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Cardiovascular Injury and Hematological Dysregulation in Chickens Exposed to Petroleum Hydrocarbon-Contaminated Environments

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Abstract **Original Research Article**

Background: *Petroleum hydrocarbon contamination of the environment is associated with systemic toxicity in exposed organisms, yet its combined effects on cardiovascular integrity and hematological function remain underexplored in avian species.*

Objective: *This study evaluated cardiovascular injury and hematological dysregulation in chickens chronically exposed to a petroleum hydrocarbon-contaminated environment.*

Methods: *A comparative experimental design was employed involving chickens exposed for 6 and 12 months and unexposed controls. Serum cardiac biomarkers (cardiac troponin I, creatine kinase-MB, brain natriuretic peptide, and atrial natriuretic peptide) and hematological parameters (erythrocyte sedimentation rate, packed cell volume, hemoglobin concentration, white blood cell count, platelet count, and differential leukocyte profiles) were assessed using standard laboratory methods. Data were analyzed using descriptive and inferential statistics at $p < 0.05$.*

Results: *Exposed chickens exhibited significant elevations in all cardiac injury and stress biomarkers, indicating myocardial injury and cardiac strain. Hematological analysis revealed anemia, leukocytosis, increased inflammatory indices, and reduced platelet counts in exposed birds. These alterations were more pronounced with prolonged exposure and demonstrated sex-dependent patterns.*

Conclusion: *Chronic petroleum hydrocarbon exposure induces significant cardiovascular injury and hematological dysregulation in chickens. The findings highlight the systemic health risks associated with environmental hydrocarbon contamination and emphasize the need for integrated cardiovascular and hematological monitoring in exposed animal populations.*

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INTRODUCTION

Petroleum hydrocarbon contamination of the environment constitutes a major ecological and public health concern, particularly in regions with sustained crude oil exploration, refining, and transportation activities. Chronic exposure to petroleum hydrocarbons occurs through contaminated soil, water, and food sources, resulting in prolonged systemic exposure in terrestrial organisms. While hepatic and renal toxicities associated with petroleum hydrocarbon exposure are well documented, increasing evidence suggests that the cardiovascular and hematopoietic systems are also critical and vulnerable targets of hydrocarbon-induced toxicity (Alao *et al*., 2025; Lahiri *et al*., 2022).

The cardiovascular system is especially susceptible to toxic injury due to its continuous exposure to circulating xenobiotics and their metabolites. Petroleum hydrocarbons and associated contaminants have been shown to induce myocardial injury through mechanisms involving oxidative stress, inflammation, endothelial dysfunction, and disruption of cellular energy metabolism. These pathological processes may culminate in cardiomyocyte damage, altered cardiac contractility, and ventricular stress. Cardiac biomarkers such as cardiac troponins, creatine kinase–MB, brain natriuretic peptide, and atrial natriuretic peptide are widely used indicators of myocardial injury, cardiac stress, and ventricular

dysfunction, and elevations in these markers have been reported following exposure to environmental pollutants, often reflecting subclinical or overt cardiovascular injury (Han *et al*., 2024; Zhao *et al*., 2025; Guo *et al*., 2022).

In parallel, the hematopoietic system plays a central role in oxygen transport, immune defense, and hemostasis, and is highly vulnerable to environmental toxicants. Chronic exposure to petroleum hydrocarbons has been associated with alterations in hematological parameters, including anemia, leukocyte dysregulation, thrombocytopenia or thrombocytosis, and disturbances in differential leukocyte counts. These changes may arise from direct bone marrow toxicity, oxidative damage to circulating blood cells, inflammatory activation, or impaired erythropoiesis. Hematological abnormalities not only reflect systemic toxicity but may also exacerbate cardiovascular stress by impairing oxygen delivery, immune regulation, and vascular integrity (Ezejiofor, 2016; Joshi et al., 2019).

Importantly, cardiovascular injury and hematological dysregulation are often biologically interconnected. Myocardial injury may provoke inflammatory and hematological responses, while altered blood cell indices can contribute to endothelial dysfunction, impaired tissue oxygenation, increased blood viscosity, and heightened cardiovascular risk. In environmental exposure settings, these interactions may amplify systemic toxicity and predispose exposed organisms to reduced physiological resilience, impaired performance, and increased mortality (McHale *et al*., 2018; Młynarska *et al*., 2025). Evaluating cardiovascular or hematological effects in isolation may therefore underestimate the true burden of petroleum hydrocarbon–induced toxicity.

