Journal of Biomedical Sciences | ISSN: 2960-9356
Research Article Peer-review, Open Access
World of Medicine: Journal of Biomedical Sciences Vol .3 No. 8 (2026)
https://wom.semanticjournals.org/index.php/biomed

Toxicant-Induced Oxidative Stress and Pregnancy Outcomes: A Study of Pregnant Women Exposed to Artisanal Refinery Emissions in Delta State

Edward Bayo Adebowale (PhD)
Department of Medical Laboratory Science, University of Benin, Benin City, Nigeria
Email: Edwardadebowale251@gmail.com

Abstract: Background: Artisanal crude oil refining ("Kpo fire") is widespread in the Niger Delta, releasing substantial amounts of particulate matter, heavy metals, and polycyclic aromatic hydrocarbons. Pregnant women residing in these host communities face an elevated risk of severe oxidative stress, which may contribute to adverse pregnancy outcomes. Objective: To investigate the association between toxicant-induced oxidative stress and pregnancy outcomes among pregnant women exposed to artisanal refinery emissions in Delta State. Methods: A comparative cross-sectional study was conducted with 250 pregnant women, comprising 125 from three artisanal refinery host communities in Warri South LGA and 125 from non-exposed control communities in Ika North LGA. Venous blood samples were evaluated for malondialdehyde (MDA), superoxide dismutase (SOD), glutathione (GSH), as well as serum lead and cadmium levels. Antenatal care (ANC) records were utilized to document pregnancy outcomes, including birth weight, gestational age at delivery, preeclampsia, and miscarriages. Data analysis was performed using SPSS version 26, employing independent t-tests, chi-square tests, and logistic regression models (significance set at $p < 0.05$). Results: Exposed participants exhibited significantly higher mean concentrations of MDA (4.82 ± 1.14 versus 2.91 ± 0.87 µmol/L , $p < 0.001$) and lead (18.4 ± 5.2 versus 7.1 ± 2.8µg/dL, $p < 0.001$), alongside depleted antioxidant markers (SOD and GSH). Adverse pregnancy outcomes were significantly elevated in the exposed cohort: low birth weight (28.0% versus 12.8%, $p = 0.002$), preterm birth (22.4% versus 9.6%, $p = 0.005$), and preeclampsia (16.8% versus 6.4%, $p = 0.009$). Elevated oxidative stress and serum lead levels emerged as significant independent predictors of low birth weight (OR = 2.87, 95% CI: 1.42 - 5.81). Conclusion: Emissions from artisanal crude oil refining induce severe oxidative stress and correlate strongly with adverse pregnancy outcomes. These findings underscore the urgent need for targeted public health interventions and sustainable alternative livelihood programs in affected regions.

Keywords: Artisanal Refinery, Oxidative Stress, Pregnancy Outcomes, Heavy Metals, Niger Delta.

Introduction

The Niger Delta region of Nigeria serves as the economic hub of the nation's crude oil exploration and production. However, socioeconomic challenges such as widespread poverty, unemployment, and militant oil bunkering have catalyzed the proliferation of artisanal crude oil refining, locally known as "Kpo fire" [1]. This illicit practice involves the crude distillation of stolen crude oil in makeshift metal drums and tanks heated over open firewood to extract adulterated petroleum products, including diesel, petrol, and kerosene. The refining process releases hazardous emissions—comprising polycyclic aromatic hydrocarbons (PAHs), benzene, particulate matter ($PM_{2.5}$), and heavy metals such as lead and cadmium—directly into the ambient air, water bodies, and soil. Because these operations frequently occur in close proximity to residential communities and waterways, local populations endure chronic, low-dose

toxicant exposure [1], [10].

Pregnancy is inherently characterized by an elevation in systemic oxidative stress driven by the high metabolic demands of the developing placenta [2]. When superimposed with environmental toxicant exposures from sources like artisanal refining, these physiological demands can severely overwhelm maternal antioxidant defenses. This imbalance triggers lipid peroxidation, cellular DNA damage, and endothelial dysfunction, which are major pathological mechanisms implicated in adverse pregnancy outcomes such as preeclampsia, preterm birth, and low birth weight [3], [11], [12]. Despite these alarming environmental health hazards, data on maternal and fetal health consequences in these host communities remain limited. Therefore, this study aims to investigate the influence of toxicant-induced oxidative stress on pregnancy outcomes among women exposed to artisanal refinery emissions in Delta State.

