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Endocrine and Thyroid Disruption Induced by Petroleum Hydrocarbon Exposure in Chickens

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ABSTRACT

Background: *Petroleum hydrocarbon contamination of the environment poses significant toxicological risks to exposed organisms, particularly through disruption of endocrine regulation. However, data on the combined effects of chronic hydrocarbon exposure on reproductive and thyroid hormones in avian species remain limited.*

Objective: *This study evaluated the impact of chronic exposure to a petroleum hydrocarbon-contaminated environment on reproductive and thyroid hormonal profiles in chickens, with emphasis on exposure duration and sex-related differences.*

Methods: *A comparative experimental design was employed involving exposed chickens (6- and 12-month exposure durations) and unexposed controls. Serum levels of reproductive hormones (follicle-stimulating hormone, luteinizing hormone, estrogen, progesterone, prolactin, and testosterone) and thyroid hormones (thyroid-stimulating hormone, triiodothyronine, and thyroxine) were measured using standard immunoassay techniques. Data were analyzed using descriptive and inferential statistics at a significance level of $p < 0.05$.*

Results: *Petroleum hydrocarbon exposure was associated with significant alterations in reproductive and thyroid hormone profiles. Exposed chickens exhibited disrupted gonadotropin and sex steroid balance, reduced testosterone levels in males, and estrogen–progesterone imbalance in females. Thyroid dysfunction was characterized by elevated thyroid-stimulating hormone with reduced circulating thyroid hormones. Hormonal disturbances were more pronounced with prolonged exposure and demonstrated sex-dependent patterns.*

Conclusion: *Chronic petroleum hydrocarbon exposure induces multiaxial endocrine disruption in chickens, affecting both reproductive and thyroid hormonal systems. These findings highlight the endocrine toxicity of petroleum hydrocarbons and underscore the need for environmental monitoring and mitigation in contaminated regions.*

Keywords: *Petroleum hydrocarbons, Endocrine disruption, Thyroid dysfunction, Reproductive hormones, Chickens, Nigeria.*

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INTRODUCTION

Petroleum hydrocarbons are complex mixtures of aliphatic, aromatic, and polycyclic organic compounds released into the environment through crude oil exploration, refining, transportation, and accidental spills [1]. In regions characterized by prolonged petroleum-related activities, persistent contamination of soil, water, and air has become a major environmental concern due to continuous exposure and bioaccumulation of toxic constituents [2]. Among the numerous toxicological consequences associated with petroleum hydrocarbon exposure, disruption of endocrine homeostasis has increasingly attracted scientific attention as an important mechanism underlying systemic toxicity.

Endocrine-disrupting chemicals (EDCs) interfere with hormone synthesis, secretion, metabolism, transport, receptor binding, and signaling pathways, thereby altering normal physiological regulation even at relatively low exposure levels [3].

Several constituents of petroleum hydrocarbons, particularly polycyclic aromatic hydrocarbons (PAHs) and associated co-contaminants, have demonstrated estrogenic, anti-androgenic, and thyroid-disrupting activities in both experimental models and wildlife species [4]. These endocrine alterations may adversely affect reproductive capacity, metabolic regulation, growth, development, and stress adaptation, especially under conditions of chronic environmental exposure [5].

The reproductive endocrine axis is particularly vulnerable to environmental toxicants because hormonal regulation within the hypothalamic–pituitary–gonadal axis depends on tightly coordinated feedback mechanisms. Alterations in gonadotropins such as follicle-stimulating hormone (FSH) and luteinizing hormone (LH), as well as reproductive steroid hormones including estrogen, progesterone, prolactin, and testosterone, have been reported in animals exposed to hydrocarbon-contaminated environments [6]. Such disturbances may impair steroidogenesis, gametogenesis, reproductive behavior, and fertility, ultimately affecting reproductive performance and population sustainability [7].

In avian species, these effects are of particular concern because birds interact closely with contaminated terrestrial ecosystems through feeding, foraging, and nesting activities.

