EFFECTS OF PROLONGED EXPOSURE TO GAS FLARES ON THE LIPID PROFILE OF HUMANS IN THE NIGER DELTA REGION, NIGERIA

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ABSTRACT

Residents of the Niger Delta Region of Nigeria have been exposed for decades now to the hazards of the oil and gas industry operations such as gas flaring. This study was aimed at assessing the potential harmful effects on the lipid profile of some of the residents exposed to prolonged gas flares. Seven hundred and ninety subjects were recruited voluntarily from two communities in Imo East Senatorial zone of Nigeria. The test community subjects have been exposed to gas flaring for more than forty five years while the control subjects were unexposed to gas flaring though residents of both communities share a lot of common characteristics. Blood samples were collected from each subject and analyzed for serum total cholesterol (TC), triglyceride(TG) and high density lipoprotein(HDL). Low density lipoprotein(LDL), very low density lipoprotein(VLDL) levels were calculated using standard formulae. Lipid profile ratios: TC/HDL, TG/HDL,LDL/HDL and HDL/VLDL were also calculated. Results showed that serum levels of TC,TG, LDL, TC/HDL, LDL/HDL and HDL/VLDL were significantly increased in the test subjects when compared with the controls ($p<0.05$).The serum level of HDL was statistically higher in the control subjects compared with the test subjects($p<0.05$). In conclusion, prolonged exposure to gas flares may contribute to increased dyslipidemia, this may increase the prevalence of cardiovascular diseases such as atherosclerosis, hypertension and ischaemic heart disease. The hyperlipidemia can be attributed to the presence of heavy metals in flared gas.

Key words: **gas flaring, lipid profile, prolonged exposure, humans, Niger Delta**.

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INTRODUCTION

Lipids are a group of fats and fat-like substances that are important constituents of cells and sources of energy. Different plasma lipids vary greatly in various populations due to differences in geographical, cultural (Hart et al, 1997); economical, social conditions (Vartiainen et al, 1997, dietary habits and genetic make-up (Abubakar et al, 2009). Age and gender differences also affect serum lipids(Malik et al, 1995; Shahid et al, 1985).

Lipid profile, also called Lipid Panel, Coronary Risk Panel or Complete Cholesterol test is mainly used to assess the risks of developing cardiovascular diseases and also to monitor management of the afflicted (Castelli et al, 1992; NCEP, 2002). A lipid profile measures the level of specific lipids in the blood. A typical lipid profile includes total cholesterolTC), triglyceride(TG), high density lipoprotein(HDL), low density lipoprotein(LDL), very low density lipoprotein(VLDL) and ratios derived from the above values.

Oil and gas industry operations include exploration, drilling, refining and distribution of the finished products to the consumers. During most of these activities, wastes either in solid, liquid or gaseous form are generated and discharged into the environment (Gobo et al, 2009). Flared gas is one of such wastes generated in the oil and gas industry.

Nigeria is the 6TH largest producer of oil in the world and it is endowed with more gas reserves than oil (Aston-Jones, 1998; NNPC, 2011; Ayoola,2012).

Gas flaring has been defined as the complex and unscientific burning and emitting of excess hydrocarbons consisting of substantial amount of soot, carbon monoxide and greenhouse gases associated with crude oil and gas production processes (Nwokezi,1992). Gas Flaring is a common practice of burning off unwanted, flammable gases via combustion in an open atmosphere, non-premixed flame(McEwen and Johnson,2012). Gas flaring is one anthropogenic activity, defined as the "wasteful emission of greenhouse gases(GHGs) that causes global warming, disequilibrium of the earth, unpredictable weather changes and major natural disasters because it emits a cocktail of benzene and other toxic substances that are harmful to humans, animals, plants and the entire physical environment"(World Bank, 1995).

According to a World Bank estimates in 2011, the annual volume of natural gas being flared and vented worldwide stood at about 140 billion cubic meters (bcm)(GGFR, 2012) , this is enough to provide for the annual gas consumption of Central and South America or that of Germany and Italy(Gerner et al, 2004). Russia still tops the world's flaring countries, followed by Nigeria, Iran and Iraq. Inconsistent data and under-reporting of gas flaring by governments and companies has complicated the global effort to track progress on flaring reduction (GGFR,2012).

