Comparative Nephrotoxicity and Hepatotoxicity Effects of Kerosene, Gasoline, Liquefied Petroleum Gas and Biomass Fuel Exposure on Male Albino Rats

Ude Tochukwu1*, Meludu Samuel Chukwuemeka1,2,3, Dioka Chudi Emmanuel1, Chikezie Onyebuchi Desmond2,4, Awalu Chimezie Joseph2 and Ibekailo Sylvester Nnaemeka5

1Department of Chemical Pathology, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria.
2Chemical Pathology Unit, Department of Medical Laboratory Science, Faculty of Health Science and Technology, College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria.
3Department of Human Biochemistry, Faculty of Basic Medical Sciences, College of Health Sciences, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria.
4Department of Chemical Pathology, Federal Medical Centre Umuahia, Abia State, Nigeria.
5Department of Physiology, Alex Ekwueme Federal University, Ndufu-Alike Ikwo, Ebonyi State, Nigeria.

Authors’ contributions

This work was carried out in collaboration among all authors. Authors UT and MSC designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors DCE and COD managed the analyses of the study. Authors ACJ and ISN managed the literature searches. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/AJRB2020/v7i430147

Editor(s): (1) Dr. Mohamed Fawzy Ramadan Hassanien, Zagazig University, Egypt.
Reviewers: (1) Ioana Stanciu, University of Bucharest, Romania.
(2) Waleed Khalid Abduljabbar, University of Fallujah, Iraq.
(3) Rama Bhat, Alva’s College, India.
Complete Peer review History: http://www.sdiarticle4.com/review-history/62523

Received 22 August 2020
Accepted 29 October 2020
Published 26 November 2020

*Corresponding author: E-mail: udetochukwu@yahoo.com;
ABSTRACT

The effects of Kerosene, gasoline, and liquefied petroleum gas and biomass fuel exposure on biomarkers of kidney and liver were investigated in male wistar rats. Fifty adult male wistar rats were randomly assigned to five groups of ten animals each. Rats in group A served as control (exposed to fresh air). Group B, C, D and E were exposed to inhalation of kerosene, gasoline, liquefied petroleum gas and biomass fuel (wood smoke) respectively. All the exposures were done using whole body exposure chambers 70 cm x 60 cm x 60 cm measurement for six weeks, 6 days per week. Five millilitres of blood sample were collected and serum extracted at the end of six weeks. The serum concentration of urea, creatinine, uric acid and activities AST, ALT, γGT were determined using Cobas reagent kits manufactured by Roche Diagnostics GmbH, Sandhofer Strasse 116, D-68305 Mannheim, Germany. Values were analysed statistically using SPSS version 23.0. The result shows significant increase in the serum levels of urea, creatinine and uric acid of test groups relative to control (p<0.05), though the effect appear to be more pronounced with exposure to kerosene, gasoline and biomass fuel. The exposure also led to significant increase in activities of AST, ALT and γGT (p<0.05). These results suggest that repeated exposure to kerosene, gasoline and liquefied petroleum gas and biomass fumes may elicit hepatic and renal toxicity, thereby impairing the normal liver and kidney function.

Keywords: Kerosene; gasoline; liquefied petroleum gas; biomass fuel; biomarkers; exposure.

