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PLASMA RENAL FUNCTIONS AMONGST 'PETROL STATION' ATTENDANTS IN OWERRI, SOUTH-EAST NIGERIA

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ABSTRACT

This study assesses the renal function of individuals who are occupationally exposed to 'petrol' vapour. It is a cohort study of 100 individuals comprising 50 'petrol station' attendants (test) in Owerri, Imo State, Nigeria, and 50 apparently healthy individuals who are 'non-petrol station' attendants (control). Information on demographic and health profiles were obtained, and venous blood samples were collected for the analysis of plasma creatinine, Na^+, K^+, Cl^- and HCO_3 using standard laboratory procedures. Results showed that plasma creatinine (1.17±0.30), K^+ (3.77 ± 0.55) and HCO_3^- (28.52±2.72) concentrations amongst 'petrol station' attendants to be significantly higher ($P<0.05$) compared to those of the control (0.87±0.18; 3.64±0.21 and 26.92±2.46 respectively). On the other hand, plasma Na^+ (131.70±4.16) and Cl^- (97.43±3.48) amongst 'petrol station' attendants were significantly lower compared to the control subjects (136.70±4.86 and 100.28±2.24 respectively). There was also a significant increase ($p<0.05$) in plasma creatinine, K^+ and HCO_3^- , and a significantly lower Na^+ and Cl^- amongst 'petrol station' with 3–6 years exposure when compared with those exposed for <1-2 years. These findings therefore, suggests that renal function impairment and nephrotoxicity, are associated with exposure to 'petroleum' vapours and its impact is time dependent.

Keywords: ***Renal/Kidney failure, Nephrotoxicity, Petroleum vapour, Owerri.***

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INTRODUCTION

Petrol (gasoline) is a volatile and inflammable petroleum derived liquid mixture primarily used for internal combustion of engines (Micyus *et al*., 2005). It consists of hydrocarbons (aromatic, saturated and unsaturated) and non hydrocarbons such as Nitrogen, Sulphur, Oxygen, Vanadium and Nickel (Micyus *et al*., 2005; Lewne et *al*., 2006). No alternative to petrol has been introduced into the Nigerian automobile industry. Therefore, millions of automobiles on Nigerian roads run on petrol or diesel fuel. Petrol contains Volatile Organic Compound (VOC) such as benzene which is limited by regulation to 6 – 8% of the content of PMS in Nigeria (Ross, 1996).

The volatility of petrol makes it readily available in the atmosphere any time it is dispensed, especially at petrol filling stations and depots (Amoore et a*l*., 1983). People are exposed to petrol fumes during fueling and refueling at petrol stations, but the petrol station attendants are at more risk by virtue of their occupational exposure (Lewne *et al*., 2006). Atmospheric concentration of petrol vapour (approximately 2000 ppm) is unsafe when inhaled even for a brief period of time (seconds). During fueling of vehicles, the concentration of petrol vapour in the air is between 20 and 200 ppm (Pranjic et a*l*., 2002; Lewne *et al*., 2006). This amount is higher when there is a long queue of cars to be fuelled, which is a usual occurrence during fuel scarcity (Rabble and Wong, 1996). Petrol inhalation is associated with dysfunctions that ange in severity from subtle cognitive impairment to encephalopathy and death (Cairney *et al*., 2002).

Occupational exposure to petrol has been reported to have toxic effects on various vital organs and systems, including respiratory, immune and nervous systems (Beckers, 1985). Organs like the heart, lungs, skin and kidneys are affected by the toxic effects resulting in various diseases and different forms of genotoxic, mutagenic (Rothman *et al*., 1996), immunotoxic, carcinogenic and neurotoxic

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manifestations (Klassen, 1990; d'Azevedo *et al*., 1996; Smith *et al*., 1996). Petrol vapour is readily detectable by most individuals at concentrations of below 1 – 2 ppm (Amoore *et al*., 1983).

Such occupational exposures to petrol vapour may increase olfactory threshold (Drinker *et al*., 1943), athough no human studies have determined the exact threshold for this effect. Nevertheless, the effects on central nervous system are readily apparent above 900 ppm (within a few minutes). The associated mobility resembles alcohol intoxication (dizziness, excitement, inco-ordination etc) and it may also act as anaesthetic, sometimes resulting in immediate loss of consciousness (Machle, 1941). This study therefore, assesses the renal function of individuals who are occupationally exposed to 'petrol' vapour.

