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Oxidative Stress, Inflammatory Responses, and Heavy Metal Burden in Chickens from Petroleum Hydrocarbon-Contaminated Environments

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ABSTRACT

Petroleum hydrocarbon contamination is frequently accompanied by heavy metal pollution, creating complex toxic environments that promote oxidative stress and inflammation in exposed organisms. This study evaluated oxidative stress status, inflammatory responses, and heavy metal burden in chickens chronically exposed to petroleum hydrocarbon-contaminated environments. Chickens exposed for 6 and 12 months were compared with unexposed controls. Antioxidant enzymes (superoxide dismutase, catalase, glutathione peroxidase), lipid peroxidation marker (malondialdehyde), inflammatory biomarkers (interleukin-6, tumor necrosis factor-alpha, C-reactive protein, nitric oxide), and heavy metals (chromium, lead, zinc) were assessed using standard laboratory techniques. Data were analyzed using descriptive and inferential statistics at $p < 0.05$. Exposed chickens showed significant depletion of antioxidant defenses and elevated malondialdehyde levels, indicating increased oxidative stress. Inflammatory biomarkers, including interleukin-6, tumor necrosis factor-alpha, and nitric oxide, were significantly elevated. Heavy metal analysis revealed marked accumulation of chromium, lead, and zinc in exposed birds. These alterations were more pronounced with prolonged exposure. Chronic petroleum hydrocarbon exposure induces integrated oxidative stress, inflammatory activation, and heavy metal accumulation in chickens, highlighting synergistic toxic mechanisms and raising concerns regarding environmental health and food safety in hydrocarbon-polluted regions.

1. Introduction

Petroleum hydrocarbon contamination of the environment remains a persistent ecological and public health challenge in regions with extensive oil exploration, refining, and transportation activities. Chronic release of hydrocarbons into soil and water is frequently accompanied by the accumulation of heavy metals such as chromium, lead, and zinc, resulting in complex chemical mixtures with enhanced toxic potential. Prolonged exposure to these contaminants poses serious risks to exposed organisms, primarily through mechanisms involving oxidative stress and inflammatory activation, which are now recognized as central pathways of petroleum hydrocarbon–induced toxicity (1;2).

Oxidative stress occurs when the generation of reactive oxygen species exceeds the capacity of endogenous antioxidant defense systems. Petroleum hydrocarbons and associated heavy metals promote excessive reactive oxygen species production through redox cycling, mitochondrial dysfunction, and inhibition of antioxidant enzymes. Disruption of key antioxidant defenses, including superoxide dismutase, catalase, and glutathione peroxidase, compromises cellular redox balance and leads to lipid peroxidation, protein oxidation, and membrane damage, commonly reflected by elevated malondialdehyde concentrations. Oxidative stress therefore represents a critical mechanistic link between environmental contamination and tissue injury (3;4).

Closely linked to oxidative stress is the activation of inflammatory pathways. Reactive oxygen species stimulate the release of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha, as well as acute-phase proteins including C-reactive protein. Nitric oxide, although essential for physiological signaling, may contribute to nitrosative stress when produced excessively during chronic inflammation. Sustained inflammatory activation not only exacerbates tissue injury but also amplifies oxidative damage, creating a self-reinforcing cycle of toxicity that accelerates systemic dysfunction (5;6).

Heavy metals commonly associated with petroleum hydrocarbon pollution further intensify oxidative and inflammatory responses. Metals such as lead and chromium are capable of depleting antioxidant reserves, inhibiting antioxidant enzymes, and directly inducing oxidative damage. In addition, heavy metals modulate immune responses by promoting cytokine release and persistent low-grade inflammation. The co-occurrence of petroleum hydrocarbons and heavy metals therefore represents a heightened toxicological burden, producing synergistic effects on oxidative stress and inflammatory pathways that exceed those observed with hydrocarbon exposure alone (7;8).

Avian species, particularly chickens, are highly relevant for investigating oxidative stress, inflammation, and heavy metal accumulation associated with environmental contamination. Their close contact with contaminated soil, water, and feed, coupled

with their physiological sensitivity and importance within the human food chain, makes chickens valuable sentinel species for environmental monitoring. However, integrated evaluations simultaneously examining oxidative stress markers, inflammatory mediators, and heavy metal burden in chickens chronically exposed to petroleum hydrocarbon-contaminated environments remain limited, especially with respect to exposure duration and sex-related differences.

