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Pro-inflammatory cytokines, markers of oxidative stress, hepatic and renal functions in individuals occupationally exposed to fumes from burning tires

Occupational exposure to fumes from burning tires

Onyekwelu Kenechukwu C.[1] Okorie Peace C.[1] Adilieje Chioma M.[1] Ejiofor Nonso C.[1] Ezechukwu Ifunanya N.[1]
Ugwu Chioma C.[1] Ikekpeazu Joy E.[1]

[1]Department of Medical Biochemistry, Faculty of Basic Medical Sciences, College of Medicine, University of Nigeria Enugu Campus, Nigeria.

Abstract

Aim: Burning of tires releases a significant amount of toxic pollutants like polycyclic aromatic hydrocarbons (PAHs), heavy metals, and volatile organic compounds into the air. This study assessed the health implications of occupational exposure to fumes from burning tires.

Methods: This was a population-based cross-sectional study. Fifty exposed and 50 control male subjects were recruited for this study. Pro-inflammatory cytokines, markers of oxidative stress, and hepatic and renal functions were assayed for in the subjects using standard methods. Data were subjected to inferential statistics and analyzed using Student's *t*-test.

Results: The results revealed significantly higher levels of urea (8.67 ± 0.45 mg/dL) and lower levels of creatinine (73.99 ± 2.87 mg/dL) compared to the controls (7.55 ± 0.31 mg/dL and 81.44 ± 2.12 mg/dL respectively); significantly elevated levels of alanine aminotransferase (ALT) (31.61 ± 3.80 U/L), aspartate aminotransferase (AST) (51.12 ± 5.07 U/L) and alkaline phosphatase (ALP) (113.14 ± 7.83 U/L); alterations in the electrolyte panel, including higher potassium (4.63 ± 0.21 mmol/L) and bicarbonate (37.91 ± 4.60 mmol/L) and lower sodium (135.80 ± 2.26 mmol/L); significant increase in the level of interleukin-1 (IL-1β) and interleukin-6 (IL-6); significant increase in serum malondialdehyde and serum total antioxidant status (TAS) levels in subjects occupationally exposed to fumes from burning tires compared to the controls.

Conclusion: The observed alterations in kidney and liver function biomarkers, along with pro-inflammatory cytokines and markers of oxidative stress, demonstrated that occupational exposure to fumes from burning tires could result in adverse health effects.

Keywords

polycyclic aromatic hydrocarbons, heavy metals, oxidative stress, renal function, hepatic function, pro-inflammatory cytokines

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Corresponding Author: Ezechukwu Ifunanya N., Department of Medical Biochemistry, Faculty of Basic Medical Sciences, College of Medicine, University of Nigeria Enugu Campus, Nigeria.
Email: ifunanya.ezechukwu@unn.edu.ng **P.**: +234 706 703 8587
Corresponding Author ORCID: https://orcid.org/0009-0006-2974-9877
Other Authors ORCID: Onyekwelu Kenechukwu C., https://orcid.org/0000-0001-8515-9456 • Okorie Peace C., https://orcid.org/0000-0003-2970-6931 • Adilieje Chioma M., https://orcid.org/0000-0001-6054-311X • Ejiofor Nonso C., https://orcid.org/0000-0003-2988-5685 • Ugwu Chioma C., https://orcid.org/0009-0008-0372-5716 • Ikekpeazu Joy E., https://orcid.org/0000-0002-4478-2660

Introduction

Burning of tires releases a range of harmful substances such as polycyclic aromatic hydrocarbons (PAHs), carbon monoxide, sulfur dioxide, nitrogen oxides, large quantities of soot, heavy metals like lead, cadmium, mercury, and many other substances into the atmosphere. Some of these pollutants are carcinogenic, and prolonged exposure to them has adverse effects on the renal, hepatic, respiratory, reproductive, cardiovascular, immunological, and inflammatory systems.[1-3] Occupational exposure to these pollutants, which gains entry into the body through inhalation, ingestion, and absorption through the skin, induces cellular oxidative stress by generating reactive oxygen species (ROS). Lipid peroxidation occurs when there is an imbalance in the generation and removal of these ROS, which leads to the breakdown of cellular components, especially phospholipids, which promotes DNA damage and cell necrosis. Studies have implicated lipid peroxidation in the underlying mechanisms of several diseases and disorders like renal, hepatic, cardiovascular, neurodegenerative diseases, and cancer.[4-6]

