

Crude Oil Polluted Water as a Vector for Heavy Metal Load and 8 Isoprostane Mediated Oxidative Damage in a Rat Model

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ARTICLE INFORMATION	ABSTRACT
<p>Article history: Published: March 2026</p> <p>Keywords: Crude oil pollution Heavy metal bioaccumulation Oxidative damage Contaminated drinking water Niger Delta Nigeria</p>	<p>Twenty male albino rats were maintained for one year and randomly assigned to two groups (n=10 each). Group 1 (control) received standard rodent chow (Coppens) and borehole water, while Group 2 was fed the same diet but with river water from the crude oil-impacted Igbeta-Ewoama community. At study endpoint, blood concentrations of manganese, lead, cadmium, selenium, mercury, chromium, and the oxidative stress biomarker 8-iso-prostaglandin F2α (8-iso-PGF2α) were. Group 2 exhibited significantly elevated levels of all six metals (p < 0.05), alongside higher 8-iso-PGF2α concentrations (p < 0.05), compared to controls. These data demonstrate heavy metal bioaccumulation and induced oxidative stress in rats chronically exposed to oil-polluted river water, highlighting environmental health risks in crude oil-drilling regions.</p>

1. Introduction

Crude oil exploration in the Niger Delta region of Nigeria has led to extensive environmental contamination, particularly of surface waters used by local communities for drinking and domestic purposes (Orisakwe et al., 2024). This pollution introduces complex mixtures of hydrocarbons and heavy metals, including cadmium (Cd), chromium (Cr), mercury (Hg), lead (Pb), selenium (Se), and manganese (Mn), which bioaccumulate in aquatic organisms and enter the food chain, posing significant health risks (Adedara et al., 2013). Chronic exposure to these contaminants induces oxidative stress through reactive oxygen species (ROS) overproduction, overwhelming endogenous antioxidant defenses and causing lipid peroxidation, DNA damage, and organ dysfunction (Ifenkwe et al., 2023; Patra et al., 2011). Despite growing evidence of ecological impacts, few studies have modeled long-term mammalian exposure to oil-impacted river water, justifying controlled rodent experiments to elucidate bioaccumulation patterns and biomarker responses.

1.1 Environmental Context

The Niger Delta, home to over 30 million people, experiences persistent crude oil spills, with over 13,000 incidents documented between 2006 and 2020, contaminating rivers like those in Igbeta-Ewoama (Orisakwe et al., 2024). These spills leach toxic metals from geological formations and drilling byproducts into waterways, exceeding WHO permissible limits for potable water—e.g., Cd (0.003 mg/L), Pb (0.01 mg/L), and Hg (0.006 mg/L)—as reported in regional surveys (Arojoye et al., 2021; Thomas et al., 2021). Residents rely on these rivers for all that water is needed for, amplifying exposure risks via direct consumption or irrigated crops, with human biomonitoring showing elevated serum metals and oxidative markers mirroring polluted sites (Ifenkwe et al., 2018; Orisakwe et al., 2024).

1.2 Heavy Metal Toxicology

Non-essential metals like Cd, Cr, Hg, and Pb disrupt cellular homeostasis by binding sulfhydryl groups, inhibiting enzymes, and generating ROS via Fenton-like reactions, leading to mitochondrial damage and apoptosis (Patra et al., 2011). Essential Mn, while regulated homeostatically, accumulates neurotoxically at high levels, depleting glutathione and elevating lipid peroxides (Milatovic et al., 2009). Se exhibits dual roles, mitigating oxidative damage at low doses but contributing to stress in excess (Wang et al., 2015). Rodent models confirm dose-dependent tissue accumulation from contaminated feeds, with liver as a primary depot (Adedara et al., 2013).

