Clinical Toxicology

Ezejiofor, J Clin Toxicol 2016, 6:1
http://dx.doi.org/10.4172/2161-0495.1000276

Research Article Open Access

Haematological Indicators of Exposure to Petroleum Products in Petroleum Refining and Distribution Industry Workers in Nigeria

Tobias I Ndubuisi Ezejiofor*

Department of Biotechnology, Occupational and environmental toxicology Unit, School of Biological Sciences, Federal University of Technology, Owerri, Nigeria

***Corresponding author:** Ezejiofor TIN, Department of Biotechnology, Occupational and environmental toxicology Unit, School of Biological Sciences, Federal University of Technology, Owerri, Nigeria, Tel: +2348036774598; E-Mail: tinezejiofor@gmail.com; favourtine@yahoo.com

Received date: Oct 18, 2015; **Accepted date:** Oct 20, 2015; **Published date:** Jan 27, 2016

Abstract

Objective: Exposures to hazardous conditions in industrial environments often results in sundry health effects among workers. The study investigated haematological effects of occupational activities in the petroleum refining and distribution industry in Nigeria.

Methodology: Adopting routine laboratory methods, haematological indices were investigated in whole blood from randomly selected workers of Port Harcourt Refining Company (PHRC) and Pipelines and Petroleum Product Marketing Company (PPMC) both in Alesa-Eleme near Port Harcourt, Nigeria, as well as non-oil work civil servants serving as control subjects.

Results: Erythrocyte Sedimentation Rate (ESR) ranged 1-100 (Mean:10.94 ± 11.82 mm/hr) in oil workers against 1-36 (Mean:6.6 ± 7.81 mm/hr) in non-oil workers ($P<0.05$); haemoglobin (Hb): 7.60-21.10 (13.19 ± 1.31 g/dL) versus 9.10-14.90 (13.01 ± 1.54 g/dL) ($P>0.05$); Parked Cell Volume (PCV): 25.00-58.00 (43.31 ± 4.09%) vs.30-49 (42.70 ± 5.01%) ($P>0.05$); Platelets: 75.00 × 10^9-430.00 × 10^9 (232.41 ± 63.18 × 10^9/L) vs. 141.00 × 10^9 -382.00 × 10^9 (239.23 ± 57.30 × 10^9/L) ($P>0.05$); White Blood Cell (WBC):3.20 × 10^9-86.00 × 10^9 (7.07 ± 6.61 × 10^9/L) vs. 4.9 × 10^9-11.00 × 109 (7.36 ± 1.64 × 10^9/L) ($P>0.05$). For the WBC differentials, the values were: lymphocytes: 18.00 × 10^9-75.00 × 10^9 (52.28 ± 9.25 × 10^9/L) vs. 25.00 ×10^9-57.00 × 10^9 (41.60 ± 10.16 × 10^9/L) ($P<0.01$); and granulocytes: 25.00 × 10^9-82.00 × 10^9 (47.72 ± 9.24 × 10^9/L) vs. 43 × 10^9-75 × 10^9 (58.40 ± 10.16 × 10^9/L ($P<0.01$).

Conclusion: Although mean values were still within parametric reference ranges, some variations were observed in the oil workers when compared to the controls: while granulocytes consistently decreased significantly ($P<0.01$), consistent significant increases in lymphocytes ($P<0.01$) and ESR ($P<0.05$) were observed, indicating a possibility of functional aberration following haematopoietic toxicity in the oil workers. Findings suggest petroleum refining and distribution industrial environments as being furnished with potentially haematotoxic substances, and haematopoietic toxicity as part of potential health effects of exposures in this industry in Nigeria. Though gender classification showed no appreciable impact, age grouping suggests that the health effects indicated by the observed variations are likely to rear up from age 40 yr. Changes observed for exposure groupings and statistically significant correlations between age, exposure (service) period and most of the parameters suggest that both age and exposure period have strong impacts in defining the patterns of variations observed in the haematological indices among the oil workers. Findings indicate a need for frequent environmental and biological monitoring for a safer and healthier workplace and workforce respectively.

Keywords: Haematological indicators; Exposures; Petroleum refining and distribution industry; Workers; Nigeria; Toxicology; Public health implications

Introduction

Petroleum refining and distribution: general environmental, ecological and health impacts

Petroleum consists of crude oils and a variety of refined oil products, and is also a significant source of polycyclic aromatic hydrocarbons (PAH) [1]. The industry of petroleum refining and distribution play an important role in terms of number of employees in the Nigerian production and overall economy. Petroleum refining has evolved continuously in response to changing consumer demands for better and different products, and involves processing of mainly oil to obtain mixtures of hydrocarbon compounds, the products of which are specified on the basis of aptitude for use. For example gasolines, are obtained by mixing of fractions of the first distillation, reforming products, and antiknock [2]. Once extracted, crude oil is transported to an oil refinery where complex hydrocarbon compounds are separated and converted through various refining operations (fractional distillation, cracking, solvent extractions, then other treatments including formulating and blending) to become useable fuel sources. Finally, impurities are removed through chemical treatment of each product. The process of refining oil manufactures nearly 2,500 useful products [3]. The major end product of oil is gasoline, followed by diesel fuel, jet fuel, fuel oil, kerosene, lubricating oil and asphalt used for road paving. Through complex network of pipelines and storage tanks, the products of the refineries are passed over for subsequent distribution by an appropriate body, which in the

Nigeria situation, is handled by the Pipelines and Petroleum products Marketing Company (ppmc).

Petroleum refining and distribution are among the occupational activities perceived to be hazardous [4]. The presence, physico-chemical properties and toxicological characteristics of other important petroleum products like benzene, toluene, xylenes, ethyl benzene, n-hexane, volatile hydrocarbons belonging to gasoline, kerosene, and diesel fuel (contents defined by the technology of the manufacturing processes), determine the extent of impacts of this industry on both the workers and environment. Apart from the products, oil refineries also contribute various forms of pollution including thermal and noise pollution. Thermal pollution involves the discharge of effluents that are significantly warmer than surrounding water, while noise level in refineries can exceed 90 decibels, posing a significant threat to the health and safety of oil refinery employees and even the surrounding community because leakage of noise pollution can have significant psychological effects on local residents, decreases aesthetics of the area and can interfere with wildlife [5]. Again, oil refineries are inherently complex in their equipment and structural design that combined with the multitude of chemicals used, there exist a continuous risk of accidents involving fires, explosions, chemical spills and burns as well as numerous other health effects [6]. Oil refinery workers are therefore continuously exposed to numerous hazardous materials and working conditions that place them at continuous risk of injury and death. Such chronic hazards include exposure to noise, heat, polluted air, varieties of hazardous substances used either as process chemicals and/or present in resultant effluents/wastes as well as in the usually invisible emissions, which include petroleum itself and other aromatic hydrocarbons (benzene, toluene, phenol, etc.), hydrogen sulphide and other natural gases (methane, propane, butane, etc.), carbon monoxide, asphalt, toxic heavy metals (arsenic, chromium, cadmium, nickel, zinc, etc.), coke dust, lead alkyls, silica, asbestos, etc. [6,7].

Exposures to these and other substances in petroleum or its associated refining operations are harmful. For instance, continuous exposure to carbon monoxide can lead to headaches and mental disturbances, and at high concentrations may bring about death from asphyxiation. Long-term exposure to coke dust, silica and hydrogen sulphide can lead to chronic lung disease, while Lead alkyls used as gasoline additives can lead to psychosis and peripheral neuropathies. Asbestos, often used in oil refineries for the thermal insulation of boilers and pipes, has long been associated with pulmonary fibrosis, lung cancer and malignant mesothelioma and other cancers among maintenance, repair and removal workers, and other workers exposed to asbestos [8,9]. Hydrocarbons which are among the major components of petroleum products are considered toxic and have been implicated in a number of human diseases [10]. A threefold increase in kidney cancer risk has been noted for exposure to hydrocarbons following occupational exposures to crude oil in oil refining activities. The risk was associated with the highest cumulative exposure category to hydrocarbons in crude oil [11]. Blood pressures and related vascular conditions had been linked to PAH exposures via residential proximities to refineries [12]. The risks are also noted even in occupational activities in which exposures are gathered by mere use of refinery products as production material (e.g., production of asphalt roofing products using asphalt from refiners and crude oils (here, PAH compounds are part of asphalt emissions). Polycyclic aromatic hydrocarbons of 4-6 rings are strongly correlated with carcinogenic activity in animal studies [13]. Asphalt, a component of petroleum hydrocarbons, for example, can cause severe burns and eye irritation, and its fumes may contain unacceptable levels of benzene, while hydrogen sulphide may lead to dermatitis, bronchitis and chemically induced pneumonia [8]. Health risk assessment for exposure to benzene in petroleum refinery environments suggest a potential cancer risk for exposure to benzene in all the scenarios [14]. Benzene exposure is known to affect many critical organs including the hematological, hepatic, renal, cardiac, and lung functions [15]. Significantly impaired lung function parameters have been noted among subjects working in petroleum refining industry and indicates obstructive lung disease among these workers [16].

