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DOI: https://doi.org/10.64388/IREV8I11-1722732

Environmental Toxicant Burden, Maternal Oxidative Stress, and Neonatal Outcomes in Crude-Oil-Impacted Communities of Warri, Delta State, Nigeria

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Abstract- ***Background: An environmental problem associated with petroleum extraction and processing activities in the Niger delta is the generation of complex mixtures of heavy metal and petroleum-derived organic pollutants. During pregnancy, the developing placenta and the maternal physiology and growing fetus may be affected by environmental toxicants, especially during pregnancy. Evidence specific to Warri that integrates environmental measurements, maternal biomonitoring, oxidative-stress biomarkers and neonatal outcomes remains limited.***

Objective: Residing in crude-oil impacted communities would be correlated with increased environmental exposures to toxicants, changes in maternal oxidative/inflammatory status, and neonatal outcomes.

Methods: The methods used was comparative cross-sectional design involving 350 pregnant women of gestation ≥14 weeks in the study community, Ijala-Ikenren, Ugborodo and Ifie-Kporo and 175 as the comparison community, Ughievwen. Lead (Pb), Cadmium (Cd) and Polycyclic aromatic hydrocarbons (PAHs) were measured in topsoil and drinking water. Maternal blood level of Pb, Cd, total PAH and plasma malondialdehyde (MDA), superoxide dismutase (SOD), reduced glutathione (GSH) and high-sensitivity C-reactive protein (hs-CRP) were evaluated. Neonatal birth weight, gestational age, 5-minute Apgar score and congenital anomalies were evaluated. Data analysed were group differences, Pearson correlations and multivariable logistic regression.

Results: The study found substantially higher Pb and Cd concentrations in exposed soil and water, including soil Pb of 68.40 ± 15.20 versus 24.70 ± 8.60 mg/kg and water Pb of 0.086 ± 0.021 versus 0.019 ± 0.008 mg/L (both p < .001). The total soil PAHs measured were 18.70 ± 5.40 mg/kg compared to 4.90 ± 1.80 mg/kg of carcinogenic PAHs, with a percentage of 72.0% versus 31.0%. In the same way, maternal blood levels of Pb, Cd and total PAHs were also higher in the exposed women. SOD and GSH were lower, while MDA and hs-CRP were higher in the exposed group of women than in the unexposed group. Exposed neonates were more likely to be low birth weight, preterm, or have a low 5-minute Apgar score or to be born with congenital anomalies. The blood lead, cadmium and MDA were positive predictors and the blood SOD was inverse predictors of LBW in the reported adjusted model (aOR = 2.85, 95% CI [1.62, 5.02], p < .001 and aOR = 0.77, 95% CI [0.63, 0.95], p = 0.02, respectively).

Conclusion: The results confirm a well-defined exposure–internal dose–oxidative stress–adverse birth outcome pattern.

Keywords: crude-oil spill, lead, cadmium, polycyclic aromatic hydrocarbons, oxidative stress, pregnancy, low birth weight, niger delta, warri, biomonitoring of the mother

I. INTRODUCTION

The risks to health from petroleum production also occur outside of acute oil-spill events. Releases from extraction, transportation and storage, combustion and artisanal refining of oil, repeated in an oil-producing area, can result in persistent mixtures of metals, particulate matter, PAHs and other contaminants on the soil, water, and air. The Niger Delta environment is particularly relevant for environmental-health research as the geographical distribution of petroleum activity is intermingled with residents' life, food systems and livelihoods. The region has recently been described as an interacting environmental, health, food-system and socioeconomic consequences of oil exploitation (Gbadamosi & Aldstadt, 2025).

The contamination issue is not just limited to one environmental compartment. A systematic review and meta-analysis of the studies of water in the Niger Delta revealed a pooled mean concentration of lead (Pb), cadmium (Cd) and other metals which often

surpass the World Health Organisation (WHO) drinking water standards (Umeoguaju et al., 2022). The latest One Health synthesis also confirms that selected cities in the Niger Delta region are heavily contaminated and bioaccumulate heavy metals (Onyena et al., 2024). Another significant part of the exposure mixture comprises PAHs. Udom et al. (2023) found that the presence of PAH cannot be ignored in the Niger Delta, and recommended concerted environmental, food-safety and human-health interventions.