1. INTRODUCTION

Gasoline, Liquefied petroleum gas, kerosene and biomass are frequently used products for the internal combustion of car engines, energy generation and for operating machines. They are also used in homes as lighting and cooking fuels. These commodities contain predominantly hydrocarbons and have been reported to have significant adverse health effects [1,2]. Pollutants from these products may be metabolically transformed into different metabolites in the body [3]. Metabolites from these products may be highly reactive, thereby interacting in different ways with the excreting and metabolizing tissues (mainly the liver and kidneys) to exert its toxic effects [4]. In Nigeria, due to the unreliable public power supply, many homes, offices and businesses rely greatly on small electric generators, biomass and liquid fuel-dependent machines for day to day activities. The fuel for operating these machines are purchased and stored in homes and offices where their fumes constitute environmental health hazards especially during refuelling. The use of kerosene and biomass in homes as cooking and lighting fuels has further increased the exposure of humans to these products [1,5]. More so, the current trend of locating filling and gas stations in residential areas exposes the residents of those areas to its fumes. It has also been noticed that the owners of filling stations attach well designed and built duplexes or bungalows to their filling stations. This also exposes the occupants of these apartments to petrol fumes. Apart from these, abuse of petroleum products as therapeutic agents for the treatment of snake bites, convulsion, arthritis, gastro-intestinal disorders and a host of other conditions has been reported [6,7]. In a developing country like Nigeria who depends mostly on crude oil exploration for her foreign earning, there is gross non-compliance to strict measures being put in place by the Government to checkmate pollution of the environment by petroleum products. The companies that explore crude oil in some parts of Niger-Delta, Nigeria pollute the most of the rivers and farms, thereby destroying the major source of living for the people who make a living by fishing. It has been reported that consumption of aquatic animals exposed to spillage possess the risk of possible bioaccumulation and bio-concentration of toxic components of petroleum [8]. There has been several cases of explosion of crude oil pipe lines due to lack of maintenance, resulting in the pollution of the immediate environment with its fractions. Individuals who are occupationally exposed like those who work in petroleum industries or with petroleum distillates are likely to be more affected than their counterparts who do not work in these industries [9,10]. Automobile mechanics, LPG pump attendants, cooks, petrol station pump attendants and petrol tanker drivers belong to this class of people. Unfortunately, they handle these products without proper protection against their possible harmful effects [5,11]. It is pertinent to evaluate the toxic potentials of these products in
terms of alteration in kidneys and liver biomarkers. Hence, this study was designed to comparatively evaluate the effect of exposure to these products on activities of AST, ALT, GGT and serum levels of urea, creatinine and uric acid of male albinos rats.

2. MATERIALS AND METHODS

2.1 Experimental Animals

Fifty adult male albinos’ rats (wistar strain) seven weeks old, that weighed 130±10 g obtained from the Animal Breeding Unit of the Faculty of Veterinary Medicine, University of Nigeria, Nsukka, were used as the experimental animals. The rats were kept in cages for two weeks allowed to acclimatize to Animal House of the College of Health Sciences, Nnamdi Azikiwe University, Nnewi campus and were allowed free access to food and water ad libitum. Thereafter, the animals were randomly (while controlling for weight differences) distributed into five groups of ten animals each based on the exposed compound.

2.2 Petroleum Products and Biomass

The petroleum products; gasoline, kerosene and Liquefied petroleum gas were purchased from Juhel filling station at Awka, Anambra State, Nigeria, while the biomass Melina wood (G. arborea) was purchased from commercial wood seller at Nnewi City, Anambra State, Nigeria.

2.3 Design of Exposure Chamber and Wood Combustor

The exposure chamber was fabricated using plywood (China OSB), while the wood combustor was made using iron, with an outlet for the release of the smoke into the chamber and a square oxygen inlet on the lid (10 cm x10 cm). The exposure chamber measures 70 cm x 60 cm x 60 cm as previously described by Uboh et al. [1] and Akintunde et al. [12], was constructed to allow at least 20% ventilation, as stipulated by the Organisation for Economic Co-operation and Development guidelines for inhalation toxicity. The burning process was initiated first before exposure by igniting 4 kg Gmelina arborea wood with a lighter in a fabricated combustor. The wood smoke was diverted from the combustor into the exposure chamber using a metal host.