Specific Objectives

To achieve this aim, the study will:

a. Compare the serum concentrations of oxidative stress markers and heavy metals between pregnant women residing in exposed artisanal refinery communities and those in non-exposed control communities.
b. Determine the prevalence of adverse pregnancy outcomes (including low birth weight, preterm birth, and preeclampsia) across both groups.
c. Assess the statistical association between elevated toxicant-induced oxidative stress and adverse pregnancy outcomes.

Literature Review

Oxidative Stress, Reproduction, and Pregnancy

Concept and Cellular Mechanics of Oxidative Stress

Oxidative stress (OS) arises from an imbalance between the excessive production of reactive species and the biological system's capacity to neutralize them through detoxification mechanisms [2]. This state is driven either by elevated levels of reactive oxygen species (ROS) and reactive nitrogen species (RNS) or by a depletion in antioxidant defense systems, frequently culminating in chronic inflammation [3]. Major ROS—such as superoxide radicals (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (•OH)—target cellular macromolecules, damaging proteins and nucleic acids while inducing lipid peroxidation (LPO). LPO compromises membrane integrity and alters DNA structures, causing mutations, cytotoxicity, and disrupted cellular metabolism [4]. Consequently, systemic oxidative stress serves as a primary or secondary driver in numerous pathologies, including diabetes mellitus, neurodegenerative disorders, cardiovascular disease, asthma, rheumatoid arthritis, and various malignancies [5].

Impact of Oxidative Stress on Reproductive Processes

Free radicals heavily influence both male and female reproductive systems. Gametes, due to their high specialization, are exceptionally vulnerable to ROS-mediated damage. In males, oxidative stress impairs sperm morphology and function, resulting in decreased viability, motility, concentration, and overall fertilization potential—key factors in male-factor infertility [3].

In females, physiological levels of ROS within follicular fluid modulate vital processes such as oocyte maturation, folliculogenesis, steroidogenesis, luteolysis, and ovulation [6]. However, pathological levels of oxidative stress contribute significantly to female reproductive disorders, including endometriosis, polycystic ovary syndrome (PCOS), premature ovarian failure, and unexplained infertility. Furthermore, oxidative stress negatively affects oocyte developmental competence during in vitro cultivation, ovarian hyperstimulation, and biological aging across both human and animal models.

Physiological Oxidative Stress and Systemic Changes in Pregnancy

Pregnancy is characterized by profound physiological and metabolic adaptations aimed at supporting fetal development while preparing the maternal body for childbirth and lactation [6]. These adaptations lead to an elevated basal oxygen consumption rate and shifts in energy substrate utilization, particularly across the feto-placental unit.

Normal pregnancy is also marked by a heightened systemic inflammatory response, particularly during the third trimester, which plays a physiological role in labor initiation but also increases susceptibility to oxidative stress [6]. This response activates peripheral granulocytes, monocytes, and lymphocytes, which release high volumes of ROS. When these reactive species attack cell membrane phospholipids and polyunsaturated fatty acids, they trigger lipid peroxidation and endothelial cell dysfunction—a core pathological mechanism in several gestational disorders. Excessive oxidation induces biochemical damage across nucleic acids, proteins, and lipids, though the complex precise mechanisms continue to be an active area of investigation [7].

Epigenetic Impacts and Fetal Programming

Oxidative stress accelerates genomic instability by promoting spontaneous DNA mutations. Furthermore, it alters DNA methylation patterns, exerting downstream effects on fetal programming. Because DNA methylation regulates gene expression without modifying the underlying sequence, it is highly sensitive to environmental stimuli. Studies evaluating global methylation in placental and cord tissues of mothers affected by gestational diabetes, preeclampsia, and obesity suggest that maternal metabolic stress profoundly influences the offspring's epigenome [6]. Animal models similarly demonstrate a direct correlation between the severity of maternal hyperglycemia and the extent of oxidative DNA damage, indicating that metabolic disruptions carry long-term consequences extending well beyond the mother.

Placental Oxidative Stress and Feto-Placental Pathology

The developing placenta represents a major endogenous source of ROS during pregnancy, influencing maternal systemic homeostasis from early gestation. Early placenta development occurs in a relatively hypoxic environment. The high metabolic rate of mitochondria-rich placental tissue generates significant levels of ROS (primarily O_2^- and nitric oxide), which are essential for regulating placental blood perfusion and fetal nutrition.