Pregnancy is one area of concern, as it is important that the fetus is developing in a controlled manner by the placenta, vascular system, endocrine and metabolic pathways. Exposure to environmental toxins can occur across the maternal–fetal barrier or they can interfere with placental signaling, which can impact oxygen and nutrient transport. Toxic metals such as lead and cadmium have no physiological roles and PAHs and their metabolites may engage redox cycling, inflammatory signalling and DNA damage. These mechanisms are more informative than residence alone when the biological effects are investigated, as these mechanisms provide more information about the maternal internal dose.

This mechanism is suggested by the presence of oxidative stress. The changes in redox biology are a normal feature of pregnancy since during pregnancy, the metabolism of the mother and the development of the placenta require more oxygen. If the generation of reactive oxygen species surpasses the antioxidant capability, lipid peroxidation, protein alteration, DNA damage, and endothelial damage are potential outcomes. MDA is a measure of lipid peroxidation and SOD and GSH are important components of antioxidant defence. This redox imbalance may coexist with inflammatory activation, which is why hs-CRP is a good systemic marker.

An increasing amount of epidemiological synthesis supports the developmental consequences of prenatal toxicant exposure. The prenatal lead exposure was investigated in a meta-analysis study, in which an inverse relationship was observed between the levels of maternal blood lead and birth weight (Vigeh et al., 2023). In a systematic review and meta-analysis of maternal PAH exposure, the pooled association with dichotomous low birth weight was not significant, yet significant associations with decreased birth weight and birth length were observed (Latifi et al., 2024). This difference is significant: continuous measures of fetal growth can detect subtle effects of toxicants that would be masked by transforming birth weight into a dichotomous clinical category.

There is also increasing evidence from the Niger Delta becoming more mechanistic. Increased levels of serum Pb and Cd, and increased lipid peroxidation and reduced antioxidant activity were reported among inhabitants of a crude-oil-producing community by Oyeyemi et al. (2024), indicating environmental contamination and measurable biological stress. In reproductive epidemiology, Oghenetega et al. (2022) tracked over 1,400 women who were pregnant and discovered an increased likelihood of selected maternal outcomes in regions that had high exposure to oil pollution. These studies reinforce the need for combined assessments of environmental exposure and biological effects, but are not completely satisfactory in clarifying the pathway from environmental burden to maternal oxidative burden to neonatal endpoints in the Warri population.

To fill in these gaps, the present study brings together environmental chemistry, maternal biomonitoring, oxidative/inflammatory markers and neonatal outcomes in a single comparative framework. The conceptual logic is deliberately parsimonious: external contamination should be correlated to internal toxicant burden, higher internal burden, to oxidative and inflammatory disturbance, and this disturbance of the internal environment should be correlated to impaired fetal growth and other adverse outcomes.

Objectives, Research Questions and Hypotheses
Research Objectives

1. To compare selected crude-oil-associated toxicants (Pb, Cd and PAHs) in environmental matrices and maternal blood between pregnant women residing in crude-oil-impacted communities and those in the comparison community.

2. To compare maternal oxidative and inflammatory status, indexed by MDA, SOD, GSH and hs-CRP, between exposed and comparison cohorts.
3. To determine whether maternal toxicant burden and oxidative-stress biomarkers are associated with selected neonatal outcomes, particularly birth weight, preterm birth, 5-minute Apgar score and congenital anomalies.

Research Questions

1. Do pregnant women in crude-oil-impacted communities have higher environmental and maternal Pb, Cd and PAH burdens than women in the comparison community?
2. Does residence in crude-oil-impacted communities correspond to a different maternal oxidative and inflammatory profile?
3. Are maternal toxicant burden and oxidative-stress biomarkers associated with adverse neonatal outcomes?

Research Hypotheses

H01: There is no significant difference in selected environmental and maternal Pb, Cd and PAH concentrations between the crude-oil-impacted and comparison communities.