Indeed, chemical pollution from refining activities is widespread, affecting not just the workers but also almost all strata of the environment. Perhaps, the extent of massive pollution and the consequent environmental and ecological degradation caused by petroleum refining and distribution activities is better captured from the report of studies conducted in the oil-rich Niger delta region of Nigeria by the United Nations Environment Programme (UNEP). The report indicated that the oil-rich Niger Delta suffers from extensive petroleum contamination. A pilot study was conducted in the region of Ogoniland where one community, Ogale, has drinking water wells highly contaminated with a refined oil product. In a 2011 study, UNEP sampled Ogale drinking water wells and detected numerous petroleum hydrocarbons, including benzene at concentrations as much as 1800 times higher than the USEPA drinking water standard, thus compelling a recommendation by UNEP for immediate provision of clean drinking water, medical surveillance, and a prospective cohort study [17]. Another study by Linden and Palsson, [18] confirmed extensive oil contamination of rivers, creeks, and ground waters in Ogoniland, Nigeria. According to their report, the levels found in the more contaminated sites are high enough to cause severe impacts on the ecosystem and human health: extractable petroleum hydrocarbons (EPHs) (>10-C40) in surface waters up to 74201/4 gL (-1), drinking water wells show up to 422001/4 gL (-1), and benzene up to 90001/4 gL (-1), more than 900 times the WHO guidelines. EPH concentrations in sediments were up to 17900 mg/kg (-1). Polycyclic aromatic hydrocarbons concentrations reached 8.0 mg/kg (-1), in the most contaminated sites. Unfortunately, the contamination has killed large areas of mangroves. Although the natural conditions for degradation of petroleum hydrocarbons are favorable with high temperatures and relatively high rainfall, the recovery of contaminated areas is prevented due to the chronic character of the contamination. Oil spills of varying magnitude originates from facilities and pipelines; leaks from aging, dilapidated, and abandoned infrastructure; and from spills during transport and artisanal refining of stolen oil under very primitive conditions.

Petroleum distribution activities also present the challenges of oil spillage as revealed in another report describing the wide spread of varieties of petroleum hydrocarbons in our environments. An accidental damage of a Nigerian National Petroleum Corporation (NNPC) pipeline that occurred in Ijegun area of Lagos, Nigeria, in May 2008 resulted in oil spillage and consequent contamination of the environment [19]. "The residual concentration of the total hydrocarbon (THC) and benzene, toluene, ethyl benzene, and xylene (BTEX) in the groundwater and soil was therefore investigated between March 2009 and July 2010. Results showed elevated THC mean levels in groundwater which were above the World Health Organization maximum admissible value of 0.1 mg/L. THC values as high as 757.97 mg/L in groundwater and 402.52 mg/L in soil were observed in March 2009. Pronounced seasonal variation in the concentration of THC in groundwater and soil samples show that

there was significant (P<0.05) difference in the measured concentration of THC between each season (dry and wet), with the highest being in the dry season and between the years 2009 and 2010. Significant hydrocarbon contamination, 500 m beyond the explosion site and 25 months after the incident, was observed revealing the extent of the spillage of petroleum products. The highest concentrations of 16.65 1/4g/L (benzene), 2.08 1/4g/L (toluene), and 4864.79 1/4g/L (xylene) were found in stations within the 100 m buffer zone. Most of the samples of groundwater taken were above the target value of 0.2 1/4g/L set for BTEX compounds by the Environmental Guidelines and Standards for Petroleum Industry in Nigeria". These observations highlight the potential risk to public health for a population where, unfortunately, oil spillages occur frequently. The public health implications of these are therefore far-reaching, since exposures to Nigerian grade of petroleum (Nigerian bonny light) has been associated with sundry effects in various species of organisms. For instance, Nigerian bonny light crude oil have been reported to induces endocrine disruption in male rats [20], as well as alteration in testicular stress response proteins and caspase-3 dependent apoptosis in albino wistar rats [21].

Refining and distribution facilities, activities and products: potential sources of hazards to the industry workers in Nigeria

Petroleum refining and distribution industry in Nigeria, as elsewhere, constitute a giant industry with many complicated systems. Alesa-Eleme near Port Harcourt, Nigeria, West Africa, is the location of the main operational facilities of both PHRC and PPMC, and lies within latitude 4.7706o and longitude 7.1056o. The PHRC has five process areas (areas1-5), each of which houses several operational units named according to the nature of activities or work performed in them [22], and accordingly, they also constitute potential sources of exposures to the workers performing those activities, and particularly so when the facilities or parts thereof become defective and consequent outlets to misty and gaseous fumes of various process materials, products or wastes/effluents. The process of production and the actual products are both sophisticated. PHRC processes crude oil (Bonny Light) into liquefied petroleum gas (LPG), Premium Motor Spirit (PMS), Dual purpose Kerosene (DPK) (Aviation and Domestic), Automotive Gas Oil (AGO- Diesel), Low Pour Fuel Oil (LPFO), and High Pour Fuel Oil (HPFO) as well as many other intermediate products that are industrially and domestically very useful. Through complex network of pipelines and storage tanks, these products of the refineries are passed over to the Pipelines and Petroleum products Marketing Company (ppmc) for subsequent distribution. Behind all these facilities and their operations are workers, who are therefore, considered liable to certain health effects and/or impacts on account of several hazards from sundry job exposures in the various work units.

The knowledge that most human diseases and sufferings are sometimes related to the hazards of their workplace meant that appropriate remedies to the situation would be possible only when these hazards are properly assessed, their very nature, extent and impacts firmly established. Unfortunately, for most of the industrial establishments in Nigeria particularly the numerous small and medium scale industries that form the bedrock of her industrial activities, and even some of the large scale industrial concerns (inclusive of the petroleum and petrochemical industry that is the mainstream of her economy), such occupational studies are yet to be carried out. In the Nigerian setting, available data regarding petroleum industry are based on studies using animal models, but not much is reported on humans, who for occupational reasons are subjected to long term, low-level continuous exposures to petroleum fractions, intermediates and finished products, as well as other hazardous conditions in their work environments. This has resulted in a dearth of data regarding the nature and extent of health effects of sundry job exposures in this industry in Nigeria. Studies done elsewhere with regard to workers in the oil and gas industry had documented some organs/systems health effects, producing various morbidities and mortalities [23-34,11]. Although most Nigerian studies have reported the effects of petroleum exposures in animal models that could possibly be extrapolated for humans [35-41], there are need for a direct human assessment of the situation using human biological samples. Results to be obtained from such direct human studies is expected to give a more assured situation with regards to human toxicology of petroleum products in Nigeria than an extrapolated result, which might be affected by species differences.

Meanwhile, risk assessment of this same petroleum refining and distribution industrial work environment revealed that the workers are furnished with sundry hazardous exposures [42], just as a study of some anthropometrical and biochemical markers also showed cardiovascular diseases, toxic nephropathy, and anicteric toxic hepatitis as part of diverse potential health risks/hazards of this work cohorts[43-45]. Presently, the impacts of these exposures, particularly as it concerns the haematological effects remain uncertain in Nigeria, and thus forms the major focus of this study aimed at exploring the haematological implications of workplace conditions, and exposures to a wide range of substances present in emissions in the environment of petroleum refining and distribution industry in Nigeria, which in the opinion of this author, are potentially haematotoxic. They have deleterious effects on human body, and have also been recognized as carcinogens by the International Agency for Research on Cancer (IARC) [23]. In relation to their haematological functions, haematopoietic system in humans, animal models or naturally exposed fauna, are target tissue for several substances and metals (at least Cd or As), and accumulations of these beyond the body's detoxification capacity portend serious dangers to the haematopoietic health, and by extension, the overall health of the individual. Meanwhile, data from the plant clinic jointly used by the two establishments being studied revealed haematological disease conditions as making reckonable contribution to the morbidities and mortalities recorded in this industry [46-47], suggesting that these diseases might be prevalent among the workers, and thus warranting further studies. In addition, there is need to provide some of the necessary data called for by Loewenson [48] and in particular, Scala [49] who had emphasized a three-fold need for toxicological data on the part of petroleum or petrochemical industry. Thus, the objective of this study is to evaluate these workers for possible health effects of occupational exposures in this industry, though with particular focus on the haematopoietic system, and to determine the role of gender, age and exposure period in defining any observed effect. Since our present attention is on the haematopoietic system, haematological markers would be assayed in the biological samples to be provided by the study participants.