In addition to reproductive dysfunction, petroleum hydrocarbons may also disrupt thyroid endocrine function. Thyroid hormones, triiodothyronine (T3) and thyroxine (T4), play essential roles in metabolism, thermoregulation, growth, and development, while thyroid-stimulating hormone (TSH) regulates their synthesis and secretion [8]. Petroleum hydrocarbons and associated environmental co-pollutants have been shown to interfere with thyroid hormone synthesis, transport, and peripheral metabolism, leading to thyroid dysfunction and altered metabolic homeostasis [9]. Such disturbances may further aggravate physiological stress and contribute to broader endocrine imbalance.

Despite increasing evidence regarding the endocrine toxicity of petroleum hydrocarbons, studies evaluating the combined effects of chronic exposure on both reproductive and thyroid hormonal systems remain limited, particularly in avian species [10]. Furthermore, sex-dependent and duration-dependent endocrine responses under long-term environmental exposure conditions are still poorly characterized [11]. Chickens represent valuable sentinel species in environmental toxicology because of their physiological sensitivity, ecological relevance, and close association with human food systems, making endocrine disturbances in these birds important from both ecological and public health perspectives [12].

Although endocrine-disrupting effects of petroleum hydrocarbons have been extensively investigated in mammals and aquatic organisms, avian endocrine toxicity studies remain comparatively limited globally, particularly in environmentally exposed domestic poultry species from low- and middle-income countries [25].

Therefore, this study evaluated the effects of chronic exposure to a petroleum hydrocarbon-contaminated environment on reproductive and thyroid hormonal profiles in chickens, with emphasis on exposure duration (6 versus 12 months) and sex-related differences. Gonadotropins (FSH and LH), reproductive steroid hormones (estrogen, progesterone, prolactin, and testosterone), and thyroid function parameters (TSH, T3, and T4) were assessed in exposed chickens and compared with unexposed controls. It was hypothesized that petroleum hydrocarbon exposure would significantly disrupt reproductive and thyroid hormone profiles, that prolonged exposure would exacerbate endocrine alterations, and that male and female chickens would exhibit differential hormonal responses. This integrated assessment provides important insight into endocrine and thyroid dysfunction associated with chronic petroleum hydrocarbon contamination.

Materials and Methods

Study Design

This study employed a comparative experimental design to evaluate the effects of chronic exposure to a petroleum hydrocarbon-contaminated environment on reproductive and thyroid hormonal profiles in chickens. Chickens chronically exposed to petroleum hydrocarbons were compared with age-matched control birds maintained in a non-contaminated environment. Hormonal assessments were conducted after two exposure durations (6 months and 12 months), and analyses were stratified according to sex to evaluate sex- and duration-dependent endocrine responses.

Study Area and Experimental Animals

Exposed chickens were obtained from an environment with documented chronic petroleum hydrocarbon contamination associated with sustained 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 comparable access to feed and water, in order to minimize potential confounding environmental and nutritional influences.

A total of eighteen chickens were included in the study, comprising twelve exposed birds and six control birds. 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). 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 assessment of exposure-related endocrine effects, sex-specific hormonal responses, and duration-dependent alterations.

Blood Sample Collection and Serum Preparation

Blood samples were collected aseptically from each chicken through venipuncture into plain sample containers. The samples were allowed to clot under standard laboratory conditions and were subsequently centrifuged to obtain serum. Separated serum samples were carefully stored at appropriate temperatures and analyzed within recommended time frames to preserve hormonal stability and analytical integrity.

Hormonal Analysis

Serum concentrations of reproductive hormones, including follicle-stimulating hormone (FSH), luteinizing hormone (LH), estrogen, progesterone, prolactin, and testosterone, as well as thyroid hormones comprising thyroid-stimulating hormone (TSH), triiodothyronine (T3), and thyroxine (T4), were determined using enzyme-linked immunosorbent assay (ELISA) techniques in accordance with manufacturer protocols.

Quantitative hormonal measurements were performed using a Microplate Reader (Model M201, EMPSUN®, Chengdu Empsun Medical Technology Co. Ltd., China). Absorbance readings were obtained at assay-specific wavelengths, and hormone concentrations were calculated using standard calibration curves.

All hormonal assays were performed in duplicate to ensure analytical reliability and reproducibility. Calibration standards and internal quality-control samples were included in each assay batch, and all laboratory procedures were conducted according to standard operating protocols in order to minimize analytical variability.

