 | <0.001 |
| Mean $NO_2$ Exposure (ppb) | 30.8 ± 6.4 | 23.2 ± 5.9 | <0.001 |
| Mean $SO_2$ Exposure (ppb) | 18.5 ± 4.8 | 13.7 ± 3.9 | <0.001 |
| Placental MDA (nmol/mg) | 5.61 ± 1.25 | 3.12 ± 0.96 | <0.001 |
| Placental SOD (U/mg protein) | 9.18 ± 1.96 | 12.74 ± 2.41 | <0.001 |

Binary logistic regression confirmed that PM2.5 publicity (OR = 1.11, 95% CI: 1.05–1.17, p < 0.001), $NO_2$ (OR = 1.07, 95% CI: 1.02–1.30, p = 0.006), $SO_2$ (OR = 1.09, 95% CI: 1.01–1.18, p = 0.032) as presented in Table 3, and living close to commercial zones (OR = 2.83, 95% CI: 1.51–5.29, p = 0.001) had been all big predictors of preterm start. Smoking (OR = 2.28, p = 0.008) and coffee SES (OR = 1.93, p = 0.014) were also independently related to expanded hazard.

**Table 3. Binary Logistic Regression Analysis of Pollution Exposure and Risk of Preterm Birth**

| Predictor | Adjusted OR (95% CI) | p-value |
|---|---|---|
| PM2.5 (per 10 μg/m³) | 2.41 (1.58 – 3.68) | <0.001 |
| $NO_2$ (per 10 ppb) | 1.92 (1.31 – 2.84) | 0.001 |
| $SO_2$ (per 10 ppb) | 1.67 (1.14 – 2.45) | 0.009 |
| Proximity to Industry | 2.89 (1.71 – 4.88) | <0.001 |
| Low SES | 1.78 (1.11 – 2.87) | 0.016 |
| Smoking | 2.91 (1.39 – 6.08) | 0.005 |

By Using PROCESS Model-4, mediation analysis tested a statistically sizable indirect effect of PM2.5 exposure on preterm start through placental MDA (oblique impact = 0.083, 95% CI: 0.031–0.154), Table 4. The direct effect of PM2.5 remained great (direct effect = 0.142, p= 0.009), whilst the whole effect turned into additionally considerable (total impact=0.225, p <0.001). Approximately 36.8% of the full effect changed into mediated by using MDA, indicating partial mediation.

Pearson correlation analysis shows strong correlations between pollutant ranges and MDA: PM2.5 (r = 0.63, p < 0.001), $NO_2$ (r= 0.51, p < 0.001), and $SO_2$ (r = 0.47, p <0.001), Table 5. Conversely, negative correlations were discovered among pollutant ranges and SOD hobby: PM2.5 (r = −0.58, p < 0.001), $NO_2$ (r= −0.94, p < 0.001), and $SO_2$ (r= −0.42, p = 0.001). A strong inverse correlation between MDA and SOD (r= −0.71, p < 0.001) become also determined.

An affords the effects of a multivariate mediation evaluation, in which the consequences of environmental pollutants (PM2.5 and $NO_2$) on preterm birth are assessed, with placental oxidative strain acting as a mediator, at the same time as controlling for capacity confounders (e.g., maternal age, smoking, SES, antenatal care). Table 6 shows 3 key results for each pollutant: direct effect (c'), which represents the effect of pollutants publicity on preterm start independent of oxidative pressure; indirect effect (a*b), which quantifies the mediating impact of oxidative pressure; and general impact (c), which captures the overall effect of pollutants exposure on preterm birth. The proportion mediated suggests the share of the whole impact of pollution on preterm birth this is explained with the aid of oxidative stress. For instance, the excessive percentage of mediation (40.4% for PM2.5) indicates a robust oblique pathway through oxidative pressure.

The stratifies contributors into four quartiles (Q1–Q4) based on their exposure to $PM_{2.5}$ (best particulate rely), revealing a dose-based relationship between pollutant stages and preterm birth threat. The lowest-exposure group (Q1: <25 μg/$m^3$) had only an 8.3% preterm birth rate, while the highest-exposure group (Q4: >45 μg/$m^3$) confirmed a 63.3% charge—an almost eight-fold increase in adjusted odds (OR=8.94, 95% CI: 4.21–18.99), Table7. Oxidative pressure biomarkers mirrored this trend: MDA (a lipid peroxidation marker) rose progressively from four.2 nmol/mg (Q1) to 10.2 nmol/mg (Q4), whilst SOD (an antioxidant enzyme) declined from 24.1 U/mg to 30.8 U/mg throughout quartiles. These effects verify that higher pollution publicity exacerbates placental oxidative pressure, which in turn escalates preterm birth threat in a graded, quantifiable manner.

## Discussion

The results of this study shed light on the complex dating among environmental pollution, placental oxidative pressure, and preterm birth. The sizeable association between pollution publicity and preterm birth threat, mediated by using placental oxidative stress, aligns with preceding studies that highlight environmental toxins as a key contributor to detrimental pregnant outcomes.

In Table 1, the analysis of socio-demographic and scientific characteristics discovered that maternal smoking, low socioeconomic popularity (SES), inadequate antenatal care visits, and proximity to commercial zones had been all drastically related to preterm delivery. This finding helps in advanced studies that have continually linked those elements with a multiplied hazard of preterm shipping. For instance, an observation using Kihal-Talnakhite et al., confirmed that women dwelling near commercial regions had a better threat of preterm delivery due to exposure to air pollutants (16). Similarly, smoking and low SES were implicated in preterm birth via a couple of resources, including a comprehensive review using Rathod et al., which noted the compounded dangers posed with the aid of socioeconomic and behavioral factors on pregnancy results (17).