Materials and Methods

Subjects

Participants in this study consisted of three hundred and thirty three (333) human subjects aged between 28 and 60 years old. Of this

number, three hundred and three (303) study participants (273 males and 30 females) were randomly drawn from the staff of the two industrial establishments studied: Port Harcourt Refining Company Ltd (PHRC) (Petroleum refiners) and Pipelines and Petroleum Products Marketing Company (PPMC) (Petroleum distributors); the remaining 30 were non-oil sector civil servants considered healthy as at the time of this study (20 males and 10 females) also randomly recruited to serve as referents or comparison group, and these were mainly classroom teachers from various Departments of an institution of higher learning, also located in a nearby neighbouring community to the study industry location, near Port Harcourt metropolis. Their age distribution matched those of the oil sector industrial workers (28-60 yr). In terms of their occupational history, information volunteered by the control subjects suggest they have been classroom teachers for most of their working life (being career academicians), which was the main reason for enlisting them as better control subjects to participate in the study. The nature and purpose of the study was explained to the participants, following which they willingly consented to participation in the study.

Inclusion and exclusion criteria

For the study participants from the industry, only those on the job for a period not less than 3 years (service period of 3 years or more) and without personal medical history of chronic ailments such as chest or genitourinary infections, renal disease, cardiovascular disease, cancer of any body site, or any other condition likely to cause abnormalities in haematological indices were included, while those with less service period and or any of the aforementioned disease conditions were excluded, since these conditions would likely present our study with the challenges of possible result confounders. For the referents, exclusion criteria included history of above medical conditions and involvement(s) in petroleum refining and distribution activities or any other activity that warrants prolonged and close contact/exposure to petroleum and gas products. For the oil workers, information regarding these was obtained from their medical records available at the plant clinic used jointly by the two establishments under study, with further clarifications volunteered by the occupational physician in-charge of the plant clinic; for the referents, same information was obtained with the help of a medical assistant in our team as she conducted physical/medical examination on each of the referents. Based on these criteria, 27 oil workers and 3 Non-oil workers were excluded. Thus, of the initial 363 persons initially recruited, only 333 persons eventually emerged as the actual participants studied.

Sample

Whole blood Sample: Using syringes and needles, venous blood (5ml) was collected from each of the participants and dripped into an anti-coagulant specimen container (containing dipotassium salt of ethylene diamine tetracetic acid (K2EDTA). The anticoagulant tube blood samples were gently but thoroughly mixed by inversion to ensure that the blood did not clot, and then stored in the refrigerator (4oC) for a maximum period of three days within which the samples were analyzed for the studied haematological parameters.

Methods

The hematological parameters determined include Erythrocyte Sedimentation Rate (ESR) and Full Blood Count- Haemoglobin (Hb), total white blood cells (WBC), granulocytes, lymphocytes and platelets. The methods adopted for the individual analysis were as given below:

Erythrocyte Sedimentation Rate (ESR): A general (non-specific) body-screening test was performed using the method of Westergreen [50]. First, the venous blood was diluted, one part of 3.13% trisodium citrate to 4 parts of blood. The diluted blood was well mixed and drawn into the top mark (0 mark) of the Westergreen ESR tube. The tube was then stood vertically for one hour, at the end of which period the level of the red cells was visually read as the erythrocyte sedimentation (ESR), the unit being millimeter per hour (mm/h).

Full Blood Count (FBC) By the QBC II plus Centrifugal Haematology System: Haematological parameters making up the full blood count (FBC) i.e. packed cell volume (PCV), Haemoglobin (Hb), total white blood cells (WBC), granulocytes, lymphocytes and platelets were determined using the QBC II plus centrifugal haematology system as described by Wardlaw and Levine [51].

Ethics

Ethical clearance was obtained from the Institutional Review Committee of the Department of Petroleum Resources (DPR), Federal Ministry of Petroleum Resources (the supervisory ministry). The procedures followed in the conduct of the study were in accordance with the ethical standards of the Institutional Review Committee of this ministry as it concerns human experimentation, and these conform to the Helsinki Declaration of 1975, as revised in 1983. Also, the nature and purpose of the study was explained to the management and staff of the establishments studied, following which approval for study and consent for voluntary participation respectively were obtained.

Statistics

Analysis of resultant data was done using statistical programme for social sciences (SPSS) Version 11. Descriptive statistics, T-test, Analyses of Variance (ANOVA), multiple comparisons analyses using Least Significant Difference (LSD (Post Hoc tests), regression and correlation analyses were some of the statistical analyses performed on the data.

Results

In Table 1 the value ranges and means of the haematological parameters (indicators) studied in the oil workers and control subjects were presented. The results showed that Erythrocyte sedimentation rate (ESR) ranged 1-100 with a mean of 10.94 ± 11.82 mm/hr in the oil workers as against 1-36 with a mean of 6.6 ± 7.81 mm/hr in the non-oil workers ($P<0.05$); hemoglobin (Hb) ranged 7.60-21.10 with a mean of 13.19 ± 1.31 g/dl as against 9.10-14.90 with a mean of 13.01 ± 1.54 g/dL for the non-oil workers ($P>0.05$). For the parked cell volume (PCV), the values were 25.00-58.00 with a mean of 43.31 ± 4.09% for the oil workers, and 30-49 with a mean of 42.70 ± 5.01% for the non-oil workers ($P>0.05$). Platelets ranged 75.00×10^9-430.00×10^9 with a mean of 232.41 ± 63.18 $\times 10^9$/L in the oil workers, and 141.00×10^9-382.00×10^9 with a mean of 239.23 ± 57.30 $\times 10^9$/L in the non-oil workers ($P>0.05$), while total white blood cell (WBC) ranged 3.20×10^9-86.00×10^9 with a mean of 7.07 ± 6.61 $\times 10^9$/L for the oil workers, and 4.9×10^9-11.00×10^9 with a mean of 7.36 ± 1.64 $\times 10^9$/L for non-oil workers($P>0.05$). For the WBC differentials, lymphocytes ranged 18.00×10^9-75.00×10^9 with a mean of 52.28 ± 9.25 $\times 10^9$/L in the oil workers, and 25.00×10^9-57.00×10^9 with a mean of 41.60 ± 10.16 ×

109/L in the non-oil workers (P<0.01), while granulocytes ranged 25.00 × 10^9-82.00 × 10^9 with a mean of 47.72 ± 9.24 × 10^9/L in the oil workers and 43 × 10^9-75 × 10^9 with a mean of 58.40 ± 10.16 × 10^9/L (P<0.01).

PARAMETER	NON-OILWORKERS (N=30) Range	Mean ± SD	OIL WORKERS (N=303) Range	Mean ± SD	P-Value (2-tailed)	Normal range and Unit of parameter
AGE	35.00-59.00	47.37 ± 6.74	28.00-60.00	43.30 ± 7.03	0.003	
SERVICE PERIOD**	3.00-34.00	20.03 ± 8.77	3.00-34.00	16.18 ± 6.42	0.003	Up to 35Yrs
ESR	1.00-36.00	6.60 ± 7.81	1.00-100.00	10.94 ± 11.82	0.05	0-7 mm/hr (M), 0-10 mm/hr (F)
Hb	9.10-14.90	13.01 ± 1.54	7.60-21.10	13.19 ± 1.31	0.487	13 g/dL-18 g/dL (M), 12 g/dL-15 g/dL (F)
PCV	30.00-49.00	42.70 ± 5.01	25.00-58.00	43.31 ± 4.09	0.448	40%-50% (M), 36%-47%(F)
PLATELETS × 109	141.00-382.00	239.23 ±57.30	75.00-430.00	232.41± 63.18	0.570	150-400 ×109 /L
TOTAL WBC × 109	4.90-11.00	7.36 ± 1.64	3.20-86.00	7.07 ± 6.61	0.813	4.0-11.0 ×109 /L
LYMPHOCYTE	25.00-57.00	41.60 ± 10.16	18.00-75.00	52.28 ± 9.25	0.000	
GRANULOCYTE	43.00-75.00	58.40 ± 10.16	25.00-82.00	47.72 ± 9.24	0.000	

T-test was the statistical tool applied. *Indicates statistically significant difference [**Service Period=number of years the worker had been engaged in the work, amounting to his/her cumulative years of exposure to the hazards of his/her workplace (i.e., exposure period). Presently, 35 yr is the maximum service period that qualifies an average civil servant for retirement in Nigeria, and is therefore considered as the upper limit of normal for service period in this report]; Key for letters in Normal Range: M = Males., F=Females.

