ASSESSMENT OF COMMON AMBIENT AIR POLLUTANTS AND RESPIRATORY HEALTH PROBLEMS AMONG ROAD CONSTRUCTION WORKERS IN IMO STATE, NIGERIA

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CERTIFICATION

This is to certify that this Project Proposal on the **"ASSESSMENT OF COMMON AIR POLLUTANTS AND RESPIRATORY HEALTH PROBLEMS AMONG ROAD CONSTRUCTION WORKERS IN IMO STATE, NIGERIA"** was written by JOHN MARK BWALA with registration number 20084774058, in partial fulfilment of the requirements for the award of Masters in Public Health (MPH) degree in the Department of Public Health, School of Health Technology, Federal University of Technology Owerri, Imo State, Nigeria.

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DEDICATION

This work is dedicated to God Almighty who gave me all it takes to start and finish the work. it is also dedicated to my family for their continuous support, and to my friends for having to drive me from Port Harcourt to Owerri early and late hours of the day.

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ABSTRACT

Road construction workers are exposed to air pollutants which can cause respiratory health problems. The general objective of this study is to assess the respiratory health problems among road construction workers in Imo state, Nigeria. This research was a cross sectional study of Road construction workers in Imo state, Nigeria. The multistage sampling technique was used to select 353 subjects from the 3 senatorial zones of Imo State. At the construction sites, gas meters were used to measure gas levels at strategic points. The blood pressure, pulse rate and forced vital capacity readings of the subjects were taken by well-qualified health personnel. The mean CO, SO_2 and SPM levels at Imo West was 182.40±6.41ppm, 516.24±55.03µg/m^3 and 1956.22±30.75µg/m^3 respectively. The mean CO, SO_2 and SPM levels at Imo East was 185.34±6.46ppm, 518.26±55.12µg/m^3 and 1960.17±30.79µg/m^3 respectively. The mean CO, SO_2 and SPM levels at Imo North was 187.51±6.49ppm, 520.14±55.34µg/m^3 and 1956.68±30.25µg/m^3 respectively. In all the senatorial zones, SPSS data analysis using the one-way ANOVA at 0.05 level of significance showed that the levels of CO, SO_2 and SPM was significantly higher ($P<0.05$) than safe levels. The mean Forced Vital Capacity among all the workers in Imo West, Imo East and Imo North was 2263.34±482.33, 2578.80±751.80, and 2382.78±610.42 respectively. The major respiratory health problems among the road construction workers in Imo state include coughing 211 (59.77%); sneezing, 290(82.15%); catarrh, 79(22.38%); sore throat, 72(20.39%); asthma, 29(8.22%); short breadth, 121(34.28%); pneumonia, 66(18.70%); headache, 133(37.68%); and wheezing, 170(48.16%). Road construction workers were advised to always wear their personal protective equipment, especially the nose mask during construction work to prevent respiratory health problems.

Keywords: Ambient Air, Pollutants, Respiratory Health Problems, Road Construction Workers, Imo State, Nigeria

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND INFORMATION

Road construction work is one of the commonest sources of air pollution (WHO, 2021). Engaging in road construction work is not an easy duty to workers whose work materials, work processes and activities may directly or indirectly expose them to health risks. The road construction work involves a wide range of road construction activities which include site clearance, earthworks, material production, crushing and movement of light and heavy-duty trucks on unpaved roads (Heidari & Marr, 2015).

The nature of their duty often exposes them to different occupational diseases and injuries including exposure to air pollutants and respiratory health problems. They are often exposed to air pollutants such as particulate matter (ultrafine: $PM_{1.0}$, fine: $PM_{2.5}$ and coarse: PM_{10}), carbon monoxide, sulfur dioxide and other pollutants that are likely to affect the air quality index (Yankson, Okyere, Wireko-Manu, Dugan, Ampomah, Acquah, Yeboah-Adjei, Okyere, Yalley, Asenso-Gyambibi, Donkor, Mock, & Afukaar, 2024). Also, they can be exposed hazardous substances including silica dust, asphalt, organic solvents, and agents, such as vibration, fumes, heat and vapors generated during the application of hot asphalt to the surface and all these are capable of causing breakdown in respiratory health (Abrar, Cheema & Mahmood, 2017; Mo, Wang, & Xiong, 2022). This is as a result of wide range of hazardous work materials, processes and activities involved in their duties that may directly or indirectly expose them to health risks.

It has been estimated that air pollution alone causes millions of deaths every year around the globe, leading to several diseases, including chronic respiratory related diseases (WHO, 2021).

As reported in a meta-analysis study, the most prevalent hazard causing respiratory conditions among road construction workers include dust, respirable crystalline silica (RCS), fumes, vapours, asbestos, fibres and gases (Boadu, Okeke, Boadi, Bonsu, & Addo, 2023). Road construction workers encounter dust in different construction activities such as during excavation or tunnelling, demolition, sandblasting, grinding, masonry works, rock drilling, road grading, milling and laying, as well as cutting, planing and sanding of wood (Boadu et al., 2023). They also encounter respirable crystalline silica (RCS) quite often from mixing of cement and concrete, demolition, rock drilling, works involving fine particles of cement, fly ash, bricks, mortar and sandstone (United States Department of Labor, 2024). Fumes mostly come across from asphalt, exhaust fumes, welding, and others (Welding Fume, 2020).

Construction workers mostly operate with heavy machinery during roads and highway repairs and construction. Most of the heavy vehicles used in road construction are powered by diesel and worker there are constantly exposed to its' fumes. Exposure to diesel fumes can cause respiratory related issues such as asthma and chronic bronchitis (Mo et al., 2022; Sulaiman, Awang, & Kamaludin, 2020). Worse still, the workers often spend lengthy of time at work which extends their period of exposure to hazardous substances, and consequently to the risk of their respiratory health (WHO, 2020). Higher prevalence of respiratory conditions has been reported on construction workers that spent expended length of exposure to the hazard substances (Fareed, Mohammad, Mahmoud, Mahmoud, Al-Dubai, & Khoja, 2018; Sulaiman et al., 2020).

In many developing countries (including Nigeria), there exist a lot of ongoing road construction activities involving road expansion or construction of new roads, all these increase air pollution risk and respiratory challenges faced by construction workers on the road (da-Silva-Filho, Botelho, Castro, Ferreira, & Silva, 2020). Working in the road construction work attracts a lot of

challenges including challenges associated with low level of education which less prepares such workers to attach seriousness to air pollution and respiratory risk and other underlying risk in their job (Boadu et al., 2020). The road construction workers in such countries are often faced with occupational hazards associated with direct or indirect exposure to hazardous materials. Therefore, addressing air pollution and respiratory health hazards associated with road construction among the workers would require research-oriented occupational health strategies.

The present study deals with the identification of potential air pollutant health hazards and respiratory risks to road construction workers associated with various construction activities. The study will also provide a base for mitigation measure design to be considered during planning phase for the minimization of health risk to workers for future road construction and reconstruction projects.

1.2 STATEMENT OF PROBLEM

Respiratory health problems such as asthma, emphysema, bronchitis, chronic obstructive pulmonary disease etc. are major problems among road construction workers. This can be attributed to the inhalation of pollutants in the air (Ana, Odeshi, Sridhar, and Ige, 2014; Sylla, Faye, Fall, & TAL-DIA, 2017). The removal of earth and rock by digging or blasting, construction of embankments, bridges and tunnels, and removal of vegetation together with emission of gases from combustion of diesel release pollutants that can exacerbate these respiratory problems. The respiratory problems present with a wide variety of symptoms ranging from coughing, wheezing, headaches, throat irritations (Thomas, & Bomar, 2023) to seizures, stroke, coma and death (Potter & Schaefer, 2020).

Road construction workers in Imo State and some other parts of Nigeria are usually faced with environmental issues such as overflooding during the raining season and excessive dust during

dry or harmattan season. At the moment, air pollution and respiratory health challenge posed by road construction workers in the area has remained unaccounted for which has prevented it from receiving the adequate attention.

1.3 OBJECTIVES OF THE STUDY

The general objective of this study is to assess the respiratory health problems among road construction workers in Imo state, Nigeria.

Specific objectives include:

(i) To determine the level of awareness of road construction workers in Imo state, Nigeria to air pollutants and the respiratory problems

(ii) To determine the level of Suspended particulate matter at

(iii) road construction sites in Imo state, Nigeria.

(iv) To determine the level of carbon monoxide at road construction sites in Imo state, Nigeria.

(v) To determine the level of sulfur dioxide at road construction sites in Imo state, Nigeria.

(vi) To determine the blood pressure and pulse rate of the road construction sites in Imo state, Nigeria.

(vii) To determine the Forced Vital Capacity of the road construction sites in Imo state, Nigeria.

(viii) To determine the respiratory health problems among road construction workers in Imo state, Nigeria.

1.4 RESEARCH QUESTIONS

(i) What is the level of awareness of road construction workers in Imo state, Nigeria to air pollutants and the respiratory problems?

(ii) What is the level of particulate matter (PM_{10}) at road construction sites in Imo state, Nigeria?

(iii) What is the level of carbon monoxide at road construction sites in Imo state, Nigeria

(iv) To determine the level of sulfur dioxide at road construction sites in Imo state, Nigeria.

(v) What is the level of blood pressure and pulse rate of the road construction sites in Imo state, Nigeria?

(vi) What is the level of Forced Vital Capacity of the road construction sites in Imo state, Nigeria?

(vii) What are the respiratory health problems among road construction workers in Imo state, Nigeria?

1.5 RESEARCH HYPOTHESIS

H_1: The level of particulate matter (PM_{10}) at road construction sites of Imo state, Nigeria has no impact on the respiratory health status of the workers.

H_2: The level of carbon monoxide at road construction sites of Imo state, Nigeria has no impact on the respiratory health status of the workers.

H_3: The level of sulfur dioxide at road construction sites of Imo state, Nigeria has no impact on the respiratory health status of the workers.

H_4: The Forced Vital Capacity of road construction workers of Imo state, Nigeria is not normal.

1.6 JUSTIFICATON OF STUDY

In Nigeria, road construction workers inhale fumes, dust particles, and other gases that are present during their construction work. This study will bring to light any potential respiratory health problems that the workers might have of which they are not aware of the consequences. Data obtained from this study will be used as a reference to educate road construction workers on the respiratory health problems associated with road construction work. This study will also provide a baseline data for which governmental and non-governmental agencies, policy makers, corporate organizations and industrial managements will use to formulate policies and make appropriate budgetary allocations toward prevention of respiratory problems from exposure to pollutants.

