Magna Scientia Advanced Research and Reviews
eISSN: 2582-9394
Cross Ref DOI: 10.30574/msarr
Journal homepage: https://msarr.magnascientiapub.com/

(RESEARCH ARTICLE)

Check for updates

QUERCETIN AND CURCUMIN ATTENUATE GASOLINE FUME-INDUCED OXIDATIVE STRESS AND MODULATE ANTIOXIDANT ENZYME ACTIVITIES IN MALE WISTAR RATS

Ucheaku Rita Soludo *, Chukwuma Walter Nwafia and Izuchukwu Charles Ifedi

Department of Human Physiology, Faculty of Basic Medical Sciences, Chukwuemeka Odumegwu Ojukwu University, Uli Campus, Anambra, Nigeria.

* Corresponding Author

ORCID Details
Izuchukwu Charles Ifedi: 0009-0002-1283-6748

Magna Scientia Advanced Research and Review, 2026, 17(02), 458–464

Publication history: Received on 14 July 2026; revised on 26 August 2026; accepted on 28 August 2026

Article DOI: https://doi.org/10.30574/msarr.2026.17.2.0172

ABSTRACT

Background: Inhalation of gasoline fumes constitutes a significant occupational and environmental health hazard capable of inducing oxidative stress and depleting endogenous antioxidant defenses in exposed individuals. Quercetin and curcumin are naturally occurring dietary polyphenols with well-established antioxidant and anti-inflammatory properties that may offer protective benefits against such toxicant-induced biochemical disruption.

Objective: This study investigated the ameliorative effects of quercetin and curcumin on serum oxidative stress markers and antioxidant enzyme activities in male Wistar rats subjected to sub-chronic leaded gasoline fume inhalation.

Methods: Sixty (60) adult male albino Wistar rats were randomly assigned into seven groups. Group A served as the negative control. Groups B1 and B2 were exposed to 20 ml leaded gasoline fumes (one hour daily) for two weeks, then treated with quercetin (50 mg/kg) and curcumin (50 mg/kg) respectively by oral gavage for one week. Groups C1 and C2 underwent three weeks of gasoline fume exposure followed by two weeks of quercetin and curcumin treatment respectively. Groups D1 and D2 were exposed for two and three weeks respectively with no antioxidant treatment. Serum malondialdehyde (MDA), reduced glutathione (GSH), superoxide dismutase (SOD), and catalase were measured using standard spectrophotometric methods. Data were analysed using GraphPad Prism 9.5.1 with one-way ANOVA followed by Fisher's LSD post-hoc test and paired t-test for body weight comparisons; significance was set at $p<0.05$.

Results: Gasoline fume exposure significantly ($p<0.05$) elevated serum MDA and significantly reduced SOD, catalase, and GSH in the untreated exposure groups (D1 and D2), with three-week exposure producing greater derangement than two-week exposure. Post-exposure quercetin and curcumin supplementation significantly attenuated MDA elevation

and restored antioxidant enzyme activities towards control values, with quercetin demonstrating superior recovery across most biochemical parameters compared to curcumin at equivalent dose.

Conclusion: Sub-chronic leaded gasoline fume inhalation produces duration-dependent oxidative stress in male Wistar rats, and post-exposure supplementation with quercetin or curcumin at 50 mg/kg significantly mitigates these effects. Quercetin demonstrated greater antioxidant efficacy than curcumin under the conditions studied, supporting its therapeutic potential against petroleum fume-induced oxidative injury.

Keywords: Gasoline Fumes; Oxidative Stress; Quercetin; Curcumin; Antioxidant Enzymes; Wistar Rats.