H02: Maternal toxicant burden and oxidative/inflammatory biomarkers are not significantly associated with adverse neonatal outcomes.

Conceptual and Theoretical Framework

The conceptual framework places crude oil related environmental contamination as the upstream exposure context, toxicant burden of the mother as the internal exposure indicator, oxidative/inflammatory disturbance as a biological response and neonatal outcomes as the health endpoints. This framework is based on the Dose–Response theory, the Oxidative-stress theory and the Developmental Origins of Health and Disease (DOHaD) approach. Contaminated water, food, soil and air may be the source of entry for petroleum-related Pb, Cd and PAHs into the body, which once absorbed can interfere with antioxidant systems, mitochondrial function, inflammatory signalling and placental physiology. There is recent evidence that shows environmental heavy metal pollution is associated with elevated serum levels of Pb and Cd, elevated lipid peroxidation and reduced antioxidant defenses in a Nigerian crude oil producing community (Oyeyemi et al., 2024). Obstetric evidence has also emerged recently, with oxidative stress and placental dysfunction emerging as two pathways connecting environmental toxicants with impaired fetal growth and preterm birth and hypertensive disorders (FIGO, 2025). Based on this, maternal MDA, SOD, GSH and hs-CRP are considered markers of biological reactions to toxicant exposure and birth weight, gestational age, Apgar score and congenital anomalies as downstream outcomes of neonatal development (see Figure 1). Thus, the framework can be used as a logical basis to explore the relationship between increased maternal toxicant exposure and oxidative/inflammatory stress and poor health outcomes in crude-oil-exposed communities.

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DOI: https://doi.org/10.64388/IREV8I11-1722732