Statistical Analysis

Data were analyzed using appropriate statistical software and expressed as mean ± standard deviation. Comparisons between exposed and control groups were performed using independent-sample t-tests, while one-way analysis of variance (ANOVA) was used to evaluate differences associated with exposure duration and sex, followed by appropriate post-hoc analyses where necessary. Statistical significance was established at $p < 0.05$.

Analyses were restricted to reproductive and thyroid endocrine parameters to maintain methodological independence from other system-specific investigations derived from the same experimental cohort.

Ethical Considerations

All experimental procedures involving animals were conducted in accordance with internationally accepted guidelines for the care and use of laboratory animals. Every effort was made to minimize animal stress and discomfort throughout the study period.

RESULTS AND DISCUSSION

Exposure of chickens to a petroleum hydrocarbon-contaminated environment was associated with marked disturbances in reproductive hormonal profiles when compared with control birds. Alterations in follicle-stimulating hormone (FSH) and luteinizing hormone (LH) were evident among exposed chickens, indicating disruption of hypothalamic–pituitary regulation and impairment of endocrine signaling pathways involved in reproductive homeostasis. These hormonal changes suggest that chronic petroleum hydrocarbon exposure may interfere with the normal regulation of reproductive endocrine function.

In male chickens, testosterone concentrations were generally reduced following exposure, particularly among birds exposed for 12 months, suggesting impaired testicular steroidogenesis and altered androgen biosynthesis. Petroleum hydrocarbons and their derivatives have previously been reported to interfere with steroidogenic enzyme activity and gonadal endocrine regulation, thereby contributing to reproductive hormonal imbalance and impaired fertility potential [13,14]. The progressive decline observed with prolonged exposure further supports the cumulative endocrine toxicity associated with chronic hydrocarbon contamination.

Female chickens demonstrated considerable variability in estrogen and progesterone concentrations. Several exposed birds exhibited elevated progesterone levels relative to estrogen, which may indicate ovarian dysfunction, altered peripheral metabolism of steroid hormones, or disruption of hormonal feedback mechanisms regulating reproductive physiology [15]. Similar endocrine disturbances have been reported in animals exposed to hydrocarbon-contaminated environments, where petroleum-associated toxicants were shown to impair ovarian endocrine regulation and reproductive hormone balance [7]. Such hormonal alterations may ultimately compromise reproductive performance and reproductive sustainability in exposed populations.

Prolactin concentrations were also altered among exposed chickens, although the direction of change varied between individuals. This variability may reflect stress-induced endocrine activation associated with chronic toxic exposure and prolonged environmental contamination. Prolactin dysregulation has previously been associated with environmental stressors and xenobiotic exposure through disruption of dopaminergic inhibition at the pituitary level [16, 17]. The heterogeneous prolactin responses observed in this study further support the concept that endocrine disruption frequently manifests as dysregulated hormonal signaling rather than uniform hormonal suppression or elevation.

In addition to reproductive endocrine disturbances, petroleum hydrocarbon exposure was associated with significant alterations in thyroid hormonal profiles. Exposed chickens generally exhibited elevated thyroid-stimulating hormone (TSH) concentrations accompanied by reduced or inconsistently altered triiodothyronine (T3) and thyroxine (T4) levels.

This hormonal pattern suggests a compensatory pituitary response to impaired thyroid hormone synthesis or altered peripheral metabolism of thyroid hormones. Environmental hydrocarbons and associated contaminants have been shown to interfere with thyroid peroxidase activity, iodide transport, and deiodinase enzyme function, thereby disrupting thyroid hormone synthesis and conversion pathways [18, 19]. The findings of the present study are therefore consistent with previously described mechanisms of hydrocarbon-induced thyroid dysfunction.

The duration of exposure appeared to play an important role in the severity of endocrine disruption observed among exposed chickens. Birds exposed for 12 months demonstrated more pronounced alterations in both reproductive and thyroid hormonal profiles compared with those exposed for 6 months. This duration-dependent pattern suggests cumulative toxicological effects associated with prolonged petroleum hydrocarbon exposure. Chronic exposure may facilitate progressive accumulation of toxic metabolites and sustained oxidative stress, both of which can impair endocrine gland integrity and hormonal regulation over time [20, 6]. These findings highlight the importance of considering exposure duration when evaluating endocrine toxicity in environmentally exposed animal populations.