Inflammation, which is the immune system's response to harmful stimuli such as toxic compounds, damaged cells, pathogens, and others, is one of the body's defense/protective mechanisms that is crucial in maintaining normal health.[7,8] Inflammation protects the body by removing injurious stimuli, initiating the healing process, and restoring the physiological functions of affected tissues and organs,[9,10] but prolonged inflammation may contribute to chronic diseases.[11,12] Studies have shown that inflammation is linked to the genesis of many diseases in humans and that heavy metals exert toxicity in humans by interfering with immune homeostasis and promoting infection. [13,14]

After gaining entry into the body, these harmful substances accumulate in various organs or other parts of the body such as the liver, kidney, lung, heart, brain, and the muscles, making them more vulnerable to attacks. Studies have shown that detectable levels of polycyclic aromatic hydrocarbons occur in almost all internal organs, especially in those organs that are adipose tissue-rich, which can serve as storage depots from which the toxicants can be gradually released.[15] Phases 1 and 2 pathways of xenobiotics biotransformation work together to help in the removal of xenobiotics from the body system. The hepatic cytochrome P450 mixed-function oxidase enzymes play a major role in the metabolism and the biotransformation of endogenous substrates as well as in the biotransformation of exogenous xenobiotics by converting lipophilic xenobiotics such as environmental pollutants, food additives, drugs, insecticides, and substances to hydrophilic compounds which are easier to excrete through the urine or feces. This process is catalyzed by a number of enzymes and generally involves oxidation, reduction, and hydrolysis reactions and is referred to as phase 1 xenobiotics metabolism reaction. Phase 2 conjugation reactions involve conjugation of lipophilic compounds with endogenous conjugation agents such as glucuronic acid, which increases the solubility of the xenobiotic compound.[16,17] In some cases, numerous xenobiotics yield toxic metabolites which are more toxic than the parent xenobiotic, a situation known as toxification.[18]

In Nigeria, flames from burning old tires are used in the processing of foods, including de-furring of slaughtered animals, because it is a cheap source of fuel. This is a common practice in most cities across Nigeria without considering the environmental effects and the impact on the health of abattoir workers and the general public who consume foods processed through such a method. De-furring of animals in abattoirs using flame from burning tires has resulted in elevated traces of heavy metals in meats.[19] Despite the ban on the use of flame from burning tires in de-furring of meat, this practice is still ongoing. The purpose of this study was to investigate the adverse health effects that butchers may encounter as a result of occupational exposure to various degrees of emission from burning tires.

Materials and Methods

This was a population-based cross-sectional study in which the renal and hepatic functions, pro-inflammatory cytokines, markers of oxidative stress, and electrolyte panel were investigated in butchers who use flame from burning tires in de-furring animals. This study was conducted between April and September 2025.

Study Location

This study was conducted at the old and new Artisan markets in Enugu metropolis. The old Artisan market, located along Ogui Road adjacent to the Ebeano tunnel driveway, stands as one of the oldest markets in Enugu, specializing in livestock trade such as cows, goats, and sheep. The new Artisan market is situated along the Enugu-Port Harcourt expressway. Butchers at these markets utilize mud blocks or old metal water/oil drums to construct fire mounds with a round base and tapering top covered with metal gauze, where animal and animal parts for de-furring are placed. Old tires are placed inside the fire mound through a side hole and ignited (Figure 1).

Figure 1: De-furring mound used by butchers in meat processing (Image created by the first author)

Subjects

Using a purposive sampling method targeted at selecting subjects occupationally exposed to fumes from burning tires, fifty (50) male butchers were selected for this study. The subjects were recruited for this study if they: were aged 18 years and above, expressed willingness to participate and gave their informed consent, had worked as a butcher and had been exposed to fumes of burning tires through inhalation, dermal absorption, or ingestion for at least 3 years, had no pre-existing liver or kidney disease and no pre-existing inflammatory conditions.

Subjects were excluded if they were below 18 years of age, were unwilling to participate or unable to give informed consent, had worked as a butcher for less than 3 years, or had pre-existing liver or kidney or inflammatory disease or conditions. Those with any form of cancerous condition and those who smoked cigarettes were also excluded from the study. The control subjects involved 50 males who were not occupationally exposed to fumes from burning tires and who did not meet any of the exclusion criteria listed above. Details of the study design and sample size determination were as described by Leidel et al. 1977.[20]

Sample Collection

After fulfilling the above inclusion criteria, blood samples (6 mL) were collected from each subject into a container for biochemical assays.

