ASSESSMENT OF GENOTOXICITY, OXIDATIVE STRESS AND HAEMATO – INFLAMMATORY MARKERS AMONG PETROLEUM PRODUCTS EXPOSED WORKERS AT NNPC LIMITED FACILITIES AT ABUJA AND ENVIRONS

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BENIN CITY.

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A THESIS WRITTEN IN THE
DEPARTMENT OF MEDICAL LABORATORY SCIENCE
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AND

SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES, UNIVERSITY OF BENIN, BENIN CITY IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF DOCTOR OF PHILOSOPHY (PhD) IN MEDICAL LABORATORY SCIENCE (CHEMICAL PATHOLOGY) OF THE UNIVERSITY OF BENIN, BENIN CITY, NIGERIA.

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CERTIFICATION

This is to certify that this thesis was carried out by **BADEJO, DAVID ADEDOTUN** with matriculation number **PG/BMS2015220** under our supervision, in partial fulfilment of the requirement for the award of Doctor of Philosophy (Ph. D) in Chemical Pathology, Department of Medical Laboratory Science.

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EXTERNAL EXAMINER	**DATE**

DEDICATION

This thesis is dedicated to the Almighty God and to the memory of my dear late parents Mr. Slyvester Bisola (JP) and Mrs. Temitayo Adenike Badejo without them, I will be nowhere today. They are my inspiration and role model. May their gentle souls continue to rest in the bosssom of the Almighty (Amen).

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ABSTRACT

The major components of petroleum are hydrocarbons which are toxic and have been implicated in a number of human diseases. The aim of this study was to assess the genotoxicity, oxidative stress and haemato – inflammatory markers among petroleum products exposed workers at NNPC Limited facilities at Abuja and environs. A total of two hundred and fifty adult males participated in this study. this research was a cross sectional study; a multivariable questionnaire was designed to provide answers to some questions. The questionnaire was divided into different sections comprises of social demographic variables such as age, sex, marital status, alcohol consumption, smoking etc. Others were awareness of hazards caused by occupational exposure to petroleum products and awareness of various ways of protection against the petroleum products with the use of personal protective equipment (PPE). Ethical approvals were obtained from Ministry of Environment, Abuja and Ethic committee of NNPC Limited. Under aseptic conditions, ten millimeter (10mls) of venous blood sample was obtained from each participating individuals at the end of the work shift on the day of exposure. The samples were processed according to each parameter requirement. Parameters such benzene and its derivatives (phenol, styrene, butanoic acid, benzene, benzene chloro, o – xylene, toluene, benze 1, 3, dimethyl, p – xylene, naphthalene and ethylbenzene), oxidative stress markers (CRP, total oxidative capacity, glutathione reductase), inflammatory and immunological markers (IL1, IL3, IL4, IL6, IL9, IL10, IFN gamma, Human LT beta, IgG and IgM), haematological parameters and deoxyribonucleic acid damage marker (8 hydroxyl 2 deoxylguanosine) were analysed using standard methods according to the manufacturer's instructions. Results showed significantly higher phenol in tanker drivers compared with petrol attendants, auto mechanics and NNPC staff. The mean serum levels of oxidative stress markers indicated significantly higher total oxidant capacity among the tanker drivers compared with the petrol attendants, auto mechanics and NNPC staff. The NNPC staff indicated statistically higher mean glutathione peroxidase compared with the petrol attendants, auto mechanics, and tanker drivers. The auto mechanics indicated higher mean C reactive protein compared with tanker drivers and NNPC staff. Results also indicated significantly higher interleukin-1 beta in auto mechanics compared with petrol attendants, and NNPC staff. The petrol attendants and the tanker drivers also indicated significantly ($p < 0.05$) higher interleukin-1 Beta compared with the NNPC staff. Tanker drivers indicated higher mean IgG level compared with the petrol attendants ($p = 0.013$), auto mechanics ($p = 0.019$) and NNPC staff ($p = 0.016$). The NNPC staff presented higher mean IgM compared with petrol attendants ($p = 0.001$) and auto mechanics ($p = 0.046$). Data further indicated a significantly ($p < 0.001$) lower mean WBC among auto mechanics compared with the petrol attendants, tanker drivers and NNPC staff respectively. The NNPC staff indicated significantly higher RBC compared with the auto mechanics and tanker drivers. The petrol attendants indicated lower mean haemoglobin (Hb) concentrations compared with the auto mechanics, tanker drivers and NNPC staff. The petrol attendants also showed significantly lower mean (haematocrit) Hct compared with the auto mechanics, tanker drivers and NNPC staff. Data also revealed that the petrol attendants had significantly higher mean 8-deoxylhydroxylguanosine compared with the auto mechanics, tanker drivers and NNPC staff. In this study the level of benzene and its derivatives, oxidative stress markers, inflammatory and immunology markers as well DNA damage markers are significantly higher compared to haematological markers among the participants. However, more works need to be done in educating the populace on the effects of petroleum products on their health.

CHAPTER ONE

INTRODUCTION

1.1 Background to the Study

Total Petroleum Hydrocarbons (TPH) is a term used to describe a broad family of several hundred chemical compounds called hydrocarbons that originally come from crude oil. Crude oil varies in how much of each chemical they contain, and so the petroleum products that are made from crude oil (Eneh, 2011). Even though exposure to supra lethal concentration of petrol vapor is rare but possible in highly confined or poor ventilation areas (Kanaly and Haryama, 2000). They are more prone to respiratory tract ailments due to the interruption by the particulate matter (PM) and fuel constituents (Begum and Rathna, 2012). The intentional inhalation of vapor has been extensively documented resulting irritation of eyes, respiratory tract and skin at low doses (Cairney *et al.,* 2002). Concentrations of vapor may affect the central nervous system (CNS) resulting in staggered gait, slurred speech and confusion and very high concentrations may result in rapid unconsciousness and death due to respiratory failure (Chilcott, 2006). Chronic exposure leads to chronic inflammation of respiratory tract and lung parenchyma (Sumathi and Neclambikai, 2016).

The inflammatory response was also studied in mice, in which inhalation of 300 μm^3 of PM 2.5 caused increase in TNF-β, interferon- β, IL-6 and transforming growth factor- β resulting in ROS synthesis (Shukla *et al.*, 2000). Benzene occupies the major composition of petroleum constituents and is a class I human carcinogen (Sumathi and Neelambikai, 2016). Activation of benzene and its reactive metabolites leads to cont

inuous production of reactive oxygen species (ROS), which leads to lipid peroxidation and damages DNA, RNA, leading to genetic modification and alterations in the functions of important enzymes and proteins (Mohammed *et al*., 2020). Evaporation of petrol is common during its handling, distribution and storage which releases benzene along with vehicle exhaust (Kumar and Tyagi, 2006). Unbalanced pro-oxidants and antioxidants cause oxidative stress. This imbalance causes cellular damage from reactive oxygen species buildup or antioxidant depletion (Biben, 2012).

The maintenance of a balanced state in animals between the production and neutralisation of reactive oxygen species (ROS). Inhalation of petrol fumes often leads to inflammation of the alveolar surfaces. However, the presence of antioxidants like glutathione (GSH), uric acid, ascorbate, and α-tocopherol in the epithelial lining fluid (ELF) can help protect the airways from damage caused by being subjected to air pollutants (Domej *et al.,* 2014).

There is currently, less documentation on the effect of hydrocarbon contact on humans. The scope of the linked investigations was restricted to either fly or mouse models, despite a few researchers attempting to broaden it. However, the sample size remained rather small, preventing the attainment of a definitive conclusion.

1.2 Statement of Problem

The emergence of several diseases, including cancer, respiratory ailments, and metal poisoning, has been linked to the progressive excretion or direct inhalation of petroleum products, depending on their amounts, in animal models. Scanty works on the effects of petroleum products have been documented in human, unlike in animal model. There is need to assess these effects in human to proffer solution to the high rising cases of cancers and other respiratory diseases ravaging people constantly being exposed to these

petroleum products. This research therefore would at end be able to address many problems associated with the effects of the petroleum products on the exposed worker in petroleum installations.

1.3 Justification of Study

Chronic exposure to petroleum products has caused lots of havoc to human including loss productive time which indirectly affecting nation economy. Heavy metals can damage cell, this can result to DNA damage and subsequently results in gene mutation and development of cancer. Infertility, skin and eye irritation as well as respiratory failure and death have all traced to the effect of petroleum products in human. All these have prompted the need for this research, to assess the health implications of effects of exposure to petroleum products on NNPC Plc workers at various facilities at Abuja and its environs.

1.4 The Study Aim

The study was aimed at assessing the genetic damage, oxidative stress, and indicators of inflammation in workers who were exposed to petroleum products at NNPC Limited sites in Abuja and its neighbouring regions.

1.5 Specific Objectives

1. To determine the serum concentrations of benzene and its derivatives (phenol, styrene, butanoic acid, benzene, benzene chloro, o – xylene, toluene, benze 1, 3, dimethyl, p – xylene, naphthalene and ethylbenzene) in the blood samples of participants who have been exposed to petroleum products at several NNPC Plc facilities in Abuja and the surrounding areas.

2. To determine the oxidative stress markers (CRP, total oxidative capacity, glutathione peroxidase) among the participants who were exposed to petroleum products at various NNPC Plc workers at Abuja and its environs.
3. To evaluate some inflammatory and other immunological markers (IL1, IL3, IL4, IL6, IL9, IL10, IFN gamma, LT beta, IgG and IgM) among the participants who were exposed to petroleum products at various NNPC Limited oil facilities within Abuja and environs.
4. To determine the haematological parameters of the participants who were exposed to petroleum products at various NNPC Plc workers at various facilities at Abuja and its environs.
5. To assess for deoxyribonucleic acid damage (8 hydroxyl 2 deoxylguanosine) among participants who were exposed to petroleum products at various NNPC Plc workers at various facilities at Abuja and its environs.

1.6 Research Questions

1. Are serum concentrations of benzene and its derivatives (phenol, styrene, butanoic acid, benzene, benzene chloro, o – xylene, toluene, benze 1, 3, dimethyl, p – xylene, naphthalene and ethylbenzene) higher among the participants who are exposed to petroleum products at various NNPC Plc facilities within Abuja and environs?
2. Can exposure to petroleum products raise the concentrations of oxidative stress markers (CRP, total oxidative capacity, glutathione peroxidase) among the participants who are exposed to the petroleum products at various NNPC Plc facilities within Abuja and environs?

3. Can exposure to petroleum products raise the concentrations of immunological including inflammatory markers (IL1, IL3, IL4, IL6, IL9, IL10, IFN gamma, LT beta, IgG and IgM) among the participants who were exposed to the petroleum products at various NNPC Plc facilities within Abuja and environs?
4. Can exposure to petroleum products alter the values of haematological parameters among the participants who were exposed to the petroleum products at various NNPC Plc facilities within Abuja and environs?
5. Can exposure to petroleum products damage the deoxyribonucleic acid among the participants who were exposed to the petroleum products at various NNPC Plc facilities within Abuja and environs?

Alternate Hypothesis (H_1)

There was significant increase in genotoxicity, oxidative stress and haemato – inflammatory markers among petroleum products exposed workers at NNPC Limited facilities at Abuja and environs.

CHAPTER TWO

LITERATURE REVIEW

2.1 Early History of Petroleum

According to Herodotus, the Babylonian walls and towers were constructed with natural asphalt over 4,000 years ago, a fact that was later corroborated by Diodorus Siculus.

China utilised petroleum two millennia ago. China's utilisation of crude oil was initiated during the first century BC, as indicated by the I Ching. Petroleum fuel was originally used by the Chinese in the fourth century BC (Gao, 1998).

The initial drilling of oil wells in China occurred in the year 347 AD. The bamboo-pole-mounted pieces reached depths of 800 feet (240 m) (Dalvi 2015). Salt was produced by evaporating brine using oil as a fuel source. By the 10th century, bamboo tubes were used to connect oil wells and salt springs.

The initial roadways in Baghdad were constructed using asphalt, which was made from locally sourced petroleum extracted from nearby petroleum deposits. In the middle of the ninth century, oil sources in the vicinity of present-day Baku, Azerbaijan were exploited. These oil fields were initially discovered by Arab geographers Abu al-Hasan, also known as 'Alī al-Mas'ūdī in the middle of the ninth century and later documented by Marco Polo in the middle of the 13th century. Polo noted that the wells produced a significant amount of oil, equivalent to hundreds of shiplArab and Persian chemists utilised distillation techniques to extract crude oil and create combustible substances for military applications. Distillation was introduced to Western Europe by the 12th century through Islamic Spain (Joseph and Gordon 2008). Since the 13th century, the presence of păcură has also been documented in Romania.

Petroleum was first mentioned in the Americas in Sir Walter Raleigh's 1595 Trinidad Pitch Lake narrative. Adventures into North America, published in 1753, by Swedish scientist and Carl Linnaeus student Peter Kalm, born in Finland, described Pennsylvania's petroleum springs.

King Pierre Ancillon de la Sablonnière, appointed by Louis XV, oversaw oil sands extraction in Merkwiller-Pechelbronn, Alsace, in 1745. New Times reported this in 1880. German Pechelbronn oil field, which functioned until 1970, helped found Antar and Schlumberger. The very first modern refinery was established there approximately 1857 (History from Pechelbronn Oil, 2009).

2.1.1 Modern history

2.1.1.1 Coal oil

Oil was refined in the 19th century to produce paraffin. The new oils were effective, but coal mine-extracted oil became scarce by 1851. Young believed the oil flowing from the coal mine's sandstone roof was formed by heat exchange between it and the coal seam. He thought it could be synthesised.

Following this notion, he conducted many tests and succeeded in distilling cannel coal at low temperatures. Thus, a petroleum-like fluid was produced. Whenever this fluid was treated in a way resembling the processing of seep oil, it produced similar results. Young ascertained that through a gradual process of distillation, he could extract multiple valuable liquids from it. One of these liquids, referred to as "paraffine oil" by the researcher, underwent solidification and transformed into a substance resembling paraffin wax when subjected to low temperatures (Russel, 2003).

On October 17, 1850, a patent was awarded for coal oil and paraffin wax extraction. In 1850, Younger & Meldrum with Edward William Binney formed a Bathgate, West Lothian partnership. This business was named E.W. Binney & Co. In addition, they founded E. Meldrum & Co. in Glasgow. Bathgate became the world's first oil refinery and commercial oil-works in 1851. Naphtha and lubricating oils were made from locally produced torbanite, shale rock, and bituminous coal. Paraffin was commercially available either a fuel and solid in 1856.

2.1.1.2 Kerosene

A process for improving a liquid fuel made from carbon dioxide, bitumen, which and oil shale was devised by Canadian geologist Abraham Pineo Gesner. Relative to substitutes like whale oil, the recently identified substance known as kerosene demonstrated better combustion properties, resulting in lower emissions and costs. Street lighting installations in Halifax and other towns started in 1850 when Gesner established the Kerosene Gaslight Corporation. He established the northern branch of Kerosene is Gas Lighting Company around Long Island, New York, after moving the organisation across the United States in 1854. Demand growth was a significant barrier to his company's capacity to produce, but petroleum's discovery provided a solution to the supply problem by enabling the production of paraffin more efficiently.

A more convenient way to purify kerosene from seepages of "rock oil" was discovered by Ignacy Łukasiewicz in 1852, which enhanced Gesner's method. Near Krosno in middle European Galician (Poland), in the town of Bóbrka, the first rock extraction of oil mine was established in 1854 (The offshore Technology 2019). In 1861, Meerzoeff established the inaugural sophisticated Russian oil refinery in Baku's highly developed oil fields,

following the discovery' rapid global dissemination. Over 90% of oil produced globally at the time was produced in Baku.

2.1.1.3 Oil wells

There's no clear-cut, widely-accepted consensus regarding the exact definition of the first commercial oil well. A candidate for the initially discovered oil well near La Brea, Trinidad, was dug up by the American Merrimac Company sometime about 1857. In offshore technology, 2019 the well was reached at an approximate depth of 280 feet. Vassiliou's paper in 2018 (Vassiliou et al., the second edition) provided the summary that follows. In addition to being drilled rather than excavated, Drake's well is notable because it used a steam engine, was connected to a business, and caused a significant surge. Sunk in 1857 with the American Merrimac Organisation in La Brea, southeast of Trinidad in the Caribbean, it was the world's first successfully completed oil well. At 280 feet, this well was deep.

2..1.1.4 Refineries

Simultaneously, the world's initial oil refineries were established in Jasło, Poland, with a more substantial facility inaugurated in Ploiești, Romania. Built in 1856 and inaugurated in 1857 by the brothers Teodor and Marin Mehedinţeanu, the Rafov Refinery, a refinery built at Ploiesti, had a surface area of four hectares, and the daily production reached over seven tons, obtained in cylindrical iron and iron casts that were heated by fire from wood; it was then called "the world's first systematic oil distillery," setting the record for being the world's first oil refinery, according to the Academy Of World Records(The city of Ploiesti, 2008).

Theodor Mehedinţeanu and Bucharest's municipal government Hall signed a contract in October 1856 giving this refinery the only authority to produce oil lamps for lighting the Wallachian capital. The arrangement came into effect on April 1st, 1857, when the goods supplied by the Rafov refinery were exchanged for the seized oil. As a result, Bucharest became the first city in the world to attain complete light via the application of refined petroleum.

Romania's crude oil production in 1857 reached a total of 275 tonnes.

With this figure, Romania was registered as the first country in world oil production statistics, before other large oil producing states such as the United States of America (1860), Russia (1863), Mexico (1901) or Persia (1913) (History of Romania oil industry, 2009).

2.1.1.5 United States

David Beaty made the discovery of crude oil at his residence in Warren, Pennsylvania in 1875. As a result, the Bradford oil field was established and, by the 180s, it accounted for 77 percent of the worldwide oil production. However, by the end of the 19th century, the Russian Empire, particularly the Branobel company in Azerbaijan, had taken the lead in production (Akiner, 2004).

Samuel Kier opened the first petroleum refinery in American territory in 1853. It was situated in Pittsburgh, close to Grant Street on Seventh Avenue. Apart from the undertakings in the state of West Virginia and Pennsylvania, James Miller Williams drilled an influential preliminary oil well in Oil Springs, such as those Ontario, Canada in 1858 (Turnbull Elford et al., 1982).. The discovery at Oil Springs touched off an oil

boom which brought hundreds of speculators and workers to the area. New oil fields were discovered nearby throughout the late 19th century and the area developed into a large petrochemical refining centre and exchange (May *et al*., 1998). The modern US petroleum industry is considered to have begun with Edwin Drake's drilling of a 69-foot (21 m) oil well in 1859 (John, 2008). The oil well was drilled on Petroleum Creek, in Titusville, in the state of Pennsylvania, for the benefit of a company called Seneca Oil Company. The rate at which oil was produced was 25 barrel per day (4.0 m3/d) in the beginning of the year, however by the final week of that year, it had decreased to 15 barrel per day (2.4 m3/d). The industry had substantial expansion throughout the 19th century, driven by the rising need of kerosene for oil lamps. The introduction of the automobile's engine with internal combustion in the early 20th century resulted in a notable nationwide fixation. This innovation has generated a demand which has primarily sustained the industry thus far. Subsequently, the formerly considered regional discoveries in the state of Pennsylvania and Ontario were eclipsed by the expanding demand, resulting in significant petroleum booms in Ohio, Oklahoma, Texas, and elsewhere and California.

2.1.2 20th century

Galician oilfields made Austria-Hungary the world's third largest oil producing country after United States and the Russian Empire, with a 5 percent share of the global oil production in 1908(Alison Frank 2009) By 1910, significant oil fields had been discovered in the Dutch East Indies (1885, in Sumatra), Persia (1908, in Masjed Soleiman), Peru (1863, in Zorritos District), Venezuela (1914, in Maracaibo Basin), and Mexico, and were being developed at an industrial level. Austria-Hungary lose its

primate on oil production which had been at the root of the 1910 Petroleum War (Alison, 2009) Significant oil fields were exploited in Alberta (Canada) from 1947. Offshore oil drilling at Oil Rocks (Neft Dashlari) in the Caspian Sea off Azerbaijan eventually resulted in a city built on pylons in 1949 (Timothy *et al*., 2010).

Up until the middle of the 1950s, coal was the most widely used fuel worldwide. After that, oil quickly surpassed it. The topic concerning crude oil supply levels received a lot of media attention throughout the oil-related booms of 1973 and 1979. This has intensified the worry that oil is a finite resource that will someday run out, especially in light of how economically viable it is as a source of energy.

2.1.3 History of oil Exploration in Africa (Nigeria)

A licence to explore for oil across the whole territory of Nigeria was first granted to the association. The original permit, however, lowered the area allotted for the business in 1951, and it was further reduced in 1955 and 1957. Starting with the first test well sunk in the Owerri area, drilling activities began in 1951. Though not enough to be deemed economically viable, a modest quantity of oil was discovered in 1953 near Eket at Akata (Frynas et al., 1999). Prior to the finding of Akata, the company had spent about £6 million on national exploration projects. While looking for oil that could be produced profitably, Shell-BP found petroleum at Oloibiri, Nigeria in 1956. Afam and Bomu, two major oil wells in the Ogoni region, were also discovered around this time. In all, 847,000 tonnes of crude products were shipped by 1960, when production of crude oil started. Licencing for oil exploration was made possible for non-British firms in the past few years. The following companies were granted licences: Agip in 1962, Elf in 1962, Gulf Oil and subsequently Chevron in 1961, Tenneco in 1960, and Mobil in 1955.

Similar to many other African countries, Nigeria's economy was primarily dependent on agricultural exports before to the discovery of oil. A considerable portion of Nigerians thought the developers were looking for palm oil, as reported by Walker et al. and colleagues (2009). Shell-BP was able to discover reserves of oil at Oloibiri in the Niger Delta, though, after a thorough search that lasted nearly 50 years and turned up oil in the nation. For example, the background information of the oil business (2012) states that oil production started in 1958.

Subsequently, it may have been anticipated that the Nigerian economy would undergo robust expansion. Nevertheless, the pursuit of oil revenues engendered significant fear and strife among the inhabitants of the region. A significant number of Nigerian citizens see a lack of tangible economic advantages resulting from the presence of oil corporations in their regionIt's also important to remember that the majority of the earnings from Nigeria's oil output have remained in the hands of government officials in Nigeria. As a result, the government has effectively taken control of almost all oil production, while the citizens have not been able to reap the socioeconomic advantages. Consequently, there is a demand from the citizens for oil companies to provide compensation (Andrew et al., 2009).

2.2 Production and exploration in Nigeria

As per the nation's nation analysis brief of 1997, for instance, the US government's Petroleum Information Administration (EIA) has estimated that Nigeria's recognised oil reserves range from 16 to 22 billion barrels (2.5×109 to 3.5×109 m3). But according to some reports, the real figure might be nearly as 35.3 billion barrel (5.61×109 m3). With its huge petroleum reserves, Nigeria is by far the most rich country in Africa and the

tenth wealthiest country overall. Nigeria produced 2,200,000 barrels (350,000 m3) of crude oil on average per day by the middle of 2001. But in 2019, this amount fell between 2.0881 million barrels per day to 1.99 thousand barrels per day.

The Nigerian Ministry of Petroleum Resources reports that as of right now, Nigeria is home to 1481 wells and 159 oil fields (the African nation of N delta the environment. survey, 2017). The Niger Delta's coastline region in Nigeria is the most productive area of the nation.

2.2.1 Offshore

African oil corporations are exploring offshore exploitation as an alternate producing area. Deepwater production mostly entails subsea drilling operations conducted at depths of 400 metres. The exploration of deep sea drilling broadens the potential avenues for discovering untapped oil reserves. Deep sea drilling has resulted in a 50% increase in oil extraction compared to previous methods of oil retrieval (McLennan et al., 2005).

The amount of oil extracted from Nigeria was expected to expand from 15,000 barrels per day (2,400 m^3/d) in 2003 to 1.27 million barrels per day (202,000 m^3/d) in 2010 (Mc Lennan *et al*., 2005). Deepwater drilling for oil is especially attractive to oil companies because the Nigerian government has very little share in these activities and it is more difficult for the government to regulate the offshore activities of the companies (Mc Lennan *et al*., 2005) .

Deepwater extraction facilities experience minimal disruptions caused by regional insurgent assaults, confiscations resulting from civil unrest, and acts of sabotage.Eighteen These technological improvements provide additional resources and options for

extracting petroleum from the Niger Delta, while minimising the risk of violence compared to onshore operations. A black market for illicit crude oil is in operation in the Niger Delta region, known as the Togo Triangle (Smith and Simon, 2014).

2.2.2 Natural gas

Three times larger than petroleum product reserves, natural gas reserves extend over 5,300 km3 (1871012 cu ft). Natural gas's biggest project is that of the Nigerian Liquid Natural Gas Company, administered by numerous firms and the state. In 1999, exploration and production began. Chevron is building the Escravos is Gas Utilisation project to produce 4,500,000 m3 (160106 cu ft) natural gas reserves daily, according to Online Nigeria. In addition, the West African Gas Pipeline export gas pipeline project has encountered many challenges. Petroleum would be transported to Benin, Ghana, Togo, and Cote d'Ivoire via the pipeline. Much of Nigeria's natural gas is flared. Due to flared gas, Nigeria loses 18.2 million US dollars everyday (Online Nigeria portal).

2.2.3 Downstream

Nigeria's petrol consumption rose from 2.3 MMT to 4.4 MMT between 1979 and 1989. Annual increase averages 7.5%. In 1989, petrol consumption rose 12.8%. According to Nigeria Businessinfo.com, Nigeria refines 450,000 barrels (72,000 m3) each day. During the 1990s, just 240,000 of them barrels (38,000 m3) were allocated daily. Sanni Abacha reduced refinery crude oil production to 75,000 barrel (11,900 m3) for each day (Misser et al., 2013). Four major petroleum refineries comprise the Warri The oil refinery and The petrochemical Plant, which can process 125,000 barrels that have been (19,900 m3) of petroleum products daily; the newly constructed Port Harcourt Refinery, which is capable of manufacturing 150,000 barrels (24,000 meters3) per day (the 'Old' refining

plant produces less); as well as the one that powers the inoperable Kaduna Refinery. According to NigeriaBusinessinfo.com, Port Harcourt and Warri refineries are operating at 30% capacity. Nigeria's total refining capacity would increase by greater over 1,000,000 barrels (160,000 m3) per day when the Dangote Refinery launches in the last quarter of 2022 (Eboh et al., 2022).

2.2.4 Extraction and processing

Underground, crude oil is at pressures defined by depth. Pressure keeps natural gas dissolved, therefore the substance may retain a lot. Along with oil and gas, water often enters oil wells. Each of these fluids are collected and separated using surface equipment. Purification crude oil is shipped to storage facilities at approximately atmospheric pressure in above-ground cylindrical steel tanks up to 30 metres (100 feet) round and 10 metres (33 feet) tall. Often, crude oil must be transported from far industrial facilities to treatment and refineries. Pipelines are the main overland transportation. Oil from remote wells is transferred to pipeline terminals by tank trucks. Some crude oil is transported using purpose-built railway carriages.

After distillation, crude oil fractions are processed into gasoline, diesel, heating oil, or asphalt. Distillation of five crude oils, comprising heavy Venezuelan Boscan or light Australian Bass Strait oil, yields the chart below.

The US petroleum industry measures capacity by volume and uses English units. The US measures crude oil in barrels, each carrying 42 gallons. Most countries measure capacity by quantity of materials processed in metric units. Thus, metric tonnes are used to quantify crude oil outside the US. An API 30° light oil barrel weighs about 139 kg. On

the other hand, a unit of measurement tonne of API 30° light oil is 252 imperial gallons or 7.2 U.S. barrels.

2.2.5 Tar Sand or Bituminous Sand

Tar sand, also known as bituminous sand, refers to a deposition of loose sand or partially cemented sandstone that contains a high concentration of highly viscous bitumen. Oil extracted from tar sands is often known as synthetic crude and has the potential to be a significant type of fossil fuel.

Below is a concise overview of tar sands. To obtain comprehensive coverage, please refer to the topics with substantial hydrocarbons and tar sands.

Bitumen deposits, like to other heavy hydrocarbons, are believed to represent deteriorated residues from accumulations of conventional oil, which is light to medium in nature. Degradation takes place when conventional oil moves upwards and comes into contact with lowering rainwater containing oxygen and microorganisms, at temperatures below 93° C (200° F). The water bacteria remove paraffins, while solution separates the volatile portions of crude oil.

Tar sands reserves at the Earth's surface can be extracted using open-pit technologies.

Following the excavation of tar sand, the bitumen must undergo a process of separation from the sand, followed by concentration and purification. The raw bitumen undergoes a refining process in a specialised coking unit, resulting in a mixture of lighter hydrocarbon fractions that can be used to generate synthesised crude, naphtha, kerosene, and petrol oil.

The most extensive documented reserves of bituminous sands are located in the Athabasca River valley in western Canada. The Athabasca region is now the exclusive site for commercial initiatives focused on the generation of synthetic oil from tar sands.

2.2.6 Fuel Oil

Fuel oil, or furnace oil, is manufactured from crude oil distillation byproducts. It is mostly used in power plant, marine, and industrial steam boilers. Industrial fuel oils are blended with petroleum fractions for uniformity and flash point. Sometimes paraffin has a lower flash point. Many definitions of fuel oil exclude kerosene.

2.2.7 Diesel Fuel

Diesel fuel, or diesel oil, powers diesel engines. Unlike petrol, it is made from less volatile crude oil fractions. In diesel engines, air compression in the cylinder starts combustion, not a spark. Fine sprays of fuel are injected into compressed, heated air. Due to its higher energy density, diesel engines often have better fuel efficiency than petrol engines. Due to fewer refining steps, diesel fuel has historically had cheaper retail prices than petrol (depending to region, season, taxes, and legislation). However, conventional diesel fuel produces more sulphur or solid carbon particles. Diesel may be more expensive than petrol due to additional refining and emission-control measures. In addition, diesel fuel emits more carbon dioxide per unit than petrol, reducing its efficiency advantages. Trucks and cars use "light-middle" and "middle" diesel fuel made for high-speed engines that change load and speed often. "Heavy" distillates are made over low- including medium-speed engines that withstand weights and speeds with ships, trains, and stationary engines. Centrane number, volatilization, and sulphur content are performance criteria. Automobiles and truck engines use the most volatile grades.

Conversely, low-speed engine grades produce the least volatility and leave a lot of carbon. These grades also have the most sulphur.

Sulphur in diesel fuel is heavily regulated. Diesel fuel with up to 5,000 ppm sulphur per weight was once used. New "lower sulphur" classifications with a 500-ppm limit were introduced in the 1990s. Following years saw tougher sulphur content limitations. Since 2010, US highway diesel fuel must be "ultra-low sulphur" (ULSD) with a maximum sulphur concentration of 15 parts per million. The EU has demanded "zero-sulfur" or "sulfur-free" diesel fuel for motor vehicles since 2009. Consequently, fuel used for diesel must have a minimum of ten ppm sulphur. Diesel gasoline with less sulphur releases less acid rain. Since higher sulphur levels damage emission-control systems, diesel vehicles can employ more efficient ones. In off-road vehicles, ships, boats, and stationary engines, higher-sulphur diesel fuel is allowed. Sulphur limits in heavier classes have been decreasing recently.

2.2.8 Kerosene

Fuel is often made from kerosene, commonly known simply paraffin or paraffin oil. Kerosene is a clear, pale yellow liquid with a pleasant scent.

Canadian physician Abraham Gesner discovered paraffin in the late 1840s from coal tar and shale oils. Petroleum quickly became the main source of paraffin after E.L. Drake dug the first well for oil in Pennsylvania in 1859. After electric lamps were introduced, its illumination value declined. Motor vehicles increased the importance of petrol as a petroleum product, reducing petroleum production. In many countries, paraffin is utilised for heating, cooking, and lighting.

Molecularly, kerosene is hydrocarbons. Based on its source, it usually has 10 hydrocarbons with 10–16 molecules of carbon per molecule. The major components include saturated straight-chain, branched, and naphthenes. Petrol is more volatile than kerosene. This substance's point of ignition, or melting point, is 38 °C (100 °F), at which it turns into combustible vapour at its surface. Petroleum has a flash point of 40 °C (40 °F). Storage and handling paraffin is safe due to its characteristics.