1 INTRODUCTION

Gasoline is a manufactured petroleum-derived fuel that does not occur naturally in the environment. It is a complex mixture of over 150 organic chemical compounds including benzene, toluene, xylene, and aliphatic hydrocarbons, and in its leaded formulation (RON 80) carries the toxic additive tetraethyllead [1,2]. While regulatory pressure has driven a global shift toward unleaded fuels, leaded RON 80 gasoline continues to be widely used across many low- and middle-income settings, including occupational environments such as filling stations, road transport, and automobile repair workshops, sustaining chronic inhalational exposure among workers and surrounding communities [3].

The respiratory system bears the brunt of gasoline fume toxicity, as inhaled volatile organic compounds (VOCs) make direct contact with airway mucosa and cross the alveolar epithelium to enter systemic circulation. Once absorbed, benzene and related aromatic hydrocarbons undergo cytochrome P450-mediated biotransformation to reactive epoxides and quinone intermediates, initiating cascades of reactive oxygen species (ROS) generation [4]. The resulting oxidative burden disrupts the delicate balance between pro-oxidant and antioxidant forces in biological tissues, leading to lipid peroxidation, protein oxidation, and DNA strand breaks that collectively drive respiratory and systemic organ injury [5,6]. Comparable oxidative biomarker derangement has been documented following inhalation of other combustion-derived pollutants, including cigarette smoke, in Wistar rat models [7]

Malondialdehyde (MDA) is a well-validated terminal biomarker of lipid peroxidation and serves as a quantitative index of oxidative membrane damage. Reduced glutathione (GSH), superoxide dismutase (SOD), and catalase constitute the core of the cell's endogenous antioxidant defense network. SOD catalyzes the dismutation of superoxide radicals to hydrogen peroxide (H_2O_2), which catalase subsequently converts to water and molecular oxygen, limiting the generation of the highly damaging hydroxyl radical [8,9]. Depletion of these defense molecules under sustained oxidative insult renders tissues progressively susceptible to cumulative damage [10].

Quercetin, a flavonoid found abundantly in onions, apples, berries, and green tea, has attracted substantial research interest for its potent antioxidant, anti-inflammatory, and cytoprotective properties [11]. It scavenges ROS directly and activates the Nrf2/antioxidant response element transcriptional pathway to amplify endogenous antioxidant enzyme biosynthesis, while also suppressing NF-κB-mediated pro-inflammatory gene expression [12,13]. Curcumin, the principal bioactive polyphenol of Curcuma longa (turmeric), engages parallel but complementary mechanisms, inhibiting NF-κB and activating Nrf2-driven antioxidant gene transcription, and has been explored for its potential to protect against toxicity from environmental pollutants including petroleum-derived compounds [14,15].

Despite growing evidence for the individual antioxidant pharmacology of quercetin and curcumin, comparative data on their post-exposure therapeutic efficacy against gasoline fume-induced biochemical perturbations in a controlled animal model remain limited. The present study was therefore designed to evaluate and compare the ameliorative effects of quercetin and curcumin supplementation on MDA, GSH, SOD, and catalase in male Wistar rats subjected to sub-chronic leaded gasoline fume inhalation at two exposure durations.

2 MATERIALS AND METHODS

2.1 Study Area and Ethical Approval

The study was carried out at the Animal House, Department of Human Physiology, Faculty of Basic Medical Sciences, College of Medicine, Chukwuemeka Odumegwu Ojukwu University, Uli Campus, Anambra State, Nigeria. Ethical approval was obtained from the Institutional Animal Ethics Committee prior to commencement of all experimental procedures. All animal work was conducted in accordance with internationally accepted principles for laboratory animal use and care.

2.2 Experimental Animals and Housing

Sixty (60) adult male albino Wistar rats with a mean initial body weight of approximately 130 g were used. Animals were housed in standard laboratory conditions (12-hour light/dark cycle; temperature 22 ± 2°C) with ad libitum access to standard rat chow and clean drinking water. A one-week acclimatization period preceded the start of experimental procedures.

2.3 Chemicals and Reagents

Quercetin (5 g, pure form) was procured from Taqwa Pharma. Curcumin (5 g) was obtained from Sigma-Aldrich. Commercial leaded gasoline (RON 80) was purchased from a licensed filling station in Uli, Anambra State, Nigeria. All biochemical assay reagents were of analytical grade.

