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Cardiovascular Risks and Impaired Lipid Metabolism in Asymptomatic Petroleum Depot Workers in Calabar Metropolis, Nigeria

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ABSTRACT

The health concerns about working in a petroleum depot without wearing a Personal Protective Equipment (PPE) warranted this study. The anthropometric, lipid profile and blood pressure status were assessed by standard methods in asymptomatic adult male depot workers (n = 64) and male university students (n = 64), in Calabar metropolis, Nigeria. The concentration (mmol L⁻¹) of total cholesterol (5.21 ± 0.90), triacylglycerol (1.23 ± 0.20) and very low density lipoprotein (0.56 ± 0.19) respectively in the serum of the depot workers were significantly (p<0.05) higher than in the control (5.09 ± 0.04 , 1.15 ± 0.04 and 0.52 ± 0.09). The body weight (73.75 ± 0.11 kg), height (1.72 ± 0.1 m) and body mass index, BMI, (24.58 ± 0.73 kg m⁻²) of the depot workers were higher (p>0.05) than that of the control. The Blood Pressure (BP) of the exposed group ($120/90\pm1.38$ mmHg) and the control ($120/80\pm1.14$ mmHg) differed only in the Diastolic Blood Pressure (DBP) by 10 mmHg and in the calculated systolic to diastolic blood pressure ratio (SBP:DBP) by 0.17. The study suggested cardiovascular risks and impaired lipid metabolism in the petroleum depot workers. The health implications of this study warrant a follow up perhaps, in a larger population and sample size. The study underscored the need for the petroleum depot workers to wear personal protective equipment and to assess their health status on a regular basis.

Key words: Lipid metabolism, body mass index, cholesterol, triacylglycerol, cardiovascular risk

INTRODUCTION

Pollution has potential adverse effect on the environment and on human health (Kampa and Castanas, 2008). Petroleum products which are derived from the fractional distillation and cracking of crude oil and used for various purposes, including cooking (Standeven and Goldsworthy, 1994), could contribute to a significant environmental pollution and adverse effect on human health. Chronic exposure of animals to petroleum products vapours resulted in adverse effects on their bone marrow, spleen and lymph nodes (Marieb, 1995) and in the detection of hydrocarbons in their blood (Zahlsen *et al.*, 1993) and urine (Burgaz *et al.*, 1992) samples. Pollutants derived from petroleum products are organic in nature hence persist and accumulate to a high concentration in the environment (Schecter *et al.*, 2006). They are volatile, hence could be readily inhaled. They bio-accumulate to a high concentration in animals due to the stability of their complexes with lipids (Kampa and Castanas, 2008). Thus, toxic concentration of the petroleum products could persist in

the environment consequently exposing humans to a high concentration of the petroleum products in the course of their daily human activities in the home and work places (Ujowundu *et al.*, 2011) with severe effect (Tietenberg, 2006).

The degree of such exposure is expectedly higher in those that work in the tank farm and gantry areas of petroleum storage depots (Rothman *et al.*, 1996). Depot workers in Calabar metropolis in Nigeria neither checked their health status nor wore the personal protective kits which could result to sudden health complications following unprotected exposure to the petroleum products vapours. These warranted this study aimed at assessing the anthropometric, lipid profile and blood pressure status of petroleum depot workers in Calabar metropolis, Nigeria, to gain an insight on their health status. These and other bioindicators have been used to assess the physiological status in animals (Egbuonu *et al.*, 2010a, 2012; Egbuonu and Osakwe, 2011; Ambrose *et al.*, 2012; Egbuonu, 2012; Egbuonu and Ezeanyika, 2013). The results of the present study may contribute to previous knowledge and form a basis for further studies on the subject.

MATERIALS AND METHODS

Chemicals and equipment: The various chemicals used for the biochemical analyses were of analytical grade and product of reputable companies. The chemicals and equipment used were provided by the Chief Technologist, Chemical Pathology Laboratory, University of Calabar Teaching Hospital, Calabar Cross River State Nigeria.

Experimental design: The study was conducted on asymptomatic male workers in 10 petroleum depots and male students of University of Calabar, in Calabar metropolis, Nigeria. The depot workers neither knew their health status nor wore personal protective equipment. The participants were aged between 18 and 35 years and must be either a staff of any of the petroleum depots or a student of the said university for 2-4 years. The age range, 18-35 years and duration, 2-4 years criteria were set for this study to fairly match the age and duration of the control. The study made use of a simple random cluster sampling (Lindell and Whitney, 2001) with standard questionnaire (Franklin *et al.*, 2000) for screening the respondents to select relevant subjects for the study. The respondents were informed with assurance of confidentiality and use of resultant information for the purpose of the study. Their oral consent was obtained before the administration of questionnaire, anthropometric measurement and collection of blood sample. Ethical clearance was obtained from the Ethical Committee of Biochemistry Department, Michael Okpara University of Agriculture, Umudike, Nigeria. All biochemical tests were conducted at the Chemical Pathology Laboratory of University of Calabar Teaching Hospital with the assistance of an appointed laboratory technologist.