2.4 Experimental Design

The fifty (50) adult male wistar rats obtained from the animal breeding unit of the faculty of Veterinary Medicine, University of Nigeria, Nsukka, after two weeks of acclimatization to the animal house were randomly (while controlling for weight differences) assigned to five groups of ten animals each designated as A, B, C, D and E. Rats in group A served as control (exposed to fresh air). Group B, C, D and E were exposed to inhalation of kerosene, gasoline, liquefied petroleum gas and biomass smoke (wood smoke) respectively. All the exposures were done using whole body exposure chambers 70 cm x 60 cm x 60 cm measurement. The animals in group A were exposed to fresh air only. Animals in group B and group C were exposed by placing the cages housing the animals into respective exposure chambers of measurement 70 cm x 60 cm x 60 cm, each containing two open calibrated beakers of 500 cm³ containing 500 cm³ of liquid Kerosene for group B and gasoline for group C respectively. The kerosene and gasoline were allowed to evaporate freely within the respective exposure chambers at ambient humidity and temperature. Animals in group B were exposed to vapours (20.2 ± 4.5 cm³ h⁻¹ Kg⁻¹m⁻³ day⁻¹) generated from direct evaporation of the liquid Kerosene, while animal in group C were exposed to vapours (30.8 ± 5.1 cm³ h⁻¹ Kg⁻¹m⁻³ day⁻¹) generated from direct evaporation of the liquid gasoline. The animals were exposed 5 h/day (9.00 a.m - 2.00 p.m), 6 day/week, to vapours for 6 weeks. During the exposure period, the initial and final volumes of liquid Kerosene and gasoline were recorded before and after daily exposure. The daily differences in volume were used to calculate relative concentrations of vapours used in this group. Group D were exposed to liquefied petroleum gas from a compressed gas cylinder with pressure gauge and mass flow controller or flow meter in a whole body exposure chamber 70 cm x 60 cm x 60 cm for 5 h/day (9.00 a.m - 2.00 p.m), 6 day/week, to LPG vapours for 6 weeks. Animals in group D were exposed to LPG vapours (202.2 ± 4.5 g h⁻¹ Kg⁻¹m⁻³ day⁻¹) generated from direct evaporation of the LPG. The initial and final volumes of LPG were recorded before and after daily exposure. The daily differences in volume were used to calculate relative concentrations of LPG used. Group E were exposed to biomass smoke by igniting 4 kg Gmelina arborea wood with a lighter in a fabricated combustor. The wood smoke was diverted from the combustor into the exposure chamber.
chamber using a metal host. The animals in group E were exposed to 1005 ug/m³ of PM₁₀ from wood smoke for 1 h/day, 6 day/week for 6 weeks. The PM concentration in the chamber was monitored using a PM₁₀ monitor (portable particulate monitor PCE-RCM 10, PCE Deutschland GmbH). At the end of each exposure day, the animals were transferred vapours-free section of the experimental animal house. Body weight of animals and mortality data were routinely monitored. The study lasted for 6 weeks and blood samples were collected at the end 6 weeks of exposure by cardiac puncture for the biochemical analysis.

2.5 Collection of Blood Sample

The animals were anesthetized with chloroform 24 h after the last exposure and blood samples collected by cardiac puncture into plain sample tubes. Serum samples were separated 1 h after extraction of blood by centrifugation at 3000 g for 10 mins and stored in at -30°C. Biochemical analyses on the serum samples were done 24 h after sample collection. Biochemical analyses were carried out for the measurement of serum activities of alanine (ALT), aspartate aminotransferases (AST), Gamma-glutamyl transferase (γGT) and serum levels of Urea, Creatinine and Uric Acid.

2.6 Biochemical Analysis

The biochemical parameters were determined using Cobas reagent kits manufactured by Roche Diagnostics GmbH, Sandhofer Strasse 116, D-68305 Mannheim, Germany. Serum AST and ALT activities was determined by the method of Bergmeyer et al. [13] and γGT by Persijn et al. [14]. Creatinine, urea and uric acid were estimated by the Jaffé reaction, diacetyl monoxime oxidase and phosphotungstate methods respectively. The concentrations of the biochemical parameters were measured using Mindray BS 120 analyser.