2.4 Gasoline Fume Exposure

Gasoline fume inhalation was conducted inside a stainless-steel exposure chamber (1.5 m × 0.9 m × 2.1 m) equipped with upper and lateral ventilation holes. Two calibrated 1000 ml beakers, each containing 500 ml of leaded gasoline, were placed at the chamber base and allowed to equilibrate for one hour prior to each session to permit fume saturation of the chamber atmosphere, following the method of Uboh et al. [16]. All exposure sessions lasted one hour per day, seven days per week.

2.5 Experimental Design and Grouping

The sixty rats were randomly distributed into seven groups as described in Table 1 below:

No.	Group	Treatment	Exposure Duration
1	A	Control — feed and water only	None
2	B1	Quercetin 50 mg/kg orally for 1 week (post-exposure)	2 weeks gasoline fume exposure
3	B2	Curcumin 50 mg/kg orally for 1 week (post-exposure)	2 weeks gasoline fume exposure
4	C1	Quercetin 50 mg/kg orally for 2 weeks (post-exposure)	3 weeks gasoline fume exposure
5	C2	Curcumin 50 mg/kg orally for 2 weeks (post-exposure)	3 weeks gasoline fume exposure
6	D1	No treatment	2 weeks gasoline fume exposure
7	D2	No treatment	3 weeks gasoline fume exposure

Table 1. Experimental grouping and treatment schedule.

All antioxidant treatments were administered by oral gavage commencing immediately after the conclusion of the gasoline fume exposure period.

2.6 Blood Collection and Biochemical Assays

Twenty-four hours after the final dose of antioxidant or the final gasoline exposure session, animals were anaesthetized with mild chloroform. Blood was collected via ocular puncture using heparinized capillary tubes following the method of Parasuraman et al. [17], transferred into plain sample tubes, allowed to clot for 5 minutes, and centrifuged at 3000 rpm for 20 minutes. The resulting serum was aspirated and used for all biochemical assays.

Serum MDA concentration was determined by the thiobarbituric acid reactive substances (TBARS) method of Aguilar Diaz De Leon and Borges [18]. SOD activity was measured by the validated spectrophotometric method of Yalcin et al. [19]. GSH concentration was quantified using a UV-VIS spectrophotometer (Model 752G) following the method of Ahmed et al. [20]. Catalase activity was determined using a UV-VIS spectrophotometer (Model 752N) following the standard protocol of Hadwan and Abed [21]. All assays were performed in duplicate and results expressed per the respective standard unit for each parameter.

2.7 Statistical Analysis

Data are presented as mean ± standard error of the mean (SEM). Body weight data were analysed using a paired t-test comparing initial and final weights within each group. Relative organ weights, MDA, SOD, catalase, and GSH were compared across groups using one-way analysis of variance (ANOVA) followed by Fisher's least significant difference (LSD) post-hoc test. All analyses were performed using GraphPad Prism 9.5.1, with statistical significance set at $p<0.05$. An F-ratio is reported for each biochemical variable. Significance indicators: * $p<0.05$ vs Group A (control); ˆ significant vs Group A (B-groups); a significant vs Group A (C-groups).

3 RESULTS

3.1 Effect on Body Weight

Table 2 presents initial and final body weights, weight differences, and paired t-test statistics for all groups. All groups demonstrated significant within-group weight gain over the study period ($p<0.05$), with the exception of Group C2 ($p=0.083$). The greatest absolute weight gain was recorded in Group C1 (quercetin, three-week exposure: 102.80 g), followed closely by Group B1 (quercetin, two-week exposure: 99.20 g), suggesting a growth-supportive effect of quercetin supplementation. Group A (control) gained 45.90 g and Group D1 (two-week exposure, untreated) gained 43.60 g, the two lowest gains among all groups. Group D2 (three-week exposure, untreated) recorded a larger gain of 94.87 g despite the longest gasoline fume exposure duration without treatment.