Avian species, particularly chickens, are well suited for investigating cardiovascular and hematological effects of environmental contaminants. Their close interaction with contaminated terrestrial environments through feeding and foraging, combined with their physiological sensitivity and relevance to human food chains, makes them valuable sentinel species for environmental health assessment. Despite this, data on the combined cardiovascular and hematological effects of chronic petroleum hydrocarbon exposure in chickens remain limited, especially under prolonged exposure conditions and with consideration of sex-related differences.

Therefore, this study aimed to evaluate cardiovascular injury and hematological dysregulation in chickens chronically exposed to petroleum hydrocarbon–contaminated environments by assessing cardiac injury and stress biomarkers alongside comprehensive hematological parameters. By integrating cardiovascular and hematological indices and examining the influence of exposure duration and sex, this study provides a more holistic understanding of systemic toxicity associated with chronic petroleum hydrocarbon exposure and contributes to environmental risk assessment in hydrocarbon-impacted regions.

MATERIALS AND METHODS

This study employed an experimental comparative design to investigate cardiovascular injury and hematological dysregulation associated with chronic exposure to a petroleum hydrocarbon-contaminated environment in chickens. Exposed chickens were compared with unexposed control chickens to evaluate alterations in cardiac injury and stress biomarkers alongside hematological parameters. Analyses were stratified by duration of exposure (6 months and 12 months) and sex to assess temporal and sex-related variations in cardiovascular and hematological responses. Chickens in the exposed group were sourced from an environment chronically contaminated with petroleum hydrocarbons as a result of sustained hydrocarbon-related activities, while control chickens were obtained from a comparable environment without known petroleum hydrocarbon contamination. All birds were maintained under similar husbandry conditions, including access to feed and water, to minimize confounding influences unrelated to environmental exposure.

A total of eighteen chickens were included in the study, comprising twelve exposed birds and six controls. The exposed group consisted of chickens exposed for 6 months (male, n = 3; female, n = 3) and 12 months (male, n = 3; female, n = 3), while the control group included chickens maintained for 6 months (male, n = 2; female, n = 2) and 12 months (male, n = 1; female, n = 1). This grouping strategy enabled evaluation of exposure-related, duration-dependent, and sex-dependent cardiovascular and hematological effects.

Blood samples were collected aseptically from each chicken via venipuncture using sterile techniques. Samples intended for hematological analysis were collected into ethylenediaminetetraacetic acid tubes, while samples for cardiac biomarker analysis were collected into plain tubes, allowed to clot, and centrifuged to obtain serum. Serum samples were stored under appropriate conditions and analyzed within recommended time frames to preserve biochemical integrity.

Cardiovascular injury and cardiac stress were assessed by measuring serum concentrations of cardiac troponin I, creatine kinase–MB, brain natriuretic peptide, and atrial natriuretic peptide. These cardiac biomarkers were quantified using enzyme-linked immunosorbent assay techniques in accordance with manufacturer instructions. Absorbance readings were obtained using a microplate reader (Model M201, EMPSUN, Chengdu Empsun Medical Technology Co. Ltd., China), and concentrations were expressed in appropriate units.

Hematological assessment included determination of erythrocyte sedimentation rate, packed cell volume, hemoglobin concentration, total white blood cell count, platelet count, and differential leukocyte counts, including neutrophils, lymphocytes, monocytes, eosinophils, and basophils. Hematological parameters were analyzed using

standard manual hematological techniques routinely employed in clinical and veterinary laboratories.

All analyses were performed in duplicate to ensure analytical reliability. Calibration standards and quality control samples were included in each analytical batch, and all laboratory procedures were conducted in accordance with established standard operating protocols to minimize analytical variability.

Data were entered and analyzed using appropriate statistical software. Results were expressed as mean ± standard deviation. Comparisons between exposed and control groups were performed using independent-sample *t*-tests where applicable, while one-way analysis of variance was used to evaluate differences based on duration of exposure and sex, followed by appropriate post-hoc tests. Statistical significance was set at $p < 0.05$, and analyses were restricted to cardiovascular and hematological parameters to maintain methodological independence from other system-specific investigations derived from the same experimental cohort.

All experimental procedures involving animals were conducted in accordance with internationally accepted guidelines for the care and use of experimental animals, and efforts were made to minimize stress and discomfort throughout the study.

RESULTS AND DISCUSSION

Chickens chronically exposed to a petroleum hydrocarbon-contaminated environment exhibited clear biochemical evidence of cardiovascular injury when compared with unexposed controls. Serum levels of cardiac troponin I and creatine kinase-MB were consistently elevated among exposed chickens, with more pronounced increases observed after 12 months of exposure. Elevation of these biomarkers reflects myocardial cell injury and membrane disruption, indicating that chronic hydrocarbon exposure compromises cardiac structural integrity. Similar elevations in cardiac injury markers following exposure to environmental pollutants have been reported and are commonly attributed to oxidative stress-mediated myocardial damage and mitochondrial dysfunction (Hicks, 2015; Liu *et al*., 2023).

