Available online on 15.04.2023 at http://jddtonline.info

Journal of Drug Delivery and Therapeutics

Open Access to Pharmaceutical and Medical Research

Open Access Full Text Article

Research Article

Effect of Exposure to Soot on Activities of Alanine Transaminase, Gamma Glutamyl Transferase and Lipase among Cooks in Nnewi

Augustine Chinedu Ihim [1]*, Yusuf Habeeb [1], Patrick Onochie Manafa [1], Emmanuel Ikechukwu Nwobodo [2], Ukamaka Chima Edward [3]. Patrick Chinedu Obi [4]

1. Department of Medical Laboratory Science, Nnamdi Azikiwe University, Nnewi Campus, Anambra State, Nigeria

2. Department of Medical Laboratory Science, Enugu State University, Nigeria

3. Department of medical Laboratory Science, Imo state University, Nigeria

4. Faculty of Medicine, Imo state University, Nigeria

Article Info:

Article History:

Received 12 Feb 2023
Reviewed 15 March 2023
Accepted 27 March 2023
Published 15 April 2023

Cite this article as:

Ihim AC, Habeeb Y, Manafa PO, Nwobodo EI, Edward UC, Obi PC, Effect of Exposure to Soot on Activities of Alanine Transaminase, Gamma Glutamyl Transferase and Lipase among Cooks in Nnewi, Journal of Drug Delivery and Therapeutics. 2023; 13(4):37-41

DOI: http://dx.doi.org/10.22270/jddt.v13i4.5793

*Address for Correspondence:

Dr Ihim Augustine Chinedu, Department of Medical Laboratory Science, Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, Awka, Nigeria

Abstract

Exposure to soot can lead to hepatic and pancreatic dysfunction. This study determined serum alanine transaminase (ALT), gamma-glutamyl transferase (GGT), and triacylglycerol lipase activities of cooks exposed to soot. Thirty (30) participants (cooks) were exposed to soot (test group), and thirty (30) participants not exposed to soot (control group) were recruited. Ethical approval was obtained from the Ethics committee of the Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, and informed consent from the participants was obtained. Alanine transaminase, gamma-glutamyl transferase, and triacylglycerol lipase activities were determined using the spectrophotometric method while the Body mass index (BMI) was calculated using weight (kg)/height2 (m2). An Independent t-test was used for statistical analysis. The results showed that the mean serum activities of alanine transaminase (14.69±1.84), of cooks exposed to soot, were significantly higher compared with the control(12.83±0.94)($p<0.05$). The mean serum activities of gamma-glutamyl transferase (7.81±4.08), of cooks exposed to soot, were significantly higher when compared with the control (3.54±2.00)($p<0.05$). The mean serum activities of triacylglycerol lipase of cooks exposed to soot were significantly low compared with the control ($p<0.05$), while there was no significant difference in the BMI, systolic pressure, and diastolic pressure levels respectively in the test group, compared to the control. This study observed higher serum activities of alanine transaminase, gamma-glutamyl transferase, and low serum activity of triacylglycerol lipase among cooks exposed to soot, this suggests a predisposition to hepatic and pancreatic dysfunction in the cooks