By the end of the first trimester, placental maturation triggers a threefold increase in local oxygen concentration, elevating ROS production predominantly within the syncytiotrophoblast. Under physiological conditions, this spike is strictly regulated by hypoxia-inducible factor 1-alpha (HIF-1α) and the upregulation of antioxidant genes, including heme oxygenase (HO-1 and HO-2), copper-zinc superoxide dismutase (Cu/Zn-SOD), catalase (CAT), and glutathione peroxidase (GPx).

However, when environmental or pathological stressors cause ROS accumulation to outpace placental antioxidant capacity, oxidative damage spreads to distal maternal tissues, precipitating severe gestational complications. Unchecked ROS accumulation leads to placental underdevelopment and dysfunction, impairing the transfer of oxygen and nutrients to the fetus. This stress triggers leukocyte and platelet adhesion to the endothelium, alongside the release of inflammatory cytokines and anti-inflammatory/antiangiogenic factors. Ultimately, this inflammatory cascade promotes generalized vasoconstriction, increases placental vascular resistance, and compromises uteroplacental blood flow [7].

Toxicant-Induced Oxidative Stress and Pregnacy Outcomes in The Niger Delta

Mechanisms of Toxicant-Induced Oxidative Stress

Toxicant-induced oxidative stress fundamentally disrupts the delicate maternal-placental-fetal equilibrium. Exposure to environmental pollutants triggers the overproduction of reactive oxygen species (ROS) that drastically overwhelm endogenous antioxidant defenses, cascading into severe pathological consequences for pregnancy [7]:

a. **Environmental Pollutants:** Chronic exposure to heavy metals (such as lead and cadmium), fine particulate matter ($PM_{2.5}$), and combustion byproducts profoundly alters maternal metabolic pathways, impairing glutathione synthesis and accelerating lipid oxidation [13], [14].
b. **Endocrine Disruption:** Toxic heavy metals can interfere with nuclear hormone receptors, disturbing the precise hormonal signaling required to establish and maintain a healthy gestation [8].
c. **Cellular and Vascular Damage:** An excess of free radicals drives lipid peroxidation—quantified via biomarkers like malondialdehyde (MDA)—along with DNA damage (such as 8-hydroxy-2'-deoxyguanosine [8-OHdG]) and systemic vascular endothelial dysfunction [7].

d. **Pathophysiological Links to Adverse Pregnancy Outcomes**

e. According to Ahsan [8], unmitigated oxidative stress during pregnancy directly instigates major obstetric complications:

f. **Preeclampsia (PE):** Oxidative injury and endothelial damage to placental blood vessels elevate vascular resistance, clinically manifesting as pregnancy-induced hypertension and proteinuria.

g. **Intrauterine Growth Restriction (IUGR) & Low Birth Weight:** Structural damage to the placental architecture impairs optimal nutrient and oxygen transfer across the feto-placental unit, restricting normal fetal development.

h. **Pregnancy Loss:** Systemic levels of high oxidative stress heavily correlate with an elevated risk of spontaneous early miscarriage and recurrent pregnancy loss.

i. **Offspring Complications:** Prenatal oxidative damage increases vulnerability to preterm birth complications, neonatal respiratory distress, and long-term neurodevelopmental deficits in the offspring.

Environmental Context: The "Kpo Fire" Phenomenon in the Niger Delta

In the Niger Delta region of Nigeria, these global toxicological pathways are severely amplified by artisanal crude oil refining, locally known as "Kpo fire" (derived from local pidgin terminology denoting "boiling" or "burning"). This illicit practice involves tapping stolen crude oil from vandalized pipelines in regional creeks, pouring it into makeshift metal drums or tanks, and heating it over open firewood for continuous periods of 24 to 72 hours. The vapors are channeled through improvised, unsealed pipes to condense and separate fractions into adulterated diesel, petrol, and kerosene.

Operating entirely without personal protective equipment or environmental pollution controls, these localized distillation sites release massive quantities of toxic fumes—including polycyclic aromatic hydrocarbons (PAHs), benzene, heavy metals, and particulate matter—directly into local air, soil, and aquatic ecosystems.