Against this background, the present study was designed to evaluate oxidative stress status, inflammatory responses, and heavy metal burden in chickens chronically exposed to a petroleum hydrocarbon-contaminated environment. Specifically, the study assessed alterations in antioxidant defense enzymes, including superoxide dismutase, catalase, and glutathione peroxidase, alongside lipid peroxidation as indicated by malondialdehyde levels. Inflammatory responses were evaluated through measurement of interleukin-6, tumor necrosis factor-alpha, C-reactive protein, and nitric oxide, while the accumulation of chromium, lead, and zinc was quantified to characterize metal burden associated with petroleum contamination. The study further examined whether these biochemical alterations varied according to duration of exposure (6 months versus 12 months) and sex.

It was hypothesized that chronic exposure to petroleum hydrocarbon-contaminated environments would result in significant depletion of antioxidant defenses, increased lipid peroxidation, elevated inflammatory biomarker levels, and accumulation of heavy metals in exposed chickens when compared with unexposed controls. It was further anticipated that the magnitude of oxidative stress, inflammatory activation, and heavy metal burden would be influenced by both duration of exposure and sex. Through this integrated approach, the study sought to establish the presence of combined oxidative, inflammatory, and metal-mediated toxicity as a central mechanism underlying petroleum hydrocarbon-induced biological effects in chickens.

2. Materials and Methods

This study employed an experimental comparative design to evaluate oxidative stress status, inflammatory responses, and heavy metal burden in chickens chronically exposed to a petroleum hydrocarbon-contaminated environment. Biochemical and immunological parameters in exposed chickens were compared with those of unexposed control chickens. Analyses were stratified by duration of exposure (6 months and 12 months) and sex to assess temporal and sex-related variations in oxidative, inflammatory, and metal-associated toxicity. Chickens in the exposed group were obtained from an environment with sustained petroleum hydrocarbon contamination resulting from hydrocarbon-related activities, while control chickens were sourced from a comparable environment without known petroleum hydrocarbon pollution. All birds were maintained under similar husbandry conditions, including access to feed and water, to minimize confounding environmental and nutritional influences unrelated to contaminant 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 enabled evaluation of exposure-related, duration-dependent, and sex-dependent effects on oxidative stress markers, inflammatory biomarkers, and heavy metal accumulation.

Blood samples were collected aseptically from each chicken via venipuncture using sterile techniques and transferred into plain sample bottles. Samples were allowed to clot and subsequently centrifuged under standard laboratory conditions to obtain serum. Serum samples were stored at appropriate temperatures and analyzed within recommended time frames to preserve biochemical and immunological integrity.

Oxidative stress status was assessed by measuring serum activities of superoxide dismutase, catalase, and glutathione peroxidase, alongside lipid peroxidation as indicated by malondialdehyde concentration. These parameters were determined using standard colorimetric and enzymatic assay methods in accordance with manufacturer instructions. Absorbance readings were obtained using a visible spectrophotometer (Model S23A, HELMREASINN, China), and results were expressed in appropriate activity or concentration units.

Inflammatory responses were evaluated by quantifying serum concentrations of interleukin-6, tumor necrosis factor-alpha, and C-reactive protein using enzyme-linked immunosorbent assay techniques according to manufacturer protocols. Optical density measurements were obtained using a microplate reader (Model M201, EMPSUN, Chengdu Empsun Medical Technology Co. Ltd., China). Nitric oxide concentration was determined using a standard colorimetric method, with absorbance measured using the same visible spectrophotometer (Model S23A, HELMREASINN, China).

Heavy metal burden was assessed by determining serum concentrations of chromium, lead, and zinc using atomic absorption spectrophotometry following appropriate sample digestion procedures. Heavy metal analysis was performed using an Agilent 240 Atomic Absorption Spectrophotometer (Agilent Technologies, USA), operated in accordance with manufacturer specifications. Calibration was achieved using certified standard solutions for each metal, and concentrations were expressed in milligrams per liter or micrograms per liter as appropriate.

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 assess 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 oxidative stress markers, inflammatory biomarkers, and heavy metal 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 all efforts were made to minimize animal stress and discomfort throughout the study.

2.1 Ethical Considerations

All experimental procedures involving animals were conducted in accordance with the ARRIVE (Animal Research: Reporting of In Vivo Experiments) guidelines and internationally accepted standards for the care and use of laboratory animals. All efforts were made to minimize animal stress and discomfort throughout the study.

