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ASSESSMENT OF LIPID PROFILE IN AUTOMOBILE MECHANICS IN EKPOMA, EDO STATE

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ABSTRACT
Automobile mechanics are professionals who specialize in the repair and maintenance of cars of various sizes. By nature of their job, they are constantly exposed to gasoline fumes or premium motor spirit. Hyperlipidaemia is well known to be one of the major basic factors for atherosclerosis and cardiovascular disease which can be modified either by proper life style changes or medical management or by the combination of both. This study was carried out to assess the plasma lipid profile in automobile mechanics in Ekpoma and environs, Edo State, Nigeria. A total of 100 individuals comprising of 60 automobile mechanics and 40 apparently healthy individuals as controls were recruited for the study. Plasma levels of total cholesterol (CHL), triglycerides (TG), high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) were determined using standard enzymatic methods and also, their body mass index (BMI) were determined. The mean±SD values of CHL, TG, HDL, LDL, VLDL and BMI of automobile mechanics were 7.51 ± 2.78, 2.08 ±1.01, 1.27 ±0.21, 5.39 ±2.70, 0.94 ±0.46 and 19.78 ±2.59 respectively. The result showed significant higher levels of CHL ($p<0.05$), TG ($p<0.05$), LDL ($p<0.05$) and VLDL ($p<0.05$) when compared with the control. There were no significant difference ($p>0.05$) in HDL and BMI level of both groups. When the lipid profile of automobile mechanics were compared according to age rang, significant different ($p<0.05$) were observed in TC and LDL. Therefore, there is a likelihood that automobile mechanics are at high risk of hyperlipidaemia which is associated with atherosclerosis and cardiovascular diseases. This effect is due to constant exposure to gasoline vapour through mouth sucking, washing of arms, inhalation and aging.

KEYWORDS: Hyperlipidaemia, mouth sucking, washing of arms, inhalation and aging.

INTRODUCTION
Automobile mechanics repair and maintain cars. Some mechanics work on all parts of cars while others specialize in one area or on one type of car. Generally, mechanics work indoors in well ventilated and lighted repair shops. Some of which are drafty and noisy. Mechanic frequently work with dirty and greasy parts, and in awkward positions. Auto-mechanics are among the job professionals in proximity to diesel and gasoline or premium motor spirit exhaust whose components are known to be toxic (Udonwa *et al.*, 2009).

Auto-mechanics are exposed to used gasoline engine oil (UGEO) that accumulates on automobile parts, tool, workbenches, floors and equipment. Given the carcinogenic potential of UGEO, they are encouraged to wash their hands regularly to remove these contaminants. Premium motor spirit is petroleum derived volatile compound mixture, gottenfrom fractionation of petroleum. It is the primary fuel in the internal combustion engine and some electrical generating machine. It consists mostly of aliphatic hydrocarbons, cyclic hydrocarbons and aromatic hydrocarbon. Additives such as tetraethyl lead, tetramethyl lead, methyclyclopentadienyl manganese carbonyl (MMT) and ethanol are added to petrol to improve its quality (octane rating) or to prevent engine knocking (Smith,1992). Polycystic aromatic hydrocarbons (PAH) compounds can be readily absorbed through the skin. For automotive mechanics, this may be the major rout of entry. Workers exposed to PAH are at an increased risk for lung, urinary tract, brain and skin cancers (Bofetta*et al.*,1997).

In general, chlorinated hydrocarbons are considered to be more potential hepato and nephro toxicants in humans (Torkelson and Rowe, 1985). Like other known xenobiotics, the chemical pollutions from gasoline vapours is metabolically transformed into various metabolites in the body. Some of these metabolites are very reactive, interactive in various ways with metabolizing, transporting and excreting tissues to elicit

toxic effects. The interaction of these various metabolites with liver tissues may cause cellular injury. Hence, the overall functionality of the liver may be compromised (Lauwerys*et al.*, 1985).