Keywords: Soot, alanine transaminase, gamma-glutamyl transferase, lipase enzymes

INTRODUCTION

The environmental soot (black carbon (BC)) causes many health issues in humans and animals [1]. Soot is considered an unwanted by-product derived from the incomplete combustion of carbon-containing materials. Soot is a powdery mass of fine black particles[2]. It consists of impure carbon, formed after the incomplete combustion of hydrocarbons [3]. The main source of environmental soot is the combustion of fossil-based fuels and biomass burning at the Earth's surface [4]. Other examples of soot may include coal, charred wood, petroleum coke, cenospheres, and tars[5]. To a smaller extent, quartz/halogen bulbs with settled dust, cooking, oil lamps, smoking of plant matter, fireplaces, candles, house fires, furnaces, and local field burning also contribute to the soot production [6]. Developed nations had been the biggest source of soot emissions until now, soot emissions are majorly from developing countries [7]. It is noted that the United States emits about 6.1% of the world's soot [8], while the largest amount of soot comes from Latin America, Asia, and Africa [9]. India and China alone may account for around 25– 35% of total global soot emissions 8Animal studies indicate that inhaled air pollutants, particularly particular matter(PM), can induce hepatotoxic effects [9]. Exposure to PM with an aerodynamic diameter ≤2.5 μm has been linked with inflammation in Kupffer cells, hepatic steatosis, and fibrosis in mice models [10]. However, evidence from human populations is scarce. Only a few epidemiological studies investigated the associations of long-term exposure to PM air pollution with hepatic steatosis or liver cancer [11] and the results are inconsistent. Liver enzymes are widely and routinely measured markers in a clinical setting for the assessment of liver function. Elevations in liver enzymes may indicate liver damage induced by various causes, such as alcohol and hepatic viruses. Mounting evidence has also shown that elevated liver enzymes, such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), and γ-glutamyl transferase (GGT), are independently associated with increased risks of metabolic syndrome, cardiovascular disease (CVD) and mortality [12]. Few studies investigated the effects of air pollution on liver enzymes but the findings are inconclusive [11]. Similar to smoke or ash, soot can be harmful to human health when inhaled. The incomplete combustion is necessary to produce soot resulting in the formation of dioxins and other toxic compounds. The small size of particulate

matter allows it to easily enter the lungs and bloodstream. This can cause serious effects including heart attacks, bronchitis, aggravated asthma, strokes, and even premature death [13].

A few studies have elucidated the effect of soot on the cardiovascular system [14], the respiratory pathways, and as a carcinogen [15] but little or no literature has been available on the effect of soot on the liver and pancreas, especially in Nnewi Metropolis. People have engaged in occupations and businesses which expose them to soot such as bean cake frying, palm fruit cooks, industrial work, etc. to make ends meet without knowing the risk associated with exposure to soot and its safety precautions thereby being vulnerable to the hazardous impact on their health. Thus, this research investigated the effect of exposure to soot on the activities of alanine transaminase, gamma-glutamyl transferase, and lipase enzymes among cooks in Nnewi to address this knowledge gap.

MATERIALS AND METHODS

The study was a cross-sectional study in the Nnewi North Local Government area of Anambra State, South Eastern, Nigeria. Nnewi is a commercial city with a large auto spare parts market and the residents are mainly businessmen and women as well as health workers who work at the Nnamdi AzikiweUniversity Teaching Hospital and other private and mission hospitals located in the town. This study was carried out amongst cooks (bean cake fryers, palm fruit cooks, groundnut fryers) exposed to soot in Nnewi, Anambra state.

Study participants

The sample size was calculated using G* Power software version 3.0.10 (Universitat Dusseldorf Germany). Power analysis for the difference between two independent means (two groups), was conducted in G* Power to determine a sufficient sample size using an alpha of 0.05, a power of 0.80, and an effect of 0.5. Based on these, the calculated total sample size of 42 has a power of 80% power to detect a difference of 0.25 at a significant level of 0.05. A total sample size of 60 was used for this study to take care of possible attrition. Sixty (60) healthy male and female participants were recruited as follows. Thirty (30) participants were exposed to soot as the test group. Thirty (30) participants were not exposed to soot as the control. A structured questionnaire was administered to determine the socio-demographic information of the participants. The study was approved by the Ethics Committee of the Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, Nnewi, Anambra State, Nigeria ERC/FHST/NAU/2021/MLS41. All the participants were informed of the project and consented participants between 18 and 65 years were recruited whereas; individuals that are sick, on drugs (except antimalaria), smokers, participants with chronic renal, liver, and cardiac failure, hypothyroidism, and obesity, and participants that declined consent were excluded from the study. Subjects younger than 18 years of age and older than 65 years of age or those outside the age range were excluded. Five milliliters (5ml) of venous blood was collected from each participant into a plain tube and allowed to clot and retract, then spun using a whisperfuge centrifuge (Germany) at 4000 revolutions per minute. The serum was separated from the plain tube using a micropipette(China) and stored at -4℃ in three aliquots using cryovials until the analysis of the activities of alanine transaminase, gamma-glutamyl transferase, and lipase. Serum alanine aminotransferase and aspartate aminotransferase activities were determined using the colorimetric method as described by [16] and modified by[17] gamma glutamyltransferase activity was determined spectrophotometrically as described by [17] while the turbidimetric method as described by [18] was employed for the evaluation of lipase.