Biochemical Assays

Liver Function

Serum levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were measured for the evaluation of liver function. The method described by Reitman and Frankel 1957 [21] was used in the determination of the levels of alanine aminotransferase and aspartate aminotransferase with slight modification, while alkaline

phosphatase was determined according to the method described by Kind and King 1954.[22]

Kidney Function

Using a colorimetric method, creatinine in the serum was determined by the Jaffe reaction, where creatinine combines with alkaline picrate to form a red-colored chromophore. The absorbance was measured in the 490–510 nm range, and the absorbance is directly proportional to the serum creatinine concentration.[23] Blood urea was determined using the direct colorimetric method as described by Barker 1944.[24]

Pro-Inflamatory Cytokines

Serum concentrations of interleukin-1β (IL-1β) and interleukin-6 (IL-6) were measured using Melsin human enzyme-linked immunosorbent assay (ELISA) kits (EKHU-0147, EKHU-0140; Melsin Medical Co. Ltd, Changchun, China).

Markers of Oxidative Stress

Malondialdehyde (MDA), a marker of lipid peroxidation, was analyzed using the method of Ohkawa et al.,[25] while the method described by Koracevic et al., 2001 [26] was employed to assay total antioxidant status (TAS).

Electrolyte Panel

Serum electrolyte levels were determined using the ion-selective electrode module of the COBAS 6000 (c501) analyzer manufactured by Roche Diagnostics, Germany.

Ethical Approval

This study was approved by the Ethics Committee of the University of Nigeria College of Medicine (Date: 03.04.2025, Decision No: COMHREC/2025/04/029).

Statistical Analysis

Statistical analyses were performed using SPSS version 22.0. Continuous variables were compared between the two groups using the independent-samples Student's *t*-test. A P value < .05 was considered statistically significant.

Reporting Guidelines

This study was reported in accordance with the STROBE guideline.

Results

Subjects exposed to fumes from burning tires showed a significant increase (P < .05) in ALT (31.61 ± 3.80 U/L), AST (51.12 ± 5.07 U/L), ALP (113.14 ± 7.83 U/L), interleukin-1 (22.17 ± 4.09 pg/mL), and interleukin-6 (13.63 ± 7.11 pg/mL) when compared with their respective controls (Table 1).

Renal function results showed higher urea levels (8.67 ± 0.45 mg/dL) and lower creatinine levels (73.99 ± 2.87 mg/dL) in individuals exposed to fumes from burning tires than in controls (7.55 ± 0.31 mg/dL and 81.44 ± 2.12 mg/dL, respectively) (Table 2).

Malondialdehyde (MDA) of subjects exposed to fumes from burning tires was elevated (10.60 ± 58 µmol/L) compared with the control (7.42 ± 0.31 µmol/L). The P value < .05 showed a statistically significant difference between the test group and the control group (Table 3). The mean TAS of 8.11 ± 0.22 mmol/L of the subjects exposed to polycyclic aromatic hydrocarbon was statistically significantly higher (P < .05) than the mean TAS of 5.16 ± 0.27 mmol/L of the control (Table 3).

Subjects exposed to fumes from burning tires showed statistically significant increases (P < .05) in potassium (4.63 ± 0.21 mmol/L) and bicarbonate (37.91 ± 4.60 mmol/L), and statistically significant decreases in sodium (135.80 ± 2.26 mmol/L) when compared with their respective controls. There was no statistically significant difference in chloride levels between the test group (102.06 ± 2.34 mmol/L) and the control group (101.19 ± 0.32 mmol/L) (Table 4).

