Journal: Texila International Journal of Public Health
Publisher: Texila International Journal
ISSN: 2520-3134
Volume 13 Issue 4, 2025
DOI: 10.21522/TIJPH.2013.13.04.Art012

# Systemic Biochemical Disruptions from Chronic Generator Exhaust Exposure: Oxidative, Hematologic, and Heavy Metal Biomarkers in Urban Nineveh, Iraq

Shahla Mohammed Farhan, Yusra M.S. Al-Shaker, Liqaa Saeed Alkhalidy*
*College of Environmental Sciences, University of Mosul, Mosul, Iraq*

***Abstract***

*In urban regions with chronic power instability—such as Mosul, Iraq—diesel generators are essential yet environmentally hazardous. Prolonged exposure to generator exhaust introduces a spectrum of toxicants, including carbon monoxide, nitrogen oxides, and heavy metals, raising significant concerns for long-term systemic health effects. This research aimed to explore changes in body systems and blood composition resulting from long-term exposure to diesel generator fumes among community members. The study used a cross-sectional comparative design with 260 participants, divided into exposed (150) and control (110). The analysis included blood gases; oxidative stress indicators like MDA and GSH; inflammatory markers such as CRP and CA; hematological parameters like HgB, and PCVs; and serum levels of trace metals including Zink, Zn, Cadmium, Nickel. The significance is at p≤0.05. Exposed Individuals showed decreases in oxygen (lower Pa02 and Sa02, and higher COHa levels), an increase in red blood cell production and hemoglobin, and a significant increase in MDA and a decrease in GSH. Also, C protein (CRP) levels increased significantly at p < 0·001). Trace metal analysis following exposure showed a buildup of lead (Pb) alongside cadmium (Cd), Copper (Cu), and Nickel (Ni), with a simultaneous decrease in zinc content, further weakening the body's antioxidant defenses. The results show that long-term exposure to generator fumes leads to health issues, including breathing problems, imbalances in the body's oxidation processes, and the accumulation of harmful metals in the system.*

***Keywords:*** *Carbon Monoxide Toxicity, Diesel Generator, Erythropoiesis, Heavy Metals, Oxidative Stress, Pollution.*

## Introduction

The growing dependence on diesel generators in low-resource and post-conflict regions has emerged as a silent yet pervasive environmental and public health challenge. In cities like Mosul, Iraq, where chronic power outages have rendered generators an infrastructural necessity, the unintended consequence has been sustained exposure to airborne pollutants, notably carbon monoxide (CO), nitrogen oxides ($NO_x$), polycyclic aromatic hydrocarbons (PAHs), and heavy metals—all by-products of incomplete fossil fuel combustion [1, 2]. Prolonged inhalation of such toxicants is known to impair pulmonary function, disrupt hematological homeostasis, and instigate systemic oxidative stress, thereby increasing the risk of chronic respiratory, cardiovascular, and neurological disorders [3–5]. Mechanistically, generator emissions exert their biological toxicity through multiple converging pathways. Carbon monoxide competitively binds to hemoglobin, forming carboxyhemoglobin (COHb), thereby reducing oxygen delivery and precipitating tissue

***Received: 16.07.2025*** ***Accepted: 23.10.2025*** ***Published on: 24.12.2025***
***Corresponding Author: liqaasaeed@uomosul.edu.iq***

hypoxia [6, 7]. In tandem, reactive oxygen species (ROS) generated by heavy metals such as lead (Pb) and cadmium (Cd) overwhelm endogenous antioxidant defenses, including glutathione (GSH) and zinc-dependent enzymes, resulting in lipid peroxidation, DNA damage, and mitochondrial dysfunction [8-10].

## Materials and Methods

### Study Design and Population Sampling

A research project comparing two populations was conducted in Mosul from September 8th to January 6th in 2024 and 2025—the widespread use of diesel generators has heavily impacted the city due to ongoing electricity shortages in the region. The primary goal of this study was to investigate the effects of long-term exposure to generator emissions on changes within the population groups under study.

The study included 110 people in the control group living in areas without electric generators in villages like Tal Khazaf, Al Kabir, Al Mawali and 150 individuals in the exposed group who either operated generators or resided near densely populated generator locations, in urban neighborhoods People were categorized based on gender and age (ranging from 10 to 80 years) and their consent was sought before participation.

### Pulse Oximetry and Pre-screening

Prior to venipuncture or arterial sampling, each subject underwent non-invasive evaluation using a calibrated pulse oximeter (Cataldo et al., 2025. This assessment included peripheral oxygen saturation ($SpO_2$) and pulse rate (PR), which served as preliminary indicators of hypoxemia and cardiovascular strain. $SpO_2$ values below 92% were flagged for potential oxygen desaturation and documented accordingly.