1.3 Oxidative Stress and 8-Isoprostane

8-iso-prostaglandin F2 α (8-iso-PGF2 α), an F2-isoprostane, serves as the gold-standard biomarker of non-enzymatic lipid peroxidation, reflecting in vivo ROS burden with superior specificity over malondialdehyde (Roberts & Morrow, 2017). Elevated urinary/plasma levels correlate with xenobiotic exposures in animals and humans, distinguishing chemical from cyclooxygenase-mediated origins via the 8-iso-PGF2 α /PGF2 α ratio (van 't Erve et al., 2016). In fish from Niger Delta rivers, oil pollution similarly boosts hepatic isoprostanes, linking to genotoxicity (Arojoye et al., 2021).

1.4 Research Gaps and Justification

While aquatic species studies abound, chronic mammalian models using authentic oil-impacted water are scarce, limiting translational insights for Niger Delta communities (Adedara et al., 2013). Prior work focused on acute crude oil gavage or diet, overlooking drinking water as the dominant exposure route (Ebokaiwe & Farombi, 2016). This one-year study addresses this by quantifying metal bioaccumulation and 8-iso-PGF₂ α in rats exposed to Igbeta-Ewoama river water versus controls, providing causal evidence of pollution-driven oxidative stress, informing remediation policies and public health interventions in oil-bearing regions.

2. Methodology

2.1 Experimental Design

Twenty male albino rats (*Rattus norvegicus*), initially weighing 52.40 ± 4.40 g, were obtained from the Small Animal Holding Unit, Department of Pharmacy, Niger Delta University, Wilberforce Island, Amassoma, Bayelsa State, Nigeria. Rats were acclimatized to laboratory conditions (12:12 h light:dark cycle, $24 \pm 2^\circ\text{C}$, $55 \pm 5\%$ humidity) for one week with ad libitum access to standard rodent chow (Coppens) and water. The rats were randomly allocated to two groups (n = 10/group) and maintained for 12 months. Group 1 (control) received standard rodent chow and borehole water. Group 2 (experimental) received identical chow but river water sourced from the crude oil-impacted Igbeta-Ewoama community, Bayelsa State, to induce chronic heavy metal exposure and oxidative stress.

2.2 Sample Collection

At study termination, rats were humanely anesthetized with chloroform vapor. Cardiac puncture via the jugular vein yielded whole blood: serum samples (for 8-iso-PGF₂ α) were collected in plain tubes, allowed to clot for 10–20 minutes at room temperature, and centrifuged at 3,000 rpm for 20 minutes to isolate serum (supernatant stored at -20°C until analysis). Whole blood for heavy metals was collected in K₃EDTA tubes, gently inverted to mix properly and stored at $1-6^\circ\text{C}$ before analysis.

2.3 Heavy Metal Analysis

Blood concentrations of manganese, lead, cadmium, selenium, mercury, and chromium were quantified using a 240 FS AA Agilent Technologies flame atomic absorption spectrometer (FAAS) following nitric acid digestion, per standard protocols (AOAC, 2019). Calibration curves were constructed with certified reference standards and the concentrations of the metals were determined from the standard curves.

2.4 Oxidative Stress Biomarker Assessment

Serum 8-iso-prostaglandin F₂ α (8-iso-PGF₂ α), was measured via competitive ELISA using Elabscience Biotechnology 8-iso-PGF₂ α ELISA kit according to manufacturer instructions. Absorbance was read at 450 nm and concentrations were determined from standard curves.

2.5 Statistical Analysis

Data are expressed as mean \pm standard deviation. Group differences were assessed via unpaired Student's t-test. Correlations employed the Pearson correlation coefficient (Pearson's r). Significance was set at $p < 0.05$ using GraphPad Prism v9.0.

4. Findings

Figure 1 illustrates heavy metal concentrations in the two experimental groups. Rats exposed to crude oil-contaminated water (Group B) exhibited significantly elevated levels of all six measured metals compared to the control group, with statistically significant differences for cadmium, chromium, mercury, lead, and selenium ($p < 0.05$). Manganese levels did not differ significantly between groups.