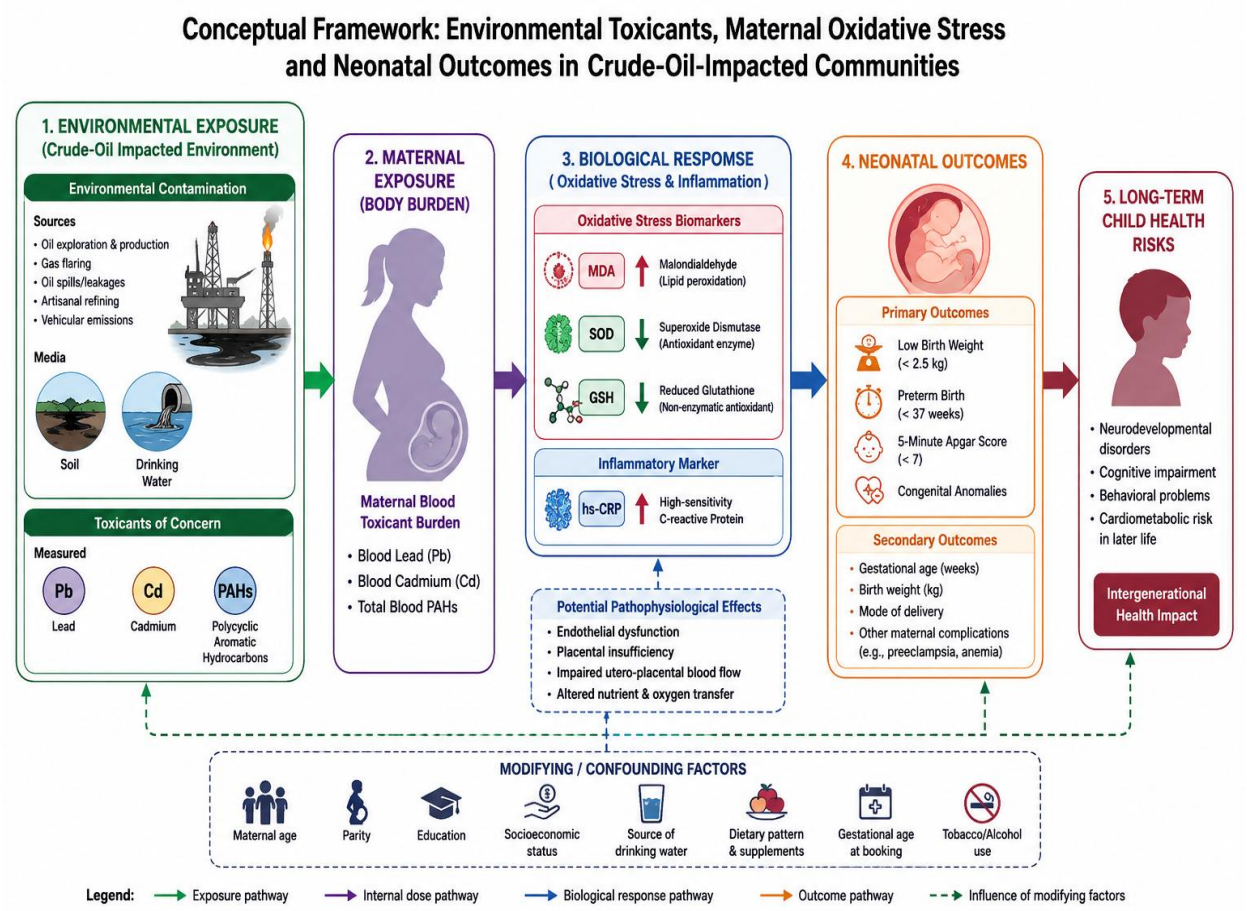

Figure 1: Conceptual Framework: Environmental Toxicant, Maternal Oxidative Stress, and Neonatal Outcomes in Crude-Oil-Impacted Communities

The hypothesized relationship between exposure to environmental toxicants from petroleum exploration and exploitation and adverse neonatal outcomes is shown in Figure 1. Lead (Pb), Cadmium (Cd) and PAHs in crude oil activities can be found in soil, water and air, which leads to an increase in the toxicant burden of mothers due to ingestion, inhalation and dermal exposure. The increased MDA and decreased SOD and GSH could be a sign of elevated toxicant levels, causing systemic inflammatory changes as measured by hs-CRP. These may affect placental function, uteroplacental circulation and fetal growth, resulting in low birth weight, preterm delivery, low 5-minute Apgar scores and congenital anomalies. Exposure, biological response and neonatal outcome may be further modified by maternal/environmental factors (socio-economic, diet and behaviour).

II. MATERIALS AND METHODS

A comparative cross-sectional design was used to investigate exposure to environmental toxicants, maternal biomonitoring markers, oxidative/inflammatory responses, and outcomes in the newborns of pregnant women living in Warri, Delta State, Nigeria, in communities affected by exposure to crude-oil (see Figure 2). The exposed group consisted of women from the communities of Ijala-Ikenren, Ugborodo and Ifie-Kporo, and Ughievwen was the comparison community. The study recruited 350 pregnant women ≥14 weeks' gestation, 175 women from each exposure category, using multistage sampling.

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Figure 2: Study setting.

Topsoil samples were taken from the 0–15 cm depth and drinking-water from the corresponding community sources. Heavy metals (lead (Pb) and cadmium (Cd)) were measured by atomic absorption spectrophotometry, and the concentration of polyaromatic hydrocarbons (PAHs) was carried out by gas chromatography–mass spectrometry after proper extraction and clean-up. Maternal venous blood was analysed for Pb, Cd, and total PAHs and plasma MDA, SOD, GSH and hs-CRP were measured as oxidative stress markers, antioxidant defense markers and systemic inflammatory markers, respectively. Neonatal outcomes assessed were birth weight, low birth weight (< 2.5 kg), gestational age, preterm birth (< 37 weeks), 5-minute Apgar score, and any visible congenital anomaly. Other conditions noted were maternal preeclampsia/gestational hypertension and anaemia. Descriptive statistics, independent-samples t tests, chi-square tests, Pearson correlation, and binary logistic regression were used for the analyses, and a p value < .05 and a 95% confidence interval were considered significant.