### Blood Collection Protocol

Venous and arterial blood samples were collected under standardized aseptic conditions by trained medical personnel. Arterial blood (1 mL) was obtained from the radial artery using heparinized syringes and analyzed for arterial blood gases (ABG), using the ABL800 FLEX analyzer (Radiometer, USA) at Al-Jumhuri and Ibn Sina Teaching Hospitals. Simultaneously, 5 mL of venous blood was drawn from the antecubital vein and fractionated as follows:

2 mL was transferred to EDTA-coated lavender-top tubes for complete blood count (CBC) analysis, measured using Sysmex XP-300 and Nihon Kohden MEK-6510K analyzers.

The remaining 3 mL was placed in dry gel-separator tubes (yellow-top) for serum separation. Samples were centrifuged at 4000 g for 10 minutes, and the resulting serum was aliquoted and stored at −20°C for biochemical assays.

### Biochemical and Oxidative Stress Assays

The oxidative stress markers were measured using enzyme-linked immunosorbent assay (ELISA) kits according to manufacturers' protocols:

Malondialdehyde (MDA): Reed Biotech Ltd. (China), read by Bio-Tek ELISA microplate reader.

Reduced Glutathione (GSH): ELK Biotechnology (China).

Uric Acid (UA): Quantified spectrophotometrically using BIOLABO S.A.S (France) kits and Jenway 7200 and JK-VS-722N instruments.

### Enzyme and Inflammatory Marker Estimation

To assess enzymatic function and systemic inflammation:

Carbonic Anhydrase (CA) was measured via FineTest ELISA kits.

C-Reactive Protein (CRP) levels were determined using turbidimetric immunoassay kits from LTA S.r.l. (Italy), Given its relevance as a biomarker of the acute-phase inflammatory response triggered by environmental pollutants.

### Heavy Metal Analysis

To quantify bioaccumulated trace metals (Zn, Cu, Pb, Cd, Ni), atomic absorption spectrophotometry (AAS) was performed at the Central Laboratory of the College of Agriculture and Forestry, University of Mosul, using validated calibration standards and internal controls to ensure analytical reliability.

### Statistical Analysis

Descriptive data were expressed as mean ± standard error (SE). A comparative analysis between the exposed and control groups was conducted using the independent Student's t-test, with statistical significance set at $p \leq 0.05$.

## Results

The comparison of blood gas (ABG) measurements between people exposed to generator smoke and the control group showed clear differences, indicating compromised respiratory function and altered coping mechanisms under stress. It was observed that the levels of oxygen pressure (Pa $O_2$), oxygen saturation (Sa $O_2$%) and oxyhemoglobin ($O_2$ Hb%) were notably lower in the exposed group (with a p value < 0.001), suggesting a disturbance in gas exchange likely caused by prolonged exposure to carbon monoxide and other pollutants, from combustion.

The levels of deoxyhemoglobin (HHB%) and carboxyhemeoglobin (COHB%) significantly increased simultaneously which supports the idea of carbon monoxide displacing oxygen at hemoglobin binding sites.The notable increase, in HHB % aligns with decreased oxygen supply and the bodys effort to compensate with desaturation.

The results support indicators of low oxygen levels in the blood and could contribute to the adverse effects on metabolism and oxidative stress observed in long-term exposure to diesel generator emissions. In total, this collection of data provides biochemical evidence of the harmful respiratory effects of prolonged exposure to diesel generator emissions (Table 1, Figure 1).

**Table 1.** Comparative Analysis of Arterial Blood Gas Parameters

| Parameter | Control (Mean ± SD) | Exposed (Mean ± SD) | p-value | Significance |
|---|---|---|---|---|
| $PaO_2$ (mmHg) | 96.84 ± 1.07 | 79.12 ± 2.05 | 0.0000 | * |
| $SaO_2$ (%) | 97.03 ± 0.12 | 94.97 ± 0.51 | 0.0000 | * |
| $O_2Hb$ (%) | 94.20 ± 0.47 | 91.40 ± 0.81 | 0.0000 | * |
| HHb (%) | 2.85 ± 0.12 | 4.84 ± 0.48 | 0.0000 | * |
| COHb (%) | 1.27 ± 0.43 | 1.93 ± 0.43 | 0.0000 | * |
| pH | 7.41 ± 0.012 | 7.27 ± 0.028 | 0.0000 | * |
| $PaCO_2$ (mmHg) | 35.69 ± 1.15 | 46.11 ± 1.55 | 0.0000 | * |
| MetHb (%) | 1.45 ± 0.09 | 1.59 ± 0.08 | 0.0252 | * |
| Lac (mmol/L) | 1.32 ± 0.09 | 1.85 ± 0.12 | 0.0000 | * |
| $HCO_3$ (mmol/L) | 22.50 ± 0.57 | 17.96 ± 0.77 | 0.0000 | * |
| Glu (mg/dL) | 105.03 ± 17.63 | 135.70 ± 23.32 | 0.1081 | ns |
| Na (mmol/L) | 136.82 ± 1.50 | 137.80 ± 1.51 | 0.4978 | ns |
| Ca (mmol/L) | 1.17 ± 0.04 | 1.15 ± 0.04 | 0.0016 | * |
| K (mmol/L) | 3.52 ± 0.18 | 3.45 ± 0.14 | 0.6317 | ns |
| Cl (mmol/L) | 105.94 ± 1.67 | 104.55 ± 1.90 | 0.1629 | ns |