Statistical analysis

The data obtained were presented as mean±SD and the mean values of the baseline and test samples were compared by Students t-test and Pearson r correlation using Statistical package for social sciences (SPSS) (Version 23) software. Statistical significance was tested at $p<0.05$.

RESULTS

Table 1.The mean values of the anthropometric indices and blood pressure of participants exposed to soot, and the control (Mean ± SD)

There was no significant difference in the mean values of the height, weight, body mass index, age, systolic, and diastolic blood pressures of participants exposed to soot compared to the control.

Table 1: The mean values of the anthropometric indices and blood pressure of participants exposed to soot and the control (Mean ± SD)

Anthropometric indices- and blood pressure	Non-soot exposed participants (test group n=30)	Soot-exposed participants (control group=30)	t-value	p-value
Age (years)	28.90±2.69	45.83±3.95	19.370	0.107
Weight (kg)	64.80±17.11	67.53±9.25	0.779	0.444
Height (m)	189.40±8.13	188.77±7.59	0.312	0.756
BMI	17.98±4.29	18.98±2.66	1.082	0.284
SBP	123.67±10.66	121.67±7.47	0.840	0.404
DBP	72.33±14.55	75.67±5.04	1.186	0.240

*Statistically significant at ($p<0.05$)
SBP=Systolic blood pressure, Diastolic blood pressure= DBP, BMI= Body Mass Index

A significantly higher difference was observed in the mean serum activities of alanine transaminase (14.69±1.84) and gamma-glutamyl transaminase (7.81±4.08) in participants exposed to soot compared with the control($p<0.05$), while a significant lower difference existed in the mean serum lipase activity (46.38±16.30) of participants exposed to soot compared with the control(65.71±33.91)($p<0.05$)

Table 2: The serum alanine transaminase, gamma-glutamyl transferase and lipase activities of participants exposed to soot and the control (Mean ± SD)

Enzymes	Non-soot exposed participants (test group n=30)	Soot-exposed participants (control group n=30)	t-value	p-value
ALT (U/L)	12.83±0.94	14.69±1.84	4.950	0.000*
GGT (U/L)	3.54±2.00	7.81±4.08	5.129	0.000*
LIPASE (U/L)	65.71±33.91	46.38±16.30	2.815	0.007*

*Statistically significant at (p<0.05).

Table 3: The association of alanine transaminase, gamma-glutamyl transferase, and lipase activities with Body Mass Index, Systolic and Diastolic blood pressures (Mean ± SD)

There is a weak negative association between GGT and systolic blood pressure (r=0.301,p=0.020)

Enzymes		BMI	Systolic blood pressure	Diastolic blood pressure
ALT	r-value	0.128	-0.200	-0.480
	p-value	0.328	0.125	0.717
GGT	r-value	0.136	-0.301	0.003
	P-value	0.301	0.020	0.979
Lipase	r-value	0.025	0.073	0.163
	p-value	0.851	0.578	0.214

*Statistically significant at (P<0.05).

ALT=alanine transaminase, GGT=gamma glutamyl transferase, SBP=Systolic blood pressure, Diastolic blood pressure= DBP, BMI= Body Mass Index