Table 2: Effect of quercetin and curcumin on body weight of male Wistar rats following gasoline fume exposure (mean ± SEM).

Group	Initial Weight (g) Mean ± SEM	Final Weight (g) Mean ± SEM	Weight Diff. (g)	p-value	t-value
A (Control)	213.60 ± 5.94	259.50 ± 7.23	45.90	0.002*	4.956
B1 (GF 2wk + Quercetin)	210.80 ± 5.65	310.00 ± 10.00	99.20	0.001*	9.154
B2 (GF 2wk + Curcumin)	227.75 ± 9.15	287.00 ± 6.22	59.25	0.002*	5.356
C1 (GF 3wk + Quercetin)	217.20 ± 11.38	320.00 ± 24.00	102.80	0.006*	4.491
C2 (GF 3wk + Curcumin)	196.00 ± 12.78	227.50 ± 58.50	81.50	0.083	2.166
D1 (GF 2wk, no treatment)	225.60 ± 4.24	269.20 ± 3.80	43.60	0.001*	7.647
D2 (GF 3wk, no treatment)	165.80 ± 7.90	260.67 ± 17.37	94.87	0.001*	5.753

* $p<0.05$, paired t-test (initial vs final weight within group). GF = gasoline fume; wk = weeks.

3.2 Effect on Serum MDA Level

Serum MDA results are presented in Table 3. Untreated gasoline-exposed groups D1 (0.26 ± 0.00 nmol/ml) and D2 (0.25 ± 0.00 nmol/ml) showed significantly elevated MDA concentrations compared with the control group A (0.11 ± 0.00 nmol/ml), confirming gasoline fume-induced lipid peroxidation. Post-exposure quercetin treatment reduced MDA to 0.15 ± 0.00 (B1) and 0.16 ± 0.00 nmol/ml (C1), while curcumin reduced it to 0.16 ± 0.00 (B2) and 0.17 ± 0.00 nmol/ml (C2), all significantly different from the control. Quercetin achieved a marginally lower MDA than curcumin at both exposure durations. The overall F-ratio of 477.73 confirms highly significant group differences.

Table 3: Effect of quercetin and curcumin on serum MDA level in male Wistar rats following gasoline fume exposure (mean ± SEM).

Group	MDA Level (nmol/ml) Mean ± SEM
A (Control)	0.11 ± 0.00
B1 (GF 2wk + Quercetin 50 mg/kg)	0.15 ± 0.00 ^
B2 (GF 2wk + Curcumin 50 mg/kg)	0.16 ± 0.00 ^
C1 (GF 3wk + Quercetin 50 mg/kg)	0.16 ± 0.00 a
C2 (GF 3wk + Curcumin 50 mg/kg)	0.17 ± 0.00 a
D1 (GF 2wk, no treatment)	0.26 ± 0.00 *
D2 (GF 3wk, no treatment)	0.25 ± 0.00 *
F-ratio	477.73

* $p<0.05$ vs Group A (control); ^ significant vs Group A (B-groups); a significant vs Group A (C-groups). GF = gasoline fume; wk = weeks.

3.3 Effect on Serum SOD, Catalase, and GSH Activities

Gasoline fume exposure significantly reduced SOD, catalase, and GSH levels in untreated groups ($p<0.05$), with 3-week exposure causing greater suppression than 2-week exposure. Post-exposure quercetin treatment produced the closest recovery to control, while curcumin was less effective, particularly in restoring catalase after 3-week exposure.

Table 4: Effect of quercetin and curcumin on serum SOD, catalase, and GSH activities in male Wistar rats following gasoline fume exposure (mean ± SEM).