During gas flaring, complete combustion though rarely achieved, releases relatively innocuous gases such as carbon dioxide and water, whereas incomplete combustion emits various compounds such as methane, propane, and hazardous air pollutants such as volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs) and soot (Kindzierski, 2000); benzene, naphthalene, styrene, acetylene, fluoranthene, anthracene pyrene, xylene and ethylene (Strosher, 1996). Chemically, VOCs created in gas

flare emissions are unsaturated cyclic(aromatic) hydrocarbons that contain one or more benzene rings(ATSDR, 2000). Benzene, Toluene, ethylbenzene and xylene (BTEX) are used as representative of this group. Benzene and toluene in particular are hazardous due to their inherent toxicity in mammals , while their wide use in industry and high volume of production lead to substantial environmental releases (Robinson et al, 1997). Flaring can also produce soot and other pollutant species that have negative effects on air quality and the environment (Strosher, 2000; Johnson and Kostiuk (2001); Johnson et al, 2001)

Idodo-Umeh (2010) reported that the Nigerian crude oil is known to contain heavy metals such as Al, Zn, As, Ba, Fe, Pb, Co, Cu, Cr, Mn, Ga, Sb, Ni and V. It has also been noted that surface and underground waters in gas flared environments tend to have more concentrations of heavy metals such as lead, barium, cadmium, selenium, manganese, magnesium and copper than non-gas flared area (Nwankwo and Ogagaure,2011; Egwurugwu et al, 2013). In addition, high concentrations of heavy metals in gas flares, soil and water in the Niger Delta Region of Nigeria have been observed (Nwankwo and Ogagaure, 2011; Idodo-Umeh, 2010). The residents of the Niger Delta Region are exposed not only to the various air and soil pollutants but also to water contaminants especially the heavy metals.

The health hazards associated with gas flaring are legion, for it affects every system in man while his plants, animals, air, soil and water are not left out. Gas flares can cause premature death (Nwafor, 2013); reduce blood production (Owu et al, 2005; Adiembo and Nwafor, 2010; Egwurugwu et al, 2013); cancer (Ruiz and Rizo, 2007; Tuntawiroon et al, 2007); immunotoxic (Olsgard et al, 2008); hypertension and kidney disease(Nawrot et al. 2002;Egwurugwu and Nwafor, 2013); neurotoxic (Argo, 2002); chronic respiratory diseases such as asthma (USEPA,2007; Ekpo and Obia, 2010); fetotoxic (Gobo et al, 2009; Argo, 2002); fetotoxic (Gobo et al, 2009; Argo, 2002); increase in temperature(thermal gradient), acid rain, low agricultural productivity(Oseji, 2011); infertility (Nwafor, 2013).

There is paucity of data on the possible effects of prolonged gas flaring on the lipid profile of Niger Delta residents in Nigeria and this prompted this research.

MATERIALS AND METHODS

Study areas

Two different communities, with very similar characteristic features, in the Imo East Senatorial zone of Nigeria were chosen for the study. Imo State is one of the nine states in the Niger Delta region of Nigeria. Egbema, an oil and gas producing community with active gas flaring by Shell Petroleum Development Company (SPDC) for more than 45 years, constitute the test group. This community is located in between many other active oil and gas flaring sites such as Ossu, Oguta and Izombe oil and gas fields operated by Addax and Akri and Ebocha oil and gas fields run by Nigeria Agip Oil Company. Thus, the residents are well exposed to the effects of gas flaring. Alaoma Owerre-Ebeiri autonomous community, a non oil and gas producing area, constitute the control group population. The residents of

both communities were mainly farmers, traders and civil servants and share many common characteristics. The study was done during the rainy season, in the months of May and June, 2012.

Selection of subjects

Apparently healthy subjects, between the ages of 18 to 80 years, who consented to in writing and/or thumb printed (after thorough explanation) to participate in the study, were randomly selected. All must have lived in their various communities consistently for more than 5 years. The research was approved by the Ethics Committee on Human Biomedical Research of the University of Port Harcourt, Nigeria and the study conforms to the Helsinki Declaration on Biomedical Research. A total of seven hundred and ninety (790) subjects of both sexes took part in the study. The test group had 475 subjects(140 males and 335 females) while the control group had 315 subjects(127 males and 188 females). All known cases of hypertension, diabetes mellitus, metabolic syndrome, dyslipidemia, renal disease, atherosclerosis and contraceptive users were excluded from the study.

Collection and analyses of blood samples

5ml of venous blood drawn from a peripheral vein in the upper limb of subjects, was put into sterile plain universal containers, allowed to clot and retract properly and then centrifuged at 5000rpm for 5minutes. The supernatant was then stored frozen at -20°C until analyzed for total cholesterol(TC), triglyceride (TG), high density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL) using Acurex-Prietest easylab biochemistry analyzer, Acurex, USA. Various ratios such as TC/HDL, TG/HDL, LDL/HDL, HDL/VLDL were calculated using Microsoft Excel.

All reagents for the analyses of biochemical parameters in this work were purchased from Randox Laboratories Ltd, Diamond Road, Crumlin, United Kingdom.