1.7 SCOPE OF STUDY

This study will involve the measurement of air pollutant levels in strategic locations at road construction sites in Imo state, Nigeria. It will also cover the investigation of respiratory problems, their signs and symptoms as a result of exposure to indoor air pollutants as well as measurement of blood pressure, pulse rate and forced vital capacity of the road construction workers.

CHAPTER TWO

LITERATURE REVIEW

2.1 CONCEPTUAL FRAMEWORK

2.1.1 Road Construction

Road construction requires the creation of an engineered continuous roadbed, overcoming geographic obstacles and having grades low enough to permit vehicle or foot travel (Helbing, Molnar, Farkas, & Bolay, 2017). It may be required to meet standards set by law or official guidelines. The process is often begun with the removal of earth and rock by digging or blasting, construction of embankments, bridges and tunnels, and removal of vegetation followed by the laying of pavement material. Roads are designed and built for primary use by vehicular and pedestrian traffic. Storm drainage and environmental considerations are a major concern. Erosion and sediment controls are constructed to prevent detrimental effects. Drainage systems must be capable of carrying the ultimate design flow from the upstream catchment with approval for the outfall from the appropriate authority to a watercourse, creek, river or the sea for drainage discharge (Boederman, 2016; Integrated Urban Drinage 2021).

A borrow pit (source for obtaining fill, gravel, and rock) and a water source should be located near or in reasonable distance to the road construction site. The topsoil and vegetation is removed from the borrow pit and stockpiled for subsequent rehabilitation of the extraction area. Old road surfaces, fences, and buildings may need to be removed before construction can begin. Trees in the road construction area may be marked for retention. These protected trees should not have the topsoil within the area of the tree's drip line removed and the area should be kept clear

of construction material and equipment. Compensation or replacement may be required if a protected tree is damaged. Much of the vegetation may be mulched and put aside for use during reinstatement. The topsoil is usually stripped and stockpiled nearby for rehabilitation of newly constructed embankments along the road. Stumps and roots are removed and holes filled as required before the earthwork begins. Final rehabilitation after road construction is completed will include seeding, planting, watering and other activities to reinstate the area to be consistent with the untouched surrounding areas (Parekh, 2024). Processes during earthwork include excavation, removal of material to spoil, filling, compacting, construction and trimming. If rock or other unsuitable material is discovered it is removed, moisture content is managed and replaced with standard fill compacted to meet the design requirements. Blasting is not frequently used to excavate the roadbed as the intact rock structure forms an ideal road base. When a depression must be filled to come up to the road grade the native bed is compacted after the topsoil has been removed. The fill is made by the "compacted layer method" where a layer of fill is spread then compacted to specifications, under saturated conditions. The process is repeated until the desired grade is reached.

2.1.2 Air Pollutants from Road Construction and Their Health Effects

The most common types of pollution found on a construction site are dust and diesel emissions. The dust types include soil dust, gravel dust, wood dust, silica dust, non-silica mineral dust and demolition dust. These dust particles are called particulate matter. Diesel emissions can be produced on a construction site when an engine combusts diesel fuel for energy, which results in the creation of diesel exhaust. Heavy duty vehicles (dump trucks, cement mixers, transport trucks), road-building machines (excavators, cranes, bulldozers), and stationary engines

(generators, pumps, compressors) are all sources of diesel exhaust emissions. Diesel exhaust gas-based pollutants include carbon dioxide, sulphur and nitrogen compounds, carbon monoxide, and hydrocarbons. Diesel exhaust particulate matter pollutants can include carbon, polycyclic aromatic hydrocarbons, and metals (Geldenhuys, Wattrus & Forbes, 2022)

Particulate matter

Particulate Matter (PM) refers to a generic class of pollutants rather than to a particular, individual pollutant with a specified chemical structure, such as SO_2. PM includes solid or liquid particles suspended in air, regardless of their chemical composition. PM can be either primary (directly emitted) or secondary (formed in the atmosphere through physical and chemical conversion of gaseous precursors such as nitrogen oxides (NO_x), sulfur oxides (SO_x), and volatile organic compounds (VOCs). PM results from the burning of fuel (for example, emissions from power plants), driving on unpaved roads, industrial activity, wood-burning stoves, and from natural sources such as pollen, dust, salt spray, erosion, and mold. PM concentrations can vary within a region or even a city (for example, concentrations will be higher near major highways). The composition of PM differs by geographic area and can vary with season, source, and meteorology (Bell, 2007). Windblown dust can be an important source of PM in desert climates. Variations can also exist at the sub regional level. Particles are generally categorized according to their size, using a measure called aerodynamic diameter. This is the diameter of a uniform sphere of unit density that would attain the same terminal settling velocity as the particle of interest. Aerodynamic diameter is determined by a particle's shape and density, and this measure permits comparison of particles having irregular shapes and different sizes and densities. PM_{10} refers to particles with an aerodynamic diameter of 10 microns or less, whereas $PM_{2.5}$, or fine PM, has an aerodynamic diameter up to 2.5 microns, and ultrafine *PM* particles

have an aerodynamic diameter up to 0.1 microns. Coarse PM ($PM_{10-2.5}$) refers to particles with an aerodynamic diameter between 2.5 and 10 microns. Total suspended particles (TSP) refer to almost all particles in the air and are typically measured as PM mass of particles up to about 45 microns in aerodynamic diameter.

A particle's size is related to its source and determines how it is transported in the atmosphere, where it is deposited in the environment, and where it is deposited in the respiratory system. Smaller particles are of special health concern because they penetrate more deeply into the lung. Such particles are typically generated through combustion processes. Diesel exhaust, a combination of gases and particles, is of particular concern because of widespread diesel use and because the resulting particles are extremely small (< 1 μm) (Kagawa, 2002). In addition to particles, the gas phase of diesel contains numerous hazardous air pollutants such as benzene, formaldehyde, and polycyclic aromatic hydrocarbons.

Size, shape and solubility: The size of the particle is a main determinant of where in the respiratory tract the particle will come to rest when inhaled. Larger particles are generally filtered in the nose and throat via cilia and mucus, but particulate matter smaller than about 10 micrometers (PM_{10}) can settle in the bronchi and lungs and cause health problems. The 10 micrometer size does not represent a strict boundary between respirable and non-respirable particles, but has been agreed upon for monitoring of airborne particulate matter by most regulatory agencies. Because of their small size, particles on the order of 10 micrometers or less can penetrate the deepest part of the lungs such as the bronchioles or alveoli.

Similarly, so called fine PM, particles smaller than 2.5 micrometers ($PM_{2.5}$) tend to penetrate into the gas exchange regions of the lung (alveolus), and very small particles (< 100 nanometers) may

pass through the lungs to affect other organs. Penetration of particles is not wholly dependent on their size; shape and chemical composition also play a part. To avoid this complication, simple nomenclature is used to indicate the different degrees of relative penetration of a PM particle into the cardiovascular system. Inhalable particles penetrate no further than the bronchi as they are filtered out by the cilia. Thoracic particles can penetrate right into terminal bronchioles whereas PM which can penetrate to alveoli, the gas exchange area, and the circulatory system and are termed respirable particles. In analogy, the inhalable dust fraction is the fraction of dust entering nose and mouth which may be deposited anywhere in the respiratory tract. The thoracic fraction is the fraction that enters the thorax and is deposited within the lung's airways. The respirable fraction is what is deposited in the gas exchange regions (alveoli).

The smallest particles, less than 100 nanometers (nanoparticles), may be even more damaging to the cardiovascular system. Nanoparticles can pass through cell membranes and migrate into other organs, including the brain. Particles emitted from modern diesel engines (commonly referred to as Diesel Particulate Matter, or DPM) are typically in the size range of 100 nanometers (0.1 micrometer). These soot particles also carry carcinogens like benzopyrenes adsorbed on their surface. Particulate mass is not a proper measure of the health hazard, because one particle of 10 μm diameter has approximately the same mass as 1 million particles of 100 nm diameter, but is much less hazardous, as it unlikely to enter the alveoli. Legislative limits for engine emissions based on mass are therefore not protective.

The site and extent of absorption of inhaled gases and vapors are determined by their solubility in water. Absorption is also dependent upon air flow rates and the partial pressure of the gases in the inspired air. The fate of a specific contaminant is dependent upon the form in which it exists (aerosol or particulate). Inhalation also depends upon the breathing rate of the subject. Another

complexity not entirely documented is how the shape of PM can affect health, except for the needle-like shape of asbestos which can lodge itself in the lungs. Geometrically angular shapes have more surface area than rounder shapes, which in turn affects the binding capacity of the particle to other, possibly more dangerous substances.

Ambient levels of PM, as indicated by PM_{10}, $PM_{2.5}$, or other measures, have been associated with health effects including increased hospital and emergency room admissions, respiratory symptoms, decline in pulmonary function, exacerbation of chronic respiratory and cardiovascular diseases, and premature mortality (U.S. EPA, 2003). Laboratory animals exposed to PM experienced a range of responses including inflammation and pulmonary injury (Dye, 2001). The health risk of PM exposure may depend on characteristics such as the particulates' content of metals, acidity, organics, sulfates, or particular combinations of these (Health Effects Institute, 2002). Identifying the aspects of PM that are harmful is a critical research need. Similarly, the biological mechanisms by which PM causes premature mortality are not well understood. Leading hypotheses focus on reflexes in the lung that lead to autonomic nervous systems changes, perhaps predisposing to arrhythmias, and on inflammation that in turn predisposes to thrombosis or related changes (Brook, 2004).

Health Effects: Increased levels of fine particles in the air as a result of anthropogenic particulate air pollution are consistently and independently related to the most serious health effects. When these particles are inhaled, the lungs produce mucous to trap the particles and tiny hairs wiggle to move the mucous and particles out of the lung. The mucous leaves the airway by coughing or swallowing. If the particle is small and it gets very far into the lungs, special cells in the lung trap the particles and then they can't get out and this can result in lung disease, emphysema, lung cancer. Because the $PM_{2.5}$ travels deeper into the lungs and because the $PM_{2.5}$

is made up things that are more toxic (like heavy metals and cancer causing organic compounds), it can have worse health effects than the bigger PM_{10}. The effects of inhaling particulate matter include asthma, lung cancer, cardiovascular disease, respiratory diseases, premature delivery, birth defects, and premature death.

A study indicated that $PM_{2.5}$ leads to high plaque deposits in arteries, causing vascular inflammation and atherosclerosis which can lead to heart attacks and other cardiovascular problems (Pope, 2002). The World Health Organization (WHO) estimated in 2005 that fine particulate air pollution ($PM_{2.5}$), causes about 3% of mortality from cardiopulmonary disease, about 5% of mortality from cancer of the trachea, bronchus, and lung, and about 1% of mortality from acute respiratory infections in children under 5 years, worldwide (Cohen, et al., 2005). Short-term exposure at elevated concentrations can significantly contribute to heart disease.