Discussion

The liver plays a vital role in almost every metabolic process in the body, including maintenance of homeostasis, and is involved in the metabolism of drugs and xenobiotics.[27] The findings of this study showed significant alterations in liver function biomarkers. The result revealed significantly elevated levels of alanine aminotransferase (ALT) (31.61 ± 3.80), aspartate aminotransferase (AST) (51.12 ± 5.07) and alkaline phosphatase (ALP) (113.14 ± 7.83) in individuals occupationally exposed to fumes from burning tires compared to the controls (20.30 ± 0.81, 21.61 ± 1.05 and 84.19 ± 3.23) respectively (Table 1); which is suggestive of liver damage. The observed increase in ALT and AST levels may indicate liver cell damage or inflammation due to PAH-induced hepatotoxicity. Additionally, the elevated ALP levels suggest cholestasis, which can occur secondary to liver injury caused by PAH exposure. This is in accordance with the study carried out by Ufelle et al. (2020), Zhang et al. 2019 and Chen et al. 2006 with elevations in liver enzymes among workers exposed to PAHs and other xenobiotics in various occupational settings.[28-30]

Xenobiotic detoxification involves multiple organs working together to remove harmful substances from the body. The kidney filters waste and toxins from the blood, which are excreted through the urine. Individuals exposed to fumes from burning tires had higher urea levels (8.67 ± 0.45 mg/dL) and lower creatinine levels (73.99 ± 2.87 mg/dL) than controls (7.55 ± 0.31 mg/dL and 81.44 ± 2.12 mg/dL, respectively), indicating possible kidney injury or dysfunction (Table 2).When the kidneys are injured as a result of polycyclic aromatic hydrocarbons, toxins, medications, and other harmful substances, their ability to filter and remove waste products like urea and creatinine is impaired, resulting in increased blood levels of the metabolites. Studies have suggested that polycyclic aromatic hydrocarbons and other environmental toxicants may play a role in the development of chronic health conditions including renal dysfunction.[31]

Oxidative stress, which is a state of imbalance between the production of reactive oxygen species and the ability of the body to detoxify these harmful substances, is caused by environmental factors such as exposure to fumes from burning tires and other air pollutants, among other factors.[32] There was a significant increase in both the serum malondialdehyde (MDA) and serum total antioxidant status (TAS) levels (Table 3), which are markers of lipid peroxidation due to oxidative stress in subjects occupationally exposed to fumes from burning tires compared to the control. Studies on the mechanism of damage caused by polycyclic aromatic hydrocarbons have shown that polycyclic aromatic hydrocarbons and heavy metals can enhance the body's oxidative stress and increase the body's reactive oxygen species.[33,34] Polycyclic aromatic hydrocarbons are known potent oxidant stressors which can contribute to inflammation and pathogenesis of chronic disease.[35] The potent oxidant stressor of PAHs has been observed in various studies, including studies on occupationally exposed adults in which higher levels of oxidative stress biomarkers and lipid peroxidation were found.[36]

The kidney is largely thought of as the organ responsible for elimination of drugs, toxins, and waste products from the body, but it performs many other important functions like maintenance of plasma osmolarity, water, and electrolyte balance.[37,38] In this study, subjects exposed to fumes from burning tires showed statistically

Table 1. Hepatic Functions and Pro-Inflammatory Cytokines of Subjects Exposed to Fumes From Burning Tyres and Their Controls

Parameters	Mean ± SD of subjects exposed to fumes from burning tyres	Mean ± SD of control group
ALT, U/L	31.61 ± 3.80*	20.30 ± 0.81
AST, U/L	51.12 ± 5.07*	21.61 ± 1.05
ALP, U/L	113.14 ± 7.83*	84.19 ± 3.23
IL-1β, pg/ml	22.17 ± 4.09*	10.69 ± 1.04
IL-6, pg/ml	13.63 ± 7.11*	7.22 ± 3.81

*ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; ALP, Alkaline phosphatase; IL -1β, interleukin-1β; IL -6, interleuki n-6; * = stastistically significant (P < .05) when compared with the control.*

Table 2. Renal Functions of Subjects Exposed to Fumes From Burning Tyres and Their Controls

Markers of renal fuction	Mean ± SD of subjects exposed to fumes from burning tyres	Mean ± SD of control group
Urea, mg/dL	8.67 ± 0.45*	7.55 ± 0.31
Creatinine, mg/dL	73.99 ± 2.87	81.44 ± 2.12

** = stastistically significant (p<0.05) when compared with the control.*

Table 3. Serum TAS and MDA of Subjects Exposed to Fumes From Burning Tyres and Their Controls

Markers of oxidative stress	Mean ± SD of subjects exposed to fumes from burning tyres	Mean ± SD of control group
MDA, µmol/l	10.60 ± 58*	7.42 ± 0.31
TAS, mmol/l	8.11 ± 0.22*	5.16 ± 0.27

*MDA, Malondialdehyde; TAS, Total antioxidant status; * = stastistically significant (P < .05) when compared with the control.*