Allain et al(1974) method was used to analyze total cholesterol and triglyceride. Albers et al (1978) method was used to analyze high density lipoprotein while Assmann et al (1984) method was used to analyze serum concentration of low density lipoprotein.

Statistical analysis

Statistical Package for Social Sciences (SPSS) (version 17 for windows, SPSS Inc., Chicago, USA) was used to analyze the data. The differences in the various parameters studied between the test and control groups were evaluated using Kolmogorov-Simirnov Z statistic. Anova was used to assess differences within the groups. Statistically significant values were determined at $p < 0.05$ or 95% confidence level.

RESULTS

Seven hundred and ninety(790) subjects participated fully in this study. The results are presented in Tables and Figures.

Table 1: Lipid profile of the subjects

Parameter	Control group (N=315)	Test group (N=475)	P Value	Percentage difference
TC(mmol/l)	4.43±0.09	5.52±0.04	0.01*	10.95
TG(mmol/l)	1.33±0.04	1.55±0.03	0.01*	7.64
HDL(mmol/l)	1.61±0.03	1.49±0.02	0.04*	-3.87
LDL(mmol/l)	2.27±0.09	3.25±0.04	0.01*	17.75
VLDL(mmol/l)	0.61±0.02	0.78±0.01	0.01*	12.23
TC/HDL ratio	3.12±0.10	4.22±0.08	0.01*	15.00
TG/HDL ratio	0.22±0.04	1.21±0.03	0.01*	69.23
LDL/HDL ratio	1.69±0.09	2.61±0.06	0.02*	21.39
HDL/VLDL ratio	1.26±0.12	2.17±0.09	0.01*	26.53

TC=total cholesterol; TG= Triglyceride; HDL=High density lipoprotein.

LDL =Low density lipoprotein; VLDL =Very low density lipoprotein; TC/HDL= total cholesterol/high density lipoprotein ratio; TG/HDL = triglyceride/high density lipoprotein ratio; LDL/HDL=low density lipoprotein ratio, HDL/VLDL=High density lipoprotein/very lipoprotein density lipoprotein.

*=statistically significant

Study of Serum Total Cholesterol Levels

The entire population of the control subjects had a mean serum total cholesterol of 4.48±0.09mmol/L(1.90-14.15mmol/L) while the total population of the test subjects had a mean serum total cholesterol level of 5.52±0.04 mmol/L (3.06-9.56mmol/L). The difference between the test and control serum total cholesterol levels is statistically significant ($p < 0.05$) (Table 1). The percentage difference between them is 10.95% (Figure 1).

The whole population of the control subjects was 315, out of this, 18 (6%), had total cholesterol <2.6mmol/L; 266 (84%) had total cholesterol within the normal range(2.6-6.0). The remaining 31 (10%) had raised total cholesterol level (> 6.00mmol/L). Among the control subjects, the males had a mean TC of 4.41±0.14mmol/l compared with 4.53±0.12mmol/l of the females, this difference is not significant statistically($p>0.05$), though the percentage difference them is 2.68% (Table 2)

The test group had a total population of 475 subjects, out of this,371 (78 %) had total cholesterol within normal range of 2.6-6.00 mmol/L; while the remaining 104 (22%) had raised total cholesterol level above 6.00 mmol/L. Among the test subjects, the males had a mean TC of 5.35±0.09mmol/l compared with 5.59±0.06mmol/l of the female subjects, this difference is significant statistically($p<0.05$), though the percentage difference between them is 2.19% (Table 2).

Study of Serum Triglyceride Levels

The mean serum triglyceride level in the control subjects was 1.33±0.04mmol/L(0.15-4.99mmol/L), while the mean serum triglyceride level in the test group was 1.55±0.03mmol/L(0.54-9.56mmol/L). The control subjects thus had lower serum levels of triglyceride compared with the test group and this difference is statistically significant ($p<0.05$) (Table 1). The percentage difference between them is 7.64%(Figure 1).

The control group had total population of 315 subjects, out of this, 268 (85%) had serum triglyceride level within normal range, while the remaining 47 (15 %) had serum triglyceride level above 1.8 mmol/L. The control male subjects had a mean TG of 1.28±0.06mmol/l compared with 1.36±0.05mmol/l of the females, this difference is not significant($p>0.05$). However, the percentage difference between them is 6.06%(Table 2).

The test group had a total population of 475 subjects, out of this, 339 (71 %), had serum triglyceride level within normal range of 0.3-1.8 mmol/L, while the remaining 136 (29%) had raised serum triglyceride level above 1.8 mmol/L. The test group males had a mean TG of 1.49±0.04mmol/l compared with 1.59±0.03mmol/l of the females, this difference is not statistically significant($p>0.05$), though the percentage difference between them is 2.93% (Table 2).