2.7 Statistical Analysis

Data collected were subjected to analysis of Variance (ANOVA). In order to test whether or not significant differences existed between groups. Pairwise comparisons were made using the Post-hoc test. The mean±SD of each parameter was taken for each group. Test probability value of p<0.05 was considered significant. The analyses were carried out on SPSS for Windows version 23.0.

3. RESULTS

Table 2 shows the comparison of serum levels of Urea, Creatinine and uric acid after 6 weeks exposure to Kerosene, gasoline, LPG and biomass smoke. The mean Urea level was highest in group B and lowest in control. The mean Urea value of the test groups B, C, D and E compared to control were statistically significantly (p=.001, .001, .032 and .001 respectively). The mean Urea value of test group B compared to groups C and E were statistically similar (p=.026). More so, the mean uraea value of test group C compared to groups D and E, and test group D compared to group E were statistically similar (p=.152, .647 and .076). The mean creatinine level was highest in group E and lowest in control. The mean creatinine value of the test groups B, C and E compared to control were statistically significant (p=.002, .001,.001 respectively), while the mean creatinine values of test groups D compared to control were statistically similar (p=.165). The mean creatinine values of test groups compared together were statistically similar (p=.89, .08, .76, .06, .85, .05). Furthermore, the mean Uric Acid level was highest in group C and lowest in control. The mean Uric Acid value of test groups B, C, D and E compared to control were significantly significant (p=.001, .001, .01 and .001 respectively). The mean uric acid value of test group B compared to C were statistically significant (p=.02), while mean uric acid value of test group B compared to groups D and E were statistically significant (p=.039 and .014), while the mean uric acid value of test group D compared to group E were statistically similar (p=.88).

Table 3 depicts the comparison of activities of AST, ALT and γGT after 6 weeks of exposure to Kerosene, gasoline, LPG and biomass smoke. The mean AST activity was highest in group C and lowest in control. The mean AST activities of the test groups B, C, D and E compared to control were statistically significantly (p=.001,.001,.006 and .001 respectively). The mean AST activity of test group B compared to test group C were statistically significant (p=0.017), while the mean AST activity of test group B compared to groups D and E were statistically similar (p=.923 and .264). More so, the mean AST activity of test group C compared
Table 1. Summary of exposures

| Groups          | Exposures                                      |
|-----------------|------------------------------------------------|
| Group A (control) | Exposed to fresh air                         |
| Group B (Kerosene) | Exposed to 20.2 ± 4.5 cm h-1 Kg-1m-3 day-1 kerosene |
| Group C (Gasoline) | Exposed to 30.8 ± 5.1 cm h-1 Kg-1m-3 day-1 gasoline |
| Group D (LPG)    | Exposed to 202.2 ± 4.5 g h-1 Kg-1m-3 day-1 LPG  |
| Group E (Biomass) | Exposed to 1005± 6.3 ug/m³ h-1 Kg-1 of biomass smoke |

Table 2. Effect of petroleum products and biomass fuel exposure on kidney biomarkers

| Parameters      | A(control) | B(Kerosene) | C(gasoline) | D(LPG) | E(Biomass) | P-value |
|-----------------|------------|-------------|-------------|--------|------------|---------|
| Urea            | 3.96±0.39  | 6.05±0.98ᵃ  | 5.64±1.14ᵃ  | 4.97±1.11ᵃ  | 5.85±1.01ᵃ  | 0.001   |
| Creatinine      | 33.90±8.84 | 54.22±9.33ᵃ | 55.00±14.95ᵃ | 42.75±17.79 | 56.12±13.87ᵃ | 0.002   |
| Uric Acid       | 2.35±0.45  | 5.62±1.13ᵃ  | 7.08±1.24ᵃ  | 5.80±1.71ᵃ  | 5.53±1.57ᵃ  | 0.001   |