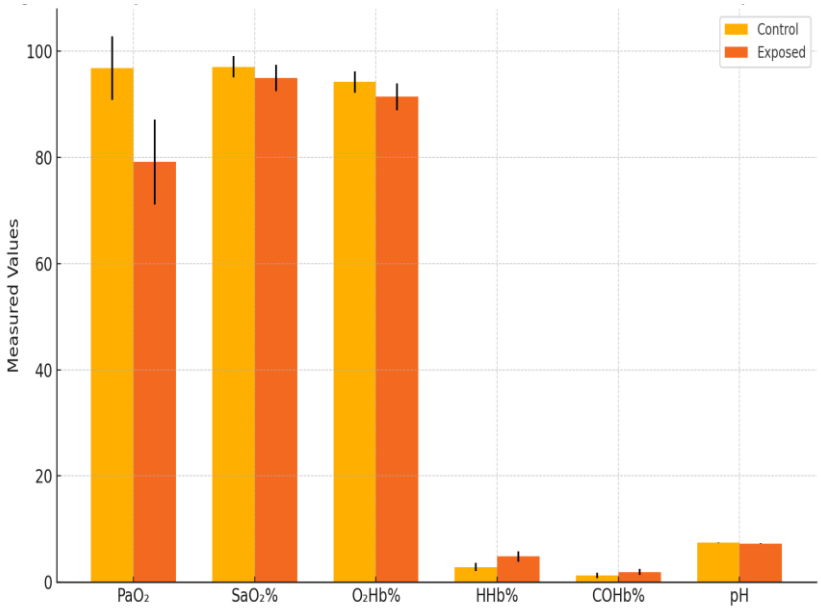


**Figure 1.** Comparison of Arterial Blood Gas Parameters between Control and Exposed Groups

The blood tests showed increases in hemoglobin levels (Hb) red blood cell count (RBCs) and packed cell volume (PCV) in people exposed to generator fumes. These results suggest a response from the body to produce red blood cells likely due to prolonged exposure to low oxygen levels caused by carbon monoxide, in the environment.

The increased Hb and RBC levels may reflect upregulated erythropoietin activity, a known adaptive mechanism that enhances the oxygen-carrying capacity of blood under sustained oxygen deprivation. Furthermore, the elevated PCV values support the evidence of hemoconcentration and an increased hematocrit fraction, reinforcing the impact of chronic environmental stress on hematopoietic dynamics.

Collectively, these hematologic alterations underscore a biologically significant shift toward oxygen delivery optimization in response to airborne pollutants, aligning with the pathophysiological profile of chronic low-grade hypoxia (Figure 2, Table 2).

As illustrated in Figure 2, significant oxidative alterations were evident in the exposed group, with elevated MDA and CA levels and reduced GSH and CRP levels, reflecting redox imbalance and a systemic inflammatory response (Table 2).

**Table 2.** Comparative Analysis of Hematological Parameters

| **Parameter** | **Control (Mean ± SD)** | **Exposed (Mean ± SD)** | **p-value** | **Significance** |
|---|---|---|---|---|
| Hemoglobin (Hb, g/dL) | 13.10 ± 0.43 | 14.52 ± 0.34 | 0.0000 | * |
| Red Blood Cells (RBCs, $10^6$/µL) | 5.07 ± 0.18 | 5.54 ± 0.06 | 0.0000 | * |
| Packed Cell Volume (PCV, %) | 40.97 ± 1.82 | 46.92 ± 0.64 | 0.0000 | * |