3. Results and Discussion

Chickens chronically exposed to a petroleum hydrocarbon-contaminated environment exhibited marked evidence of oxidative stress when compared with unexposed controls. Antioxidant defense enzymes, including superoxide dismutase, catalase, and glutathione peroxidase, were generally reduced in exposed chickens, particularly following prolonged exposure. The observed depletion of these enzymes indicates compromised antioxidant capacity and reduced ability to neutralize reactive oxygen species. Concurrently, malondialdehyde levels were elevated among exposed birds, reflecting increased lipid peroxidation and oxidative damage to cellular membranes. These findings are consistent with established evidence that petroleum hydrocarbons promote excessive generation of reactive oxygen species and impair endogenous antioxidant systems (9; 10).

Duration of exposure played a critical role in oxidative stress severity. Chickens exposed for 12 months demonstrated greater reductions in antioxidant enzyme activities and higher malondialdehyde concentrations compared with those exposed for 6 months, indicating cumulative oxidative damage with prolonged exposure. Chronic persistence of hydrocarbons and associated contaminants in biological tissues may overwhelm antioxidant defenses over time, leading to progressive cellular injury. Similar duration-dependent oxidative stress patterns have been reported in animals exposed to hydrocarbon-polluted environments (12;13).

In parallel with oxidative stress, petroleum hydrocarbon exposure elicited significant inflammatory responses. Serum concentrations of interleukin-6, tumor necrosis factor-alpha, and C-reactive protein were elevated in exposed chickens relative to controls, indicating activation of systemic inflammatory pathways. Increased nitric oxide levels were also observed, suggesting enhanced nitrosative stress and inflammatory signaling. These inflammatory responses are likely triggered by oxidative damage and tissue injury, as reactive oxygen species can activate transcription factors that promote cytokine production and acute-phase protein synthesis (14; 15).

The inflammatory response was more pronounced in chickens exposed for 12 months, further supporting the cumulative nature of petroleum hydrocarbon toxicity. Sustained elevation of pro-inflammatory cytokines and nitric oxide may exacerbate tissue damage, disrupt immune homeostasis, and contribute to the progression of chronic disease. Persistent low-grade inflammation is increasingly recognized as a key mediator linking environmental contamination to long-term health impairment (10;16).

Heavy metal analysis revealed significant accumulation of chromium, lead, and zinc in chickens exposed to petroleum hydrocarbon-contaminated environments. Exposed birds, particularly those with longer exposure duration, exhibited higher concentrations of these metals compared with controls, indicating bioaccumulation from contaminated soil, water, and feed sources. Heavy metals commonly co-occur with petroleum hydrocarbons and may intensify toxicity by directly generating reactive oxygen species and inhibiting antioxidant enzymes (18;19).

The coexistence of heavy metal accumulation with oxidative stress and inflammation suggests synergistic toxicity. Lead and chromium are known to disrupt redox balance and promote inflammatory cytokine release, thereby amplifying oxidative and inflammatory injury initiated by petroleum hydrocarbons. Zinc, although an essential trace element, may exert toxic effects at elevated concentrations by disturbing cellular metal homeostasis and antioxidant regulation. The combined presence of hydrocarbons and heavy metals therefore represents a heightened toxicological burden compared with exposure to hydrocarbons alone.

Sex-related differences were evident across oxidative stress, inflammatory, and heavy metal parameters. Female chickens tended to exhibit greater depletion of antioxidant enzymes and higher inflammatory marker levels following prolonged exposure, whereas males showed relatively higher accumulation of certain heavy metals. These differences may be attributed to sex-dependent variations in metal metabolism, hormonal modulation of antioxidant systems, and immune responsiveness, as reported in previous environmental toxicology studies (20).

Collectively, the findings of this study demonstrate that chronic exposure to petroleum hydrocarbon-contaminated environments induces integrated oxidative stress, inflammatory activation, and heavy metal burden in chickens. Oxidative damage, persistent inflammation, and metal accumulation appear to act synergistically to exacerbate systemic toxicity, potentially contributing to the dysfunction of multiple organ systems observed in hydrocarbon-exposed animals. Given the ecological relevance of chickens and their role in the human food chain, these results raise important concerns regarding environmental health, food safety, and long-term ecological consequences of petroleum hydrocarbon pollution.

Statistical Interpretation: Independent-sample *t*-test analysis demonstrated significant depletion of antioxidant defense enzymes (SOD, CAT, and GPx) and a concomitant elevation of malondialdehyde levels in petroleum hydrocarbon-exposed chickens compared with controls ($p < 0.05$), indicating pronounced oxidative stress. Inflammatory analysis revealed significantly increased serum levels of interleukin-6, tumor necrosis factor-alpha, and nitric oxide in exposed birds ($p < 0.05$), while C-reactive protein showed no statistically significant difference. Heavy metal assessment showed marked accumulation of chromium, lead, and zinc in exposed chickens relative to controls ($p < 0.05$), confirming bioaccumulation and metal-associated toxicity in hydrocarbon-contaminated environments.