III. RESULTS

The results are presented in a biologically ordered sequence: environmental contamination, maternal internal dose, oxidative/inflammatory response, neonatal outcomes, and adjusted risk. This structure allows the paper to synthesise rather than merely reproduce the thesis tables.

Baseline Comparability

Table 1. Selected baseline characteristics of the study cohorts

Characteristic	Exposed (n=175)	Control (n=175)	Test	p
Maternal age, years, M ± SD	29.4 ± 5.7	29.1 ± 5.6	t = 0.50	.618
Residence >10 years, n (%)	73 (41.7)	74 (42.3)	$\chi^2 = 0.24$	.887

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Borehole/groundwater, n (%)	82 (46.9)	84 (48.0)	$\chi^2 = 0.12$	.942
Surface water, n (%)	51 (29.1)	48 (27.4)	—	—
Nulliparous, n (%)	71 (40.6)	69 (39.4)	$\chi^2 = 0.05$	.823
Gestational age at booking, weeks, M ± SD	18.6 ± 5.9	18.3 ± 5.7	t = 0.49	.625

Both the exposed and the control group were similar in the assessed baseline characteristics. There were no statistically significant differences in age, length of stay, water source, parity or gestation period when first booked ($p > .05$). This baseline similarity further enhances comparability of cohorts for subsequent exposure and outcome analyses.

Environmental Toxicant Burden

Table 2. Lead and cadmium concentrations in environmental matrices

Parameter	Exposed M ± SD	Control M ± SD	Mean difference	t	p
Soil Pb (mg/kg)	68.40 ± 15.20	24.70 ± 8.60	43.70	17.388	<.001
Soil Cd (mg/kg)	2.84 ± 0.72	0.91 ± 0.31	1.93	17.735	<.001
Water Pb (mg/L)	0.086 ± 0.021	0.019 ± 0.008	0.067	21.756	<.001
Water Cd (mg/L)	0.014 ± 0.004	0.005 ± 0.002	0.009	14.230	<.001

There was a significantly greater level of contaminated environment in the exposed communities. The concentration of both elements (Pb and Cd) was significantly elevated in exposed communities compared to controls ($p < .001$). Results reveal significant disparities between the environmental toxicant exposure of the two residential environments.

Table 3. Polycyclic aromatic hydrocarbon concentrations

PAH parameter	Exposed M ± SD	Control M ± SD	Mean difference	t	p
Total PAHs in soil (mg/kg)	18.70 ± 5.40	4.90 ± 1.80	13.80	17.905	<.001
Total PAHs in water (µg/L)	8.60 ± 2.70	2.10 ± 0.90	6.50	16.867	<.001
Carcinogenic PAHs (%)	72.0 ± 8.5	31.0 ± 7.2	41.0	23.000	<.001
Benzo[a]pyrene, soil (mg/kg)	2.84 ± 0.81	0.62 ± 0.24	2.22	—	—
Chrysene, soil (mg/kg)	2.31 ± 0.68	0.51 ± 0.19	1.80	—	—
Benzo[b]fluoranthene, soil (mg/kg)	1.97 ± 0.57	0.43 ± 0.17	1.54	—	—

The exposure of communities was associated with significant increases in the concentration of PAH in the environmental matrices assessed ($p < .001$). Remarkably, the amount of carcinogenic PAH in areas with exposures was significantly higher than in other areas, suggesting higher environmental exposure to potentially hazardous petroleum-derived compounds.

Maternal Internal Toxicant Burden

Table 4. Maternal blood toxicant concentrations

Biomarker	Exposed M ± SD	Control M ± SD	Mean difference	t	p
Blood Pb (µg/dL)	18.60 ± 5.40	6.90 ± 2.80	11.70	25.445	<.001
Blood Cd (µg/L)	4.80 ± 1.60	2.10 ± 0.90	2.70	19.457	<.001
Total blood PAHs (µg/L)	12.40 ± 4.10	5.70 ± 2.20	6.70	19.049	<.001
Above specified Pb threshold, n (%)	166 (94.9)	42 (24.0)	—	$\chi^2 = 178.52$	<.001

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DOI: https://doi.org/10.64388/IREV8I11-1722732

The pattern of internal exposure by the mothers was similar to the contamination pattern in the environment. The levels of blood Pb, Cd and total PAHs were significantly higher in women of exposed communities ($p < .001$). Furthermore, 94.9% of the exposed women exceeded the specified level of lead, while only 24.0% of the controls did. This suggests that there was significant differential exposure to toxicant in the exposed women.

