Unique Research Journals
Unique Research Journal of Medicine and Medical Sciences Vol. 1(4), pp. 038-043, October, 2013
Available online@http://www.uniqueresearchjournals.org/URJMMS

Unique Research Journal of Medicine and Medical Sciences

Full Length Research Paper

Blood dyscrasia in women of child bearing age exposed to PMS in Okada–Ore Local Community Edo State, Nigeria

Adesina O. O.[1], Olayanju A. O.[2], Oduola T.[2], Ademosun A. A.[2], Ezimah A. C.[1]and Olayanju A. J.[2]

[1]College of Health Sciences, University of Maiduguri, Borno State. Nigeria.
[2]Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Nigeria.

*Corresponding author. E-mail: ddddickson@yahoo.com

Accepted 27 July, 2013

Occupational exposure to petroleum has been reported to have toxic effects on various organs and systems. The purpose of this research is to investigate the potential risk associated with exposure of non-pregnant women of child bearing age selling PMS (WSP) in Okada-Ore Road, Edo State, Nigeria using batteries of haematological parameters and level of MetHb as an indirect blood biomarker of exposure to benzene in PMS. 100 women each for both test and control population within the age 18-34 years were recruited into the study.We observed that women exposed to petroleum had a significant higher Met Hb than control group. The red cell indices decreased with increase in years of exposure. Red blood picture of subject within 1-3 years of exposure shows that of dimorphic blood picture with microcytic normocytic hypochromic cells as predominant features.Platelet count decreased with level of Subjects above 5 years of exposure resulting in mild thrombocytopenia with few blasts. The route of exposure to PMS is by mouth suction due to conservation of time for maximum profit making and skin contamination.

Key words: Blood dyscrasia, benzene, anaemia, lead poisoning.

INTRODUCTION

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). In 2011, total crude oil and condensate production was 866,245,232 barrels, giving a daily average of 2.37 mmb/pd, while in the gas sector, a total of 2,400.40 Billion Standard Cubic Feet(BSCF) of Natural Gas was produced bysixteen companies; of the quantity produced, 1,781.37 BSCF (74%) was utilized, while 619.03BSCF (26%) flared (NNPC, 2011).

Despite the effort of the government in restructuring the petroleum industry, most of the refineries in the country are working at a sub-optimal level due to constant vandalization of oil pipelines, oil spillage, poor maintenance, illegal refineries and other illegal activities. During these processes the handlers of crude oil and its products are exposed to various cytotoxic chemicals that may induce anaemia and other pathologic effects. Refined products from these activities are traded by touts or peddlers including women and children without observing any safety measures in most local communities in Nigeria. The unavailability of the products in the organized market and periodic fuel price hikes further promotes patronage of these 'black markets'.

Occupational exposure to petroleum has been reported

to have toxic effects on various organs and systems, and these include respiratory, immune and nervous systems. Organs such as the heart, lungs, skin andkidneys are affected by these toxic effects resulting in various diseases and different forms of genotoxic, mutagenic, immunotoxic, carcinogenic and neurotoxic manifestations (Becker, 1985; Klassen, 1990; d'Azevedo et al., 1996; Smith et al., 1996; Rabble and Wong, 1996; Ross 1996; Rothman et al., 1996).

Gasoline or petrol is a clear but slightly yellowish liquid mixture which is primarily used as fuel in internal combustion engine (Ron, 2004). Petrol comes from crude oil and it is a mixture of organic hydrocarbon. It is consist mostly of aliphatic hydrocarbons (HCs) obtained by fractional distillation of petroleum, enhanced with isooctane or the aromatic HCs like toluene or benzene to increase its octane rating. The bulk of a typical gasoline consist of HCs with below 4-12 Carbon atom (Collins, 2007), molecule commonly referred to as C4-C12. Many of this HCs are hazardous substances and are regulated in the united states by the occupational safety health administration.PMS contains volatile organic compounds like benzene and the content is regulated to 6 to 8% of the content of PMS in Nigeria (Rowat, 1998), and between 1%(v/v) to 5% in the USA and Europe (Menkes et al., 1997; Wallace, 1996).

Although, leaded petrol is no longer available, unleaded however still contain small traces of lead. The material safety for unleaded gasoline shows at least 15 hazardous chemicals occurring in various amounts, including benzene (up to 5% by volume, toluene (up to 35% by volume), trimethylbenzene (up to 7% by volume), naphthalene (up to 1% by volume), trimethylbutyether (MTBE) up to 18% by volume, in some states and about 10 others. Benzene and lead content of PMS poses health risks for everyone (Dean, 1985). Exposure to benzene causes various cancers, skin, lungs and kidney damage and death upon exposure to very high concentration (Rinsky et al., 1981). Others includes auto immunity (regressing to diseases of the joint e.g. Rheumatoid arthritis), and Central nervous involvement. Others include, mucous membrane irritation .However, it is worthy to note that there is acute and chronic toxicity (Carballo et al., 1995).