Sulfur dioxide

Sulfur dioxide (SO_2), is a water-soluble gas that was a primary component of the 1952 London fog. Sulfur oxides are produced from the combustion of sulfur-containing fuels and materials, such as coal and metal ores. Power plants are the main source of SO_2 emissions. Other sources are industrial boilers, trains, ships, and metal-processing facilities. Household use of coal can contribute significant amounts of SO_2 as well. In some areas, such as parts of China, coal is the primary fuel for cooking and heating and causes high levels of SO_2 indoors. Natural sources of SO_2 include volcanoes. SO_2 can be converted to sulfuric acid, and therefore contributes to acid deposition, which harms vegetation, other materials, and wildlife. SO_2 also contributes to the formation of particulate matter. Sulfate aerosols, a major component of fine particulate matter, can travel far from their sources. Tall stacks of power plants often release pollution above the

inversion layer, which reduces local pollution but allows pollutants to migrate long distances and undergo chemical transformation.

Health effects: Because SO_2 is highly soluble in water, most inhaled SO_2 is absorbed by the mucous membranes of the upper airways with little reaching the lung; however, increased ventilation and oral breathing, such as from exercise, can raise the dose delivered to the lung (Agency for Toxic Substances and Disease Registry, 2008). SO_2 exposure has been associated with reduced lung function, bronchoconstriction (increased airway resistance), respiratory symptoms, hospitalizations from cardiovascular and respiratory causes, eye irritation, adverse pregnancy outcomes, and mortality. However, it is difficult to attribute these reported associations to SO_2 itself, because it is a precursor to particulate matter and generally exists as a component of a complex, combustion-related pollutant mixture. Experimental studies suggest that some persons with asthma may be particularly sensitive to SO_2 itself. Controlled exposure studies have shown that effects can occur with very short-term exposure (for example, ten minutes) in some people with asthma, whereas epidemiological research has shown effects associated with long-term exposure (for example, yearly levels). Short-term exposures to high levels of sulfur dioxide can be life-threatening. Exposure to 100 parts of sulfur dioxide per million parts of air (ppm) is considered immediately dangerous to life and health. Previously healthy nonsmoking miners who breathed sulfur dioxide released as a result of an explosion in an underground copper mine developed burning of the nose and throat, breathing difficulties, and severe airway obstructions. Long-term exposure to persistent levels of sulfur dioxide can also affect your health. Lung function changes have been observed in some workers exposed to 0.4–3.0 ppm sulfur dioxide for 20 years or more. However, these workers were also exposed to other chemicals, making it difficult to attribute their health effects to sulfur dioxide exposure alone.

Additionally, exercising asthmatics are sensitive to the respiratory effects of low concentrations (0.25 ppm) of sulfur dioxide. Long-term studies surveying large numbers of children have indicated possible associations between sulfur dioxide pollution and respiratory symptoms or reduced breathing ability. Children who have breathed sulfur dioxide pollution may develop more breathing problems as they get older, may make more emergency room visits for treatment of wheezing fits, and may get more respiratory illnesses than is typical for children. However, studies like these are unable to provide conclusive evidence about sulfur dioxide's effects on children's health because many other pollutants are also present in the air.

Carbon monoxide

Carbon monoxide (CO) is a colorless, odorless gas formed by incomplete combustion of organic matter due to insufficient oxygen supply to enable complete oxidation to carbon dioxide (CO_2). Motor vehicles contribute the majority of CO emissions to outdoor air, and consequently CO concentrations tend to be higher in areas with high traffic density and during times of high traffic volume. Carbon monoxide levels may also be high in congested urban areas with slow-moving traffic. Automobiles emit more CO during periods of colder temperature and while idling or moving slowly. Other sources include off-road vehicles and wildfires. Wood burning also produces significant CO emissions in some areas. Elevated CO levels typically occur during colder periods because of increased vehicular emissions and inversion conditions that prevent pollution from dispersing.

Health effects: Mild acute CO poisoning can cause light-headedness, confusion, headaches, vertigo, and flu-like effects. Larger exposures can lead to significant toxicity of the central nervous system and heart, and death. Following acute poisoning, long-term sequelae often occur.

Carbon monoxide can also have severe effects on the fetus of a pregnant woman. Chronic exposure to low levels of carbon monoxide can lead to depression, confusion, and memory loss. Carbon monoxide mainly causes adverse effects in humans by combining with hemoglobin to form carboxyhemoglobin (HbCO) in the blood. This prevents hemoglobin from carrying oxygen to the tissues, effectively reducing the oxygen-carrying capacity of the blood, leading to hypoxia. Additionally, myoglobin and mitochondrial cytochrome oxidase are thought to be adversely affected. Carboxyhemoglobin can revert to hemoglobin, but the recovery takes time because the HbCO complex is fairly stable.

Signs and symptoms: Inhaling CO gas can lead to hypoxic injury, nervous system damage, and even death. Different people and populations may have different carbon monoxide tolerance levels. On average, exposures at 100 ppm or greater is dangerous to human health (Prockop and Chichkova, 2007). Carbon monoxide exposure may lead to a significantly shorter life span due to heart damage (Henry, et al., 2006). The carbon monoxide tolerance level for any person is altered by several factors, including activity level, rate of ventilation, a pre-existing cerebral or cardiovascular disease, cardiac output, anemia, sickle cell disease and other hematological disorders, barometric pressure, and metabolic rate (Lipman, 2006).

Acute Poisoning: The main manifestations of carbon monoxide poisoning develop in the organ systems most dependent on oxygen use, the central nervous system and the heart. The initial symptoms of acute carbon monoxide poisoning include headache, nausea, malaise, and fatigue. These symptoms are often mistaken for a virus such as influenza or other illnesses such as food poisoning or gastroenteritis (Goldfrank, et al., 2002). Headache is the most common symptom of acute carbon monoxide poisoning; it is often described as dull, frontal, and continuous. Increasing exposure produces cardiac abnormalities including fast heart rate, low blood pressure,

and cardiac arrhythmia (Choi, 2001). Central nervous system symptoms include delirium, hallucinations, dizziness, unsteady gait, confusion, seizures, central nervous system depression, unconsciousness, respiratory arrest, and death (Weaver, 2009). Less common symptoms of acute carbon monoxide poisoning include myocardial ischemia, atrial fibrillation, pneumonia, pulmonary edema, high blood sugar, lactic acidosis, muscle necrosis, acute kidney failure, skin lesions, and visual and auditory problems (Gandini, et al., 2001). One of the major concerns following acute carbon monoxide poisoning is the severe delayed neurological manifestations that may occur. Problems may include difficulty with higher intellectual functions, short-term memory loss, dementia, amnesia, psychosis, irritability, a strange gait, speech disturbances, Parkinson's disease-like syndromes, cortical blindness, and a depressed mood. Depression may occur in those who did not have pre-existing depression. These delayed neurological sequelae may occur in up to 50% of poisoned people after 2 to 40 days (Goldfrank, 2002). It is difficult to predict who will develop delayed sequelae; however, advanced age, loss of consciousness while poisoned, and initial neurological abnormalities may increase the chance of developing delayed symptoms.

Chronic poisoning: Chronic exposure to relatively low levels of carbon monoxide may cause persistent headaches, lightheadedness, depression, confusion, memory loss, nausea and vomiting (Fawcett, et al., 1992). It is unknown whether low-level chronic exposure may cause permanent neurological damage. Typically, upon removal from exposure to carbon monoxide, symptoms usually resolve themselves, unless there has been an episode of severe acute poisoning. Chronic exposure may worsen cardiovascular symptoms in some people and might increase the risk of developing atherosclerosis. Long-term exposures to carbon monoxide present the greatest risk to persons with coronary heart disease and in females who are pregnant.

Pathophysiology: The precise mechanisms by which the effects of carbon monoxide are induced upon bodily systems, are complex and not yet fully understood (Hardy and Thom, 1994). Known mechanisms include carbon monoxide binding to hemoglobin, myoglobin and mitochondrial cytochrome oxidase and restricting oxygen supply, and carbon monoxide causing brain lipid peroxidation (Weaver, 2009).

Hemoglobin: Carbon monoxide has a higher diffusion coefficient compared to oxygen and the only enzyme in the human body that produces carbon monoxide is heme oxygenase which is located in all cells and breaks down heme. Under normal conditions carbon monoxide levels in the plasma are approximately 0 mmHg because it has a higher diffusion coefficient and the body easily gets rid of any CO made. When CO is not ventilated it binds to hemoglobin, which is the principal oxygen-carrying compound in blood; this produces a compound known as carboxyhemoglobin. The traditional belief is that carbon monoxide toxicity arises from the formation of carboxyhemoglobin, which decreases the oxygen-carrying capacity of the blood and inhibits the transport, delivery, and utilization of oxygen by the body. The affinity between hemoglobin and carbon monoxide is approximately 230 times stronger than the affinity between hemoglobin and oxygen so hemoglobin binds to carbon monoxide in preference to oxygen (Bateman, 2003). Hemoglobin is a tetramer with four oxygen binding sites. The binding of carbon monoxide at one of these sites increases the oxygen affinity of the remaining three sites, which causes the hemoglobin molecule to retain oxygen that would otherwise be delivered to the tissue (Gorman, et al., 2003). Because of the increased affinity between hemoglobin and oxygen during carbon monoxide poisoning, little oxygen will actually be released in the tissues. This causes hypoxic tissue injury (Goldfrank, et al., 2002). Hemoglobin acquires a bright red color when converted into carboxyhemoglobin.

Myoglobin: Carbon monoxide also binds to the hemeprotein myoglobin. It has a high affinity for myoglobin, about 60 times greater than that of oxygen. Carbon monoxide bound to myoglobin may impair its ability to utilize oxygen (Bateman, 2003). This causes reduced cardiac output and hypotension, which may result in brain ischemia. A delayed return of symptoms have been reported. This results following a recurrence of increased carboxyhemoglobin levels; this effect may be due to a late release of carbon monoxide from myoglobin, which subsequently binds to hemoglobin (Omaye, 2002).