Table 4. Mean Serum Electrolyte of Subjects Exposed to Fumes From Burning Tyres and Their Controls

Markers of oxidative stress	Mean ± SD of subjects exposed to fumes from burning tyres	Mean ± SD of control group
MDA, µmol/l	10.60 ± 58*	7.42 ± 0.31
TAS, mmol/l	8.11 ± 0.22*	5.16 ± 0.27

*Na^+, sodium ions; K^+, Potassium ions; HCO_3^-, Bicarbonate ions; Cl^- , Chloride ions; * = stastistically significant (p<0.05) when compared with the control.*



Figure 1. *Tire-Burning De-Furring Mound Used by Butchers for Meat Processing*

significant increases (*P* < .05) in potassium (4.63 ± 0.21 mmol/L) and bicarbonate (37.91 ± 4.60 mmol/L), and a statistically significant decrease in sodium (135.80 ± 2.26 mmol/L) when compared with their respective controls (Table 4). Exposure to PAH has been linked to kidney injury, which can impair the ability of the kidneys to filter and balance electrolytes, leading to imbalances such as hyperkalemia (high potassium) and hyponatremia (low sodium), as was observed in this study.

The immune system is continually exposed to various environmental xenobiotics, including polycyclic aromatic hydrocarbons and heavy metals. As a result of this, various inflammatory signaling pathways are activated, along with reduced disease resistance and inflammation.[39] This study showed that fumes from burning tires had a deleterious impact on the immune system. It impacts several pro-inflammatory cytokines, such as interleukins (IL-1β and IL-6), which were significantly increased when compared with their respective controls (Table 1), contributing to inflammatory disorders. Occupational exposure to heavy metals has consistently increased the levels of the pro-inflammatory cytokines.[40]

Limitations

This study was limited by difficulty in isolating tire smoke exposure from lifestyle factors and general ambient urban air pollution.

Conclusion

The observed alterations in kidney and liver function biomarkers, along with pro-inflammatory cytokines and markers of oxidative stress, indicated that occupational exposure to fumes from burning tires could lead to adverse health effects. Burning tires releases a range of harmful substances, including polycyclic aromatic hydrocarbons (PAHs), heavy metals such as lead, cadmium, and

mercury, and other compounds, into the atmosphere, which can have adverse effects on the renal, hepatic, and inflammatory systems after prolonged exposure.

Declarations

Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Declaration of Helsinki and its later amendments, or comparable ethical standards.

Informed Consent

Written informed consent was obtained from all participants prior to their enrollment in the study.

Data Availability

The authors confirm that the data supporting the findings of this study are available within the manuscript.

Conflict of Interest

The authors declare no conflict of interest.

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None.

Author Contributions (CRediT Taxonomy)

Conceptualization: O.K.C., I.J.E.
Methodology: O.K.C., O.P.C., A.C.M.
Investigation: O.K.C., O.P.C., A.C.M., E.N.C., E.I.N., U.C.C.
Data Curation: O.K.C., E.I.N.
Formal Analysis: O.K.C., I.J.E.
Writing – Original Draft: O.K.C.
Writing – Review & Editing: O.K.C., O.P.C., A.C.M., E.N.C., E.I.N., U.C.C., I.J.E.
Supervision: I.J.E.

AI Usage Disclosure

The authors confirm that the data supporting the findings of this study are available within the manuscript.

Abbreviations

ALP: Alkaline phosphatase
ALT: Alanine aminotransferase
AST: Aspartate aminotransferase
ELISA: Enzyme-linked immunosorbent assay
IL: Interleukin
MDA: Malondialdehyde
ROS: Reactive oxygen species
SD: Standard deviation
SPSS: Statistical package for the social sciences
TAS: Total antioxidant status

References

1. *US Environmental Protection Agency. Air Emissions From Scrap Tire Combustion. US Environmental Protection Agency; 1997.*

2. *Jankowska-Kieltyka M, Roman A, Nalepa I. The air we breathe: air pollution as a prevalent proinflammatory stimulus contributing to neurodegeneration. Front Cell Neurosci. 2021;15:647643. doi:10.3389/fncel.2021.647643*

3. *Oliveira M, Slezakova K, Delerue-Matos C, Pereira MC, Morais S. Children environmental exposure to particulate matter and polycyclic aromatic hydrocarbons and biomonitoring in school environments: a review on indoor and outdoor exposure levels, major sources, and health impacts. Environ Int. 2019;124:180-204. doi:10.1016/j.envint.2018.12.052*

