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Original Article

Integrated Hepatic And Renal Toxicity Following Chronic Petroleum Hydrocarbon Exposure In Chickens

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Abstract - *Petroleum hydrocarbons are persistent environmental contaminants capable of inducing multi-organ toxicity following chronic exposure. This field-based comparative study evaluated integrated hepatic and renal biochemical alterations in domestic chickens residing in a petroleum hydrocarbon–impacted environment. Exposed birds (n = 12; 6- and 12-month environmental residence) were compared with unexposed controls (n = 6). Serum hepatic enzymes, protein indices, bilirubin fractions, renal function markers, and electrolytes were analyzed using standard enzymatic and colorimetric methods. Exposed chickens exhibited significantly elevated AST, ALT, and ALP activities, reduced albumin and total protein concentrations, and increased total and conjugated bilirubin levels relative to controls. Urea concentrations were significantly higher in exposed birds, accompanied by significant reductions in sodium, chloride, and bicarbonate. Creatinine and potassium showed non-significant increases. Biomarker deviations appeared greater after 12 months compared with 6 months of residence, although duration effects were interpreted descriptively. These findings indicate that chronic environmental petroleum hydrocarbon exposure under field conditions is associated with coordinated hepatic dysfunction and renal filtration–electrolyte dysregulation in domestic chickens. The integrated hepato-renal biochemical profile observed highlights the systemic impact of sustained environmental hydrocarbon exposure in poultry populations.*

Keywords - *Petroleum hydrocarbons, hepatotoxicity, nephrotoxicity, biochemical markers, chickens, environmental exposure, Nigeria.*

1. Introduction

Petroleum hydrocarbons are among the most widespread environmental contaminants, released through crude oil exploration, refining, transportation, and accidental spills (Daâssi & Qabil Almaghribi, 2022). In petroleum-producing regions, chronic contamination of soil and water creates continuous exposure pathways for terrestrial organisms through ingestion of contaminated feed and water, inhalation of volatile fractions, and dermal contact (Ola et al., 2024). Prolonged exposure to petroleum-derived mixtures, including polycyclic aromatic hydrocarbons (PAHs) and related compounds, has been associated with multisystem toxicity, with the liver and kidneys particularly vulnerable because of their roles in xenobiotic metabolism, detoxification, and excretion (Fowles et al., 2016; Montano et al., 2025).

The liver is the principal site for biotransformation of petroleum hydrocarbons and PAHs, and toxic injury can occur when reactive metabolites and oxidative stress overwhelm antioxidant defenses. Mechanistically, PAHs and petroleum-associated contaminants can promote reactive oxygen species generation, lipid peroxidation, and inflammatory signaling, which together contribute to hepatocellular damage and functional impairment (Sombiri et al., 2024; Montano et al., 2025). Such injury is commonly reflected by changes in serum hepatic biomarkers, including aminotransferases (AST, ALT), alkaline phosphatase (ALP), albumin, total protein, and bilirubin fractions (Huang et al., 2017; Gudiso et al., 2019).

The kidneys complement hepatic clearance by eliminating water-soluble metabolites and maintaining electrolyte and acid–base homeostasis. Petroleum hydrocarbon exposure may disrupt glomerular filtration and tubular transport processes, leading to alterations in serum urea, creatinine, and electrolytes (Dey et al., 2015; Oleforuh-Okoleh et al., 2023). When renal handling of electrolytes and bicarbonate is impaired during toxic stress, systemic biochemical homeostasis may be disturbed, potentially compounding multi-organ dysfunction.

Hepatic and renal toxicities are biologically interconnected and may act synergistically. Hepatic dysfunction can increase circulating toxic intermediates that burden renal clearance, while impaired renal function can prolong systemic exposure to hepatotoxic compounds.

This hepato-renal crosstalk is increasingly recognized as an important determinant of clinical and toxicological outcomes in acute and chronic injury states (Clementi et al., 2025; Pickkers & Darmon, 2021).

However, many environmental toxicology studies still assess hepatic and renal injury separately, which can underestimate the combined biochemical burden associated with chronic exposure.