Gasoline is one of the distilled fractions of crude petroleum which contains aliphatic, aromatic and a variety of other branched saturated and unsaturated hydrocarbons (Ihediaha and Chineme, 2005). The toxicological effect of gasoline is due to as an interference with the cellular or subcellular processes, which leads to a disruption of the normal metabolism of a living organism upon exposure to it (Beckles *et al.*, 1985). Petroleum hydrocarbon magnified their toxic effects by competing with some endogenous metabolites or blocking some pathways. This interference may or may not be lethal (Dede and Kagbo, 2001). Aliphatic and aromatic hydrocarbons are the major constituents of petroleum and petrochemical products, like other xenobioticsthey are metabolized in the liver to a greater extent. Acute-duration inhalations of gasoline or PMS have been associated with irritation, headache, dizziness, nausea, euphoria and drowsiness (Schaumburg and Spencer, 1978). Studies with rats and mice with chronic inhalation exposure to gasoline vapours have found hepatocellular tumors in female mice and nephropathy andrelated renal tumors in male rats, based on these petrol fumes-induce injury (Uboh *et al., 2005*). There is a significant increase in the level of serum total cholesterol and triglyceride which is an indication that oral exposure to gasoline affect lipid metabolism (Robertson *et al.*, 2007). On one hand, lipid metabolism is affected once there is liver damage since the disturbance of cell membrane integrity is likely to cause some membrane lipids to be released into circulation and can also cause the tissue to compromise its effectiveness in regulating lipid metabolism. Such imbalance is associated with increased β-oxidation of fatly acids by mitochondria, peroxisomal and cytochromP450 ZEI (CYP2EI) pathways. The oxidative processes produce free element, H_2O_2 and reactive oxygen species (ROS) which depletes the potent ant-oxidants, glutathione and vitamin E (McCullough, 2002). Increased lipid per-oxidation and oxidative stress in hepatocytes of male and female rats have been reported to be gasoline vapour exposure (Uboh *et al.*, 2007). Such reactive oxygen species (ROS) as hydroxyl and superoxide radicals are known to provoke severe cellular alteration resulting in cell damage or death, due to their high reactivity. These species attack such important cell constituent as protein, lipid and nucleic acid and lipid peroxides that accumulate due to lipid per-oxidation are known to be very harmful to cells and tissue (Linden *et al.*, 2008). There is therefore a likelihood that exposure to gasoline predisposes the subject to atherosclerotic condition, due to its generation of free radicals that affect lipid metabolism in the liver (Zahlsen *et al.*, 1993). Because the liver has a central role in the maintenance of lipid homeostasis, the presence of gasoline alters membrane integrity of the liver and increases serum lipids concentration. The increased total cholesterol, triglyceride, LDL cholesterol and decreased HDL-Cholesterol are implicating risk factors for atherosclerosis and related cardiovascular diseases (Halim *et al.*, 1997).

On the other hand, lipid profile or lipid panel is a panel of blood tests that serves as an initial broad medical screening tool for abnormalities in lipids, such as cholesterol and triglycerides. The results of this test can identify certain genetic diseases and can determine approximate risks for cardiovascular disease, certain forms of pancreatitis and other diseases (Sidhu and Naugler, 2012). Lipid profile is a group of tests that are often ordered together to determine risk of coronary heart disease (Ugonabo *et al.*, 2007). It is a good indicator of whether someone will have a heart attack or stroke caused by blockage of blood vessels or atherosclerosis (Nnodim *et al.*, 2012).

Since exposure to gasoline and PMS is linked to alteration of lipid profile which is a good indicator of whether someone is predisposed to stroke or heart attack, this work is therefore aimed at determining the level of lipid profile in automobile mechanics.

MATERIALS AND METHODS

AREA OF STUDY

This study was carried out in Ekpoma, the administrative headquarters of Esan West Local Government Area of Edo State, Nigeria. The area proper lies between latitude 60C 45 North of equator and longitudes 60 5 and 60 8 East of the Greenwhich meridian. Ekpoma area falls within the rain forest/savannah transitional zone of South Western Nigeria. Ekpoma has a population of 89,628 and 127,718 at the 1991 and 2006 population census respectively (NPC, 2006). Majority people in this area are civil servants, traders, business men and women, transporters, farmers, teacher/lecturers and students by occupation. Ekpoma since after its designation as headquarters and as the host of state owned university (Ambrose Alli University), the town has grown into an urban centre. Ekpoma is made up of many quarters, including Eguare, Iruekpen, Emaudo, Ujuolen, Ihumudumu, Illeh, Uke, Uhiele, Ujemen, ukpenu, Egoro, Emuhi, Igor and Idumebo (Aziegbe, 2006).