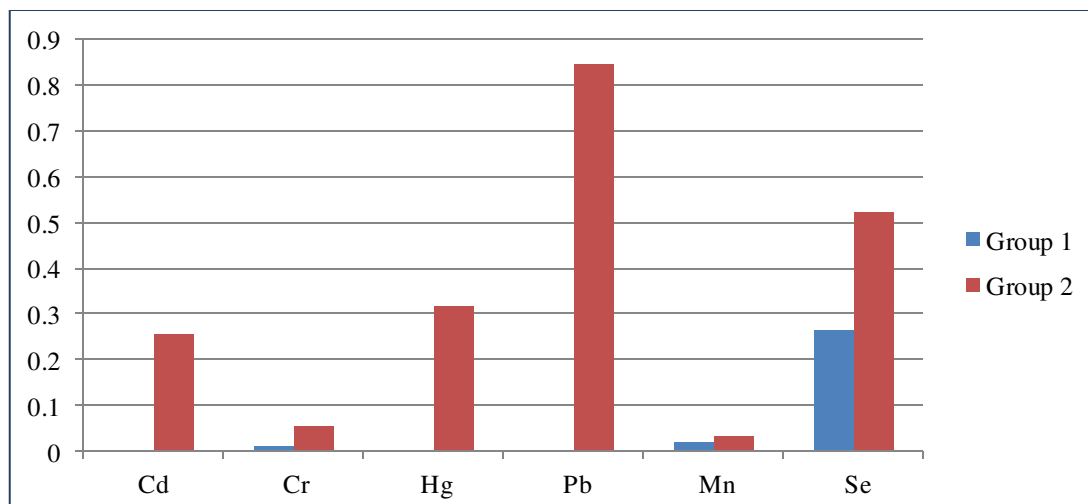


Figure 1: Mean Values of Heavy Metal Levels in the two Animal Groups.

Figure 2 depicts 8-iso-prostaglandin F2 α concentrations, in the two experimental groups. Rats exposed to river water from the oil-producing community (Group 2) exhibited significantly higher levels compared to controls (p < 0.05).