MATERIALS AND METHODS

Study area: This cross-sectional study was carried out in Owerri, the capital of Imo State. It has an estimated population of 400,000 and lies within latitude $4^0 45^{'}$ N and $7^o 15^{'}$ N and longitude $6^o 50^{'}$ E and $7^o 25^{'}$ Eof the Greenwich Meridian.

It also occupies the area between the lower River Niger, upper Imo and middle Imo regions of Imo state (FRNOG, 2007).

Study population and Sampling: The study population comprised a total of one hundred (100) subjects distributed into fifty (50) 'petrol station' attendants (test group) and fifty (50) 'non-petrol station' attendants (control group). The sampled population comprised young male and female adults between the age of 22 – 33 years.

Subjects were recruited using simple randomly sampling method after giving inform consent to be included in the study area.

Ethical consideration: The principle of the declaration on the right of the subject was employed for this study. Before enrolment for the study, the participants were informed on the significance of the study and their consents were sort for and obtained.

Sample collection: After an informed consent was obtained from the subjects, four millilitres of blood samples were collected by venipuncture technique from subjects (both test and control) into lithium heparinized vacuum tubes, mixed gently and spun at 1000 rpm for 10 minutes at room temperature to obtain plasma which was extracted into plain tubes and frozen at -4^oC until required for further analysis.

Sample analysis: Determination of plasma creatinine was carried out using the Jaffe's method reported by Cheesbrough, (2005), plasma sodium, potassium and chloride using Biolyte 2000. A liquid based ion-selective electrode method while plasma bicarbonate was estimated using the acid-base back titration described by VanSlyke, (1922).

Statistical analysis: Data obtained was analyzed using SPSS software package version 16.0. The Students t- test was performed at 95% confidence level and a $p \leq 0.05$ was considered significant. Result was represented as Mean ± Standard Deviation and presented using suitable table.

RESULTS

The mean ages of the test and control were 26.62±3.74 years and 27.81±5.22 years respectively. Interestingly, all the test group study subjects have one form of education or the other, but majority (58.0%) have secondary education as their highest level of education.

Table 1 shows the results obtained in the investigation in their SI unit and reveals the mean ± standard deviation of plasma creatinine, Na^+, K^+, Cl^- and HCO_3^- of petrol station attendants and their control subjects respectively. Plasma creatinine and HCO_3^- were significantly higher ($p<0.05$) in the petrol station attendants than the control while plasma Na^+ and Cl^- were significantly higher ($p<0.05$) in the control than the petrol station attendant's values. Although the mean plasma K^+ was higher in the petrol station attendants than the control, the different was not statistically significant ($p>0.05$).

DISCUSSION

The kidney's role in maintaining a constant extracellular environment, which is required for adequate functioning of the cells, is achieved by excretion of waste products of metabolism (such as urea, creatinine and uric acid) and by specifically adjusting the urinary excretion of water and electrolytes, to compensate the net intake and endogenous production (Nwankwo *et al*., 2006). However, a deviation from the normal levels of these waste products/electrolytes in blood, due to several factors, indicates renal impairment (Gidado *et al*., 2001; Nwankwo *et al.,* 2006; Zannan *et al*., 2008), and subjects with kidney dysfunction may have a variety of different clinical presentations (Cotran *et al*., 1999). Some of these presentations may be asymptomatic, only detected on routine laboratory

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examinations from abnormal serum/ plasma catabolites (such as creatinine, urea, uric acid, blood urea nitrogen) and electrolytes (such as Na^+, K^+, Cl^-, HCO_3 etc) (Cotran *et al*., 1999).