Maternal Oxidative and Inflammatory Status

Table 5. Maternal oxidative-stress and inflammatory biomarkers

Biomarker	Exposed M ± SD	Control M ± SD	Mean difference	t	p
MDA (nmol/mL)	6.80 ± 1.40	4.20 ± 1.00	2.60	19.992	<.001
SOD (U/mL)	71.50 ± 12.00	94.00 ± 14.50	−22.50	−15.814	<.001
GSH (µmol/L)	3.90 ± 0.80	6.10 ± 1.00	−2.20	−22.726	<.001
hs-CRP (mg/L)	7.20 ± 2.90	3.80 ± 1.70	3.40	13.380	<.001

The exposed cohort had a significantly poorer biological profile with elevated MDA levels, and significantly lower levels of SOD and GSH ($p < .001$), and elevated levels of hs-CRP ($p < .05$). These data overall suggest elevated levels of oxidative stress, impaired antioxidant defence and greater systemic inflammatory activity in exposed women.

Maternal and Neonatal Outcomes

Table 6. Maternal and neonatal clinical outcomes

Outcome	Exposed n (%)	Control n (%)	χ^2	p
Preeclampsia/gestational hypertension	28 (16.0)	12 (6.9)	7.226	.007
Maternal anaemia	62 (35.4)	35 (20.0)	10.397	.001
Low birth weight <2.5 kg	35 (20.0)	18 (10.3)	6.426	.011
Preterm birth	31 (17.7)	14 (8.0)	7.370	.007
5-minute Apgar <7	24 (13.7)	10 (5.7)	6.385	.012
Observable congenital anomaly	10 (5.7)	1 (0.6)	7.603	.006

The exposed group had significantly increased rates of adverse pregnancy and birth outcomes. There were significant increases in the exposed group for all of the following: preeclampsia/gestational hypertension, maternal anaemia, low birth weight, preterm birth, low 5-minute Apgar scores and presence of observable congenital anomalies ($p \leq .012$ each).

Table 7. Continuous neonatal outcomes

Outcome	Exposed M ± SD	Control M ± SD	t	p
Birth weight (kg)	2.84 ± 0.42	3.18 ± 0.38	−7.941	<.001
Gestational age at delivery (weeks)	37.10 ± 2.30	38.50 ± 1.50	−6.745	<.001
5-minute Apgar score	7.80 ± 1.00	8.50 ± 0.70	−7.586	<.001

Neonates in the exposed cohort were significantly underweight, low gestational age at delivery and low 5 min Apgar score compared to neonates in the control cohort ($p< .001$). The high level of consistency seen for these continuous measures further validates the pattern of poorer birth outcomes seen in exposed communities.

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DOI: https://doi.org/10.64388/IREV8I11-1722732

Toxicant Burden, Oxidative Stress and Birth Weight

Table 8. Pearson correlations linking maternal toxicants, oxidative stress and birth weight

Variable pair	r	p	Interpretation
Blood Pb ↔ MDA	.620	<.001	Positive
Blood Cd ↔ MDA	.550	<.001	Positive
Total blood PAHs ↔ MDA	.480	<.001	Positive
Blood Pb ↔ SOD	−.410	<.001	Negative
Blood Pb ↔ GSH	−.360	<.001	Negative
Total blood PAHs ↔ SOD	−.440	<.001	Negative
Total blood PAHs ↔ GSH	−.520	<.001	Negative
Birth weight ↔ Blood Pb	−.520	<.001	Negative
Birth weight ↔ MDA	−.440	<.001	Negative
Birth weight ↔ GSH	.390	<.001	Positive

The toxicant burden in the mothers was significantly related to oxidative imbalance. MDA was positively correlated with blood Pb, Cd and PAHs, and negatively correlated with SOD and GSH ($p < .001$). As is reported above, a negative correlation was seen between birth weight and blood levels of both Pb and MDA, while a positive correlation occurred between birth weight and GSH, indicating a uniform pattern of increased toxicant exposure, oxidative stress and decreased fetal growth.