INCLUSION AND EXCLUSION CRITERIA

Automobile mechanics in steady state involving repair and maintenance of automobile vehicles for minimum of seven years and above were used while those that were not up to seven years in repair and maintenance were excluded from the study.

SAMPLE SIZE

A total number of one hundred samples were collected comprising of sixty automobiles mechanics and forty apparently healthy individuals (control).

RESEARCH DESIGN

This project work was carried out within three months. Blood samples were collected from steady-state automobile mechanics in Ekpoma, Edo State. The blood samples were centrifuged and plasma was immediately separated from the cells into plain containers with label corresponding to initial blood sample bottle. The serum samples were stored frozen until the time for analysis. Laboratory analysis was carried out for Cholesterol, HDL and triglycerides, VLDL and LDL were calculated.

SAMPLE COLLECTION

Five millilitres of venous fasting blood was collected from the subjects and dispensed into a lithium heparin container and mixed gently. The blood samples were centrifuged at 4000rpm for 5minutes. The plasma was separated into a clean dry plain container and stored frozen until analysed at room temperature.

ANALYTICAL METHODS

ESTIMATION OF SERUM TOTAL CHOLESTEROL

Enzymatic Endpoint method (CHOD-PAP) (Richmond, 1973).

Procedure

Ten microlitres of distilled water, standard and samples were dispensed into tubes labelled blank, standard and sample respectively. One millilitre of cholesterol reagent was added into the respective test tubes, and the contents were mixed and incubated at 37^{o}C for five minutes. The absorbance of standard and samples were measured against blank at a wavelength of 500nm using spectrophotometer.

Formula

$$\text{TC Conc. in sample} = \frac{\text{Absorbance of sample} \times \text{Conc. Std (5.33mmol/l)}}{\text{Absorbance of standard}}$$

Where: TC= Total cholesterol, Conc.= Concentraion and Std=Standard

SERUM TRIGYCERIDE ESTIMATION**:** Colorimetric method (Trinder, 1969).

Procedure

Ten microlitres of distilled water, standard and samples were dispensed into tubes labelled blank, standard and sample respectively. One millilitre of triglyceride reagent was added into the respective test tubes and the contents were mixed and incubated at 37^{o}C for five minutes. The absorbance of standard and samples were measured against blank at a wavelength of 500nm using spectrophotometer.

$$\text{Conc. of TG in sample} = \frac{\text{Absorbance of sample} \times \text{Conc. of Std (2.17mmol/l).}}{\text{Absorbance of standard}}$$

SERUM HIGH DENSITY LIPOPROTEIN-CHOLESTEROL (HDL-C) ESTIMATION:

Precipitation method (Lopes-virella, 1972).

Procedure

Stage I

Two hundred microlitres of standard/sample was dispensed into test tube labelled standard/sample. Five hundred microlitres of precipitant was added into the test tubes and mixed well, the contents were allowed to stand for ten minutes at room temperature and then centrifuged for ten minute at 4000rpm. The supernatant was separated and the cholesterol content was estimated using CHOD-PAP method.

Stage II

One hundred microlitres of distilled water, standard supernatant and sample supernatant was added into test tubes labelled blank, standard and sample respectively. One millilitre of cholesterol reagent was added into the respective test tubes; the contents were mixed and incubated at 37^{o}C for five minutes in the water bath. The absorbance of standard and sample were measured against blank at the wavelength of 500nm using spectrophotometer.

Formula

$$\text{Conc. of HDL-C in sample} = \frac{\text{Absorbance of sample} \times \text{Conc. of Std (5.33mmol/l).}}{\text{Absorbance of standard}}$$

SERUM LOW DENSITY LIPOPROTEIN-CHOLESTEROL (LDL-C) ESTIMATION

Low density lipoprotein (LDL) was calculated from total cholesterol, triglyceride and HDL cholesterol using the Warnick equation (Warnick *et al.*, 1990).

LDL-Cholesterol(mmol/l) = Total Cholesterol – Triglyceride/ 2.2 – HDL-cholesterol

LDL-Cholesterol(mg/dl) = Total Cholesterol – Triglyceride/5 – HDL-Cholesterol.