Table 1: Value ranges and means of haematological parameters studied in the oil workers (PPMC and PHRC put together) compared with the non-oil workers.

A cursory look at the results showed that although the mean values for the studied haematological indices were still within their parametric reference ranges, there were reasonable variations in the values of these same parameters when compared with what obtained in the non-oil work referents. A close look at the haematological parameters showed erythrocyte sedimentation rate (ESR) to be significantly higher in oil workers than non-oil workers (P<0.05). Subsequently, the oil workers and their Non-oil work counterparts were separated according to gender, age, and exposure periods (service years), and the mean values for the studied parameters following these groupings were as presented in table 2 (Males), table 3 (Females), table 4 (Age groups) and table 5 (Exposure periods). ESR was also higher in both male and female oil workers than their corresponding sexes among the referent group, with the difference being very significant for the females (P<0.01) and non-significant for the males (P>0.05) (Table 1). For all the age groups, ESR was higher in oil workers than the referents, but at age groups 30-39 yr and 50-59 years, the observed increase of ESR in oil workers was significant (P<0.01), whereas the increases in the various exposure groups were not significant (P>0.05).

PARAMETER	NON-OILWORKERS (N=20) Mean ± SD	OIL WORKERS (N=273) Mean ± SD	P-Value (2-tailed)	Normal range and Unit of parameter
AGE	49.75 ± 5.15	43.63 ± 6.98	0.000	
SERVICE PERIOD	23.20 ± 7.81	16.32 ± 6.47	0.000	Up to 35 yr
ESR	5.45 ± 7.57	9.26 ± 8.97	0.065	0-7 mm/hr (M), 0-10 mm/hr (F)
Hb	13.72 ± 0.93	13.38 ± 1.16	0.202	13 g/dL-18 g/dL (M), 12 g/dL-15 g/dL (F)
PCV	45.05 ± 3.05	43.94 ± 3.54	0.172	40%-50%(M), 36%-47%(F)
PLATELETS ×109	243.35 ± 57.94	232.45 ± 64.32	0.462	150-400 ×109/L
TOTAL WBC × 109	7.38 ± 1.67	7.18 ± 6.94	0.898	4.0-11.0 ×109/L
LYMPHOCYTE	44.45 ± 8.46	52.30 ± 9.33	0.000	
GRANULOCYTE	55.55 ± 8.46	47.69 ± 9.31	0.000	

T-test was the statistical tool applied. *Indicates statistically significant difference

Table 2: Haematological parameters studied in male oil workers and male non-oil workers.

Discussion

There was consistent increase in the values for ESR in all segments of oil workers over their non-oil work peers, and this might be a pointer to some yet unspecified effects in the oil workers. The reality of this fact was corroborated by the correlation relationship that existed between ESR and most of the parameters studied in the oil workers, either at 95% or 99% confidence levels. Raised ESR is said to indicate for general health defects of non-specific nature [52]. Erythrocyte Sedimentation Rate (ESR) is a very useful non-specific test that in general, is increased in conditions associated with fevers and increased pulse rate, having the great advantage that certain chronic infections without fever often cause an increase; moreover, it is very often raised in inflammatory conditions and neoplastic conditions, particularly if there is tissue degeneration or if there is extensive metastases. It is also of great value in the assessment of rheumatic activity, and high values are common in the so-called collagen vascular diseases. Exceedingly high values are found in the immunoglobinaemias (a group of conditions which have in common the production of excessive amounts of monoclonal immunoglobulin) caused by proliferation of sometimes frankly malignant, sometimes relatively benign cells whose normal function is the elaboration of immunoglobulins. In this class belong the myelomatosis (a condition characterized by a malignant proliferation of plasma cells in the bone marrow, often regarded as an aleukaemic plasma cell leukaemia), and usually there are skeletal lesions, and renal involvement (renal failure) is one of the common causes in myelomatosis. Thus, multiple myeloma is often the first suspect when there is exceedingly high value of ESR. Values are also increased in anaemia. In some instances however, particularly among elderly patients, there is a marked elevation of the ESR and yet no abnormality can be found to account for it, though some of these later develop active disease, perhaps rheumatoid arthritis, but in others the ESR gradually reverts to normal though the process may take many months. Still, in yet other instances, even within normal ESR values, active infections such as for instance tuberculosis cannot be ruled out. However, the possibilities of the aforementioned conditions underscore the non-specificity of this parameter [52]. Although ESR is a non-specific marker diagnostically, it however gives a pointer (a kind of blowing an alarm) that something is wrong within the system, which calls for thorough investigation needing the application of a more specific diagnostic tool or marker. Inflammatory conditions of various aetiologies very often bring about raised ESR values. Thus, the consistently raised ESR values among the oil workers might be suggestive of a variety of clinical conditions occurring as effect(s) of occupational exposures to an equally variety of toxicants inherent within oil and gas work environment. Such occurrences, though might still remain latent for now, are still stealthily and progressively taking roots in these workers, especially those of the age groups 40-49 years (possibly the initiation point of the effects) and 50-59 years(possibly the maturation point for the full establishment and manifestations of effects). Olusi [53] had pointed out that most petroleum related chemicals can cause deleterious effects after long exposure, and the latency period varies from ten to twenty-five years. Of particular interest in this regard are inflammatory conditions such as arthritis, malignancies and other effects of the various body tissues/organs including those of blood-forming systems. Thus, subsequent differential diagnoses aided by appropriate markers and judicious interpretation of these parametric results surely would enable us come out with specific isolations from the very many possible clinical conditions (indicated by the observed raised ESR values) that may have occurred among these oil workers following exposures to petroleum hydrocarbons and other substances and/or conditions possibly present in the oil and gas work environment. Risk assessment of petroleum refining and distribution industrial work environment confirmed that the workers are indeed furnished with sundry hazardous exposures [4,42], while a study of some anthropometrical and biochemical markers indicated cardiovascular diseases, toxic nephropathy and anicteric hepatoxicity as being part of the diverse potential health hazards of this work cohorts [43-45]. Also, data from the plant clinic jointly used by the two establishments under study revealed haematological disease conditions as making some contribution to the morbidities and mortalities recorded in this industry [46,47]. These specific health conditions noted within this industrial sector have the potential to spur a significant rise in ESR values as observed in this study.

Looking at the haematological parameters generally, one observed that the mean values for the individual parameters fell within their parametric reference ranges. Again, with the exception of the significant increase observed in ESR ($P<0.05$) and lymphocytes ($P<0.01$) and a significant decrease in granulocytes ($P<0.01$), values for other parameters did not show any appreciable difference in oil workers compared with those of the non-oil workers ($P>0.05$) (Table 1). Same presentation trend was also observed when both the oil workers and the non-oil workers were separated according to gender and age with comparisons done for the corresponding sexes (Tables 2 and 3), and for equivalent age groups (Table 4) respectively. Though the values for all parameters were higher in the oil workers compared to the non-oil workers in the age group 30-39 years, only those of ESR ($P<0.05$) and lymphocytes ($P<0.01$) respectively increased significantly, even as the granulocytes decreased significantly ($P<0.05$) among the oil workers compared with the non-oil workers. However, at age groups 40-49 years and above, a pattern of general depression in most of the blood cell types was observed. With the exception of the lymphocytes that showed significant increase ($P<0.01$), all other blood cell types showed a decrease in the oil work cohorts, the decrease being significant ($P<0.01$) only for the granulocyte (Table 4.), indicating that whatever the stimulus exacting the effect on the bone marrow and causing the aplasia starts showing up from the age of 40 years, a trend that continued into the age group 50 yr and above for most of the blood cell types, albeit insignificantly ($P>0.05$). To determine if exposure period (service years) had any effect on the haematological parameters, the oil workers were separated into their various exposure periods and the resultant exposure groups compared with the referent group (Table 5). The observation was that in all exposure periods, ESR, Hb, PCV, and Lymphocytes remained higher in oil workers than in the referents, the difference was significant for ESR ($P<0.05$) only in the 1 yr-5 yr exposure group, while the difference was consistently significant for the lymphocyte ($P<0.01$) in all the exposure groups; the values for the granulocytes decreased significantly ($P<0.01$) in all the exposure groups of the oil workers compared with those of the referent