Group	SOD (U/l) Mean ± SEM	Catalase (U/l) Mean ± SEM	GSH (U/ml) Mean ± SEM
A (Control)	20.72 ± 0.60	83.70 ± 0.31	16.22 ± 0.31
B1 (GF 2wk + Quercetin)	18.79 ± 0.06	81.50 ± 0.87	14.91 ± 0.02
B2 (GF 2wk + Curcumin)	18.32 ± 0.18	80.14 ± 0.51	14.45 ± 0.25
C1 (GF 3wk + Quercetin)	18.35 ± 0.39 a	79.33 ± 1.72 a	14.59 ± 0.22 a
C2 (GF 3wk + Curcumin)	16.89 ± 1.09 a	71.28 ± 4.52 a	14.04 ± 0.28 a
D1 (GF 2wk, no treatment)	17.76 ± 0.60 *	77.37 ± 1.19 *	14.03 ± 0.35 *
D2 (GF 3wk, no treatment)	14.79 ± 0.56 *	71.58 ± 0.55 *	12.66 ± 0.57 *
F-ratio	9.535	6.105	10.74

* $p<0.05$ vs Group A (control); a significant vs Group A (C-groups). GF = gasoline fume; wk = weeks.

4 DISCUSSION

The present study demonstrates that sub-chronic inhalation of leaded gasoline fumes (RON 80) in male Wistar rats induces significant, duration-dependent oxidative stress, evidenced by markedly elevated serum MDA concentrations and concurrent significant reductions in SOD, catalase, and GSH activities in the untreated positive control groups (D1 and D2). These biochemical derangements align with the established toxicological understanding that volatile aromatic hydrocarbons in gasoline fumes, particularly benzene and its alkyl derivatives, undergo pulmonary and hepatic cytochrome P450-mediated biotransformation to reactive epoxide and quinone metabolites that generate superoxide radicals and initiate lipid peroxidation cascades targeting polyunsaturated fatty acid-rich cellular membranes [4,6].

The significant elevation in MDA in exposed groups confirms that lipid peroxidation is a central mechanism of gasoline-fume-induced cellular damage. The greater MDA elevation in D1 (0.26 ± 0.00 nmol/ml) compared with D2 (0.25 ± 0.00 nmol/ml) is notable and may reflect increased antioxidant enzyme consumption and partial compensatory responses in the three-week exposure group; however, the concurrent greater enzyme suppression in D2 suggests an overall more severe oxidative state in the longer exposure group, consistent with the pattern reported by Abdullahi et al. [22] in a comparable rodent gasoline fume inhalation model, and with oxidative marker derangement reported in a Wistar rat model of chronic cigarette smoke inhalation [7]. The simultaneous depletion of GSH reflects its heightened utilization in conjugating electrophilic metabolites via glutathione transferase and in supporting glutathione peroxidase-mediated lipid hydroperoxide neutralization, while SOD and catalase suppression indicates progressive overwhelm of the enzymatic antioxidant scaffolding with increasing duration of exposure [8,9].

Post-exposure administration of quercetin at 50 mg/kg significantly attenuated MDA and restored SOD, catalase, and GSH activities in both the two-week and three-week exposure groups (B1 and C1), with the two-week quercetin group (B1) demonstrating the closest biochemical recovery to control values across all four parameters. Quercetin's antioxidant efficacy in this context is mechanistically consistent with its capacity to directly scavenge superoxide radicals and hydroxyl radicals through its catechol B-ring and 3-hydroxyl functional groups, while concurrently activating the Nrf2/antioxidant response element transcriptional pathway to drive upregulation of SOD, catalase, glutathione peroxidase, and glutamate-cysteine ligase, the rate-limiting enzyme in de novo GSH biosynthesis [11,12]. The metal-chelating capacity of quercetin additionally limits Fenton-type hydroxyl radical generation by sequestering redox-active iron and copper, further reducing the oxidant burden at source [12].