Central nervous system effects: The mechanism that is thought to have a significant influence on delayed effects involves formed blood cells and chemical mediators, which cause brain lipid peroxidation (degradation of unsaturated fatty acids). Carbon monoxide causes endothelial cell and platelet release of nitric oxide, and the formation of oxygen free radicals including peroxynitrite (Hardy and Thom, 1994). In the brain, this causes further mitochondrial dysfunction, capillary leakage, leukocyte sequestration, and apoptosis (Blumenthal, 2001). The result of these effects is lipid peroxidation, which causes delayed reversible demyelinization of white matter in the central nervous system known as Grinker myelinopathy, which can lead to edema and necrosis within the brain (Gorman, et al., 2003). This brain damage occurs mainly during the recovery period. This may result in cognitive defects, especially affecting memory and learning, and movement disorders. These disorders are typically related to damage to the cerebral white matter and basal ganglia. Hallmark pathological changes following poisoning are bilateral necrosis of the white matter, globus pallidus, cerebellum, hippocampus and the cerebral cortex (Fukuhara, et al., 1996).

Pregnancy: Carbon monoxide poisoning in pregnant women may cause severe adverse fetal effects. Poisoning causes fetal tissue hypoxia by decreasing the release of maternal oxygen to the

fetus. Carbon monoxide also crosses the placenta and combines with fetal hemoglobin, causing more direct fetal tissue hypoxia. Additionally, fetal hemoglobin has a 10 to 15% higher affinity for carbon monoxide than adult hemoglobin, causing more severe poisoning in the fetus than in the adult (Omaye, 2002). Elimination of carbon monoxide is slower in the fetus, leading to an accumulation of the toxic chemical. The level of fetal morbidity and mortality in acute carbon monoxide poisoning is significant, so despite mild maternal poisoning or following maternal recovery, severe fetal poisoning or death may still occur.

2.1.3 Respiratory Diseases Caused By Air Pollutants

Respiratory disease is a medical term that encompasses pathological conditions affecting the organs and tissues that make gas exchange possible in higher organisms, and includes conditions of the upper respiratory tract, trachea, bronchi, bronchioles, alveoli, pleura and pleural cavity, and the nerves and muscles of breathing. Respiratory diseases range from mild and self-limiting, such as the common cold, to life-threatening entities like bacterial pneumonia, pulmonary embolism, and lung cancer. In humans the anatomical features of the respiratory system include airways, lungs, and the respiratory muscles. Molecules of oxygen and carbon dioxide are passively exchanged, by diffusion, between the gaseous external environment and the blood (Powers & Dhamoon, 2023). This exchange process occurs in the alveolar region of the lungs. The respiratory system can be subdivided into an upper respiratory tract and a lower respiratory tract based on anatomical features. The upper respiratory tract includes the nasal passages, pharynx and the larynx, while the lower respiratory tract is comprised of the trachea, the primary bronchi and lungs.

The primary function of the respiratory system is to supply the blood with oxygen in order for the blood to deliver oxygen to all parts of the body. The respiratory system does this through breathing. When we breathe, we inhale oxygen and exhale carbon dioxide. This exchange of gases is the respiratory system's means of getting oxygen to the blood. The respiratory system lies dormant in the human fetus during pregnancy. At birth, the respiratory system becomes fully functional upon exposure to air, although some lung development and growth continues throughout childhood. Pre-term birth can lead to infants with under-developed lungs. Smoking and air pollution are two common causes of respiratory problems. Disorders of the respiratory system can be classified into four general areas (Wikipedia Contributors, 2019):

(i) Obstructive conditions (e.g., emphysema, bronchitis, asthma attacks)

(ii) Restrictive conditions (e.g., fibrosis, sarcoidosis, alveolar damage, pleural effusion)

(iii) Vascular diseases (e.g., pulmonary edema, pulmonary embolism, pulmonary hypertension)

(iv) Infectious, environmental and other "diseases" (e.g., pneumonia, tuberculosis, asbestosis, particulate pollutants): Coughing is of major importance, as it is the body's main method to remove dust, mucus, saliva, and other debris from the lungs. Inability to cough can lead to infection. Deep breathing exercises may help keep finer structures of the lungs clear from particulate matter, etc.

The respiratory tract is constantly exposed to microbes due to the extensive surface area, which is why the respiratory system includes many mechanisms to defend itself and prevent pathogens from entering the body. Common Respiratory Disorders Include:

(i) Chronic Obstructive Pulmonary Disease (COPD) - Irritation of the lungs can lead to asthma, emphysema, and chronic bronchitis and people can develop two or three of these together.

(ii) Chronic Bronchitis - Any irritant reaching the bronchi and bronchioles will stimulate an increased secretion of mucus. In chronic bronchitis the air passages become clogged with mucus, and this leads to a persistent cough.

(iii) Emphysema - The delicate walls of the alveoli break down, reducing the gas exchange area of the lungs. The condition develops slowly and is seldom a direct cause of death.

(iv) Asthma - Periodic constriction of the bronchi and bronchioles makes it more difficult to breathe.

(v) Pneumonia - An infection of the alveoli. It can be caused by many kinds of both bacteria and viruses. Tissue fluids accumulate in the alveoli reducing the surface area exposed to air. If enough alveoli are affected, the patient may need supplemental oxygen.

2.1.3.1 Chronic Obstructive Pulmonary Disease (COPD)

Chronic obstructive pulmonary disease (COPD) is a type of obstructive lung disease characterized by long term poor airflow. The main symptoms include shortness of breath and cough with sputum production. COPD typically worsens over time (Vestbo, 2013). Tobacco smoking is the most common cause of COPD, with a number of other factors such as air pollution and genetics playing a smaller role (Decramer, et al., 2012). In the developing world, one of the common sources of air pollution is poorly vented heating and cooking fires. Long-term exposure to these irritants causes an inflammatory response in the lungs resulting in narrowing of the small airways and breakdown of lung tissue. The diagnosis is based on poor airflow as measured by lung function tests (Nathell, et al., 2007). In contrast to asthma, the

airflow reduction does not improve much with the use of a bronchodilator. Most cases of COPD can be prevented by reducing exposure to risk factors. This includes decreasing rates of smoking and improving indoor and outdoor air quality. While treatment can slow worsening there is no cure. COPD treatments include stopping smoking, vaccinations, respiratory rehabilitation, and often inhaled bronchodilators and steroids (Decramer, et al., 2012). Some people may benefit from long-term oxygen therapy or lung transplantation. In those who have periods of acute worsening, increased use of medications and hospitalization may be needed.

Signs and symptoms

Wheezing: The most common symptoms of COPD are sputum production, shortness of breath, and a productive cough (Vestbo, 2013). These symptoms are present for a prolonged period of time and typically worsen over time.

Cough: A chronic cough is often the first symptom to develop. When it persists for more than three months each year for at least two years, in combination with sputum production and without another explanation, there is by definition chronic bronchitis. This condition can occur before COPD fully develops. The amount of sputum produced can change over hours to days. In some cases, the cough may not be present or may only occur occasionally and may not be productive. Some people with COPD attribute the symptoms to a "smoker's cough". Sputum may be swallowed or spat out, depending often on social and cultural factors. Vigorous coughing may lead to rib fractures or a brief loss of consciousness. Those with COPD often have a history of "common colds" that last a long time (Vestbo, 2013).

Shortness of breath: Shortness of breath is often the symptom that most bothers people. Typically the shortness of breath is worse on exertion of a prolonged duration and worsens over

time. In the advanced stages, it occurs during rest and may be always present. It is a source of both anxiety and a poor quality of life in those with COPD. Many people with more advanced COPD breathe through pursed lips and this action can improve shortness of breath in some (Nathan and Sean, 2013).

Etiology

The primary cause of COPD is tobacco smoke, with occupational exposure and pollution from indoor fires being significant causes in some countries. Typically these exposures must occur over several decades before symptoms develop. A person's genetic makeup also affects the risk.

Air pollution: Poorly ventilated cooking fires, often fueled by coal or biomass fuels such as wood and animal dung, lead to indoor air pollution and are one of the most common causes of COPD in developing countries (Kennedy, et al., 2007). These fires are a method of cooking and heating for nearly 3 billion people with their health effects being greater among women due to more exposure. They are used as the main source of energy in sub-Saharan Africa.

Occupational exposures: Intense and prolonged exposure to workplace dusts, chemicals and fumes increase the risk of COPD in both smokers and nonsmokers. Workplace exposures are believed to be the cause in 10–20% of cases (Devereux, 2006). A number of industries and sources have been implicated, including high levels of dust in coal mining, gold mining, and the cotton textile industry, occupations involving cadmium and isocyanates, and fumes from welding. Working in agriculture is also a risk. Silica dust exposure can also lead to COPD, with the risk unrelated to that for silicosis (Rushton, 2007). The negative effects of dust exposure and cigarette smoke exposure appear to be additive or possibly more than additive.

Pathophysiology

COPD is a type of obstructive lung disease in which chronic incompletely reversible poor airflow (airflow limitation) and inability to breathe out fully (air trapping) exist. The poor airflow is the result of breakdown of lung tissue (known as emphysema) and small airways disease (known as obstructive bronchiolitis). The relative contributions of these two factors vary between people. Severe destruction of small airways can lead to the formation of large air pockets known as bullae that replace lung tissue. This form of disease is called bullous emphysema (Devereux, 2006). COPD develops as a significant and chronic inflammatory response to inhaled irritants. Chronic bacterial infections may also add to this inflammatory state. The inflammatory cells involved include neutrophil granulocytes and macrophages, two types of white blood cell. Those who smoke additionally have Tc1 lymphocyte involvement and some people with COPD have eosinophil involvement similar to that in asthma. Part of this cell response is brought on by inflammatory mediators such as chemotactic factors. Other processes involved with lung damage include oxidative stress produced by high concentrations of free radicals in tobacco smoke and released by inflammatory cells, and breakdown of the connective tissue of the lungs by proteases that are insufficiently inhibited by protease inhibitors. The destruction of the connective tissue of the lungs is what leads to emphysema, which then contributes to the poor airflow and, finally, poor absorption and release of respiratory gases. General muscle wasting that often occurs in COPD may be partly due to inflammatory mediators released by the lungs into the blood. Narrowing of the airways occurs due to inflammation and scarring within them. This contributes to the inability to breathe out fully. The greatest reduction in air flow occurs when breathing out, as the pressure in the chest is compressing the airways at this time.

2.1.3.2 Bronchitis

Bronchitis is inflammation of the bronchi (large and medium-sized airways) in the lungs. Symptoms include coughing up mucus, wheezing, shortness of breath, and chest discomfort (Albert, 2010). Bronchitis is divided into two types: acute and chronic.