population. While the values for Hb and PCV remained insignificant (P>0.05) in all the exposure periods, those of both the WBC and Platelets showed variable decreases or depressions that also remained insignificant (P>0.05) in all the exposure groups of oil workers compared with those of the referents. Thus, apart from the lymphocytes increasing significantly(P>0.01) and granulocytes decreasing significantly (P<0.01) in the oil workers (ostensibly because of the mathematical relationship shared by both fractions of the total WBC count), and the insignificant depression observed for WBC and Platelets, no appreciable difference was noted for most of the parametric values in the exposed(oil work) relative to the unexposed(non-oil work) populations, neither was there any dose-dependent gradations across the various exposure periods(Table 5). These observations appeared to be corroborated by the correlation relationships between age, service period and some haematological indices, since Pearson's (2-tailed) correlation analyses of oil workers showed that strongly positive correlations existed between exposure period and age (P<0.01), and age is also strongly correlated with some parameters such as granulocytes (P<0.01) and lymphocytes (P<0.01); significantly negative correlation existed between exposure period and platelets (P<0.01), exposure period and lymphocytes (P<0.05); and there was significantly positive correlation between exposure period and granulocytes (P<0.05). This is quite understandable, since an older worker is more likely to have accumulated a longer service period, and therefore larger cumulative exposures to the conditions in his/her work place (see legend under Table1), with greater chances of manifesting the consequent effects where and when conditions for such manifestations are ripe, and of course, taking into account the latency period for some of such health effects. The changes observed for exposure groupings and the statistically significant correlations demonstrated between age, exposure (service) period and most of the parameters suggest that both age and exposure period have strong impacts in defining the patterns of variations observed in the haematological indices among the oil workers. The implications of these variations in the haematological profiles are great for the oil workers, given the wide array of conditions or circumstances that could precipitate such deviations from normal.

PARAMETER	NON-OILWORKERS (N=10) Mean ± SD	OIL WORKERS (N=30) Mean ± SD	P-Value (2-tailed)	Normal range and Unit of parameter
AGE	42.6 ± 7.25	40.33 ± 6.9	0.379	
SERVICE PERIOD	13.7 ± 7.21	14.97 ± 5.84	0.579	Up to 35Yrs
ESR	8.9 ± 8.16	26.27 ± 20.74*	0.014	0-7mm/hr (M), 0-10mm/hr (F)
Hb	11.59 ± 1.55	11.42 ± 1.31	0.741	13 g/dL-18 g/dL (M), 12 g/dL-15 g/dL (F)
PCV	38 ± 4.94	37.57 ± 4.29	0.791	40%-50% (M), 36%-47%(F)
PLATELETS × 109	231 ± 58.14	232 ± 52.59	0.960	150-400 ×109/L
TOTAL WBC × 109	7.32 ± 1.65	6.1 ± 1.61*	0.046	4.0-11.0 ×109/L
LYMPHOCYTE	35.9 ± 11.27	52.03 ± 8.65*	0.000	
GRANULOCYTE	64.1 ± 11.27	47.97 ± 8.65*	0.000	
T-test was the statistical tool applied. *Indicates statistically significant difference				

Table 3: Mean values of haematological parameters studied in female oil workers and female non-oil workers.

Haematological indices: Implications for the oil workers

The leucocytes fractions are known to either increase or decrease at different health conditions, depicting the dynamics of the immune system to respond to variable health challenges at various times in the life of the individual, following sundry exposures. The leucocytes fractions consist of the non-granular lymphocytes, the granulocytes (composed of polymorphonuclear neutrophilic granulocytes, eosinophilic granulocytes and basophilic granulocytes) and the monocytes. In any case, it is noteworthy that all forms of blood cells in the haematopoietic system primarily originated as marrow precursors in the bone marrow, taking their root from a single pluripotent stem cell, which has the potential to develop into any particular cell line (erythrocytes, leucocytes and thrombocyte or platelets) depending on the need of the body at a particular point in time [52,54,55]. The lymphocytes on their part, according to experts [51,53,54], are usually markedly elevated in conditions, such as:

-Acute infections: infectious mononucleosis, infectious lymphocytosis, mumps, rubella, pertussis, and various viral infections.

-Chronic infections: tuberculosis, syphilis, brucellosis, and infectious hepatitis. -Chronic lymphocytic leukaemia.

AGE GROUP	PARAMETER	NON-OILWORKERS Mean ± SD	OIL WORKERS Mean ± SD	P-Value
30-39 (Non-oil workers, n=5), (Oil workers, n=97)	AGE	36.60 ± 1.52	35.55±2.69	0.651
	SERVICE PERIOD	10.00 ± 4.18	11.46 ± 4.34*	0.000
	ESR	6.20 ± 4.02	11.84 ± 16.04*	0.031
	Hb	10.96 ± 1.48	13.24 ± 1.31	0.405
	PCV	35.80 ± 4.38	43.64 ± 4.47	0.287
	PLATELET×109	224.20 ± 75.32	236.16 ± 64.46	0.814
	WBC-T ×109	6.52 ± 1.36	7.05 ± 8.11	0.818

	LYMPHOCYTE	41.00 ± 12.43	52.88 ± 8.08*	0.000
	GRANULOCYTE	59.00 ± 12.43	47.12 ± 8.08*	0.000
40-49 (Non-oil workers, n=13), (Oil workers, n=142)	AGE	45.46 ± 2.60	44.27 ± 2.71	
	SERVICE PERIOD	19.62 ± 7.30	16.82 ± 5.34*	0.005
	ESR	5.08 ± 6.96	9.99 ± 8.48	0.146
	Hb	13.67 ± 0.97	13.11 ± 1.23	0.705
	PCV	44.85 ± 3.18	43.13 ± 4.05	0.613
	PLATELET×109	251.85 ± 43.41	235.28 ± 62.84	0.752
	WBC-T ×109	7.59 ± 1.93	7.16 ± 6.93	0.878
	LYMPHOCYTE	40.00 ± 11.28	53.33 ± 9.63*	0.000
	GRANULOCYTE	60.00 ± 11.28	46.65 ± 9.61*	0.000
50-59 (Non-oil workers, n=13), (Oil workers, n=64)	AGE	53.92 ± 2.78	53.23 ± 2.61	
	SERVICE PERIOD	24.67 ± 8.29	21.97±5.64	0.106
	ESR	8.42 ± 9.77	11.83 ± 11.07*	0.040
	Hb	13.15 ± 1.40	13.26 ± 1.46	0.405
	PCV	43.25 ± 4.56	43.17 ± 3.48	0.610
	PLATELET×109	231.83 ± 64.71	216.83 ± 56.92	0.110
	WBC-T ×109	7.46 ± 1.39	6.86 ± 2.13	0.734
	LYMPHOCYTE	43.58 ± 8.33	48.97 ± 9.55*	0.000
	GRANULOCYTE	56.42 ± 8.33	51.03 ± 9.55*	0.000

Multiple comparisons using Least Significant Difference (LSD) was the statistical method of analysis applied. *indicates statistically significant difference.

Table 4: Value ranges and means of haematological health parameters for the various age groups of the oil workers and non-oil workers.

On the other hand, since the value for the lymphocytes is always a differential of the total leucocyte count, an increase in lymphocytes will surely reflect a decrease in the granulocytes differential component or fraction. And this was clearly the pattern observed with respect to these parameters in the result of oil vs non-oil workers. There was a depression of the granulocytes among the oil workers (Table 1). Decreased levels of granulocytes (Granulocytopenia) (inclusive of Neutropenia) are caused by a variety of conditions such as:

-Infections: influenza, malaria, kala-zar, milliary tuberculosis.

-Diffuse marrow disease- particularly if marrow is aplastic, or if there is an extensive infiltration with malignant cells or leukaemia. Neutropenia in this case occurs as part of the general depression of the whole bone marrow.

-In megaloblastic anaemia where neutropenia is a common finding, and in Addisonian pernicious anaemia.

-In hypersplenism: red blood cells, white blood cells and platelets are generally sequestrated in the spleen leading to depression of the three cell types- a condition generally termed "pancytopenia"

-Miscellaneous causes include disseminated systemic lupus erythematosus (SLE), anaphylactoid shock, myxoedema, iron deficiency anaemia, and idiopathic and cyclic neutropenia.

-Drugs/Toxins: Here, neutropenia occurs due to direct toxic action, which has direct depressive effect on the marrow. Apart from various drugs employed for therapeutic purposes particularly cytotoxic ones, many other natural and environmental chemicals/agents have been listed as inducers of neutropenia [52,54,55]. These include benzene, benzene hexachloride, Carbon tetrachloride, DDT, dinitrophenol, glue, hair dyes, industrial solvents, quinidine, sulphamethoxypyridozine, thiosemicarbazole (Neo-mercazole), propylthiouracil, and potassium perchlorate.