Curcumin at 50 mg/kg similarly produced significant reductions in MDA and partial restoration of SOD, catalase, and GSH in groups B2 and C2; however, the recovery of catalase in Group C2 (71.28 ± 4.52 U/l) was not significantly different from the untreated D2 group (71.58 ± 0.55 U/l), indicating insufficient curcumin-mediated protection against catalase suppression following three weeks of gasoline fume exposure at this dose. This differential outcome compared with quercetin is likely attributable to the well-documented pharmacokinetic limitations of curcumin, including poor aqueous solubility, rapid phase II hepatic metabolism via glucuronidation and sulfation, and intestinal P-glycoprotein efflux, all of which reduce the effective serum and tissue concentration achievable at 50 mg/kg in rodents [14]. Despite

this, curcumin demonstrated meaningful ameliorative activity across most parameters, consistent with the reports of Abd El-Hady et al. [22] and Rahmani et al. [15] showing significant curcumin-mediated antioxidant protection in rodent models of environmental oxidant exposure.

The overall pattern of quercetin outperforming curcumin at an equivalent dose across several biochemical endpoints in this study has practical implications for antioxidant intervention strategies in occupationally exposed populations. Both compounds engage the Nrf2 and NF-κB pathways, but structural differences in their polyphenolic backbone confer different ROS-scavenging efficiencies and pharmacokinetic profiles. Future investigations employing higher curcumin doses, nanoformulations, phospholipid complexes, or piperine co-administration to enhance curcumin bioavailability may achieve outcomes more comparable to quercetin, and a combined quercetin-curcumin regimen may generate synergistic protection not captured by the single-agent arms of the present design [23].

Limitations of this study include evaluation of only a single dose level for each antioxidant, which precludes full characterization of dose-response relationships. Additionally, the biochemical assessments were confined to serum parameters; tissue-level measurements of oxidative stress in pulmonary homogenates, as well as molecular assessments of Nrf2 and NF-κB pathway activation, would provide more mechanistically complete insight. These represent priorities for future studies in this research line.

5 CONCLUSION

Sub-chronic inhalation of leaded gasoline fumes induces significant duration-dependent oxidative stress in male Wistar rats, characterized by elevated serum MDA and suppressed antioxidant enzyme activities. Post-exposure oral supplementation with quercetin or curcumin at 50 mg/kg significantly attenuated these biochemical perturbations, with quercetin demonstrating superior overall antioxidant efficacy compared with curcumin under the experimental conditions employed. These findings support the therapeutic potential of dietary polyphenols, particularly quercetin, as post-exposure protective agents against the oxidative consequences of petroleum fume inhalation, and provide a scientific basis for further investigations employing optimized dosing regimens, combined polyphenol protocols, and molecular mechanistic endpoints.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

Acknowledgments

The authors gratefully acknowledge the technical support of the Animal House Facility staff, Department of Human Physiology, Faculty of Basic Medical Sciences, Chukwuemeka Odumegwu Ojukwu University, Uli Campus.

Funding Source

The authors declare that no grants, financial support, or sponsorship was received for this research. The entire study was self-funded by the authors.

Disclosure of conflict of interest

The authors declare no conflict of interest.

Statement of ethical approval

Ethical approval was obtained from the Institutional Animal Ethics Committee of Chukwuemeka Odumegwu Ojukwu University, Uli Campus, prior to commencement of the study. This study did not involve human participants.

REFERENCES

[1] Agency for Toxic Substances and Disease Registry. Toxicological profile for automotive gasoline. Washington (DC): U.S. Department of Health and Human Services; 2025.

[2] Masuk NI, Hossen I, Islam MN. Health and environmental impacts of leaded gasoline: current scenario and future challenges. J Hazard Mater. 2021;401:123458.

[3] Alses M, Alzeer S. Evaluation of some biological parameters of gasoline station attendants in Damascus, Syria. Heliyon. 2021;7:e07056.

[4] Abdrabouh AE. Inflammatory and proapoptotic effects of inhaling gasoline fumes on the lung and ameliorative effects of fenugreek seeds. Sci Rep. 2022;12:14446.