While driven by socioeconomic factors such as widespread youth unemployment, easy access to stolen crude, and high commercial fuel prices, the persistence of "Kpo fire" ensures continuous, community-wide exposure. Pregnant women residing in these host environments face chronic, high-dose toxicant inhalation, accelerating maternal-placental oxidative stress and driving the disproportionate burden of adverse pregnancy outcomes documented in the region.

Health & Environmental Problems

Table 1. Health & Environmental Problems

Toxicant Released	Health Effect
PAHs - Polycyclic Aromatic Hydrocarbons	Causes oxidative stress, DNA damage, cancer, pregnancy complications
Heavy Metals: Lead, Cadmium, Mercury	Neurotoxicity, preeclampsia, low birth weight, miscarriage
PM2.5 & Soot	Respiratory problems, asthma, black soot rain
Benzene, Toluene	Blood disorders, reproductive toxicity
Oil spills + Acid rain	Contaminates water, fish, and farmland

Source: UNEP [1]

Materials and Methods

Study Design and Setting

This study utilized a comparative cross-sectional design conducted between January and June 2026. The research was carried out in Delta State, Nigeria, comparing an exposed population from Warri South Local Government Area (LGA) an area with documented artisanal crude oil refining activities along its creeks with a non-exposed control population from Ika North LGA.

Study Population and Eligibility Criteria

The target population comprised pregnant women aged 18 to 45 years attending Antenatal Care (ANC) clinics at primary health centers within the respective study areas.

a. **Inclusion Criteria:** Pregnant women with a gestational age between 14 and 40 weeks who had resided in the study area for a minimum of 2 years.
b. **Exclusion Criteria:** Women with a history of active smoking, pre-existing chronic medical conditions (such as diabetes mellitus), or human immunodeficiency virus (HIV) infection.

Sample Size Determination

The minimum sample size was calculated using the formula for comparing two independent proportions, setting the statistical power at 80% ($\beta = 0.20$), a significance level (α) of 0.05, and anticipating a low birth weight prevalence of 25% in the exposed group versus 12% in the control group. This yielded a minimum requirement of 224 participants. To account for a potential 10% attrition or incomplete data rate, the sample size was rounded up to 250 participants, equally distributed as 125 participants per group.

Sampling Technique

A multi-stage sampling technique was adopted for this study:

a. **Stage 1:** Three primary health centers (PHCs) were purposefully selected from the exposed LGA (Warri South), and three comparative PHCs were selected from the non-exposed control LGA (Ika North).
b. **Stage 2:** Eligible pregnant women attending ANC clinics in the selected facilities were recruited consecutively until the required sample size for each group was achieved.

Data and Biological Sample Collection

Data were collected using a structured interviewer-administered questionnaire divided into sections covering socio-demographics, obstetric history, and environmental exposure history.

For laboratory analysis, 5 mL of venous blood was aseptically collected from each participant. Serum was separated and analyzed using standard laboratory protocols:

a. **Malondialdehyde (MDA):** Quantified via the Thiobarbituric Acid Reactive Substances (TBARS) assay method.
b. **Superoxide Dismutase (SOD):** Assessed using spectrophotometry.
c. **Glutathione (GSH):** Measured via Ellman's method.
d. **Lead and Cadmium:** Analyzed using an Atomic Absorption Spectrophotometer (AAS).

Outcome Measures

Pregnancy and neonatal outcomes—including birth weight, gestational age at delivery, APGAR scores, incidence of preeclampsia, and history of miscarriage—were extracted directly from official ANC records and delivery logs.

Statistical Analysis

Data were cleaned, coded, and analyzed using SPSS version 26. Descriptive statistics were computed and summarized as means and standard deviations (for continuous variables) or frequencies and percentages (for categorical variables). Bivariate analyses were performed using independent t-tests for continuous variables and chi-square tests for categorical variables. Binary logistic regression models were subsequently fitted to determine independent predictors of adverse pregnancy outcomes. Statistical significance was established a priori at $p < 0.05$.

Results

Socio-Demographic Characteristics of Participants

A total of 250 pregnant women completed the study, with 125 participants in each group (exposed and non-exposed controls). As shown in Table 1, the mean age of participants was comparable between the two cohorts, recording 27.8 ± 5.2 years in the exposed group and 28.1 ± 4.9 years in the control group ($p > 0.05$). The majority of participants across both groups attained secondary-level education.

Significant variations were observed in occupational distributions between the two populations (p = 0.008): the majority of women in the artisanal refinery host communities were engaged in trading and fishing (62.4%), whereas civil service employment was more prevalent among the control cohort (45.6%).