Domestic chickens are relevant sentinels for environmental toxicology because they are continuously exposed to soil-, water-, and feed-borne contaminants and can bioaccumulate environmental toxicants, making them useful for monitoring contamination in human-shared environments (Kribi-Boukhris et al., 2020). In petroleum-producing settings where free-ranging poultry may reside near contaminated soils and water bodies, integrated assessment of liver and kidney function is important not only for animal health but also for food production and community exposure concerns.

Although petroleum hydrocarbon exposure has been linked to biochemical disruption in poultry, available studies often focus on single-organ outcomes, controlled conditions, or narrowly defined endpoints rather than coordinated hepato-renal profiles assessed within the same animals under field-relevant chronic exposure (Oleforuh-Okoleh et al., 2023; Harrison & Melford, 2026).

Consequently, duration-related progression and sex-associated variability in integrated hepatic and renal biochemical responses under real-world environmental exposure remain insufficiently characterized. Addressing this gap is necessary for a more comprehensive risk assessment of poultry populations residing in petroleum-impacted regions.

1.1. Aim and Research Questions

Aim: To evaluate whether chronic residence in a petroleum hydrocarbon-impacted environment is associated with coordinated hepatic and renal biochemical alterations in chickens.

Research questions:

1. Do exposed chickens differ from controls in hepatic enzymes, protein indices, and bilirubin fractions?
2. Do exposed chickens differ from controls in urea/creatinine and electrolyte indices?
3. Are biochemical deviations descriptively greater after 12 months of exposure compared with 6 months?

2. Materials and Methods

2.1. Study Design and Exposure Classification

A comparative field-based observational design was employed. Chickens residing in a petroleum hydrocarbon–impacted environment (exposed group) were compared with chickens obtained from an environment without documented hydrocarbon contamination (control group). Exposure status was defined based on environmental residence rather than experimental dosing. No laboratory administration of petroleum hydrocarbons, sham exposure, or controlled dosing protocols were undertaken, reflecting real-world chronic environmental conditions.

2.2. Animals and Sample Size

A total of eighteen domestic chickens were included: twelve environmentally exposed birds and six controls. Within the exposed group, six birds had resided in the contaminated environment for approximately 6 months (3 males, 3 females) and six for approximately 12 months (3 males, 3 females). In the control group, four birds were evaluated at 6 months (2 males, 2 females) and two at 12 months (1 male, 1 female).

Sample size was constrained by the availability of birds meeting predefined residence criteria within the field setting. Given the exploratory nature of this environmental field study, a formal a priori power calculation was not performed.

2.3. Husbandry and Control of Confounding Variables

Birds in both groups were maintained under comparable husbandry conditions typical of local poultry management practices, including access to feed and water. Control birds were sourced from a geographically distinct setting without known petroleum hydrocarbon contamination. Efforts were made to minimize major differences in management practices, housing conditions, and nutritional access between groups to reduce potential confounding effects inherent to field-based designs.

2.4. Sample Collection and Serum Preparation

Blood samples were collected aseptically via venipuncture using sterile disposable syringes into plain (additive-free) collection tubes. Samples were allowed to clot at room temperature and were subsequently centrifuged at 3,000 rpm for 10 minutes to obtain serum. Serum aliquots were stored at 4 °C for short-term analysis and processed within recommended analytical time frames to preserve analyte stability.

2.5. Biochemical Analyses

Hepatic function markers assessed included aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), albumin, total protein, total bilirubin, and conjugated bilirubin. Renal and electrolyte parameters included sodium, potassium, chloride, bicarbonate, urea, and creatinine.

All analytes were measured using standard enzymatic and colorimetric methods according to manufacturer instructions. Absorbance readings were obtained using a visible spectrophotometer (Model S23A, HELMREASINN, China).

All assays were performed in duplicate. Calibration standards and commercially prepared quality-control sera were included in each analytical batch to ensure assay precision and reliability.

2.6. Statistical Analysis

Data were analyzed using IBM SPSS Statistics (Version 26.0; IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean ± standard deviation (SD). Between-group comparisons were performed using Welch's independent-samples t-test to account for unequal variances and small sample sizes.