Table 1: Mean ± standard deviation of plasma creatinine, Na^+, K^+, Cl^- and HCO_3^- of Petrol Station Attendants

Parameters	Control (n = 40)	Test (n = 60)
Creatinine (mg/dl)	0.87±0.18	1.17±030*
Na^+ (mmol/L)	136.70±4.86	131.70±4.16*
K^+ (mmol/L)	3.64±0.21	3.77±0.55
Cl^- (mmol/L)	100.28±2.24	97.43±3.48*
HCO_3^- (mmol/L)	26.92±2.46	28.52±2.72*

Results are Mean ± Standard Deviation; Values in a row having different superscript are statistically different at p≤0.05; Na^+ = Sodium ion; K^+ = Potassium; Cl^- = Chloride; HCO_3^- = Bicarbonate

Azotaemia is one of the clinical manifestations of renal disorders characterized by elevation of serum/ plasma urea, creatinine, and blood urea nitrogen levels (Cotran *et al*., 1999). In fact, a persistent elevation of serum creatinine levels in blood has been reported to be a risk factor for progression of chronic kidney disease to kidney failure (Mortada *et al*., 2001; Appel *et al*., 2003). In this regard, the observed increase in plasma creatinine and bicarbonate levels, and the slight increase in the levels potassium ion, as well as the decrease in sodium and chloride ions, amongst the petrol attendants following exposure to petrol, are in agreement with the work by Nwanjo and Ojiako, (2007), who reported a rise in serum creatinine levels in petrol station attendants, and that of Uboh *et al*. (2010), whose report showed a rise in plasma creatinine and potassium ion, and a decrease in sodium and chloride ions in rats exposed to petrol vapour.

The rise in creatinine levels might be due to a fall in Glomerular Filtration Rate (GFR) resulting in creatinine retention in blood, while the elevation of HCO_3^- levels might be due to acid-base imbalance triggered by CO_2 retention following impaired alveolar ventilation. This causes a rise in PCO_2 and the compensatory changes ultimately trigger plasma bicarbonate ion level elevation (Vander *et al*., 2001). On the other hand, the low Na^+ and Cl^-levels could be due to the loss of Na^+ and concomitant loss of Cl^- in the urine. The slight rise in K^+ may be due to reduced renal excretion of K^+ and the exchange of Na^+ for H^+ to compensate for the rise in HCO_3.

These results suggest that the absorbed constituents of petrol vapour might have reacted with the renal tissues to cause injury to the glomerulus, the tubules, or both. As such, nephrons are lost leading to kidney function impairment. Although the effect of inhaled petrol on renal tissues might not be dependent on age and sex, it is probable that the effect of inhaled petrol on renal tissues could depend on the exposure time, possibly as a result of the phenomenon of bioaccumulation, which is associated with direct transfer of compounds through body surface into the circulatory fluids in a process known as biconcentration (WHO, 1998). Thus, exposure to petrol should be considered as a predisposing factor for renal function impairment in humans.

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REFERENCES

Amoore, J., Von Burg, R. and Whittemore, I. (1983): Detectibility of gasoline by its odor. *The Toxicologist*.22nd Annual meeting of the Society of Toxicologists. March 6-11.

Appel, L. J., Middleton, J., Miller, E. R., Lopkowitz, M., Norris, K., Agodoa, L.Y., Bakris, G. and Douglas, J. G. (2003): Human Biology and health. Englewood Cliffs, New Jersey, USA: Prentice Hall.

Becker, C.E. (1985): Principles of occupational medicine, In: Cecil Textbook of Medicine, 17th ed., J.B. Wyngaarden and L.H. Smith, (jr eds.). W.B. Saunders Co. Pp. 2277-2279.

Cairney, S., Maruff, P., Burns, C.B. and Currie, B.J. (2002): The neurobehavioural consequences of petrol sniffing. *NeurosciBiobehav rev.,* 26:81-89.

Cheesbrough, M. (2005): Clinical chemistry tests. In: District laboratory practice in tropical countries. Cambridge university press.Low price edition. 2nd ed. Pp. 333-337.

Cotran, R. S., Kumar, V. and Collins, T. (1999): The kidney. In: Robbins pathologic Basis if disease, 6th edition. WB.Saunder Co. Philadelphia. Pp. 930 – 996.

d'Azevedo, P.A., Tannhauser, A.L. and Tannhauser, S.L. (1996): Haematological alternations in rats from xylene and benzene. *Vet. Human Toxicol.,*38(5):340-344.

Drinker, P., Yaglou. C.P. and Warren, M.F. (1943): The threshold toxicity of gasoline vapour. *J. Ind. HygToxicol.,* 25:225-232.