After acute inhalation of the PMS fumes, they are rapidly absorbed into the blood and distributed throughout the body resulting in mucous membrane irritation, neurological and other symptoms due to respiratory failure (Hunter, 1966; Carballo et al., 1995). Chronic toxicity on the other hand has been reported to cause Bone-Marrow (BM) depression, a plastic and leukaemia, cardiac abnormalities, heart attack and other cancer of the lungs, brain and stomach (Travis et al., 1990).

High doses however can results into developmental retardation of foetus. It poses a higher risk in children, unborn babies and women of child bearing age. Children exposed to high level of lead after birth may develop behavioural and learning problem, slow growth and finally hearing loss (EPA, 2009). Exposure to high levels of lead during pregnancy contributes to miscarriages, preterm birth, low birth weights and developmental delays in infants (Kunitz and Levy, 1974).

Lead, when absorbed by the bone and fat in the brain can remain for 10years.Over this time, it is released back into the body after sniffing stops (Bass, 1970). Thus accounting for the social and behavioural problem often associated with workers handling PMS (Nurcome et al., 1970). Further study revealed that most women who are exposed to higher than average levels of air pollution have healthy babies.However, studies from the United States and other countries suggest that pregnant women exposed to high levels of certain air pollutants may be slightly more likely than pregnant women living in less polluted area to have a premature or small –for- age children (Stillerman et al., 2008).

Continuous exposure of PMS vapour has been reported in both experimental animal and human to cause harmful effects on reticuloendothelial system by destroying or inhibiting the haematopoietic components of the BM and induction of leukaemia during occupational exposure (Wixtron and Brown, 1992). In a recent publication, benzene when inhaled into the body produces met haemoglobin (methb) which differs from Haemoglobin based on the absence of electron that is needed in the formation of a strong bond with oxygen needed for oxygen transport. Increase in level of methb results in symptoms like shortness of breath, palpitation, anxiety, and confusion. PMS, also cause a reduction in Haematological indices which become chronic with prolong exposure. The measurement of serum methb is an indirect method of its evaluation of this form of poisoning (Okoro et al., 2006).

OBJECTIVE OF THE STUDY

The purpose of this research is to investigate the potential risk associated with exposure of non-pregnant women ofchild bearing age selling PMS (WSP) in Okada-Ore Road, Edo State, Nigeria using batteries of haematological parameters and level of MetHb as an indirect blood biomarker of exposure to benzene in PMS. This study is worthwhile because no studies have been reported on blood dyscrasia among this study group.

SUBJECT AND METHODS

Women recruited to this study are age matched for control and study population. The study was conducted between January, 2008 to January, 2009. The length of the work in their business was taking as the duration of

Table 1. Showing the Age distribution of subject (Women Selling Pms) and control (women not selling PMs.

Variable	Age (years)	
Age group (years)	**PMS Seller (%)**	**Control / Non-Seller (%)**
18-21	15(15.0)	15(15.0)
22-25	40(40.0)	40(40.0)
26-29	35(35.0)	35(35.0)
≥30	10(10.0)	10(10.0)
Total	100(100.0)	100(100.0)

Table 2. Blood cell counting relation to year of exposure to PMs.

Years of exposure	WBC (10^9/L)	Platelets (10^9/L)	RBC (10^{12}/L)
≤ 1	4.20±0.10	260±10.0	3.80±0.05
2-3	3.60±0.20	230±20.0	3.65±0.05
3-4	3.40±0.15	160±10.0	3.20±0.05
≥5	2.80±0.10	130±10.0	3.00±0.05
Control	4.60±1.50	285±30.0	4.70±1.50

exposure to PMS.This cross-sectional survey was carried out among women of child bearing age selling PMS in Okada- Ore Road as test subject and apparently healthy, non PMS seller as control population.100 women each for both test and control population within the age 18-34 years were recruited into the study after an informed consent through an oral interview and structured questionnaire. 5Milliliters of the venous blood from the ante-cubital vein was collected into an EDTA Bottle using standard methods. It was stored in a cool pack before taken to the laboratory for analysis. Blood sample were analysed within 24 h of collection. Haematological parameters were determined according to method of Dacie and Lewis, 1984. Analysis of Met-Hb was done using a method according to Evelyn and Malloy, 1938.