Similar to diesel fuel, petroleum serves as an intermediate distillate from crude oil with a boiling point within 150 and 300 °C (300–575 °F). Distillation or cracking can be used to obtain kerosene. Distillation physically separates kerosene from other crude oil fractions.

2.2.9 Biolith

Biolith refers to any silt that is derived form the body parts of living organisms or is created by the physiological processes of organisms. Fossilised organisms, known as bioliths, can occasionally be recognised as ancient plants or animals. In addition to petroleum-based diesel fuel, Fischer-Tropsch diesel can be manufactured from natural gas, coal, or biomass. Soybeans and oil palms are prime sources of biodiesel. These low-sulphur petroleum-based fuels can be combined with ordinary diesel fuel or used alone in diesel-powered vehicles without modifications. Diesel alternatives are often recommended to reduce petroleum use and pollutants. However, only biodiesel can reduce carbon dioxide over its lifetime.\

Petrochemical refers to chemical compounds derived from petroleum or natural gas. Petrochemicals, unlike fuels, are a variety of molecules made from petroleum and natural gas for industrial use. Nevertheless, the scope of the notion has been broadened to include all organic molecules including aliphatic, aromatic, and naphthenic, along with inorganic

substances such as ammonia, sulphur, and carbon black. A specific chemical belonging to the petrochemical category is often present in alternative sources such as coal, cola, or vegetable products.

Petrochemicals make plastics, laundry detergent and other solvents, medicines, fertilisers, insecticides, explosive compounds, synthetic fibres other rubbers, colours, resins made from epoxy, flooring, and insulation substances. Petrochemicals are present in aspirin, baggage, cars, planes, boats, polyester clothes, even CDs and tapes.

Petrochemicals, like oil and gas, are mostly carbon and hydrogen. All carbon atoms in saturated molecules are covalently linked. Multiple double bonds make molecules unsaturated. Chemical reactivity and convertibility make unsaturated molecules ideal for petrochemicals.

Feedstocks are petroleum parts used to make other compounds. Fatty acids, aromatics, synthesis gas, and inorganics are petrochemical feedstock sources. Ethylene, propylene, and butadiene are olefins. The petrochemical sector relies on ethylene. Ethyl alcohol is a type of solvent or chemical substance, which is while ethylene glycol makes polyester dyes, resins, and antifreezes. Syrene makes synthetic rubber-based polyesters, resins, and polyesters, while polyethylene makes films and plastics.

Aromatics refer to unsaturated hydrocarbon compounds that adopt a ring structure. The primary aromatic feedstocks consist of xylene, naphthalene, toluene, and benzene. The main constituent of polystyrene polymers, styrene, is obtained from benzene. Additionally, it finds application in the manufacturing of adhesives such as glues, paints, epoxy resins, and other bonding agents. Tallow is primarily utilised in the manufacturing

of explosives, as well as in the creation of petrol additives and solvents. Xylene is required for both the refining of petrol and the manufacturing of synthetic fibres and plastics. It is worth mentioning that insecticides include naphthalene.

Synthesis gas is utilised in the production of methanol and ammonia. Ammonia is mostly utilised in the production of ammonium nitrate, a type of fertiliser. A significant proportion of the manufactured methanol is utilised in the production of formaldehyde. The remaining portion is utilised for the production of silicone rubber, polymers, and polyester fibres.

The development of thermal-cracking technology in 1913 was a significant catalyst for the growth of the petrochemical industry. The approach generated gaseous by-products, initially used exclusively as fuel or lighting gas, but later found to be valuable as chemical feedstocks in the 1920s and 1930s. The sector had sustained growth following the expansion of natural gas sources and the introduction of catalytic cracking.

2.3 Overview of the Toxicological Effect of Petrol

Combining crude oil fractions with brand-specific additives creates a complex mixture of the chemical and aliphatic hydrocarbons. Petroleum composition varies depending on its origin, manufacturing process, and production batch.

Except as stated, this article only covers petrol without lead and does not discuss combustion products or chemical components like toluene, benzene, xylene, butadiene, etc. Traditional methods of determining concentration might be inaccurate and should only be used as an estimate. The technical product's temperature, brand, and batch can affect the average weight in molecules, which is used to determine parts per million (ppm).

2.3.1 Summary of Health Effects of Petrol

Petrol fumes can irritate the skin, eyes, and respiratory system in little amounts. Increased vapour exposure can cause CNS symptoms as disorientation, slurred speech, and staggered walking. Extremely high doses have the potential to cause respiratory collapse, fast unconsciousness, and death.

Prolonged dermal exposure to liquid petrol or inhalation of vapour has been associated with renal dysfunction, attributed to lipid degeneration of the proximal convoluted tubules and glomeruli (Hansbrough *et al*., 1985; Schneider *et al*., 1991), the clinical manifestations of which include haematuria, proteinuria and myoglobinuria. A late-onset autoimmune glomerulonephritus has also been described (Simpson and Cruse, 1981).

Pulmonary sequelae following inhalation of petrol vapour or secondary to pulmonary elimination of volatile hydrocarbons (following ingestion or dermal absorption) include persistent atelectasis (Hansbrough *et al*., 1985) and petachial haemorrhage. This may be associated with concomitant 'hydrocarbon hepatitis' secondary to vascular endothelial damage, possibly due to hydrocarbon-induced degeneration of fatty tissue (Simpson and Cruse, 1981; Binns *et al*., 1978; Walsh *et* al., 1974).

The critical health effect of petrol is chemical pneumonitis, arising from aspiration of liquid petrol or inhalation of petrol-contaminated vomitus (Toxicology update, 1989).

2.3.2 Kinetics and metabolism

Animal and human ADME data is unavailable since petrol is a complicated hydrocarbon combination. Local or systemic symptoms after cutaneous, oral, and inhalation exposure suggest petrol vapour or liquid penetration.

After solvent-based extraction or hydrocarbons from the skin, petrol exposure can be determined.

However, the profile of hydrocarbons found in the systemic circulation after cutaneous exposure is markedly different to that extracted from the skin (Hieda *et al*., 2005), indicating selective uptake and distribution of individual hydrocarbon components.

There is some evidence to suggest that petrol exposure may alter hepatic enzyme activity in rats and humans (Harman *et al*., 1981; Rao and Pandya, 1980), although the clinical relevance of such observations has not been established.

The primary method by which unstable components of petrol are eliminated is through exhaled air. This notion is derived from clinical observation; there is a lack of experimental investigations to validate this mode of elimination in humans.

2.3.3 Sources and Route of Exposure

Petrol consists of a blend of easily evaporating hydrocarbons, making inhalation the primary method of exposure (Cecil et al., 1997). Petrol vapour can accumulate to levels that are extremely toxic in enclosed or inadequately ventilated spaces, but instances of such exposures are infrequent (Takamiya et al., 2003). Table 2.1 provides a comprehensive overview of petrol vapour concentrations under various exposure circumstances, based on a typical sample. Petrol vapour has a distinct hydrocarbon profile than liquid petrol. The majority (>70%) of petrol vapour consists of light

hydrocarbons (C4 & C5) (Halder et al., 1986), while liquid petrol is primarily constituted of C6-12 compounds (>80%) (Carter et al., 2002). The deliberate act of inhaling vapour, commonly known as 'sniffing' or 'huffing', has been thoroughly documented in many studies (Edminster and Bayer, 1985; Cairney et al., 2002; Flanagan and Ives, 1994; Fortenberry, 1985; Remington and Hoffman, 1984; Poklis and Burkett, 1977).

2.4 Human Data on Health Effects of Acute / Single Exposure

General Toxicity

The potential health hazards associated with the handling and utilisation of petrol are negligible, as long as the product(s) are handled in compliance with proper health and safety protocols (Henry, 1998).

The primary health consequence linked to petroleum being subjected is chemical pneumonitis, which occurs when vomit is inhaled into the lungs after consumption (Litovitz and Greene, 1988). Heart arrhythmia and ventricular fibrillation can occur as an uncommon problem of petrol intoxication. This is caused by an enhanced sensitivity of the heart muscle to naturally occurring catecholamines, as stated by Litovitz in 1988.

Intensive intoxication can damage lungs, the kidneys, liver, and spleen vascular endothelium. This damage is particularly observed in the proximal tubules of the kidney, where it leads to renal lipid degeneration.

2.4.1 Inhalation

As evaluated by the Toxicology Update in 1989, most people can easily identify petrol vapour at concentrations of less than 1 to 2 parts per millilitre (Amoore et al., 1983). However, olfactory thresholds may be raised by repeated exposure within a 24-hour period or by long-term occupational exposure.

Some have proposed that the main factor influencing acute toxicity is not the length of exposure but rather the amount of petrol vapour (Wang and Irons, 1961). This presumption pertains to a single study in which three dogs were dosed with various petrol products and depends on exposures lasting less than thirty minutes.

After inhalation, effects on the central nervous system (CNS) become noticeable beyond 900 ppm in a matter of minutes; the symptoms have similarities to alcohol intoxication in terms of morbidity (feeling dizzy, excitement, incoordination, etc.).

When petrol concentrations are high enough (>10,000 ppm), it can serve as an anaesthetic and occasionally cause instant unconsciousness. As Takamiya et al. (2003) have noted, one important contributing factor to fatal occurrences is the effect's quickness.

2.4.2 Ingestion

Consuming petrol can lead to immediate and widespread symptoms of irritation in the gastrointestinal tract, such as nausea, vomiting, abdominal pain, and diarrhoea (Toxicology update, 1989). The oral toxicity of petrol is relatively low, similar to other petrochemical products like kerosene (2 – 17 g kg-1) (Chilcott, 2006). Considering negligible respiratory consequences from breathing or vomiting petrol, 7.5 g kg-1 is the fatal dose for adults. ..

2.5 Animal and In-Vitro Data

2.5.1 General toxicity

The immediate toxicity of petroleum in various animal species is generally similar to that observed in humans, primarily affecting the central nervous system, lungs, and kidneys (IARC, 1989). Table 2.2 presents the LD50 data as well as information on skin and ocular irritation.

Table 2.1: Acute Toxicity Data for Petrol

Test	Species	Result
Acute oral (LD_{50})	Rat	13.6 g kg^{-1}
Sensitisation	Guinea pig	Not sensitising
Primary dermal irritancy	Rabbit	slight
Acute dermal	Rabbit	No mortalities
Primary eye	Rabbit	Non irritating

Source: (IARC, 1989)

2.5.2 General Toxicity of Petrol

The main pathological condition associated with chronic exposure to high levels of inhalant abuse is dysfunction of the central nervous system. This condition has been extensively documented in cases of frequent recreational exposure, also known as "sniffing" or "huffing". (Edminster and Bayer, 1985; Cairney et al., 2002; Flanagan and Ives, 1994; Fortenberry, 1985; Remington & Hoffman, 1984; Poklis and Burkett, 1977; Valpey et al., 1978). Currently, there is not enough information to definitively establish a clear connection between long-term (work-related) exposure to petrol and other pathological diseases (Harrington, 1987). This could be attributed to the fact that petrochemical workers are susceptible to a diverse array of chemicals, along with other factors that may complicate the situation (IARC, 1989).

Lead has traditionally been recognised as the primary element in gasoline that causes neurotoxicity. Studies have shown a connection between the amount of lead in the body and neurological impairments resulting from the abuse of gasoline through inhalation (known as 'sniffing' or 'huffing'). It is important to mention that tetraethyl lead (TEL) has a relatively low volatility (0.4 mm Hg at 25ºC). Therefore, chronic exposure to TEL through the skin is more likely to occur than inhalation, particularly in the case of petrol sniffing (Toxicology update, Until). Until 2000, UK and most of Europe have sold only 'unleaded' petrol. The policy sets a maximum allowable amount of lead in marketable petrol, which is defined as less than 0.005 g L-1 in Annex I of the 1998 EU Directive (EU, 1998).

Although there is a recognised correlation between long-term exposure to petrol and cancer of the kidneys in male rats (Halder et al., 1985; Olson et al., 1987; Short et al., 1987), there's is presently no proof to establish a connection between petrol exposure and kidney cancer in people (Trump et al., 1984; IARC, 1989). The susceptibility of male rats is commonly believed to be influenced by a unique protein called α-2-microglobin, which is not found in other mammals (Olson et al., 1987; Olson et al., 1990).

2.5.3 Genotoxicity

Petrol exhibited carcinogenic properties in drosophila melanogaster when present in cell culture medium at doses ranging from 1000 to 2500 ppm, as demonstrated by Nylander et al. in 1978.

The investigation of petrol's potential to cause gene alterations in bacteria (Salmonella assay) and mammalian cells using the mouse lymphoma assay (Conaway et al., 1984) yielded no positive findings. Additionally, a different mammalian cell assay for gene

mutation utilising a human lymphoblastoid cell line yielded unfavourable outcomes (Richardson et al., 1986). Conaway et al.'s 1984 in vitro bone marrow experiment showed no clastinogenic consequences in rats.

A research indicates that the UDS (unscheduled DNA biosynthesis) assay has yielded positive outcomes. Limited in vitro investigations, One dose level showed a beneficial effect on rats treated with hepatocytes and a minimal effect on human and mouse hepatocytes. The investigation found that in vivo UDS testing in rats yielded null results, while mice showed a modest rise in UDS (Loury et al., 1986). In summary, it may be inferred that petrol does not possess substantial mutagenic properties.

2.5.4 Carcinogenicity

Multiple epidemiological studies have failed to establish a statistically significant connection between malignancies and professional contact with petrol (Dement et al., 1997; Enterline, 1993; Enterline & Viren, 1985; McLaughlin, 1993; McLaughlin et al., 1983; McLaughlin et al., 1985; McLaughlin et al., 1984; Norell et al., 1986; Rushton and Alderson, 1983).

2.5.5 Reproductive and Developmental Toxicity

No evidence about the specific effects of petrol on human reproductive or developmental health was found. The study conducted by McKee et al. (2000) showed a NOAEL (No Observed Adverse Effect Level) of 20,000 mg m-3 for reproductive harm in an two-generation rat study. Petrol is not a CHIPS Chemical Hazardous Information and Packaging Supply concern to reproduction or development.

Benzene is a chemical compound.

Benzene serves as a solvent and fundamental chemical precursor in industries such as shoemaking, paint production, insecticide manufacturing, and others. Consequently, workers in these sectors may be exposed to benzene throughout their daily activities. Benzene, a compound generated by industrial emissions, tobacco smoke, and car fumes, is another contributor to air pollution. Additionally, it can cause a decrease in blood cell counts, result in long-term poisoning, and potentially trigger the development of acute myelogenous leukaemia (AML). The citation is from Loomis et al. (2017). However, prolonged and consistent exposure to low levels of benzene can also lead to a decrease in the quantity of blood cells and an increased likelihood of developing myelodysplastic syndrome, also known as (MDS) (Schnatter et al., 2012, Koh et al., 2015, Lan et al., 2004). This has been the subject of significant research and scrutiny. Prior toxicological investigations have examined the methods by which benzene causes damage to blood cells (Chen et al., 2019, Guo et al., 2019, Liang et al., 2018). However, there is still a deficiency in our ability to detect and identify early signs of toxicity and provide timely warnings.

Benzoene is a transparent, colourless, and flammable substance that has a scent resembling petrol. It evaporates easily to create vapours in the air. The predominant source of this organic molecule in the atmosphere is attributed to a range of factors including motor vehicle emissions, wood-burning fires, petrol vapours, combustion of coal and oil, and tobacco smoke. Improper transportation and industrial usage are the primary contributors to benzene contamination in the environment. Transportation-related pollutants are the main cause of benzene emissions in urban ambient air. However, the benzene concentrations within smoke-filled houses are significantly greater.

Aside from breath and skin absorption, benzene can also potentially be ingested through food and drink, as stated by Wilbur et al. (2008). The presence of higher levels of benzene in groundwater can potentially impact the variety and organisation of biological systems and constitute a threat to human well-being (Fahy et al., 2005). Moreover, the contamination of groundwater with benzene has been associated with natural gas, as indicated by Kerfoot et al. (2009). Tobacco smoking, whether active or passive, is an additional means by which benzene exposure occurs in both the general public and occupational groups (Arnold et al., 2013).

The liver metabolises benzoene into several toxic byproducts, including hydroquinone (HQ), phenol, benzene oxide, catechol, 1,2,4-benzenetriol, and trans,trans-muconic acid, whose is a ring-opened derivative (Snyder et al., 1999). The concentration of these hepatic metabolites in the bone marrow seems to be associated with benzene's specific toxicity towards haematological tissue (Rickert et al., 1979).

Benzene is classified to be a Group 1 carcinogenic for both people and animals due to its carcinogenic qualities. Consequently, the substance is related with health hazards and stress (IARC, 1982). For instance, studies conducted in the laboratory and field of epidemiology have established a robust correlation between benzene exposure and the development of leukaemia, specifically acute myeloid leukaemia (AML) (Baan et al., 2009). In addition, there is limited evidence establishing a connection between benzene and the development of several types of myeloma, aplastic anaemia, non-Hodgkin's lymphoma, as well as acute and chronic lymphocytic leukaemia (Baan et al., 2009).

Schnatter et al. found in 2012 that even occasional benzene exposure increases the risk of myelodysplastic syndrome.

For example, benzene can harm the immunological, neurological, or reproductive functions (ATSDR, 2007). Human peripheral lymphocytes can develop chromosomal abnormalities, animals exposed to benzene lose tolerance to infection, and B- and T-cells grow slower.

2.6 Structure of Benzene

C6H6 is benzene, an organic molecule. Benzene has a planar ring of six carbon atoms joined by one hydrogen atom. Fundamental petrochemical benzene is in crude oil. Cycles of unbroken pi bonds between carbon atoms make benzene a hydrocarbon with aromatic properties. In petrol stations, benzene, a colorless, extremely combustible chemical, gives off a pleasant smell. This chemical is mostly used to synthesize complex molecules like cumene and ethylbenzene. Each year, billions of kilogrammes of these chemicals are created. Industrial uses use benzene, but consumer products rarely use it due to its harmful effects (Folkins and Hillis 2005).

2.6.1 Discovery of Benzene

From the greasy residue of lighting gas generation, Michael Faraday extracted and characterized benzene as hydrogen bicarburet in 1825. Mitscherlich extracted lime and gum benzoin benzoic acid in 1833. It was called "benzin" by him. In 1836, French chemist Auguste Laurent termed the substance "phène".

2.6.2 Occurrence of Benzene

Both coal and petroleum contain small amounts of benzene. It is the outcome of many components undergoing incomplete combustion. Before World War II, the primary source of benzene for commercial application was derived from the byproducts of coke manufacture, specifically known as "coke-oven light oil," which was primarily utilised by the steel industry. In the 1950s, the plastics sector needed more benzene, thus petroleum was used to make it. In the present era, the petrochemical industry is responsible for the vast majority of benzene production, whereas coal contributes only a minor fraction to the overall output (Hillis 2005).

2.6.3 Historical Utilisation of Benzene in Various Applications

Benzene was utilised as an aftershave lotion during the late 19th and early 20th century due to its pleasant fragrance. Benzene was a prevalent industrial solvent employed until the 1920s, specifically for the purpose of removing grease from metal surfaces. As the risks associated with benzene became evident, toluene (methylbenzene), a less cancer-causing solvent with similar physical properties, was used as a substitute.

Treatment was stopped. Polyurethane The cement industry, Paint Strippers, Spot Removing agent, The hydraulic Torque wrench, and other consumer products used benzene.

2.6.4 Benezene Markers In Human trans-trans-Nucleic acid

Benzene exposure is measured using it as a biomarker at 1 ppm. You can also make muconic acid using the acid sorbic or its salts. The meal's 500 mg of sorbic acid boosts urine trans,trans-muconic acid elimination. After careful research, 0.12% of sorbic acid is eliminated in urination as trans, trans-muconic acid. Consuming 6-30 mg of sorbic acid

daily eliminates 10-50% accumulated trans, trans-muconic acid in nonsmokers and 5-25% in smokers.

2.6.5 Phenol

An hydroxyl (—OH) group attached to a carbon atom is a characteristic feature of an aromatic ring's structure. In addition to being the general term for the entire family, phenol is also the particular term of its most basic member, monohydroxybenzene (C_6H_5OH), which is also referred to as benzenol or carbolic acid. Phenols exhibit similarities to alcohols, although they have the ability to generate more robust hydrogen bonds. Consequently, they exhibit greater solubility in water compared to alcohols and possess higher boiling temperatures. Phenols exist as either colourless fluids or white solids at normal room temperature and can be quite poisonous and corrosive.

2.6.6 S-Phenylmercapturian acid

In the body, benzene exposure produces S-Phenylmercapturic acid (S-PMA). The biomarker is often used to evaluate benzene exposure (van Sittert et al., 1993). Industrial workers may have urinary amounts of up to 543 µg/g creatinine. In the study, smokers had a urine content of 9.1, which was recorded µg S-PMA/g creatinine, while non-smokers had 4.8 µg

2.6.7 Hydroquinone

It belongs to the phenol group and is derived from benzene. Its chemical formula is $C_6H_4(OH)_2$. The compound features two hydroxyl groups attached to a benzene ring in the para position. The substance is a solid with a white colour and a granular texture. Hydroquinones are alternative derivatives of this parent chemical.

2.6.8 Toluene, also known as methylbenzene

Gasoline and unprocessed petroleum contain toluene. Furthermore, it is used to synthesize benzene, graphic coloring agents, paints, and solvents. The lipophilic neurotoxic in this chemical degrades myelin in the brain's white matter. It also causes substantial cerebral and cerebellar degeneration.$20, $21. When someone intentionally inhales toluene vapours from a paint-saturated cloth or paper grocery pouch containing paints or lacquer thinners, which include toluene, they committed intentional abuse. Approximately ten percent to fifteen percent of Americans have used inhalants, but the exact prevalence is unknown. Dementia, ataxia, brain-stem damage, and corticospinal paralysis can result from prolonged toluene inhalation. Apathy, impairment of memory, visual perception issues, and maintained language skills are the main symptoms of dementia. MRIs and postmortems can reveal toluene-induced leukoencephalopathy.

2.6.9 Benzene synthesis

Industry produces benzene through catalytic transformation, toluene hydrodealkylation, imbalance, and steam cracking. Catalytic reformates made for 44-50% of US benzene production between 1978 and 1981, in accordance the findings of the ATSDR Toxicological Profile.

Low-octane hydrocarbons are converted to high-octane molecules by catalytic reformation.

Catalytic reforming mixes hydrogen gas with hydrocarbons, which boil at 60–200 °C. Then, a bifunctional catalyst such platinum itself hydrochloride or rhenium chloride is used at 500–525 °C and 8–50 atm. Aliphatic hydrocarbons cyclize and dehydrogenate to

become aromatic under these conditions. Extraction with diethylene glycol (DEG) or sulfolane separates the aromatic components from the reformate. After distillation, benzene is separated from aromatic chemicals. Aromatics are extracted from reformate to produce aromatic compounds with few non-aromatic components. Extracting and distilling the BTX compound (benzene, the aforementioned to and xylene isomers) yields aromatic compounds.

Like catalytic reforming, UOP and BP converted LPG, principally the gases butane and propane, into aromatic chemicals.

2.6.10. Toluene hydrodelkylation

Benzene is produced by hydrodealkylation of toluene. The chromium in molybdenum, or platinum oxide catalyst reacts toluene with hydrogen in this hydrogen-intensive process. At 500–650 degrees Celsius and 20–60 atmospheres, the reaction occurs. Sometimes greater temperatures replace catalysts in similar reactions. Toluene dealkylates into benzene and methane at these circumstances.

Toluene Toluene is disproportionated into benzene and xylene.

Benzene and xylene are produced by toluene disproportionation (TDP).

Since para-xylene (p-xylene) along with other xylene isomers have different demand, then Select TDP (STDP) method, an upgraded TDP process, can be used. Nearly 90% of TDP effluent is p-xylene. Some systems prioritise xylenes over benzene.

Steam cracking uses high temperatures and steam to break down hydrocarbon molecules.

Aliphatic hydrocarbons are steam-cracked to make ethylene and other alkenes. Depending on the starting material used for olefin synthesis, steam cracking can create pyrolysis petrol, a benzene-rich liquid byproduct. Pyrolysis petrol can be combined with other hydrocarbons to supply petrol or extracted to produce BTX aromatic substances (benzene, toluene, and xylenes).

2.6.11 Different methods

Although these alternate paths to benzene may not have significant commercial significance, they are nonetheless accessible. Metals possess the ability to reduce phenol and halobenzenes. Benzoic acid and its salts undergo decarboxylation, a chemical process that leads to the production of benzene. The diazonium complex generated from aniline reacts with hypophosphorous acid, leading to the production of benzene. Alkyne trimerisation refers to the chemical process in which three molecules of acetylene are combined to produce benzene.

2.6.12 Utilisations of Benzene

Ethylbenzene, the chemical cumene, a substance called and nitrobenzene are mostly produced from benzene's intermediary role. Close to two-thirds of 1988 American Chemical Society compounds had benzene rings (Brown, 1988). The chemical transformation all ethanol, a precursor to styrene, for polymers and plastics like polystyrene uses over 50% of benzene production. Nylons, which are made into fabrics and technical polymers, are made from cyclohexane. It is 10% of global benzene production. Rubber, lubricants, dyes, detergents and prescription medications, explosives, and insecticides utilize less benzene. Chinese benzene consumption surpassed US

consumption in 2013. Apparently African continent and the Middle East are producing more benzene than the United States and Western Europe.

Modern fuel additives employ toluene instead of benzene. The other solvent dissolves similar liquids, but toluene is safer and dissolves more. Toluene-to-benzene conversion is another method.

Petrol contains benzene: A constituent part

The fuel addition benzene decreases engine knocking and boosts octane. Previous to the 1950s, fuel commonly had a significant amount of benzene, until tetraethyl lead became the preferred antiknock additive. Benzene has been reintroduced as a petrol additive in some nations after the global discontinuation of leaded petrol. In the United States, the amount of benzene in petrol is closely controlled and often limited to roughly 1% due to concerns about its detrimental impact on health and its ability to contaminate groundwater (Kolmetz, 2007). The current fuel requirements in Europe impose a maximum benzene concentration restriction of 1%. New EPA restrictions in 2011 reduced fuel benzene to 0.62%.

2.6.12 Benzene Reactions

The majority of benzene reactions involve the substitution of a proton with another group (Stranks et al., 1970). Benzene can undergo electrophilic aromatic substitution as a general method of derivatization. Benzoene undergoes substitution reactions with acylium ions and alkyl carbocations, resulting in the formation of substituted derivatives. This is due to the nucleophilic nature of benzoene. Benzene can undergo nitration, sulfonation, and chlorination reactions.

Various functional groups are incorporated into the benzene structure through electrophilic aromatic substitution. Sulfonating benzene uses oleum, a mixture of sulphuric and sulphur trioxide acids. Decaying sulfonated benzene is detergent-like. Nitration occurs between benzene and nitronium ions. The precursor of aniline is nitrobenzene. Chlorobenzene is produced by utilising chlorine with the assistance or an acidic Lewis catalyst, like aluminium trichloride.

2.6.13 Hydrogenation of Benzene

Hydrogenation turns benzene into cyclohexane. With high hydrogen pressures and heterogeneous catalysts like finely divided nickel, this reaction occurs. Room-temperature alkenes hydrogenate. In contrast, benzene and related compounds are less reactive and require temperatures exceeding 100 °C for hydrogenation to occur. This reaction is frequently employed in widespread industrial applications. Benzene is resistant to hydrogen in the absence of a catalyst. Due to the enhanced reactivity of cyclohexene and cyclohexadienes, it is not possible to halt the hydrogenation process in order to generate these compounds. However, benzene undergoes selective hydrogenation to diene via the non-catalytic Birch reduction process.

2.6.14.1The health effects of benzene

Bone marrow failure and malignancy are linked to benzene. It is classified as a carcinogen. Benzene is strongly associated with cardiovascular disease, acute leukaemia, aplastic anaemia, and abnormalities in the bone marrow, as supported by a substantial body of epidemiological, clinical, and laboratory research (Kasper et al., 2004; Merck, 2011; Bard, 2014). Smith (2010) has established a correlation between benzene and various types of blood cancers, including acute myeloid leukaemia (AML), aplastic

anaemia, myelodysplastic syndrome (MDS), acute lymphoblastic leukaemia (ALL), and chronic myeloid leukaemia.

Only zero benzene content is safe, according to the API in 1948. Even low doses can cause harm (Smith, 2010). According to the US Agency for Health and Human Services, benzene causes cancer. AML and ANLL are specifically induced by benzene, according to the WHO (2008). The IARC classified benzene as carcinogenic. Benzene poses a worldwide health risk to humans due to its widespread use in petroleum and hydrocarbon fuels. Benzoene causes chromosome damage and DNA strand breaking, as well as impacting the liver, kidney, lung, heart, and brain. Both humans and animals are susceptible to developing cancer as a result of exposure to benzene. Studies have shown that exposure to benzene, regardless of the method, can induce cancer in both male and female experimental animals of various species (Huff, 2007; Rana and Verma, 2005).

2.6.15 Benzene exposure

Benzene can be swallowed or absorbed through the skin when water is contaminated. Benzoene, which undergoes hepatic metabolism, is excreted through the urine. Activated charcoal (AC) tubes are employed for the collection of benzene in both air and water, while analysis is conducted using a gas chromatograph. Urine, blood, and breath tests can be employed to measure the amount of benzene in individuals. However, their usefulness is restricted because benzene is rapidly metabolised within the human body (Yardley-Jones et al., 1991).

Benzene exposure can lead to the progressive development of aplastic anaemia, leukaemia, and multiple myeloma (OSHSTHS, 2015).

OSHA limits workplace benzene. In a forty-hour workweek or eight-hour workday, benzene in the workroom air cannot exceed 1 part per million. NIOSH recommends that personnel utilise respiratory protection where there is a potential for exposure to benzene levels exceeding the recommended 0.1 ppm (8-hour) contact limit due to its carcinogenic properties (CSIBA, 2015).

2.6.15.1 Benzene's exposure limits

The Environmental Protection Agency (EPA) of the United States has set the highest level of contaminants (MCL) for benzene in drinking water at 0.005 mg/L (5 parts per billion) with the United States National Primary Drinking Water Regulations (BT, 2000). The primary objective of this rule is to prevent the development of leukaemia caused by benzene exposure. This health aim is not enforceable but provides a sufficient safety buffer to prevent harmful consequences. The Environmental Protection Agency (EPA) requires the documentation of spills or accidental discharges containing benzene in the environment that weigh 10 pounds (4.5 kg) or greater.

Occupational brief exposure limit for benzene in the air is 5 parts per million for a duration of 15 minutes (NIOSH, 2004). Legal restrictions were implemented based on compelling data indicating significant health hazards for workers exposed to benzene. According to predictions, there is an estimated increase of five fatalities caused by leukaemia for every 1,000 employees exposed to a concentration of 1 part per million (ppm) over their entire working lives. (This estimate assumes that there is no maximum limit to the carcinogenicity of benzene.) In addition, OSHA established a threshold of 0.5 parts per million (ppm) as a measure to further reduce worker exposure levels

As per NIOSH (2004), an IDLH circumstance is presently described as a situation where there is a potential for being exposed to airborne contaminants that can cause mortality or immediate or delayed persistent ill health consequences, or that hinders the ability to escape from a situation like this. The previously NIOSH Respirator Selection Logic provides a clear and concise definition of this concept. At the highest IDLH value, only the most trustworthy breathing apparatus that protects workers is allowed. Its primary function is to provide a means of escape for the worker in case their respiratory protection equipment fails, therefore assuring their safety in a hazardous workplace (NIOSH 2004). In September 1995, NIOSH released a new guideline for the establishment of suggested limits of exposure (RELs) for a range of substances, including carcinogens. Since benzene is carcinogenic, NIOSH advises workers to wear protective respiratory gear when exposed to levels above the REL of 0.1 ppm. NIOSH sets a 15-minute short-term occupational exposure limit (STEL) of 1 ppm.