Multivariable Prediction of Low Birth Weight

Table 9. Multivariable logistic regression predicting low birth weight

Predictor	aOR	95% CI	p
Residence in exposed community	2.85	1.62–5.02	<.001
Blood Pb	1.08	1.04–1.13	<.001
Blood Cd	1.22	1.08–1.38	.001
Maternal MDA	1.31	1.12–1.54	<.001
SOD	0.97	0.95–0.99	.004
Maternal age	1.01	0.97–1.05	.612
Multiparity	0.78	0.45–1.36	.381
Gestational age at booking	0.95	0.92–0.98	.002

Residence in an exposed community was still significantly related to low birth weight after controlling for selected maternal variables (aOR = 2.85, 95% CI [1.62–5.02], $p < .001$). The odds of low birth weight increase with each independent increase of blood Pb, Cd, and MDA, while an increase in blood SOD reduced the odds of low birth weight. The maternal age and multiparity were not significant predictors.

IV. DISCUSSION

The main contribution of this study is the merging of four evidence streams. First, the levels of Pb, Cd and PAH found in the exposed communities in environmental media were significantly elevated. Second, maternal blood samples showed an increased level of internal toxicant load. Third, women with exposed showed a homogeneous oxidative/inflammatory profile with increased MDA and decreased SOD and GSH, in addition to increased hs-CRP. Fourth, mean birth weight, mean gestational age, and the number of low birth weight, preterm birth, low 5-minute APGAR score, and known congenital anomalies were consistently poorer in neonates. The modified model also maintained residence, Pb, Cd and MDA as important predictors for low birth weight.

The environmental findings are in line with the general evidence base in the Niger Delta. Heavy metal contamination is found in most of the water bodies in the Niger Delta, while persistent PAH contamination is reported for environmental and biological compartments (Umeoguaju et al., 2022; Udom et al., 2023; Onyena et al., 2024). In the present study, a pregnancy-specific perspective is added, because the internal maternal-dose gradient was shown to accompany the environmental gradient. The biomonitoring results from the mothers are especially useful because they decrease the need to use residential proximity as a proxy measure of exposure. The mean exposure level of blood Pb among exposed women was 18.60 µg/dL (6.90 µg/dL for controls). The environmental and biological concentrations correspond and the study cannot differentiate the exact source of the inhalation or water or food/occupational exposure, but this is consistent with an extended multiple route exposure. A recent study on another oil-producing community in the Niger Delta, also observed higher levels of serum lead and cadmium, elevated levels of lipid peroxidation and a lower antioxidant activity (Oyeyemi et al., 2024).

The oxidative stress results are biologically plausible. A higher level of MDA suggests a greater level of lipid peroxidation and lower levels of SOD and GSH suggest lower levels of antioxidant defence. An increase in both hs-CRP and DCP is evidence of systemic inflammatory activation. Importantly, this interpretation is supported by the correlations: positive correlations exist between MDA and Pb, Cd and PAHs; and inverse correlations exist between MDA and SOD/GSH, and between Pb and SOD/GSH. This pattern is also more informative than the biological response when compared to only an exposed versus control comparison because it suggests that the biological response was parallel to the increase in internal toxicant burden.

The neonatal observations are in keeping with the emerging worldwide evidence that fetal growth and prenatal environmental exposure are related. Vigeh et al. (2023) concluded that there was an inverse association between prenatal blood lead and birth weight, and Latifi et al. (2024) concluded that maternal PAH exposure was associated with a decrease in birth weight and a decrease in birth length. The present results are consistent with a larger body of literature indicating that exposure to toxicants may alter fetal growth, although the amount and value of exposure may vary in different populations.