-Metals- including Gold, Organic Arsenicals.

-Physical agents like Ionizing radiation.

Indeed, most of the conditions listed above are obtainable given the very nature, requirements, production processes and products of oil refining and distribution industry. The workers of this industry have more than adequate exposures to myriad of chemical, physical, biological and ergonomical hazards [4,42] that furnish them with most of the listed agents.

Exposure Period	N	Parameter	Range	Mean ± SD	P-Value	Normal range and unit of parameter
1-5	23	ESR	1-74.00	12.70 ± 16.85	0.058	0 mm/hr-7 mm/hr (M), 0 mm/hr-10 mm/hr (F)
6-10	14		2-22.00	8.64 ± 6.63	0.586	
11-15	128		1-100.00	10.86 ± 13.30	0.071	
16-20	56		2-38.00	10.77 ± 8.79	0.113	
21-25	61		2-53.00	10.54 ± 8.87	0.113	
26-35	21		2-62.00	12.71 ± 13.61	0.128	

CONTROL	30		1-36.00	6.60 ± 7.81		
Total	333		1-100.00	10.55 ± 11.57		
1-5	23	Hb	10-15.80	13.54 ± 1.60	0.149	13 g/dL-18 g/dL (M), 12 g/dL-15 g/dL (F)
6-10	14		11.6-14.60	13.52 ± 0.83	0.236	
11-15	128		7.6-16.80	13.09 ± 1.32	0.763	
16-20	56		10.6-16.70	13.07 ± 1.14	0.843	
21-25	61		10.6-16.40	13.14 ± 1.14	0.659	
26-35	21		11.9-21.10	13.61 ± 1.91	0.114	
CONTROL	30		9.1-14.90	13.01 ± 1.54		
Total	333		7.6-21.10	13.17 ± 1.33		
1-5	23	PCV	33-57.00	44.78 ± 5.72	0.073	40%-50% (M), 36%-47%(F)
6-10	14		38-48.00	44.36 ± 2.76	0.221	
11-15	128		25-58.00	43.05 ± 4.36	0.683	
16-20	56		35-55.00	43.04 ± 3.68	0.723	
21-25	61		35-54.00	43.28 ± 3.69	0.535	
26-35	21		39-49.00	43.38 ± 2.82	0.567	
CONTROL	30		30-49.00	42.70 ± 5.01		
Total	333		25-58.00	43.25 ± 4.17		
1-5	23	PLATELET ×109	153-430.00	250.13 ± 77.83	0.530	150-400 ×109 /L
6-10	14		108-320.00	217.93 ± 51.56	0.293	
11-15	128		75-423.00	239.71 ± 62.81	0.970	
16-20	56		115-420.00	224.57 ± 65.50	0.301	
21-25	61		114-370.00	225.92 ± 60.34	0.340	
26-35	21		133-318.00	217.86 ± 52.31	0.230	
CONTROL	30		141-382.00	239.23 ± 57.30		
Total	333		75-430.00	233.02 ± 62.62		
1-5	23	WBC ×109	3.5-10.50	7.17 ± 1.43	0.916	4-11 ×109 /L
6-10	14		3.8-9.10	6.51 ± 1.60	0.680	
11-15	128		3.2-86.00	7.36 ± 9.89	1.000	
16-20	56		3.2-17.20	7.00 ± 2.51	.804	
21-25	61		3.2-15.70	6.84 ± 2.14	0.713	
26-35	21		3.6-10.40	6.47 ± 1.69	0.625	
CONTROL	30		4.9-11.00	7.36 ± 1.64		
Total	333		3.2-86.00	7.10 ± 6.32		
1-5	23	LYMPHOCYTE	33-63.00	53.96 ± 8.22*	0.000	
6-10	14		45-68.00	55.71 ± 6.24*	0.000	

11-15	128		30-72.00	52.59 ± 8.39*	0.000	
16-20	56		18-69.00	51.45 ± 11.21*	0.000	
21-25	61		27-75.00	52.18 ± 9.72*	0.000	
26-35	21		28-73.00	48.71 ± 9.37*	0.008	
CONTROL	30		25-57.00	41.60 ± 10.16		
Total	333		18-75.00	51.32 ± 9.81		
1-5	23	GRANULOCYTE	37-67.00	46.04 ± 8.22*	0.000	
6-10	14		32-55.00	44.29 ± 6.24*	0.000	
11-15	128		28-70.00	47.39 ± 8.36*	0.000	
16-20	56		31-82.00	48.55 ± 11.21*	0.000	
21-25	61		25-73.00	47.82 ± 9.72*	0.000	
26-35	21		27-72.00	51.29 ± 9.37*	0.008	
CONTROL	30		43-75.00	58.40 ± 10.16		
Total	333		25-82.00	48.68 ± 9.80		
Multiple comparisons using Least Significant Difference (LSD) was the statistical method of analysis applied. *Indicates statistically significant difference.						

Table 5: Value ranges and means of haematological parameters for the various exposure periods of the oil workers and the control subjects.

A careful consideration of all these potential causes of variations in the blood cell lines, as it concerned the oil workers revealed that chemical aetiology than other clinical reasons appeared most highly favoured, since as at the time of this study all the participants were apparently clinically healthy, and the only denominator common to all of them was occupational circumstance (i.e., being commonly exposed to factors in their oil and gas work environment), and this may therefore be responsible for the observed variations, since the workers cum participants with clinical conditions that could likely act as confounders were excluded from the study abinitio. Although our failure to adjust for other possible confounders such as, for instance, their smoking habit, could be considered a major weakness of this study, the possible effect (s) of these on our study results are deemed to cancel out, as such failure apply equally in both the oil work population and their control peers.

Variations in different blood cell lines following exposure to petroleum products had been reported previously. Using animal models, Orisakwe et al. [36] reported significantly decreased values in the packed cell volume (PCV) and total white blood cell (WBC) in a group of rats after 7days of treatment with 200 mg/kg Bonny Light crude oil compared to the respective control and before-treatment groups. Chu et al. [56] in an earlier 14-day study reported that light gas oil caused a decrease in Hb, PCV, and RBC, with bone marrow myeloid hyperplasia and dyserythropoiesis. Except the lymphocytes and granulocytes that showed significant variations in our study on the oil work cohorts (human subjects), the variations recorded in the values for Hb, PCV, WBC and Platelets were not significant when compared with the non-exposed population. However, unlike these animal studies where the observed effects were produced from specific petroleum product(s) directly and invasively/perenterally introduced into the system of the laboratory animal, our study are targeted at the effects likely to have been produced from general possible exposures that could have occurred through respiratory, dermal contact or sundry other exposure routes. Olusi [53] had pointed out that most petroleum related chemicals can cause deleterious effects after long exposure, and the latency period varies from ten to twenty-five years.

Yamato et al. [57] also reported blood disorders as one of the most frequently observed effects of long-term petroleum pollution on individual organisms. Petrol (gasoline) is believed to be unique among petroleum products in being capable of destroying the blood forming elements in the body [58]. Most of the effects attributable to petroleum products have been linked directly or indirectly to some of the primary constituents of petroleum products including particularly benzene, toluene, ethyl benzene, and xylene- the components often referred to as the BTEX complex [59,60,10-16]. Of these, benzene is unquestionably the most dangerous hydrocarbon used in industry, and some petroleum solvents present a major long-term hazard for man. Regarding these, though various studies investigating link between benzene exposures and various forms of leukaemias in various occupational settings and group produced variable results, most results revealed that high benzene exposure causes acute myeloid leukaemia (AML) Three petroleum case-control studies identified 60 cases (241 matched controls) for AML and 80 cases (345 matched controls) for chronic lymphoid leukaemia (CLL) [61]. However, the study of Rushton et al., [62] reported a much lower benzene exposures than previous studies. The study also does not persuasively demonstrate a risk between benzene and AML. They concluded that a previously reported strong relationship between myelodysplastic syndrome (MDS) (potentially previously reported as AML) at their study's low benzene levels suggests that MDS may be the more relevant health risk for lower exposure. Higher CLL risks in refinery workers may be due to more diverse exposures than benzene alone. Despite remaining within the parametric reference range, the finding in our study of significant ($P<0.01$) increase in the lymphocytes fraction of the WBC

among the oil work cohorts relative to the control group suggests a tendency towards lymphocytosis and possibly lymphocytic leukaemia in the oil workers (under appropriate stimulus). Though these workers still remained clinical healthy as at the time of this study, there is need to monitor them on long term basis (even post retirement from active service) specifically for lymphocytic leukaemic disease, given the long latency period required for full establishment of some of the effects of petroleum hydrocarbons, particularly benzene and other members of the BTEX complex.