Acute bronchitis

Acute bronchitis, also known as a chest cold, is short term inflammation of the bronchi of the lungs. The most common symptom is a cough. Other symptoms include coughing up mucus, wheezing, shortness of breath, fever, and chest discomfort. The infection may last from a few to ten days. The cough may persist for several weeks afterwards with the total duration of symptoms usually around three weeks. Some have symptoms for up to six weeks. In more than 90% of cases the cause is a viral infection (Albert, 2010). These viruses may be spread through the air when people cough or by direct contact. Risk factors include exposure to tobacco smoke, dust, and other air pollution. A small number of cases are due to high levels of air pollution or bacteria such as *Mycoplasma pneumoniae* or *Bordetella pertussis*. Diagnosis is typically based on a person's signs and symptoms. Other causes of similar symptoms include asthma, pneumonia, bronchiolitis, bronchiectasis, and COPD. A chest X-ray may be useful to detect pneumonia. Prevention is by not smoking and avoiding other lung irritants. Frequent hand washing may also be protective. Treatment of acute bronchitis typically involves rest, paracetamol (acetaminophen), and NSAIDs to help with the fever (Tackett and Atkins, 2012).

Chronic bronchitis

Chronic bronchitis is defined as a productive cough that lasts for three months or more per year for at least two years (Vestbo, 2013). Most people with chronic bronchitis have chronic obstructive pulmonary disease (COPD). Tobacco smoking is the most common cause, with a number of other factors such as air pollution and genetics playing a smaller role. Additionally, chronic inhalation of air pollution or irritating fumes or dust from hazardous exposures in occupations such as coal mining, grain handling, textile manufacturing, livestock farming, and metal moulding may also be a risk factor for the development of chronic bronchitis. Symptoms of chronic bronchitis may include wheezing and shortness of breath, especially upon exertion and low oxygen saturations. The cough is often worse soon after awakening and the sputum produced may have a yellow or green color and may be streaked with specks of blood (Cohen and Powderly, 2004). Treatments include quitting smoking, vaccinations, rehabilitation, and often inhaled bronchodilators and steroids. Some people may benefit from long-term oxygen therapy or lung transplantation (Rabe, et al., 2007).

2.1.3.3 Asthma

Asthma is a common long term inflammatory disease of the airways of the lungs. It is characterized by variable and recurring symptoms, reversible airflow obstruction, and bronchospasm. Symptoms include episodes of wheezing, coughing, chest tightness, and shortness of breath (Martinez, 2007). These episodes may occur a few times a day or a few times per week. Depending on the person they may become worse at night or with exercise. Asthma is thought to be caused by a combination of genetic and environmental factors. Environmental factors include exposure to air pollution and allergens. Other potential triggers include

medications such as aspirin and beta blockers. Diagnosis is usually based on the pattern of symptoms, response to therapy over time, and spirometry (Lemanske and Busse, 2010). Asthma is classified according to the frequency of symptoms, forced expiratory volume in one second (FEV_1), and peak expiratory flow rate (Yawn, 2008). It may also be classified as atopic or non-atopic where atopy refers to a predisposition toward developing a type 1 hypersensitivity reaction. There is no cure for asthma. Symptoms can be prevented by avoiding triggers, such as allergens and irritants, and by the use of inhaled corticosteroids.

Signs and symptoms

Wheezing: Asthma is characterized by recurrent episodes of wheezing, shortness of breath, chest tightness, and coughing. Sputum may be produced from the lung by coughing but is often hard to bring up (Jindal, 2011). During recovery from an attack, it may appear pus-like due to high levels of white blood cells called eosinophils. Symptoms are usually worse at night and in the early morning or in response to exercise or cold air. Some people with asthma rarely experience symptoms, usually in response to triggers, whereas others may have marked and persistent symptoms.

Associated conditions: A number of other health conditions occur more frequently in those with asthma, including gastro-esophageal reflux disease (GERD), rhinosinusitis, and obstructive sleep apnea (Boulet, 2009). Psychological disorders are also more common, with anxiety disorders occurring in between 16–52% and mood disorders in 14–41% (Andrew and Harry, 2010).

Etiology

Asthma is caused by a combination of complex and incompletely understood environmental and genetic interactions (Miller and Ho, 2008). These factors influence both its severity and its

responsiveness to treatment. It is believed that the recent increased rates of asthma are due to changing epigenetics (heritable factors other than those related to the DNA sequence) and a changing living environment (Dietert, 2011). Onset before age 12 is more likely due to genetic influence, while onset after 12 is more likely due to environmental influence.

Environmental: Many environmental factors have been associated with asthma's development and exacerbation including allergens, air pollution, and other environmental chemicals (Kelly and Fussell, 2011). Smoking during pregnancy and after delivery is associated with a greater risk of asthma-like symptoms. Low air quality from factors such as traffic pollution or high ozone level has been associated with both asthma development and increased asthma severity (Gold and Wright, 2005). Exposure to indoor volatile organic compounds may be a trigger for asthma. Formaldehyde exposure, for example, has a positive association (McGwin, et al., 2010). Asthma is associated with exposure to indoor allergens. Common indoor allergens include dust mites, cockroaches, animal dander, and mold.

Exacerbation: Some individuals will have stable asthma for weeks or months and then suddenly develop an episode of acute asthma. Different individuals react to various factors in different ways. Most individuals can develop severe exacerbation from a number of triggering agents. Home factors that can lead to exacerbation of asthma include dust, animal dander (especially cat and dog hair), cockroach allergens and mold (Baxi and Phipatankul, 2010). Perfumes are a common cause of acute attacks in women and children. Both viral and bacterial infections of the upper respiratory tract can worsen the disease. Psychological stress may worsen symptoms. It is thought that stress alters the immune system and thus increases the airway inflammatory response to allergens and irritants (Gold and Wright, 2005).

Pathophysiology

Asthma is the result of chronic inflammation of the conducting zone of the airways (most especially the bronchi and bronchioles), which subsequently results in increased contractability of the surrounding smooth muscles. This among other factors leads to bouts of narrowing of the airway and the classic symptoms of wheezing. The narrowing is typically reversible with or without treatment. Occasionally the airways themselves change. Typical changes in the airways include an increase in eosinophils and thickening of the lamina reticularis (Kumar, et al., 2008). Chronically the airways' smooth muscle may increase in size along with an increase in the numbers of mucous glands. Other cell types involved include: T lymphocytes, macrophages, and neutrophils. There may also be involvement of other components of the immune system including: cytokines, chemokines, histamine, and leukotrienes among others (Mason, et al., 2010).

Diagnosis

There is currently no precise test for asthma with the diagnosis typically based on the pattern of symptoms and response to therapy over time. A diagnosis of asthma should be suspected if there is a history of: recurrent wheezing, coughing or difficulty breathing and these symptoms occur or worsen due to exercise, viral infections, allergens or air pollution. Spirometry is then used to confirm the diagnosis. In children under the age of six the diagnosis is more difficult as they are too young for spirometry.

Spirometry: Spirometry measures the amount of airflow obstruction present and is generally carried out after the use of a bronchodilator, a medication to open up the airways. Two main components are measured to make the diagnosis: the forced expiratory volume in one second

(FEV_1), which is the greatest volume of air that can be breathed out in the first second of a breath, and the forced vital capacity (FVC), which is the greatest volume of air that can be breathed out in a single large breath. If the FEV_1 measured by this technique improves more than 12% following administration of a bronchodilator such as salbutamol, this is supportive of the diagnosis. It however may be normal in those with a history of mild asthma, not currently acting up (mason, et al., 2010). It is reasonable to perform spirometry every one or two years to follow how well a person's asthma is controlled.

2.1.3.4 Pneumonia

Pneumonia is an inflammatory condition of the lung affecting primarily the microscopic air sacs known as alveoli (McLuckie, 2009). Typical signs and symptoms include a cough with phlegm, chest pain, fever, and trouble breathing. Symptoms can vary from mild to severe. People who are old or very young may not have typical symptoms. Usually people begin improving within three days of starting treatment; however, they may feel tired for more than a month afterwards (Ashby and Turkington, 2007). Pneumonia is usually caused by infection with viruses or bacteria and less commonly by other microorganisms, certain medications and conditions such as autoimmune diseases (Jeffrey, 2010). Risk factors include other lung diseases such as cystic fibrosis, COPD, asthma, diabetes, heart failure, a history of smoking, a poor ability to cough such as following a stroke, or a weak immune system. Diagnosis is often based on the symptoms and physical examination. Chest X-ray, blood tests, and culture of the sputum may help confirm the diagnosis. Vaccines to prevent certain types of pneumonia are available. Other methods of prevention include hand washing and not smoking. Treatment depends on the underlying cause. Pneumonia believed to be due to bacteria is treated with antibiotics. If the pneumonia is severe,

the affected person is generally hospitalized. Oxygen therapy may be used if oxygen levels are low.

Signs and symptoms

People with infectious pneumonia often have a productive cough, fever accompanied by shaking chills, shortness of breath, sharp or stabbing chest pain during deep breaths, and an increased rate of breathing (Hoare and Lim, 2006). The typical signs and symptoms in children under five are fever, cough, and fast or difficult breathing (Singh and Aneja, 2011). Fever is not very specific, as it occurs in many other common illnesses, may be absent in those with severe disease, malnutrition or in the elderly. In addition, a cough is frequently absent in children less than 2 months old. More severe signs and symptoms in children may include blue-tinged skin, not willing to drink, convulsions, ongoing vomiting, extremes of temperature, or a decreased level of consciousness (Nair and Niederman, 2011).

Etiology

Pneumonia is due to infections caused primarily by bacteria or viruses and less commonly by fungi and parasites. Although there are more than 100 strains of infectious agents identified, only a few are responsible for the majority of the cases. Mixed infections with both viruses and bacteria may occur in up to 45% of infections in children and 15% of infections in adults (Ruuskanen, et al., 2011). The term pneumonia is sometimes more broadly applied to any condition resulting in inflammation of the lungs, however, this inflammation is more accurately referred to as pneumonitis. Conditions and risk factors that predispose to pneumonia include smoking, immunodeficiency, alcoholism, chronic obstructive pulmonary disease, asthma, chronic kidney disease, and liver disease (Nair and Niederman, 2011).