Table 2 exhibits drastically better exposure to PM2.5, $NO_2$, and $SO_2$ among ladies who delivered preterm, together with increased placental markers of oxidative strain. These findings align with a frame of literature that links air pollutants publicity to oxidative harm, a key mechanism riding irritation and pregnancy complications. A study by Klepac et al. observed similar institutions among excessive PM2.5 publicity and improved stages of oxidative pressure biomarkers in pregnant women (18). Additionally, higher placental malondialdehyde (MDA) ranges and

reduced superoxide dismutase (SOD) interest in preterm births replicate an imbalance among pro-oxidants and antioxidants, helping the hypothesis that oxidative stress performs an essential position inside the pathogenesis of preterm exertions (19,26).

The binary logistic regression in Table three suggests publicity to PM2.5, $NO_2$, $SO_2$, and living close to commercial zones are considerable predictors of preterm birth, even after adjusting for smoking and SES. These consequences are consistent with previous research which has discovered an expanded risk of preterm birth related to various air pollution. A look at using Bekkar et al., similarly showed that improved publicity of air pollution was independently associated with a better threat of preterm start, emphasizing the significance of controlling environmental elements all through pregnancy (20). The odds ratios determined on this look propose that environmental pollution like PM2.5 has a more potent association with preterm beginning than traditional hazard factors like smoking, which in addition underscores the importance of environmental elements in reproductive fitness.

Mediation analysis offered in Table four shows that placental oxidative pressure in part mediates the relationship between PM2.5 publicity and preterm delivery, with approximately 36.8% of the impact mediated through placental MDA stages. This finding is in step with the work of Lembo et al., who proved that oxidative stress performs a mediating role within the consequences of environmental pollution on preterm delivery (21). The large indirect effect of PM2.5 exposure via placental oxidative strain affords mechanistic insight into how air pollutants contribute to adverse pregnancy results. It is properly documented that pollutants like PM2.Five induce oxidative pressure, leading to inflammatory responses that could cause preterm hard work (22).

Table five's correlation evaluation in addition helps the findings, displaying sturdy positive correlations between pollutant degrees and MDA, whilst poor correlations among pollution and SOD interest emphasize the dangerous results of pollution on antioxidant defenses. These consequences are consistent with research by Zhang et al., who discovered that publicity to pollution consists of $NO_2$ and PM2.5 is associated with better ranges of lipid peroxidation markers (e.g., MDA) and reduced antioxidant hobby in pregnant ladies (23). The inverse correlation among MDA and SOD on this examination is, in particular, top-notch, as it reinforces the concept that environmental pollutants impair the body's potential to counteract oxidative stress, thereby exacerbating the risk of preterm birth.

The multivariate mediation analysis in Table 6, which incorporates covariates inclusive of smoking and SES, further strengthens the argument that oxidative stress mediates the effects of pollution publicity on preterm birth. The findings that each PM2.5 and $NO_2$ have a substantial indirect impact on preterm beginning through oxidative stress (with proportions of mediation of 40.4% and 35.0%, respectively) are consistent with the speculation that placental oxidative strain is an important mediator within the relationship between environmental pollution and unfavorable pregnancy effects. This mediation effect indicates that decreasing pollutants publicity may additionally help mitigate a number of the oxidative stress-pushed mechanisms contributing to preterm start, supporting public fitness tasks aimed at reducing air pollution exposure at some point of pregnancy.Finally, Table 7 sensitivity analysis highlights a dose-established courting among PM2.5 exposure and preterm delivery, with gradually higher quotes of preterm start, MDA ranges, and decrease SOD pastime discovered in higher exposure quartiles. This dose-response dating is consistent with preceding research which has determined a better prevalence of preterm start in populations uncovered to extra stages of air pollution (20, 24,25). The strong affiliation between higher pollution tiers and elevated oxidative stress underscores the critical need for targeted interventions to lessen the publicity of environmental pollutants, mainly for pregnant women living in high-pollutant regions.

This has a look at provides compelling proof that environmental pollution publicity, mainly to PM2.5, $NO_2$, and $SO_2$, is related to an expanded danger of preterm beginning, with placental oxidative stress performing as a huge mediator. These findings are supported by a developing body of literature that emphasizes the unfavorable results of air pollution on maternal and fetal fitness. Policymakers need to prioritize air high-quality development and public health training to mitigate the risks of pollutants-associated pregnancy headaches.

This takes a look at several barriers, including its go-sectional design, which limits the ability to set up causal relationships between pollution exposure, oxidative pressure, and preterm beginning. Additionally, the look at trusted self-stated information for some variables, together with smoking and antenatal care visits, can introduce considered bias. The sample turned into additionally geographically limited, which might also affect the generalizability of the findings to different populations. Lastly, whilst oxidative strain markers have been measured in placental tissue, different potential biomarkers or confounding elements may not have been completely accounted for.

## Conclusions

This study underscores the important association between environmental pollution, especially PM2.5, $NO_2$, and $SO_2$, and increased risk of preterm birth, with placental oxidative stress acting as a mediator. To mitigate these risks, public health policies must focus on reducing pollution, especially in industrial areas, and improving access to antenatal care. Healthcare providers should integrate environmental risk assessments into antenatal care. Implementation can include air quality monitoring, educational campaigns, and specialized training for healthcare professionals. Regular evaluation of these efforts will help track improvements in maternal health outcomes and preterm birth rates.

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### Conflicts of interest

There are no conflicts of interest.

### Author contribution

H.A.S.A: Methodology, H.K.H.: Data curation, original draf t preparation, A.A.H.K: Writing, Reviewing and Editing.

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