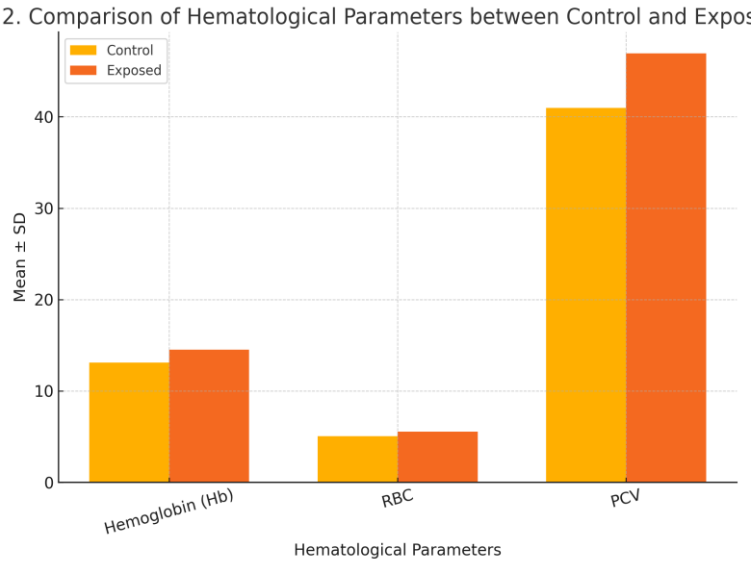


**Figure 2.** Comparison of Hematological Parameters between Control and Exposed Groups

The integrated interpretation of Table 3 reveals a striking biochemical scenario in individuals exposed to chronic generator exhaust, highlighting a multifaceted disruption of redox homeostasis and elemental balance. Notably, the marked elevation in malondialdehyde (MDA) levels, a decisive indicator of lipid peroxidation, unequivocally reflects extensive membrane lipid injury, which, in turn, suggests widespread propagation of oxidative damage across various cellular compartments. In contrast, the substantial reduction in glutathione (GSH), a principal intracellular antioxidant, illustrates a state of defensive depletion, thereby confirming that endogenous antioxidant systems are profoundly overwhelmed under persistent reactive oxygen species (ROS) pressure.

Moreover, the remarkable increase in carbonic anhydrase (CA), an enzyme fundamental to acid-base equilibrium, suggests an adaptive respiratory response to prolonged oxidative and metabolic stress, potentially serving as a compensatory mechanism to maintain systemic pH stability despite ongoing cellular insults. The parallel rise in C-reactive protein (CRP) levels, often considered a hallmark of systemic inflammation, further corroborates the presence of a sustained inflammatory milieu, which might reflect a chronic rather than merely acute immunological challenge in these individuals.

The significant decline in uric acid (UA) levels also warrants attention, as it may signal increased utilization of this secondary antioxidant reserve to buffer ongoing oxidative stress. In examining the heavy metal profile, the conspicuous accumulation of copper (Cu), lead (Pb), cadmium (Cd), and nickel (Ni) clearly indicates an exogenous toxicological load, one that not only exacerbates ROS generation but also interferes with essential mitochondrial and enzymatic functions, further amplifying oxidative and inflammatory cascades.

Conversely, the sharp decrease in zinc (Zn), an indispensable cofactor for antioxidant enzymes such as superoxide dismutase, profoundly compromises cellular defense mechanisms, rendering the organism more susceptible to cumulative oxidative injuries. Collectively, these interrelated biochemical deviations delineated in Table 3 construct a coherent narrative of systemic vulnerability, emphasizing the hidden yet potent health risks posed by continuous exposure to environmental pollutants and underscoring the critical need for preventive and mitigative strategies.

Conversely, the profound reduction in zinc (Zn), a critical cofactor for antioxidant enzymes such as superoxide dismutase, further amplifies systemic vulnerability to oxidative injury, thereby fostering a microenvironment conducive to chronic degenerative processes. Taken together, these multifaceted biochemical perturbations provide compelling evidence of the covert yet substantial health risks posed by prolonged exposure to generator emissions, underscoring an urgent imperative for robust public health interventions to curb environmental pollutant exposure and safeguard community health (Table 3, Figure 3).