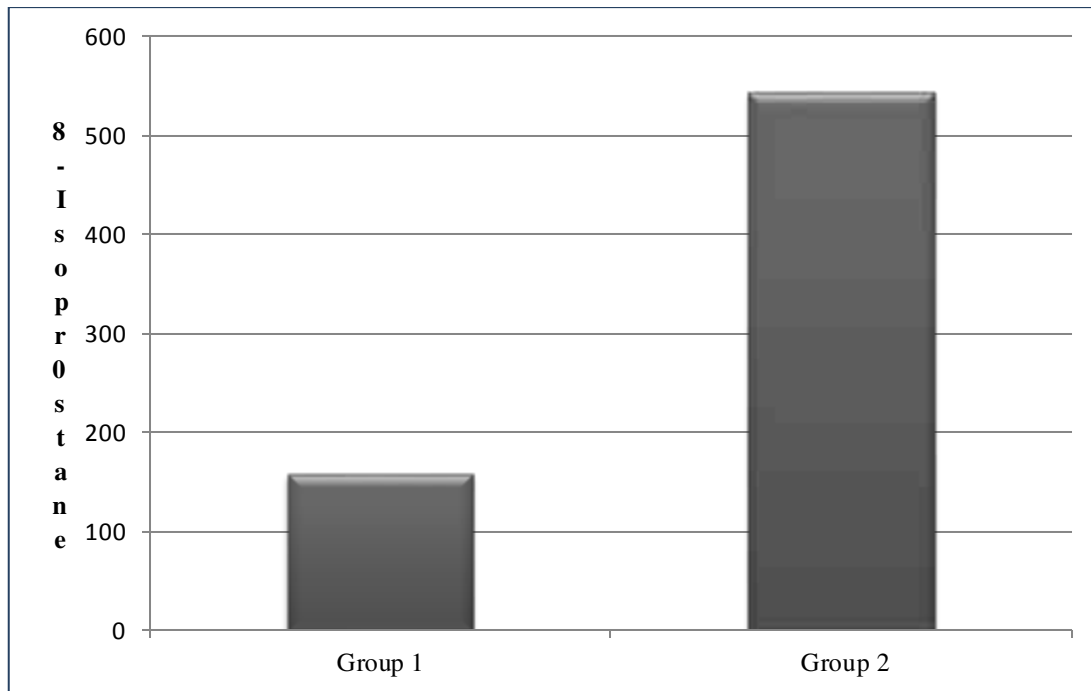


Figure 2: 8-Iso-Prostaglandin F2 α Concentrations in the Two Animal Groups

Table 1 presents the Pearson correlation coefficients between 8-isoprostane levels and the measured metals in experimental group 2. A strong positive correlation was observed with mercury (r = 0.653), a moderate positive correlation with manganese (r = 0.330), and weak positive correlations with cadmium (r = 0.240), chromium (r = 0.290), lead (r = 0.221), and selenium (r = 0.089). Statistical significance was attained only for the correlation with manganese.

Table 1: Correlation between the Measured Metals and and 8-Iso-Prostaglandin F2 α in Group 2

8-Isoprostane	Cadmium		Chromium		Mercury		Lead		Selenium		Manganese	
	R	P	R	p	R	P	R	P	R	P	R	P
	0.24	0.416	0.29	0.352	0.653	0.54	0.221	0.807	0.089	0.766	0.33	0.041

4. Discussion

These results demonstrate that crude oil-contaminated water serves as a potent vector for heavy metal bioaccumulation in rats, significantly elevating levels of non-essential metals (cadmium, chromium, mercury, lead, and selenium) while preserving essential manganese homeostasis (Adedara et al., 2013). Exposure to Nigerian Bonny Light Crude Oil similarly promotes metal leaching into water, facilitating uptake and tissue accumulation in rodents, which parallels groundwater contamination patterns in the Niger Delta and amplifies systemic toxicity (Adedara et al., 2013 ; Ebokaiwe & Farombi, 2016 ; Orisakwe et al., 2024).

Elevated non-essential metal concentrations indicate heightened risks of oxidative stress, endocrine disruption, and organ damage, including mercury- and lead-induced neurotoxicity as well as cadmium- and chromium-associated carcinogenicity. These effects align with contaminated feed studies showing induced lipid peroxidation (Ebokaiwe & Farombi, 2016). The non-significant changes in manganese levels suggest effective homeostatic regulation, preventing overload (Milatovic et al., 2009).

Exposure to oil-polluted river water also induced significant oxidative stress in rats, as evidenced by markedly elevated 8-isoprostane F2 α levels - a validated biomarker of lipid peroxidation (van 't Erve et al., 2016). In the Niger Delta, oil contamination introduces hydrocarbons and heavy metals that drive reactive oxygen species (ROS) overproduction, increasing 8-isoprostane levels comparable to those in human serum from affected communities (Orisakwe et al., 2024; Arojokoye et al., 2021). Such elevations portend inflammation, DNA damage, organ dysfunction, and carcinogenesis, necessitating water remediation and antioxidant strategies in impacted ecosystems (Adedara et al., 2013; van 't Erve et al., 2016, Ifenkwe et al., 2023).

Correlation analyses further identified mercury and manganese as primary drivers of oxidative stress, exhibiting strong to moderate positive associations with 8-isoprostane levels (Roberts & Morrow, 2017; Milatovic et al., 2009). Mercury promotes ROS generation and lipid peroxidation, leading to cellular injury (Wang et al., 2015), while manganese excess impairs mitochondria, elevates F2-isoprostanes, and induces inflammation and neurodegeneration (Aschner et al., 2013; Milatovic et al., 2009). Cadmium, chromium, lead, and selenium displayed weaker positive correlations, reflecting subtler contributions at tested doses; notably, cadmium and lead deplete antioxidants such as glutathione peroxidase, with selenium exerting modulatory effects (Patra et al., 2011; Wang et al., 2015). These insights prioritize mercury and manganese mitigation in polymetallic exposures, guiding neuroprotective interventions and environmental biomonitoring (Milatovic et al., 2009; Wang et al., 2015).