2.1.3.5 Tuberculosis

Tuberculosis (TB) is an infectious disease caused by the bacterium *Mycobacterium tuberculosis* (MTB). Tuberculosis generally affects the lungs, but can also affect other parts of the body. Most infections do not have symptoms, known as latent tuberculosis. About 10% of latent infections progress to active disease which, if left untreated, kills about half of those infected (Gerald, et al., 2010). The classic symptoms of active TB are a chronic cough with blood-containing sputum, fever, night sweats, and weight loss. Tuberculosis is spread through the air when people who have active TB in their lungs cough, spit, speak, or sneeze. People with latent TB do not spread the disease. Active infection occurs more often in people with HIV/AIDS and in those who smoke. Diagnosis of active TB is based on chest X-rays, as well as microscopic examination and culture of body fluids. Prevention of TB involves screening those at high risk, early detection and treatment of cases, and vaccination with the bacillus Calmette-Guérin vaccine (Konstantinos, 2010). Those at high risk include household, workplace, and social contacts of people with active TB. Treatment requires the use of multiple antibiotics over a long period of time.

Signs and symptoms

Tuberculosis may infect any part of the body, but most commonly occurs in the lungs (known as pulmonary tuberculosis). Extrapulmonary TB occurs when tuberculosis develops outside of the lungs, although extrapulmonary TB may coexist with pulmonary TB. General signs and symptoms include fever, chills, night sweats, loss of appetite, weight loss, and fatigue (Gerald, et al., 2010).

Causes

The main cause of TB is *Mycobacterium tuberculosis*, a small, aerobic, nonmotile bacillus. The high lipid content of this pathogen accounts for many of its unique clinical characteristics (Southwick, 2007). It divides every 16 to 20 hours, which is an extremely slow rate compared with other bacteria, which usually divide in less than an hour. MTB can withstand weak disinfectants and survive in a dry state for weeks. In nature, the bacterium can grow only within the cells of a host organism, but M. tuberculosis can be cultured in the laboratory.

Risk factors

A number of factors make people more susceptible to TB infections. The most important risk factor globally is HIV; 13% of all people with TB are infected by the virus (World Health Organization, 2011). This is a particular problem in sub-Saharan Africa, where rates of HIV are high. Tuberculosis is closely linked to both overcrowding and malnutrition, making it one of the principal diseases of poverty (Lawn and Zumla, 2011). Those at high risk include: people who inject illicit drugs, inhabitants and employees of locales where vulnerable people gather (e.g. prisons and homeless shelters), medically underprivileged and resource-poor communities, high-risk ethnic minorities, children in close contact with high-risk category patients, and health-care providers serving these patients. Chronic lung disease is another significant risk factor. Silicosis increases the risk about 30-fold. Those who smoke cigarettes have nearly twice the risk of TB compared to nonsmokers (van Zyl, Yew, Leung, and Zumla, 2010).

2.1.3.6 Lung Cancer

Lung cancer, also known as lung carcinoma, is a malignant lung tumor characterized by uncontrolled cell growth in tissues of the lung. If left untreated, this growth can spread beyond the lung by the process of metastasis into nearby tissue or other parts of the body (Falk and

Williams, 2010). Most cancers that start in the lung, known as primary lung cancers, are carcinomas. The two main types are small-cell lung carcinoma (SCLC) and non-small-cell lung carcinoma (NSCLC). The most common symptoms are coughing (including coughing up blood), weight loss, shortness of breath, and chest pains (Horn, et al., 2015). The vast majority (85%) of cases of lung cancer are due to long-term tobacco smoking. About 10–15% of cases occur in people who have never smoked (Thun, et al., 2008). These cases are often caused by a combination of genetic factors and exposure to radon gas, asbestos, second-hand smoke, or other forms of air pollution. Lung cancer may be seen on chest radiographs and computed tomography (CT) scans. The diagnosis is confirmed by biopsy which is usually performed by bronchoscopy or CT-guidance. Prevention is by avoiding risk factors including smoking and air pollution. Treatment and long-term outcomes depend on the type of cancer, the stage (degree of spread), and the person's overall health. Most cases are not curable. Common treatments include surgery, chemotherapy, and radiotherapy.

Signs and symptoms

Signs and symptoms which may suggest lung cancer include (Kasper, et al., 2015): respiratory symptoms: coughing, coughing up blood, wheezing, or shortness of breath. Systemic symptoms include weight loss, weakness, fever, or clubbing of the fingernails. Symptoms due to the cancer mass pressing on adjacent structures include chest pain, bone pain, superior vena cava obstruction, or difficulty swallowing. If the cancer grows in the airways, it may obstruct airflow, causing breathing difficulties. The obstruction can lead to accumulation of secretions behind the blockage, and predispose to pneumonia.

Causes

Cancer develops following genetic damage to DNA and epigenetic changes. These changes affect the normal functions of the cell, including cell proliferation, programmed cell death (apoptosis) and DNA repair. As more damage accumulates, the risk of cancer increases]

Smoking: Smoking, particularly of cigarettes, is by far the main contributor to lung cancer (Hecht, 2012). Cigarette smoke contains at least 73 known carcinogens, including benzopyrene, NNK, 1,3-butadiene and a radioactive isotope of polonium, polonium-210 (Kumar, et al., 2013). Passive smoking (the inhalation of smoke from another's smoking) is a cause of lung cancer in nonsmokers. A passive smoker can be defined as someone living or working with a smoker. Those who live with someone who smokes have a 20–30% increase in risk while those who work in an environment with secondhand smoke have a 16–19% increase in risk. Investigations of sidestream smoke suggest it is more dangerous than direct smoke (Schick and Glantz, 2005).

Radon gas: Radon is a colorless and odorless gas generated by the breakdown of radioactive radium, which in turn is the decay product of uranium, found in the Earth's crust. The radiation decay products ionize genetic material, causing mutations that sometimes turn cancerous. Radon is the second-most common cause of lung cancer in the USA, causing about 21,000 deaths each year (Choi and Mazzone, 2014). The risk increases 8–16% for every 100 Bq/m^3 increase in the radon concentration.

Asbestos: Asbestos can cause a variety of lung diseases, including lung cancer. Tobacco smoking and asbestos have a synergistic effect on the formation of lung cancer. In smokers who work with asbestos, the risk of lung cancer is increased 45-fold compared to the general population (Tobias and Hochhauser, 2010).

Air pollution: Outdoor air pollution has a small effect on increasing the risk of lung cancer. Fine particulates ($PM_{2.5}$) and sulfate aerosols, which may be released in traffic exhaust fumes, are associated with slightly increased risk. For nitrogen dioxide, an incremental increase of 10 parts per billion increases the risk of lung cancer by 14%. Outdoor air pollution is estimated to account for 1–2% of lung cancers (Chen, et al., 2008). Tentative evidence supports an increased risk of lung cancer from indoor air pollution related to the burning of wood, charcoal, dung or crop residue for cooking and heating (Lim and Seow, 2012). Women who are exposed to indoor coal smoke have about twice the risk and a number of the by-products of burning biomass are known or suspected carcinogens. This risk affects about 2.4 billion people globally, and is believed to account for 1.5% of lung cancer deaths (Sood, 2012).

2.1.3.7 Emphysema

Emphysema is a long-term, progressive disease of the lungs that primarily causes shortness of breath due to over-inflation of the alveoli (air sacs in the lung). In people with emphysema, the lung tissue involved in exchange of gases (oxygen and carbon dioxide) is impaired or destroyed. Emphysema is included in a group of diseases called chronic obstructive pulmonary disease or COPD. Emphysema is called an obstructive lung disease because airflow on exhalation is slowed or stopped because over-inflated alveoli do not exchange gases when a person breaths due to little or no movement of gases out of the alveoli (Schiffman, 2016). Emphysema changes the anatomy of the lung in several important ways. This is due to in part to the destruction of lung tissue around smaller airways. This tissue normally holds these small airways, called bronchioles, open, allowing air to leave the lungs on exhalation. When this tissue is damaged, these airways collapse, making it difficult for the lungs to empty and the air (gases) becomes trapped in the alveoli.

Normal lung tissue looks like a new sponge. Emphysematous lung looks like an old used sponge, with large holes and a dramatic loss of "springy-ness" or elasticity. When the lung is stretched during inflation (inhalation), the nature of the stretched tissue wants to relax to its resting state. In emphysema, this elastic function is impaired, resulting in air trapping in the lungs. Emphysema destroys this spongy tissue of the lung and also severely affects the small blood vessels (capillaries of the lung) and airways that run throughout the lung. Thus, not only is airflow affected but so is blood flow. This has dramatic impact on the ability for the lung not only to empty its air sacs called alveoli but also for blood to flow through the lungs to receive oxygen.

2.2 THEORETICAL FRAMEWORK

2.2.1 Air Quality Dispersion Model

The air quality dispersion model is part of models that are used to determine compliance with National Ambient Air Quality Standards (NAAQS), and other regulatory requirements such as New Source Review (NSR) and Prevention of Significant Deterioration (PSD) regulations. The model was originally published in April 1978 to provide consistency and equity in the use of modeling within the U.S. air quality management system (ENVIRONMENTAL PROTECTION AGENCY 40 CFR Part, n.d.). It is an estimation model that uses allows for estimation of the concentration of pollutants at specified ground-level receptors surrounding an emissions source (US EPA,OAR. 2019). The present study applied the air quality dispertion model to enable it estimate the concentration level of the air quality for the study air pollutants and respiratory flow of the participants at the assessed road construction sites in Imo State Nigeria

2.2.2 The Capability, Opportunity, Motivation - Behaviour (COM-B)

The COM-B model is a behavior change model that identifies three factors the has the capability to effect behavioral changes. The three factors include capability (C), opportunity (O), and motivation (M).

The **c**apability as recognized in the model refers to the ability to participate in an act or action, opportunity refers to some external factors of influence in lifestyle while motivation refers to the perceptive reasoning stages that encourages behavior (Michie, van Stralen and West, 2011).

The application of COM-B model to this study is an warning that respiratory health problems emanating from road construction activities is a sort man made problems that can be tackled through behavioral changes such as through the use of personal protective equipment (PPE) and other regulatory interventions to achieve more effective results in averting the consequential respiratory health issues (Stevely et al., 2018).The present study adopted the COM-B model to expose the underlying respiratory health problems in road construction and how they can be averted by the workers through behavioral adjustments to recommended guides.

2.3 EMPIRICAL STUDIES

The outdoor particulate matter (PM_{10}) concentration and the pulmonary function status of residents in four selected communities in Ibadan were studied using a cross-sectional design between January and March 2008 (Ana, et al., 2014). Lung function status (FEV_1) of 140 randomly selected participants was measured. Daily mean of PM_{10} levels were compared with WHO guideline limits. Data analysis was done using descriptive, $\chi(2)$, ANOVA and Spearman-rank correlation tests at 5% level of significance. For all sites, PM_{10} concentration was generally

higher in the afternoon. There was a significant negative correlation between PM_{10} burden and FEV_1 across the study locations ($r = -0.371$, $p < .05$). It was concluded that most of the locations with higher particulate burden were observed to have declining lung function status. A longitudinal study to establish more robust associations was advocated.