RESULTS

Determination of methhaemoglobin

About 0.2 ml of blood was lysed in a mixture of 4 ml of phosphate buffer (pH 6.8) and 6 ml of non-ionic detergent. The lysate was then divided into two equal volumes in separate tubes (A and B). The absorbance of 1 part was read spectrophotometrically at 630 nm (Tube A) and recorded as DI. The absorbance spectrum of met-Hb exhibits a small characteristic peak of 630 nm. The addition of cyanide eliminates the peak and the decrease in absorbance is proportional to the metHb concentration. Addition of ferricyanide measures total Hb after conversion to metHb. One drop of potassium ferricyanide was added to the 1 part A, mixed and absorbance recorded as D2. One drop of potassium cyanide was added to B, mixed and allowed to stand for 5 min and absorbance recorded as D3. One drop of potassium cyanide was added to B, mixed and read as absorbance D4 (Tables 1 to 4). 4 ml of phosphate buffer plus 6 ml non-ionic determine was used as blank. Result was expressed as:

MetHb (%) = D1-D2/D3-D4) × 100

DISCUSSION

In the study of the acute toxicological effects of diesels and crude oil that contains benzene in an experimental animal, it was observed that an increase in the dose of fuel administered into the animals caused a dose dependent decrease in Hb and PCV (Dede and Kagbo, 2002). Haematological changes in women of child bearing age expose to PMS have not been well assessed in Nigeria. Observation from our study revealed that Hb and all other haematological parameters decreases with increase level of exposure resulting in progressive anemia. This observation is married to constant and repeated unproductive exposure to PMS thereby eliciting gradual iron depletion in the iron store via inhalation of fumes resulting is serious public health concern (Hotz and Lauwerys, 1992).

A study by Akintonwa and his colleagues revealed that chronic exposure to petroleum products may have deleterious effects on the health of the filling station

Table 3. Showing Hbg/dl, PCV and Red cell indices with year of exposure to PMs.

Exposure (Years)	Hbg/dl	PCV (L/L)	Red cell count	MCV (fl)	MCHC (g/dl)	MCH (pg)
≤ 1	13.20±0.06	0.39±0.01	3.80±0.05	102.63	33.85	34.74
2-3	11.30±0.12	0.36±0.02	3.65±0.05	98.63	31.39	30.96
3-4	10.60±0.14	0.34±0.01	3.20±0.05	106.25	31.18	33.13
≥5	9.46±0.12	0.30±0.01	3.00±0.05	100.00	31.53	31.53
Control	13.50±0.01	0.42±0.01	4.70±1.50	89.36	33.33	28.72

Normal value Hbg/dl 12 g/dl (women non-pregnancy); MCV (Men and women 92±9 fl), MCH 29.5±2.5 Pg; MCHC (33±15 g/dl) as expressed as a mean±2SD (95% range) (Evatt et al., 1983)[34].

Table 4. Exposure to PMS and percentage (%) of Met Hb.

Exposure (years)	Test subject Met Hb %
≤1	2.4
2-3	2.9
3-5	8.0
≥5	10.5

Normal Range - Met Hb< 2.0%.

attendants studied. They however, concluded that exposure may have little or no effect on the red and white cell but cause a significant decrease of 43% in platelet count as compare with the control group (Akintonwa et al., 2005). Their finding is contrary to a study conducted in the same group of subject in Southern Nigeria with significantly higher effect on red, white, and platelet cellas compared with control (Hayes et al., 1997). These finding equally agree with result obtained from similar work conducted by (Dede and Kagbo, 2002; Udonwa et al., 2009).From our study also, it was equally observed that women exposed to petroleum had a significant higher Met Hb than control group.

Other Study confirms that most women who are exposed to higher than average levels of air pollution have healthybabies, However, studies, from the united states and other countries suggest that pregnant women exposed to high levels of certain air pollutant as benzene may be slightly more likely than pregnant women living in less polluted areas to have a premature or small for gestational-age baby (Stillerman et al., 2008; Wilson and Schwarzman, 2009).

From other similar work done in southern Nigeria in a separate group handling PMS, it was reported that the major route of exposure to PMS is by mouth suction due to conservation of time for maximum profit making and skin contamination 29.33. In develop countries as USA exposure has been limited to the use of rubber hood over the delivery pump to use of ‘’self service’’ at the stations. Mohorovic (2003); Wilson and Schwarzman (2009) in their findings observed in its chronic toxicity summary that the inhalation reference exposure level of 60 ug/m^3 causes a critical effect on lowering red and white cell counts in occupationally exposed humans. The hazard index targets include haemopoietic system, development, and nervous system (Aksoy et al., 1972).