P-value is significant at p<0.05.ᵃ = significantly higher compared to control.ᵇ = significantly higher compared to group B.ᶜ = significantly higher compared to group C.ᵈ = significantly higher compared to group D.ᵉ = significantly higher compared to group E. Units of measurements: Urea= mmol/l, creatinine=umol/l, uric acid=mg/dl

Table 3. Effect of petroleum products and biomass fuel exposure on liver biomarkers

| Parameters | A( Control) | B(Kerosene) | C(gasoline) | D(LPG) | E(Biomass) | P-value |
|------------|-------------|-------------|-------------|--------|------------|---------|
| AST        | 38.60±10.95 | 73.33±10.88ᵃ| 95.60±24.01ᵇ | 74.25±17.06ᵇ | 84.00±28.63ᵇ | 0.001   |
| ALT        | 37.70±8.62  | 54.22±9.33ᵃ | 92.50±16.93ᵇ | 73.75±17.52ᵇ | 99.00±10.04ᵇ | 0.001   |
| γ-GT       | 24.30±5.63  | 55.77±8.55ᵃ | 64.20±11.00ᵃ | 58.75±13.62ᵃ | 87.57±7.44ᵃ | 0.001   |

P-value is significant at p<0.05.ᵃ = significantly higher compared to control.ᵇ = significantly higher compared to group B.ᶜ = significantly higher compared to group C.ᵈ = significantly higher compared to group D.ᵉ = significantly higher compared to group E

Exposure to petroleum products and biomass smoke is widespread and frequent in our environment as man has invented a lot of equipment that makes use of these products in order to make his life better. Exposures to these products are considered environmental toxicant with attendant health effects [15]. This study evaluated comparatively the effects of persistent exposure to kerosene, gasoline, liquefied petroleum gas and biomass smoke on serum levels of urea, creatinine, uric acid and serum activities of AST, ALT and GGT of male albino wistar rats. The result showed that exposure to kerosene, gasoline, liquefied petroleum gas and biomass smoke significantly increase serum...
levels of urea, creatinine and uric acid which are indicator of kidney impairment. This effect appears to be more pronounced with exposure to gasoline, kerosene and biomass smoke. The kidney functions in maintaining homeostasis in the body by the reabsorption of important materials and excretion of waste products. Diseases of the kidney which diminish the glomerular filtration can lead to the retention of urea, creatinine and uric acid. The increase in urea, creatinine and uric acid found in this study could be attributed to high lipophilicity of petroleum compound and induction of oxidative stress. Petroleum products has been reported to bind to the membrane lipid bilayer and proteins due to its high lipophilicity, leading to damage to the membrane bilayer and proteins, which causes inhibition of Na+/K+/ATPase activity, which in turn leads to disruption of ion homeostasis and cell injury [16]. It could also be due to biotransformation of petroleum and particulate compound to reactive intermediates that bind covalently to macromolecules and in turn alter their activity resulting in cell injury [4,17]. More so, induction of oxidative stress by petroleum products oxidative metabolites has also been reported to cause disruption of the immune system, including induction of autoimmunity, effects on T-cell function, suppression of immunofunction, increased post-glomerular resistance, and decreased glomerular filtration rate (GFR) [16]. The increase in urea, creatinine and uric acid found in this study agrees with previous reports of Uhegbu et al. [18] and Uboh et al. [19]. Alanine transaminase (ALT), Aspartate transaminase (AST) and Gamma glutamyl transferase (γ-GT) activities have been used in the diagnosis hepatic injury. The activity of these enzymes is known to increase in the liver and serum as a result of hepatic damage or injury [20]. The result of this study also showed that exposure to kerosene, gasoline, liquefied petroleum gas and biomass smoke significantly increased serum activities of ALT, AST and γ-GT which is indicative of possible hepatocellular injury. The increase in activities of ALT, AST and γ-GT found in this study could be attributed to induction of oxidative stress by petroleum metabolites and particulate compounds. The metabolism of petroleum compounds has been reported to generate reactive metabolites which interact with the membrane lipids of hepatocytes to produce lipid peroxide and ROS leading to lipid peroxidation and damage to the hepatic membrane and tissues; hence leakage of hepatic enzymes [21,17]. This effect could also be attributed to induction of cellular degeneration and down regulation of gene expression. Hydrocarbons from petroleum products and biomass has been reported to cause down regulation or impairment of CYP450 enzyme activity leading to decreased bile acid-independent bile flow and accumulation of cholestatic reactions, which subsequently increase serum activities of ALP, γ-GT, AST and ALT [22,23,24]. The increase in activities of γ-GT, AST and ALT found in this study agrees with previous reports of Uboh et al. [1], Patrick-Iwuanyanwu et al. [6], Odunola et al. [25], Ravnskov [26], Awodele et al. [27] and Iyanda [28].