DISCUSSION

Soot is an impure and unrefined carbonaceous substance produced as a result of incomplete combustion of wood, oil, etc., and rising in particles and adhering to the sides of the chimney or pipe conveying the smoke in the atmosphere to other locations [14]. Most cooks are ignorant of the health-dilapidating effect of exposure to soot or particulate matter(PM). Unfortunately in Nigeria and especially in Nnewi, most kitchen and houses lack chimneys or pipes conveying the smoke from the kitchens to the atmosphere, and large-scale cooking for parties and other ceremonies are done in the open ground thereby making soot more concentrated and readily available for the cooks' inhalation. In this study, there was no significant difference in the anthropometric indices and blood pressure of the participants exposed to soot compared with the control(p<0.05).This was expected as the participants were healthy and age-matched. However a significantly higher difference was observed in the mean serum activities of alanine transaminase and gamma-glutamyl transaminase in participants exposed to soot compared with the control(p<0.05), This study agrees with the cross-sectional study in Taiwan by [22] which investigated long-term exposure to ambient fine particulate matter on the liver enzymes in adults which observed higher alanine transaminase (ALT), gamma-glutamyl transferase (GGT) and aspartate aminotransferase activities in participants exposed to particulate matter. An earlier study in Italy found that police employees exposed to urban air pollution had higher AST and ALT than the controls (office workers)[19]. A panel study among 545 elderly participants in Korea reported a positive association between short-term exposure to PM2.5 and AST, ALT, and GGT [20,21] investigated the associations of AST, ALT, and GGT with long-term exposure to PM2.5, PM with oxides (NOx) and nitrogen dioxide (NO2). Only PM2.5 was positively associated with GGT (5.1% increase (95% CI: 0.1% to 10.4%) per 2.8 μg/m3 increment in annual average PM2.5) and no significant associations were found for other air pollutants affecting liver enzymes[21]. A Taiwanese study revealed that long-term exposure to PM2.5 was positively associated with activity(44.17% increase (95% CI: 23.21% to 68.68%) per 12.2 μg/m3 PM2.5 increment) and the ALT significantly mediated the association between PM2.5 and incident hepatocellular carcinoma observed in this study 11. On the contrary, a study among 74 American children found that air-related pollution was not associated with ALT or AST Increase but was associated with cytokine [18], a marker of hepatocellular apoptosis[22]. This may be due to factors, such as the genetic variation in the study participants, the variation of PM levels and constituents, changes in the study design, and methods of enzyme activity determination used. The mechanism by which PM air pollution attacks liver enzymes is yet to be understood. Some researchers have suggested the induction of oxidative stress and inflammation as a major pathway [9]. Animal experiments found that PM exposure could lead to oxidative damage in hepatocytes [23]. Mice models also showed PM could induce genotoxicity in the liver, [24] although the molecular mechanisms remain to be elucidated. In addition, PM may also indirectly affect liver function by contributing to the pathogenesis of steatohepatitis through the alteration of lipid metabolism and induction of a pro-inflammatory milieu, resulting in the exacerbation of non-alcoholic steatohepatitis [9]. Moreover, in this research, a significantly lower difference existed in the mean serum lipase activity of participants exposed to soot compared with the control (p<0.05). The pancreas and lipase are essential for the digestion of lipids in our diet, and any dysfunction or disease affecting the pancreas or lipase production can lead to problems with lipid digestion and absorption, such as pancreatic insufficiency or cystic fibrosis 10 Low levels of lipase may be a sign of permanent

damage to cells in the pancreas that make lipase. This may happen in certain chronic (long-lasting) diseases, such as cystic fibrosis, and chronic pancreatitis. In this study, it is observed that long-term exposure to soot can predispose to chronic pancreatitis, this may be due to oxidative damage to pancreatic cells by particulate matter 2.5 (PM2.5). In addition to insulin resistance, PM exposure has been shown to exert toxic effects directly on the pancreas [25]. In a streptozotocin-induced mouse model of type-1 diabetes, PM from diesel exhaust fumes exacerbated pancreatic cell vacuolation and islet cell apoptosis, increased pancreatic amylase activity, increased expression of oxidative stress markers 8-isoprostane and superoxide dismutase and reduced levels of the antioxidant glutathione peroxidase [25]. In a rat model of gestational diabetes PM exposure induced maternal pancreatic inflammation indicated by diminished pancreatic glucose transporter-2 expression [26]. The previous study also observed an association between particulate matter 2.5 and pancreatic cancer [26]. Finally, there is a weak negative association between GGT and systolic blood pressure. The implication could be that GGT activity reduction may trigger an increase in systolic blood pressure but this may not have clinical significance as the association is very weak.

CONCLUSION

This study revealed higher Alanine transaminase (ALT) and gamma-glutamyl transferase (GGT) activities, and low pancreatic lipase activity of cooks exposed to soot. This suggests that exposure to soot could predispose to hepatic and pancreatic dysfunction

Acknowledgement

The authors would like to pay their most profound gratitude to the management and staff of Nnamdi Azikiwe University Teaching Hospital, Nnewi, and Reene Medical Diagnostic Laboratory, Awada, Anambra State, for the laboratory analyses of all biochemical parameters

Conflict of interest: None declared.