Biomarkers of Oxidative Stress and Heavy Metal Concentrations

Table 2. Comparison of Biomarkers

Biomarker	Exposed	Control	T
MDA	4.82 ± 1.14	2.91 ± 0.87	14.92
SOD	12.4 ± 3.1	18.7 ± 4.2	-13.41
GSH	3.21 ± 0.98	5.14 ± 1.21	-13.85
Lead	18.4 ± 5.2	7.1 ± 2.8	21.03
Cadmium	2.87 ± 0.91	1.12 ± 0.54	18.24

From the table we can see that exposed women had significantly higher oxidative damage and lower antioxidants.

Pregnancy Outcomes

Table 3. Pregnancy Outcomes

Outcome	Exposed n	Control n	$\chi 2$
Low Birth Weight <2.5kg	35	16	9.42
Preterm <37 weeks	28	12	7.81
Preeclampsia	21	8	6.78
Miscarriage	14	6	3.54

Predictors of Low Birth Weight (Logistic Regression)

Multivariable logistic regression analysis was conducted to identify independent risk factors associated with low birth weight (LBW) among the participants. After adjusting for confounding variables such as maternal age, educational attainment, and parity, exposure to artisanal refining (OR = 2.34, 95% CI: 1.31 - 4.18, p = 0.004), elevated malondialdehyde (MDA) levels (OR = 2.87, 95% CI: 1.42 - 5.81, p = 0.003), and high blood lead concentrations (OR = 1.96, 95% CI: 1.12 - 3.44, p = 0.018) emerged as significant independent predictors of low birth weight.

Discussion

This study demonstrates that pregnant women residing in artisanal crude oil refining host communities experience severe oxidative stress and a significantly elevated burden of adverse pregnancy outcomes compared to unexposed controls. The biochemical findings marked by significantly elevated concentrations of malondialdehyde (MDA) alongside depleted antioxidant enzymes (superoxide dismutase [SOD] and glutathione [GSH]) reflect extensive lipid peroxidation and overwhelmed antioxidant defense systems. This physiological disruption is primarily driven by maternal exposure to polycyclic aromatic hydrocarbons (PAHs), volatile organic compounds, and heavy metals released during open crude oil combustion [5].

The findings reveal that elevated oxidative stress markers and high serum lead levels increase the odds of low birth weight nearly threefold. Lead is a well documented reproductive and developmental toxicant capable of readily crossing the placental barrier. Once fetal exposure occurs, lead induces systemic vascular endothelial dysfunction and vasoconstriction, severely restricting uteroplacental blood flow and contributing directly to intrauterine growth restriction (IUGR) and preeclampsia [9], [15]. These patterns mirror established literature on ambient air pollution and adverse birth outcomes, while highlighting the magnified risks unique to localized, unregulated petro-industrial activities.

Socioeconomically, the persistence of the "Kpo fire" phenomenon is fueled by systemic poverty, high youth unemployment, and commercial fuel scarcity. This dynamic creates a dangerous "double burden," where vulnerable populations trade immediate economic survival for chronic environmental toxicity. Unlike formal industrial refining, artisanal refining operations occur directly within residential backyards and community creeks, resulting in continuous, high-dose maternal inhalation and dermal absorption.

Strengths and Limitations

A primary strength of this study lies in its objective integration of biochemical biomarkers (MDA, SOD, GSH) and toxic heavy metal assays (Lead and Cadmium), moving beyond purely questionnaire-based exposure estimates. However, certain limitations must be acknowledged. Due to its cross-sectional design, temporal sequence and definitive causality cannot be definitively established. Additionally, the study relied on community residence rather than continuous personal air-monitoring telemetry to quantify individual pollutant inhalation.

Conclusion and Recommendations

Conclusion

Emissions originating from artisanal crude oil refining ("Kpo fire") induce profound oxidative stress and heavy metal accumulation, which are strongly associated with a high prevalence of low birth weight, preterm delivery, and preeclampsia among pregnant women in Delta State, Nigeria.