Conclusion

The study revealed that though the levels of the assayed haematological markers were still within the parametric reference ranges as at the time of this study, however, relative to the non-oil work referent group, variations observed in the markers among the oil workers suggest that haematotoxic effects are part of the potential health effects of oil workers following sundry job exposures in the petroleum oil refining and distribution industry in Nigeria. The study findings thus suggest that petroleum refining and distribution work environment in Nigeria is furnished with some haematotoxic substances. Changes observed for exposure groupings and statistically significant correlations between age, exposure (service) period and most of the parameters suggest that both age and exposure period have strong impacts in defining the patterns of variations observed in the haematological indices among the oil workers. However, the inability to obtain and adjust data for the smoking habit of the study participants was a major deficiency of the study, which needs to be taken care of in any future study of these industrial workers. However, despite what might be considered as the shortcomings of the study, the results offer far reaching contributions in the area of occupational and environmental toxicology, given that most of what was known of petroleum toxicology in Nigeria were based on studies using animal models. To the best of my knowledge, this is the first study on this thematic area conducted directly on humans in Nigeria. While earlier studies have reported the effects of petroleum exposures in animal models for possible extrapolation to humans, this study was carried out using direct human biological samples. Thus, the results give a more assured situation than an extrapolated result, which might be affected by species differences.

Summary of Study Findings and Suggestions

The following are among the major highlights/summary of this study findings, which also represent major contributions in this thematic area to the scientific community:

*The study findings suggest that petroleum refining and distribution work environment is furnished with some haematotoxic substances.

*Haematopoietic toxicity is a potential health effect of long term occupational exposures in the petroleum refining and distribution industry in Nigeria, hence, long-serving staff might be victims of haematopoietic cellular pathology.

*Exposure (service) period and Age are among the variables defining the observed health effects.

*For the management of the establishments within this industrial sector, the study findings provided data-based evidence for upgrading the industrial/occupational health measures aimed at improving health of workers and eventually their productivity. In this regard, the study results indicated a need for frequent environmental and biological monitoring to ensure a safer and healthier workplace and workforce respectively in this very critical sector of the Nigerian economy.

*The study findings also offer immense benefit in the realms of forensic toxicology, since it provided scientific evidence for resolution of health compensation litigations by providing a firmer ground to sue (or not to sue) for health compensation for the retired/disengaged or estranged workers who may have already fallen victims of various organic ailments, particularly those involving occupationally-facilitated haematological diseases conditions following several years of service in this sector

*Finally, It is important to note that data on the haematological health indices of the petroleum refining and distribution industry workers in Nigeria provided by this study is indeed part of the response to the call by Loewenson [48] regarding industrially furnished exposures and their health effects, and also by Scala [47] for toxicological data on the petroleum/petrochemical industry. Data regarding other health hazards of this industry in Nigeria and their organ/system effects has been provided in our previous works [4,42-47], while unresolved aspects remains the focus of on-going studies.

*In terms of recommendation, a major deficiency of this study which is the inability to obtain and adjust data for the smoking habit of the study participants, warrants that this be taken care of in any future study of these industrial workers. It is also being recommended that the population of the referents (control group) in such study be enlarged to draw better statistical power than is seemingly the case in the present study whereby 10% of the study population was allotted to the referents (another deficiency pointed out). Again, although gender, age and exposure (Service) period were among the variables affecting the distribution of the observed effects in the oil workers, data relating to these were however variable, and therefore requires further elucidation/validation in a future study in which smoking habit of participants will be adjusted for. Finally, despite remaining within the parametric reference range, the finding in our study of significant ($P<0.01$) increase in the lymphocytes fraction of the WBC among the oil work cohorts relative to the control group suggests a tendency towards lymphocytosis and possibly lymphocytic leukaemia in the oil workers (given appropriate stimulus). Though these workers still remained clinical healthy as at the time of this study, there is still need to monitor them on long term basis (even post retirement from active service) specifically for the variants of lymphocytic leukaemic disease, given the long latency period required for full establishment of some of the effects of petroleum hydrocarbons, particularly benzene and other members of the BTEX complex.

Acknowledgements

The author is grateful to the Department of Petroleum Resources (DPR), Federal Ministry of Petroleum Resources for authorizing not only this study but also release of relevant data by the studied establishments under her supervision. The management and staff of PHRC and PPMC are hereby appreciated for permitting and participating in this study. Credit for the success of this study goes to the entire staff of the plant clinic (used by both establishments) that were at the time of this study conducting periodic medical examination for the entire staff, an exercise that greatly smoothened this study. Special interest shown in the study and the various assistance rendered by Dr. Shaibu M, an occupational physician and then head (Pant Clinic), Dr. Idris F (who latter succeeded him) and

their office secretaries, particularly miss Evangel Njoku were all very highly appreciated.