Authors Contribution

ACI, YH, and POM conceived and designed the research proposal. EUC and EIN performed sample collection, experiments, and data analysis. ACI, YH, and PCO contributed to the final version of the manuscript. All authors have read and approved the final manuscript

Data Availability

The data used to support the findings of this study are available from the corresponding author upon reasonable request

Funding: No funding sources

Ethical approval : The study was approved by the Ethics Committee of the Faculty of Health Sciences and Technology, Nnamdi Azikiwe University, Nnewi, Anambra State, Nigeria ERC/FHST/NAU/2021/MLS41

REFERENCES

1. Agarwal R, Awasthi A, Singh N, Mittal SK, Gupta PK, "Epidemiological study on healthy participants affected by agriculture crop-residue burning episodes and its relation with their pulmonary function tests" Int J Environ Res Public Health 2013; 23(4):281-295. https://doi.org/10.1080/09603123.2012.733933

2. Chuang HC, Jones TP, Lung SC, BéruBé KA, "Soot-driven reactive oxygen species formation from incense burning" Sci Total Environ 2011; 409(22):4781-4787. https://doi.org/10.1016/j.scitotenv.2011.07.041

3. Canagaratna MR, Onasch TB, Wood EC, Herndon SC, Jayne JT, Cross ES, Miake-Lye, R C, Kolb CE, Worsnop DR, "Evolution of vehicle exhaust particles in the atmosphere" J Air Waste Manag Assoc 2010; 60(10):1192-1203. https://doi.org/10.3155/1047-3289.60.10.1192

4. Glaser B, Dreyer A, Bock M, Fiedler S, Mehring M, Heitmann T, "Source Apportionment of Organic Pollutants of a Highway-Traffic-Influenced Urban Area in Bayreuth (Germany) Using Biomarker and Stable Carbon Isotope Signatures" Environ. Sci. Technol. 2005; 39(11):3911-3917. https://doi.org/10.1021/es050002p

5. Birky MM, Voorhees KJ, "The use of soot analysis as an investigative tool in aircraft fires" Aviat Space Environ Med. 1989; 60(10 Pt 2):B72-77.

6. Kamboures MA, Hu S, Yu Y, Sandoval J, Rieger P, Huang SM, Zhang S, Dzhema I, Huo D, Ayala A, Chang MCO, "Black carbon emissions in gasoline vehicle exhaust: a measurement and instrument comparison" J Air Waste Manag Assoc 2013; 63(8):886-901. https://doi.org/10.1080/10962247.2013.787130

7. Victor DG, Ramanathan V, Zaelke D,"Harmful soot spurs climate-policy action" Nat 2014; 517(7532):21-21. https://doi.org/10.1038/517021b

8. Wang R, Tao S, Shen H, Huang Y, Chen H, Balkanski Y, Boucher O, Ciais P, Shen G, Li W, Zhang Y, Chen Y, Lin N, Su S, Li B, Liu J, Liu W, "Trend in Global Black Carbon Emissions from 1960 to 2007" Environ. Sci. Technol. 2014; 48(12):6780-6787. https://doi.org/10.1021/es5021422

9. Kim, JW, Park S, Lim CW, Lee K, Kim B, "The Role of Air Pollutants in Initiating Liver Disease" Toxicol. Res. 2014; 30(2):65-70. https://doi.org/10.5487/TR.2014.30.2.065

10. Tan HH, Fiel MI, Sun Q, Guo, J, Gordon RE, Chen LC, Friedman SL, Odin JA, Allina J, "Kupffer cell activation by ambient air particulate matter exposure may exacerbate non-alcoholic fatty liver disease" J. Immunotoxicol. 2009c; 6(4):266-275. https://doi.org/10.3109/15476910903241704

11. Pan WC, Wu CD, Chen M.J, Huang YT, Chen CJ, Su HJ, Yang HI, "Fine Particle Pollution, Alanine Transaminase, and Liver Cancer A Taiwanese Prospective Cohort Study (REVEAL-HBV)" J Natl Cancer Inst 2016; 108(3):1-4. https://doi.org/10.1093/jnci/djv341