5. Conclusion and Recommendations

5.1 Conclusion

This study provides compelling evidence that chronic exposure to water from the crude oil-impacted river induces significant heavy metal bioaccumulation and oxidative stress in male albino rats. Thus, crude oil-contaminated water poses a significant risk to environmental and human health through heavy metal bioaccumulation and oxidative stress. The findings underscore the urgent need for stringent water quality monitoring, remediation strategies, and regulatory frameworks to mitigate the adverse effects of oil pollution in regions like the Niger Delta. Prioritizing mercury and manganese mitigation, alongside antioxidant interventions, may help alleviate the toxicological impacts of polymetallic exposure, protecting vulnerable ecosystems and communities exposures (Milatovic et al., 2009). These findings validate rodent models for extrapolating Niger Delta environmental risks, revealing pollution-driven pathways to cellular damage, inflammation, and potential organ dysfunction that mirror human health threats in oil-producing regions.

5.2 Recommendations

Based on the results and preceding discussion, the following evidence-based recommendations are proposed for all stakeholders involved in this process.

- Government agencies and state Ministries of Environment and Health should institute routine monitoring of surface and groundwater in crude oil-producing communities, prioritizing mercury, manganese, lead, cadmium, chromium, and other petroleum-related metals.
- Relevant regulatory bodies should establish and enforce clear maximum allowable limits for these metals in drinking water, aligned with WHO and national standards, with mandatory public reporting of any exceedances.
- An early-warning system should be created so that once contaminant levels exceed thresholds, immediate public advisories are issued, alternative safe water is provided, and contaminated sources are temporarily closed.
- In highly impacted areas, government should phase out direct consumption of river and shallow well water and provide safe alternatives such as treated municipal supply, boreholes with proven low contamination, or community-scale treatment units.
- Vulnerable groups (children, pregnant women, elderly, chronically ill) should be prioritized for access to safe water, health screening, and follow-up, given their increased susceptibility to heavy metal toxicity and oxidative damage.
- Community-level risk communication programs should be developed in local languages, explaining the dangers of consuming contaminated water, pathways of exposure (drinking, cooking, bathing, fishing), and practical protective behaviors.
- Oil operators should conduct independent environmental audits to identify point and non-point sources of metal contamination (e.g., leaking pipelines, produced-water discharge, artisanal refining sites) and implement source-control measures to minimize ongoing pollution.
- Evidence-based remediation technologies—such as constructed wetlands, adsorption/filtration systems, phytoremediation, and depth-targeted sediment removal—should be deployed in heavily impacted river segments used for domestic purposes.
- Post-remediation monitoring must be mandatory, with long-term tracking of metal levels in water, sediments, and biota to verify that interventions actually reduce exposure and ecological risk.
- Screening programs should be established for residents of oil-impacted communities, including periodic measurements of blood heavy metals and oxidative-stress/organ-function markers (e.g., liver, kidney, and neurological assessments).
- Based on the present findings and mechanistic work on manganese and mercury neurotoxicity and oxidative damage, antioxidant-supportive strategies (dietary antioxidants, micronutrient supplementation where appropriate) should be incorporated into community health programs, while emphasizing that these do not replace exposure reduction.
- Clinicians in Niger Delta health facilities should receive targeted training to recognize subclinical heavy-metal-related syndromes (neurocognitive changes, renal and hepatic dysfunction, reproductive issues) and to integrate environmental exposure history into routine clinical assessment.
- Federal and state governments should update and strictly enforce environmental and public-health regulations on crude oil exploration, production, transport, and spill response, with explicit provisions for heavy metals and chronic low-dose exposure.
- An independent Environmental and Toxicology Surveillance Unit for the Niger Delta should be created, tasked with integrating environmental data, health outcomes, and laboratory research, and empowered to recommend enforcement actions and community protections.
- Oil companies should be mandated to contribute to a dedicated Environmental Health Trust Fund used to finance water infrastructure, remediation projects, medical screening, and research in affected communities.

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