References

1. Neff JM (1985) Polycyclic aromatic hydrocarbons. In: Rand GM, Petrocelli SR (eds). Fundamentals of Aquatic Toxicology. Bristol, PA: Taylor and Francis pp. 416-454.
2. Cottica D, Grignani E (2013) [Evolution of technology and occupational exposures in petrochemical industry and in petroleum refining]. G Ital Med Lav Ergon 35: 236-243.
3. Gennaro V, Finkelstein MM, Ceppi M, Fontana V, Montanaro F, et al. (2000) Mesothelioma and lung tumors attributable to asbestos among petroleum workers. Am J Ind Med 37: 275-282.
4. Ezejiofor TIN (2014) Risk Assessment: Re-appraisals for Potential Hazards in the Operational Environment and Facilities of Petroleum Refining and Distribution Industry in Nigeria-Research and Review. Occup Med Health Aff 2: 187.
5. Runion HE (1988) Occupational exposures to potentially hazardous agents in the petroleum industry. Occup Med 3: 431-444.
6. Engler R (1975) Oil refinery health and safety hazards: their causes and the struggle to end them. Philadelphia Area Project on Occupational Safety and Health, Philadelphia, USA.
7. Gennaro V, Ceppi M, Boffetta P, Fontana V, Perrotta A (1994) Pleural mesothelioma and asbestos exposure among Italian oil refinery workers. Scand J Work Environ Health 20: 213-215.
8. Epstein PR, Selber J (2002) Oil- a life cycle analysis of its health and environmental impacts. Harvard Medical School Boston, and Massachusetts: Report of center for Health and the global Environment 73.
9. Van den Borre L, Deboosere P (2015) Enduring health effects of asbestos use in Belgian industries: a record-linked cohort study of cause-specific mortality (2001-2009). BMJ Open 5: e007384.
10. Awodele O, Sulayman AA, Akintonwa A (2014) Evaluation of haematological, hepatic and renal functions of petroleum tanker drivers in Lagos, Nigeria. Afr Health Sci 14: 178-184.
11. Anttila A, Pokhrel A, Heikkilä P, Viinanen R, Pukkala E (2015) Kidney cancer risk in oil refining in Finland: a nested case-referent study. J Occup Environ Med 57: 68-72.
12. Trasande L, Urbina EM, Khoder M, Alghamdi M, Shabaj I, et al. (2015) Polycyclic aromatic hydrocarbons, brachial artery distensibility and blood pressure among children residing near an oil refinery. Environ Res 136: 133-140
13. Trumbore DC, Osborn LV, Johnson KA, Fayerweather WE (2015) Airborne Exposures to Polycyclic Aromatic Compounds Among Workers in Asphalt Roofing Manufacturing Facilities. J Occup Environ Hyg 12: 564-576.
14. Edokpolo B, Yu QJ, Connell D (2015) Health risk assessment for exposure to benzene in petroleum refinery environments. Int J Environ Res Public Health 12: 595-610.
15. D'Andrea MA, Reddy GK (2014) Hematological and hepatic alterations in nonsmoking residents exposed to benzene following a flaring incident at the British petroleum plant in Texas City. Environ Health 14: 115.
16. Meo SA, Alrashed AH, Almana AA, Altheiban YI, Aldosari MS, et al. (2015) Lung function and fractional exhaled nitric oxide among petroleum refinery workers. J Occup Med Toxicol 10: 37.
17. Kponee KZ, Chiger A, Kakulu II, Vorhees D, et al. (2015) Petroleum contaminated water and health symptoms: a cross-sectional pilot study in a rural Nigerian community. Environ Health 14: 86.
18. Lindén O, Pålsson J (2013) Oil contamination in Ogoniland, Niger Delta. Ambio 42: 685-701.
19. Doherty VF, Otitoloju AA (2013) Monitoring of soil and groundwater contamination following a pipeline explosion and petroleum product spillage in Ijegun, Lagos Nigeria. Environ Monit Assess 185: 4159-4170.
20. Adedara IA, Ebokaiwe AP, Mathur PP, Farombi EO (2014) Nigerian bonny light crude oil induces endocrine disruption in male rats. Drug Chem Toxicol 37: 198-203.
21. Ebokaiwe AP, D'Cruz CS, Jubendradass R, Amala Rani JS, Mathur PP, et al. (2015) Nigerian bonny-light crude oil induces alteration in testicular stress response proteins and caspase-3 dependent apoptosis in albino wistar rats. Environ Toxicol 30: 242-252.
22. PHRC Brochure (2008) PHRC. Nigeria: Premier Petroleum Refining Company 20.
23. International Agency for research on cancer (IARC) (1989) Occupational exposures in petroleum refining; crude oil and major petroleum fuels. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. IARC Monogr Eval Carcinog Risks Hum 45: 1-322.
24. Hayes RB, Yin SN, Dosemeci M, Li GL, Wacholder S, Chow WH (1996) Mortality among benzene-exposed workers in China. Environ Health Perspect 104: 1349-1352.
25. Health Effects Institute (HEI) (1995) Particulate air pollution and daily mortality: replication and validation of selected studies. Boston, M A: The phase I report of the particle epidemiology evaluation Project
26. Pukkala E, Auvinen A, Wahlberg G (1995) Incidence of cancer among Finnish airline cabin attendants, 1967-92. BMJ 311: 649-652.
27. Dement JM, Hensley L, Kieding S, Lipscomb H (1998) Proportionate mortality among union members employed at three Texas refineries. Am J Ind Med 33: 327-340.
28. Clapp RW, Coogan PF (1999) Leukemia in petroleum refinery workers: a review of recent studies. New Solut 9: 375-387.
29. National Institute of Environmental Health Science (NIEHS) (2000) 9th Annual report of suspected human carcinogens; NIEHS, Washington DC.
30. Sebastian San M, Armstrong B, Cordoba JA, Stephens C (2001) Exposures and cancer incidence near oil fields in Amazon basin of Ecuador. Occup Environ Med 58: 517-522.
31. Satin KP, Bailey WJ, Newton KL, Ross AY, Wong O (2002). Updated epidemiological study of workers at two California petroleum refineries, 1950-1995. Occup Environ Med 59: 248-256.
32. Tsai SP, Wendt JK, Cardarelli KM, Fraser AE (2003) A mortality and morbidity study of refinery and petrochemical employees in Louisiana. Occup Environ Med 60: 627-633.
33. Parodi S, Vercelli M, Stella A, Stagnaro E, Valerio F (2003) Lymphohaematopoietic system cancer incidence in an urban area near a coke oven plant: an ecological investigation. Occup Environ Med 60: 187-194.
34. Parodi S, Montanaro F, Ceppi M, Gennaro V (2003) Mortality of petroleum refinery workers (Letters). Occup Environ Med 60: 304-307.
35. Orisakwe OE, Njan AA, Afonne OJ, Akumka DD, Orish VN, et al. (2004) Investigation into the nephrotoxicity of Nigeria bonny light crude Oil in albino rats. Int J Environ Res Public Health 1: 106-110.
36. Orisakwe OE, Akumka DD, Njan AA, Afonne OJ, Okechi OO (2005) Hepatotoxic and haematological effects of Nigerian bonny light crude oil in male albino rats. Toxicol Environ Chem 87: 215-221.
37. Igbo NM, Dede EB, Ayalogu OE (2001) Acute toxicity effects of crude petroleum (bonny light), kerosene, and gasoline in albino rats. J Appl Sci Environ Mgt 5: 73-75.
38. Dede EB, Kagbo HD (2001) Investigation of acute toxicological effects of diesels fuel in rats (Rattus Rattus). J Appl Sci Environ Mgt 5: 83-84.
39. Gabriel UU, Allison ME, Alagoa KJ (2001) Effects of crude oil water dispersion on the haemoglobin and haematocrit of the African catfish, clarias gariepinus. J Appl Sci Environ Mgt 5: 9-11.
40. Ayalogu OE, Igbo NM, Dede EB (2001) Biochemical changes in the plasma and liver of albino rats exposed to petroleum samples (gasoline, kerosene, and crude petroleum). J Appl Sci Environ Mgt 5: 97-100.
41. Uba EC, Onyekonwu MO, Mbeledogu IU (2001) Modeling environmental contamination: case study of a potentially polluted area in the Niger delta. J Appl Sci Environ Mgt 5: 77-84.

42. Ezejiofor TIN, Iwuala MOE, Osuala FOU, Nwigwe HC (2012) Risk assessment: Prevalent occupational hazards in Nigerian petroleum oil refining and distribution industry. J Med Invest Pract 8: 24-29.
43. Ezejiofor TIN, Ezejiofor AN, Iwuala MOE (2013) Anthropometrical and Biochemical Markers of Cardiovascular Health Risks among Petroleum Oil Refining and distribution Industry Workers, Nigeria. J Med Lab Sci 22: 21-32.
44. Ezejiofor TIN, Ezejiofor AN, Iwuala MOE (2013) Toxic Nephropathy: A Potential Health Effect of Occupational Exposures in Petroleum Oil Refining and Distribution Industry Workers in Nigeria. The J Toxicol Health. Photon 103: 330-344.
45. Ezejiofor TIN, Ezejiofor AN, Orisakwe OE, Nwigwe HC, Osuala FOU, et al. (2014) Anicteric hepatoxicity: A potential health risk of occupational exposures in Nigerian petroleum Oil refining and distribution industry. J Occup Med Toxicol 9: 3.
46. Ezejiofor TIN (2010) Patterns of accidents and injuries in Nigerian petroleum Oil refining and distribution industry. Mary Slessor J Med 10: 23-28.
47. Ezejiofor TIN (2010a) Morbidity and mortality patterns in Nigerian petroleum Oil refining and distribution industry. Mary Slessor J Med 10: 8-22.
48. Loewenson R (1995) Occupational health in small-scale industries in Africa. Afri Newsl on Occup Health and Safety 13: 44-45.
49. Scala RA (1988) Motor Gasoline Toxicity. Toxicol Sci 10: 553-562.
50. Westergreen A (1921) Studies of suspension stability of the blood in pulmonary tuberculosis. Acta Medica Scand 54: 247-282.
51. Wardlaw SC, Levine RA (1983) Quantitative Buffy Coat (QBC) analysis-a new laboratory tool functioning as screening for complete blood cell count. JAMA 249: 617-620.
52. Thompson RB, Proctor SJ. A Short Textbook of Haematology, (6thedn) (ELBS). Pitman Publishing Ltd, London, pp. 518.
53. Olusi O (1981) Human health hazards associated with Petroleum related pollution. Proceedings of the 1981 seminar on Petroleum industry and the Nigerian Environment Lagos, pp. 195-200.
54. Ludlam CA (Ed) (1992) Clinical Haematology, ELBS edition, Longman Singapore Publishers Pte Ltd, Singapore, pp. 478.
55. Hoffbrand AV, Pettit JE (1993) Essential Haematology. (3rd.edn), Blackwell Science, USA, pp. 437.
56. Chu I, Villeneuve DC, Secours V, Otson R, Valli VE (1991) Short-term dermal toxicity and mutagenicity of Coal processing products in rat. J Toxicol Environ 33: 317-326.
57. Yamato O, Goto I, Maede YJ (1996) Hepatotoxic and Haematological effects of Nigerian Bonny light crude oil on rats. J Wild Dis 32: 381-384.
58. Bramley J, Turner L (1986) Petroleum hydrocarbon solvents In: Hobson GD (ed) Modern Petroleum Technology II, John Wiley and Sons, New York, pp. 933-962.
59. Sherertz PC (1998) Petroleum Products: Report of the Virginia Department of Health, Division of Health Hazards Control.
60. Henry JA (1998) Composition and toxicity of petroleum products and their additives. Human & Experimental Toxicol 17: 111-123.
61. Brain BJ (1999). The relationship between the Myelodysplastic/ myeloproliferative syndromes and the Leuk Lymphoma 34: 443-449.
62. Rushton L, Schnatter AR, Tang G, Glass DC (2014). Acute myeloid and chronic lymphoid leukaemias and exposure to low-level benzene among petroleum workers. Br J Cancer 110: 783-787.