5. CONCLUSION

In conclusion, the results of this work suggest that repeated exposure to kerosene, gasoline, and liquefied petroleum gas and biomass fumes may elicit hepatic and renal toxicity, thereby impairing the normal liver and kidney function. Petroleum workers and those occupationally exposed to biomass smoke should ensure appropriate use of personal protective equipment, in addition should have regular medical check-up to ascertain their health condition.

ETHICAL APPROVAL

The protocol was in line with the guidelines of the National Institute of Health (NIH) (NIH Publication 85-23, 1985) for laboratory animal care and use.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Uboh FE, Akpanabiatu MI, Eyong EU, Ebong PE, Eka OO. Evaluation of toxicological implications of inhalation exposure to kerosene fumes and petrol fumes in rats. Acta Biol Szeged. 2005;49(3-4):19-22.
2. Okoye JO, Ude T, Ibekaike SN, Awalu CJ. Histopathological, hemochromatotic, hypercholesterolemic, and androgenic effects of escravos crude oil on the testis in male chinchilla rabbits. Bri. J. Biotechnol. 2014;4(6):649-658.
3. Hu Z, Wells PG. Modulation of benzo (a) pyrene bioactivation by glucuronidation in
lymphocytes and hepatic microsomes from rats with a hereditary deficiency in bilirubin UDP-glucuronosyl transferase. Toxicol. Appl. Pharmacol. 1994;127:306-313.

4. Nygren J, Cedewal B, Erickson S, Dusinska M, Kolman A. Induction of DNA strand breaks by ethylene oxide in human diploid fibroblasts. Environ. Mol. Mutagen. 1994;24:161-167.

5. Oluwole O, Arinola GO, Huo D, Olopade CO. Biomass fuel exposure and asthma symptoms among rural school children in Nigeria. J Asthma. 2017b;54:347-356.

6. Patrick-Iwuanyanwu KC, Onyemaenu CC, Wegwu MO, Ayalogu EO. Hepatotoxic and nephrotoxic effect of kerosene and petrol contaminated diets in wistar albino rats. J. Environ. Toxicol. 2011;5(1):49-57.

7. Chilcott RP, Chapd HQ. Summary of health effects of diesel. Sci. Total Environ. 2007;20:129-138.

8. Eyang EU, Umoh IB, Ebong PE, Eteng MU, Antai AB, Akpa AO. Haemotoxic effects following ingestion of Nigerian crude oil and crude oil polluted shellfish by rats. Nig J. Physiol. Sci. 2004;19:1-6.

9. Okoro AM, Ani EJ, Ibu JO, Akpogomeh BA. Effect of petroleum products inhalation on some haematological indices of fuel attendants in Calaber Metropolis, Nigeria. Nig J Physiol Sci. 2006;21:71-75.

10. Sirdah MM, Allaham NA, El Madhoun RA. Possible health effects of liquefied petroleum gas on workers at filling and distribution stations of Gaza governorates. EMHU. 2013;19(3):289-294.

11. Udronwa NE, Uko EK, Ikpeme BM, Ibanga IA, Okon BO. Exposure of petrol station attendants and auto mechanics to premium motor spirit fumes in Calabar, Nigeria. J En-viron Public Health. 2009;281876.