[5] Halliwell B, Gutteridge JMC. Free radicals in biology and medicine. 5th ed. Oxford: Oxford University Press; 2015.

[6] Adeyemi OS, Olayaki LA. Petroleum fumes exposure and oxidative stress responses in Wistar rats. Environ Toxicol Pharmacol. 2023:129-32.

[7] Afuberoh FC, Umeji JC, Anyaogu CC, Okoye OF. "Impacts of chronic cigarette smoke exposure on lung histology and oxidative stress markers in adult male Wistar rats." Eur J Biomed Pharm Sci. 2026;13(1):100–106. doi:10.5281/zenodo.18094443

[8] Yuan J, Zhao S, Li Y. Catalase as a therapeutic target in oxidative stress-related diseases. Free Radic Biol Med. 2021;170:214-26.

[9] Islam MT, Ali ES, Das AK. Superoxide dismutase in health and disease. Int J Biol Macromol. 2022;138:649-57.

[10] Akinpelu M, Olayanju A, Adeniyi TD, Atere AD, Moronkeji AI, Igunbor MC, et al. Oxidative stress response to gasoline generator exhaust emission in adult male Wistar rats. Environ Anal Health Toxicol. 2024:24-9.

[11] Li Y, Yao J, Han C, Yang J, Chaudhry MT, Wang S, et al. Quercetin, inflammation, and immunity. Nutrients. 2016;8(3):167.

[12] Kumar V, Pandey A, Sharma C. Quercetin as a protective phytochemical against environmental toxicant-induced tissue injury: mechanisms and evidence. Phytother Res. 2020;34(11):2846-62.

[13] Al-Zharani M, Mubarak M, Rudayni HA. Quercetin alleviates oxidative stress induced by toxicants in male Wistar rats. Nutrients. 2023;15(8):1888.

[14] Hewlings SJ, Kalman DS. Curcumin: a review of its effects on human health. Foods. 2017;6(10):92.

[15] Rahmani AH, Alsahli MA, Aly SM, Khan MA, Aldebasi YH. Role of curcumin in disease prevention and treatment. Adv Biomed Res. 2018;7:38.

[16] Uboh FE, Akpanabiatu MI, Alozie Y, Ebong PE, Ufot U. Comparative evaluation of effect of vitamins E and C on gasoline vapour-induced hepatotoxicity and hormonal changes in rats. Internet J Toxicol. 2005;4(1).

[17] Parasuraman S, Raveendran R, Kesavan R. Blood sample collection in small laboratory animals. J Pharmacol Pharmacother. 2010;1(2):87-93.

[18] Aguilar Diaz De Leon J, Borges CR. Evaluation of oxidative stress in biological samples using the thiobarbituric acid reactive substances assay. J Vis Exp. 2020;(159):e61122.

[19] Yalcin M, Karakoc Y, Akyuz F. A validated spectrophotometric method for serum superoxide dismutase activity measurement. J Clin Biochem Nutr. 2020;66(3):198-204.

[20] Ahmed OM, Ahmed RR, El-Gareib AW. Antioxidant and anti-inflammatory roles of quercetin in ameliorating oxidative tissue injury. Biomed Pharmacother. 2021;140:111738.

[21] Hadwan MH, Abed HN. Data supporting the spectrophotometric method for the estimation of catalase enzyme activity. Data Brief. 2016;6:194-9.

[22] Abdullahi S, Wali CC, Ochayi MO, Archibong BE. Effects of gasoline fume inhalation on oxidative stress biomarkers and pulmonary histopathology in Wistar rats. Calabar J Sci Technol. 2025;7(2):264-71.

[23] Abd El-Hady AM, Azzoz RM, Soliman SM, Abdelrahman IY, Khalil WM. Protective effects of curcumin and quercetin in irradiated rats. Histochem Cell Biol. 2024;162:299-309.