2.6.15.2 Benzene exposure routes:

(a) Inhalation

Vehicle service stations, wood smoke, smoke from cigarettes, petroleum transfer, motor vehicle exhaust, and industrial emissions are all potential sources of benzene in outdoor air, leading to low concentrations (GBTA, 2010). As per the ToxGuide (2010), About 50% of US benzene exposure comes from smoking or being near tobacco smoke. Smokers inhaled 1.8 milligrams of benzene per day from 32 cigarettes. The quantity mentioned is approximately tenfold the normal daily consumption of benzene by nonsmokers (PHSB, 2007).

Exhalation is the primary mechanism through which benzene, when breathed, is expelled without undergoing any changes. Between 16.4% and 41.6% for the benzene that was kept in the body was eliminated through the lungs within a time span of five to seven hoursAbout 52 and 87% of benzene exposed to 63 and 405 mg/m3 for between one and five hours was eliminated form polyphenol in urine within 23 to 50 hours of exposure. In another human investigation, 30% of topically administered benzene was eliminated as polyphenol in the urine. Most benzene metabolism occurs in the liver. CASRN (2011) stands for CAS Registry Number.

2.6.16 Soft drink consumption

Ascorbic acid (vitamin C) particular benzoic acid (preservative) can react to create benzene in certain conditions and media. A March 2006 British National Standards Agency assessment examined 150 soft drink brands. It was determined that four people had benzene levels above WHO guidelines. Removal of affected lot numbers from shelf. The US FDA reported similar issues (2006).

(b) Contamination of Water Supply

In 2005, a substantial amount of benzene was released, leading to the cessation of water showed that they supply to the city of Harbin in China, which has a population of approximately nine million people (Guardian, 2005). Benzoene was released onto the Songhua River after a CNPC facility blast in Jilin city on November 13, 2005. City drinking water comes from this river. Exposure to extreme heat might result in the contamination of plastic water pipes with benzene (Isaacson et al., 2021).

(d) The act of deliberate and systematic extermination of a certain group of people, known as genocide.

The Nazis employed benzene injections as one of their diverse methods for executing individuals (Auschwitz-Birkenau State Museum, 2020).

2.6.17 The Toxicology of Benzene Exposure: Indicators and Biomarkers

Benzene exposure levels can be determined by a range of assays. Direct measurement of benzene can be conducted in breath, blood, or urine. However, due to its rapid elimination through inhalation or biotransformation, these studies are typically limited to the initial 24 hours following exposureIn wealthy countries, most people have blood benzene along with other aromatic petroleum compounds.Since the vast majority of these metabolites build in the urine in a correlation with exposure and can stay in considerable amounts for a few days after exposure ends, they may be indicators for human exposure. Based on Ashley et al. (1994), Fustinon et al. (2005), ACGIH (2009), Baselt (2008), as well as other credible sources, the ACGIH has set occupational biological exposure limits. Set limitations of 500 μg/g creatinine as muconic acid or 25 μg/g creatinine for phenylmercapturic acidic compounds in end-of-shift urine samples.

(b) Biochemical alterations

Most bacteria and eukaryotes have the ability to undergo the process of oxidising benzene, despite it not being a commonly used substance for metabolism. The bacterial ring destabilizes and is rapidly reduced by NADH to create a cyclical diol containing two double bonds when exposed to dioxygenase. This disturbs molecular aroma. Then, NADH helps create a second reduction process that turns diol into catechol. Catechol

metabolism produces acetyl as well as succinyl CoA, chemicals are used for energy production within the Citric Acid Cycle.

The liver serves as the initial site for the complex benzene metabolic pathway. Numerous enzymes are involved. The enzymes that are included are quinine oxidoreductase (NQ01), also known as DT-diaphorase or NAD(P)H dehydrogenase (quinone 1), as well as glutathione (GSH) and myeloperoxidase (MPO). Cytochrome P450 2E1 (CYP2E1) is one such example. CYP2E1 participates in the transformation of phenol into hydroquinone, benzene into oxepin (benzene oxide), and hydroquinone into both benzenetriol and catechol. Polyphenols are synthesised from catechol, benzenetriol, and hydroquinone. MPO converts these polyphenols into benzoquinones within the bone marrow. The aforementioned chemical intermediates and metabolites cause genotoxicity by inhibiting a number of others II, which maintains chromosome structure, disrupting microtubules, which organize and stabilize cells, and generating oxygen-containing radicals, or free radicals, which can cause point mutations, higher oxidative stress, DNA strand fragmentation, and altered GSH and NQ01 redirect metabolic processes away from harmful effects. Benzoquinone undergoes metabolism by NQ01, resulting in the formation of polyphenols, which counteract the detrimental effects of MPO. Phenylmercapturic acid is partially synthesised by GSH (Smith, 2010; Snyder and Hedli, 1996).

These enzymes' genetic variations can increase or decrease function. CYP2E1 mutations boost its activity and hazardous metabolite production. NQ01 mutations affect detoxification and function. Mutations in myeloperoxidase diminish function and minimize harmful metabolites. Eliminations or mutations in the gene encoding

glutathione (GSH) result in impaired functionality and diminished capacity for detoxification. Dougherty et al. (2008) suggest that these genes could be the primary targets for genetic screening to determine vulnerability to benzene toxicity. Chemical and hazardous compounds' molecular impacts are studied in molecular toxicology (c).Due to its advanced understanding of biological principles, molecular toxicology is becoming more essential in benzene toxicity. Glutathione is increasingly acknowledged as a new biomarker for exposure and effect due to its notable involvement in mitigating benzene-induced DNA breaks (Fracasso et al., 2010). In addition to haematological assays, fluorescence in situ hybridization (FISH) using DNA probes can be used to monitor these abnormalities and assess the hematotoxic consequences of benzene (Eastmond et al., 2000). Enzymes possessing polymorphic gene codes participate in the metabolic processes of benzoene. Research indicates that an individual's genetic makeup at these specific locations on their DNA may influence their vulnerability to the detrimental effects of benzene exposure. Individuals with variations of NAD(P) H:quinone oxidoreductase 1 (NQO1), microsomal epoxide hydrolase (EPHX), and deletion of glutathione S-transferase T1 (GSTT1) exhibited a higher prevalence of DNA single-stranded breaks (Garte et al., 2000). (d) Carcinogenic Activity and Biological Oxidation Benzene's carcinogenic effects can be understood by studying biological oxidation metabolites. When pure benzene is oxidized in the body, it becomes benzene oxide, an epoxide that can damage DNA. Moreover, this compound is not easily cleared from the body.

2.6.18 Analysis of Biomarkers for Benzene Exposure: Sources and Laboratory Testing

Benzene samples can be obtained from humans breath, urine, and blood metabolites. The following are the biomarkers associated with benzene:

1. Phenol is a chemical compound.

2. Hydroquinone is a chemical compound.

3. Catechol is a chemical compound.

1. UPLC-MS/MS can be employed to analyse 1, 2, and 4 benzenetriol.

The following cytokines were measured: MCP-1, TNF-α, VEGF, IL-9, MIP1-α, 1L-4, IL-10, and IL-15. The ELISA technique can be employed for their analysis.

8. Parameters pertaining to the study of blood and its components. Study of blood and its disorders An Autoanalyzer can be utilised for the analysis of these substances.

9. DNA damage can be assessed using the PCR and ELISA techniques.

2.6.19 Minimising Benzene Exposure

Efforts should be made to minimise people exposure to benzene regardless of its form, as the pathways of exposure are well recognised. At the workplace, adherence to regulations and protocols is crucial when it comes to the handling and utilisation of chemicals. It is obligatory to use safety equipment such as PPE when dealing with compounds containing benzene, especially in petroleum refineries and stations. It is recommended to utilise items such as dungarees, boots, gloves, and goggles. Given the proven fact that there is no acceptable level of benzene, it is imperative to strictly monitor and regulate the usage of benzene derivatives, such as benzoic acid, which are commonly used as preservatives.

2.6.20 Impact on Benzene Exposure on the Immune System

2.6.20.1 Cytokines

Hematopoietic progenitor cells are vulnerable to direct harm caused by benzene, which can lead to programmed cell death or diminish their responsiveness to cytokine and cellular adhesion molecules. However, the harmful effects of benzene on fully developed blood cells or stromal cells can disrupt the intricate system of adhesion molecules, chemokines, and cytokines that govern hematopoiesis, including processes such as haematological commitment, maturation, or mobilisation (Kalf et al., 1996: King et al., 1989). Therefore, individuals with genetic variants that alter crucial mechanisms governing hematopoiesis may have a higher vulnerability to hematotoxic effects following exposure to benzene (Lan et al., 1995).

A complete literature review on cytokine and benzene exposure was conducted in this study.

Zhang et al. (2010) found that even at low concentrations, benzoene can produce hematotoxicity by many mechanisms, such as altering gene and protein expression, patterns of DNA methylation, and miRNA profiles.

Toxicogenomic studies on benzene-exposed humans must understand gene-environment interactions and create biomarkers indicating exposure, immediate effect, and vulnerability. The research encompass genome-wide examination of genes associated with susceptibility (genomics), the expression of genes (transcriptomics), the expression of proteins (proteomics), and epigenetic changes (epigenomics) (Zhang et al., 2010).

Examining the cytokines and chemokines expression can serve as a means to investigate the impact on benzene or its byproducts on the immune system. Following exposure to benzene, specific cases major human peripheral blood mononuclear cells (PBMC) demonstrated an increase in the production of inflammatory cytokines, while in other cases, a decrease was observed (Gillis et al., 2007). The scientists showed that the generation of TNF-α by activated PBMC increased in a dose-dependent manner for each benzene metabolite. In addition, the only method to increase the concentration of IL-6 was through treatment with catechol, benzenetriol, and BQ. However, treatment with HQ enhanced the production of IFN-γ. In contrast, catechol and HQ reduced GM-SCF and IL-1β secretion. As Gillis et al. showed in 2007, BQ therapy decreased IL-2 synthesis.

Renz and Kalf (1991) demonstrated that using separate murine stromal macrophages, HQ hinders the enzymatic transformation of 31 kDa pre-IL-1α into the fully developed cytokine through the action of the digestion protease calpainThe study revealed that HQ caused a drop in IL-1 secretion, and this decrease was directly related to the dosage of HQ. Additionally, a reduction in overall protein content was also attributed to HQ. Therefore, the suppression of cytokine production by mononuclear phagocytes, which are important in regulating hematopoiesis, can worsen myelotoxicity (Carbonnelle et al., 1995). The impact of HQ on IL-1 was recorded in a separate study carried out during the previous year (Niculescu et al., 1995). Niculescu et al. (1995) showed that 1,4-benzoquinone, the oxidation product of HQ in the cell, has a concentration-dependent inhibitory effect on extremely pure human platelet calpain. When HQ was introduced into Bl human cells, which are self-stimulated by interleukin-lβ, the cells ceased autonomous growth and released interleukin-lβ into the culture media (Niculescu et al., 1995). There is a significant amount of similarity between both cytokines, as GM-CSF plays a role in supporting the growth and differentiation of myeloid hematopoietic progenitor cells (Irons and Stillman, 1996). Previous research by Irons and Stillman (1996) has demonstrated that treating human bone marrow cells, namely those containing hematopoietic progenitor cells (HPC) referred to as CD34+ cells, with HQ enhances their ability to form colonies in response to GM-CSF, but not IL-3. It seems that HQ stimulates any number of secondary signals that, in combination with GM-CSF, synergistically induce HPC to enter the cell cycle, even if these signals are individually insufficient to do so. Proliferation or survival alterations can make vulnerable target cells more likely to experience replication-dependent harm and subsequent development of

cancer in a rapidly dividing tissue, such as bone marrow, where regulating the proliferation of stem and progenitor cells is of utmost importance (Irons and Stillman, 1996).

TNF-α inhibits bone marrow cell growth and colony formation. Additionally, it is known to stimulate NF-κB activation in several cell types. Haematological precursor cells (HPC), which rely on active NF-κB, must evade apoptosis. HQ affects cytokine responsiveness, promotes HPC cellular death, and suppresses NF-κB activation in T and B cells, according to Kerzic et al. (2003). In 2003, Kerzic et al. observed that HQ exposure at various levels suppresses TNF-α-induced activation of NF-κB in haematological progenitors and primary HPC. HQ enhanced cell sensitivity to TNF-α's proapoptotic actions. These results suggest that NF-κB is necessary for the survival of HPCs and that these cells are susceptible to apoptosis produced by cytokines when NF-κB suppression caused by HQ occurs (Kerzic et al., 2003).

MDS is a chronic disorder characterised by abnormal bone marrow development and inflammation, typically observed in individuals previously exposed to benzene. The study also examined the temporary toxic effects of benzene exposure, known as benzene poisoning (BP). According to Kerzic and Lin (2007), just one specific polymorphism, the −238 (G→A) polymorphism, was found to be associated with the development of BID. Neither de novo MDS nor BP was linked to this polymorphism. Evidence suggests that genetic diversity in TNF-α expression may shield injured blood cell progenitors from CD8+ T-cells, promoting the selection of particular cell clones in blood-related malignancies. Furthermore, there is a possibility that the gene −238A is associated with

other genes in a larger haplotype that influences the generation of TNF-α within progenitor cells of the hematopoietic system (Kerzic and Lin, 2007).

Omatsu et al. (2010) found that temporarily removing these cells greatly impairs the bone marrow cells' capacity to transform into adipogenic and osteogenic forms. This also leads to a notable decline in the number of cycling lymphoid and erythroid progenitors. The material itself and associated reactive metabolites, or p-benzoquinone (BQ) and HQ, were tested for their impact on MSC survival (Zolghadr et al., 2012). CXCL12 expression decreased with HQ but increased somewhat with BQ. The heightened attraction and adherence of hematopoietic stem cells (HSCs) to the hematopoietic niche are probably due to increased expressions of CXCL12, whereas reducing the levels of CXCL12 might have the opposite impact. Moreover, alterations in the expression of CXCL12 have been demonstrated to disturb the hematopoietic stem cell (HSC) niche, resulting in haematological failure and contributing to the progression of leukaemia (Zolghadr et al., 2012).

In another investigation, peripheral blood mononuclear cells (PBMC) that were exposed to four benzene metabolites (catechol, hydroquinone, 1,2,4-benzenetriol, and 1,4-benzoquinone) exhibited notable and concentration-dependent enhancements in the production of various chemokines released outside the cells, such as IL-8, Eotaxin, MIP1-α, and RANTES (Gillis et al., 2007). Cells exposed to HQ, benzenetriol, and benzoquinone exhibited an increase in MCP-1 chemokine synthesis, however catechol did not have the same effect (Gillis et al., 2007). Moreover, these treatments resulted in an increase in the inflammatory cytokine IL-6. This study aligns with prior research indicating that several cell types exhibit increased production of IL-8 while exposed to

benzene compounds (Bironaite et al., 2004). Gillis et al.'s study found that stimulating PBMCs by administering the PMA ionomycin mixture resulted in a substantial increase in cytokine production, ranging from 10 to several thousand times higher. This led to a large elevation in the levels of cytokines in the medium. Various metabolites of benzene exert distinct impacts on the capacity of activated PBMCs to release soluble cytokines. Benzenetriol or BQ did not diminish IL-8 and MCP-1 chemokine production at a given concentration, whereas HQ and catechol did. The concentrations of other chemokines either remained mostly unchanged or increased (Gillis et al., 2007).

Genetic variations that affect NAD(P) function enhance the risk of benzene-induced myeloid cell injury. A study by Miyazawa et al. (2007) found that suppressing or inhibiting NQO1 in the endothelium of human bone marrow leads to a decrease in the production of adhesion molecules caused by TNF-α and a reduction in the adherence of progenitor cells to endothelial cells. This effect is mediated through the NF-κB pathway.

Atopy refers to a genetic predisposition to develop allergic reactions to certain substances.

The reactive metabolites of benzene, namely hydroquinone (HQ) and benzoquinone, exhibit various consequences beyond their immediate harmful properties. Benzene and its metabolites have the ability to directly or indirectly impact mast cells and basophils, which are important cells involved in respiratory allergens such as rhinitis and bronchial asthma (Triggiani et al., 2011). These substances can have a direct effect or work in conjunction with functionally relevant T lymphocytes, macrophages, and monocytes. These chemicals have been found to prevent the release of substances from mast cells and reduce the inflammation in the lungs produced by IgE in rats (Triggiani et al: 2011). Furthermore, benzene metabolites alter the chemical composition and physiological

functions associated with other immunocompetent cells and have the potential to compromise lung immune responses. The primary mechanism of benzene metabolites is to hinder early transduction signals. Triggiani et al. (2011) found that benzene metabolites have distinct effects on the innate immunity of basophils and mast cells, as well as on chronic inflammation in the lungs.

The study conducted by Boscolo et al. (2000) found a significant correlation between urinary trans, trans-muconic acid, a metabolite of benzene, and NK CD16+CD56+ lymphocytes in unnoticed atopic and nonatopic women. The correlation between NK cells and urine trans, trans-muconic acid in women with low environmental exposure suggests that benzene might stimulate NK cell function (Boscolo et al., 2000).

A study conducted three years ago discovered that aromatic compounds such as chlorobenzene might elevate the occurrence of milk and egg white allergies (Lehmann et al., 2001). Ethylbenzene and chlorobenzene increased IL-4-producing CD3+ T-cell populations. The presence of allergens can lead to the development of a type 2 skewed memory, which is characterised by an elevated number of type 2 T cells releasing IL-4 and a reduced number of type 1 T cells producing IFN-gamma (Lehmann et al., 2001).

2.6.21. Effects of Benzene on the Lungs

When tobacco leaf pigments are burned, tar includes a lot of hydroquinone. A study by Geiselhart and al. (1996) found that HQ in cigarette tar inhibits IL-2-dependent T-cell proliferation. HQ blocked 90% of IL-2-induced main human T lymphoblast (HTL) proliferation in vitro without killing them. HTL's S phase cell cycle progression was reduced by HQ without affecting IL-2 binding.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Study Location

This investigation was undertaken at Abuja-area NNPC Limited oil facilities. Abuja is a Nigeria federal capital territory which houses federal ministries and parastatals. Abuja is a civil service state with larger percentage of the population gainfully employed in both the government and private establishments, including NNPC Limited. However, many others engage in trading, bronze casting, commercial bus and petroleum tank driving, automobile mechanics and gasoline and diesel repairs. Although, NNPC is company of international repute, which its workers are exposed to petroleum products on daily basis, however, these workers ought to follow safety rules, it is expected that the level of their adherence to these rules will be determined through their health profiles.

3.2 Study Area

Study areas included all the marked areas where all the participants in this study are located. They include the petrol stations, automobile mechanic workshops and gasoline and diesel mechanics workshops.

3.3 Research Design

This cross-sectional study required consent before completing a questionnaire. The questionnaire aimed to gather data and demographic data from the participants. The questionnaire had two main sections: demographic data (age, their sexual orientation, marital status, as well as educational attainment) and exposure duration and form, underlying health challenges, alcohol and cigarette consumption tradition, and basic health indicators.

3.4 Ethical Approval

Ethical approval was sought and obtained at Ministry of Health, Abuja and Ethics committee of NNPC Limited, Abuja.

3.5 Consent

Prior to sending the questionnaire to the participants to gather their demographic information, their consent was obtained and the benefits and potential drawbacks of the study were clearly outlined to them.

3.6 Study Subjects

The subjects included all workers of NNPC Limited at Abuja and environs who had been exposed to petroleum products and were willing to participate in the research. The participants were those who had been exposed to petroleum products between zero to fifteen years.

3.7 Participants Grouping

1. Petroleum attendants
2. Automobile mechanics
3. Tanker drivers
4. N.N.P.C. workers
5. Control

3.8 Inclusion Criteria

1. Study participants who gave written consent.

2. Participants with petroleum exposure.
3. Participants with no related sickness, like hypertension, cough, heart related diseses.

3.9 Exclusion Criteria

1. Participants who had not given written consent for the study.
2. Participants who had never been exposed to petroleum products.
3. Participants who were having underlining illness.

3.10 Sample Size

$n = \frac{z^2 pq}{d^2}$ (This formula is used when the prevalence of variable of interest is unknown)

n = sample size z = 95% confidence level (1.96) p = 50% or 0.50 prevalence

q = (1-p)

d = Tolerated margin of error, 5% or 0.05

$$\frac{(1.96)^2 \times 0.5\,(1 - 0.5)}{0.05 \times 0.05} = \frac{3.84 \times 0.25}{0.0025} = \frac{0.96}{0.0025} = \mathbf{384}$$

For the purpose of this research 250 participants were able to particxipate till the end.

3.11 Sample Collection and Preparation

3.11.1 Blood Sample

Each participant's intravenous fasting blood sample of 10 millilitres (10mls) was obtained using sterile methods at the conclusion of their work shift on the day of exposure. Each

obtained blood sample was dispensed into an ethylenediaminetetraacetic acid container, with a volume of 5 millilitres per sample. Carefully combined, the mixture was refrigerated for examination. In the plain receptacle (5 ml), the material was coagulated subsequently centrifugation after fifteen minutes at 1000 RPM. Sterilized and stored at -200C, the extracted serum was analyzed.

3.12 BTEX Analysis (Alegretti *et al.*, 2004)

The measurement of benzene & its byproducts in human blood samples was performed utilising the BTEX Extraction procedure, as specified in EPA Method 5021. 0.5 grammes of each sample were added to with 0.2 grammes or anhydrous sodium sulphate (NaSO4) and stirred using a stirring rod. Subsequently, a volume of 25 ml of methanol was introduced into the sample and agitated using a stirrer with a magnet for approximately 20 to 30 minutes. Subsequently, the extract was prepared for purification and subjected to separation utilising silicon gel Solid Phase Extraction (SPE).

3.13 Silica Gel Clean Ups:

At the bottom of the syringe cartridge, glass wool was positioned. Next, a precise measurement of 5 grammes of silica gel was carefully weighed and placed into the cartridge. The cartridge was prepared by washing it with 5ml of methanol. The methanol was permitted to pass throughout the pores of the column until the liquid level in the column was slightly higher than the column frit. The accumulated methanol was disposed of. The sample was introduced into the column and the resulting liquid was promptly collected in a 25ml flask. Before exposing the column's frit to air, an extra 5ml of methanol was used to elute the column again, ensuring complete removal of the small amount of BTEX contained in the sample. The last portion was put into 2ml flask vials

that were labelled and had Teflon-Lined rubber covers. The BTEX chemical was analyzed using a US-made Agilent Technologies Varian 3800/4000 GC-MS. The specifications of the column were 30.0 metres in length, 320 micrometres in width, and 0.00 micrometres in height. The temperature of the detector was 300 degrees Celsius. The temperature at the inlet was 200 degrees Celsius. The pressure at the setting was 15.0 psi, while the carrier gas used was nitrogen. After 5 minutes at 50°C, the combustion temperature was increased by 15°C per minute to 200°C. Increased 2°C each minute until 210°C, it was held for 10 minutes. For each 2ml injection, 1.0 µl was injected, with an 80-minute cycle length. The external calibration used BTEX standards. BTEX chemicals were identified and quantified using chromatogram standards' retention lengths. The remaining solvents utilised was of high purity and met the standards for analytical grade.

3.14 Haematological Parameters (XN – 1000 Sysmex Operating)

concept: The XN concept employs laser flow cytometry to enumerate blood cells. Various signal intensities are gathered based on the cellular properties, and scattergrams of the corresponding measurement channels are generated. The Sysmex Haematology Analyzer employs a cyanide-free approach to measure haemoglobin.

3.15 Analyse Method

The machine aspirates 88 µl of the complete blood sample and mixes it homogeneously with immediately available cell fluids, resulting in dilution. The mixture then flows through a narrow tube where cells pass through individually. Lasers are utilised to measure the characteristics of the cell.

3.15.1 Oxidative Stress Biomarkers

Total Antioxidant Capacity

C – Reactive Protein (Zhang *et al*., 2018)

Test Principle

We use Sandwich-ELISA for ELISA. Each Immunoassay plate is pre-coated with a human CRP antibody. The matching antibody is added to micro ELISA plate wells with reference materials, or specimens. Each microplate well receives a detection antibody that has been biotinylated targeting Human CRP and an Avdin-Horseradish (HRP) conjugate. We then incubate the mixture. Wash away loose parts. In each well, substrate solution is added. A well with Human CRP, which is biotinylated detection antibody, or avidin-HRP conjugate will be blue. inhibit solutions inhibit substrate-enzyme reactions, turning them yellow. The optical density, also referred to as OD, is measured at 450 ± 2 nm using the spectrophotometer. Ocular density (OD) is proportional to human CRP. A traditional curve comparing optical density (OD) can be used to determine Human CRP levels in samples.

Procedure for conducting an analysis

3.15.2 Glutathione S peroxidase

Water to serve as the dilution accepted, blank, as well as sample was chosen. To each standard, blank, or sample dilution, a hundred microliters were added and placed in the

wells. The sealer plate was incubated at 37°C about 90 minutes. Solution was carefully deposited towards the bottom of the microscopic ELSA plate to avoid coming in contact with the inner layer in order to reduce foam formation. Without rinsing, each well was decanted. A hundred microliters of Biotinylated Detection Antibodies was added to each well immediately. Following a new sealer, the plate was placed in an incubator at 37°C for 1 hour. Each well was emptied and 350 ml of wash buffer added. For one minute, the well was submerged. Aspirating each well's liquid, emptying it, and drying it on immaculate absorbent paper. There were three further attempts at this method. Washing the testing strips prevented desiccation. Wells received 100 microliters of HRP conjugated work solution. After that, the plate was placed inside an incubator at 37°C for 30 minutes with a fresh lid. Decant each well and rinse five times. Ninety microliters in order of Substrate Reagent per well. An additional sealer was applied onto the plate, and was then placed in an incubator at 37°C for 15 minutes. Avoiding light exposure protected the plate. To measure optical density, each Microplate Reader was warmed for 15 minutes. Wells received fifty microliters or Stop Solution. A microplate reader set to 450 nm quickly measured each well's OD value.

3.15.3 Inflammatory and Immunological Markers

Interlukin 1 (Cetin *et al.,* 2018)

The reagent was procured from elabscience and utilised in line with the instructions provided by the manufacturer.

Sandwich-ELISA is used in this instrument. A Human IL-1 antibody was pre-coated on each micro ELISA plate in this kit. In the micro ELISA plate, reference materials—specimens—are mixed with the appropriate antibody. A detection antibody modified with

biotin that recognizes Human IL-1, Avidin, as well as Horseradish is Peroxidase (HRP), are then added to each microplate well and incubated. Water moves loose particles. Wells receive substrate solution. Biotinylated detecting antibody, Avidin-HRP conjugate, and Human IL-1 wells are blue. An enzymatic reaction is stopped by a stop solution, turning the color yellow. Spectrophotometry measures optical density (OD) at a particular section wavelengths of 450 ± 2 nm.

Procedure for conducting an analysis

Dilute acceptable, blank, and test wells were labeled. Each well received 100 microliters each of the expected, so blank, and sample dilution. The sealer plate was incubated at 37 degrees Celsius for 90 minutes. Solution was carefully put into the bottom of every micro ELSA plate, avoiding contact with the inner wall, to reduce foam formation. No-rinse draining of well water. For each well, 100 microliters of Biotinylated Detection Antibody solution that worked was added immediately. Afterward, the plate was sealed and placed in a 37°C incubator for 1 hour. We decanted each well and added 300 microliters of liquid wash buffer. Submerging the well for 1 minute followed. Every well's solution was withdrawn, drained, and let to evaporate on clean absorbent paper. Three more times, the step was taken. Use these tested strips after washing to avoid desiccation. Each well received 100 μL HRP conjugate working solution. A disposable cover was applied to cover the plate, that was subsequently set in a 37°C incubator for 30 minutes. Decant each well and rinse five times. Each well received 90 μL Substrate Reagent. The plate was then sealed and placed in a 37°C incubator for 15 minutes. Avoiding light exposure protected the plate. To measure optical density, our Microplate

Reader was warmed for 15 minutes. 50 microliters of Stop Solution went to each well. Using a 450 nm micro-plate reader, each well's OD value was quickly determined.

Interlukin 3 (Dehnabeh *et al.*, 2014)

Manufacturer instructions were followed for elabscience reagent use. Using test concept.

Kit uses Sandwich-ELISA. These kits' micro ELISA plates were pre-coated with Human IL-3 antibody. The previously micro Immunoassay plate's small cavities blend reference materials with the right antibody. Each microplate well is then progressively treated with a biotinylated antibody for Human IL-3 detection and the Avidin-Horseradish This enzyme (HRP) conjugate. Water eliminates loose bits. Into every hole, the a solution of substrate is added. Blue wells contain recombinant IL-3, biotinylated detecting antibody, and Avidin-HRP conjugate. Stop solutions are yellow and stop the enzyme-substrate reaction. Around 450 ± 2 nm wavelength, spectrophotometers calculate optical density (OD).

Procedure for analyzing

Identified diluted standard, blank, and sample channels. Every standard, vacant, and sample dilution received 100 microliters and was placed in the respective wells. A sealer-sealed plate was incubated at 37 degrees Celsius for 90 minutes. Solutions were carefully provided toward the bottom of the micro ELSA program plate to minimize foaming and inner wall contact. Each well was decanted without rinsing. Each well received 100 microliters of Biotinylated Detecting Antibodies working solution immediately. After applying a new sealant, the plate was incubated at 37°C for 1 hour. Each well was

decanted and given 300 ml of wash buffer. Then the well was completely submerged for 1 minute. The liquids inside of each of the wells were retrieved, emptied, and evaporated on a clean absorbent sheet. This step was repeated three times. These tested strips were used after washing to prevent drying. Each well received 100 microliters of HRP-conjugated working solution. After adding a new cover, the plate was incubated at 37°C for 30 minutes. Each well was decanted and rinsed five times. All wells received Ninety microliters of Substrates Reagent. After adding a new sealant, the plate was incubated at 37°C for 15 minutes. Protecting the plate from light. Light density was measured after 15 minutes of preheating the Microplate Reader. fifty microliters or Stop Solution was added to each well. The density of light (OD value) for each of the wells was measured simultaneously using a 450 nm micro-plate reader.

Interlukin - 4 (Eini *et al.*, 2020)

The reagent was purchased from elabscience and used as directed.

Testing idea

Kit uses Sandwich-ELISA. A Human IL-4 antibody is pre-coated on this kit's micro ELISA plate. Reference materials, or specimens, are inserted in the micro ELISA plate's tiny holes with the compatible antibody. A biotinylated detection antibody that targets Human IL-4 and a Avidin-Horseradish Peroxidase (HRP) combination are then sequentially added to each microplate well and incubated. The unpaid are deleted completely. Each well gets solution. Human IL-4, biotinylated detection antibody, with Avidin-HRP conjugate blue wells. The enzyme reaction is stopped by a stop solution,

turning the solution yellow. Optical density (OD) is determined using spectrophotometry at 450 ± 2 nm wavelength.

An experiment or analytic procedure

We chose diluting standard, blank, and sample wells. Each mixture of standard, blank, and sample received 100 microliters and was placed in the wells. Plate with sealer was incubated at 37 degrees Celsius for 90 minutes. To avoid foaming, solutions were carefully guided on the bottom of every micro ELSA plate without touching the inner wall. We decanted each well without rinsing. 100 microliters of Biotinylated Detecting Antibodies working solution was added to each well immediately. The plate was then sealed and placed in a 37°C incubator for 1 hour. Then, 350 uL of a wash buffer was added to each well. For one minute, the well was submerged. Each well was emptied or left for evaporation on a clean absorbent surface. It was repeated three times. Washed testing strips were given to prevent desiccation. The human resources administration conjugate working solution was 100 microliters per well. For 30 minutes, the plate was covered and set up in an incubator at 37 degrees Celsius. Decant each well and rinse five times. Each well received 90 μL Substrate Reagent. Following a new sealer, the plate was placed in a 37°C incubator for 15 minutes. Avoiding light exposure protected the plate. Prior measuring the optical density, the Microplate Reader was warmed for 15 minutes. Wells received fifty microliters of Stopping Solution. A micro-plate reader with a wavelength of 450 nm measured each well's optical density (OD value).