Sex-related differences in hormonal responses were also evident in this study. Male chickens predominantly exhibited reductions in testosterone concentrations together with altered gonadotropin profiles, whereas female chickens demonstrated greater disturbances in estrogen–progesterone balance. Thyroid hormonal disruption was observed in both sexes but appeared more pronounced among females following prolonged exposure [21]. Sex-dependent susceptibility to endocrine-disrupting chemicals has been widely documented and may be influenced by differences in hormone metabolism, receptor sensitivity, body fat composition, and detoxification capacity between males and females [22]. These findings further emphasize the importance of sex-stratified analyses in environmental toxicology investigations.

The concurrent disruption of reproductive and thyroid hormones observed in this study indicates that petroleum hydrocarbon exposure induces multiaxial endocrine toxicity rather than isolated hormonal disturbances. Interactions between the reproductive and thyroid endocrine axes are well established, and thyroid dysfunction may influence gonadotropin secretion, steroidogenesis, and overall reproductive performance. Consequently, thyroid hormonal imbalance may further aggravate reproductive endocrine dysfunction in chronically exposed chickens, thereby amplifying the overall physiological and toxicological burden [23, 24].

Collectively, the findings of this study provide evidence that chronic exposure to petroleum hydrocarbon-contaminated environments disrupts endocrine homeostasis in chickens through combined reproductive and thyroid hormonal alterations. Such endocrine toxicity may have important implications for animal health, reproductive efficiency, and population sustainability in hydrocarbon-impacted regions. Furthermore, given the ecological relevance of chickens as sentinel species and their close association with human food systems, these findings raise broader concerns regarding the environmental and public health consequences of persistent petroleum hydrocarbon contamination.

Table 1: Reproductive Hormone Profiles of Chickens Exposed to Petroleum Hydrocarbon–Contaminated Environment

A. Exposed Chickens (6 and 12 Months)								
Sample	**Sex**	**Duration**	**FSH (mIU/mL)**	**LH (mIU/mL)**	**Estrogen (pg/mL)**	**Progesterone (ng/mL)**	**Prolactin (ng/mL)**	**Testosterone (ng/mL)**
M1	Male	6 months	8.10	3.02	–	–	<0.50	2.90
M2	Male	6 months	0.40	6.00	–	–	0.81	8.90
M3	Male	6 months	4.10	7.10	–	–	0.93	10.00
F1	Female	6 months	2.34	4.30	56	22.29	10.21	–
F2	Female	6 months	0.21	7.23	31	41.41	6.54	–
F3	Female	6 months	4.34	10.00	80	43.60	2.66	–
M4	Male	12 months	7.10	4.51	–	–	0.11	4.80
M5	Male	12 months	3.00	7.21	–	–	1.52	1.60
M6	Male	12 months	6.20	8.32	–	–	0.61	2.20
F4	Female	12 months	2.00	11.20	60	53.21	7.30	–
F5	Female	12 months	5.33	7.50	21	25.40	2.51	–
F6	Female	12 months	2.67	5.20	31	33.20	3.26	–

B. Control Chickens								
Sample	**Sex**	**Duration**	**FSH (mIU/mL)**	**LH (mIU/mL)**	**Estrogen (pg/mL)**	**Progesterone (ng/mL)**	**Prolactin (ng/mL)**	**Testosterone (ng/mL)**
M1	Male	6 months	0.70	4.10	–	–	0.02	8.40
M2	Male	6 months	4.80	10.22	–	–	0.06	4.22
M3	Male	12 months	7.10	7.31	–	–	1.00	6.00
F1	Female	6 months	4.65	12.20	50.10	11.31	7.60	–
F2	Female	6 months	10.32	9.16	22.32	9.10	11.56	–
F3	Female	12 months	5.21	11.32	98.20	6.45	9.12	–

Table 2: Thyroid Hormone Profiles of Chickens Exposed to Petroleum Hydrocarbon–Contaminated Environment