Study of High Density Lipoprotein Levels

The entire population of the control subjects had a mean serum high density lipoprotein (HDL) value of 1.61±0.03mmol/L(0.50-3.80mmol/L). The total population of the test subjects had a mean HDL level of 1.49±0.02mmol/L(0.30-3.50mmol/L). The control subjects, therefore, had raised serum levels of HDL when compared with the test subjects and the difference is statistically significant ($p<0.05$)(Table 1) and the percentage difference between them is 3.87% (Figure 1).

The control group had a total population of 315 subjects, out of this, 18 (6 %) had serum HDL values less than normal; 178 (56 %) had serum levels of HDL within the normal range of 0.8-1.7 mmol/L), while the remaining 119 (38 %) had serum values of HDL above the normal range. The male control subjects had a mean HDL value of 1.65±0.05mmol/l compared with 1.59±0.04mmol/l of the females, the difference is not significant ($p>0.05$), though the percentage difference between them is 3.70% (Table 2).

The entire population of the test group was 475, 23(4 %) of this, had serum levels of HDL below normal range; 316 (67%) had serum levels of HDL within normal range of 0.8-1.7mmol/L while the remaining 136(29%) had levels above normal range. The test group males had a mean HDL value of 1.49±0.05mmol/l compared with 1.49±0.03mmol/l of the female subjects, this difference is not significant statistically($p>0.05$), there is no percentage difference between them(Table 2).

Study of Low Density Lipoprotein Levels

The mean serum low density lipoprotein (LDL) value for the control subjects was 2.27±0.09mmol/L(0.04-11.38mmol/L). The mean value of LDL for the test subjects was 3.25±0.05mmol/L(2.49-5.45mmol/L). The test subjects thus had increased serum levels of LDL more than the control subjects and this increment is statistically significant ($p<0.05$) (Table 1). Also, the percentage difference between them is 17.75%(Figure 1).

Out of the total control subjects of 315, 143 (45%) had serum levels of LDL lower than normal range; 142(45%) had serum values within normal range, the remaining 30 (10%) had levels of LDL above the normal range. The control group male subjects had a mean LDL value of 2.16±0.13mmol/l compared with 2.34±0.12mmol/l of the female test subjects, this difference is not significant (p>0.05), though the percentage difference between them is 8% (Table 2).

In the test group of 475 subjects, 417 (87%) had values within the normal range, the remaining 58 (13 %) had levels above the normal range of 1.9-3.6mmol/L. The test group males had a mean LDL value of 3.11±0.07mmol/l compared with 3.31±0.06mmol/l of the female test subjects, this difference is significant statistically(p<0.05), though the percentage difference between them is 3.12% (Table 2).

Study of Very Low Density Lipoprotein.

The entire population of the control subjects had a mean serum VLDL level of 0.61±0.02mmol/L(0.07-3.10mmol/L) while the test group subjects had a mean value of 0.78±0.01mmol/L(0.40-1.92mmol/L). Thus, the test subjects had increased serum levels of VLDL when compared with the control subjects and this increment is statistically significant (p <0.05)(Table 1).The percentage difference between them is 12.23%(Figure 1).

The control subjects had a total population of 315, out of this, 304 (96%) had serum values of VLDL within normal range of 0.4-1.3mmol/l while the remaining 11 (4%) had raised levels of VLDL above normal range. The control male subjects had a mean VLDL of 0.61±0.03mmol/l compared with 0.62±0.02mmol/l of the females, this difference is not significant (p<0.05), though the percentage difference between them is 1.61%(Table 2).

The test group had a population of 475 subjects, out of this, 439 (92%) had values within normal range of 0.4-1.3mmol/L; the remaining 36(8%) had levels above the normal range. The test group males had a mean VLDL value of 0.75±0.02mmol/l compared with 0.79±0.01mmol/l of the females, this difference is not significant statistically (P>0.05), however, the percentage difference between them is 2.60%(Table 2).