Sample size determination: A total of 210 male petroleum depot workers who met the set criteria (age range, 18-35 years, depot staff for 2-4 years) served as the exposed study population. Out of the 210 questionnaires administered to the exposed study population, 132, representing 62.86%, were returned. A total of 46 of the returned questionnaires were discarded for various reasons, including improper filling. Twenty two respondents declined consent for anthropometric measurements and sample collection. The remaining 64 respondents representing 30.47% of the exposed study population, that gave their consent for anthropometric measurements and sample collection the exposed group sample size. Based on the exposed

group sample size, the control group sample size was set at 64, applying the set criteria (age range, 18-35 years, student in university for 2-4 years).

Measurement of blood pressure, height and weight: Blood pressure of the control and exposed respondents was measured at the University of Calabar and the various depots, respectively by the method of Wesseling (1995). Aneroid Sphygmomanometer model OGO2, Kenzmedico Company Limited, Saitama Japan was used, with each of the participant seated in a quiet place on a chair with a back support. Four readings were taken at 5 min intervals and the mean value of the two closest readings used as the final value.

Each of the respondent's height was measured using a measuring tape, U.S. metric tape, model 52717, Texas USA. The tape was placed vertically on a smooth wall surface at a right angle to the floor with the participant standing without shoes and backing the measuring tape. The height reading was taken as the right angle contact point with a horizontal line from the base of the head (not hair). Four readings were taken and mean value of two closest readings used as the final value.

The weight (kg) of the respondents was measured with a weighing scale (Analogue weighing scale, Model Jindal 35, Delhi, India, with a maximum capacity of 150 kg). The scale was placed on a smooth horizontal surface with each participant stepping on the scale without shoes. Four readings were taken and the final value calculated as the mean value of the two closest readings.

Body mass index calculation: The Body Mass Index (BMI) was calculated from data obtained in this study using the formula:

BMI (kg m⁻²) = $\frac{\text{Weight (kg)}}{\text{Square of the height (m}^2)}$

Blood sample collection: Venous blood was taken from a peripheral vein on any of the arms of each respondent and transferred into a sterile 10 mL sample bottle, allowed to clot and centrifuged at 2000 rpm for 10 min (using Universal 320 Centrifuge, Hettich-Germany) to obtain the serum. The serum was separated using a Pasteur pipette into a labeled 5 mL sample container and stored in the freezer until used for the determination of each of the respondent's total cholesterol and triacylglycerol concentrations. The serum samples were thawed (brought out from the freezer and allowed to stand at room temperature in order to attain the ambient temperature), before commencing the biochemical determinations.

Determination of serum total cholesterol and triacylglycerol concentrations: The serum total cholesterol concentration was determined with Giesse diagnostic kit based on the enzymatic colorimetric estimation of cholesterol ester hydrolysis (following series of reactions catalysed by cholesterol esterase, cholesterol oxidase and cholesterol peroxidase) to release oxygen. The oxygen subsequently reacted with 4-amino antipyrine to form a red coloured compound (dye) that was measured with a colorimeter (Lasany Auto Colorimeter, Model 13586, India) at 520 nm and the concentration calculated from the relation:

 $\frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{Concentration of standard} (5.2 \text{ mmol } \text{L}^{-1})$

The triacylglycerol concentration in the serum was determined with Giesse diagnostic kit based on the enzymatic colorimetric estimation of triacylglycerol on conversion by the actions of lipoprotein lipase, glycerol kinase glycerol-3-phosphate oxidase and peroxidase to release oxygen which reacted with 4-amino antipyrine and phenol derivative to give a red colour that was measured with a colorimeter (Lasany Auto Colorimeter, 13586, India) at 520 nm and the concentration calculated from the relation:

 $\frac{\text{Absorbance of test}}{\text{Absorbance of standard}} \times \text{Concentration of standard} (2.3 \,\text{mmol}\,\text{L}^{-1})$

Very low density lipoprotein calculation: The very low density lipoprotein was estimated as in Roger *et al.* (1997) based on the Friedewald formula (Friendwald *et al.*, 1972) thus:

Very low density lipoprotein (mmol L^{-1}) = 0.456×Total triacylglycerol concentration

Data analysis: Data was analyzed by simple student t- test using Statistical Package and Service Solutions (SPSS) for windows version 7. Mean differences at p<0.05 were considered statistically significant. Results were presented as Mean±Standard Deviation (SD).

RESULTS

As shown in Fig. 1-3 the serum total cholesterol $(5.21\pm0.90 \text{ mmol } \text{L}^{-1})$, triacylglycerol $(1.23\pm0.20 \text{ mmol } \text{L}^{-1})$ and very low density lipoprotein $(0.56\pm0.19 \text{ mmol } \text{L}^{-1})$ for the exposed group were significantly (p<0.05) higher than that in the control group 5.09 ± 0.04 (mmol L^{-1}), 1.15 ± 0.04 (mmol L^{-1}) and $0.52\pm0.09 \text{ mmol } \text{L}^{-1}$, respectively.