12. Akinunde JK, Abioye JB, Ebinama ON. Potential protective effects of Naringin on Oculo-pulmonary injury induced by PM10 (Wood Smoke) exposure by modulation of oxidative damage and acetylcholine esterase activity in a rat model. Curr Ther Res Clin Exp. 2020;92:100586.

13. Bergmeyer HU, Scheibe P, Wahlefeld AW. Methods for aspartate and alanine amino transferase. Clin Chem. 1979;125:1487.

14. Persijn JP, Van der Slik W. A new method for the determination of gamma-glutamyltransferase in serum. J. Clin Chem Clin Biochem. 1976;9(14):421-427.

15. Uboh FE, Akpanbiatu MI, Eteng PMU, Ebong E, Umoh IB. Toxicological effects of exposure to gasoline vapours in male and female rats. Internet J Toxicol. 2008;4:40–45.

16. Ekpenyong CE, Asuquo EA. Recent advances in occupational and environmental health hazards of workers exposed to gasoline compounds. IJOMEH. 2017;30(1):1-26.

17. Kurmi OP, Dunster C, Ayres JG, Kelly FJ. Oxidative potential of smoke from burning wood and mixed biomass fuels. Free Radic Res. 2013;47:829–835.

18. Uhegbu FO, Imo C, Ifeanacho NG. Effect of exposure of male albino rats to kerosene, diesel and petrol on kidney function. Int. Res. J. Environment Sci. 2015;4(11):12-18.

19. Uboh FE, Akpanbiatu MI, Ndem JI, Alozie Y, Ebong PE. Comparative nephrotoxic effect associated with exposure to diesel and gasoline vapours in rats. J. Toxicol. Environ. Health Sci. 2009;1(4):068-074.

20. Lum G, Gambino SR. Serum gamma glutamyl transeptidase activity as an indicator of diseases of liver, pancreas or bone. Clin. Chem. 1972;18:358.

21. Dere E, Ari F. Effect of benzene on liver function in rats (Rattus norvegicus). Environ Monit Assess. 2009;154:23–27.

22. George J, Liddle C, Murray M, Byth K, Farrell GC. Pre translational regulation of cytochrom P450 gene is responsible for disease changes of individual P450 enzymes among patients with cirrhosis. Biochem Pharmacol. 1995;9:873–881.

23. Tanaka T, Uchiimi T, Hinoshita E, Inokuchi A, Toh S, Wada M. The human multi drugs resistance protein 2 gene: Functional characterization of the 5-flanking region and expression in hepatic cells. Hepatology. 1999;30:1507–1512.

24. Al-Olayan EM, El-Khadragy MF, Aref AM, Othman MS, Kassab RB, Abdel Moneim AE. The potential protective effect of Physalis peruviana L. against carbon tetrachloride induced hepatotoxicity in rats is mediated by suppression of oxidative stress and down regulation of MMP-9 expression. Oxd Med Cell Longev. 2014;381413.

25. Odunola OA, Uka E, Kazeem A, Akinwumi KA, Gbadegesin MA, Osifeso OO, Ibegebui MD. Exposure of laboratory mice to domestic cooking gas: - Implications for...
toxicity. Int. J. Environ. Res. Public Health. 2008;5(3):172-176.
26. Ravnskov U. Experimental glomerulonephritis induced by hydrocarbon exposure: A systemic review. BMC Nephrol. 2005;6:15–18.
27. Awodele O, Sulayman AA, Akintonwe A. Evaluation of hematological hepatic and renal function of petroleum tanker drivers in Lagos, Nigeria. Afr Health Sci. 2014;14:178–84.
28. Iyanda AA. Effects of oral and dermal sub-chronic exposure of kerosene on biochemical parameters in male wistar rats. Annu. Res. Rev. Biol. 2013;3(3):188-194.

© 2020 Tochukwu et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sdiarticle4.com/review-history/62523