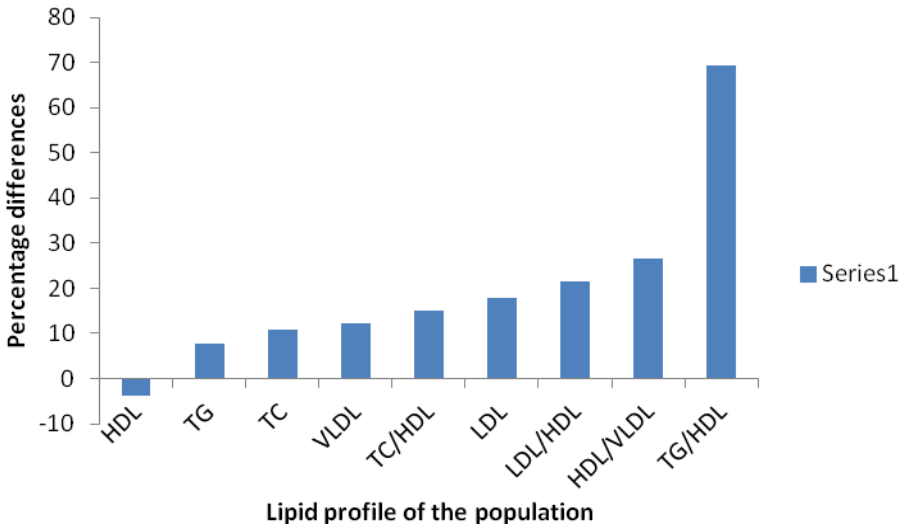

Figure 1: Lipid profile of the subjects in percentage differences.

Table 2: Comparison of the lipid profiles in males and females

Parameter	Research group	Male	Female	P value	Percentage difference
TC(mmol/l)	Control	4.41±0.14	4.53±0.12	0.94	2.68
	Test	5.35±0.07	5.59±0.05	0.02*	2.19
TG(mmol/l)	Control	1.28±0.06	1.36±0.04	0.47	6.06
	Test	1.49±0.04	1.58±0.03	0.12	2.93
HDL(mmol/l)	Control	1.65±0.05	1.59±0.04	0.45	3.70
	Test	1.49±0.04	1.49±0.03	0.94	0.00
LDL(mmol/l)	Control	2.16±0.13	2.34±0.11	0.43	8.00
	Test	3.11±0.07	3.31±0.06	0.04*	3.12
VLDL(mmol/l)	Control	0.61±0.03	0.62±0.02	0.62	1.61
	Test	0.75±0.02	0.79±0.01	0.12	2.60
TC/HDL ratio	Control	3.01±0.14	3.19±0.12	0.26	5.81
	Test	3.98±0.12	4.32±0.11	0.08	4.09
TG/HDL ratio	Control	0.90±0.06	0.98±0.04	0.12	8.51
	Test	1.13±0.05	1.24±0.04	0.18	4.64
LDL/HDL ratio	Control	1.59±0.12	1.76±0.11	0.04	9.52
	Test	2.42±0.11	2.70±0.09	0.08	5.47
HDL/VLDL ratio	Control	3.41±0.15	3.20±0.16	0.20	6.06
	Test	2.23±0.08	2.15±0.05	0.42	1.83

*Statistically significant.

Study of the total cholesterol/ high density lipoprotein ratio

The control subjects had a mean TC/HDL ratio of 3.12±0.10(1.18-14.71) while the test subjects had a mean value of 4.22±0.08. Therefore, the test subjects had a higher value compared with the control and this difference is statistically significant ($p < 0.05$)(Table 1). The percentage difference between the control and test subjects is 15%(Figure 1).

The control males had a mean TC/HDL ratio of 3.01±0.15 compared with 3.19±0.13 of the females, this difference is not statistically significant ($p > 0.05$), however, the percentage difference between them is 5.81%(Table 2)

The test group males had a mean TC/HDL ratio of 3.98±0.12 compared with 4.32±0.11 of the females, this difference is not significant ($p > 0.05$), though the percentage difference between them is 4.09%(Table 2).

Study of Triglyceride/high density lipoprotein (TG/HDL) ratio

The entire population of the control subjects had a mean serum TG/HDL ratio of 0.22±0.01(0.09-4.99) compared with 1.21±0.03(0.92-2.11) of the test subjects, this difference is significant ($p < 0.05$)(Table 1) and the percentage difference between the two is 69.23%(Figure 1).

Among the control subjects, the males had a mean value of 0.90±0.06 while the females had a mean value of 0.98±0.04, this difference is not statistically significant, though the percentage difference the two is 8.51%(Table 2).

Among the test group subjects, the males had a mean value of 1.13±0.05 while the females had a mean value of 0.89±0.03, this difference is not significant statistically ($p > 0.05$), however, the percentage difference between them is 4.64%(Table 2).

Study of Low density lipoprotein/High density lipoprotein (LDL/HDL) ratio

The whole population of the control subjects had a mean serum LDL/HDL ratio of 1.69±0.08(0.01-13.15) while the test subjects had a mean serum LDL/HDL ratio of 2.61±0.06(1.18-8.30), the difference is statistically significant ($p<0.05$) (Table 1), and the percentage difference between them is 21.39% (Figure 1)

Among the control subjects, the males had a mean value of 1.59±0.13 while the females had a mean value of 1.76±0.11, this difference is significant statistically ($p<0.05$) and the percentage difference between them is 9.52% (Table 2).