Recommendations

Based on these findings, the following multi-tiered interventions are urgently recommended:

a. **Public Health Interventions:** Healthcare facilities in oil-producing and artisanal refining regions should incorporate routine antenatal screening for heavy metal toxicity and oxidative stress markers among pregnant women for early risk stratification.
b. **Policy and Regulatory Action:** Government authorities must rigorously enforce the ban on illegal artisanal refining while concurrently implementing robust alternative livelihood and economic empowerment programs for youths in the Niger Delta.
c. **Community-Level Support:** Non-governmental organizations and public health agencies should distribute dietary antioxidant supplements and facilitate access to clean, unpolluted water resources for pregnant women residing in vulnerable host communities to mitigate oxidative damage.

Reference:

[1] UNEP, Environmental Assessment of Ogoniland. United Nations Environment Programme, 2011.

[2] G. J. Burton and E. Jauniaux, “Oxidative stress,” Best Practice & Research Clinical Obstetrics & Gynaecology, vol. 25, pp. 287–299, 2011.

[3] K. H. Al-Gubory, “Environmental pollutants and oxidative stress in reproductive organs,” Reproduction, vol. 147, pp. R229–R240, 2014.

[4] W. Dröge, “Free radicals in the physiological control of cell function,” Physiol. Rev., vol. 82, pp. 47–95, 2002.

[5] J. E. Klaunig and L. M. Kamendulis, “The role of oxidative stress in carcinogenesis,” Annual Review of Pharmacology and Toxicology, vol. 44, pp. 239–267, 2014.

[6] K. Krumova and G. Cosa, “Overview of Reactive Oxygen Species,” in Singlet Oxygen: Applications in Biosciences and Nanosciences, vol. 1, pp. 1–21, 2016.

[7] A. Phaniendra, D. B. Jestadi, and L. Periyasamy, “Free radicals: Properties, sources, targets, and their implication in various diseases,” Indian J. Clin. Biochem., vol. 30, pp. 11–26, 2015.

[8] H. Ahsan, “3-Nitrotyrosine: A biomarker of nitrogen free radical species modified proteins in systemic autoimmunogenic conditions,” Hum. Immunol., vol. 74, pp. 1392–1399, 2013.

[9] D. M. Stieb, L. Chen, M. Eshoul, and S. Judek, “Ambient air pollution, birth weight and preterm birth,” Occupational and Environmental Medicine, vol. 69, pp. 694–704, 2012.

[10] A. Onakpohor, B. S. Fakinle, J. A. Sonibare, M. A. Oke, and F. A. Akeredolu, "Investigation of air emissions from artisanal petroleum refineries in the Niger-Delta Nigeria," Heliyon, vol. 6, no. 11, Art. no. e05608, 2020, doi: 10.1016/j.heliyon.2020.e05608.

[11] O. B. Oghenetega, G. R. E. E. Ana, M. A. Okunlola, and O. A. Ojengbede, "Miscarriage, stillbirth, and infant death in an oil-polluted region of the Niger Delta, Nigeria: A retrospective cohort study," Int. J. Gynecol. Obstet., vol. 150, no. 3, pp. 361–367, 2020, doi: 10.1002/ijgo.13259.

[12] O. B. Oghenetega, M. A. Okunlola, G. R. E. E. Ana, O. Morhason-Bello, and O. A. Ojengbede, "Exposure to oil pollution and maternal outcomes: The Niger Delta prospective cohort study," PLoS ONE, vol. 17, no. 3, Art. no. e0263495, 2022, doi: 10.1371/journal.pone.0263495.

[13] L. Yang, L. Shang, S. Wang, W. Yang, L. Huang, C. Qi, A. Gurcan, Z. Yang, and M. C. Chung, "The association between prenatal exposure to polycyclic aromatic hydrocarbons and birth weight: A meta-analysis," PLoS ONE, vol. 15, no. 8, Art. no. e0236708, 2020, doi: 10.1371/journal.pone.0236708.

[14] A. K. Amegah, C. Sewor, and J. J. K. Jaakkola, "Cadmium exposure and risk of adverse pregnancy and birth outcomes: A systematic review and dose–response meta-analysis of cohort and cohort-based case–control studies," J. Expo. Sci. Environ. Epidemiol., vol. 31, no. 2, pp. 299–317, 2021, doi: 10.1038/s41370-021-00289-6.

[15] Z. Zhong, Q. Yang, C. Li, X. Chen, and F. Zhou, "A global perspective of correlation between maternal blood lead levels and risks of preeclampsia: An updated systematic review and meta-analysis," Front. Public Health, vol. 10, Art. no. 1072052, 2022, doi: 10.3389/fpubh.2022.1072052.