**Table 3.** Comparative Analysis of Oxidative Stress, Inflammatory, and Heavy Metal Biomarkers in Control and Exposed Groups

| Parameter | Control (Mean ± SD) | Exposed (Mean ± SD) | p-value | Significance |
|---|---|---|---|---|
| MDA (ng/mL) | 313.93 ± 10.17 | 554.11 ± 73.13 | 0.0000 | * |
| GSH (ng/mL) | 94.81 ± 3.26 | 72.70 ± 4.77 | 0.0000 | * |
| Uric acid (mg/dL) | 7.19 ± 0.65 | 5.22 ± 0.47 | 0.0000 | * |
| Carbonic Anhydrase (pg/mL) | 447.66 ± 38.47 | 1137.01 ± 56.36 | 0.0000 | * |
| CRP (mg/L) | 0.002 ± 0.001 | 8.29 ± 2.16 | 0.0000 | * |
| Zinc (ppm) | 0.20 ± 0.03 | 0.04 ± 0.006 | 0.0000 | * |
| Copper (ppm) | 0.009 ± 0.0007 | 0.17 ± 0.0199 | 0.0000 | * |
| Lead (ppm) | 0.02 ± 0.005 | 0.13 ± 0.008 | 0.0000 | * |
| Cadmium (ppm) | 0.008 ± 0.0011 | 0.024 ± 0.0008 | 0.0000 | * |
| Nickel (ppm) | 0.03 ± 0.0022 | 0.04 ± 0.0018 | 0.0000 | * |

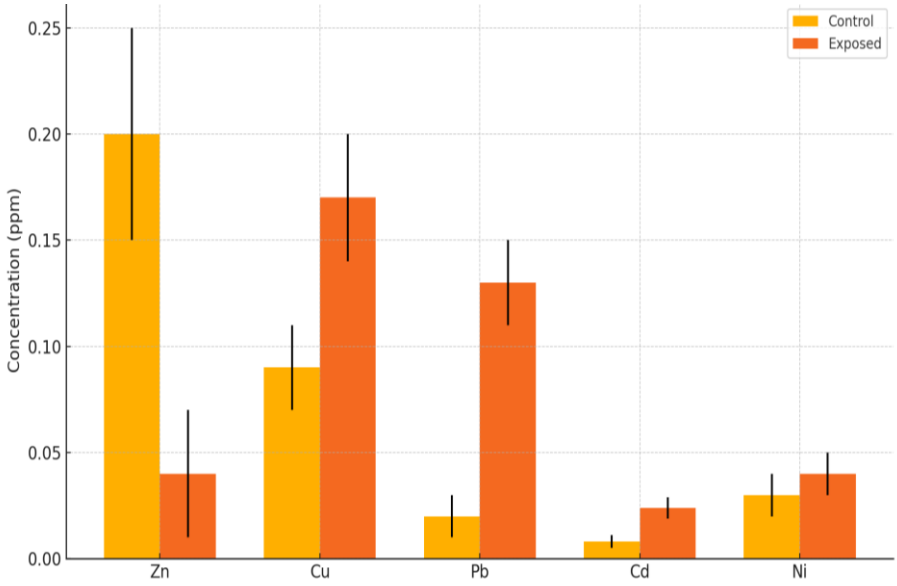


**Figure 3.** Serum Heavy Metal Concentrations in Control vs Exposed Groups

In Figure 4 of the study shows a connection where lower levels of glutathione are linked to higher malondialdehyde levels in people constantly exposed to generator fumes. It indicates a relationship between antioxidant reduction and lipid oxidation in individuals facing long-term exposure to generator exhaust fumes.

Journal: Texila International Journal of Public Health
Publisher: Texila International Journal
ISSN: 2520-3134
Volume 13 Issue 4, 2025
DOI: 10.21522/TIJPH.2013.13.04.Art012