Among the test group subjects, the males had a mean serum LDL/HDL ratio of 2.42±0.11 compared with 2.70±0.09 of the females, this difference is not significant statistically ($p>0.05$) and the percentage difference between them is 5.47% (Table 2).

Study of High density lipoprotein/Very low density lipoprotein (HDL/VLDL) ratio

The entire population of the control subjects had a mean serum HDL/VLDL ratio of 1.26±0.02 while the test subjects had a mean serum HDL/VLDL ratio of 2.17±0.04, the difference is statistically significant ($p< 0.05$)(Table 1) and the percentage difference between them is 26.53%.(Figure 1)

Among the control subjects, the males had a mean value of 3.41±0.15 while the females had a mean value of 3.20±0.11, this difference is not significant statistically ($p>0.05$) and the percentage difference between them is 6.06% (Table 2).

Among the test group subjects, the males had a mean serum HDL/VLDL ratio of 2.23±0.08 compared with 2.15±0.11 of the females, this difference is not significant statistically ($p>0.05$) and the percentage difference between them is 1.83% (Table 2).

DISCUSSION

Lipid profile, also called Lipid Panel, Coronary Risk Panel or Complete Cholesterol test is mainly used to assess the risks of developing cardiovascular diseases and also to monitor management of the afflicted (Castelli et al, 1992; NCEP, 2002). Lipid profile can also be used to assess the effects of environmental contaminants on the health and well being of persons exposed to such pollutants.

This study revealed that the serum concentrations of total cholesterol(TC), triglyceride(TG), low density lipoprotein(LDL), very low density lipoprotein(VLDL) plus the following ratios: TC/HDL, TG/HDL, LDL/HDL and HDL/VLDL were significantly increased in subjects chronically exposed to gas flares when compared with the non-exposed subjects($p<0.05$). The exposed persons also had reduced serum concentrations of high density lipoproteins(HDL) compared with the non-exposed subjects($p<0.05$).

Lipid metabolism occurs in the liver. Cholesterol is an essential constituent in cell membranes and it is used by gland cells to make steroid hormones (Ganong, 2005). Liver damage can lead to lipid derangement with possible disturbances of cell membrane integrity which may cause some membrane lipids to be released into the circulation. Studies have demonstrated a correlation between environmental pollution and the development of cardiovascular disease (CVD)(Jennrich, 2013). The observed increase in the lipid profile of the subjects exposed to prolonged gas flares may be due to the various harmful constituents of flared gas.

The aliphatic and aromatic hydrocarbons are major constituents of gas flares, their metabolism as well as other xenobiotics can result to changes in cell membranes due to reactive free radical species (Bondy et al, 1995). Photo induced lipid peroxidation by poly aromatic hydrocarbons (PAH) is mediated by reactive oxygen species (ROS), forming singlet oxygen and superoxide (Xia et al, 2006; Yu et al, 2006). Singlet oxygen itself can react with amino acids, proteins, lipids and DNA resulting in cell damage and

diseases (Tyrrell, 2000). ROS can also damage DNA and proteins leading to aging, inflammation, cardiovascular diseases, cancer and age-related diseases (Loft et al, 1996).

Increased serum cholesterol was also seen in females receiving 350 mg/kg/day acenaphthene (EPA 1989). Increased liver weight and dose-related centrilobular pigmentation accompanied by an increase in liver enzymes were observed in both male and female mice receiving 250 mg/kg/day fluoranthene by gavage for 13 weeks (EPA 1988). The induction of carboxylesterase activity has also been observed in animals exposed to PAHs (Nousiainen et al. 1984).

Experimental studies have also shown that cadmium and lead can contribute to oxidative stress by (a)catalyzing the formation of reactive oxygen species(Richard et al, 1991; Vaziri et al, 2001),(b) increasing lipid peroxidation(Ding et al, 2000; Yiin et al, 1999) and(c) depleting the glutathione and protein bound sulfhydryl groups(Ding et al, 2000). In vivo and in vitro studies suggest that lead –induced oxidation contributes to red blood cell damage (Yiin et al, 1999). Depletion of intracellular glutathione is related with the mechanism of oxidized low density lipoprotein, which is a risk factor for atherosclerosis and CVD (Kuzuya et al, 1989).

It has been established that both acute and chronic lead poisoning cause impairment of heart and vessel function(Kopp et al, 1988; Wojtczak-Jaroszowa and Kubow, 1989). An association between atherosclerosis and lead exposure is biologically plausible (Kristal-Boneh et al, 1999). And one of the underlying mechanisms in the association between cardiovascular damage and lead exposure is the induction or acceleration of atherosclerosis(Wojtczak-Jaroszowa and Kubow, 1989). Lead can induce atherosclerosis by three pathophysiological mechanisms: inhibition of superoxide dismutase, resulting in the elevation of serum lipid peroxide (Ito et al, 1985; Qinlan et al, 1988); formation of atherosclerotic plaques from a single mutated proliferating cell(monoclonal hypothesis); and inhibition of the activity of

cytochrome P-450(Alveres et al, 1977), leading to an increase in serum lipids and their accumulation in vessel walls.