Observation from our study stained thin film of subject exposure for and above 5 years showed a leucoerythroblastoid picture with decrease total leucocyte count this finding is similar in the work done by Carballo et al. (1995). The red cell indices decrease with increase in years of exposure. Red blood picture of subject within 1-3 years of exposure shows that of dimorphic blood picture with microcytic normocytic hypo chromic cells as compared with the pattern of result of red cell indices. Platelet count decrease with level of Subjects above 5 years of exposure resulting in mild thrombocytopenia with few blasts.

This finding is similar to recent report by Wixtron and Bown, 1992; Aksoy et al., 1972has demonstrated the Impairment of immune function and/or various anaemia may result from the haematotoxicity with hematologic lesion and bone marrow involvement leading to peripheral lymphocytopaenia and / or pancytopenia following chronic exposure (Aoyama, 1986).These lesions can lead to the induction and or development of leukaemia years after apparent exposure (Aoyama, 1986).

This study suggested increased exposure to PMS fumes among its seller while the primary route is oral suction and skin contact. Met Hb is a useful biomarker in determining the level of exposure. In Nigeria,

PMSdispensing station is scarcely inadequate in most rural and some urban-rural communities. It is therefore recommended to enlighten campaign to educate PMS seller on negative health implication of exposure to fumes. Close regulatory monitoring of individual selling petroleum and petroleum products is also very important.This can be achieved by mandatory provision and enforcement of the use of protective fitting s and or adequate stationing of filling station with dispenser in local communities to reduce the fumes inhalation.

REFERENCES

Agency for Toxic Substances and disease Registry (ATSDR) (2007). Lead toxicity: Patient information Sheet. Retrieved August 10, 2009.

Agency for Toxic Substances and Disease Registry (ATSDR) (1990). Toxicological profile for nitrobenzene, U.S Department of Health and Human Services. Public Health Service, Atlanta, Ga, USA, 1990.

Akintonwa A, Ojo BA, Emeka P, Coker HAB, Sofola O (2005). Effect of Chronic Exposure to petroleum products on some Hematological and Biochemical parameters JHM, 15(2): 77-82.

Aksoy M, Dincol K, Erdem S, Akgun T, Dincol G (1972). Details of blood changes in patients with pancytopenia associated with long-term exposure to benzene. Br. J. Ind. Med., 29: 56-64.

Anderson, D, Yu TW, Schmeizer P (1995). An investigation of the DNA .damaging ability of benzene and its metabolites in human lymphocytes using the comet Assay. Environ. Mol. Mutat., 26: 305-314.

Aoyama K (1986). Effects of benzene inhalation on lymphocyte subpopulations and immuneresponse in mice. Toxicol. Appl. Pharmacol., 85: 92-101.

Aston-Jones N (1998). The Human Ecosystem of the Niger Delta: An Era Handbook, Environmental Rights Action, Lagos, Nigeria., pp. 136-138.

Bass M (1970). Sudden Sniffing Death. J. Am. Med. Asso., 212(12): 2075-2079.

Becker CE (1985). Principles ofoccupational Medicine, In; CecilTextbook of Medicine, 17th ed. (J.B. Wyngaarden and L. H. Smith, Jr. eds.) pp. 2277-2279. W. B. Saunders CoPhiladalphia.

Carballo MA, Nigro ML, Dicarlo MB, Gasparini S, Campos S, Negri G, Gadano A (1995). Ethylene oxide II :Cytogenic and biochemical studies in persons occupationally exposed : Environ. Mol. Mutat., 25(25): 81-97(S).

Collins C (2007). Implementing phytoremediation of petroleum Hydrocarbons Methods of Biotechnology. 23. Humana Press. p. 100.

d'Azevedo PA, Tannhauser AL, Tannhauser SL (1996). Haematologicalalternations in rats from xylene andbenzene Vet. Human Toxicol., 38(5): 340-344.

Dacie JV, Lewis SM (1984). Practical Haematology .6th ed. Churchill Living Stone London.

Dean BJ (1985). "Recent findings on the genetic toxicology of benzene, toluene, xylenes and phenols," Mutation Res., 154(3): 153-181.

Dede EB, Kagbo HD (2002). "A study on the acute toxicological effects of commercial diesel fuel in Nigeria in rats (*Ratus ratus*) using hematological parameters." J. Appl. Sci. Environ. Manage. 6(1): 84–86.