4. *Halliwell B. Oxidative stress and neurodegeneration: where are we now? J Neurochem. 2006;97:1634-1658. doi:10.1111/j.1471-4159.2006.03907.x*

5. *Halliwell B, Gutteridge JMC. Free Radicals in Biology and Medicine. 4th ed. Oxford University Press; 2007.*

6. *Tchounwou PB, Yedjou CG, Patlolla AK, Sutton DJ. Heavy metal toxicity and the environment. Exp Suppl. 2012;101:133-164. doi:10.1007/978-3-7643-8340-4_6*

7. *Medzhitov R. Inflammation 2010: new adventures of an old flame. Cell. 2010;140:771-776. doi:10.1016/j.cell.2010.03.006*

8. *Nathan C, Ding A. Nonresolving inflammation. Cell. 2010;140:871-882. doi:10.1016/j.cell.2010.02.029*

9. *Ferrero-Miliani L, Nielsen O, Andersen P, Girardin S. Chronic inflammation: importance of NOD2 and NALP3 in interleukin-1β generation. Clin Exp Immunol. 2007;147:227-235. doi:10.1111/j.1365-2249.2006.03261.x*

10. *Olszowski T, Baranowska-Bosiacka I, Gutowska I, Chlubek D. Pro-inflammatory properties of cadmium. Acta Biochim Pol. 2012;59(4):475-482. doi:10.18388/abp.2012_2080*

11. *Giovanni M, Yue J, Zhang L, Xie J, Ong CN, Leong DT. Pro-inflammatory responses of RAW264.7 macrophages when treated with ultralow concentrations of silver, titanium dioxide, and zinc oxide nanoparticles. J Hazard Mater. 2015;297:146-152. doi:10.1016/j.jhazmat.2015.04.081*

12. *Wu Y, Antony S, Meitzler JL, Doroshow JH. Molecular mechanisms underlying chronic inflammation-associated cancers. Cancer Lett. 2014;345(2):164-173. doi:10.1016/j.canlet.2013.08.014*

13. *Brucker N, Moro A, Charão M, et al. Relationship between blood metals and inflammation in taxi drivers. Clin Chim Acta. 2015;444:176-181. doi:10.1016/j.cca.2015.02.032*

14. *Jin Y, Liu L, Zhang S, et al. Cadmium exposure to murine macrophages decreases their inflammatory responses and increases their oxidative stress. Chemosphere. 2016;144:168-175. doi:10.1016/j.chemosphere.2015.08.084*

15. *Pavanello S, Fustinoni S. Urinary profiles to assess polycyclic aromatic hydrocarbons exposure in coke-oven workers. Toxicol Lett. 2010.*

16. *Ortiz de Montellano PR. Cytochrome P450: Structure, Mechanism, and Biochemistry. Plenum Press; 1986. Black SD. Membrane topology of the mammalian P450 cytochromes. FASEB J. 1992;6:680-685. doi:10.1096/fasebj.6.2.1537456*

17. *Baranczewski P, Stańczak A, Sundberg K, et al. Introduction to in vitro estimation of metabolic stability and drug interactions of new chemical entities in drug discovery and development. Pharmacol Rep. 2006;58(4):453-472.*

18. *Testa B, Pedretti A, Vistoli G. Reactions and enzymes in the metabolism of drugs and other xenobiotics: a meta-analysis of the primary literature. Drug Discov Today. 2012;17(11-12):549-560. doi:10.1016/j.drudis.2012.01.017*

19. *Beetseh C, Onum D. Chemical implications of metal toxicity of meat processed through tire fire. Chem Mater Res. 2013;3:79-90.*

20. *Leidel NA, Busch KA, Lynch JR. Occupational Exposure Sampling Strategy Manual. DHEW/NIOSH Publication No. 77-173. National Institute for Occupational Safety and Health; 1977.*

21. *Reitman S, Frankel S. A colorimetric method for the determination of serum glutamic oxalacetic and glutamic pyruvic transaminases. Am J Clin Pathol. 1957;28(1):56-63.*

22. *Kind PR, King EJ. Estimation of plasma phosphatase by determination of hydrolysed phenol with amino-antipyrine. J Clin Pathol. 1954;7(4):322-326. doi:10.1136/jcp.7.4.322*