Extensive studies in both humans and animals have established increment in serum lipids following lead exposure(Skoczynska et al, 1993; Khan et al, 1993; Dessi et al, 1989; El-Gazzer et al, 1989; Gatagonova, 1994). Kristal-Boneh et al. 1999, had observed that lead exposure is associated with increased serum values of TC and HDL in occupationally exposed workers. They speculated that the increase in serum triglycerides could have been caused by lead-induced inhibition of lipoprotein lipase activity or decreased activity of hepatic lipase.

Cadmium exposure has also been implicated in hypertension and atherosclerosis(Revis et al, 1981; Ross et al, 1993; Houtmann, 1993). Hypertriglyceridemia was also observed in male rats exposed to 18 mg Cd/kg/day as cadmium chloride in the drinking water for 8 weeks (Larregle et al. 2008); the increase in plasma triglycerides was likely due to a decrease in lipoprotein lipase activity.

Human studies following inhalation of xylene suggest that acute-duration exposure to high levels of xylene may result in hepatic toxicity (Klaucke et al. 1982; Uchida et al. 1993.

Animal model studies using rats have also shown that mixed xylene, *m*-xylene, *o*-xylene, or *p*-xylene generally induce a wide variety of hepatic enzymes, as well as increased hepatic cytochrome P-450 content in rats (Elovaara 1982; Elovaara et al. 1980; Selgrade et al. 1993).

Many similar hepatic effects appear after intermediate-duration exposure to mixed xylene or *o*-xylene. They include increased absolute and/or relative hepatic weight in rats (Kyrklund et al. 1987; Ungvary 1990;), increased cytochrome P-450 (Tatrai et al. 1981; Ungvary 1990); increased microsomal enzyme

activity (Elovaara et al. 1980, 1987; Tatrai et al. 1981; Toftgard et al. 1981; Ungvary 1990;), proliferation of the smooth and rough endoplasmic reticulum (Rydzynski et al. 1992; Tatrai and Ungvary 1980; Tatrai et al. 1981; Ungvary 1990).

Exposure by inhalation to hydrogen sulfide led to increased liver enzyme activities in some exposed persons (Burnett et al. 1977). Hayden et al (1990) had also demonstrated increased maternal liver cholesterol levels in Sprague-Dawley dams exposed to 75 ppm, but not 50 ppm, for 7 hours/day from gestation day 6 to postpartum day 21.

Toluene has been identified in brain, liver, lung, and blood in humans following toluene exposure (Paterson and Sarvesvaran 1983; Takeichi et al. 1986). Within the human brain, toluene has a greater affinity for areas of the brain that contain lipid-rich white matter,such as the brain stem, rather than the areas with larger amounts of grey matter (Ameno et al. 1992).

Some studies of workers occupationally exposed to average concentrations between about 30 and 350 ppm toluene have reported liver effects such as increased serum levels of enzymes leaked from the liver (Guzelian et al. 1988; Svensson et al. 1992).Animals studies following chronic exposure revealed increased liver weights (NTP 1990; Poon et al. 1994; Ungvary et al. 1982) and induction of hepatic cytochrome P450 levels (Ungvary et al. 1982; Wang et al. 1996).

Several studies have described changes in the serum lipid profile of humans exposed to **zinc** sulfate or gluconate for 3–12 months (ATSDR, 2005). Ingestion of 2.3–4.3 mg zinc/kg/day for 5– 6 weeks (Chandra 1984; Hooper et al. 1980) or 0.71 mg zinc/kg/day for 12 weeks (Black et al. 1988) reduced levels of high-density lipoprotein (HDL) cholesterol. In the study by Chandra (1984), a slight increase in

low-density lipoprotein (LDL) cholesterol was observed in subjects who served as their own controls; measurements were taken prior to zinc supplementation and after a 10-week post exposure period. Serum cholesterol, triglyceride, and LDL cholesterol levels were not affected by zinc supplementation in the study by Black et al. (1988). However, in another study, zinc supplements depressed HDL cholesterol levels and raised LDL cholesterol levels in elderly subjects (>60 years of age), especially in those who exercised. (Goodwin et al. 1985). Young women with a total daily intake of 1.6 mg zinc/kg/day in a 2-month study had a transient decrease in HDL cholesterol (Freeland-Graves et al. 1980).