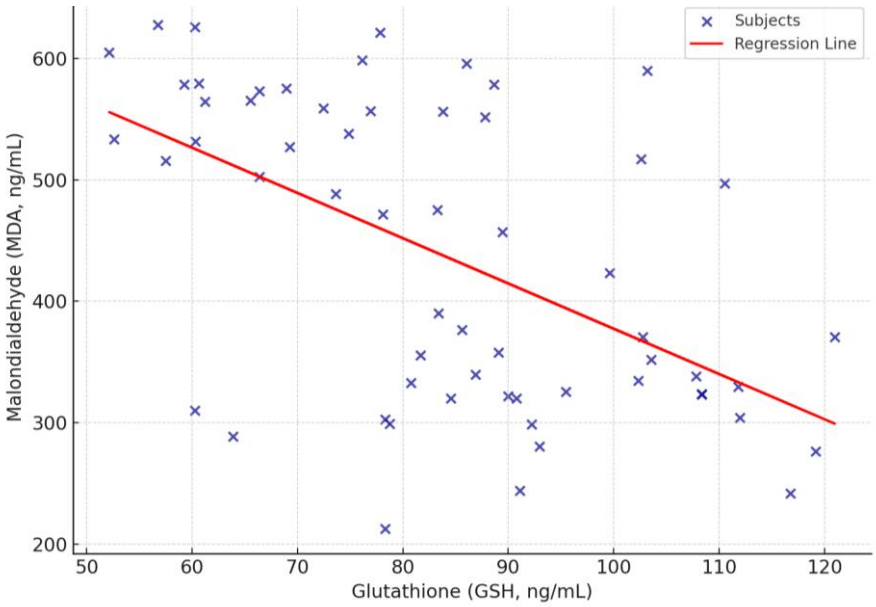


**Figure 4.** Negative Correlation between GSH and MDA Levels

## Discussion

The current investigation delineates a compelling biochemical and hematophysiological portrait of individuals chronically exposed to diesel generator emissions—an exposure pattern increasingly prevalent in post-conflict, infrastructure-deficient urban regions such as Mosul. Notably, the observed decrements in $PaO_2$, $SaO_2$, and $O_2Hb\%$ in the exposed group ($p<0.001$) underscore a disruption in alveolar-capillary gas exchange, plausibly mediated by the sustained inhalation of carbon monoxide (CO) and nitrogen oxides ($NO_x$), both of which competitively bind to hemoglobin and impair oxygen transport efficiency [11, 12].

Thus, the concomitant rise in COHb and HHb levels in exposed individuals is not merely statistically significant but mechanistically indicative of systemic hypoxemia, triggering compensatory erythropoietic activation, as evidenced by elevated hemoglobin, RBC count, and PCV. These hematological shifts likely reflect upregulation of erythropoietin, a hypoxia-sensitive hormone, in a physiological attempt to restore tissue oxygenation [13, 14].

This adaptation, while compensatory in the short term, may predispose individuals to hyperviscosity syndromes and cardiovascular strain under prolonged exposure scenarios.

The notable increase in malondialdehyde (MDA), a product of lipid peroxidation, along with the decrease in reduced glutathione (GSH), indicates a significant imbalance in redox levels within the system. This characteristic is representative of damage caused by oxidative stress, where reactive oxygen species (ROS) surpass the body's natural antioxidant defenses, leading to impairment of cell membranes and mitochondrial activity, as highlighted by Napolitano et al. [15], Al-Abdaly [16], and Zayani et al. [17].

The relationship observed between GSH and MDA levels in the research (as shown in Figure 4) supports the idea that glutathione reduction may indicate and contribute to lipid damage. Furthermore, the significant increase in carbonic anhydrase (CA), a metalloenzyme essential for the transport of $CO_2$ and the

regulation of pH levels in the body, could be a response to maintaining balance in cases of lung acidosis or reduced alveolar ventilation. Studies have suggested that an elevated level of CA is linked to conditions such as obstructive pulmonary disease and pulmonary hypertension induced by hypoxia [18, 19], which might have similarities in how they affect the body with exposure to smoke from generators.

The notable rise in C-reactive protein (CRP) levels among individuals exposed to generator emissions (8.29 ± 2.16 mg/L) clearly indicates an active and ongoing inflammatory response. This elevation aligns with the well-documented role of CRP as a sensitive marker for systemic inflammation, often seen in cases of chronic exposure to environmental toxins. The increase suggests that the liver's acute-phase response has been robustly activated, most likely driven by the release of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha. This systemic reaction not only confirms the presence of oxidative stress but also highlights a state of chronic immune activation that may predispose exposed individuals to further vascular and metabolic complications [20, 21].

The detected metal composition in the group exposed indicates a biochemical hazard. High blood levels of lead, copper, and nickel—all confirmed to trigger the formation of oxygen species and interfere with enzymatic redox cycles—suggest a continual accumulation of toxic substances, with the potential to harm multiple organs [22].

The noticeable reduction in zinc levels is concerning, as zinc supports superoxide dismutase and metallothioneins, which are essential for managing oxidative stress in the body's natural state. A lack of zinc could worsen vulnerability and hinder DNA repair over time, increasing the risk of genetic damage in the long run [23].

From a public health perspective, these results align with the concept of environmental fairness. This suggests that urban communities facing challenges are more likely to be exposed to environmental risks without adequate ways to reduce them. The combined biological disruptions mentioned here—including acidosis, oxidative stress, compensation by red blood cells, and accumulation of heavy metals—may lead to an increased vulnerability among these people toward long-term heart and lung diseases, degenerative neurological disorders, and cancer.

In summary, the information presented here offers a basis for understanding the impact of diesel generator emissions on environmental health in a discreet yet significant manner. It is crucial to take action at the policy level to reduce generator use, improve urban electricity provision, and establish community-based screening initiatives to detect early health issues caused by pollutants.

The discoveries have two implications—one related to health and the other to the environment—that go beyond just Mosul's specific situation. On the health front, the combination of low oxygen levels in the blood, imbalanced oxidative processes, and the accumulation of harmful metals can serve as an early indication needing regular medical checkups in people relying on generators for power. It's crucial to check blood oxygen levels, markers of oxidative stress such as MDA and GSH, and trace metal levels to intervene promptly before irreversible harm sets in [24].