In addition to markers of myocardial injury, exposed chickens demonstrated altered levels of brain natriuretic peptide and atrial natriuretic peptide, suggesting increased cardiac wall stress and functional adaptation to injury. Elevated natriuretic peptides are indicative of hemodynamic overload and myocardial strain and have been widely used as biomarkers of cardiac dysfunction in both clinical and experimental settings (Yu *et al*., 2021; Sandefur & Jialal, 2023). The observed increases in BNP and ANP among exposed chickens suggest that petroleum hydrocarbon exposure may induce subclinical cardiac stress that progresses with prolonged exposure duration.

Duration of exposure played a significant role in the severity of cardiovascular alterations. Chickens exposed for 12 months showed greater elevations in cardiac biomarkers compared with those exposed for 6 months, indicating cumulative myocardial injury with prolonged exposure. Chronic petroleum hydrocarbon exposure is known to promote persistent oxidative stress and inflammatory signaling, which can progressively impair cardiac tissue and exacerbate functional decline over time (Albakri, 2019; Akbarian *et al*., 2016). The duration-dependent pattern observed in this study underscores the importance of long-term exposure assessment when evaluating cardiovascular toxicity in contaminated environments.

Hematological analysis revealed marked dysregulation of blood indices in petroleum hydrocarbon-exposed chickens. Exposed birds exhibited reduced packed cell volume and hemoglobin concentrations, particularly following prolonged exposure, suggesting the development of anemia. Such reductions may result from impaired erythropoiesis, increased destruction of red blood cells, or direct toxic effects of hydrocarbons on bone marrow function. Previous studies have documented anemia and reduced erythrocyte indices following exposure to petroleum hydrocarbons, linking these effects to oxidative damage and disruption of hematopoietic processes (Yasin & Salih, 2025; Shree & Tyagi, 2022).

Total white blood cell counts and differential leukocyte profiles were also altered in exposed chickens. Leukocytosis with shifts in neutrophil and lymphocyte proportions was evident, reflecting activation of inflammatory and immune responses. Chronic exposure to environmental hydrocarbons has been shown to stimulate systemic inflammation and immune dysregulation, resulting in altered leukocyte distribution and function (Mank et al., 2024; Chen *et al*., 2023). Such hematological changes may contribute to increased susceptibility to infection and impaired immune competence in exposed animals.

Platelet counts were variably altered among exposed chickens, with some individuals exhibiting thrombocytosis, particularly after prolonged exposure. Platelet dysregulation may reflect inflammatory activation or endothelial injury associated with chronic toxic exposure. Altered platelet indices have been linked to increased cardiovascular risk and may further exacerbate myocardial stress by promoting microvascular dysfunction (Nwaogu & Onyeze, 2014; Asegbeloyin et al., 2015; Oleforuh-Okoleh *et al*., 2024).

Sex-related differences were evident in both cardiovascular and hematological responses. Male chickens tended to exhibit more pronounced elevations in cardiac injury markers, while female chickens demonstrated greater alterations in hematological parameters, particularly erythrocyte indices and leukocyte differentials. Sex-dependent susceptibility to environmental toxicants has been attributed to differences in hormonal regulation, antioxidant capacity, and xenobiotic metabolism, which may modulate cardiovascular and hematopoietic responses to chronic exposure (Panigrahy *et al*., 2017; Sokolenko *et al*., 2024).

The concurrent presence of cardiovascular injury and hematological dysregulation observed in this study highlights the integrated nature of systemic toxicity induced by petroleum hydrocarbons. Hematological abnormalities may exacerbate cardiovascular injury by impairing oxygen delivery and promoting inflammatory stress, while myocardial injury may further stimulate hematological and immune responses. This bidirectional interaction underscores the importance of evaluating cardiovascular and hematological parameters collectively when assessing environmental toxicity (Jabbar & Ali, 2020).

Overall, the findings of this study provide compelling evidence that chronic exposure to petroleum hydrocarbon-contaminated environments induces significant cardiovascular injury and hematological dysregulation in chickens. These alterations were influenced by both duration of exposure and sex, emphasizing cumulative toxicity and differential vulnerability. Given the ecological relevance of chickens and their close association with human food systems, the observed cardiovascular and hematological disturbances raise important concerns regarding animal health, productivity, and potential implications for environmental and public health in hydrocarbon-impacted regions.