12. Ghouri N, Preiss D, Sattar N, "Liver enzymes, nonalcoholic fatty liver disease, and incident cardiovascular disease' A narrative review and clinical perspective of prospective data" Hepatol. 2010; 52(3):1156-1161. https://doi.org/10.1002/hep.23789

13. Center for American Progress. Center for American Progress; Center for American Progress. Charlton M. 2004, "Nonalcoholic fatty liver disease: A review of current understanding and future impact" Clin. Gastroenterol. Hepatol. 2012; 2(12):1048-1058. https://doi.org/10.1016/S1542-3565(04)00440-9

14. Ihim AC, Okeke O, Manafa PO, Ezejindu DN, Nnodim J, Obi PC," Effect of Exposure To Soot On Troponin I And Lipid Profile Levels" European j. Biomed. pharm. sci 2016;3(2):05-10.

15. Niranjan R, Thakur AK, "The Toxicological Mechanisms of Environmental Soot (Black Carbon) and Carbon Black, Focus on Oxidative Stress and Inflammatory Pathways" Front. Immunol. 2017; 1(1)1-3. https://doi.org/10.3389/fimmu.2017.00763

16. 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. https://doi.org/10.1093/ajcp/28.1.56

17. Ihim AC, Iloesie SC, Ogbodo EC, Oguaka VN, Meludu SC, " Effects of regular exercise on the liver function tests of male subjects in the college of health sciences, Nnamdi Azikiwe University, Nnewi campus, Anambra state, Nigeria" Int. J. Curr. Res. Med. 2018; 4(3):73-79. 18. Tietz NW, Shuey DF, Astles JR, "Turbidimetric measurement of lipase activity--problems and some solutions"

Clin. Chem.1987; 33(9):1624-1629. https://doi.org/10.1093/clinchem/33.9.1624

19. Tomao E, Baccolo TP, Sacchi L, Sio SD, Tomei F, "Harm to the liver among employees of the Municipal Police Force" Int J Environ Health Res, 2002;12(2):145-151. https://doi.org/10.1080/09603120220129300

20. Kim KN, Lee H, Kim JH, Jung K, Lim YH, Hong YC," Physical Activity- and Alcohol-dependent Association Between Air Pollution Exposure and Elevated Liver Enzyme Levels: An Elderly Panel Study" Int J Prev Med 2015; 48(3):151-169. https://doi.org/10.3961/jpmph.15.014

21.Markevych I, Wolf K, Hampel R, Breitner S, Schneider A, von Klot S, Cyrys J, Heinrich, J, Döring A, Beelen R, Koenig W, Peters A," Air Pollution and Liver Enzymes" Epidemiol. 2013; 24(6):934-935. https://doi.org/10.1097/EDE.0b013e3182a77600

22. Hsieh S, Leaderer BP, Feldstein AE, Santoro N, McKay LA, Caprio S, McConnell R, "Traffic-related air pollution associations with cytokeratin-18, a marker of hepatocellular apoptosis, in an overweight and obese pediatric population" Pediatr. Obes. 2017; 13(6):342-347. https://doi.org/10.1111/ijpo.12228

23. Meng Z, Zhang Q, "Effects of dust storm fine particles instillation on oxidative damage in hearts, livers, lungs of rats" J.Hyg. Res. 2006; 35(6):690-693.

24. Liu X, Meng Z," Effects of Airborne Fine Particulate Matter on Antioxidant Capacity and Lipid Peroxidation in Multiple Organs of Rats" Inhal Toxicol. 2005; 17(9):467-473. https://doi.org/10.1080/08958370590964467

25. Nemmar A, Al-Salam S, Beegam S, Yuvaraju P, Yasin J, Ali BH, "Pancreatic Effects of Diesel Exhaust Particles in Mice with Type 1 Diabetes Mellitus" Cell. Physiol. Biochem. 2014; 33(2):413-422. https://doi.org/10.1159/000356680

26. Yi L, Wei C, Fan W, Fine particulate matter (PM2.5), a risk factor of rat gestational diabetes with altered blood glucose and pancreatic GLUT2 expression. Gynecol. Endocrinol. 2017:1-6. https://doi.org/10.1080/09513590.2017.1318368