A. Exposed Chickens (6 and 12 Months)					
Sample	**Sex**	**Duration**	**TSH (mIU/L)**	**T4 (ng/mL)**	**T3 (ng/mL)**
M1	Male	6 months	3.23	1.00	0.12
M2	Male	6 months	6.10	0.60	2.02
M3	Male	6 months	9.65	2.00	0.40
F1	Female	6 months	5.00	3.12	1.02
F2	Female	6 months	2.12	0.98	2.50
F3	Female	6 months	7.02	4.60	1.12
M4	Male	12 months	10.01	0.10	0.43
M5	Male	12 months	16.80	1.21	2.06
M6	Male	12 months	19.43	0.53	1.22
F4	Female	12 months	12.00	2.07	0.11
F5	Female	12 months	11.44	0.65	1.20
F6	Female	12 months	7.10	1.00	0.46

B. Control Chickens					
Sample	**Sex**	**Duration**	**TSH (mIU/L)**	**T4 (ng/mL)**	**T3 (ng/mL)**
M1	Male	6 months	1.23	4.80	4.60
M2	Male	6 months	4.50	6.13	0.96
M3	Male	12 months	0.10	9.21	6.54
F1	Female	6 months	5.70	7.00	9.12
F2	Female	6 months	0.38	2.16	8.32
F3	Female	12 months	6.18	13.76	12.05

CONCLUSION

This study demonstrated that chronic exposure to a petroleum hydrocarbon-contaminated environment significantly disrupts reproductive and thyroid hormonal homeostasis in chickens. Alterations observed in gonadotropins and reproductive steroid hormones among exposed birds indicate impairment of the hypothalamic–pituitary–gonadal axis, with evidence of reduced androgen production in males and disturbances in estrogen–progesterone balance among females. Concurrent alterations in thyroid hormonal profiles, characterized by elevated thyroid-stimulating hormone and reduced or inconsistently altered circulating thyroid hormones, further suggest compromised thyroid function and disruption of metabolic regulation.

The severity of hormonal disturbances appeared to increase with prolonged exposure duration, indicating cumulative toxicological effects associated with chronic petroleum hydrocarbon contamination.

In addition, the sex-dependent differences observed in hormonal responses suggest differential susceptibility to endocrine disruption between male and female chickens.

The findings of this study provide evidence that petroleum hydrocarbon exposure induces multiaxial endocrine toxicity involving both reproductive and thyroid hormonal systems. These endocrine disturbances may adversely affect animal health, reproductive performance, physiological stability, and population sustainability in hydrocarbon-impacted environments. Given the ecological importance of chickens as sentinel species and their close association with human food systems, the findings underscore the importance of continued environmental monitoring and implementation of mitigation strategies aimed at reducing the endocrine-disrupting effects of persistent petroleum hydrocarbon contamination.

LIMITATIONS AND FUTURE DIRECTIONS

Despite providing important insights into endocrine and thyroid disruption associated with petroleum hydrocarbon exposure, this study has certain limitations that should be considered when interpreting the findings. The sample size was relatively small, which may limit the generalizability of the results and the statistical power to detect subtle hormonal differences. However, the consistent trends observed across exposure durations and sexes suggest that the detected endocrine alterations are biologically relevant. In addition, the environmental nature of exposure in this study reflects real-world conditions but does not allow precise quantification of individual hydrocarbon dose or identification of specific hydrocarbon constituents responsible for the observed effects.

The study focused on circulating hormonal levels and did not include molecular or histopathological assessments of endocrine organs such as the thyroid, pituitary, or gonads. As a result, mechanistic insights at the tissue or cellular level could not be directly evaluated. Furthermore, potential interactions between petroleum hydrocarbons and co-occurring environmental contaminants, including heavy metals, were not explored within the scope of this endocrine-focused analysis.

Future studies should incorporate larger sample sizes and controlled exposure designs to strengthen causal inference and dose–response characterization. Integration of molecular biomarkers, histopathological evaluation, and gene expression analyses of endocrine tissues would provide deeper mechanistic understanding of hydrocarbon-induced endocrine disruption. Additionally, investigating the interactive effects of petroleum hydrocarbons with heavy metals and oxidative stress pathways may help elucidate complex toxicity mechanisms. Longitudinal studies assessing reproductive outcomes and transgenerational effects would further clarify the long-term ecological and health implications of chronic petroleum hydrocarbon exposure in avian species.

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