Odeshi, Ana, Sridhar, Olatunji, and Abimbola, (2014) studied the outdoor air particle-bound trace metals in four selected communities in Ibadan, Nigeria. Trace metal concentrations were determined in particulate matter (PM_{10}) in ambient air of four purposively selected residential areas in Ibadan, Nigeria using a volumetric sampler following standard procedures and levels compared with WHO guideline limits. Glass-fibre filter papers exposed to the particulate matter were digested using appropriate acid mixtures, and the digest analyzed for trace metals including Ni, Cr, Mn, Zn, and Pb using ICPMS method and levels compared with WHO limits. Data was analyzed using ANOVA and Pearson correlation test at 5 % level of significance. The highest mean PM_{10} concentrations 502.3 ± 39.9 $\mu g/m^{(3)}$ were recorded in the afternoon period, while the lowest concentration 220.6 ± 69.9 $\mu g/m^{(3)}$ was observed in the morning hours. There was a significant difference between the PM_{10} levels across the various locations ($p < 0.05$), and all the levels were higher than WHO limit of 50 $\mu g/m^{(3)}$. There was a significant correlation between PM_{10} and Ni ($p < 0.05$). Urban communities with increased human activities especially motor traffic recorded both higher levels of PM_{10} and toxic trace metals.

Fifty-nine mother-child pairs from 59 households that used firewood exclusively for cooking in three rural communities in southwest Nigeria underwent blood test for albumin, pre-albumin, retinol-binding protein (RBP), superoxide dismutase (SOD), vitamins C, vitamin E, malondialdehyde (MDA) and C-reactive protein (CRP). Spirometry was performed and indoor levels of $PM_{2.5}$ were determined. The median indoor $PM_{2.5}$ level was 1575.1 $\mu g/m^3$ (IQR 943.6--

2847.0, $p<0.001$), which is substantially higher than the World Health Organization (WHO) standard of 25 $\mu g/m^3$. The mean levels of pre-albumin (0.21±0.14 g/dL) and RBP (0.03±0.03 g/dL) in women were significantly lower than their respective normal ranges (1-3 g/dL and 0.2-0.6 g/dL, respectively, $p<0.05$). Similarly, the mean levels of pre-albumin (0.19±0.13 g/dL) and RBP (0.01±0.01 g/dL) in children were significantly lower than the respective normal ranges (1-3 g/dL and 0.2-0.6 g/dL, respectively, $p<0.05$). Mean serum concentrations of MDA in children (5.44±1.88 µmol/L) was positively correlated to serum concentrations of CRP ($r=0.3$, $p=0.04$) and negatively correlated to lung function (FEV_1/FVC) in both mothers and children (both $r=-0.3$, $p<0.05$). Also, regression analysis indicates that CRP and SOD are associated with lung function impairment in mothers (-2.55±1.08, $p<0.05$) and children (-5.96±3.05, $p=0.05$) respectively. It was concluded that exposure to HAP from biomass fuel is associated with pulmonary dysfunction, reduced antioxidant defense and inflammation of the airways (Oluwole, Arinola, Ana, Wiskel, and Huo, 2013).

A study was carried out to assess the respiratory health effect of city ambient air pollutants on transit and non-transit workers and compare such effects by transportation mode, occupational exposure and socio-demographic characteristics of participants. The adjusted odds ratio (OR) for respiratory function impairment (force vital capacity (FVC) and/or FEV(1)<80% predicted or FEV(1)/FVC<70% predicted) using Global Initiative for Chronic Obstructive Lung Diseases (GOLD) and National Institute for Health and Clinical Excellence (NICE) criteria were calculated. In order to investigate specific occupation-dependent respiratory function impairment, a comparison was made between the ORs for respiratory impairment in the three occupations. Adjustments were made for some demographic variables such as age, BMI, area of residence, etc. Results showed that exposure to ambient air pollution by occupation and

transportation mode was independently associated with respiratory functions impairment and incident respiratory symptoms among participants. Motorcyclists had the highest effect, with adjusted OR 3.10, 95% CI 0.402 to 16.207 for FVC<80% predicted and OR 1.71, 95% CI 0.61 to 4.76 for FEV(1)/FVC<70% predicted using GOLD and NICE criteria. In addition, uneducated, currently smoking transit workers who had worked for more than 1 year, with three trips per day and more than 1 h transit time per trip were significantly associated with higher odds for respiratory function impairment at $p<0.001$, respectively (Ekpenyong, et al., 2012).

Ana, et al. (2009a) carried out a study to assess the sources of air pollution in and around schools as a surrogate for air quality and report adverse health effects among students at selected secondary schools in urban Ibadan, Nigeria. The study was a descriptive cross-sectional survey involving eight secondary schools. The pre-tested, self-administered questionnaires, observational checklists to assess certain environmental health indicators, and interviews were used to collect data. A total of 400 students from senior secondary classes, 50 from each school, were selected through stratified random sampling. The school's location, especially if close to high traffic roadways, contributed to reported perceived poor air quality of school environments. The majority of students believed air pollution sources in the school environment were mainly refuse burning and car emissions from nearby roadways. Cough and asthma were the most frequently reported adverse health outcomes. They concluded that proximity of study schools to certain sources and activities such as refuse burning and major roadways seemed to present substantial risk factors for reported respiratory morbidity among secondary students in urban Ibadan, Nigeria.

In a study to assess the prevalence of various health outcomes associated with exposure to environmental risk factors including industrial pollution in selected communities of Nigeria's oil-

rich Niger delta area (NDA), a total of 14 air samples, 16 grab soil samples and 18 surface water samples were collected and analyzed for physicochemical parameters including heavy metals and polycyclic aromatic hydrocarbons (PAHs) using standard methods. A 77-item questionnaire was administered on randomly selected 349 subjects. A five-year record was collected from health facilities located in the two communities. The laboratory results indicated that the median PAH level at Eleme as compared to Ahoada East was higher than the guideline limit 50 ng/l for surface waters. The mean TSP level at Eleme was higher than the level at Ahoada East and the guideline limit 100 microg/m^3. The survey results showed that at Eleme air pollution in the community was significantly associated with painful body outgrowths ($p = 0.027$) and the effect the air contaminants was significantly associated with respiratory health problem ($p = 0.044$). At Ahoada East commonly consumed aquatic food was highly significantly associated with painful body outgrowth ($p < 0.0001$) while use of domestic cooking fuel types was also highly significantly associated with child deformities ($p < 0.0001$). Hospital records showed high proportions of respiratory disorder among males (3.85%) and females (4.39%) at Eleme as compared to the proportion of respiratory disorder among males (3.68%) and females (4.18%) at Ahoada East. The study showed that industrial communities such as Eleme, which are exposed to higher levels of air pollution, are more predisposed to respiratory morbidities, skin disorders and other related health risks (Ana, et al., 2009b).

A cross-sectional study aimed at assessing the association between particulate concentrations, respiratory symptoms and lung function was carried out at 3 communities at Ile-Ife, Nigeria. Assessment of the indoor PM_{10} levels was done by filtration using the Gent stacked filter unit sampler for collection of atmospheric aerosol in two size fractions ($PM_{2.5}$ and PM_{10}). The Medical Research Council (MRC) questionnaire was administered followed by spirometry test.

Results showed that the mean PM_{10} concentration in participants using liquefied petroleum gas (LPG), kerosene and firewood was 80.8 ± 9.52 μg/m(3), 236.9 ± 26.5 μg/m(3) and 269 ±93.7 μg/m(3), respectively. A similar trend was found in the forced vital capacity (FVCs). Users of firewood had significantly lower FEV1 and FVC compared with LPG users ($P < 0.05$). The participants using firewood had the highest prevalence of pulmonary and non-pulmonary symptoms (57.1%), whereas subjects using LPG had the lowest (23.8%). It was concluded that there are high levels of particulate matter pollutions with respiratory effects in residential indoor environments in Ile-Ife, Nigeria (Ibhafidon, 2014).

In a study of the relationship of biomass fuel use with asthma symptoms and lung function in Nigerian children (Thacher, et al., 2013), data was collected from 299 village children regarding the cooking fuels used and duration of daily smoke exposure in the cooking area. Asthma symptoms were assessed with a modified International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire, and lung function was assessed with spirometry. The prevalence of a lifetime history of wheeze was 9.4% (95% CI: 6.3%-13.2%). Fourteen children (4.7%) had airway obstruction (FEV1/FEV6 <85%). Female subjects had lower FEV1 and FEV6 (110 % and 120% percent predicted, respectively) than males (121% and 130%, respectively, $P <0.001$ for both differences). Advancing age was associated with a relative decline in the predicted value of FEV1 of 7.8 % per year ($r = -0.61$; $P < 0.001$). Children in families that used firewood daily did not have a significantly increased likelihood of asthma-related symptoms (OR = 2.36, 95% CI: 0.66-8.44). Similarly, airway obstruction did not differ significantly between children in households that did and did not use firewood daily (mean FEV1/FEV6 of 0.95 and 0.97, respectively; $P = 0.41$).

Mustapha, et al. (2011) investigated associations between respiratory health and outdoor and indoor air pollution in school children 7-14 years of age in low socioeconomic status areas in the Niger Delta. Exposure to home outdoor and indoor air pollution was assessed by self-report questionnaire. School air pollution exposures were assessed using traffic counts, distance of schools to major streets, and particulate matter and carbon monoxide measurements, combined using principal components analysis. Hierarchical logistic regression was used to examine associations with reported respiratory health, adjusting for potential confounders. Traffic disturbance at home (i.e., traffic noise and/or fumes evident inside the home vs. none) was associated with wheeze [odds ratio (OR) = 2.16; 95% confidence interval (CI), 1.28-3.64], night cough (OR = 1.37; 95% CI, 1.03-1.82), phlegm (OR = 1.49; 95% CI, 1.09-2.04), and nose symptoms (OR = 1.40; 95% CI, 1.03-1.90), whereas school exposure to a component variable indicating exposure to fine particles was associated with increased phlegm (OR = 1.38; 95% CI, 1.09-1.75). No significant positive associations were found between cooking with wood/coal (OR = 2.99; 95% CI, 0.88-10.18) or kerosene (OR = 2.83; 95% CI, 0.85-9.44) and phlegm compared with cooking with gas. They concluded that traffic pollution is associated with respiratory symptoms in the school children.