23. *Toora BD, Rajagopal G. Measurement of creatinine by Jaffe reaction: determination of concentration of sodium hydroxide required for maximum color development in standard, urine, and protein-free filtrate of serum. Indian J Exp Biol. 2002;40(3):352-354.*

24. *Barker SB. The direct colorimetric determination of urea in blood and urine. J Biol Chem. 1944;152(2):453-463. doi:10.1016/s0021-9258(18)72073-5*

25. *Ohkawa H, Ohishi N, Yaji K. Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. Anal Biochem. 1979;95:351-356. doi:10.1016/0003-2697(79)90738-3*

26. *Koracevic D, Koracevic G, Djordjevic V, Andrejevic S, Cosic V. Method for the measurement of antioxidant activity in human fluids. J Clin Pathol. 2001;54:356. doi:10.1136/jcp.54.5.356*

27. *Singh A, Bhat TK, Sharma OP. Clinical biochemistry of hepatotoxicity. J Clin Toxicol. 2011;S4:1-3.*

28. *Ufelle S, Onyekwelu K, Aneke C, Ibegbu D, Okoli U, Ikekpeazu J. Assessment of hepatic functions, hematopoietic cytokines, and haematological parameters in people occupationally exposed to volatile petroleum hydrocarbons. Arch Environ Occup Health. 2020;76(8):567-671.*

29. *Zhang Z, Guo C, Chang LY, et al. Long-term exposure to ambient fine particulate matter and liver enzymes in adults: a cross-sectional study in Taiwan. Occup Environ Med. 2019;76(7):488-494. doi:10.1136/oemed-2019-105695*

30. *Chen B, Zheng LX, Pan JS, Wang XJ. Elevation of some serum liver enzymes in coke oven workers and the possible relationship with exposure to polycyclic aromatic hydrocarbons. Wei Sheng Yan Jiu. 2006;35(3):264-268.*

31. *Kataria A, Trasande L, Trachtman H. The effects of environmental chemicals on renal function. Nat Rev Nephrol. 2015;11(10):610-625. doi:10.1038/nrneph.2015.94*

32. *Ikekpeazu JE, Ikekpeazu AJ, Eke CN, et al. Oxidative stress, lipid peroxidation, and antioxidant enzymes in apparently healthy individuals involved in physical exercise. Asian J Med Sci. 2016;7(6):16-19.*

33. *Moller P, Scholten RH, Roursgaard M, Krais AM. Inflammation, oxidative stress, and genotoxicity responses to biodiesel emissions in cultured mammalian cells and animals. Crit Rev Toxicol. 2020;50(5):383-401.*

34. *Vogel CFA, Van Winkle LS, Esser C, Haarmann-Stemmann T. The aryl hydrocarbon receptor as a target of environmental stressors: implications for pollution-mediated stress and inflammatory responses. Redox Biol. 2020;34:101530. doi:10.1016/j.redox.2020.101530*

35. *Araujo JA, Barajas B, Kleinman M, et al. Ambient particulate pollutants in the ultrafine range promote early atherosclerosis and systemic oxidative stress. Circ Res. 2008;102(5):589-596. doi:10.1161/circresaha.107.164970*

36. *Kuang D, Zhang W, Deng Q, et al. Dose-response relationships of polycyclic aromatic hydrocarbons exposure and oxidative damage to DNA and lipid in coke oven workers. Environ Sci Technol. 2013;47(13):7446-7456. doi:10.1021/es401639x*

37. *Bougle A, Duranteau J. Pathophysiology of sepsis-induced acute kidney injury: the role of global renal blood flow and renal vascular resistance. Contrib Nephrol. 2011;174:89-97.*

38. *Brown D, Wagner CA. Molecular mechanisms of acid-base sensing by the kidney. J Am Soc Nephrol. 2012;23:774-780. doi:10.1681/asn.2012010029*

39. *Harshitha P, Bose K, Dsouza HS. Influence of lead-induced toxicity on the inflammatory*

cytokines. Toxicology. 2024;503:153771. doi:10.1016/j.tox.2024.153771

40. *Turksoy VA, Tutkun L, Iritas SB, Gunduzoz M, Deniz S. The effects of occupational lead exposure on selected inflammatory biomarkers. Arch Ind Hyg Toxicol. 2019;70:36-41. doi:10.2478/aiht-2019-70-3214*

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