Federal Republic of Nigeria Official Gazette (2007): Summing the 3 L.G.A.s making up Owerri as per: Legal notice on publication of the details of the breakdown of the national and state provisional totals of 2006 census.

Gidado, A., Bashirat, J., Gana, G. M., Ambi, A. A., Milala, M. and Zanna, H. (2001): Effects of aqueous extract of the seeds of *Daturastramonium* on some indices of liver and kidney function in rats. *Nig. J. Exp. Applied Biol*., 2 (2): 123-127.

Klassen, C.D. (1990): Non metallic environmental toxicants; air pollutants, solvents, vapour and particles. In: Goodman and Gillman's textbook, the pharmacological Basis of Therapeutics, 8th ed., A.G. Gillman, T.W. Rall, A.S. Niuo and P. Taylor (eds.) NY, Pergamon Press., Pp. 1596-1614.

Lewne, M., Nise, G., Lind, M.L. and Gustavsson, P. (2006): exposure to particles and nitrogen dioxide among taxi, bus lorry drivers. *Int. Arch. Occup. Environ. Health.,*79: 220-226.

Machle, W. (1941): Gasoline intoxication. *Journal of the American Medical Association.,* 117: 1965-1971.

Micyus, N.J., McCurry, J.D. and Seeley, J.V. (2005): Analysis of aromatic compounds in gasoline with flow-switching comprehensive two-dimensional gas chromatography. *J. chromatogr. A*., 1086: 115-121.

Mortada, W. I., Sobh, M. A., El-Defrawy, M. M. and Farahat, S. E. (2001): Study of lead exposure from automobile exhaust as a risk for nephrotoxicity among Traffic policemen. *AM J. Nephrol.,* 21:274 – 279.

Nwanjo, H. U. and Ojiako, O. A. (2007): Investigation of the potential health hazards of petrol station attendants in Owerri, Nigeria. *J. Appl. Sci. Environ. Manage.* Vol. 11 (2) 197 – 200.

Nwankwo, E. A., Nwankwo, B. and Mubi, B. (2006): Prevalence of impaired kidney in hospitalized hypertensive patients in Maiduguri, Nigeria. *Int. J. intern med.,* 6(1).

Pranjic, N., Mujagic, H., Nurkic, M., Karamehic, J. and Pavlovic, S. (2002): Assessment of health effects in workers at gasoline station. *Basn J. Basic Med. Sci.,* 2:35-45.

Ross, D. (1996): Metabolic basis of benzene toxicity (Review). *Euro. J. Haematol.,* 60:111-118.

Rothman, N., Li, G.L.,Dosemeci, M., Bechtold, W.E., Marti, G.E. and Wang, Y.Z. (1996): Haematotoxicity among Chinese-workers heavily exposed to benzene. *Am, J. Ind. Med*., 29(3):236-246.

Smith, T.J., Mallet, A.K. and Brantom, P.G. (1996): Ninety day feeding study in Fischer of 344 rats of highly refined petroleum derived food grade white oils and waves. *Toxicol.Pathol.,* 24: 214-230.

Uboh, F., Akpanabiatu, M., Ekaidem, I., Eteng, M. and Eyong, E. (2010): Exposure to gasoline and KerosineVapours: a risk factor for nephrotoxicity in rats. *The int. Journal of Toxicology.*Vol 7. No 2.

Vander, A., Sherman, J. and Luciano, D. (2001): Human physiology: The mechanism of body's function. McGraw Hill 8th ed. Pp. 506 – 551.

VanSlyke, D. (1922): Titration method of bicarbonate estimation, *J. Biol. Chem.,* 52:495.

World Health Organistion (1998): Environmental health criteria 206- methyl tetra-butylether. *Int. prog. Chem. Safe* II.

Zannan, H., Adeniji, S., Shehu, B. B., Modu, S. and Ishaq, G. M. (2008): Effects of aqueous suspension of the root of Hyphaenethebacia (L): mart on some indicators of liver and kidney function in rats. *Journal of pharmacolToxicol.,*3 (4):330-334.

AUTHOR'S CONTRIBUTION

Festus O.O. supervised this work with the assistance of Dada F.L., Iweka F.K., Eyaufe A.O., Osagie R.N., Akiyang E.E. All authors contributed to the study.