Interleukin 6 (Hang 2020 *et al.*, 2020)

Use of the reagent from elabscience was per manufacturer's instructions.

Principle of testing

ELISA kit uses Sandwich-ELISA. Each micro ELISA plate in this kit is pre-coated with a Human IL-4 antibody. Samples, or reference materials, are inserted in the micro ELISA plate's tiny holes accompanied by the matching antibody. After that, a biotinylated antibody that acts as a detection that targets Human IL-4 and the Avidin-Horseradish Peroxidase (HRP) conjugate are sequentially applied to each microplate well and incubated. Unpaid workers are totally removed. The solution is then added to all of them well. Biotinylated detecting antibody, Avidin-HRP conjugate, and Human IL-4 wells are blue. Stop solution stops the enzyme reaction, turning the solution yellow. To measure optical density (OD), spectrophotometry is used at a wavelengths of 450 ± 2 nm.

The Testing Principle

ELISA kit uses Sandwich-ELISA. This kit includes a Human IL-6 antibody-coated micro ELISA plate. Reference materials are inserted in micro ELISA plate wells with the appropriate antibody. Then, a biotinylated detection antibody that binds to Human IL-6 and Avidin-Horseradish Peroxidase (HRP) are added to each well of the micro plate and incubated. Water moves broken parts. Each well receives substrate solution. Wells with Human IL-6, biotinylated antibody, and Avidin-HRP conjugate are blue. Stop solution ends the enzyme-substrate reaction, turning it yellow. The spectrophotometer measures the density of light (OD) at 450 ± 2 nm accurately.

3.16 Protocol for analyzing

We selected dilute standard, empty, and sample wells. One hundred microliters have been added to each standard, blank, or sample dilution and placed in the wells. The kit

included a sealer plate incubated at 37 degrees Celsius for 90 minutes. To avoid foaming, the solutions were carefully added to permeate the bottom of this micro ELSA plate without touching the inner wall. Without rinsing, each well was decanted. In each well, 100 microliters of Biotinylated Detection Antibodies functioning solution was added immediately. Following a new sealant, the plate was placed in an incubator at 37°C for 1 hour. Each well was decanted and 300 microliters of liquid wash buffer added. Well was submerged for 1 minute. Aspirating each well, emptying it, and drying it on immaculate absorbent paper. Three times, this process was repeated. The examination strips were cleaned to prevent desiccation. We added 100 microliters of HRM conjugate working solution to each well. The plate was then sealed with a new cover and set up in an incubator at a temperature of 37°C for 30 minutes. Five times, each well was decanted and rinsed. The Substrate Reagent order was 90 microliters per well. After applying new sealant, the plate was placed in a 37°C incubator for 15 minutes. Protecting the plate from light. After 15 minutes of preheating, the Microplate Reader measured optical density. Stop Solution added 50 microliters to each well. Our micro-plate reader adjusted at 450 nm detected each well's OD value simultaneously.

Interlukin -9 (Khalaf *et al.,* 2023)

3.17 Test concept

ELISA kit uses Sandwich-type technique. This kit includes a Human IL-9 antibody-coated micro ELISA plate. Reference materials—specimens—are blended with the appropriate antibody in the micro ELISA plate's tiny pores. In a sequential fashion, a biotinylated antibody that acts as a detection that targets Human IL-9 and the Avidin-Horseradish Peroxidase (HRP) combination are applied to each microplate well and

incubated. Water moves loose particles. Wells receive substrate solution. Only Humans IL-9, biotinylated detecting antibody, and Avidin-HRP conjugate do wells turn blue. The enzyme-substrate reaction is stopped by a stop solution, turning it yellow. The optical density, also known as OD, is measured at 450 ± 2 nm using the spectrophotometer.

3.18 Analytical procedure

Various water sources were chosen for the purpose of dilute standard, blank, and specimen. Each standard, blank, or sample dilution received 100 microliters and was placed in the properly wells. Kits incubated the sealer plate at 37 degrees Celsius for 90 minutes. Solutions were gently introduced to enter the bottom underneath the microscopic ELSA plate without touching the inner wall to reduce foam formation. Wells were decanted without rinsing. For each well, 100 microliters of Biotinylated Detecting Antibody working solution was added immediately. The plate was then renewed with sealant and placed in an incubator at 37°C for 1 hour. Wells were decanted and 350 microliters of liquid wash buffer added. Well submerged for 1 minute. Extract, empty, and let each well's contents evaporate on a clean absorbent sheet. Three times, this phase played out. To prevent desiccation, the assessed strips were available after cleaning. Every well received 100 microliters of HRP conjugate-containing working solution. Using a fresh cover, the plate was sealed and set up in an incubator at a temperature of 37°C for 30 minutes. Descanting each well and rinsing five times followed. Per well, Ninety microliters of Substrates Reagent was added. New sealant was applied to the prepared plate, which was incubated at 37°C for 15 minutes. Light wasn't allowed on the plate. Before measuring OD, its Microplate Reader was warmed for 15 minutes. 50

microliters of Stop Solution per well. Each well's OD value was measured simultaneously using a 450 nm-calibrated micro-plate reader.

Interleukin 10 (Kim et al., 2021)

Using elabscience's reagent per manufacturer's instructions.

Test Principle

Test principle for the kit is Sandwich enzyme immunoassay. This kit includes an IL10-binding antibody pre-coated on a microtiter plate. In selected water sources of a microtiter plate, standards or samples are added, followed by 3 biotin-conjugated IL10-targeting antibodies. Each microwell plate well receives Avidin-Horseradish The enzyme per oxide (HRP) conjugate and is incubated. Only Interleukin 10 (i L10), biotin-conjugated antibodies, and and enzyme-conjugated Avidin wells will change color after adding TMB substrate solution. To determine the amount, the reaction between the enzyme and the substrate is stopped with a solution of water or sulphuric acid solution, and the color change is determined using spectrophotometry at 450nm ±10nm. By comparing the samples' optical density (OD) to the standard curve, IL10 concentration is measured.

Assay Procedure

diluted standard, blank, or sample require 7 reference standard wells and 1 blank well. Each well received 100 µL of Standardized Working Solution. Plate sealer then coated it. Afterward, the solution was incubated at 37°C for 80 minutes. Every well was drained. A 200 µl wash solution was added to each well and left to settle for 1-2 minutes before suctioning and washing. Each well was totally drained before securely connecting the plate bottom to liquid-absorbing paper. Three times, the solution was rinsed. Any residual Wash Buffer was aspirated and decanted after the last wash. Inverted dish on absorbent

paper to remove excess liquid. Each well has 100 microliters of Biotinylated Antibody Working Solution. A plate sealer closed the wells, which were then incubated at 37°C for 50 minutes. As usual, aspiration washing was done three times. The wells received 100 microliters containing Streptavidin-HRP Work Solution. For 50 minutes, the wells were sealed with a piece of sealer and incubated at 37 degrees Celsius. As usual, aspiration washing was done five times. A fresh Plate sealer sealed each well after 90 microliters of TMB Substrates Solution is added. Next, the plate put in a 37°C incubator for 20 minutes. When TMB Substrates Solution was added, the solution turned blue. OD was measured after 15 minutes of Microplate Reader preheating. In each well, 50 microliters diluted Stop Reagents were applied. When Stop Reagent was added, the liquid turned yellow. Gently tapping the dish edge swirled the liquid. Eliminate any water or fingerprints from the plate's bottom and check for liquid bubbles. At 450 nm, this microplate reader quickly assessed the sample.

Interferon Gamma (Mahittikorn *et al.*, 2020)

Using elabscience's reagent per manufacturer's instructions.

Test Principle

This kit uses Sandwich enzyme immunoassay. This kit includes a microtiter plate coated with an antibody that targets interferon gamma (IFN-γ). Standards or samples and 3 biotin-conjugated autoantibodies that specifically bind at Interleukin 10 (IL10) are placed in the microtiter plate wells. Avidin-Horseradish Peroxidase (HRP) conjugated is then applied to each microwell plate well and incubated. Adding TMB substrate solution,

especially in wells with IFN-γ, biotin-conjugated antibody, or enzyme-conjugated Avidin will cause a dramatic color change. Overall enzyme-substrate reaction is halted by adding sulphuric acid suspension, and the color change is quantified using spectrophotometry at 450nm ±10nm. The amount of individual interferon Gamma (IFN-γ) in samples is evaluated by comparing their optical density (OD) with the standard curve.

3.19 Experimental Method

Diluting standard, blank, and sample require 7 reservoir for the benchmark and 1 well for the blank. Each well received one hundred microliters of Regular Working Solution. This was then covered with Plate sealer. The solution was then incubated at 37°C for 80 minutes. The liquid was then taken from each well. After aspirating and washing approximately 200 µl of an additional washing solution per well, the solution was incubated for 1-2 minutes. The plate was securely fastened to absorbent paper to drain all the liquid from the wells. The solution was thoroughly rinsed three times. Aspiration and decanting removed anything remaining Wash Buffer after the last wash. Turning the dish over and pressing it against absorbent paper. Each well received a hundred microliters or Biotinylated Antibody Working Solution. After that, the surface of the plate cover was placed on the top of each of the wells and the mixture was incubated at 37°C for 50 minutes. As before, aspiration washing was done three times. One hundred microliters Streptavidin-HRP Solution was applied to each well. The wells were sealed utilizing a layer of sealer and incubated at 37°C for 50 minutes. As before, aspiration washing was done five times. After adding Ninety microliters of TMB Substrates Solution, each well was sealed with a fresh Plate sealer. After that, the plate was incubated at 37°C for 20 minutes. After adding TMB Substrates Solution, the liquid turned blue. This Microplate

Reader was warmed for 15 minutes before measuring OD. Each well received 50 microliters of Stop Reagent. Addition of Stopping Reagent turned the liquid yellow. The dish edge was gently tapped to swirl the liquid. Completely eliminate No air bubbles were found on the liquid after removing the water droplets and fingerprint off the plate's underside. After that, the Microplate reader quickly assessed the sample at 450 nm.

LT Beta (Ullah *et al.*, 2022)

The reagent was purchased from elabscience and used as directed.

Test Principle

Sandwich enzymes immunoassay is the kit's test principle. This package includes a microtiter plate pre-coated using an antibody that preferentially binds to LT beta. Standards or samples are placed in predetermined wells in a microtiter plate, and 3 biotin-conjugated IL10-targeting antibodies are added. After that, each microwell plate well receives Avidin-Horseradish The enzyme per oxide (HRP) conjugate and is incubated. Only wells with the combination of tumor necrosis factor (LT beta), biotin-conjugated antibodies, and enzyme-conjugated Avidin change color when the TMB substrate solution is introduced. To end the enzyme-substrate reaction, a sulphuric acid solution is added and the color change is detected using quantitative spectrophotometry at 450nm ± 10nm. Comparing the samples' optical density (OD) to the standard curve determines their LT beta content.

Protocol for Analysis By preparing 7 baseline wells and 1 blank well, water sources are allocated for the diluted expected, blank, and specimen. The appropriate wells received a hundred microliters of Standardized Working Solution. After that, a plate sealer sealed it.

The solution was then incubated at 37°C for 80 minutes. Each well's fluid was extracted. Post-aspiration, the solution was rinsed with 200 milliliters of 1 x wash solutions in each well and incubated for 1-2 minutes. Attaching the plate with absorbent paper totally drained all wells. The solution was thoroughly rinsed three times. Suction and pouring eliminated any residual Wash Buffer following the previous washing phase. The surface of the plate was turned upside down and secured on absorbent paper. Each well received 100 microliters of Biotinylated Antibody Working Solution. After enclosing the wells with the plate sealer, they were incubated at 37°C for 50 minutes. As before, aspiration washing was done three times. Each well received a hundred milliliters of Streptavidin-HRP Standard Solution. After closing the wells with a plate sealer, they were incubated at 37°C for 50 minutes. As before, aspiration washing was done five times. After adding Ninety micro Liters of TMB The substrate Solution, each well was sealed with a fresh Plate sealer. The plate was then incubated at 37°C for 20 minutes. TMB Substrates Solution turned the liquid blue. Before measuring the optical density, the Microplate Reader was warmed for 15 minutes. After that, each well received 50 microliters of Stopping Reagent. Introduction of Stop Reagent turned the liquid yellow. The dish edge was gently tapped to swirl the liquid. No air bubbles on the liquid confirmed that the bottom of the plate was clean of water and fingerprints. After that, this microplate reader quickly assessed the sample at 450 nm.

IgG and IgM (Lachenauer *et al.*, 200)

The spectrophotometric method was used to analyze IgG and IgM, following manufacturer instructions.

Tes Procedure

A dilution of protein standard from the the kit manufacturer was done using IgG/IgM constituent in a test tubes. Ten microliters of the diluted IgG/IgM standard were dispensed into a tube and labeled appropriately. Three hundred of IgG/IgM stabilizing solution was then added and incubated for 15 minutes at room temperature. 200 microliters of colour developer was then added and mixed. The absorbance of the solution was then read at 420nm after 10 minutes of incubation. Samples was treated same as the the standard. The concentrations of the IgG/IgM in the samples were extrapolated using the calibration curve.

DNA Damage Marker 8-hydroxy-2-deoxyguanosine (8OHDG) (Soini *et al.,* 2011)

The reagent was purchased from elabscience and used as directed.

Test Principle

Sandwiches enzyme immunoassay is used in these equipment. This kit includes a microtiter plate pre-coated with an antibody that preferentially recognizes tumor necrosis factor (LT beta). After filling predetermined wells within a microtiter plate with benchmarks or samples, 3 biotin-conjugated autoantibodies that preferentially target 8OHDG are added. Avidin-Horseradish The enzyme per oxide (HRP) conjugated is then applied to each microwell plate well and incubated. Only wells containing 8OHDG, biotin-conjugated antibody, or enzyme-conjugated Avidin will change color when the substrate for the TMB solution is introduced. To end the enzyme-substrate reaction, a

sulphuric acid solution is added and the color change is detected using quantitative spectrophotometry at 450nm ±10nm. The samples' optical density (OD) is compared to a standard curve to estimate their 8OHDG content.

Procedure for Assaying

To prepare dilute standard, blank, and sample, use 7 standard wells and 1 blank well. Each well received a hundred microliters or Standard Working Solution. After that, the plate's sealer covered it. The solution was then incubated at 37°C for 80 minutes. Each well's fluid was extracted. Take a powerful breath. After aspirating the solution, 200 µl of 1 x Wash Solution was added to each well and left to settle for 1-2 minutes. Comfortably connecting the stainless steel plate with absorbent paper emptied each well of all liquid that remained. The solution was properly cleaned three times. Aspiration and decanting removed whatever remaining Wash Buffer after the last washing process. Turning the dish over and pressing it against absorbent paper. Each well received 100 microliters or Biotinylated Antibodies Working Solution. The plate sealer was placed over the wells, and the samples were incubated at 37°C for 50 minutes. As before, aspiration washing was done three times. Each well received a hundred microliters or Streptavidin-HRP Working Solution. After sealing with a plate sealer, the wells were incubated at 37°C for 50 minutes. As before, aspiration washing was done five times. After adding 90 microliters of TMB Substrates Solution, each well was sealed with a fresh Plate sealer. The plate was then incubated at 37°C for 20 minutes. After adding TMB Substrates Solution, the liquid turned blue. A Microplate Reader was warmed 15 minutes before OD measurement. Next, fifty milliliters of Stopping Reagent was applied to each well. The addition of Stopping Reagent turned the liquid yellow. The dish edge was gently tapped

to swirl the liquid. No air bubbles were seen on the liquid after the plate's underside was cleaned of water and fingerprints. After that, this microplate reader quickly assessed the sample at 450 nm.

3.19 Data analysis

Data averages and variability were shown. ANOVA was used to compare several groups, whereas the t-test with independent samples was used to compare two groups. Group differences were assessed using an LSD post-hoc multiple comparing test. A statistically significant level of $p < 0.05$ was used to evaluate statistical significance. Version 25.0 of IBM/SPSS was used for statistical analysis.

CHAPTER FOUR

RESULTS

Table 4.1 displays the sociodemographic features and lifestyles of those who have been exposed to petroleum products, as well as those who have not been exposed (controls). Control group had 68% males, automotive mechanics group had 100% males, tanker drivers group had 100% males, and NNPC personnel group had 100% males. Most study participants were 40 or older. In particular, the control group had 76% 40-year-olds, fuel attendants 96%, automobile mechanics 68%, and tanker drivers 54%. The majority of NNPC workers, 92%, were aged 40–59. Most of the control subjects (54%) and petrol attendants (82%) were single, while the majority of the auto-mechanics (62%), tanker drivers (74%) and NNPC staff (100%) were married. A greater percentage of the control subjects (56%) and petrol attendants (82%) had no children, while the majority of the auto-mechanics (52%), tanker drivers (70%) and NNPC staff (100%) indicated a high level of parity (≥5 children). Majority of the married participants (control, 47.8%; petrol attendants, 33.3%; tanker drivers, 43.2% 32.3%), have spent 2 years in marriage. Most of the married auto-mobile mechanics (41.9%) and NNPC staff (44%) have spent 4 years in marriage. A greater percentage (74%) of the control subjects had BSc degree, while majority of the petrol attendants (84%), mechanics (94%), tanker drivers (88%) had secondary school education, while most of the NNPC staff (66%) had GRA. Majority of the control, 82%; petrol attendants, 70%; NNPC staff (60%) were Christians, while most of the tanker drivers were Muslims (94%). The participants were predominantly non-smokers, non-alcoholics and do not take hard drugs. All the petrol attendants that smoke reported they take 1 stick of cigarette per day, 66.7% of the smoking mechanics reported they take not more than 2 sticks per day; all the smoking tanker drivers and NNPC staff

indicated they take more than 2 stick per day. Majority of those who drink alcohol across the study population, reported they take just one bottle of alcoholic beverage per day.

Table 4.1 Sociodemographic Characteristics and Lifestyles of Subjects Exposed to Petroleum Products and the Unexposed Controls

Characteristics		Control N (%)	Petrol Attendants N (%)	Automobile Mechanics N (%)	Tanker Drivers N (%)	NNPC Staff N (%)
Sex	Females	16 (32.0)	25 (50.0)	0 (0)	0 (0)	0 (0)
	Males	34 (68.0)	25 (50.0)	50 (100)	50 (100)	50(100)
Age Group	<40 years	38 (76.0)	48 (96.0)	34 (68)	27 (54)	1 (2)
	40-59 years	12 (24.0)	2 (4.0)	15 (30)	22 (44)	46 (92)
	≥60 years	0 (0)	0 (0)	1 (2)	1 (2)	3 (6)
Marital Status	Married	23 (46.0)	9 (18.0)	31 (62)	37 (74)	50(100)
	Single	27 (54.0)	41 (82.0)	19 (38)	13 (26)	0 (0)
Parity	None	28 (56.0)	41 (82.0)	19 (38)	13 (26)	0 (0)
	Low	4 (8.0)	3 (6.0)	2 (4)	0 (0)	0 (0)
	Moderate	1 (2.0)	1 (2.0)	3 (6)	2 (4)	0 (0)
	High	17 (34.0)	5 (10.0)	26 (52)	35 (70)	50(100)
Years of Marriage	1 year	5 (21.7)	1 (11.1)	3 (9.7)	5(13.5)	3 (6)
	2 years	11 (47.8)	3 (33.3)	10 (32.3)	16(43.2)	8 (16)
	3 years	7 (30.4)	2 (22.2)	4 (12.9)	2 (5.4)	12 (24)
	4 years	0 (0.0)	3 (33.3)	13 (41.9)	11(29.7)	22 (44)
	≥5 years	0 (0)	0 (0)	1 (3.2)	3 (8.1)	5 (10)
Educational Qualification	B.Sc	37 (74.0)	0 (0.0)	0 (0)	0 (0)	2 (4)
	GRA	1 (2.0)	1 (2.0)	0 (0)	0 (0)	33 (66)
	NCE	0 (0.0)	1 (2.0)	0 (0)	0 (0)	0 (0)
	ND	2 (4.0)	6 (12.0)	0 (0)	0 (0)	1 (2)
	SEC	10 (20.0)	42 (84.0)	47 (94)	44 (88)	14 (28)
	Primary	0 (0)	0 (0)	3 (6)	6 (12)	0 (0)
Religion	Christianity	41 (82.0)	35 (70.0)	25 (50)	3 (6)	30 (60)
	Islam	9 (18.0)	15 (30.0)	25 (50)	47 (94)	20 (40)
Smoking Habit	No	50(100.0)	48 (96.0)	44 (88)	48 (96)	48 (96)
	Yes	0 (0.0)	2 (4.0)	6 (12)	2 (4)	2 (4)
Number of Sticks per Day	1 stick/day	0 (0.0)	2 (100)	2 (33.3)	0 (0)	0 (0)
	2 Sticks/day	0 (0)	0 (0)	4 (66.7)	0 (0)	0 (0)
	>2 Sticks/day	0 (0)	0 (0)	0 (0)	2 (100)	2 (100)
Drinking of Alcohol	No	50(100.0)	46 (92.0)	38 (76)	46 (92)	42 (84)
	Yes	0 (0.0)	4 (8.0)	12 (24)	4 (8)	8 (16)

Number of Bottles Per Day	1 bottle/day	0 (0.0)	4 (100)	6 (50)	2 (50)	5 (63)
	2 bottles/day	0 (0)	0 (0)	5 (41.7)	2 (50)	2 (25)
	5 bottles/day	0 (0)	0 (0)	1 (8.3)	0 (0)	1 (12)
Use of Hard Drugs	No	50(100.0)	50 (100.0)	49 (98)	49 (98)	49 (98)
	Yes	0 (0)	0 (0)	1 (2)	1 (2)	1 (2)

Table 4.2 displays the work-related attributes of individuals who were exposed to petroleum products, as well as their control groupThe research shows that 48% of the surveyed individuals, 94% of petroleum staff members, and 76% of NNPC officials had 1-5, 6-10, and 11-15 years of work experience. A higher proportion of the control group (96%), petrol attendants (58%), automotive mechanics (44%), and NNPC staff (82%) have a work duration of ≤10 hours per day, whereas every one of the tanker drivers (100%) reported working for more than 10 hours everyday. The majority of the control group (100%), petroleum attendants (56%), and NNPC staff (84%) expressed awareness regarding the impacts of petroleum products. Conversely, a majority of auto mechanics (54%) or tanker drivers (64%) reported lacking awareness regarding the impacts of petroleum products.

Table 4.2 Work Related Characteristics of Subjects Exposed to Petroleum Products and their Control

Characteristics		**Control**	**Petrol Attendants**	**Automobile Mechanics**	**Tanker Drivers**	**NNPC Staff**
		N (%)	**N (%)**	**N (%)**	**N (%)**	**N (%)**
Years of working experience	1 – 5	24 (48)	47 (94)	17 (34)	10 (20)	3 (6)
	6 – 10	18 (36)	3 (6)	16 (32)	25 (50)	9 (18)
	11 – 15	8 (16)	0 (0)	17 (34)	15 (30)	38(76)
Working hours/day	≤10	48 (96)	29 (58)	22 (44)	0 (0)	41(82)
	>10	2 (4)	21 (42)	28 (56)	50(100)	9 (18)
Knowledge of petroleum products effects	No	0 (0)	22 (44)	27 (54)	32 (64)	8 (16)
	Yes	50(100)	28 (56)	23 (46)	18 (36)	42(84)
Types of	None	50(100)	15 (30)	14 (28)	9 (18)	12(24)
	Difficulty in	0 (0)	2 (4)	0 (0)	0 (0)	16(32)

petrol	Breathing					
product	Dizziness	0 (0)	1 (2)	1 (2)	0 (0)	0 (0)
effects	Eye Irritation	0 (0)	3 (6)	1 (2)	2 (4)	7 (14)
experienced	Nasal Irritation	0 (0)	1 (2)	0 (0)	2 (4)	0 (0)
	Skin Irritation	0 (0)	27 (54)	18 (36)	36 (72)	13(26)
	Others	0 (0)	1 (2)	16 (32)	1 (2)	2 (4)
Measures	Medical	0 (0)	11 (22)	3 (6)	0 (0)	23(46)
taken to	attention					
treat effects	Disappearance of Symptom	0 (0)	24 (48)	33 (66)	24 (48)	16(32)
	None	50(100)	15 (30)	14 (28)	26 (52)	11(22)
Routes of	Eyes	0 (0)	3 (6)	3 (6)	1 (2)	4 (8)
exposure to	Nostril	0 (0)	10 (20)	8 (16)	2 (4)	21(42)
petroleum	Skin	0 (0)	23 (46)	31 (62)	22 (44)	20(40)
products	None	50(100)	14 (28)	8 (16)	25 (50)	5 (10)
Sucking	No	50(100)	48 (96)	13 (26)	37 (74)	39(78)
petrol with	Yes	0 (0)	2 (4)	37 (74)	13 (26)	11(22)
the mouth						
Washing	No	50(100)	43 (86)	9 (18)	22 (44)	34(68)
hands with	Yes	0 (0)	7 (14)	41 (82)	28 (56)	16(32)
petrol						
Knowledge	No	12 (24)	21 (42)	13 (26)	1 (2)	3 (6)
of PPE	Yes	38 (76)	29 (58)	37 (74)	49 (98)	47(94)
Use of PPE	No	12 (24)	30 (60)	16 (32)	1 (2)	5 (10)
	Yes	38 (76)	20 (40)	34 (68)	49 (98)	45(90)
Types of	Boot	-	2 (10)	0 (0)	0 (0)	10(22.2)
PPE used	Facial Mask	-	8 (40)	0 (0)	1 (2)	3 (6.7)
	Helmet	-	0 (0)	0 (0)	43(85.7)	4 (8.9)
	Hand Gloves	-	9 (45)	1 (3)	0 (0)	4 (8.9)
	Coverall	-	1 (5)	33 (97)	6 (12.3)	24(53.3)

Table 4.3 compares the blood levels of petrol attendants and unexposed controls for benzene and its metabolites. Compared to the control group, petrol attendants had higher average phenol and styrene levels ($p = 0.001$). Petrol attendants had much higher levels in their blood including butanoic acid, hydrocarbon chloride, among others o-xylene, the compound benzoic acid, benzene, benzene dimethyl, p-xylene, or ethylene benzene ($p < 0.001$). The petrol attendants had a significantly higher average naphthalene level than the control group ($p=0.042$).

Table 4.3 Petrol attendant blood samples compared to unexposed controls for benzene and its derivatives.

Variables (ppm)	Groups	Number of Subjects	Mean	Std. Deviation	T - Statistics	P – Value
Phenol	Control	50	0.18	0.14	-3.44	0.001
	Petrol Attendant	50	0.26	0.08		
Styrene	Control	50	0.20	0.19	-3.54	0.001
	Petrol Attendant	50	0.31	0.07		
Butanoic Acid	Control	50	0.32	0.26	-7.02	0.000
	Petrol Attendant	50	0.81	0.42		
Benzene	Control	50	1.00	0.75	-10.50	0.000
	Petrol Attendant	50	2.45	0.60		
Benzene Chloride	Control	50	0.48	0.31	-17.98	0.000
	Petrol Attendant	50	1.80	0.40		
O – Xylene	Control	50	0.37	0.09	-12.14	0.000
	Petrol Attendant	50	0.86	0.27		
Benzoic Acid	Control	50	0.35	0.17	-22.37	0.000
	Petrol Attendant	50	1.38	0.27		
Toluene	Control	50	1.44	0.75	-16.77	0.000
	Petrol Attendant	50	3.69	0.57		
Benzene Dimethyl	Control	50	0.29	0.12	-15.32	0.000
	Petrol Attendant	50	0.97	0.28		
P – Xylene	Control	50	0.39	0.21	-10.87	0.000
	Petrol Attendant	50	0.97	0.30		
Naphthalene	Control	50	0.18	0.06	-2.05	0.042
	Petrol Attendant	50	0.25	0.24		
Ethylene Benzene	Control	50	2.18	0.46	-7.36	0.000
	Petrol Attendant	50	2.96	0.59		

Table 4 shows car mechanics' blood and urine benzene and byproduct levels compared to unexposed controls. Four The t-test for independent samples demonstrated a substantial ($p < 0.001$) rise in average the chemical st butanoic acids, benzene, benzoic acid, the aforementioned to the chemical the presence of dimethyl, p-xylene, or naphthalene levels among auto mechanics in comparison with the control group. Car mechanics had a statistically significant higher average o-xylene level than the control group ($p = 0.034$). The average ethylene benzene levels of auto mechanics were substantially lower than controls. No significant changes were found in phenolic and benzene chloride ($p = 0.363$ and 348).

Table 4.4 Automobile Mechanics' Blood Benzene and Derivative Concentrations Compared to Unexposed Controls

Variables (ppm)	Groups	Number of Subjects	Mean	Std. Deviation	T – Statistics	P – Value
Phenol	Control	50	0.18	0.14	0.91	0.363
	Mechanics	50	0.25	0.49		
Styrene	Control	50	0.20	0.19	-5.08	0.000
	Mechanics	50	0.41	0.21		
Butanoic Acid	Control	50	0.32	0.26	-6.65	0.000
	Mechanics	50	0.74	0.36		
Benzene	Control	50	1.00	0.75	-2.37	0.000
	Mechanics	50	1.53	1.37		
Benzene	Control	50	0.48	0.31	-0.94	0.348
Chloride	Mechanics	50	0.60	0.83		
O – Xylene	Control	50	0.37	0.09	-2.15	0.034
	Mechanics	50	0.68	1.01		
Benzoic Acid	Control	50	0.35	0.17	-5.75	0.000
	Mechanics	50	0.57	0.20		
Toluene	Control	50	1.44	0.75	-12.19	0.000

	Mechanics	50	3.79	1.12		
Benzene	Control	50	0.29	0.12	-9.75	0.000
Dimethyl	Mechanics	50	2.25	1.42		
P – Xylene	Control	50	0.39	0.21	-6.77	0.000
	Mechanics	50	1.78	1.43		
Naphthalene	Control	50	0.18	0.06	-4.51	0.000
	Mechanics	50	0.54	0.57		
Ethylene	Control	50	2.18	0.46	5.23	0.000
Benzene	Mechanics	50	1.04	1.46		

Tanker driver blood samples were tested for benzene and its compounds. A control group not exposed to these chemicals was compared to these levels. For findings, see Table 4.5. Compared to the control group, the mean levels of phenol, synthetic sty butanoic acidic substances, benzene chloride, among others o-xylene, benzoic acids, benzene dimethyl, p-xylene, or an auto mechanics chemical increased significantly ($p < 0.001$). The average amounts of benzene (0.646), toluene (0.266), and ethylene benzene (0.607) were similar.

Table 4.5 Tanker Drivers' Serum Benzene and Derivative Concentrations Compared to Unexposed Controls

Variables (ppm)	Groups	Number of Subjects	Mean	Std. Deviation	T – Statistics	P – Value
Phenol	Control	50	0.18	0.14	-19.49	0.000
	Tanker drivers	50	1.85	0.58		
Styrene	Control	50	0.20	0.19	-6.92	0.000
	Tanker drivers	50	0.43	0.13		
Butanoic Acid	Control	50	0.32	0.26	-6.90	0.000
	Tanker drivers	50	0.98	0.61		
Benzene	Control	50	1.00	0.75	0.46	0.646
	Tanker drivers	50	0.94	0.60		
Benzene	Control	50	0.48	0.31	-5.76	0.000
Chloride	Tanker drivers	50	0.85	0.31		
O – Xylene	Control	50	0.37	0.09	-14.89	0.000
	Tanker drivers	50	2.34	0.93		
Benzoic Acid	Control	50	0.35	0.17	-3.92	0.000
	Tanker drivers	50	0.79	0.76		
Toluene	Control	50	1.44	0.75	-1.12	0.266
	Tanker drivers	50	1.69	1.33		
Benzene	Control	50	0.29	0.12	-11.42	0.000
Dimethyl	Tanker drivers	50	2.85	1.58		
P – Xylene	Control	50	0.39	0.21	-12.63	0.000
	Tanker drivers	50	2.25	1.01		
Naphthalene	Control	50	0.18	0.06	-8.42	0.000

	Tanker drivers	50	1.13	0.79		
Ethylene	Control	50	2.18	0.46	0.51	0.607
Benzene	Tanker drivers	50	2.04	1.78		

NNPC employees' blood samples were tested for benzene and related constituents. The amounts of these chemicals were then compared to a control group without exposure. Table 4.6 shows results. Results show NNPC personnel had considerably greater quantities of phenol, synthetic sty o-xylene, the acid benzoic, p-xylene, and naphthalene ($p < 0.001$) than the control group. Compared to NNPC personnel, control individuals had considerably higher amounts of the chemical and benzene chloride ($p < 0.001$). Butanoic acid and benzene dimethyl concentrations did not vary ($p = 0.883$ and 0.270, respectively).