Evelyn KA, Malloy HT (1938). "Micro determination of met haemoglobin and sulphaemoglobin in a sample of blood," Br. J. Haematol., 126: 555–652.

Hayes RB, Yin SN, Dosemeci M (1997). "Benzene and the dose-related incidence of hematologic neoplasms in China," J. Nat. Can. Institute, 89(14): 1065–1071.

Hotz P, Lauwerys RR (1997). "Hematopoietic and lymphatic malignancies in vehicle mechanics," Crit. Rev. Toxicol., 27(5): 443-494.

Hunter CG (1966). "Aromatic solvents," Ann. Occupational Hyg., 9(4): 191-198.

IARC (1982). "Some industrial Chemicals and dyestuffs, "IARC Monogr Eval. Carcinog Risks Hum., 29: 95-148.

Kunitz SJ, Levy JE (1974). Changing ideas of alcohol use among Navaho Indians. Quarterly J. Stud. Alcohol, 35(1): 243-259.

Material and Safety data Sheet (MSDS) (2003). Tesoro Petroleum Companies, Inc, US, 8 February.

Menkes DB, Fawcett JP (1997). "Too easily lead? Health effects of gasoline additives" Environ. Health Perspect., 105(3): 270-273.

Mohorovic L (2003). The level of maternal methemoglobin during pregnancy in an air-polluted environment," Environ. Health Perspect., 111(16): 1902-1905.

NNPC (Nigerian National Petroleum Corporation) (2011). Draft Annual Statistical Bulletin.www.nnpcgroup.com. Accessed on August 12, 2012.

Nurcombe B, Bianchi GN, Money J, Cawte JE (1970). A hunger for stimuli: Psychosocial Background of petrol inhalation.Br. J. Med. Physiol., 43: 367-374

Okoro AM, Ani EJ, Ibu JO, Akpogomeh BA (2006). Effect of petroleum products inhalation on some haematological indices of fuel attendants in calabarmetropolis,Nigeria. Nig. J. Physiol. Sci., 21(1-2): 71-75.

Oxford English Dictionary Online edition, entry 'Gasoline'

Perera F, Li Z, Whyatt R, Hoepner L, Wang S (2009). Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. Paediatrics, 124(2): e195-e202.

Rabble GK, Wong O (1996). Leukemia mortality by cell type in petroleum workers with potential exposure tobenzene. Environ. Health Perspect., 104: 1381-1392.

Rinsky RA, Young RJ, Smith AB (1981). "Leukaemia in benzene workers," Am. J. Ind. Med., 2(3): 217-245.

Ron H (2004). "Our Monitoring Heritage. Petrol and oil.

Chrysler Collector. (154):16-20
Rothman N, Li GL, Dosemeci M, Bechtold WE, Marti GE, Wang YZ (1996). Haematotoxicity among Chinese Workers-heavily exposed to benzene. Am, J. Ind. Med., 29(3): 236-246.
Rowat SC (1998). “Integrated defence system overlaps as a disease model withexamples for multiple chemical sensitivity.” Environ. Perspect., 106: 85-109.
Smith JH, Mallet AK, Brantom PG (1996). Ninety days feeding study inFischer – 344 rats of highly refinedpetroleum-derived food grade white oilsand waves. Toxicol Pathol., 24: 214-230.
Stillerman, K, Mattison DR, Giudice LC, Woodruff TJ (2008). Environmental Exposures and adverse pregnancy outcomes. A review of the Science. Reprod. Sci., 15(7): 631-650.
Travis CC, Quillen JL, Arms AD (1990). “Pharmacokinetics of benzene.” Toxicol. Appl. Pharmacol., 102: 400–420,.
US Environmental Protection Agency (EPA) (2009). Lead in paint, dust and Soil. Retrieved August 10, 2009.
Udonwa NE, Uko EK. Ikpeme M, Ibanga A, Okon BO (2009). Journal of Environmental and Public Health Volume, Article ID 281876, 5 pages Exposure of Petrol Station Attendants and Auto Mechanics to Premium Motor Sprit Fumes in Calabar, Nigeria.
Wallace L (1996). “Environmental exposure to benzene: an update,” Environ. Health Perspect., 104: 1129-1136.
Wilson M, Schwarzman M (2009). Towards a new U.S Chemicals policy: Rebuilding the foundation to advance new science ,green chemistry, and environment health. Environ. Health Perspect., 117(8): 1202-1209.
Wixtron RN, Brown SL (1992). Individual and population exposure to gasoline. J. Expo. Environ. Epidemiol.. 2: 23-28. PMID: 1380368 URL:http://www.icbi.nlm.nih.gov(s)