Results are reported as mean difference (exposed − control) with corresponding 95% confidence intervals (CI), t-statistics with degrees of freedom (df), exact p-values, and Cohen's d effect sizes. Statistical significance was defined as $p < 0.05$ (two-tailed). Given the limited subgroup sizes, exposure-duration comparisons (6 vs 12 months) were interpreted descriptively rather than inferentially.

2.7. Ethical Considerations

All procedures involving animals were conducted in accordance with internationally recognized guidelines for the care and use of experimental animals and followed the ARRIVE reporting recommendations (Norecopa, 2025). Measures were taken to minimize stress and discomfort during handling and sample collection.

3. Results

3.1. Hepatic Biochemical Alterations

Chickens residing in the petroleum hydrocarbon–impacted environment demonstrated significant alterations in hepatic biomarkers compared with controls (Table 1).

AST was markedly elevated in exposed birds (MD = 30.92 IU/L; 95% CI: 20.76–41.08; t = 6.52; df = 14.27; $p < 0.001$; d = 2.41). ALT (MD = 17.25 IU/L; 95% CI: 2.43–32.07; p = 0.026; d = 0.92) and ALP (MD = 27.67 IU/L; 95% CI: 8.50–46.84; p = 0.008; d = 1.28) were also significantly higher in exposed chickens.

In contrast, hepatic synthetic indices were reduced. Albumin was significantly lower in exposed birds (MD = −42.08 g/L; 95% CI: −61.73 to −22.43; $p < 0.001$; d = −1.94), as was total protein (MD = −32.50 g/L; 95% CI: −52.45 to −12.55; p = 0.004; d = −1.52).

Markers of bilirubin metabolism were elevated in exposed chickens, with significant increases in total bilirubin (MD = 7.68 µmol/L; 95% CI: 4.58–10.78; $p < 0.001$; d = 2.01) and conjugated bilirubin (MD = 2.21 µmol/L; 95% CI: 0.81–3.61; p = 0.004; d = 1.28).

3.2. Renal Function and Electrolyte Alterations

Renal parameters also differed between groups (Table 2). Urea concentrations were significantly higher in exposed chickens (MD = 10.98 mmol/L; 95% CI: 4.87–17.09; t = 3.89; df = 12.67; p = 0.002; d = 1.40). Although creatinine was higher in exposed birds, the difference did not reach statistical significance (MD = 1.69 mmol/L; 95% CI: −0.96 to 4.34; p = 0.195; d = 0.57).

Electrolyte disturbances were evident in exposed chickens. Sodium (MD = −46.17 mmol/L; 95% CI: −88.52 to −3.82; p = 0.035; d = −1.20), bicarbonate (MD = −31.75 mmol/L; 95% CI: −44.71 to −18.79; $p < 0.001$; d = −2.92), and chloride (MD = −50.42 mmol/L; 95% CI: −93.18 to −7.66; p = 0.027; d = −1.63) were significantly lower in exposed birds. Potassium was higher but did not reach statistical significance (MD = 1.56 mmol/L; 95% CI: −0.16 to 3.28; p = 0.070; d = 1.15).

3.3. Exposure Duration Patterns

Across most hepatic and renal biomarkers, deviations from control values appeared greater in birds residing in the contaminated environment for 12 months compared with 6 months. However, inferential subgroup analysis was not performed due to small sex-by-duration subgroup sizes; therefore, duration-related trends are interpreted descriptively.

4. Discussion

This study demonstrated coordinated biochemical alterations involving hepatic enzymes, hepatic synthetic indices, bilirubin fractions, urea, and electrolyte parameters in chickens chronically residing in a petroleum hydrocarbon–impacted environment. The pattern observed across multiple organ-related biomarkers supports the presence of systemic biochemical stress rather than isolated single-organ perturbation.