Table 4.6 Comparing NNPC Staff Blood Samples for Benzene or Its Derivatives to Unexposed Controls

Variables (ppm)	Groups	Number of Subjects	Mean	Std. Deviation	T - Statistics	P – Value
Phenol	Control	50	0.18	0.14	-10.07	0.000
	NNPC Staff	50	0.41	0.06		
Styrene	Control	50	0.20	0.19	-14.04	0.000
	NNPC Staff	50	2.51	1.14		
Butanoic Acid	Control	50	0.32	0.26	-0.14	0.883
	NNPC Staff	50	0.33	0.20		
Benzene	Control	50	1.00	0.75	5.41	0.000
	NNPC Staff	50	0.42	0.04		
Benzene	Control	50	0.48	0.31	5.37	0.000
Chloride	NNPC Staff	50	0.24	0.04		
O – Xylene	Control	50	0.37	0.09	-5.38	0.000
	NNPC Staff	50	1.71	1.77		
Benzoic Acid	Control	50	0.35	0.17	-28.30	0.000
	NNPC Staff	50	1.78	0.31		
Toluene	Control	50	1.44	0.75	-14.43	0.000
	NNPC Staff	50	3.13	0.32		
Benzene	Control	50	0.29	0.12	1.10	0.270
Dimethyl	NNPC Staff	50	0.26	0.08		
P – Xylene	Control	50	0.39	0.21	-10.40	0.000

	NNPC Staff	50	0.72	0.07		
Naphthalene	Control	50	0.18	0.06	-15.63	0.000
	NNPC Staff	50	0.77	0.25		
Ethylene	Control	50	2.18	0.46	-21.72	0.000
Benzene	NNPC Staff	50	4.90	0.75		

Table 4.7 compares blood serum benzene and derivative levels between groups. The results of the Analysis for Variance (ANOVA) showed that tanker drivers had significantly higher phenol concentrations ($p < 0.001$) than petrol staff members, automotive technicians, and NNPC officials. NNPC workers had a considerably higher average phenol level ($p = 0.037$) than auto mechanics. Serum styrene levels in NNPC workforce were significantly higher ($p < 0.001$) than in petroleum attendants, auto mechanics, particularly tanker drivers. The average butanoic acid content was substantially higher in tanker driver ($p = 0.007$) as compared to petroleum attendants and NNPC staff ($p < 0.001$). The total amount of the chemical butanoic acid were substantially higher ($p < 0.001$) among petroleum attendants and car mechanics compared to NNPC staff. There were significant differences ($p < 0.001$) in median hydrocarbon concentrations among the experimental groups. The harshest mean value was 2.45 mg/m3 for petrol attendants, followed by auto mechanics at 1.53, tanker drivers at 0.94, and NNPC officials at 0.42. Benzene chloride levels differed greatly between experimental groups. The mean measurement of 1.80 mg/m3 for petrol attendants was highest, followed by tanker drivers (0.85 mg/m3), auto mechanics (0.60 mg/m3), and NNPC officials (0.24 mg/m Tanker drivers had considerably higher o-xylene levels ($p < 0.01$, $p < 0.001$) than petrol attendants, auto technicians, and NNPC workers. The NNPC staff had a considerably greater average ($p < 0.001$) than petroleum attendants and car mechanics. NNPC staff had the highest concentration of benzoic acid (1.78 mg/m3), followed by petroleum workers (1.38 mg/m3), tankers drivers (0.79 mg/m3), and vehicle mechanics (0.57 mg/m3). Auto technicians had considerably higher toluene levels than

tanker drivers ($p < 0.001$) or NNPC managers ($p < 0.01$). NNPC personnel had significantly higher values than fuel attendant ($p = 0.003$) or tankers operators ($p < 0.001$). Compared to petrol attendants, tanker drivers had significantly greater ($p < 0.001$) median toluene levels. The tanker drivers had the highest average benzene dimethyl concentration, at 2.85 mg/m3, followed by automotive mechanics without 2.25 mg/m3, petroleum attendant at 0.97 mg/m3, and the National Nuclear Power Corporation executives with 0.26 mg/m3. Compared to auto technicians, petroleum attendants, and NNPC officials, tanker drivers had considerably higher mean p-xylene levels ($p < 0.01$ or $p < 0.001$). Auto mechanics had a considerably greater average ($p < 0.001$) than petroleum attendants and NNPC employees. Tanker drivers had the highest average naphthalene level (1.13 mg/m3), next to the NNPC administrators (0.77 mg/m3), mechanics working on cars (0.54 mg/m3), and petrol attendants (0.25 mg/m3). NNPC workers showed the highest average ethylene benzene level (4.9 mg/m3), followed by fuel attendants (2.96 mg/m3), tankers personnel (2.04 mg/m3), and auto mechanics (1.04 mg/m3).

Table 4.7 Benzene and Derivative Mean Serum Concentrations Compared by Experimental Group

Variables (ppm)	Petrol Attendant	Auto Mechanics	Tanker Drivers	NNPC Staff	F-Stat	P – Value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD		
Phenol	0.26 ± 0.08	0.25 ± 0.49	1.85 ± 0.58	0.41 ± 0.06	199.8	<0.001
Styrene	0.31 ± 0.07	0.41 ± 0.21	0.43 ± 0.13	2.51 ± 1.14	163.4	<0.001

Butanoic Acid	0.81 ± 0.43	0.74 ± 0.36	0.98 ± 0.61	0.33 ± 0.20	20.6	<0.001
Benzene	2.45 ± 0.60	1.53 ± 1.37	0.94 ± 0.60	0.42 ± 0.04	57.7	<0.001
Benzene Chloride	1.80 ± 0.40	0.60 ± 0.83	0.85 ± 0.31	0.24 ± 0.04	93.3	<0.001
O-xylene	0.86 ± 0.27	0.68 ± 1.01	2.34 ± 0.93	1.71 ± 1.77	23.4	<0.001
Benzoic Acid	1.38 ± 0.27	0.57 ± 0.20	0.79 ± 0.76	1.78 ± 0.31	76.7	<0.001
Toluene	3.69 ± 0.57	3.79 ± 1.12	1.69 ± 1.33	3.13 ± 0.32	53.9	<0.001
Benzene Dimethyl	0.97 ± 0.28	2.25 ± 1.42	2.85 ± 1.58	0.26 ± 0.08	60.4	<0.001
P-xylene	0.97 ± 0.30	1.78 ± 1.43	2.25 ± 1.01	0.72 ± 0.07	31.6	<0.001
Naphthalene	0.25 ± 0.24	0.54 ± 0.57	1.13 ± 0.79	0.77 ± 0.25	25.3	<0.001
Ethylene Benzene	2.96 ± 0.59	1.04 ± 1.46	2.04 ±1.78	4.90 ± 0.75	86.2	<0.001

Table 4.8 shows bloodstream levels of oxidative stress-related indicators in petroleum-exposed gasoline attendants and the control group. Independent sample t-test showed that the control group had a higher mean glutathione peroxidase level (p = 0.013) than the petrol attendants. However, petrol attendants had considerably greater C reactive protein concentrations than the control group (p-value < 0.001). The standard deviation of the combined oxidant capability of the two groups was similar (p = 0.945).

Table 4.8 Petroleum Product-Exposed Petrol Attendants and Unexposed Controls' Serum Oxidative Stress Markers

Variables	Groups	N	Mean	Std. Deviation	T - Statistics	P – Value
Total Oxidant Capacity (U/mL)	Control	50	2.00	0.36	-.069	.945
	Petrol Attendant	50	2.00	0.46		
Glutathione Peroxidase (U/mL)	Control	50	11.36	20.38	2.542	.013
	Petrol Attendant	50	3.17	10.17		
C Reactive Protein (mg/l)	Control	50	5.38	3.15	-4.756	.000
	Petrol Attendant	50	11.26	8.14		

Table 4.9 shows oxidative stress marker values in petroleum-exposed auto-mechanics and controls. According to data, the comparison group had a considerably higher average total oxidant capacity ($p < 0.001$) than auto mechanics. The auto mechanics had a greater median C reactive protein ($p = 0.001$) than the control group. Auto mechanics and controls had similar mean glutathione peroxidase ($p = 0.337$).

Table 4.9 Petroleum-Exposed Auto-Mechanics and Unexposed Controls' Serum Oxidative Stress Markers

Variables	Groups	N	Mean	Std. Deviation	T - Statistics	P – Value
Total Oxidant Capacity (U/mL)	Control	50	2.00	0.36	3.749	0.000
	Mechanics	50	1.76	0.27		
Glutathione Peroxidase (U/mL)	Control	50	11.36	20.38	-0.965	0.337
	Mechanics	50	15.26	19.93		
C- Reactive Protein (mg/L)	Control	50	5.38	3.15	-3.430	0.001
	Mechanics	50	9.22	7.24		

Table 4.10 compares serum oxidative stress indicators in petroleum-exposed tanker drivers and controls. Tanker drivers had considerably higher mean total oxidant capacity than the control group. Comparisons across groups showed no significant differences in average peroxidase activity of glutathione (p = 0.321) or C activated protein (p = 0.182).

Table 4.10 Petroleum Product-Exposed Tanker Drivers and Unexposed Controls' Serum Oxidative Stress Markers

Variables	Groups	N	Mean	Std. Deviation	T - Statistics	P – Value
Total Oxidant Capacity (U/mL)	Control	50	2.00	0.36	-4.321	0.000
	Tanker Drivers	50	2.72	1.12		
Glutathione Peroxidase (U/mL)	Control	50	11.36	20.38	0.997	0.321
	Tanker Drivers	50	7.61	17.04		
C - Reactive Protein (mg/L)	Control	50	5.38	3.15	-1.345	0.182
	Tanker Drivers	50	6.37	4.12		

Table 4.11 displays the levels in the blood of oxidative stress markers in NNPC staff members who were exposed to petroleum products, as well as in the unexposed control group. The data suggests that the average total oxidant capacity was significantly greater ($p = 0.037$) within the untreated group compared to the NNPC workers. Conversely, NNPC employees had a significantly higher average glutathione peroxidase level than the control group ($p = 0.010$). Differences within C reactive protein quantities between groups were not significant ($p = 0.342$).

Table 4.11. NNPC Staff Exposure to Petrol Products and Unexposed Controls' Serum Oxidative Stress Markers

Variables	Groups	N	Mean	Std. Deviation	T - Statistics	P – Value
Total Oxidant Capacity (U/mL)	Control	50	2.00	0.36	2.11	0.037
	NNPC Staff	50	1.82	0.45		
Glutathione Peroxidase (U/mL)	Control	50	11.36	20.38	-2.61	0.010
	NNPC Staff	50	64.65	142.60		
C - Reactive Protein (mg/L)	Control	50	5.38	3.15	-0.95	0.342
	NNPC Staff	50	6.08	4.04		

In Table 4.12, the experiment groups' blood oxidative stress levels were compared. Analysis of variance showed an important rise in the overall oxidant capacity among tanker drivers compared to petroleum attendants, car mechanics, or the National Petroleum Corporation personnel ($p < 0.001$). In comparison to petrol attendants, automotive technicians, and tanker drivers, NNPC workers had significantly greater levels of glutathione peroxidase ($p < 0.001$). The average C reactive protein level of car mechanics was greater than hydrocarbon transporters ($p = 0.022$) or NNPC was the executives ($p = 0.012$). The CRP levels of petrol attendants were significantly greater ($p < 0.001$) than those of tanker drivers or NNPC officials.

Table 4.12. The Mean Serum Levels of Oxidative Stress Markers Compared among the Experimental Groups

Variables	Petrol Attendant	Auto Mechanics	Tanker Drivers	NNPC Staff	F-Stat	P – Value
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD		
Total Oxidant Capacity (U/mL)	2.00 ± 0.46	1.76 ± 0.27	2.72 ± 1.12	1.82 ± 0.45	22.13	<0.001
Glutathione Peroxidase (U/mL)	3.17 ±10.17	15.26 ± 19.93	7.61 ± 17.04	64.65 ± 14.2	7.65	<0.001
C - Reactive Protein (mg/L)	11.26 ± 8.14	9.22 ± 7.24	6.37 ± 4.12	6.08 ± 4.04	7.99	<0.001

Table 4.13 compares blood levels about inflammation-related and immunologic markers in petroleum-exposed gasoline attendants with unexposed controls. With p-values of less than 0.01 or 0.001, gasoline attendants exhibited significantly greater average concentrations of IL-Beta, IL-3, IL-6, IL-8, IL-10, or Human LT beta. Unlike gas attendants, the control group had higher average IL-4 levels. However, IL-9 (p = 0.143), gamma interferon (p = 0.173), immunoglobin G (p = 0.839), or immunoglobin M (p = 0.207) were not significantly different across groups.

Table 4.13. The Serum Levels of Inflammatory and Immunological Markers among the Petrol Attendants Exposed to Petroleum Products and the Unexposed Controls

Variables	Groups	N	Mean	Std. Deviation	T - Stat	P – Value
Interleukin1 Beta (pg/ml)	Control	50	31.57	10.88	-6.34	0.000
	Petrol Attendants	50	44.82	9.99		
Interleukin 3 (pg/ml)	Control	50	65.52	41.20	-5.96	0.000
	Petrol Attendants	50	165.01	110.56		
Interleukin 4 (pg/ml)	Control	50	4.97	1.11	6.32	0.000
	Petrol Attendants	50	3.32	1.47		
Interleukin 6 (pg/ml)	Control	50	3.92	1.90	-2.66	0.009
	Petrol Attendants	50	24.50	54.66		
Interleukin (pg/ml)	Control	50	12.76	14.38	-16.13	0.000
	Petrol Attendants	50	487.17	207.45		
Interleukin 9 (pg/ml)	Control	50	9.48	3.43	1.47	0.143
	Petrol Attendants	50	8.36	4.09		
Interleukin 10 (pg/ml)	Control	50	14.55	7.05	-5.04	0.000
	Petrol Attendants	50	22.94	9.39		
LT Beta (pg/ml)	Control	50	97.44	69.19	-8.13	0.000
	Petrol Attendants	50	244.90	107.90		
Interferon Gamma (pg/ml)	Control	50	4.81	2.92	-1.37	0.173
	Petrol Attendants	50	5.43	1.33		
Immunoglobin G (g/L)	Control	50	1.76	1.01	-0.20	0.839
	Petrol Attendants	50	1.81	1.34		

Immunoglobin M (g/L)	Control	50	4.62	5.18	1.27	0.207
	Petrol Attendants	50	3.41	4.22		

Table 4.14 shows blood levels about inflammation-related or immunological markers in car mechanics exposed to petroleum compounds and unexposed controls. Data suggest that automotive technicians had significantly higher levels on IL-Beta, the IL-3, IL-6, IL-8, IL-10, LT-Beta, gamma interferon, and immunoglobin M ($p < 0.05$, $p < 0.01$, or $p < 0.001$). Comparing the two groups, IL-4, also ($p = 0.165$), IL-9 ($p = 0.258$), and immunoglobin G ($p = 0.201$) were not significantly different.

Table 4.14. The Serum Levels of Inflammatory and Immunological Markers among Auto-Mechanics Exposed to Petroleum Products and the Unexposed Controls

Variables	Group	N	Mean	Std. Deviation	T – Stat	P – Value
Interleukin 1 Beta (pg/ml)	Control	50	31.57	10.88	-8.148	0.000
	Mechanics	50	47.81	8.95		
Interleukin 3 (pg/ml)	Control	50	65.52	41.20	-4.538	0.000
	Mechanics	50	166.14	151.28		
Interleukin 4 (pg/ml)	Control	50	4.97	1.11	1.398	0.165
	Mechanics	50	4.35	2.96		
Interleukin 6 ((mg/L)	Control	50	3.92	1.90	-4.816	0.000
	Mechanics	50	9.29	7.65		
Interleukin 8 (pg/ml)	Control	50	12.76	14.38	-11.90	0.000
	Mechanics	50	359.93	205.65		
Interleukin 9 (pg/ml)	Control	50	9.48	3.43	-1.137	0.258
	Mechanics	50	13.47	24.56		
Interleukin 10 (pg/ml)	Control	50	14.55	7.05	-2.874	0.005
	Mechanics	50	18.41	6.32		
LT Beta (pg/ml)	Control	50	97.44	69.19	-7.683	0.000
	Mechanics	50	211.24	78.62		
Interferon Gamma (pg/ml)	Control	50	4.81	2.92	-2.519	0.013
	Mechanics	50	5.99	1.61		
Immunoglobin G (g/L)	Control	50	1.76	1.01	-1.289	0.201
	Mechanics	50	2.02	1.00		
Immunoglobin M (g/L)	Control	50	4.57	5.22	-3.214	0.002
	Mechanics	50	11.66	14.70		

Tanker drivers who were subjected to petroleum products and a control group without exposure are shown in Table 4.15 for serum inflammatory and immunological markers. LT Beta, IL-9, and IL-4 levels differ significantly between the participants in the control groups and tanker drivers ($p < 0.05$, $p = 0.002$, $p < 0.001$). At the p-value lower than 0.001, 0.01, or 0.05, tanker drivers had significantly greater median amounts of the gamma interferon, which IL Beta, Interleukin-3, IL-6, IL-8, and IL-10 than the control group. For immunoglobin G ($p = 0.072$) and immunoglobin M ($p = 0.060$), no significant changes were found.

Table 4.15. The Serum Levels of Inflammatory and Immunological Markers among Tanker Drivers Exposed to Petroleum Products and the Unexposed Controls

Variables	Group	N	Mean	Std. Deviation	T – Stat	P – Value
Interleukin 1 Beta (pg/ml)	Control	50	31.57	10.88	-6.384	0.000
	Tanker Drivers	50	43.97	8.38		
Interleukin 3 (pg/ml)	Control	50	65.52	41.20	-2.212	0.029
	Tanker Drivers	50	94.91	84.46		
Interleukin 4 (pg/ml)	Control	50	4.97	1.11	5.055	0.000
	Tanker Drivers	50	3.26	2.13		
Interleukin 6 (pg/ml)	Control	50	3.92	1.90	-2.862	0.005
	Tanker Drivers	50	10.86	17.05		
Interleukin 8 (pg/ml)	Control	50	12.76	14.38	-3.869	0.000
	Tanker Drivers	50	93.91	147.60		
Interleukin 9 (pg/ml)	Control	50	9.48	3.43	3.142	0.002
	Tanker Drivers	50	6.89	4.71		
Interleukin 10 (pg/ml)	Control	50	14.55	7.05	-4.150	0.000
	Tanker Drivers	50	30.03	25.41		
LT Beta (pg/ml)	Control	50	97.44	69.19	1.983	0.049
	Tanker Drivers	50	74.68	42.40		
Interferon Gamma (pg/ml)	Control	50	4.81	2.92	-3.774	0.000
	Tanker Drivers	50	6.54	1.42		
Immunoglobin G (g/L)	Control	50	1.76	1.01	-1.816	0.072
	Tanker Drivers	50	5.53	1.46		

Immunoglobin (g/L)	M	Control	50	4.57	5.22	-1.906	0.060
		Tanker Drivers	50	20.74	59.76		

The personnel employed by the Nigerian National Petroleum Corporation (NNPC) who had been in contact with petroleum products were asked to analyze the average levels with immunological and inflammatory indicators in their blood and compare them to the levels of the control group who were not exposed (Table 4.16). In juxtaposition with the control group, the NNPC staff showed a notable rise in average levels from immunoglobin M, interferon Gamma, which Interleukin Beta, Interleukin-3, IL-4, IL-6, IL-8, and IL-10. These differences were statistically significant at $p < 0.001$, $p < 0.01$, or $p < 0.05$. Conversely, the untreated group had a greater mean level of IL-9 in comparison to the specific NNPC personnel ($p < 0.001$). Upon compared the two groups, there were no significant differences observed with respect to either immunoglobin G ($p = 0.339$) as well as average LT beta ($p = 0.149$).

Table 4.16. The Serum Levels of Inflammatory and Immunological Markers among the NNPC Staff Exposed to Petroleum Products and the Unexposed Controls

Variables	Groups	N	Mean	Std. Deviation	T – Stat	P – Value
Interleukin 1 Beta (pg/ml)	Control	50	31.57	10.88	-5.269	0.000
	NNPC Staff	50	40.63	5.45		
Interleukin 3 (pg/ml)	Control	50	65.52	41.20	-2.528	0.013
	NNPC Staff	50	97.52	79.48		
Interleukin 4 (pg/ml)	Control	50	4.97	1.11	-3.336	0.001
	NNPC Staff	50	8.03	6.38		
Interleukin 6 (mg/L)	Control	50	3.92	1.90	-2.874	0.005
	NNPC Staff	50	8.20	10.36		
Interleukin 8 (pg/ml)	Control	50	12.76	14.38	-5.032	0.000
	NNPC Staff	50	63.45	69.76		

Interleukin 9 (pg/ml)	Control	50	9.48	3.43	7.743	0.000
	NNPC Staff	50	5.15	1.96		
Interleukin 10 (pg/ml)	Control	50	14.55	7.05	-2.338	0.021
	NNPC Staff	50	17.91	7.29		
LT Beta (pg/ml)	Control	50	97.44	69.19	-1.454	0.149
	NNPC Staff	50	113.54	36.66		
Interferon Gamma (pg/ml)	Control	50	4.81	2.92	-3.977	0.000
	NNPC Staff	50	6.64	1.45		
Immunoglobin G (g/L)	Control	50	1.760	1.01	-0.962	0.339
	NNPC Staff	50	1.96	1.00		
Immunoglobin M (g/L)	Control	50	4.5	5.22	-9.793	0.000
	NNPC Staff	50	24.32	13.26		

Comparing the mean blood levels of immunological and inflammatory markers between the experimental groups is shown in Figure 4.17. When comparing auto technicians to petroleum attendant ($p = 0.023$) and NNPC employees (p 0.001), assessment of variance (ANOVA) revealed considerably (p 0.001) lower interleukin-1 beta. A tanker drivers or petrol attendants had considerably greater quantities of interleukin-1 Beta compared to NNPC personnel, with a statistical significance of $p < 0.05$. Auto technicians had statistically higher mean interleukin-3 levels than NNPC personnel ($p = 0.002$) or tanker drivers ($p = 0.001$). Petrol station attendants also displayed significantly higher IL-3 than tanker drivers ($p = 0.002$) and NNPC workers ($p = 0.003$). In comparison to petrol attendants, car mechanics, and tanker drivers, respectively, NNPC workforce had a considerably higher ($p < 0.001$) level of IL-4. When comparing the IL-6 of auto technicians, tanker drivers, and NNPC employees, the petrol attendants showed higher values ($p < 0.05$). In comparison to car technicians, tanker drivers, and NNPC employees, petrol attendants had significantly ($p < 0.001$) higher levels of interleukin 8. Along with petroleum drivers and NNPC employees, car technicians also had greater ($p < 0.001$) IL-8. Compared to gas station personnel ($p = 0.046$), tanker drivers ($p = 0.010$), and NNPC employees ($p = 0.001$), the mean IL-9 was substantially higher among auto mechanics. Tanker drivers had higher mean IL-10 than petroleum attendant ($p = 0.014$), auto technicians (p 0.001), and NNPC employees (p 0.001). Car mechanic ($p = 0.021$), tankers drivers ($p < 0.001$), and NNPC personnel ($p < 0.001$) all exhibited statistically lower mean LT-betas than petrol attendants. Along with the tankers drivers and NNPC

employees, the auto technicians' IL-8 was higher ($p < 0.001$) than theirs. The mean LT-beta value was higher ($p < 0.001$) when NNPC workers were compared to tanker drivers. In comparison to car mechanics ($p = 0.028$) and gas station attendants (p 0.001), NNPC employees had a considerably higher baseline interferon gamma level. Petrol attendants and tanker drivers had lower levels of interferon gamma ($p < 0.001$). In comparison to the NNPC employees ($p = 0.016$), auto technicians ($p = 0.019$), and gas station employees ($p = 0.013$), tanker drivers had a higher mean IgG level. The NNPC staff had a significantly higher average IgM level compared to vehicle technicians ($p = 0.046$) or the gas station personnel ($p = 0.001$). When examining the immunoglobulin M measurements obtained from the tanker drivers with the petrol attendants, it was found that the tanker drivers had significantly greater values ($p = 0.007$).

Table 4.17. The Mean Serum Levels of Inflammatory and Immunological Markers Compared among the Experimental Groups

Variables	Petrol Attendant	Auto Mechanics	Tanker Drivers	NNPC Staff	F-Stat	P – Value
Interleukin 1 Beta (pg/ml)	44.82 ± 9.99	47.81 ± 8.95	43.97 ± 8.38	40.63 ± 5.45	6.21	<0.001
Interleukin 3 (pg/ml)	165.01±110.5	166.14±15.12	94.91±84.46	97.52±79.48	6.61	<0.001
Interleukin 4 (pg/ml)	3.32 ± 1.47	4.35 ± 2.96	3.26 ± 2.13	8.03 ± 6.38	18.01	<0.001
Interleukin 6 (pg/ml)	24.50 ± 5.46	9.29 ± 7.65	10.86±17.05	8.20 ± 10.36	3.35	0.020
Interleukin 8 (pg/ml)	487.1 ± 207.4	359.9 ± 20.56	93.91±14.76	63.45 ± 6.97	75.9	<0.001
Interleukin 9 (pg/ml)	8.36 ± 4.09	13.47 ± 2.45	6.89 ± 4.71	5.15 ± 1.96	3.97	<0.001
Interleukin 10 (pg/ml)	22.94 ± 9.39	18.41 ± 6.32	30.03±25.41	17.91 ± 7.29	7.62	<0.001
LT Beta (pg/L)	244.9 ± 107.9	211.24 ± 78.6	74.68 ± 42.4	113.5 ± 36.6	61.2	<0.001
Interferon Gamma (pg/ml)	5.43 ± 1.33	5.99 ± 1.61	6.54 ± 1.42	6.64 ± 1.45	7.33	<0.001
Immunoglobin G (g/L)	1.81 ± 1.34	2.02 ± 1.0	5.53 ± 1.46	1.96 ± 1.0	2.97	0.033
Immunoglobin M (g/L)	3.41 ± 4.22	11.66 ± 14.70	20.74 ± 5.97	24.32±13.26	4.43	0.005

The average sample measurements of haemoglobin levels for the gasoline station employees who were subjected for petroleum products or those unexposed controls are presented in Table 4.18. A t-test conducted on separate samples demonstrated that regular untreated group exhibited significantly elevated granulocyte and basophil counts ($p = 0.004$ and p 0.001, respectively) comparing to untreated gas attendants. In contrast, the petrol attendants demonstrated markedly elevated mean MCHC ($p < 0.001$), lymphocytes ($p = 0.009$), or white blood cells ($p = 0.036$) compared to the control group. When comparing white blood cells (WBC), hematocrit (Hct), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), platelets, white blood cells, and monocytes across the two groups, no significant differences were observed ($p > 0.05$).

Table 4.18. The Serum Levels of Hematological Parameters among the Petrol Attendants Exposed to Petroleum Products and the Unexposed Controls

Variables	Groups	N	Mean	Std. Deviation	T – Stat	P – Value
WBC	Control	50	5.31	0.83	-0.696	0.488
(10^3/µL)	Petrol Attendant	50	5.48	1.55		
RBC	Control	50	4.71	0.65	-1.490	0.139
(10^6/µL)	Petrol Attendant	50	4.91	0.67		
HB (g/dL)	Control	50	13.26	0.95	0.000	1.000
	Petrol Attendant	50	13.26	1.34		
HCT (%)	Control	50	40.41	4.05	0.352	0.726
	Petrol Attendant	50	40.12	4.13		
MCV (µm^3)	Control	50	82.82	4.87	0.616	0.539
	Petrol Attendant	50	82.14	6.15		
MCH (pg)	Control	50	26.62	3.88	-0.896	0.372
	Petrol Attendant	50	27.20	2.42		
MCHC	Control	50	29.67	3.05	-7.365	0.000
(g/dL)	Petrol Attendant	50	33.09	1.20		
Platelet	Control	50	214.70	41.95	-0.810	0.420
(cells/µL)	Petrol Attendant	50	225.44	83.12		
Neutrophil	Control	50	45.68	5.49	2.909	0.004
(%)	Petrol Attendant	50	41.12	9.64		
Lymphocyte	Control	50	41.88	4.88	-2.670	0.009
(%)	Petrol Attendant	50	45.62	8.59		
Monocyte	Control	50	8.50	2.38	0.724	0.471
(%)	Petrol Attendant	50	8.18	1.96		
Eosinophil	Control	50	2.83	2.17	-2.123	0.036
(%)	Petrol Attendant	50	4.33	4.48		
Basophil (%)	Control	50	1.31	0.74	4.877	0.000
	Petrol Attendant	50	0.74	0.38		

Here are some examples of acronyms: The abbreviations MCV, MCH, MCHC, HB, and HCT represent the phrases mean cell volume, mean cell haemoglobin, average cell haemoglobin concentration, haemoglobin, and hematocrit, respectively**.**

Petroleum-exposed auto-mechanics or controls have haematological characteristic blood levels in Table 4.19. levels in Table 4.19. The team of automobile mechanics group had considerably lower WBC, platelets, and white blood, particularly neutrophil ($p < 0.001$) compared to the control group. Haemoglobin, MCV, MCHC, monocytes in particular lymphocytes, or eosinophils were significantly greater among those in the auto mechanics group compared to the untreated group ($p < 0.001$ or $p < 0.05$). RBC (p 0.357) and Hct ($p = 0.075$) were similar between groups.

4.19. The Serum Levels of Hematological Parameters among the Auto-Mechanics Exposed to Petroleum Products and the Unexposed Controls

White blood cells, blood cells with red blood cells, haemoglobin, or mean cell volume,

Variables	Groups	N	Mean	Std. Deviation	T – Stat	P – Value
WBC (10^3/µL)	Control	50	5.31	0.83	6.106	0.000
	Mechanics	50	3.93	1.35		
RBC (10^6/µL)	Control	50	4.71	0.65	-0.925	0.357
	Mechanics	50	4.84	0.72		
HB (g/dL)	Control	50	13.26	0.95	-4.820	0.000
	Mechanics	50	14.26	1.11		
HCT (%)	Control	50	40.41	4.05	-1.803	0.075
	Mechanics	50	42.36	6.50		
MCV (µm^3)	Control	50	82.82	4.87	-2.601	0.011
	Mechanics	50	87.13	10.65		
MCH (pg)	Control	50	26.62	3.88	-4.053	0.000
	Mechanics	50	29.90	4.20		
MCHC (g/dL)	Control	50	29.67	3.05	-5.592	0.000
	Mechanics	50	34.03	4.58		
Platelet	Control	50	214.70	41.95	3.087	0.003
(cells/µL)	Mechanics	50	181.03	64.92		
Neutrophil (%)	Control	50	45.68	5.49	7.581	0.000
	Mechanics	50	30.47	13.08		
Lymphocyte	Control	50	41.88	4.88	-4.767	0.000
(%)	Mechanics	50	51.62	13.59		
Monocyte (%)	Control	50	8.50	2.38	-2.633	0.010
	Mechanics	50	11.59	7.94		
Eosinophil (%)	Control	50	2.83	2.17	-2.330	0.022
	Mechanics	50	4.39	4.21		
Basophil (%)	Control	50	1.31	0.74	-2.938	0.004
	Mechanics	50	2.41	2.54		

and mean cell concentration of haemoglobin are acronyms.