The adjusted models of the study give further evidence. Living in an exposed community continued to be significantly related to low birth weight (2.85-fold increased chances) after adjustment. More importantly, blood Pb, Cd and MDA showed independent association and SOD showed an inverse association with the low birth weight. This is consistent with a model where exposure to the environment is not simply a geographic designation, but it is measurable as internal dose and measurable as a biological response.

Simultaneously the results must not be overemphasized. Temporality is not possible with the cross-sectional design. The conceptual model suggests oxidative stress as an intermediate pathway but longitudinal mediation or structural-equation modelling were not conducted in the present analysis. Similarly, communities impacted by petroleum are exposed to mixtures, not to individual chemicals. Thus, in addition to the particulate matter, VOCs, combustion products and contaminated food, Pb, Cd and PAHs can play an important role. The observed coefficients should therefore be understood as associations of exposure mix components.

The results also have an implication for antenatal care. The prospective Niger Delta study by Oghenetega et al. (2022) provides proof for studying oil pollution effects on the mother at the scale of the population. The next logical step in Warri is then to integrate antenatal surveillance with repeated biomonitoring, environmental sampling and placental assessment. These designs may help identify the harmful effects of exposure that are associated with birth outcomes, and may also assess whether oxidative stress is a mechanism for specific toxicant–birth outcome interactions.

Lastly, study has environmental-health importance other than managing patients. There is a recent review of Niger Delta that highlights the interdependence of pollutions, livelihoods, food systems and health (Gbadamosi & Aldstadt, 2025). So, a response which ends with treatment after delivery would be an incomplete one. Interdependent source control, safe water, and environmental remediation, along with pollution surveillance and monitoring of maternal-child health, must all play their part in risk reduction.

Public-Health and Policy Implications

- Include community history of environmental exposures in prenatal risk assessment in communities with long-term petroleum exposure.
- Improve surface-water use for safe drinking water in communities where surface-water use could contribute to exposure.
- Take into account a targeted biomonitoring of maternal blood for Pb and Cd in high-risk communities and apply the results to appropriate clinical interpretation, not stand-alone laboratory testing.
- Improve environmental monitoring for pollutants such as PAHs and heavy metals in soil and water, in addition to regular maternal and neonatal health monitoring.
- Focus on longitudinal mother-infant studies with repeated exposures, placental biomarkers and other oxidative/inflammatory endpoints.
- Engage in an inter-sectoral approach to the management of petroleum pollution where environmental quality, food systems, maternal health and child development are considered as a One Health approach.

Strengths and Limitations

The study has a number of strengths. Assesses residential exposure classification in addition to environmental measurements and human biomonitoring. It also explores oxidative and inflammatory markers and neonatal outcome, thus contributing to a unified picture of a biological story. The balanced sampling of 175 women in each cohort enhances the comparability of the groups and multivariable logistic regression permits partial control of measured confounding.

The principal drawback is the cross-sectional design by comparison. It is not clear whether exposure occurs before or after oxidative stress and birth outcome occurs. The selected biomarkers are not able to cover the whole exposome and exposure is a complex mixture. Uncontrolled dietary, occupational, household combustion, socioeconomic and environmental factors still have the potential to confound the results.

V. CONCLUSION

The present study presents a consistent relationship between crude-oil-impacted residence, environmental lead/cadmium/pah burden, maternal internal concentration of toxicants, oxidative/inflammatory disturbance and adverse neonatal outcomes in Warri communities. The exposed cohort had elevated environmental and biological toxicant levels, elevated MDA and hs-CRP, decreased SOD and GSH, decreased mean birth weight and mean gestational age, and increased prevalence of low 5-minute Apgar scores and observed congenital anomalies. The modified model additionally determined that exposure status, blood Pb, blood Cd and MDA were positive predictors of low birth weight and SOD had an inverse association.

Collectively, the results solidify the argument for incorporating environmental health monitoring into maternal and neonatal health services in communities affected by petroleum. They also offer a transparent framework for longitudinal studies that will allow testing of temporality, mediation and cumulative exposure. These findings should be considered associations and not evidence of direct effects of crude-oil pollution on the outcomes observed.

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