The research underscores the importance of shifting away from diesel-powered energy sources in densely populated urban areas due to environmental concerns. To tackle this issue effectively and prevent long-term exposure to emissions in these areas, it is essential to implement policies such as controlled zoning for generator locations, raising public awareness of exhaust fume exposure, and providing incentives to switch to renewable energy sources. Neglecting these foundational

issues could lead to environmental inequality and worsen health challenges for vulnerable populations [25–27].

## Conclusion

This research uncovers physical and chemical changes associated with ongoing exposure to diesel generator emissions. Decreased oxygen levels in the body and increased oxidative stress markers, such as MDA levels and lower GSH levels, indicate potential health issues related to the exposure. Moreover, elevated blood hemoglobin levels and red blood cell count suggest a response by the body, while the accumulation of heavy metals such as lead, cadmium, and nickel, along with reduced zinc levels, hints at a broader health impact on individuals living in urban areas reliant on generators for power. The findings indicate the need for continuous health monitoring of residents living near generators, as well as for implementing policies and regulations to limit their use.

## Limitations

Although the results of this study are both statistically and biologically convincing, its sectional design limits the ability to establish causal relationships definitively. The use of proximity-based exposure classification without measuring airborne pollutant levels like CO, NOx and PM2.5 could lead to misclassification bias in exposure assessment. Additionally, the study was conducted within an urban area, geographically potentially restricting how broadly the findings can be applied to other regions, with similar post-conflict or low-resource settings.

The absence of follow-up information hinders our ability to grasp the ongoing progression of pollutant-triggered biochemical alterations over time. It is essential for upcoming studies to incorporate long-term research plans, exposure monitoring devices, and diverse functional tests across multiple organs, such as cognitive function and liver profiling, to confirm and build upon these preliminary findings.

## Conflict of Interest

The authors declare that there is no conflict of interest.

## Acknowledgements

The authors thank the staff of Al-Jumhuri and Ibn Sina Hospitals, and the Central Laboratory at the University of Mosul, for their technical support. Deep appreciation is extended to the participants, whose cooperation amid environmental hardship made this research possible.

## References

[1]. Mathew, N., Tirpude, A., Pillai, A. M., Mondal, P., & Arfin, T., 2023, Emerging contaminants in air pollution and their sources, consequences, and future challenges. *Bioremediation Technologies: For Wastewater and Sustainable Circular Bioeconomy*, pp. 235–274. https://doi.org/10.1515/9783110773848-009

[2]. Odubo, T. C., & Kosoe, E. A., 2024, Sources of air pollutants: impacts and solutions. *Air Pollutants in the Context of One Health: Fundamentals, Sources, and Impacts*, pp. 75–121. https://doi.org/10.1007/978-3-031-36213-2_4

[3]. Priyadarshanee, M., Mahto, U., & Das, S., 2022, Mechanism of toxicity and adverse health effects of environmental pollutants. *Microbial Biodegradation and Bioremediation*, pp. 33–53. https://doi.org/10.1016/B978-0-323-85347-6.00003-8

[4]. Sierra-Vargas, M. P., Montero-Vargas, J. M., Debray-García, Y., Vizuet-de-Rueda, J. C., Loaeza-Román, A., & Terán, L. M., 2023, Oxidative stress and air pollution: its impact on chronic respiratory diseases. *International Journal of Molecular Sciences*, 24(1), 853. https://doi.org/10.3390/ijms24010853

[5]. Al-Mallah, K. H., Al-Iraqi, O. M., & Saeed, M. G., 2022, Detection of urinary tract lesions of buffalos at Mosul city. *Indian Veterinary Journal*, 99(08), 43–49.

[6]. Wang, T., & Zhang, Y., 2024, Mechanisms and therapeutic targets of carbon monoxide poisoning: a focus on reactive oxygen species. *Chemico-Biological Interactions*, 111223. https://doi.org/10.1016/j.cbi.2024.111223

[7]. Otterbein, G. E., Tift, M. S., & Lee, G. R., 2022, Carbon Monoxide, Oxygen, and Pseudohypoxia. *In: Carbon Monoxide in Drug Discovery: Basics, Pharmacology, and Therapeutic Potential*, pp. 118–135.

[8]. Udeigwe, E. J., 2021, Biological Activities of Nonenzymatically Oxidized Lipids in RPE and Microglial Cells: HOHA Lactone and Pseudo Leukotriene C. *Case Western Reserve University*.