Table 4.20 shows the standard deviations of serum haematological parameters of petroleum-exposed tanker drivers and controls. The groups had similar mean WBC, RBC, MCH, neutrophils, which as well as lymphocyte ($p = 0.814$, 0.493, 0.126, 0.053, and 0.415). Compared to the untreated group, tanker drivers had significantly higher levels for Hb, Hct, MCV, which is MCHC, monocytes, also eosinophils, and basophils ($p <$ 0.001 or $p < 0.01$ or $p <$ The control group had greater platelet counts than tanker drivers.

Table 4.20. The Serum Levels of Hematological Parameters among the Tanker Drivers Exposed to Petroleum Products and the Unexposed Controls

Variables	Groups	N	Mean	Std. Deviation	T – Stat	P – Value
WBC	Control	50	5.28	0.83	0.236	0.814
(10^3/µL)	Tanker Drivers	50	5.22	1.39		
RBC	Control	50	4.71	0.65	-0.688	0.493
(10^6/µL)	Tanker Drivers	50	4.82	0.84		
HB (g/dL)	Control	50	13.26	0.95	-3.079	0.003
	Tanker Drivers	50	13.99	1.35		
HCT (%)	Control	50	40.41	4.05	-2.353	0.021
	Tanker Drivers	50	42.74	5.72		
MCV (µm^3)	Control	50	82.82	4.87	-3.364	0.001
	Tanker Drivers	50	87.02	7.35		
MCH (pg)	Control	50	26.62	3.88	-1.543	0.126
	Tanker Drivers	50	35.21	39.20		
MCHC	Control	50	29.67	3.05	-4.850	0.000
(g/dL)	Tanker Drivers	50	32.08	1.73		
Platelet	Control	50	214.70	41.95	2.843	0.005
(cells/µL)	Tanker Drivers	50	190.68	42.81		
Neutrophil	Control	50	45.68	5.49	1.956	0.053
(%)	Tanker Drivers	50	42.72	9.18		
Lymphocyte	Control	50	41.88	4.88	0.818	0.415
(%)	Tanker Drivers	50	40.75	8.47		
Monocyte	Control	50	8.50	2.38	-3.735	0.000
(%)	Tanker Drivers	50	10.68	3.38		
Eosinophil	Control	50	2.83	2.17	-4.366	0.000
(%)	Tanker Drivers	50	4.96	2.66		
Basophil (%)	Control	50	1.31	0.74	-2.556	0.012
	Tanker Drivers	50	1.96	1.61		

White and red cells in the blood, haemoglobin, also pack cell volume, mean volume of cells, and concentration are acronyms.

The red blood cells, HB, Pack cell volume,, MCV, the MCH, MCHC Petroleum-exposed NNPC staff and controls are compared in Table 4.21 for haematological parameters. Average WBC, MCV, platelets, neutrophils, and lymphocytes did not change ($p = 0.479$, 0.577, 0.106, 0.524, and 0.01).

Table 4.21. The Serum Levels of Hematological Parameters among the NNPC Staff Exposed to Petroleum Products and the Unexposed Controls

Variables	Groups	N	Mean	Std. Deviation	T – Stat	P – Value
WBC	Control	50	5.31	0.83	0.710	0.479
	NNPC Staff	50	5.12	1.67		
RBC (10^6/µL)	Control	50	4.71	0.65	-3.755	0.000
	NNPC Staff	50	5.16	0.51		
HB (g/dL)	Control	50	13.26	0.95	-6.176	0.000
	NNPC Staff	50	14.43	0.93		
HCT (%)	Control	50	40.41	4.05	-4.264	0.000
	NNPC Staff	50	43.49	3.12		
MCV (µm^3)	Control	50	82.82	4.87	-0.560	0.577
	NNPC Staff	50	83.66	9.39		
MCH (pg)	Control	50	26.62	3.88	-2.331	0.022
	NNPC Staff	50	28.12	2.37		
MCHC (g/dL)	Control	50	29.67	3.05	-7.556	0.000
	NNPC Staff	50	33.22	1.30		
Platelet (cells/µL)	Control	50	214.70	41.95	-1.631	0.106
	NNPC Staff	50	229.60	48.65		
Neutrophil (%)	Control	50	45.68	5.49	0.639	0.524
	NNPC Staff	50	44.57	11.00		
Lymphocyte (%)	Control	50	41.88	4.88	-0.218	0.828
	NNPC Staff	50	42.23	10.08		
Monocyte (%)	Control	50	8.50	2.38	-1.784	0.077
	NNPC Staff	50	9.31	2.16		
Eosinophil (%)	Control	50	2.83	2.17	0.186	0.853
	NNPC Staff	50	2.75	1.90		
Basophil (%)	Control	50	1.31	0.74	5.185	0.000
	NNPC Staff	50	0.71	0.34		

Table 4.22 shows the outcome of the experiment groups' average serum haematological parameters. Auto technicians had significantly lower WBC ($p < 0.001$) when compared with petrol staff members, tankers drivers, or NNPC officials. RBC was higher in NNPC staff than automobile employees ($p = 0.025$) or tanker operators ($p = 0.016$). Petroleum attendants experienced lower mean Hb levels than car engineers ($p < 0.001$), tankers drivers ($p = 0.003$), or The National Petroleum Corporation (executives ($p < 0.001$). Petrol personnel had considerably lower mean mean Hct compared to car mechanics ($p = 0.028$), tankers drivers ($p = 0.010$), or NNPC officials ($p < 0.001$).

Car mechanic and tanker drivers had a considerably higher mean MCV (p 0.010) than petrol station personnel. The MCV of automotive mechanics was higher above that of NNPC personnel ($p = 0.044$). Tankers had higher MCH values ($p = 0.044$) than petrol station staff. Mean MCHC for tanker drivers was substantially lower than automobile technicians ($p < 0.001$) and NNPC personnel ($p = 0.030$). The mean platelet count of NNPC employees was higher than automobile technicians ($p < 0.001$) and tanker drivers ($p = 0.002$). Platelet counts were greater in petrol attendants compared to tanker operators ($p = 0.005$) or vehicle mechanics ($p < 0.001$). Petrol station personnel, tanker drivers, and NNPC officials had higher neutrophil counts than auto technicians (p 0.001). NNPC officials, tanker drivers, or petrol station attendants exhibited reduced levels of lymphocytes than auto technicians (p 0.001, 0.004. Lymphocyte counts were lower in tanker drivers than petrol station personnel ($p = 0.020$).

Monocyte levels were higher in car mechanics than NNPC employees ($p = 0.013$) or petrol station personnel (p 0.001). Additionally, tanker drivers' monocyte levels were

higher than those of the petrol station workers (p = 0.007). When compared to petrol station workers (p = 0.025), automotive technicians (p = 0.020) or tankers drivers (p = 0.002), the NNPC employees showed lower eosinophil levels. When compared to tanker drivers and auto technicians, the basophil level was lower (p 0.001) in the petrol station attendants. Likewise, when NNPC employees were compared to car mechanics and tanker drivers, their basophil levels were found to be lower (p < 0.001).

Table 4.22. The Mean Serum Levels of Hematological Parameters Compared among the Experimental Groups

Variables	Petrol Attendant	Auto Mechanics	Tanker Drivers	NNPC Staff	F-Stat	P – Value
WBC ($10^3/\mu L$)	5.48 ± 1.55	3.93 ± 1.35	5.22 ± 1.39	5.12 ± 1.67	10.59	<0.001
RBC ($10^6/\mu L$)	4.91 ± 0.67	4.84 ± 0.72	4.82 ± 0.84	5.16 ± 0.51	2.46	0.064
HB (g/dL)	13.26 ± 1.34	14.26 ± 1.11	13.99 ± 1.35	14.43 ± 0.93	9.16	<0.001
HCT (%)	40.12 ± 4.13	42.36 ± 6.50	42.74 ± 5.72	43.49 ± 3.12	4.13	0.007
MCV (μm^3)	82.14 ± 6.15	87.13 ± 10.65	87.02 ± 7.35	83.66 ± 9.39	4.22	0.006
MCH (pg)	27.20 ± 2.42	29.90 ± 4.20	35.21 ± 39.2	28.12 ± 2.37	1.64	0.181
MCHC (g/dL)	33.09 ± 1.20	34.03 ± 4.58	32.08 ± 1.73	33.22 ± 1.30	4.70	0.003
Platelet	225.4 ±	181.0 ±	190.68 ±	229.6 ± 48.6	7.79	<0.001

(cells/μL)	83.1	64.92	42.8			
Neutrophils (%)	41.12 ± 9.64	30.47 ± 13.08	42.72 ± 9.18	44.57 ± 11.0	17.04	<0.001
Lymphocytes (%)	45.62 ± 8.59	51.62 ± 13.59	40.75 ± 8.47	42.23 ±10.08	10.98	<0.001
Monocytes (%)	8.18 ± 1.96	11.59 ± 7.94	10.68 ± 3.38	9.31 ± 2.16	5.41	0.001
Eosinophils (%)	4.33 ± 4.48	4.39 ± 4.21	4.96 ± 2.66	2.75 ± 1.90	3.68	0.013
Basophils (%)	0.74 ± 0.38	2.41 ± 2.54	1.96 ± 1.61	0.71 ± 0.34	15.96	<0.001

We compared petroleum attendants' average 8-deoxylhydroxylguanosine levels to the not exposed control group (Figure 4.1). The petroleum station attendants and unexposed control group had similar 8-deoxylhydroxylguanosine levels (p = 0.172).

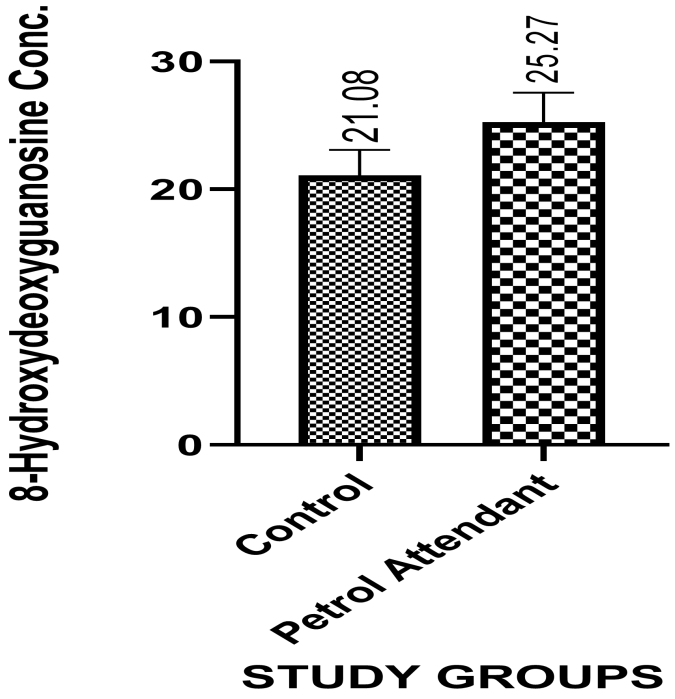

Figure 4.1: Petroleum Participants or Unexposed Control Deoxylhydroxylguanosine Mean

The average concentration of 8-deoxylhydroxylguanosine in auto mechanics is displayed in Figure 4.2, along with the unexposed control. The mean 8-deoxylhydroxylguanosine did not significantly differ (p = 0.122) between the exposed automotive technicians and the unexposed control group.

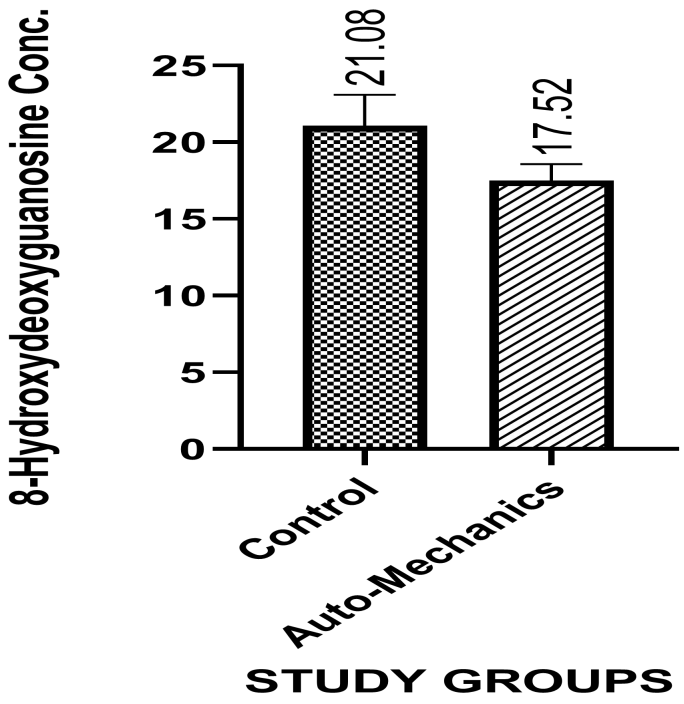

Figure 4.2: The Unexposed Control and Mean 8-Deoxylhydroxylguanosine in Automotive Mechanics

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Figure 4.3 displays the mean levels and contrast between 8-deoxylhydroxylguanosine between the unexposed control and tanker drivers. No significant difference in mean 8-deoxylhydroxylguanosine between unexposed control group and tanker drivers ($p = 0.276$).

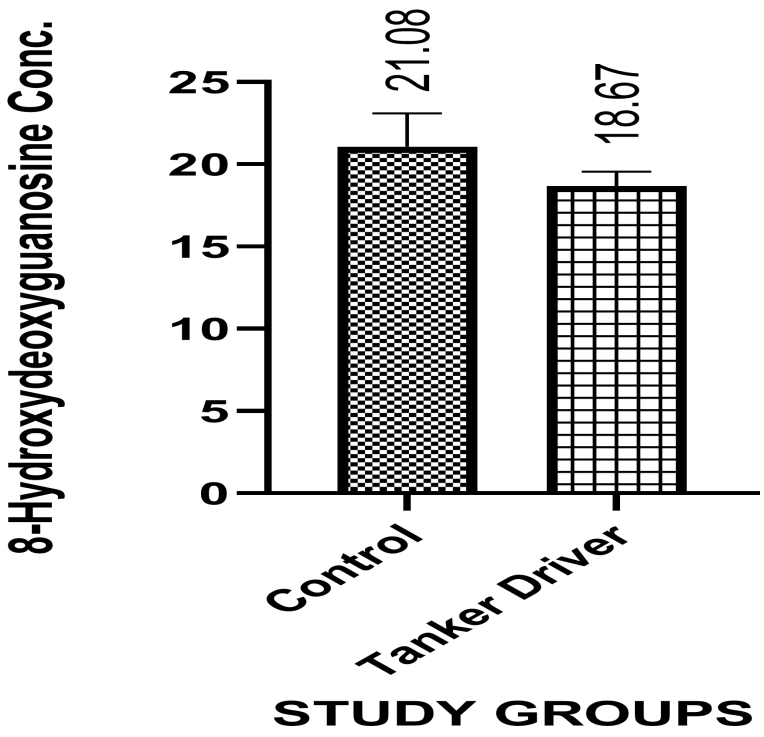

Figure 4.3 Mean 8-deoxylhydroxylguanosine in Tanker Drivers and the Unexposed Control

Figure 4.4 for the standard deviation 8-deoxylhydroxylguanosine in the NNPC staff as well as the unexposed control. The unexposed control group had a significantly higher mean 8-deoxylhydroxylguanosine level ($p < 0.001$) compared to the NNPC workforce.

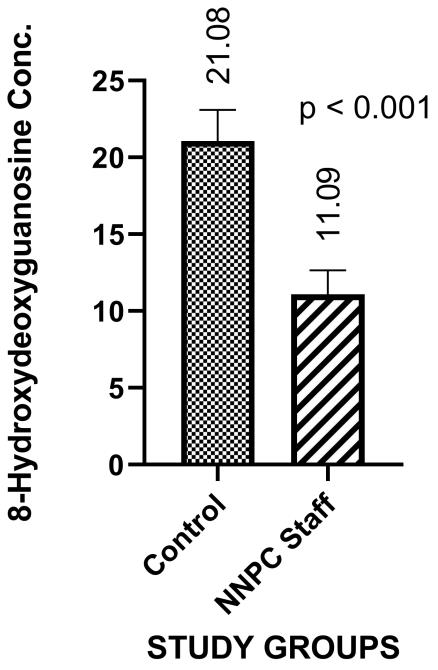

Figure 4.4 Mean 8-Deoxylhydroxylguanosine in NNPC Staff and the Unexposed Control

The standard deviations of the blood concentrations of 8-deoxylhydroxylguanosine for each experimental group are compared in Table 4.23. According to the data, the mean 8-deoxylhydroxylguanosine of petrol attendants was significantly greater than that of car technicians ($p < 0.001$), tankers drivers ($p = 0.003$), and NNPC officials ($p < 0.001$). When NNPC employees were compared to mechanics working on vehicles ($p = 0.004$) or tanker drivers ($p = 0.001$), their 8-deoxylhydroxylguanosine levels were lower.

Table 4.23. The Mean Serum Levels of 8-Deoxylhydroxylguanosine Compared among the Experimental Groups

Groups	N	Mean	Std. Deviation	F- Statistics	P – Value
Petrol Attendants	50	25.27	16.13		
Auto Mechanics	50	17.52	7.49	14.14	<0.001
Tanker Drivers	50	18.67	6.23		
NNPC Staff	50	11.09	11.02		

CHAPTER FIVE

DISCUSSION, CONCLUSION AND RECOMMENDATION

5.1: Discussion of Findings

On the job and in the surrounding environment, benzoene is a common contaminant. Given that benzene is a Group 1 carcinogen to people and animals, health risks and stress levels associated with the molecule have long been of concern. Leukaemia and benzene exposure are closely related, particularly in the case of acute myeloid leukaemia. Furthermore, the neurological, reproductive, and immune systems may all be negatively impacted by benzene exposure. Hematopoietic progenitor cells are susceptible to direct benzene damage, which may lower their reactivity to cytokines and cellular adhesion molecules or cause apoptosis. However, the toxic effects of benzo on stromal cells or mature blood cells can disturb the generation of hem management, specifically affecting haematological determination, maturation, or mobilisation. This disruption is caused by the interconnected system of cytokines, chemokines, and adhesion molecules.

The present investigation examined the impact of exposure to petroleum products on the assessed biomarkers of oxidative stress, inflammation, and immunological function.

The study population was predominantly males (control, 68%; automobile mechanics, 100%; tanker drivers, 100% and NNPC staff 100%). These employees are carrier workers who sporadically deployed to the field to keep an eye on other temporary workers. While the majority of auto technicians (62%), tanker drivers (74%), and NNPC employees (100%) were married, the majority of the controlled subjects (54%) and petrol station attendants (82%) were single. A greater percentage of the control subjects (56%) and petrol attendants (82%) had no children, while the majority of the auto-mechanics (52%),

tanker drivers (70%) and NNPC staff (100%) indicated a high level of parity (≥5 children). This could be attributed to the geographical location of the study, which was conducted in the Northern region of the country. In this region, a significant proportion of the participants adhere to the Islamic faith, which promotes polygamy and having a large number of children. Majority of the married participants (control, 47.8%; petrol attendants, 33.3%; tanker drivers, 43.2% 32.3%), have spent 2 years in marriage. Most of the married auto-mobile mechanics (41.9%) and NNPC staff (44%) have spent 4 years in marriage. A greater percentage (74%) of the control subjects had Bsc degree, while majority of the petrol attendants (84%), mechanics (94%), tanker drivers (88%) had secondary school education, while most of the NNPC staff (66%) had GRA. Majority of the control, 82%; petrol attendants, 70%; NNPC staff (60%) were Christians, while most of the tanker drivers were Muslims (94%). The participants were predominantly non-smokers, non-alcoholics and do not take hard drugs. All the petrol attendants that smoke reported they take 1 stick of cigarette per day, 66.7% of the smoking mechanics reported they take not more than 2 sticks per day; all the smoking tanker drivers and NNPC staff indicated they take more than 2 stick per day. For the entire research population, the majority of alcohol consumers said they only drank one bottle a day. In the words of Spatari et al., the fuel's aromatic hydrocarbons are the active chemicals that reduce the immune response. Our analysis revealed that personal benzene exposure was the sole component that had a significant impact on the elevated amounts of IL-1β, IL-6, TNF-, which and IFN-γ, as indicated by the model using multiple linear regression.

The study revealed that 50% of tanker drivers had a work experience ranging from 6 to 10 years, while 94% of petrol attendants had a work experience ranging from 1 to 5 years.

Additionally, 76% of NNPC personnel had a work experience ranging from 11 to 15 years. A greater percentage of the petrol attendants (58%), automobile mechanics (44%) and NNPC staff (82%) work for ≤10 hours daily, while all the tanker drivers (100%) reported they put in more than 10 hours of work per day. The long working hours of petroleum tanker drivers may be due to the fact that in this part of the world, there is no clear work ethics that spell out the duration or the distance a driver should cover, unlike in the developed countries where two drivers are usually attached to a vehicle to enable them swap after driving for certain period of time or cover certain distance. This practice and work schedule allow the drivers adequate rest which promotes mental alertness. The majority of the control group (100%) as well as the NNPC officials (84%) and fuel station attendants (56%) said they were aware of the impacts of petroleum products. But the majo 0020rity of tanker drivers (64%) and car mechanics (54%) claimed they were unaware of the dangers of petroleum products. The prevalence of ignorance regarding the impact of petroleum on the skin is particularly evident in the numerous mechanic workshops in Nigeria. In these workshops, motor mechanics, specifically those working with petrol engines, often resort to syphoning fuel with their mouths and subsequently use the same fuel to clean their hands after work.

Skin irritation was suggested as the type of petroleum product effect most experienced by the participants (petrol attendants, 54%; mechanics, 36%; tanker driver, 72%). The NNPC staff stated that difficulty in breathing (32%) was most experienced by them as the effect of petroleum products. Most of the petrol attendants (48%), mechanics (66%), and tanker drivers (48%) reported that the symptoms of the petrol product effect they experienced disappeared without treatment. This may be due to the fact that majority of

work place and site of exposure are well ventilated, allowing cross ventilations which prevents the effect of the petroleum exposure especially benzene vapour to be felt. Most of the NNPC staff (46%) said they sought medical attention to treat the petrol product effects. The skin was the most identified route of exposure to petroleum products (petrol attendants, 46%; mechanics, 62%; tanker drivers, 44%). Majority (42%) of the NNPC staff chose nostril as the main route of exposure to petroleum products. A greater percentage of the control (100%), petrol attendants (96%), tanker drivers (74%), and NNPC staff (78%) said they are not in the habit of sucking petrol with their mouth, whereas majority of the auto mechanics (74%) admitted they suck petrol with their mouth. Similarly, the majority of control subjects (100%) and employees of NNPC (86%) and petrol attendants (68%) said they did not wash their hands with petrol frequently, while the majority of auto mechanics (82%), tanker drivers (56%) and other workers said they do. With regard to personal protective equipment (PPE), the vast majority of the controlled subjects (76%), gas station attendants (58%), auto technicians (74%), tankers drivers (98%) and NNPC employees (94%) reported knowing about it. However, a greater percentage of the respondents (control, 76%; auto mechanics, 68%; tanker drivers, 98%; and NNPC, 90%) did not make use of these PPE. On the other hand, majority (60%) of the petrol attendants claimed they made use of the PPE. When asked to mention some of the PPE used, data showed that the petrol attendants stated more use of hand gloves (45%); auto mechanics and NNPC staff used more of coverall (97% and 53.3%), while tanker drivers used helmets (86.7%) more than other PPE.

In this study, analysis of variance (ANOVA) indicated significantly higher phenol in tanker drivers compared with petrol attendants, auto mechanics and NNPC staff. The

NNPC staff also indicated higher mean phenol compared with auto mechanics. Serum styrene level was also higher in NNPC staff compared with petrol attendants, auto mechanics and tanker drivers. In comparison to petroleum attendants and NNPC employees, tanker drivers had a statistically higher mean butanoic acid. In comparison with the NNPC workers, the butanoic acid levels of auto mechanics and petrol attendants were also greater. Petroleum attendants (2.45 mg/m3), mechanics working on cars (1.53 mg/m3), tankers drivers (0.94 mg/m3), and NNPC staff (0.42 mg/m3) had the highest mean benzene levels across the experimental groups, with statistically significant variations observed. Benzene chloride also showed significant differences among the experimental groups with the highest mean value seen in the petrol attendants (1.80 mg/m^3), followed by the tanker drivers (0.85 mg/m^3), auto mechanics (0.60 mg/m^3) and NNPC staff (0.24 mg/m^3). NNPC employees, car mechanics and petrol attendants did not exhibit a substantially (p 0.001) higher o-xylene level than did tanker drivers. In comparison to petrol station attendants and car mechanics, NNPC employees had a higher mean value. Benzoic acid indicated significant differences among the experimental groups with the NNPC staff showing the highest value (1.78 mg/m^3), followed by the petrol attendants (1.38 mg/m^3), tanker drivers (0.79 mg/m^3), and auto mechanics (0.57 mg/m^3). Auto mechanics had significantly higher toluene level compared with tanker drivers ($p < 0.001$), and NNPC staff. NNPC staff showed higher values compared with petrol attendants and tanker driver. Tanker drivers on the other hand indicated higher mean toluene compared with the petrol attendants. Benzene dimethyl indicated significant differences among the experimental groups with the highest mean value seen in the tanker drivers (2.85 mg/m^3), followed by the auto

mechanics (2.25 mg/m^3), petrol attendants (0.97 mg/m^3) and NNPC staff (0.26 mg/m^3). Tanker drivers had significantly higher mean levels of p-xylene compared to auto mechanics, petrol attendants, and NNPC personnel. NNPC employees and petrol station attendants were found to have lower mean values than auto mechanics. NNPC employees (0.77 mg/m3), car mechanics (0.54 mg/m3), petrol station attendants (0.25 mg/m3), and tanker drivers (1.13 mg/m3) had the highest naphthalene levels across the study groups. Significant differences were also observed in ethylene benzene levels with the NNPC staff indicating the highest mean level (4.9 mg/m^3), followed by the petrol attendants (2.96 mg/m^3), tanker drivers (2.04 mg/m^3) and auto mechanics (1.04 mg/m^3). Results from this study agrees with previous works; Moro *et al.,* 2018. They recorded increase value of BTX (tables 4.3, 4.4 and 4.5) among petrol attendants.

In petrol fumes, aliphatic chemicals make up around 95% of the compounds, whereas aromatics make up less than 2%. According to Odewabi et al. (2014), it is therefore always easily accessible in the atmosphere, particularly at petrol stations.

Cell damage and excessive ROS production have been related in numerous studies. The imbalance between the pace at which ROS are generated and the rate at which antioxidants consume them results in oxidative stress (Livingstone, 2001). There are two main ways that ROS is produced. First off, dust particle iron concentration and intrinsic particle characteristics are what cause ROS to be produced. According to Mittel et al. (2014), the production of reactive oxygen species (ROS) is mostly caused by the oxidative burst of neutrophils and macrophages that are stimulated during phagocytosis and prolonged inflammation. Oxidation processes result in the production of free radicals, which then harm cells through a chain reaction. Antioxidants stop these chemical events

by preventing these molecules from oxidising. Antioxidants are generally reducing agents like thiols, polyphenols, and the antioxidant ascorbic acid (vitamin C) since they accomplish this by oxidising themselves. The intricate network of many antioxidants that is prevalent in animals as well as plants comprises glutathione, vitamin C, as well A, and E, along with enzymes such as catalase, super oxide dismutase, and several peroxidases.

The results of this experiment demonstrated that tankers drivers had significantly higher total oxidant capacity compared to NNPC personnel, auto mechanics, or petrol attendants. When juxtaposed with petrol station personnel, car mechanics, and tankers operators, the members of the NNPC staff exhibited significantly higher average levels of glutathione peroxidase. The auto mechanics indicated higher mean C reactive protein compared with tanker drivers and NNPC staff. The petrol attendants also had higher mean values of CRP compared with tanker drivers and NNPC staff. Benzene occupies the major composition of petroleum constituents and is a class I human carcinogen (Sumathi and Neelambikai, 2016). Activation of benzene and its reactive metabolites leads to continuous production of reactive oxygen species (ROS), which leads to lipid peroxidation and damages DNA, RNA, leading to genetic modification and alterations in the functions of important enzymes and proteins (Mohammed *et al*., 2020). Individuals who come into contact with these constituents included in petroleum products are at danger of encountering a diverse range of adverse health consequences. The total antioxidant capacity considers the collective influence of all antioxidants present in body fluids, including bloodstream (Verma and Rana, 2001). The elevated levels on total antioxidant capacity seen in this study amongst tanker drivers may not be attributed solely to exposure to petroleum compounds, but rather to other metabolic processes occurring inside their system.

According to this survey, the majority of tanker drivers (50%) had 6–10 years of work experience, while the majority of NNPC employees (76%) had 11–15 years. Petrol attendants made up the largest group with 94% of those with 1–5 years of experience. While all tankers drivers (100%) reported working more than 10 hours a day, a higher percentage of control workers (96%), petroleum attendants (58%), auto mechanics (44%) or NNPC employees (82%) work less than 10 hours a day.. The participants were predominantly non-smokers, non-alcoholics and do not take hard drugs. All the petrol attendants that smoke reported they take 1 stick of cigarette per day, 66.7% of the smoking mechanics reported they take not more than 2 sticks per day; all the smoking tanker drivers and NNPC staff indicated they take more than 2 stick per day. In addition, the results demonstrated a statistically significant increase in antioxidant activities between the petroleum product-exposed and non-exposed groups. Their outcome is comparable to Aida et al.'s (2015). The study's findings concur with those of earlier research projects; Moro et al., 2018. The amount of glutathione S transferase in petrol station workers was found to have increased. Ibrahim et al.'s 2021 assessment of antioxidant biomarkers among Bauchi metropolis's petroleum vendors produced excellent findings.. Though, malondialdehyde (MAD), catalase (CAT) and reduce glutathione (GSSH) were assayed in their work, compared to this study where C reactive protein, total antioxidant capacity (TAC) and glutathione S peroxidase were analysed.

Total Antioxidant Capacity results in this study (figure 4.12) disagree with the work of Malini and Maithily, 2012 in chronic exposure to petroleum in India. They obtained decreased values in both tanker drivers and petrol attendants, but higher in petrol attendants. Other researchers' results include those of Owagboriaye et al., who evaluated

the impact of petrol fumes on albino rats' antioxidant status and lipid peroxidation and found that the concentration of serum MDA was significantly greater and the concentration of serum GSH was significantly lower. Additionally, Odewabi et al. found that among petrol station workers in Ogun, Nigeria, there were considerably higher serum MDA levels and significantly lower serum GSH levels. However, the results is not in concordance with work of Malini and Maithily, (2017), who reported finding that showed no significant difference in superoxide dismutase and levels of Malondialdehyde and total antioxidant capacity among 165 males divided into three groups were the petrol fillers, tanker drivers and the controls.