Table 1. Cardiovascular Biomarkers of Chickens Exposed to Petroleum Hydrocarbon–Contaminated Environments

Parameter	**Exposed Chickens (n = 12) Mean ± SD**	**Control Chickens (n = 6) Mean ± SD**	**Statistical Outcome**
Cardiac Troponin I (ng/mL)	5.64 ± 2.55	0.48 ± 0.60	↑ Significant ($p < 0.05$)
CK-MB (ng/mL)	49.58 ± 20.77	21.00 ± 9.70	↑ Significant ($p < 0.05$)
BNP (pg/mL)	59.75 ± 13.87	21.83 ± 5.64	↑ Significant ($p < 0.05$)
ANP (pg/mL)	48.17 ± 16.42	10.67 ± 2.73	↑ Significant ($p < 0.05$)

↑ *Increase relative to control.*

Table 2. Hematological Parameters of Chickens Exposed to Petroleum Hydrocarbon–Contaminated Environments

Parameter	**Exposed Chickens (n = 12) Mean ± SD**	**Control Chickens (n = 6) Mean ± SD**	**Statistical Outcome**
ESR (mm/h)	14.50 ± 7.22	1.17 ± 1.17	↑ Significant ($p < 0.05$)
PCV (%)	20.33 ± 8.32	54.17 ± 8.04	↓ Significant ($p < 0.05$)
Hemoglobin (g/dL)	6.68 ± 2.76	18.05 ± 2.71	↓ Significant ($p < 0.05$)
WBC ($\times 10^9$/L)	21.48 ± 7.07	9.62 ± 3.83	↑ Significant ($p < 0.05$)
Platelets ($\times 10^9$/L)	18.83 ± 8.67	53.33 ± 14.25	↓ Significant ($p < 0.05$)

↑ *Increase;* ↓ *Decrease relative to control.*

Statistical Interpretation: Independent-sample *t*-test analysis revealed significant elevations in all measured cardiovascular biomarkers among petroleum hydrocarbon–exposed chickens compared with controls ($p < 0.05$), indicating myocardial injury and cardiac stress. Hematological analysis demonstrated significantly increased erythrocyte sedimentation rate and white blood cell count alongside marked reductions in packed cell volume, hemoglobin concentration, and platelet count in exposed chickens ($p < 0.05$). These findings indicate anemia, inflammatory activation, and hematopoietic dysregulation associated with chronic petroleum hydrocarbon exposure.

CONCLUSION

This study demonstrates that chronic exposure to a petroleum hydrocarbon-contaminated environment induces significant cardiovascular injury and hematological dysregulation in chickens. Elevated levels of cardiac troponin I, creatine kinase-MB, brain natriuretic peptide, and atrial natriuretic peptide among exposed birds provide strong evidence of myocardial injury and cardiac stress. These alterations suggest compromised cardiac integrity and adaptive responses to sustained toxic insult.

Concomitantly, marked hematological abnormalities were observed, including reduced packed cell volume, hemoglobin concentration, and platelet counts, alongside elevated erythrocyte sedimentation rate and white blood cell counts. These findings indicate the development of anemia, inflammatory activation, and hematopoietic disruption in exposed chickens. The severity of cardiovascular and hematological alterations increased with prolonged exposure, highlighting the cumulative nature of petroleum hydrocarbon toxicity.

Overall, the concurrent manifestation of cardiovascular injury and hematological dysregulation underscores the systemic toxicity of petroleum hydrocarbons and suggests that impairment of blood composition may further exacerbate cardiac stress. These findings have important implications for animal health, productivity, and ecological stability in hydrocarbon-impacted environments, and they reinforce the need for integrated cardiovascular and hematological assessment in environmental toxicology studies.

LIMITATIONS AND FUTURE DIRECTIONS

Although this study provides compelling evidence of cardiovascular and hematological toxicity associated with petroleum hydrocarbon exposure, certain limitations should be acknowledged. The relatively small sample size may limit statistical power and generalizability; however, the consistency and magnitude of the observed alterations across multiple biomarkers support the biological relevance of the findings. Additionally, environmental exposure conditions did not permit precise quantification of individual hydrocarbon doses or identification of specific petroleum constituents responsible for the observed effects.

The study relied on serum biomarkers and hematological indices without histopathological examination of cardiac tissue or bone marrow, limiting direct assessment of structural damage and mechanistic pathways. Furthermore, potential interactions between cardiovascular injury, hematological changes, oxidative stress, and inflammatory mediators were not explored within the scope of this analysis.

Future studies should incorporate larger cohorts, controlled exposure models, and histopathological evaluation of cardiac and hematopoietic tissues to strengthen causal inference. Integration of oxidative stress and inflammatory biomarkers may further elucidate mechanistic links between myocardial injury and hematological dysregulation. Longitudinal studies examining recovery potential and long-term cardiovascular outcomes would also enhance understanding of the chronic health consequences of petroleum hydrocarbon exposure.

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