[9]. Mahmoud, O., Mammdoh, J., & Saeed, M. G., 2022, Histopathological and Scores Assessment of Using Omega-3 for Improvement Gingival Wound Healing Process in Rabbit Model. *Egyptian Journal of Veterinary Sciences*, 53(4), 517–527.

[10]. Pena, A. C., & Pamplona, A., 2022, Heme oxygenase-1, carbon monoxide, and malaria–the interplay of chemistry and biology. *Coordination Chemistry Reviews*, 453, 214285. https://doi.org/10.1016/j.ccr.2021.214285

[11]. Bauer, N., Mao, Q., Vashistha, A., Seshadri, A., Du, N. Y. C., Otterbein, L., Tan, C., de Caestecker, M. P., & Wang, B., 2025, A Critical Update on the Therapeutic Potential of Carbon Monoxide. *Medicinal Research Reviews*, 45(4), 1275–1301.

[12]. Devalla, L., Ghewade, B., Jadhav, U., Annareddy, S., & others, 2024, A comprehensive review on carbon monoxide diffusion capacity in COPD patients. *Cureus*, 16(2).

[13]. Yook, J. S., You, M., Kim, J., Toney, A. M., et al., 2021, Systemic iron mobilization and redistribution for adaptive thermogenesis through HIF2-α/hepcidin axis. *Proceedings of the National Academy of Sciences*, 118(40), e2109186118.

[14]. Locatelli, F., Minutolo, R., De Nicola, L., & Del Vecchio, L., 2022, Strategies in the treatment of anaemia in chronic kidney disease: the HIF-prolyl hydroxylase inhibitors. *Drugs*, 82(16), 1565–1589.

[15]. Napolitano, G., Fasciolo, G., & Venditti, P., 2021, Mitochondrial management of reactive oxygen species. *Antioxidants*, 10(11), 1824.

[16]. Al-Abdaly, Y. Z., 2023, Neurobehavioral evaluation of quail treated with omega-3 and chlorpyrifos. *Iraqi Journal of Veterinary Sciences*, 37(5 Suppl I–IV).

[17]. Zayani, Z., Matinahmadi, A., Tavakolpournegari, A., & Bidooki, S. H., 2025, Exploring Stressors: Impact on Cellular Organelles and Implications for Cellular Functions. *Stresses*, 5(2), 26.

[18]. Swenson, E. R., Kumar, A., Kumar, N., & Alvarez, B. V., 2021, Targeting Carbonic Anhydrases in Cardiovascular and Pulmonary Disease. *In: The Carbonic Anhydrases: Current and Emerging Therapeutic Targets*, pp. 37–77.

[19]. Stevens, R. P., 2024, Carbonic Anhydrase IX Promotes Acute Lung Injury and Mortality During Metabolic Acidosis and Pneumonia. *Doctoral dissertation, University of South Alabama*.

[20]. Teissier, T., Boulanger, E., & Cox, L. S., 2022, Interconnections between inflammageing and immunosenescence during ageing. *Cells*, 11(3), 359.

[21]. Camps, J., Iftimie, S., Jiménez-Franco, A., Castro, A., & Joven, J., 2025, Metabolic Reprogramming in Respiratory Viral Infections: A Focus on SARS-CoV-2, Influenza, and RSV. *(Journal name not provided—please specify if known)*.

[22]. Parui, R., Nongthombam, G. S., Hossain, M., Adil, L. R., et al., 2024, Impact of heavy metals on human health. *In: Remediation of Heavy Metals: Sustainable Technologies and Recent Advances*, pp. 47–81.

[23]. Liu, N., Du, J., Ge, J., & Liu, S. B., 2024, DNA damage-inducing endogenous and exogenous factors and research progress. *Nucleosides, Nucleotides and Nucleic Acids*, pp. 1–33.

[24]. Munteanu, C., Galaction, A. I., Onose, G., Turnea, M., & Rotariu, M., 2025, The Janus Face of Oxidative Stress and Hydrogen Sulfide. *Antioxidants*, 14(3), 360.

[25]. Egede, L. E., Walker, R. J., & Williams, J. S., 2024, Addressing structural inequalities and social determinants of health. *Journal of General Internal Medicine*, 39(3), 487–491.

[26]. Alfathi, M., Alabdaly, Y., & Al-Hayyali, F., 2023, Spirulina against gentamicin toxicity in rat liver and kidney. *Egyptian Journal of Histology*, 46(4), 1666–1675.

[27]. Alabdaly, Y. Z., 2021, Effect of diclofenac on the pharmacokinetics of ciprofloxacin in quail. *Iraqi Journal of Veterinary Sciences*, 35(4), 777–781.