SERUM VERY LOW DENSITY LIPOPROTEIN-CHOLESTEROL (VLDL) ESTIMATION

Very low density lipoprotein was calculated from trtglyceride using theWiarnick equation (Warnick*et al.*, 1990).

VLDL-Cholesterol (mmol/l)=Triglyceride÷2.2

STATISTICAL ANALYSIS

Student's t – test and Pearson correlation at $p<0.05$ level of significance were used to compare results in both the control and the test group (all results were reported as mean + standard deviation) using Window SPSS version 20.0, confident interval of values $p<0.05$ was considered significant.

RESULTS

Table 1 shows a significance increase ($p<0.05$) in the values of CHL (7.51±2.78mmol/L), TG

(2.08±1.01mmol/L), LDL (5.39±2.70mmol/L) and VLDL (0.94±0.46mmol/L) in the automobile mechanics when compared with the control groups of values of 2.60±0.50mmol/L, 1.29±0.32mmol/L, 0.84±0.57mmol/L and 0.57±014mmol/L respectively. There was no significant increase ($p>0.05$) in the values of HDL (1.27±0.21mmol/L) in automobile mechanics when compared with the control groups of 1.24±0.24mmol/L (Table 1).

Table 2 shows the mean±SD values TC, TG, HDL, LDL and VLDL of automobile mechanics according to the age range. There was significant different ($p<0.05$) in the level of TC among age group 31-40 years (7.84±2.58mmol/l) and 41-50 years (9.10±3.21mmol/l) when compared with those within age range 20-30 years (6.07±1.46mmol/l). There was no significant difference ($p>0.05$) in the level of TC between those within age range 31-40 and 41-50 years. There was significant different ($p<0.05$) in the level of LDL of age group 31-40 years (5.78±2.71mmol/l) and 41-50 years (6.67±3.11mmoml/l) when compared with age range 20-30 years (3.81±1.19mmol/l). There was no significant difference ($p>0.05$) in the level of LDL between age range 31-40 and 41-50 years. There was no significant difference in the level of TG, HDL and VLDL across the age ranges understudied.

APPENDICES

4.1 Table 1: Comparison of lipid profile parameters and BMI between auto mechanics and control groups in the study.

Parameters (Mmol/L)	Auto mechanics mean±SD. (N=60)	Control mean±SD. (N=40)	t-value	p-value
CHL	7.51±2.78*	2.60±0.50	13.35	P<0.05
TG	2.08±1.01*	1.29±0.32	5.65	P<0.05
HDL	1.27±0.21	1.24±0.24	0.530	P>0.05
LDL	5.39±2.70*	0.84±0.57	12.65	P<0.05
VLDL	0.94±0.46*	0.57±0.14	5.76	P<0.05

mean±SD* = Statistically Significant.

4.2 Table 2: Comparison Lipid Profile of Automobile Mechanics According to the Age Group

Parameters (mmol/L)	Age 20-30 N=20	Age 31-40 N=23	Age 41-50 N=17	F-value	P-value
TC	$6.07±1.46_a$	$7.84±2.58_b$	$9.10±3.21_b$	7.026	P<0.05
TG	$2.25±1.07_a$	$1.80±0.94_a$	$2.33±0.97_a$	1.752	P>0.05
HDL	$1.24±0.19_a$	$1.30±0.26_a$	$1.26±0.17_a$	0.466	P>0.05
LDL	$3.81±1.19_a$	$5.78±2.71_b$	$6.67±3.11_b$	7.103	P<0.05
VLDL	$1.01±0.50_a$	$0.81±0.43_a$	$1.06±0.44_a$	1.866	P>0.05

Values with different alphabets are significantly different at $p<0.05$.