Petroleum attendants and tanker drivers exhibited a statistically significant increase in interleukin-1 Beta levels compared to the NNPC workers, with a p-value of less than 0.05. The levels of interleukin-3 were found to be significantly greater in automobile mechanics compared to both tankers operators as well as and NNPC officials. Petrol attendants exhibited markedly elevated IL-3 levels in comparison to both the tanker's operators or NNPC officials. The IL-4 level in NNPC workforce was significantly greater compared with the levels of petrol attendants, auto mechanics, and tanker drivers. The petrol attendants had significantly elevated levels of IL-6 compared with those of auto mechanics, tanker drivers, and NNPC officials, with a p-value of less than 0.05. Interleukin 8 levels were significantly higher ($p < 0.001$) amongst petrol attendants compared to car technicians, tanker drivers, and NNPC officials. The auto mechanics exhibited significantly elevated levels of IL-8 comparing against the tanker drivers or NNPC officials, with a p-value of less than 0.001. The average IL-9 level was substantially greater in car technicians compared to petroleum personnel, tankers drivers,

and NNPC officials. Tanker drivers exhibited a significantly higher average IL-10 level compared to petroleum attendant, automotive mechanics, and NNPC officials. The average LT-beta was significantly higher in petroleum attendants compared to vehicle mechanics, tankers drivers, and NNPC officials. The auto mechanics exhibited significantly greater levels of IL-8 compared to the a tanker operators and NNPC officials, with a p-value of less than 0.001. The mean LT-beta value of the NNPC workforce was significantly greater compared to that of the tanker drivers. The NNPC workforce had a significantly elevated average gamma interferon gamma concentration in comparison with those of petrol attendant or automotive mechanics. Higher interferon gamma level was also found in tanker drivers compared with the petrol attendants. Tanker drivers indicated higher mean IgG level compared with the petrol attendants, auto mechanics and NNPC staff. The NNPC staff presented higher mean IgM compared with petrol attendants and auto mechanics. The tanker drivers also showed higher values of IgM compared with the petrol attendants. Human exposure to benzene can occur not only through inhalation and dermal absorption, but also through ingestion of food and drinking water (Wilbur *et al*., 2008). Exposure to benzene can cause harmful effects on immunological, neurological, and reproductive systems (ATSDR, 2007). Benzene can reduce both B-cell and T-cell proliferation, decrease the host resistance to infection in animals exposed to it, and produce chromosomal aberrations in human peripheral lymphocytes (IPCS, 1993). Results from this study agrees with previous works; Moro *et al.,* 2018. proinflammatory cytokines among gasoline station attendants. Several researchers have reported comparable findings (higher IL-6 and TFN), including

References: Bahadar et al. (2014); Ducos et al. (1990); Mosser and Zhang (2008) (lower IL-10); Moro et al. (2017); Carrieri et al. (2018); Moro et al. (2015); Salem et al. (2018).

Hematopoietic progenitor cells that have been directly damaged by benzoene may undergo apoptosis or become less receptive to cytokines and cellular adhesion molecules. On the other hand, benzene toxicity to mature blood cells or stromal cells may interfere with the network of chemokines, adhesion molecules, and cytokines that regulates hematopoiesis, including hematopoietic commitment, maturation, or mobilisation (King *et al.*, 1989; Miller *et al.,* 1994; Kalf *et al.,* 1996; Ross *et al.*, 1996 and Kalf *et al.*, 2000). Accordingly, hematotoxic effects could be enhanced among individuals exposed to benzene who have genetic variants that alter key pathways thats regulate hematopoiesis (Lan *et al.,* 2005). Growing corpus of research suggests that benzoene may impact the synthesis, processing, or gene expression of many cytokines in vitro as well as in vivo. NNPC officials, tanker drivers, and petrol attendants had considerably higher WBCs than car mechanics, according to the examination of haematological data in this study. In comparison to car mechanics along with tanker drivers, the NNPC workforce showed considerably greater RBC. The average haemoglobin concentrations of petroleum attendants were lower compared to vehicle technicians, tanker operators, or NNPC workers. In addition to automobile mechanics, tanker operators, or NNPC staff, The average Hct of the petrol station personnel was also significantly lower. There were statistically significant disparities between the mean MCVs of the tanker drivers and automotive technicians and the petrol attendants. Additionally, the MCV of the auto technicians was higher than that of the NNPC employees. Tanker drivers' MCH values were higher than those of petrol attendants.

Compared to tanker drivers, the mean MCHCs of NNPC personnel and mechanics working on cars were statistically greater. NNPC employees reported a greater mean platelet level than mechanics who work on automobiles as well as tanker drivers. Overall platelet level was similarly higher among gas station employees than among mechanics working on vehicles among tanker drivers. Auto technicians had significantly reduced neutrophil counts in comparison to The National Petroleum Corporation (staff members, tankers drivers, and petrol attendants. NNPC personnel, tankers drivers, or gas station workers exhibited lymphocyte levels that were lower compared to auto technicians. A notable disparity was seen when examining lymphocyte concentrations among petroleum attendants and tanker drivers. The auto mechanics exhibited elevated monocyte levels in comparison with those of petrol station attendant or the National Petroleum Corporation staff. Tanker drivers had significantly elevated monocyte levels in comparison to gas attendant. The eosinophil levels of NNPC staff were found to be significantly lower than those of petroleum attendants, car mechanics, or tanker tankers. The petrol attendants had significantly lower the basophil levels in comparison to the automobile mechanics and tanker drivers. Similarly, the NNPC staff indicated lower basophil level compared with auto mechanics and tanker drivers. Results in tables 18 - 21 indicate increased in haematological parameters (PCV, Hb, TRBC, TWBC, MCHC, MCH and MCV) among the participants. However, petroleum attendants had reduced hematological parameters when all of them were compared. This disagrees with Okoro *et al*., 2006 whom got reduced haematological parameters (PCV, Hb, TRBC, TWBC, MCHC, MCH and MCV) among fuel attendants at Calabar. The increase in haematology parameters among some participants may be due to shorter period of exposure. Also, some group of the

participants (NNPC officials) are chief executives, who rarely go out for field work, hence reduced contacts are made with the petroleum products. Similar findings were reported by Marieb et al. (1995) using an animal model. According to their suggestion, petroleum chemicals induce changes in blood chemistry and lead to anaemia by causing underdevelopment of the bone marrow in mice used for experiments.

This study points to a comparable outcome for people, particularly for the participants who worked as fuel attendants. Crude oil's hazardous components, like lead and benzoene, have been shown to activate in the bone marrow. The cytotoxic effects are believed to be caused by the disruption of DNA function. The resultant bone marrow depression is characterized by inadequate production of red cell and other formed elements (Rabble *et al,* 1996; Synder and Hedli, 1996). White blood cells function primarily in body defense against foreign bodies and this is often achieved through leucocytosis and antibody production (Marieb, 1995; Robbin and Angel, 1976).

This study found that individuals whose bodies were exposed to hydrocarbon vapours for a duration exceeding two years experienced a notable decrease in their total wbc, a phenomenon observed in both males and females. According to reports, benzoene's myelotoxic activity causes haematological alterations that range from pancytopenia to complete bone marrow aplasia (d'Azevedo *et al.,* 1996). Xylene is also reported to cause leukocytopaenia (d'Azevedo *et al.,* 1995). The decrease in WBC observed in this study is possibly as a result of pancytopaenia and leukocytopaenia, which may result in impaired migration of phagocytic cells, lower resistance to viruses, bacteria and foreign bodies (Marieb, 1995). The observation in this study is similar to previous findings attributed to toxicity from constituents of crude oil , combined with stress imposed by crude oil

hydrocarbons (Ndodigha *et al,* 1999; Dede and Kagbo, 2002; Ovuru and Ekweozor, 2004).

The study's findings revealed that the average level of 8-deoxylhydroxylguanosine, a marker for DNA damage, was significantly higher in petrol attendants compared to vehicle mechanics, tanker operators, and NNPC personnel ($p < 0.001$). The NNPC staff indicated lower 8-deoxylhydroxylguanosine compared with auto mechanics and tanker drivers. Epidemiological studies have not demonstrated a statistically significant link between cancer and occupational exposure to petrol (McLaughlin *et al*., 1983; Rushton and Alderson, 1983; McLaughlin *et al*., 1984; McLaughlin *et al*., 1985; Enterline and Viren 1985Norell *et al*., 1986; McLaughlin, 1993; Dement *et al*., 1997; Enterline, 1993). But the IARC has categorised petrol as "possibly carcinogenic to humans" mostly because there is insufficient data to support the carcinogenicity of the chemical in people but plenty to support its carcinogenicity in laboratory animals. It was also mentioned that several of the ingredients in petrol, like benzene and 1,3-butadiene, are recognised or suspected human carcinogens (IARC, 1989). Chemicals Hazard Identification and Processing for Supply gives petrol the Risk Phrase R45, which means "may cause cancer." (CHIPS) Regulations. Besides oxidative damage to protein, the continuous production of reactive species during benzene metabolism could induce toxicity in key cellular components, such as DNA (Badham, 2010). It is well known that oxidative stress determines disturbance of intracellular metabolic processes which cause alterations including oxidation of lipids, proteins and DNA with consequent long-lasting modifications, as suggested by several studies (Uzma *et al.*, 2010); resultant oxidation products include 8-OHdG, an oxidized nucleoside originated from DNA oxidative

damage and repair an excreted with urine (Sciskalska *et al.,* 2014). Higher concentration of 8OHDG obtained in this study among the petrol attendants (figure 4.1) agrees with Fenga *et al*., 2017, whom in their study obtained increased 8OHDG among workers exposed to low dosage of benzene.

5.2 Limitations

This study experienced some limitations which is listed as follows:

1. Inability to measure the environmental concentrations of benzene around the study areas.
2. Refusal of vast majority of the participants to offer their urine samples due to one reasons or the other, ranging from personal, beliefs, culture to religion.
3. Many participants opted out at the middle of the study due to the reason stated above, hence reducing the sample size.

5.3 Conclusion

The study indicates that the toxic properties of petrol fumes, acting as a substance that damages genetic material, likely played a role in the elevated levels in oxidative stress (OS), blood-related, inflammatory, immune-related, and DNA damage markers observed in the individuals. To facilitate expansion and exploration of uncharted areas, it is imperative to provide increased funding for further research in this domain. Medical monitoring requires meticulous focus on the individuals who are exposed. Enforcing stringent standards is essential to safeguard the well-being of individuals who are exposed to petroleum products. These workers should also be ensured that the workplace is safe and secure to mitigate any possible health hazards.

5.4 Recommendations

1. Government should activate the policy that mandate the use of PPE, and where it is absent, should be formulated.
2. All the concerned agencies responsible for educating the public on the dangers of petroleum products to health should be made to perform their obligations.

5.5 Contribution to Knowledge

This study revealed concentrations of benzene and its derivatives among the participants which has never been done in this part of the world to the best of our knowledge.

REFERENCES

Aggarwal S., van de Loosdrecht A. A., C. Alhan, Ossenkoppele G. J., Westers T. M. and Bontkes H. J. (2011). Role of immune responses in the pathogenesis of low-risk MDS and highrisk MDS: implications for immunotherapy," *British Journal of Haematology* 153(5):568–581.

Aida, A.H. Sahar, A.A. Amal, F.G. and Sarah, A.B. (2013). Assessment of oxidative stress and antioxidant status among petrol stations' workers exposed to benzene in Zagazig city. *Zagazig University Medical Journal*, 19:5. 446-457.

Alegretti AP, Thiesen FV and Maciel GP. (2004). Analytical method for evaluation of exposure to benzene, toluene, xylene in blood by gas chromatography preceded by solid phase microextraction. *Journal of Chromatography B Analytical Technology Biomedical Life Science*;809(1):183-7.

Alison Frank (2009). The Petroleum War of 1910: Standard Oil, Austria, and the Limits of the Multinational Corporation". The American Historical Review. Oxford University Press. ***114*** (1):16–41.

Amoore, J., Von Burg, R. and Whittemore, I. (1983). Detectibility of Gasoline by its Odor. The Toxicologist. 22nd Annual Meeting of the Society of Toxicologists. 6-11.

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

Andrew W. (2009). The day oil was discovered in Nigeria. BBC News. Retrieved 11 August 2021.

Arnold, S. M., Angerer, J. and Boogaard P. J. (2013). "The use of biomonitoring data in exposure and human health risk assessment: benzene case study," *Critical Reviews in Toxicology*, 43(2):119–153.

Austin, H, Delzell E. and Cole, P. (1988). Benzene and Leukemia: a review of the literature and risk assessment. *America Journal of Epidemiology.* 127:419 - 425.

Awasthi G, Joshi D, Swarup A, Mandal TK. and Awasthi DK. (2016). Epidemiological studies on Petroleum toxicity. *International Journal of Pharmaceutical Drug Analysis,* 4: 251-7.

Awoyokun, D. (2013). *"BIAFRA: The Untold Story of Nigeria's civil war". P.M. News*

Baan, R., Grosse, Y. and Straif, K. (2009). A review of human carcinogens. Part F. chemical agents and related occupations," *The Lancet Oncology*. 10 (12):1143–1144.

Badham H.J., Renaud S.J., Wan J. and Winn L.M. (2010). Benzene-initiated oxidative stress: effects on embryonic signaling pathways. Chem Biol Interact. ;184(1–2):218–21.

Bahadar H, Mostafalou S. and Abdollahi M (2014). Current understandings and perspectives on non-cancer health effects of benzene: a global concern. *Toxicol Appl Pharmacol.* 2014;276(2):83–94.

Bandolier (2006).http://www.jr2.ox.ac.uk/bandolier/ band25/b25-6.html

Bank, World (2004). Taxation and State Participation in Nigeria's Oil and Gas Sector.

Banner, W., Jr. and Walson, P. D. (1983). Systemic toxicity following gasoline aspiration. *America Journal of Emergency Medicine* **1**, 292-294.

Bard, D. (2014). Traffic-related air pollution and the onset of myocardial infarction: disclosing benzene as a trigger? A small-area case-crossover study". *Plos One*. **9** (6): 6. .

Barrientos, A., Ortuno, M. T., Morales, J. M., Tello, F. M. and Rodicio, J. L. (1977). Acute renal failure after use of diesel fuel as shampoo. *Archives of internal medicine* **137**, 1217.

Beck, L. S., Hepler, D. I. and Hansen, K. L. (1984) The acute toxicity of selected hydrocarbons. Princeton Scientific Publishers, Inc.,

Becker, C. E. (1985). Principles of occupational Medicine, In; Cecil Textbook of Medicine, 17th ed. (J. B. Wyngaarden and L. H. Smith, Jr. eds.) pp. 2277-2279. W. B. Saunders Co, Philadalphia.

Begum SS, Rathna MB (2012). Pulmonary function tests in petrol filling workers in Mysore city. *Pak Journal of Physiology* ;8:12-4.

Benzene Toxicity (2000). Standards and Regulations". Agency for Toxic Substances and Disease Registry (ATSDR); Environmental Medicine & Environmental Health Education – CSEM. Archived from the original on June 10, 2010. Retrieved October 9, 2010.

Binns, H., Gursel, E. and Wilson, N. (1978). Gasoline contact burns. *Jacep* **7**, 404-405.

Binns, H., Gursel, E. and Wilson, N. (1978). Gasoline contact burns. *Jacep* **7**, 404-405

Birben E, Sahiner UM, Sackesen C, Erzurum S. and Kalayci O. (2012). Oxidative Stress and Antioxidant Defense. *The World Allergy Organization journal,* 5:9–19.

Bironaite, D., Siegel, DMoran, J. L., Weksler, B. B. and Ross, D. (2004). "Stimulation of endothelial IL-8 (eIL-8) production and apoptosis by phenolic metabolites of benzene in HL-60 cells and human bone marrow endothelial cells," *Chemico-Biological Interactions*. 149 (2-3): 177–188.

Boele, R. Fabig, H., and Wheeler D. (2001). Shell, Nigeria and the Ogoni. A study in unsustainable development: I. The story of Shell, Nigeria and the Ogoni people - environment, economy, relationships: conflict and prospects for resolution. *Sustainable Development*. **9** (2): 74–86.

Boscolo, P., Di, M. Gioacchino, E. and Sabbion. (2000). "Lymphocyte subpopulations, cytokines and trace elements in asymptomatic atopic women exposed to an urban environment". *Life Sciences*. 67 (10). 1119–1126.

Burke M. (2008). Nanotechnology: *The Business. p. 3. ISBN 9781420053999.*

Cairney, S., Maruff, P., Burns, C. and Currie, B. (2002). The neurobehavioural consequences of petrol (gasoline) sniffing. *Neuroscience and Biobehavoral Review* **26**, 81-89.

Cairney, S., Maruff, P., Burns, C. B., Currie, J. and Currie, B. J. (2004). Neurological and cognitive impairment associated with leaded gasoline encephalopathy. *Drug Alcohol Dependent* **73**, 183-188.

Cairney, S., Maruff, P., Burns, C. B., Currie, J. and Currie, B. J. (2005). Neurological and cognitive recovery following abstinence from petrol sniffing. *Neuropsychopharmacology* **30**, 1019-1027.

Carbello, M. A., Nigro, M. L, Fraga, I, and Gadano, A. (1994) Ethylene oxide: cytogenic and biochemical studies in persons occupationally exposed. *Environ. Mol. Mutagenesis* 23 (23): 7. d'Azevedo, P. A. Tannhauser, A. L.

Carbonnelle, P., Lison, D., Leroy, J. Y. and Lauwerys, R. (1995). "Effect of the benzene metabolite, hydroquinone, on interleukin-1 secretion by human monocytes in vitro". *Toxicology and Applied Pharmacology*. 132 (2):220–226.

Carrieri M, Spatari G, Tranfo G, Sapienza D, Scapellato ML, Bartolucci GB. (2018). Biological monitoring of low level exposure to benzene in an oil refinery: effect of modulating factors. *Toxicology Letter*.; 298:70-5.

Carter M., Claydon M., Giacopetti D., Money C., Pizzella G., Margary A., and Viinanen R (2002). *A Survey of European Gasoline Exposures for the Period 1999–2001.* European Oil Company Organisation for Environment, Health and Safety (CONCAWE); Brussels, Belgium: 2002. CONCAWE Report nr. 9.

Cecil, R., Ellison, R. J., Larnimaa, K., Margary, S. A., Mata, J. M., Morcillo, L., Muller, J.-M., Peterson, D. R. and Short, D. (1997). Exposure profile: gasoline. *CONCAWE Report* **97/52**.

Çetin, A., Şen, A., Çetin, I., Çimen, B., Cimen, L., Savas, G., Öztürk, A. and Koker, M. (2018). Comparison of ELISA and flow cytometry for measurement of interleukin-1 beta, interleukin-6 and tumor necrosis factor-α. *Turkish Journal of Biochemistry*, *43*(5), 540-548. https://doi.org/10.1515/tjb-2017-0164.

Chang, K. (2018). "Life on Mars? Rover's Latest Discovery Puts It 'On the Table'". The New York Times. Archived from the original on May 28, 2019. Retrieved June 8, 2018. The identification of organic molecules in rocks on the red planet does not necessarily point to life there, past or present, but does indicate that some of the building blocks were present.

Chen Y, Zhang Guo X, Ren J. and Gao A. (2019). The crosstalk between autophagy and apoptosis was mediated by phosphorylation of Bcl-2 and beclin1 in benzene-induced hematotoxicity. *Cell Death Diease.,* 10 (10); p. 772 – 730.

Coker, D. T., Christian, F., Claydon, M. F., Irvine, D., Portail, C., Tindle, P., Webb, C. L. F. and Eyres, A. R. (1987). A Survery of exposures to gasoline vapour. CONCAWE Report 4/87.

Conaway, C. C., Schreiner, C. A. and Cragg, S. T. (1984). Mutagenicity evaluation of petroleum hydrocarbons. Princeton Scientific Publishers Inc, New Jersey page 115 – 120.

Dalvi, S. (2015). Fundamentals of Oil and Gas Industry for Beginners. *ISBN 978-9352064199*.

David T. (1995)."Niger Delta Oil Production, Reserves, Field Sizes Assessed". Industry Briefs. Oil and Gas Journal.

Dede, E. B. and Kagbo, H. D. (2002). A study on the acute toxicological effect of commercial diesel fuel in Nigeria in rats (Ratus ratus) using hematological parameters. *Journal of Applied. Science. Environ. Management*. 6: 84 – 86.

Dehnabeh S.R., Mahdian R., Ajdary S., Mostafavi E. and Khatami S., (2014). Correlation between Plasma Interleukin-3, the *α/β* Globin Ratio, and Globin mRNA Stability", *Anemia*, vol. , Article ID 640203, pages 6 - 10 https://doi.org/10.1155/2014/640203

Dement, J. M., Hensley, L. and Gitelman, A. (1997). Carcinogenicity of gasoline: a review of epidemiological evidence. *Annal of New York Academic Science* **837**, 53-76.

Domej W, Oettl K. and Renner W. (2014). Oxidative stress and free radicals in COPD – implications and relevance for treatment. *International Journal Of Chronic Obstructive Pulmonary Disease*, 9: 1207–1224.

Donkor O. N., Ravikumar M. and Proudfoot O. (2012). Cytokine profile and induction of T helper type 17 and regulatory T cells by human peripheral mononuclear cells after microbial exposure," *Clinical and Experimental Immunology*, vol. 167, no. 2, pp. 282–295.

Dougherty, D; Garte, S; Barchowsky, A; Zmuda, J. and Taioli, E (2008). "NQO1, MPO, CYP2E1, GSTT1 and STM1 polymorphisms and biological effects of benzene exposure—a literature review". *Toxicology* Letters. **182** (1–3): 7–17. doi:10.1016/j.toxlet.2008.09.008. PMID 18848868.

Ducos P, Gaudin R, Robert A, Francin JM and Maire C (1990). Improvement in HPLC analysis of urinary trans,trans-muconic acid, a promising substitute for phenol in the assessment of benzene exposure. *International Archioulogy Occupational Environment Health*. ;62(7):529–34.

Eade, N. R., Taussig, L. M. and Marks, M. I. (1974). Hydrocarbon pneumonitis. *Pediatrics* **54**, 351-357.

Eastmond, D.A.; Rupa, DS. and Hasegawa, LS (2000). "Detection of hyperdiploidy and chromosome breakage in interphase human lymphocytes following exposure to the benzene metabolite hydroquinone using multicolor fluorescence in situ hybridization with DNA probes". *Mutation* Resource **322** (1): 9–20. doi:10.1016/0165-1218(94)90028-0. PMID 7517507.

Eboh, Camillus (2022). "Dangote says it will complete its Nigerian oil refinery in the fourth quarter". *Reuters*. Retrieved.

Edminster, S. C. and Bayer, M. J. (1985). Recreational gasoline sniffing: acute gasoline intoxication and latent organolead poisoning. Case reports and literature review. *Journal of Emergency Medicine* **3**, 365-370.

Eigenbrode J. L. (2018). Organic matter preserved in 3-billion-year-old mudstones at Gale crater, Mars" (PDF). *Science*. **360** (6393): 1096–1101. Bibcode:2018Sci...360.1096E. doi:10.1126/science.aas9185. PMID 29880683. S2CID 46983230. Archived (PDF) from the original on August 25, 2021. Retrieved January 4, 2021.

Eini P., Majzoobi M.M., Ghasemi Basir H.R., Moosavi Z. and Moradi A. (2020). Comparison of the serum level of interleukin-4 in patients with brucellosis and healthy controls. *Journal of Clinical Laboratory Analysis* ;34(7): e23267 - e23681 doi: 10.1002/jcla.23267. Epub PMID: 32100374; PMCID: PMC7370742.

Ekeinde, Austin (2007). *"Convicted governor cheered in Nigerian oil delta". Reuters.*

Eneh O. C (2011). A Review on Petroleum: Source, Uses, Processing, Products and the Environment. *Journal of Applied Science*; 11:2084 -2091.

Enterline, P. E. (1993). Review of new evidence regarding the relationship of gasoline exposure to kidney cancer and leukemia. *Environmental Health Perspective* **101(6)**, 101-103.

Enterline, P. E. and Viren, J. (1985). Epidemiologic evidence for an association between gasoline and kidney cancer. *Environmental Health Perspective* **62**, 303-312.

Esosa U, Osebhahiemen O. and Aanuoluwa S. (2017). Effects of Methanol Leaf Extract of Alchornea laxiflora on Antioxidant Enzymes, Triacylglycerol and Cholesterol Levels in Male Wistar Rats. *International Journal of Life Sciences Research*, 5, (2), 93-97.

EU (1998). Directive 98/70/EC of the European parliament and of the Council: Amendment to Coucnil Directive 93/12/EEC. *Official Journal of the European Communities* **350/59**.

Eyres, A. R. (1987). *A Survery of exposures to gasoline vapour. CONCAWE Report 4/87.*

Fahy, A., Lethbridge, G., Earle, R., Ball, A. S., Timmis, K. N. and McGenity, T. J. (2005). "Effects of long-term benzene pollution on bacterial diversity and

community structure in groundwater," *Environmental Microbiology*. 7(8): 1192–1199.

FDA (2007). Too Much Benzene In Some Drinks" Archived 2007-02-18 at the Wayback Machine, CBS News, May 19, 2006. Retrieved July 11, 2006.

Fenga C, Gangemi S, Teodoro M, Rapisarda V, Golokhvast K, Docea AO, Tsatsakis AM. and Costa C. (2017). 8-Hydroxydeoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to low-dose benzene. *Toxicol Rep.* 31;4:291-295. doi: 10.1016/j.toxrep.2017.05.008. PMID: 28959652; PMCID: PMC5615153.

Flanagan, R. J. and Ives, R. J. (1994). Volatile substance abuse. *Bull Narcotic* **46**, 49-78.

Fortenberry, J. D. (1985). Gasoline sniffing. *America Journal of Medicine* **79**, 740-744.

Fracasso ME, Doria D, Bartolucci GB, Carrieri M, Lovreglio P, Ballini A, Soleo L, Tranfo G. and Manno M (2010). "Low air levels of benzene: Correlation between biomarkers of exposure and genotoxic effects". *Toxicology* Letter. **192** (1): 22–28. doi:10.1016/j.toxlet.2009.04.028. PMID 19427373.

Fraumeni, J. F., Jr. (1984). A population--based case--control study of renal cell carcinoma. *Journal of the National Cancer Institute* **72**, 275-284.

Frynas, J. G. (1999). Oil in Nigeria: Conflict and Litigation Between Oil Companies and Village Communities. *Münster: Lit Verlag. p. 81.*

Gao, Z. (1998). *Environmental Regulation of Oil and Gas*. Kluwer Law International. p. 8.

Garte, S; Taioli, E; Popov, T; Bolognesi, C; Farmer, P. and Merlo, F (2000). "Genetic susceptibility to benzene toxicity in humans". J Toxicol Environ Health A. **71** (22): 1482–1489. doi:10.1080/15287390802349974. PMID 18836923. S2CID 36885673.

Georgieva, T., Michailova, A., Panev, T. and Popov, T. (2002). Possibilities to control the health risk of petrochemical workers. *International archives of occupational and environmental health,* 75(1): S21–S26.

Gillis, B. Gavin, I. M. Arbieva Z., King S. T., Jayaraman, S. and Prabhakar, B. S. (2007). "Identification of human cell responses to benzene and benzene metabolites". *Genomics*. 90 (3):324–333.

Goodheart, R. S. and Dunne, J. W. (1994). Petrol sniffer's encephalopathy. A study of 25 patients. *Medical Journal of Australia* **160**, 178-181.

Guo X. Zhong W, Chen Y. Zhang W, Ren J. and Gao A. (2019). Benzene metabolites trigger pyroptosis and contribute to haematotoxicity via TET2 directly regulating the Aim2/Casp1 pathway *EBioMedicine*, 47; pp. 578-589.

Halder, C. A., Holdsworth, C. E., Cockrell, B. Y. and Piccirillo, V. J. (1985). Hydrocarbon nephropathy in male rats: identification of the nephrotoxic components of unleaded gasoline. *Toxicology Industrial Health* **1**, 67-87.

Halder, C. A., Van Gorp, G. S., Hatoum, N. S. and Warne, T. M. (1986). Gasoline vapor exposures. Part II. Evaluation of the nephrotoxicity of the major C4/C5 hydrocarbon components. *American Industrial Hygiene Association Journal* **47**, 173-175.

Hansbrough, J. F., Zapata-Sirvent, R., Dominic, W., Sullivan, J., Boswick, J. and Wang, X. W. (1985). Hydrocarbon contact injuries. *Journal of Trauma* **25**, 250-252.

Harman, A. W., Frewin, D. B. and Priestly, B. G. (1981). Induction of microsomal drug metabolism in man and in the rat by exposure to petroleum. *British Journal of Industrial Medicine* **38**, 91-97.

Haro-García, L., CJuárez-Pérez, C. A. and Aguilar-Madrid, G. (2012). "Production of IL-10, TNF and IL-12 by peripheral blood mononuclear cells in Mexican workers exposed to a mixture of benzene-toluene-xylene". *Archives of Medical Research*. 43(1): 51–57.

Harrington, J. M. (1987). The health experience of workers in the petroleum manufacturing and distribution industry. *CONCAWE report number 2/87.*

Hegazy RM. and Kamel FM. (2014). Oxidant Hepatic and/or Haem. Injury on Fuel-Station Workers Exposed to Benzene Vapor, Possible Protection of Antioxidants. *American Journal of Medicine and Medical Sciences,* 4:35-46.

Henderson, R. F., Sabourin, P. J., Bechntold, W. E., Steinberg, B. and Chang, I. Y. (1993). Isobutene (2-methylpropene). *Toxicology Applied Pharmacolology 123*: 50 - 61.

Henry, J. A. (1998). Composition and toxicity of petroleum products and their additives. *Human Experimental Toxicolology* **17**, 111-123.

Hieda, Y., Tsujino, Y. and Takeshita, H. (2005). Skin analysis to determine causative agent in dermal exposure to petroleum products. *Journal of Chromatography B, Analytical Technologies in the Biomedical and Life Sciences* 823, 53-59.

Hillis O. F. (2005). Benzene. Ullmann's Encyclopedia of Industrial Chemistry. Weinheim: Wiley-VCH. doi:10.1002/14356007.a03_475. ISBN 978-3527306732

History of the Oil Industry (2012). LUBCON INTERNATIONAL". *Archived from* the original *on 10 April 2012*. Retrieved 4 December 2012. Retrieved 26 November 2012

Huang D, Ying H, Jiang D, Liu F, Tian Y, Du C, Zhang L. and Pu X. (2020). Rapid and sensitive detection of interleukin-6 in serum via time-resolved lateral flow immunoassay. *Analysis Biochemistry* 1;588:113468.

Huckabay, P., Wendy D., VanCleave C. and Ostrander, J. (1995). Petroleum sector notebook paper. Cameron University. *Journal of Applied Science Environmental Management*, 6: 84 – 86.

Huff J. (2007). Benzene-induced cancers: abridged history and occupational health impact". *International Journal of Occupational and Environmental Hygiene.* **13** (2): 213–221.

Lachenauer C. S., Baker C. J., Baron M. J., Kasper D. L., Lawrence C. G., Madoff C. (2002). Quantitative Determination of Immunoglobulin G Specific for Group B Streptococcal β C Protein in Human Maternal Serum, *The Journal of Infectious Diseases*,185(3):368–374.

I.A.R.C. (1982). "Some industrial chemicals and dyestuffs," *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*. 29:1–398.

I.A.R,C. (1989). Occupational Exposure in Petroleum Refining; Crude Oil and Major Petroleum Fuels. *IARC Monographs on the Evaluation of Carcinogenic Risks in Human* **45**.

International Agency for the Research on Cancer (IARC) (1989). Occupational exposures in petroleum refining: Crude oil and major petroleum fuels. IARC Monographs on the evaluation of carcinogenic risks to humans. Volume 45. Lyon.

IPCS (1993). "Benzene," International Programme on Chemical Safety (Environmental Health Criteria 150), World Health Organization, Geneva, Switzerland, 1993, http://www.inchem.org/documents/ehc/ehc/ehc150.htm.

Irons, R. D. and Stillman, W. S. (1996). “Impact of benzene metabolites on differentiation of bone marrow progenitor cells”. *Environmental Health Perspectives*. 104 (6):1247–1250.

Isaacson, Kristofer P.; Proctor, Caitlin R.; Wang, Q. Erica; Edwards, Ethan Y.; Noh, Yoorae; Shah, Amisha D.; Whelton, Andrew J. (2021). "Drinking water contamination from the thermal degradation of plastics: Implications for wildfire and structure fire response". Environmental Science: Water Research & Technology. **7** (2): 274–284. doi:10.1039/D0EW00836B.

Josiah, U. E. (2004). Agglutination, Ideophones and Reduplication as Linguistic Features of the Lower Cross Sub-Family of Languages: The Ibibio Dimension. 11th Biennial Conference of Modern Languages Association of Nigeria. Ajara, Badgry, Lagos State.

Kalf G. F. (2000). “Utility of a mouse model for studying the effects of benzene on the myeloid lineage: effects of hydroquinone on a model myeloid system,” *Journal of Toxicology and Environmental Health A*, vol. 61, no. 5-6, pp. 399–411.

Kalf, G., FRenz, J. F. and Niculescu, R. (1996). “p-Benzoquinone, a reactive metabolite of benzene, prevents the processing of pre-interleukins-1α and -1β to active cytokines by inhibition of the processing enzymes, calpain, and interleukin-1β converting enzyme”. *Environmental Health Perspectives*. 104 (6):1251–1256.