Chromium(VI) has been reported to cause severe liver effects in four of five workers exposed to chromium trioxide in the chrome plating industry. Derangement of the cells in the liver, necrosis,lymphocytic and histiocytic infiltration, and increases in Kupffer cells were reported(ATSDR,2012). Glaser et al (1985) had observed increase in triglycerides and phospholipids in a group exposed to 0.2 mg chromium(VI)/m^3 for 90 days.

The increase in total cholesterol in the test group may have resulted from the damage to the hepatic cells by the toxic pollutants. The observed decrease in HDL in the test group population may have resulted from the accumulation of cholesterol released into the plasma from the dying cells and from membranes undergoing turnover (Ubani et al, 2010). HDL acts as a shuttle that moves cholesterol throughout the body, binds esterified cholesterol released from the peripheral tissues and then transfer cholesteryl esters to the liver or to tissues that use cholesterol to synthesize steroid hormones (Berg et al, 2007). HDL is called “good cholesterol” because it carries cholesterol and phospholipids from tissues and organs back to the liver for degradation and elimination. Thus, it prevents the deposition of cholesterol on the walls of arteries by carrying cholesterol away from arteries to liver. High level of HDL

is a good indicator of a healthy heart because it reduces the blood cholesterol level (Sembulingam and Sembulingam, 2010; Ganong, 2005).

The results also showed significantly increased values of LDL in the test group population compared with the control population ($p<0.05$). LDL-cholesterol is referred to as "bad cholesterol" because it carries cholesterol and phospholipids from the liver to the peripheral tissues and organs like the heart. It is responsible for the deposition of cholesterol on the walls of arteries causing atherosclerosis. The observed increase in serum LDL-cholesterol in the test group subjects may result from the impairment in the receptor-mediated endocytosis which prevents the binding of LDL to specific receptors that could lead to its degradation and release of cholesterol (Ubani et al, 2010). LDL receptors are present in all cells but most abundant in hepatic cells and adrenal cortex. The liver has a major role in the control of plasma levels of LDL cholesterol because most of the LDL receptors are present in the liver which also synthesizes cholesterol and removes cholesterol from lipoprotein remnants (Vasudevan and Sreekumari, 2005). Hyperlipidemia constitutes a major etiopathological factor for atherosclerosis (Sanjay and Subir, 2002). It has been established that total plasma cholesterol and LDL-Cholesterol are the best markers of plasma lipemia for the evaluation of cardiovascular diseases (CVD) (Bravo et al, 2012)

The observed statistically significantly increase in the following lipid ratios:TC/HDL, TG/HDL, LDL/HDL and HDL/VLDL in the test group subjects when compared to the control subjects ($p<0.05$) in this study tend to indicate the greater risk in the exposed population to coronary artery disease and other cardiovascular diseases following prolonged exposure to gas flares. Lipid profile ratios such as total cholesterol/HDL ratio can be explored as diagnostic tool for metabolic syndrome and atherosclerosis (Arthur et al, 2012).

There were no statistically significant differences between the males and females in the mean serum concentrations of triglyceride, High density lipoprotein, very low density lipoprotein,TC/HDL,

TG/HDL,and HDL/VLDL ratios among the control and test subjects (P>0.05). However, the female test subjects had increased values of total cholesterol, low density lipoprotein and LDL/HDL ratio(p<0.05) when compared with the males. This agrees with the findings of Nwafor(2013). The increased dyslipidemia among the females in the test group population when compared with the males may be due to the fact that the domiciled females at home as house wives and/or are mainly farmers that spent most of their time in the gas flared environment and therefore more exposed to the pollutants than the more migratory males. In addition, alcohol dehydrogenase which detoxifies carbohydrates, sugar, alcohol and chemicals and butyl cholinesterase that scavenge chemicals are both lower in females than in males. Furthermore, females have a greater total percentage body fat which stores chemicals, use more fragrances, hair coloring, hair sprays, lipsticks and other make-ups with known toxic ingredients; women typically do the house cleaning, and are exposed daily to toxic products even in their kitchens(Grout, 2012).

In conclusion, residents of Egbema, a community in the Niger Delta Region of Nigeria, have increased serum concentrations of total cholesterol, triglyceride, low density lipoprotein, TC/HDL ratio, TG/HDL ratio, LDL/HDL ratio and HDL/VLDL ratio compared with the referents. The observed deleterious effects may be due to the various pollutants present in flared gas, especially the heavy metals. They are thus more likely to develop cardiovascular diseases such as atherosclerosis, coronary heart disease and hypertension. The oil and gas companies should as a matter of urgency put in place measures to reduce the burden already inflicted on the exposed persons while instituting actions to minimize gas flaring and other public health challenges due to gas flaring and related issues.

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