DISCUSSION

Hyperlipidaemia is well known to be one of the major basic factors for atherosclerosis and cardiovascular disease. Hence any condition in life that causes hyperlipidaemia may therefore be considered to be among the risk factor for atherosclerosis. The present study assessed the lipid profile of automobile mechanics. The results of this study showed that exposure to petrol vapour, or premium motor spirit caused significant increase in serum total cholesterol (hypercholesterolaemia), LDL–C, VLDL-C, triglycerides and as well as decrease in serum HDL–C in the automobile mechanics. It has been reported that hypercholesterolaemia is a major risk factor for the development of atherosclerosis and it has been shown to be associated with impaired endothelial function (Zeiher *et al.*, 1995 and Heitzer *et al.*, 2001). The increased lipid profile observed in this study agrees with previous report that exposure to gasoline vapour alters the normal serum lipid profile and cause oxidative stress in the hepatocytes of male and female rats (Uboh *et al.*, 2007b). The Oxidative modification of LDL-C has also been shown to be an important step in promoting atherosclerosis (Witzum, 1994, Stocker and Keaney, 2004). Also increase in the vascular production of supper-oxide anion has been implicated as a contributing factor to impaired endothelium-dependent vascular relaxation in animal (rat) models of hypercholesterolaemia (Ohara*et al.*, 1993). In previous study, a high extend of lipid peroxidation, evidenced in elevated level of malonyldiadehyde (MDA) and reduced superoxide dismutase (SOD) activity in rats exposure to gasoline vapours was reported (Uboh*et al.*, 2007a). Hence, the atherosclerotic condition observed in this study may be

attributed to oxidative modification of lipoprotein which promotes endothelial dysfunction.

The present study suggest an increased risk of atherosclerosis in automobile mechanic who occupationally are more exposed to diesel and gasoline exhaust. It must be noted that the triglyceride level of the automechanics were observed to be significantly higher than that of the control. As such, this could indicate an increased hepatic secretion and decrease clearance of triglyceride in the automechanics (xve*et al.,* 2001). This is largely contributed by exposure to exhaust from gasoline and diesel and to other pollutants in the workshop environment. This may have caused an abnormal ratio of triglyceride (TG)/HDL-Cholesterol which indicates a risk of coronary disease (Prasad *et al.,* 2009). Automobile mechanics are constantly exposed to petroleum motor spirit by sucking it with their mouth through a tube in an attempt to siphon the fuel from the vehicle tank. In addition, they often wash vehicle parts and their hands with PMS without any gloves (Birtch*et al.,* 2000). In the process, auto mechanic inhale the fuel fume. Thus, refueling and repairing of automobile significantly expose them to benzene (a volatile chemical compound) (Dede and kagbo, 2001). And this could possibly account for the significant increase in atherogenic risk index and coronary risk index observed in this study.

When the lipid profile of automobile mechanics was compared based on age range, there was significant different ($p<0.05$) in the level of TC of age group 31-40 years and 41-50 years when compared with age range 20-30 years. There was also significant different ($p<0.05$) in the level of LDL of age group 31-40years and 41-50 years when compared with age range 20-30 years. This significant difference observed is in agreement with the definition of aging as the progressive loss of function accompanied by decreasing fertility increasing mortality and disability. As individual advances in age, some pathways in which cholesterol serves as a substrate are blocked and result hyperlipidaemia (Kirkwood and Austad, 2000). The prevalence of disease increases exponentially in old age (American Heart Association, 2002).

When the body mass index (BMI) of automechanics and control population were compared from this study, there was no significant variation. This could an indication that the body mass index of automobile mechanics has no influence in their hyperlipidaemia observed when compared with the control groups.

CONCLUSION

The result of this work suggests that reported exposure to gasoline vapour, diesel and petroleum motor spirit may elicit hepatotoxicity, thereby impairing the normal liver function. The effect influences the increased lipid profile (TC, TG, LDL and VLDL) that was observed in auto mechanics when compared with control groups. Based on previously reported data we propose that the presence of gasoline increase the intracellular oxidative stress, perhaps exceeding the protective capacity of the antioxidant systems. The effect brings about significant increases in TG, LDL and total cholesterol.

RECOMMENDATION

It is hereby recommended that automobile mechanics should have regular medical check-up to ascertain their health condition. It becomes very important that government and other stake holders in Nigeria health sector and communication media should rise up to the challenge of providing detail data base on the use, constituents, health and environment hazard of petrol vapour and automechanics should be advised to observe good hygiene protocol of keeping their tools, work cloths and environment as neat as possible. The use of face-mask should be encouraged to protect the eyes, nose and mouth and as well as prevent the passage of petrol effluents through these channel into the body. However, mouth sucking should be avoided.

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