Elevations in AST, ALT, and ALP in exposed birds are consistent with hepatocellular injury and possible cholestatic involvement. Petroleum hydrocarbons and associated polycyclic aromatic hydrocarbons (PAHs) undergo hepatic biotransformation that can generate reactive metabolites and reactive oxygen species, promoting oxidative stress, membrane lipid peroxidation, and inflammatory signaling (Huang et al., 2017; Montano et al., 2025). Sustained oxidative burden may compromise hepatocyte membrane integrity, leading to leakage of intracellular enzymes into circulation. Concurrent reductions in albumin and total protein suggest impaired hepatic synthetic function or altered protein metabolism under chronic toxic stress. Increased total and conjugated bilirubin further indicate disruption of

hepatobiliary handling and clearance mechanisms, reinforcing the interpretation of hepatic dysfunction.

Renal involvement was evidenced by significantly elevated urea and by electrolyte disturbances characterized by reduced sodium, chloride, and bicarbonate concentrations. These findings are compatible with impaired glomerular filtration and altered tubular reabsorption processes under toxicant exposure (Dey et al., 2015; Oleforuh-Okoleh et al., 2023). Decreased bicarbonate levels may reflect disturbance in acid–base regulation, while reduced sodium and chloride suggest compromised tubular transport. Although creatinine and potassium increases did not reach statistical significance, their directional trends align with a broader pattern of renal stress. The absence of statistical significance for certain markers likely reflects limited statistical power rather than the absence of a biological effect, given the small sample size inherent to field-based designs.

Importantly, hepatic and renal alterations were observed concurrently in the same animals. This integrated profile supports the concept of hepato-renal interaction during chronic toxic exposure, wherein hepatic dysfunction may increase systemic toxic intermediates while renal impairment prolongs circulating exposure to hepatotoxic compounds (Clementi et al., 2025; Pickkers & Darmon, 2021). Evaluating these organ systems simultaneously provides a more comprehensive assessment of systemic biochemical burden compared with single-organ analyses.

Duration-related trends suggested greater deviations after 12 months compared with 6 months of environmental residence, consistent with cumulative toxic effects expected during sustained exposure. However, formal subgroup inference was not undertaken due to small sex-by-duration subgroup sizes. These patterns should therefore be interpreted cautiously and confirmed in larger, adequately powered studies with direct environmental exposure quantification.

Collectively, the findings indicate that chronic environmental petroleum hydrocarbon exposure in field conditions is associated with coordinated biochemical disturbances affecting hepatic function and renal filtration–electrolyte regulation in domestic chickens.

5. Conclusion

Chickens residing in a petroleum hydrocarbon–impacted environment exhibited biochemical alterations consistent with combined hepatic dysfunction and renal filtration–electrolyte dysregulation. Exposed birds showed significant elevations in hepatic enzymes and bilirubin fractions, reductions in albumin and total protein, increased urea concentrations, and decreased sodium, chloride, and bicarbonate levels. Although creatinine and potassium changes were not statistically significant, their directional shifts supported the overall pattern of renal involvement.

These results provide evidence of coordinated hepato-renal biochemical stress under chronic environmental petroleum hydrocarbon exposure in a field setting.

5.1. Implications

Chronic biochemical disruption in poultry raised in hydrocarbon-impacted environments may compromise animal health, productivity, and potentially food-system safety in petroleum-producing communities. Integrated monitoring of livestock biochemical markers alongside environmental remediation strategies may strengthen risk management in contaminated regions.

Limitations and Future Directions

This field-based investigation was constrained by limited sample availability, resulting in small subgroup sizes and reduced statistical power for duration- and sex-specific analyses. Environmental hydrocarbon concentrations and specific contaminant profiles (e.g., total petroleum hydrocarbons [TPH], polycyclic aromatic hydrocarbons [PAHs]) were not directly quantified, and exposure classification was based on environmental residence rather than measured dose. Additionally, the study relied on serum biochemical indices without histopathological or molecular confirmation of tissue injury.

Future studies should incorporate environmental chemical profiling, larger sample sizes, controlled exposure quantification, and tissue-level analyses to clarify mechanistic pathways and strengthen causal inference.