4. Conclusion

This study demonstrates that chronic exposure to petroleum hydrocarbon-contaminated environments induces pronounced oxidative stress, inflammatory activation, and heavy metal accumulation in chickens. Significant depletion of antioxidant enzymes alongside elevated malondialdehyde levels indicates impaired redox homeostasis and increased lipid peroxidation in exposed birds. These oxidative disturbances were accompanied by marked increases in pro-inflammatory cytokines and nitric oxide, reflecting sustained immune activation and nitrosative stress. The accumulation of chromium, lead, and zinc further highlights the role of co-occurring heavy metals in intensifying hydrocarbon-associated toxicity.

The convergence of oxidative stress, inflammation, and heavy metal burden suggests a synergistic toxicological mechanism in which petroleum hydrocarbons and associated metals jointly disrupt cellular integrity and immune regulation. Prolonged exposure exacerbated these effects, emphasizing the cumulative nature of environmental contamination. Given the ecological relevance of chickens and their role within the food chain, these findings underscore the broader environmental and public health implications of petroleum hydrocarbon pollution and highlight oxidative and inflammatory biomarkers as sensitive indicators of environmental toxicity.

5. Limitations and Future Directions

Despite the strength of the observed biochemical and immunological alterations, this study has certain limitations. The relatively small sample size may limit generalizability, although the consistency of changes across multiple oxidative, inflammatory, and metal parameters supports the biological relevance of the findings. Environmental exposure conditions did not allow precise quantification of individual hydrocarbon fractions or metal speciation, which may influence toxicity profiles. Additionally, the absence of tissue-level histopathological assessment limited direct evaluation of organ-specific oxidative and inflammatory damage.

Future studies should incorporate larger cohorts, controlled exposure models, and tissue histopathology to strengthen causal inference. Investigation of molecular signaling pathways linking heavy metal accumulation, oxidative stress, and inflammatory activation would further elucidate underlying mechanisms. Longitudinal studies assessing reversibility of oxidative and inflammatory damage following remediation or removal from contaminated environments would also provide valuable insight into recovery potential and long-term health outcomes.

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Appendices

Table 1. Oxidative Stress Markers in Chickens Exposed to Petroleum Hydrocarbon–Contaminated Environments

Parameter	Exposed Chickens (n = 12) Mean ± SD	Control Chickens (n = 6) Mean ± SD	Statistical Outcome
SOD (U/mL)	101.8 ± 21.5	218.2 ± 48.5	↓ Significant ($p < 0.05$)
CAT (U/mL)	73.3 ± 55.8	180.3 ± 47.5	↓ Significant ($p < 0.05$)
GPx (U/mL)	102.9 ± 57.9	237.8 ± 41.8	↓ Significant ($p < 0.05$)
MDA (U/mL)	7.69 ± 4.13	3.02 ± 2.47	↑ Significant ($p < 0.05$)

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

Table 2. Inflammatory Biomarkers in Chickens Exposed to Petroleum Hydrocarbon–Contaminated Environments

Parameter	Exposed Chickens (n = 12) Mean ± SD	Control Chickens (n = 6) Mean ± SD	Statistical Outcome
IL-6 (pg/mL)	75.60 ± 31.92	36.90 ± 17.00	↑ Significant ($p < 0.05$)
TNF-α (pg/mL)	45.3 ± 14.4	33.7 ± 11.6	↑ Significant ($p < 0.05$)
CRP (mg/L)	3.83 ± 1.81	4.03 ± 1.91	NS
NO (µmol/L)	42.83 ± 11.33	34.17 ± 15.15	↑ Significant ($p < 0.05$)

NS = Not significant.

Table 3. Heavy Metal Concentrations in Chickens Exposed to Petroleum Hydrocarbon–Contaminated Environments

Parameter	Exposed Chickens (n = 12) Mean ± SD	Control Chickens (n = 6) Mean ± SD	Statistical Outcome
Chromium(mg/L)	3.78 ± 3.06	0.91 ± 1.55	↑ Significant ($p < 0.05$)
Lead (mg/L)	8.23 ± 4.40	3.10 ± 1.53	↑ Significant ($p < 0.05$)
Zinc (mg/L)	3.11 ± 1.92	1.20 ± 1.16	↑ Significant ($p < 0.05$)