Kanaly RA, Harayama S. (2000). Biodegradation of high-molecular-weight polycyclic aromatic hydrocarbons by bacteria. *Journal of Bacteriology*; 182:2059-2067.

Kasem A. (1992). The Miracle of Islam Science (2nd ed.). Knowledge House Publishers. *ISBN 0-911119-43-4*. *OCLC 26084778*.

Kasper, D. L. (2004). Harrison's Principles of Internal Medicine, 16th ed., McGraw-Hill Professional, p. 618, ISBN 0071402357.

Kato, M. Rocha, M. L., Carvallio, A.. B., Chaves, M.. E., Rana, M. C. and Oliverra, F. C., (1993). Occupational exposure to neutratoxicants- preliminary survey in five industries of camacari petrochemical complex, Brazil, *Environmental Resource* 61: 133139.

Kearney, C. A. and Dunhzam, D. B. (1986). Gasoline vapor exposures at a high volume service station. *America Industrial Hygiene Association Journal* **47**, 535-539.

Kerfoot, H. B., Brady, W. D., Schramm, W. H. and Allendorf, M. A. (2009). “Natural gas as the source of benzene in groundwater,” *Environmental Forensics*.10:(1)60–67.

Kerzic, P. J., Pyatt, D. W., Zheng, J. H. Gross, S. A., Le, A. and Irons, R. D. (2003). "Inhibition of NF-*κ*B by hydroquinone sensitizes human bone marrow progenitor cells to TNF-*α*-induced apoptosi". *Toxicology*. 187 (2-3): 127–137.

Khadija Sharife. "How Nigeria's lucrative oil profits disappear". #Panama Papers. ANCIR.

Khalaf, N.F., Al-rikabi, A.H. and Salman, I.N. (2023). Pre-diabetes and diabetic neuropathy are associated with low serum levels of interleukin-9. *Beni-Suef University Journal Basic Applied Science* **12**, 75. https://doi.org/10.1186/s43088-023-00412-6.

Kim SJ, Lim J, Nam GE. and Park HS. (2021). Correlation between Serum Lipid Parameters and Interleukin-10 Concentration in Obese Individuals. *Journal Obesity Metabolism Syndrome* ;30(2):173-177.

Kinawy, A.A. (2009). Impact of gasoline inhalation on some neurobehavioural characteristics of malerats. *BMC Physiology***,** 9:21.

King A. G., Landreth K. S., and Wierda D (1989). "Bone marrow stromal cell regulation of B-lymphopoiesis. II. Mechanisms of hydroquinone inhibition of pre-B cell maturation," *Journal of Pharmacology and Experimental Therapeutics*, vol. 250, no. 2, pp. 582–590.

Kirkeleit, J., Riise, T., Gjertsen, B.T., Moen, B.E., Braveit, M. and Bruserua, Q. (2008). Effect of benzene in human hematopoiesis. *Open Hematology Journal,* 2:87–102.

Klassen, C. D. (1990). Non metallic environmental toxicant: Air pollutants, solvents, vapour and particles. In: *Goodman and Gillman's Textbook, The Pharmacological Basis of Therapeutics* 8th ed,, A. G. Gilman, T. W. Rall, A. S Niuo and P. Taylor (eds.) NY, Pergamon Press, Pp 1596-1614.

Klieman, K. A. (2012). U.S. Oil Companies, the Nigerian Civil War, and the Origins of Opacity in the Nigerian Oil Industry". *Journal of American History*. **99** (1): 155–165. doi:10.1093/jahist/jas072.

Koh D.H., Jeon H.K., Lee S.G. and Ryu H.W. (2015). The relationship between low-level benzene exposure and blood cell counts in Korean worker. *Occupational of Environmental Med*icine, 72 (6), pp. 421-427.

Kolmetz, G. (2007). Guidelines for BTX Revamps, AIChE . Spring Conference.

Kovarik, W. (2005). Ethyl-leaded gasoline: how a classic occupational disease became an international public health disaster. *Internal Journal Occupation Environmental Health* **11**, 384-397.

Kumar A. and Tyagi S. K. (2006). Benzene and toluene profiles in ambient air of Delhi as determined byactive sampling and GC analysis. *Journal of Science Industrial Resource* 65:252-257.

Kurutas, E.B. (2015). The importance of antioxidants which play the role in cellular response against oxidative/nitrosative stress: current state. *Nutrition Journal* 15, 71.

Lan Q., Zhang L. and. Shen M. (2005). Polymorphisms in cytokine and cellular adhesion molecule gene and susceptibility to hematotoxicity among workers exposed to benzene," *Cancer Research*, vol. 65, no. 20, pp. 9574–9581.

Lehmann I, Rehwagen M., and Diez U. (2001). Enhanced in vivo IgE production and T cell polarization toward the type 2 phenotype in association with indoor exposure to VOC: results of the LARS study," *International Journal of Hygiene and Environmental Health*, vol. 204, no. 4, pp. 211–221.

Li Q., Geiselhart L., Mittler J. N., Mudzinski S. P., Lawrence D. A. and Freed B.M. (1996). Inhibition of human T lymphoblast proliferation by hydroquinone," *Toxicology and Applied Pharmacology*, vol. 139, no. 2, pp. 317–323.

Li, D.M. and Han, X.D. (2006). Evaluation of toxicity of methyl tertbutyl ether (MTBE) on mouse spermatogenic cells in vitro. *Toxicology and Industrial Health,* 22 (7):291–299.

Liang B., Chen Y., Yuan W., Qin F., Zhang Q., N. Deng, X. Liu, Ma X., Zhang X., Zhang B., Deng Q., Huang M., Tang H., Liu L., Chen W., Xiao Y. (2018). Down-regulation of miRNA-451a and miRNA-486-5p involved in benzene-induced inhibition on erythroid cell differentiation in vitro and in vivo. *Archieve of Toxicol*ology, 92 (1), pp. 259-272.

Litovitz, T. (1988). Mycardial sensitization following inhalation abuse of hydrocarbons. *Occupation Medicine* **3**, 567-568.

Litovitz, T. and Greene, A. E. (1988). Health implications of petroleum distillate ingestion. *Occupation Medicine* **3**, 555-568.

Livingstone DR (2001). Contaminant-stimulated reactive oxygen species. Production and oxidative damage in aquatic organisms. *Marine Pollution Bull*; 42:656–66.

Loury, D. J., Smith-Oliver, T., Strom, S., Jirtle, R., Michalopoulos, G. and Butterworth, B. E. (1986). Assessment of unscheduled and replicative DNA synthesis in hepatocytes treated in vivo and in vitro with unleaded gasoline or 2,2,4trimethylpentane. *Toxicology* and *Applied Pharmacology* **85**, 11-23.

Lv, L. Kerzic, P. and Lin, G. (2007). "The TNF-*α* 238A polymorphism is associated with susceptibility to persistent bone marrow dysplasia following chronic exposure to benzene". *Leukemia Research*. 31 (11):1479–1485.

Mahittikorn, A., Mala, W. and Masangkay, F.R. (2022). Increased interferon-γ levels and risk of severe malaria: a meta-analysis. *Science Report* **12**, 18917 - 18925. https://doi.org/10.1038/s41598-022-21965-z

Malini, S., and Maithily, K. (2017). Analysis of Oxidative Stress in Chronic Exposure to Petroleum Hydrocarbons in Karnataka, India. *Asia Pacific Journal of Medical Toxicology*, *6*(1), 6-11.

Marieb, E. N. (1995). *Human Anatomy and Physiology*. 3rd ed. Benjamin and Cummnings Pub Co, California 585-611.

Maruff, P., Burns, C. B., Tyler, P., Currie, B. J. and Currie, J. (1998). Neurological and cognitive abnormalities associated with chronic petrol sniffing. *Brain* **121 (Pt 10)**, 1903-1917.

May G. and Hard O. (1998). The Story of Early Canadians' Quest for Oil at Home and Abroad. Dundurn Press, p. 59

McDermott, H. J. and Vos, G. A. (1979). Service station attendants' exposure to benzene and gasoline vapors. *America Industrial Hygiene Association Journal* **40**, 315-321.

Mchale, C. M., Zhang, L. and Smith, M. T. (2012). "Current understanding of the mechanism of benzene-induced leukemia in humans: implications for risk assessment," *Carcinogenesis*. 33 (2): 240–252.

McKain, D. L., and Bernard L. A. (1994). Where It All Began: The Story of the People and Places Where the Oil Industry Began—West Virginia and Southeastern Ohio. Parkersburg, W.Va.: David L. McKain, . Akiner (2004), p. 5

McKee, R. H., Dally, S., Dmytrasz, B. A., Gonnet, J. F., Hagemann, R. E., Mackerer, C. R., Nessel, C. S., Priston, R. A. and Riley, A. J. (2000). An assessment of the reproductive toxicity of gasoline vapour. CONCAWE Report number 00/53. Brussels.

McLaughlin, J. K. (1993). Renal cell cancer and exposure to gasoline: a review. *Environmental Health Perspective* **101 Suppl 6**, 111-114.

McLaughlin, J. K., Blot, W. J., Mandel, J. S., Schuman, L. M., Mehl, E. S. and Fraumeni, J. F., Jr. (1983). Etiology of cancer of the renal pelvis. *Journal National Cancer Institute* **71**, 287-291.

McLaughlin, J. K., Blot, W. J., Mehl, E. S., Stewart, P. A., Venable, F. S. and Fraumeni, J. F., Jr. (1985). Petroleum-related employment and renal cell cancer. *Journal Occupation Medicine* **27**, 672-674.

McLennan, J. and Williams, S. (2005). Deepwater Africa reaches turning point". Oil & Gas Journal. **103** (6): 18. INIST 16556065.

Melikian, A.A., O'Connor, R., and Prahalad, A.K.. (1999). Determination of the urinary benzene metabolites S-phenylmercapturic acid and trans,trans-muconic acid by liquid chromatography-tandem mass spectrometry. Carcinogenesis 20(4), 719-726.

Mendez-Ferrer S., Michurina T. V., and Ferraro F. (2010). Mesenchy-´ mal and haematopoietic stem cells form a unique bone marrow niche," *Nature*, vol. 466, no. 7308, pp. 829–834.

Miller A. C. K., Schattenberg D. G., Malkinson A. M., and Ross D. (1994). "Decreased content of the IL1α processing enzyme calpain in murine bone marrow-derived macrophages after treatment with the benzene metabolite hydroquinone," *Toxicology Letters*, vol. 74, no. 2, pp. 177–184.

Misser, F. (2013), Amuwo, Kunle; Bach, Daniel C.; Lebeau, Yann (eds.), "European Interests in Nigeria", Nigeria during the Abacha Years (1993-1998): The Domestic and International Politics of Democratization, African Dynamics, Ibadan: IFRA-Nigeria, pp. 235–257, ISBN 979-10-92312-08-9, retrieved 31 May 2021

Mittal M, Siddiqui MR, Tran K, Reddy SP, and Malik AB (2014). Reactive Oxygen Species in Inflammation and Tissue Injury. *Antioxidant Redox Signal* ;20:1126 – 1167.

Miyazawa, M., Ito, Y., Yoshida, Y., Sakaguchi, H. and Suzuki, H. (2007). "Phenotypic alterations and cytokine production in THP-1 cells in response to allergens," *Toxicology in Vitro*. 21. (3): 428–437.

Mohammed, A., Muhammad, I.U., Wudil, A.M., Alhassan, A.J., Abubakar, S.M and Lat, N.A. (2020). *In vitro* and *in vivo* Antioxidant Properties of Extracts from the Root of *Curcuma longa* Linn. *European Journal of Medicinal Plants,* 31 (6): 6-12.

Moro AM, Brucker N, Charao MF, Baierle M, Sauer E. and Goethel G. (2017). Biomonitoring of gasoline station attendants exposed to benzene: effect of gender. *Mutatin Resourse*. ;813:1–9.

Moro A.M., Brucker N., Charao M.F., Sauer E., Freitas F., Durgante J., et al (2015). Early hematological and immunological alterations in gasoline station attendants exposed to benzene. *Environmental Resource*.; 137:349–356.

Mosser D.M. and Zhang X. (2008). Interleukin-10: new perspectives on an old cytokine. *Immunololgy Review*.; 226:205–218.

Ndodigha, E. M., Olayimika, F. O., Oruwari, B. M., Ekweozor, I. K. E. and Wekhe, S. N. (1999). Toxic effect of crude oil on organs and blood cells of West Africa dwarf goat. *Nigerian Vetenary Journal* 20:82- 91.

Niculescu, R., Bradford, H. N., Colman, R. W. and Kalf, G. F. (1995). 'Inhibition of the conversion of pre-interleukins-1α and 1β to mature cytokines by p-benzoquinone, a metabolite of benzene". *Chemico-Biological Interactions*. 98 (3): 211–222.

Norell, S., Ahlbom, A., Olin, R., Erwald, R., Jacobson, G., Lindberg-Navier, I. and Wiechel, K. L. (1986). Occupational factors and pancreatic cancer. *British Journal of Industrial Medicine* **43**, 775-778.

Nylander, P. O., Olofsson, H., Rasmuson, B. and Svahlin, H. (1978). Mutagenic effects of petrol in Drosophila melanogaster I. Effects of benzene and 1,2dichloroethane. *Mutation Research* **57**, 163-167.

Occupational Safety and Health Standards, Toxic and Hazardous Substances (2015). Wayback Machine. Osha.gov. Retrieved on 2011-11-23.

Odewabi, A.O, Ogundahunsi, O.A. and Oyalowo, M. (2014). Effect of Exposure to Petroleum Fumes on Plasma Antioxidant Defense System in Petrol Attendants. *British Journal of Pharmacology and Toxicology,* 5(2):83-87.

Olson, M. J., Garg, B. D., Murty, C. V. and Roy, A. K. (1987). Accumulation of alpha 2u-globulin in the renal proximal tubules of male rats exposed to unleaded gasoline. *Toxicology Applied Pharmacoogyl* **90**, 43-51.

Olson, M. J., Johnson, J. T. and Reidy, C. A. (1990). A comparison of male rat and human urinary proteins: implications for human resistance to hyaline droplet nephropathy. *Toxicology Applied Pharmacology* **102**, 524-536.

Omatsu, Y., Sugiyama, T. and Kohara H. (2010). “The essential functions of adipo-osteogenic progenitors as the hematopoietic stem and progenitor cell niche”. *Immunity*. 33 (3):387–399.

Ovuru, S. S. and Ekweozor, I. K. E. (2004). Haematological changes associated with crude oil

Owagboriaye, F.O., Dedeke, G.A., Aladesida, A.A., Bamidele, J.A. and Olooto, W.E. (2016). Assessment of the effect of gasoline fume on stress hormones, antioxidant status and lipid peroxidation in albino rat; *Journal of king Saud university-science,* 30:(3), 393-399.

Periago, J. F. and Prado, C. (2005). Evolution of occupational exposure to environmental levels of aromatic hydrocarbons in service stations. *Annals of Occupation Hygiene* **49**, 233-240.

Poklis, A. (1976). Death resulting from gasoline "sniffing": a case report. *Journal of Forensic Science Society* **16**, 43-46.

Poklis, A. and Burkett, C. D. (1977). Gasoline sniffing: a review. *Clinical Toxicology* **11**, 3541 - 3545.

Poljsak B, Suput D. and Milisav I. (2013). Achieving the balance between ROS and Antioxidants: When to Use the Synthetic Antioxidants. *Oxidative medicine and cellular longevity*, 9; 450 - 500.

Rabble, G. K. and Wong, O. (1996). Leukemia mortality by cell type in petroleum workers with potential exposure to benzene. *Environmental Health Perspective* 104: 1381 – 1392.

Rana SV; Verma Y. (2005). "Biochemical toxicity of benzene". *Journal of Environmental Biology* **26** (2): 157–168. PMID 16161967.

Rao, G. S. and Pandya, K. P. (1980). Hepatic metabolism of heme in rats after exposure to benzene, gasoline and kerosene. *Archieve of Toxicology* **46**, 313-317.

Rapp, George (1985). Archaeomineralogy. Springer. p. 237.

Rekhadevi, P.V., Rahman, M.F., Mahboob, M. and Grover, P. (2010). Genotoxicity in filling station attendants exposed to petroleum hydrocarbons. *The Annals of Occupational Hygiene,* 54(8): 944-954.

Remington, G. and Hoffman, B. F. (1984). Gas sniffing as a form of substance abuse. *Can Journal of Psychiatry* **29**, 31-35.

Renz, J. F. and Kalf, G. F. (1991). "Role for interleukin-1 (IL-1) in benzene-induced hematotoxicity: inhibition of conversion of pre-IL-1α to mature cytokine in murine macrophages by hydroquinone and prevention of benzene-induced hematotoxicity in mice by IL-1α". *Blood*. 78 (4): 938–944.

Richardson, K. A., Wilmer, J. L., Smith-Simpson, D. and Skopek, T. R. (1986). Assessment of the genotoxic potential of unleaded gasoline and 2,2,4trimethylpentane in human lymphoblasts in vitro. *Toxicology Applied Pharmacology* **82**, 316-322.

Rickert, D. E., Baker, T. S., Bus, J. S., Barrow, C. S. and Irons, R. D.(1979). "Benzene disposition in the rat after exposure by inhalation," *Toxicology and Applied Pharmacology*. 49 (3):417–423.

Ritchie, G. D., Still, K. R., Alexander, W. K., Nordholm, A. F., Wilson, C. L., Rossi, J., 3rd and Mattie, D. R. (2001). A review of the neurotoxicity risk of selected hydrocarbon fuels. *Journal of Toxicology Environmental Health B Critical Review* **4**, 223-312.

Ross D., Siegel D., Schattenberg D. G., Sun X. M., and Moran J. L. (1996). "Cell-specific activation and detoxification of benzene metabolites in mouse and human bone marrow: Identification of target cells and a potential role for modulation of apoptosis in benzene toxicity," *Environmental Health Perspectives*, vol. 104, no. 6, pp. 1177–1182, 1996.

Ross, D. (1996). Metabolic basis of benzene toxicity (Review). *European Journal of Haematology,* 60*:* 111 – 118.

Rothman, N., Li, G. L., Dosemeci, M. Bechtold, W. E., Marti G. E. and Wang, Y. Z. (1996). Haematotoxicity among Chinese Workers-heavily exposed to benzene from *Americal, Journal industrial Medicine* 29 (3): 236-246.

Rothman, N., Smith, M. T. and Hayes, R. B. (1996). "An epidemiologic study of early biologic effects of benzene in Chinese workers". *Environmental Health Perspectives*. 104 (6):1365–1370.

Ruppert T, Scherer G, Tricker AR, Adlkofer F (1997). trans,trans-muconic acid as a biomarker of non-occupational environmental exposure to benzene. *International Archive Occupational Environmental Health*; 69(4):247-251. doi: 10.1007/s004200050143. PMID: 9137998.

Rushton, L. and Alderson, M. R. (1983). Epidemiological survey of oil distribution centres in Britain. *British Journal of Industrial Medicine* **40**, 330-339.

Russell, Loris S. (2003). A Heritage of Light: Lamps and Lighting in the Early Canadian Home. University of Toronto Press. *ISBN 0-8020-3765-8*.

Salem E, El-Garawani I, Allam H, El-Aal BA, Hegazy M. (2018). Genotoxic effects of occupational exposure to benzene in gasoline station workers. *Industrial Health*. 56(2):132–140.

Salim A. (2008). 1000 Years of Missing Industrial History". In Emilia Calvo Labarta; Mercè Comes Maymo; Roser Puig Aguilar; Mònica Rius Pinies (eds.). A shared legacy: Islamic science East and West. Edicions Universitat Barcelona. pp. 57–82 [63]. ISBN 978-84-475-3285-8.

Schnatter, A. R., Glass, D. C., Tang, G., Irons, R. D. and Rushton, L. (2012). "Myelodysplastic syndrome and benzene exposure among petroleum workers: an international pooled analysis," *Journal of the National Cancer Institute*. 104 (22): 1724–1737.

Schneider, M. S., Mani, M. M. and Masters, F. W. (1991). Gasoline-induced contact burns. *Journal Burn Care Rehabilitation* **12**, 140-143.

Schneider, M. S., Mani, M. M. and Masters, F. W. (1991). Gasoline-induced contact burns. *Journal Burn Care Rehabilitation* **12**, 140-143.

Sciskalska M., Zalewska M., Grzelak A., Milnerowicz H. (2014). The influence of the occupational exposure to heavy metals and tobacco smoke on the selected oxidative stress markers in smelters? *Biology Trace Element Res*ource, 159 (1-3), pp. 59-68

Seshia, S. S., Rjani, K. R., Boeckx, R. L. and Chow, P. N. (1978). The neurological manifestations of chronic inhalation of leaded gasoline. *Development Medical Child Neurology* **20**, 323-334.

Seymour, F. K. and Henry, J. A. (2001). Assessment and management of acute poisoning by petroleum products. *Human Experimental Toxicology* **20**, 551-562.

Short, B. G., Burnett, V. L., Cox, M. G., Bus, J. S. and Swenberg, J. A. (1987). Sitespecific renal cytotoxicity and cell proliferation in male rats exposed to petroleum hydrocarbons. *Laboratory Investigation* **57**, 564-577.

Shukla A, Timbin C, BeruBe K, Gordan T, Mckinney W. and Driscoll K. (2000). Inhaled particulate matter causes expression of nuclear factor (NF)-kappa Brelated genes and oxidant-dependent NF-kappa B activation in vitro. *American Journal Respiratory Cell Molecular Biolology*; 23: 182-187.

Simpson, L. A. and Cruse, C. W. (1981). Gasoline immersion injury. *Plastic and Reconstructive Surgery* **67**, 54-57.

Smith, J. H, Mallet, A. K. and Brantom, P. G, (1996). Ninety days feeding study in Fischer – 344 rats of highly refined petroleum-derived food grade white oils and waves. *Toxicology Pathology* 24: 214-230.

Smith, M. T. (2010). Advances in understanding benzene health effects and susceptibility. *Annual Review Public Health*. **31**: 133–148.

Smith, R and Simon, J. (2014). How To Steal A Million Barrels of Oil. Planet Money. NPR. Retrieved 1 November 2014.

Smith. T..J., Hammand, S. K., Wond, O. (1993). Health effect of gasoline exposure 1: Exposure assessment for US. Distributions workers. *Environmental Health Perspectives* 101:13021 - 13030

Snyder R. and Hedli CC (1996). An overview of benzene metabolism. *Environmental Health Perspect*. 104(06):1165-1171. doi: 10.1289/ehp.961041165. PMID: 9118888; PMCID: PMC1469747.

Snyder, R., Witz, G. and Goldstein, B. D. (1993). "The toxicology of benzene," *Environmental Health Perspectives*. 100: 293–306.

Soini Y, Haapasaari KM, Vaarala MH, Turpeenniemi-Hujanen T, Kärjä V, Karihtala P. (2011). 8-hydroxydeguanosine and nitrotyrosine are prognostic factors in urinary bladder carcinoma. *International Journal of Clinical Experimental Pathology* ;4(3):267-275. PMID: 21487522; PMCID: PMC3071659.

Spatari G., Saitta S., and Giorgianni C. (2013). Interleukin-10 involvement in exposure to low dose of benzene," *Toxicology and Industrial Health*. 31(4):351–354.

Stranks, D. R.; Heffernan M. L., Lee Dow K. C., McTigue P. T., Withers G. R. A. (1970). Chemistry: A structural view. Carlton, Victoria: Melbourne University Press. p. 347. ISBN 978-0-522-83988-3.

Sumathi P. & Neelambikai N. (2016). Evaluation of pulmonary functions in petrol pump workers. *Indian Journal of Clinical Anatomy and Physiology*, 3:189-194.

Takamiya, M., Niitsu, H., Saigusa, K., Kanetake, J. and Aoki, Y. (2003). A case of acute gasoline intoxication at the scene of washing a petrol tank. *Leg Medicine (Tokyo)* **5**, 165-169.

Tan, B. L., Norhaizan, M. E., Liew, W. P. and Sulaiman Rahman, H. (2018). Antioxidant and Oxidative Stress: A Mutual Interplay in Age-Related Diseases. *Frontiers in Pharmacology*, *9*, 1162.

Tannhauser, S. L. (1996). Haematological alternations in rats from xylene and benzene *Vetenary Human Toxicology* 38 (5): 340 - 344.

ten K. and Inge L. (2018). Organic molecules on Mars. *Science*. **360** (6393): 1068–1069. Bibcode:2018Sci...360.1068T. doi:10.1126/science.aat2662. PMID 29880670. S2CID 46952468.

The Guardian (2005). 100 tonnes of pollutants spilled into Chinese river". 25 November 2005. Archived from the original on 10 March 2020. Retrieved 7 January 2020.

The History Of Romanian Oil Industry Archived (2009). Wayback Machine

ToxGuide for Benzene (2010). Agency for Toxic Substances and Disease Registry, Department of Health and Human Services.

Toxicology update. Gasoline. (1989). *Journal of Application Toxicology* **9**, 203-210.

Triggiani, M. Loffredo, S. Granata, F Staiano, R. I. and Marone, G. (2011). "Modulation of mast cell and basophil functions by benzene metabolites," *Current Pharmaceutical Design*, vol. 17, no. 34, pp. 3830–3835.

Trump, B. F., Lipsky, M. M., Jones, T. W., Heatfield, B. M., Higginson, J., Endicott, K. and Hess, H. B. (1984). An Evaluation of the significance of experimental hydrocarbon toxicity in man. Princeton Scientific Publishers Inc., New Jersey

Turnbull E. J. (1982). Canada West's Last Frontier. *Lambton County Historical Society*, p 110

Uche, C. (2008). "Oil, British Interests and the Nigerian Civil War". T*he Journal of African History*. **49** (1): 111–135.

Ullah MI, Alzahrani B, Alsrhani A, Atif M, Alameen AAM, Ejaz H. (2021). Determination of serum tumor necrosis factor-alpha (TNF-α) levels in metabolic syndrome patients from Saudi population. *Pak Journal of Medical*

Science ;37(3):700-705. doi: 10.12669/pjms.37.3.3897. PMID: 34104151; PMCID: PMC8155431.

Ulrich V. H. (1993). The Great Well of China". *Scientific American.* **268** (6): 116–121. Bibcode:1993SciAm.268f.116U. doi:10.1038/scientificamerican0693-116.

Uzma, N., Kumar, B.S. & Hazari, M.A. (2010). Exposure to benzene induces oxidative stress, alters the immune response and expression of p53 in gasoline filling workers. *American Journal of Industrial Medicine.* 53:1264-7120.

Valpey, R., Sumi, S. M., Copass, M. K. and Goble, G. J. (1978). Acute and chronic progressive encephalopathy due to gasoline sniffing. *Neurology* **28**, 507-510.

van Sittert, N.J., Boogaard, P.J., and Beulink, G.D. (1993). Application of the urinary S-phenylmercapturic acid test as a biomarker for low levels of exposure to benzene in industry. *British Journal of Industrial Medicine* 50(5), 460-469.

Vassiliou, M. (2018). Historical Dictionary of the Petroleum Industry, 2nd Edition. Lanham, Maryland: Rowman and Littlefield. Page 50 – 60.

Verma Y. and Rana S.V. (2001). Biological Monitoring of Exposure to Benzene in Petrol Pump Workers and Dry Cleaners. *Industrial Health;* 39:330–333.

Vermeulen, R., Lan, and Q., Zhang. (2005). "Decreased levels of CXC-chemokines in serum of benzene-exposed workers identified by array-based proteomics," *Proceedings of the National Academy of Sciences of the United States of America*. 10 (47). 17041–17046.

Viinanen, R. (2002). A survey of european gasoline exposures for the period 1999 - 2001. *CONCAWE Report* **9/02**.

Walsh, W. A., Scarpa, F. J., Brown, R. S., Ashcraft, K. W., Green, V. A., Holder, T. M. and Amoury, R. A. (1974). Gasoline immersion burn. *New England Journal of Medicine* **291**, 830.

Wang, C. C. and Irons, G. V., Jr. (1961). Acute gasoline intoxication. *Archieve of Environmental Health* **2**, 714-716.

WHO (2008). International Agency for Research on Cancer, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Archived 2008-03-06 at the Wayback Machine, Volumes 1 to 42, Supplement 7.

Wilbur, S., Wohlers, D., Paikoff, S., Keith, L., S. and Faroon, O. (2008). "ATSDR evaluation of potential for human exposure to benzene," *Toxicology and Industrial Health*. 24 (5-6): 399–442.

World's first oil refinery (2018). *The city of Ploiesti". 12 November 2018.*

Xiang, J., Bi, P., Pisaniello, D. and Hansen, A. (2014). Health impact of workplace heat exposure: an epidemiological review. *Industrial Health*. 52 (2): 91-101.

Yardley-Jones A., Anderson, D. and Parke, D. V. (1991). The toxicity of benzene and its metabolism and molecular pathology in human risk assessment". *British Journal of Industrial Medicine.* **48** (7): 437–444.

Zayn Bilkadi (1995). The Oil Weapons. *Saudi Aramco World*, , pp. 20-27

Zhang L, Li HY, Li W, Shen ZY, Wang YD, Ji SR, Wu Y. (2018). An ELISA Assay for Quantifying Monomeric C-Reactive Protein in Plasma. *Front Immunolology* 12;9:511. doi: 10.3389/fimmu.2018.00511. PMID: 29593741; PMCID: PMC5857914.

Zhang, L. McHale, C. M. and Rothman, N. (2010). "Systems biology of human benzene exposure". *Chemico-Biological Interactions*. 184 (1-2):86–93.

Zhou, H., Dehn, D. and Kepa, J. K. (2010). "NAD(P)H: quinone oxidoreductase 1-compromised human bone marrow endothelial cells exhibit decreased adhesion molecule expression and $CD34^{+}$ hematopoietic cell adhesion," *Journal of Pharmacology and Experimental Therapeutics*. 334 (1)260–268.

Zolghadr F., Sadeghizadeh M., Amirizadeh N., Hosseinkhani S. and Nazem S., (2012). How benzene and its metabolites affect human marrow derived mesenchymal stem cells," *Toxicology Letters*, vol. 214, no. 2, pp. 145–153.

ETHICAL APPROVAL

FEDERAL MINISTRY OF ENVIRONMENT

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ENVIRONMENTAL ASSESSMENT DEPARTMENT

Ref: FMEnv/EA/OM3/69/Vol.1/66

Date: 25th January, 2024

Dr. Badejo, David Adedotun,
Plot 39, Biltmore Garden Estate,
Airport Road,
Abuja – FCT.

RE: APPLICATION FOR ETHICAL APPROVAL

Please refer to your letter dated 22nd December, 2023 on the above subject matter.

2. Consequent upon the review of your request, approval is hereby granted for you to embark on research on "Assessment of Health Implication of effects of exposure to petroleum products on oil workers at various NNPC Limited facilities at Abuja and Environs".

3. You are to ensure confidentiality of respondents and abide by standards governing researches of this nature.

4. Thank you for your cooperation.

Abbas.

Dr. Abbas O. Suleiman
Director, Environmental Assessment Department
For: Honourable Minister

ETHICAL CLEARANCE

NNPC

INTERNAL MEMO

To: LEAD HSSE ADVISOR, PSC, NUIMS
Ref: HPRS/23/01/01
Date: 6th January, 2023

Attention: Badejo David Adedotun

Department of Medical Laboratory Science.

University of Benin,

Benin City.

RE: APPLICATION FOR RESEARCH ETHICAL CLEARANCE

I am directed to acknowledge the receipt of your request on the above stated matter.

Consequently, upon the review of your proposal and recommendations by our ethical clearance committee, you are hereby given approval by the MD NMSL to conduct the research on "**Assessment of health implications of effects of exposure to petroleum products on oil workers at various NNPC Limited facilities at Abuja and environs**" at no cost to NNPC.

You are to ensure confidentiality of the respondents, abide by standard ethical requirements and make available to the library of NMSL, a copy of your research findings.

Accept the assurance of the highest esteem of the MD NMSL.

Dr. O. J. Ojo

Manager Health Planning Research and Statistics

For: MD NMSL