The slightly higher value in the exposed than in the control groups, respectively for the body weight $(73.75\pm0.11, 73.80\pm0.04 \text{ kg})$, height $(1.72\pm0.1, 1.70\pm0.08 \text{ m})$ and body mass index, BMI, $(24.58\pm0.73, 24.54\pm0.68 \text{ kg m}^{-2})$ was not significant (p>0.05) (Fig. 4-6).

The Blood Pressure (BP) of the exposed group (120/90±1.38 mmHg) and the control (120/80±1.14 mmHg) differed only in the Diastolic Blood Pressure (DBP) by 10 mmHg (Fig. 7) and in the calculated systolic to diastolic blood pressure, ratio (SBP:DBP) by 0.17 (Fig. 8).



Fig. 1: Serum total cholesterol concentration (mmol L^{-1})



Fig. 2: Serum triacylglycerol concentration (mmol L⁻¹)



Fig. 3: Serum very low density lipoprotein cholesterol concentration (mmol L^{-1})



Fig. 4: Weight (kg) of the test and control groups



Fig. 5: Height (m) of the test and control groups



Fig. 6: Body mass index (kg m^{-2}) of the test and control groups



Fig. 7: Diastolic blood pressure (mmHg) of the test and control groups

DISCUSSION

Petroleum products which are used for various purposes, including cooking (Standeven and Goldsworthy, 1994) could contribute to a significant environmental pollution and adverse effect on human health. For instance, benzene a major constituent of petroleum products is a known toxicant



Fig. 8: Blood pressure ratio (SBP:DBP) of the test and control groups

(Kirkeleit *et al.*, 2008). Petroleum products are volatile, hence are readily inhaled. Depot workers in Calabar metropolis, Nigeria neither checked their health status nor wore personal protective equipment to minimize the adverse health effects of petroleum products vapours, warranting this research. Preliminary survey response revealed that the petroleum depot workers did experience dizziness, respiratory tract and eye irritation which are in consonance with previous reports (Klassen, 1990; Ross, 1996; Rothman *et al.*, 1996; Smith *et al.*, 1996).

Body Mass Index (BMI), significantly predicted health risks in animals, including metabolic syndrome (Indhavivadhana *et al.*, 2010), diabetes (Chaabo *et al.*, 2010) and has been used in assessing animal health status (Egbuonu *et al.*, 2013; Ezeanyika *et al.*, 2008). High weight could indicate obesity (Van Herpen and Schrauwen-Hinderling, 2008). The BMI and the corresponding determinants, weight and height were higher in the exposed group compared to the control group. This result agreed with the findings of Anikeh *et al.* (2014), who reported a significant increase in body mass index in female petrol station attendants. However, the observed differences in this study were not significant (p>0.05) hence negligible.

In the present study, the total cholesterol concentration was significantly higher (p<0.05) in the petroleum depot workers, suggesting impaired lipid metabolism and possibly liver disease. This result agrees with previous result and suggestion, though in rats exposed to gasoline fumes (Uboh *et al.*, 2005), warranting similar study and assessment on biomarkers of liver functions. In apparent support of the present result on cholesterol concentration, the triacylglycerol concentration was higher (p<0.05) in the petroleum depot workers. This further suggests the compromised function of the Very Low Density Lipoprotein (VLDL) cholesterol in transporting the accumulated triacylglycerol back to the adipose tissue (Egbuonu and Osakwe, 2011). This probably resulted in higher serum VLDL cholesterol in the depot workers than in the control observed in this study.

Compromised function of VLDL is a risk factor for arteriosclerosis (Ochei and Kolhatkar, 2008) by impairing lipid metabolism. Impaired lipid metabolism resulting in higher total cholesterol and triacylglycerol concentrations in the serum may predispose animals to cardiac diseases (Egbuonu *et al.*, 2010b; Uboh *et al.*, 2005). This could imply cardiac dysfunction risk in the depot workers. Lipids are atherogenic hence could cause high blood pressure by forming plaques that narrow the blood vessels (Egbuonu and Ezeanyika, 2012). Interestingly, the diastolic blood pressure in the petroleum depot workers group was higher while the systolic to diastolic blood pressure ratio (SBP:DBP) was lower than that in the control, suggesting stage 1 hypertension and pre-hypertension (Tanne, 2003).

CONCLUSION

The study suggested cardiovascular risks and impaired lipid metabolism in the petroleum depot workers. The health implications of this study warrant a follow up perhaps in a larger population and sample size. The study highlighted the need for the petroleum depot workers (and petrol station pump attendants) to wear a personal protective equipment and to assess their health status on a regular basis.

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