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Table 1. Liver function parameters of chickens exposed to a petroleum hydrocarbon–contaminated environment

Parameter	Exposed (n=12) Mean ± SD	Control (n=6) Mean ± SD	Mean diff (E−C)	95% CI	t (df)	p-value	Cohen's d
AST (IU/L)	43.92 ± 15.14	13.00 ± 4.52	30.92	20.76 to 41.08	6.52 (14.27)	<0.001	2.41
ALT (IU/L)	25.42 ± 22.15	8.17 ± 6.46	17.25	2.43 to 32.07	2.49 (14.16)	0.026	0.92
ALP (IU/L)	52.67 ± 24.37	25.00 ± 13.84	27.67	8.50 to 46.84	3.07 (15.54)	0.008	1.28
Albumin (g/L)	53.42 ± 24.23	95.50 ± 14.73	−42.08	−61.73 to −22.43	−4.56 (15.11)	<0.001	−1.94
Total protein (g/L)	59.00 ± 23.61	91.50 ± 15.60	−32.50	−52.45 to −12.55	−3.48 (14.42)	0.004	−1.52
Total bilirubin (µmol/L)	11.08 ± 4.46	3.40 ± 1.67	7.68	4.58 to 10.78	5.27 (15.37)	<0.001	2.01
Conjugated bilirubin (µmol/L)	3.06 ± 2.01	0.85 ± 0.76	2.21	0.81 to 3.61	3.36 (15.42)	0.004	1.28

Values are mean ± SD. Group comparisons used Welch's t-test. Mean difference is exposed − control. p-values are two-tailed; significance at $p < 0.05$.

Table 2. Renal function and electrolyte parameters of chickens exposed to a petroleum hydrocarbon–contaminated environment

Parameter	Exposed (n=12) Mean ± SD	Control (n=6) Mean ± SD	Mean diff (E−C)	95% CI	t (df)	p-value	Cohen's d
Sodium (mmol/L)	113.33 ± 38.97	159.50 ± 37.82	−46.17	−88.52 to −3.82	−2.42 (10.39)	0.035	−1.20
Potassium (mmol/L)	3.54 ± 1.23	1.98 ± 1.61	1.56	−0.16 to 3.28	2.09 (8.03)	0.070	1.15
Bicarbonate (mmol/L)	37.08 ± 10.37	68.83 ± 11.94	−31.75	−44.71 to −18.79	−5.55 (8.91)	<0.001	−2.92
Chloride (mmol/L)	83.58 ± 25.28	134.00 ± 40.54	−50.42	−93.18 to −7.66	−2.79 (7.01)	0.027	−1.63
Urea (mmol/L)	13.40 ± 9.40	2.42 ± 1.89	10.98	4.87 to 17.09	3.89 (12.67)	0.002	1.40
Creatinine (mmol/L)	3.21 ± 3.34	1.52 ± 1.94	1.69	−0.96 to 4.34	1.35 (15.41)	0.195	0.57

Values are mean ± SD. Group comparisons used Welch's t-test. Mean difference is exposed − control. p-values are two-tailed; significance at $p < 0.05$.

Table 3. Summary of key biochemical findings in exposed chickens compared with controls

Organ/System	Marker	Direction	Statistical evidence	Interpretation
Liver	AST	↑	$p < 0.001$	Hepatocellular injury
Liver	ALT	↑	$p = 0.026$	Hepatocellular injury
Liver	ALP	↑	$p = 0.008$	Cholestatic/enzymatic stress
Liver	Albumin	↓	$p < 0.001$	Reduced synthetic function
Liver	Total protein	↓	$p = 0.004$	Reduced synthetic/overall protein status
Liver	Total bilirubin	↑	$p < 0.001$	Impaired bilirubin handling
Liver	Conjugated bilirubin	↑	$p = 0.004$	Hepatobiliary dysfunction
Kidney	Urea	↑	$p = 0.002$	Reduced filtration/renal stress
Electrolytes	Sodium	↓	$p = 0.035$	Electrolyte dysregulation
Electrolytes	Chloride	↓	$p = 0.027$	Tubular/acid–base disturbance
Electrolytes	Bicarbonate	↓	$p < 0.001$	Acid–base disturbance

Arrows indicate statistically significant direction of change in exposed chickens relative to controls based on Welch's t-test ($p < 0.05$). Potassium and creatinine showed increases that did not reach statistical significance and are not listed as significant findings.