The prevalence of chronic bronchitis (CB) and its association with socio-demographic factors and tobacco smoking was carried out in some rural communities in Ekiti state, Nigeria. The subjects were selected by multistage cluster sampling method. The European Coal and Steel Community (ECSC) questionnaire was adapted and administered by trained health workers to obtain socio-demographic information, respiratory symptoms, history of tobacco smoking, occupational exposure to dust and housing. Of the 391 subjects that participated in the study, none of the current smokers had CB, while 36.4% of those with CB were former smokers. The

multivariate logistic regression analysis showed that aged 65-74 (OR= 9.66, 95% C.I 3.43-27.20), aged e"75 years (OR= 3.88, 95% C.I 1.08-13.98) and tobacco smoking (OR= 6.37 95% C.I 2.12-19.14) had the strongest association with CB. Poor housing (OR=1.80; 95% C.I 0.56-6.51), occupational exposure to organic and inorganic dust (OR= 1.74; 95% C.I 0.67-4.60) and Low socioeconomic status (OR=1.72; 95% C.I 0.29-10.88) were also independently associated with CB. Sex and level of education were not associated with CB. The prevalence of chronic bronchitis in this study was 5.6% and is comparable to previous studies worldwide (Desalu, 2011).

A study was done to determine the magnitude of the problem among woodworkers in south-eastern Nigeria exposed to high level of wood dust. Five hundred and ninety-one woodworkers were selected using a stratified random sampling. The prevalence of woodwork-related rhinitis and asthma were then observed in the study population. Also the peak expiratory flow rate (PEFR) of each woodworker was obtained. The prevalence of occupational rhinitis was 78%, while that of asthma was 6.5%. As period of woodwork increased the prevalence of rhinitis and asthma increased (rhinitis: chi2 trend = 53.015, df = 1, P = 0.000). For asthma, chi2 trend = 19.721, df = 1, P = 0.000). Also the PEFR significantly became low with increasing years of exposure to woodwork (chi2 trend = 75.965, df = 1, P = 0.000). In conclusion the prevalence of rhinitis and asthma in woodworkers was high and significantly increased with years of working as a woodworker (Aguwa, et al., 2007).

A study was done to assess the influence of smoking on respiratory symptoms and respiratory function in sawmill workers in Benin City. 150 sawmill workers who were all males and aged between 18 and 50 years, and had been in continuous employment in sawmill factories for a minimum of one year were studied. They were selected by a two-stage random sampling process

from sawmills in Benin City. These were compared to 150 age and sex matched controls in order to determine the effect of sawdust exposure on the respiratory system. A questionnaire was used to elicit morbidity patterns and anthropometric measurements were also made. Respiratory rates, Peak Expiratory Flow Rates and Blood Pressures were measured in both groups. Although blood pressure was similar in both groups, respiratory rates were higher and Peak Flow Rates were lower in the sawmill workers compared to the controls (20.83 +/- 2.02 cycles/minute and 516.72 +/- 38.48 L/minute for the sawmill workers; 15.45 +/- 1.23 cycles/minute and 575.37 +/- 27.34 L/minute for the controls, respectively). Less than 5% of the sawmill workers wore protective devices/clothing, and health and safety standards were neither practiced nor enforced. The findings suggest that respiratory symptoms especially sputum production and chest pain are common in sawmill workers (Ugheoke, et al., 2006).

The effect of chronic exposure to dust from local woods such as ebony, achi, and iroko on lung function of timber market workers in Calabar, Nigeria, was studied by Okwari, et al. (2005). Forced vital capacity (FVC), Forced Expiratory Volume in one second, (FEV1), Forced Expiratory Volume as a percentage of forced vital capacity (FEV1 %), and Peak Expiratory Flow Rate (PEFR) were measured in 221 workers (aged 20-25 years) exposed to wood dust to assess their lung function and compared with 200 age- and sex- matched control subjects who were not exposed to any known air pollutant. The concentration of respirable dust was significantly higher in the test ($P<0.001$) than in control site. The mean values of FVC, FEV1, FEV1% and PEFR of the timber workers were significantly lower ($P<0.01$) than in control subjects. Respiratory symptoms such as cough, chest pain and nasal irritation had higher prevalence in the test group than in the control group. Non-respiratory symptoms (skin and eye irritation) were prevalent in

the test group but not found in the control group. Workers exposed to wood dust had restrictive pattern of ventilatory function impairment.

Maduka, et al. (2009) studied the effect of occupational exposure to local powdered tobacco (snuff) on pulmonary function. Snuff industry workers in Onitsha and Enugu markets in Nigeria were studied and compared with age, weight, and height-matched control not exposed to any known air pollutant. The pulmonary indices studied include forced vital capacity [FVC], forced expiratory volume in one second [FEV1] and ratio of FEV1/FVC as percentage using a vitalograph spirometer and Peak Expiratory Flow Rate [PEFR], using a mini Wright Peak Expiratory Flow Meter. The respiratory and non-respiratory symptoms frequently associated with these workers were also analyzed and dust sampling in both test and control environments was also done. The results obtained showed statistically significant impairment of lung function of workers chronically exposed to snuff. FVC, FEV1 and PEFR in the exposed [test] subjects were significantly decreased in comparison with the control subjects [$P<0.05$]. However, the mean value of FEV1/FVC [%] of the test subjects was 86.8% which was within the normal range and was not significantly different from control. This signified that the test subjects had restrictive pattern of lung function defect. All respiratory symptoms, such as cough, chest tightness had a higher prevalence in test subjects than their control group. The lung function indices of snuff-producing workers proportionately decreased with their length of exposure in the industry. The respirable dust level in the vicinity [indoor] of the snuff-workers [1.11+/-0.35 mg/m3] was significantly [$P<0.001$] higher than in the control environment [0.37+/-0.086 mg/m3]. The dust sampling result showed that chronic exposure to Nigerian snuff [powered tobacco] dust impairs lung function and the effect is progressive with time.

Ige and Awoyemi (2002) studied five hundred bakery workers were to assess occupational induced lung impairment as a result of exposure to grain and flour dust. Occupational related symptoms were recorded using structured questionnaire. Age and sex matched controls consisting of 500 University College Hospital (UCH) Ibadan workers and students were used. They were apparently healthy and work and live at places free of fumes and smoke. Peak expiratory flow rate (PEFR) was measured in all subjects. However, full spirometry work up was done on 100 bakery workers and 100 control subjects that had been selected using simple random sampling technique. The most frequent pulmonary symptoms among the bakery workers were sneezing and running nose (53.30%) and periodic breathlessness/chest tightness (23.16%) while the symptom of cough/phlegm present in (21.53%) of the subjects. The mean PEFR of the bakery workers (463.20 + 51.39 L/ min) was significantly lower ($P < 0.0001$) than that of the control subjects (538.0 + 47.23 L/min). Similarly, the mean values of FEV1, FVC and FEV1% were also significantly lower than the control subjects. The findings indicate that respiratory symptoms are common during the working hours among the bakery workers and 23.16% of the subjects studied suffered some degree of airway obstruction.

A total of five hundred saw millers in Ibadan, Nigeria were studied to assess occupation-induced lung impairment as a result of exposure to saw dust (Ige and Awoyemi, 2002). Occupation-related symptoms were recorded using structured questionnaire. Age and sex matched controls consisting of 500 University College Hospital (UCH) Ibadan workers or students were used. They were apparently healthy and work and live at places free of fumes and smoke. Peak expiratory flow rate (PEFR) was measured in all subjects. However, full spirometry work-up was done on 120 saw millers and 120 control subjects that had been selected using simple random sampling technique. The most frequent pulmonary symptoms among the sawmill workers were

running nose and sneezing (57.40%) and productive cough (34.30%), while the symptoms of dyspnoea and wheezing were each present in (4.10%) of the subjects. The mean PEFR of the sawmillers (463.8 +/- 63.4 L/min) was significantly lower ($P < 0.0001$) than that of the control subjects (537.7 +/- 71.5 L/min). Similarly, the mean values of FEV1, FVC and FEV1% (FEV1%) were also significantly lower in the FVC sawmillers than the control subjects. The findings indicate that respiratory symptoms are common during the working hours among the saw millers and 4.1% of the subjects studied suffered some degree of airway obstruction.

CHAPTER THREE

MATERIALS AND METHODS

3.1 AREA OF STUDY

Imo State

Imo state was created in 3rd February 1976 with Owerri as its capital and largest city. The State lies within latitudes 4°45'N and 7°15'N, and longitude 6°50'E and 7°25'E with an area of around 5,100 sq km. The 2006 census puts its population at 3,934,899 (National Bureau of Statistics of Nigeria, 2007). Located in the south-eastern region of Nigeria, it occupies the area between the lower River Niger and the upper and middle Imo River. Imo State is bordered by Abia State on the East, River Niger and Delta State to the West, Anambra State on the North and Rivers State to the South. The 3 senatorial zones of Imo State include Imo North, Imo East and Imo West. The state is rich in natural resources including crude oil, natural gas, lead and zinc. Economically exploitable flora including iroko, mahogany, obeche, bamboo, rubber tree and oil palm predominate. Other natural resources found in the State are white clay, fine sand and limestone.

Many trade and investment opportunities abound in the peaceful state including oil and gas exploration, chemical plants, brewery plants, hydroelectricity and gas-fired power plants, grain milling, starch production, cashew product industry, fruit and vegetable juice concentrate production, integrated multi-oil seeds processing plant, ceramic industry, inland waterway transport, integrated palm produce industry etc. The economy of the state depends primarily on agriculture and commerce. The chief occupation of the people is farming. Their cash crops include oil palm, raffia palm, rice, groundnut, melon, cotton, cocoa, rubber, maize, etc. Food

crops such as yam, cassava, cocoyam and maize are also produced in large quantities. Imo State consists of twenty-seven Local Government Areas. They are:

(i) Aboh Mbaise

(ii) Ahiazu Mbaise

(iii) Ehime Mbano

(iv) Ezinihitte Mbaise

(v) Ideato North

(vi) Ideato South

(vii) Ihitte/Uboma

(viii) Ikeduru

(ix) Isiala Mbano

(x) Isu

(xi) Mbaitoli

(xii) Ngor Okpala

(xiii) Njaba

(xiv) Nkwerre

(xv) Nwangele

(xvi) Obowo

(xvii) Oguta

(xviii) Ohaji/Egbema

(xix) Okigwe

(xx) Onuimo

(xxi) Orlu

(xxii) Orsu

(xxiii) Oru East

(xxiv) Oru West

(xxv) Owerri Municipal

(xxvi) Owerri North

(xxvii) Owerri West

Each LGA is made up of autonomous communities which are headed by a traditional ruler. In addition to English being official language, Imo state is a predominantly Igbo speaking state, with Christianity being the predominant religion.

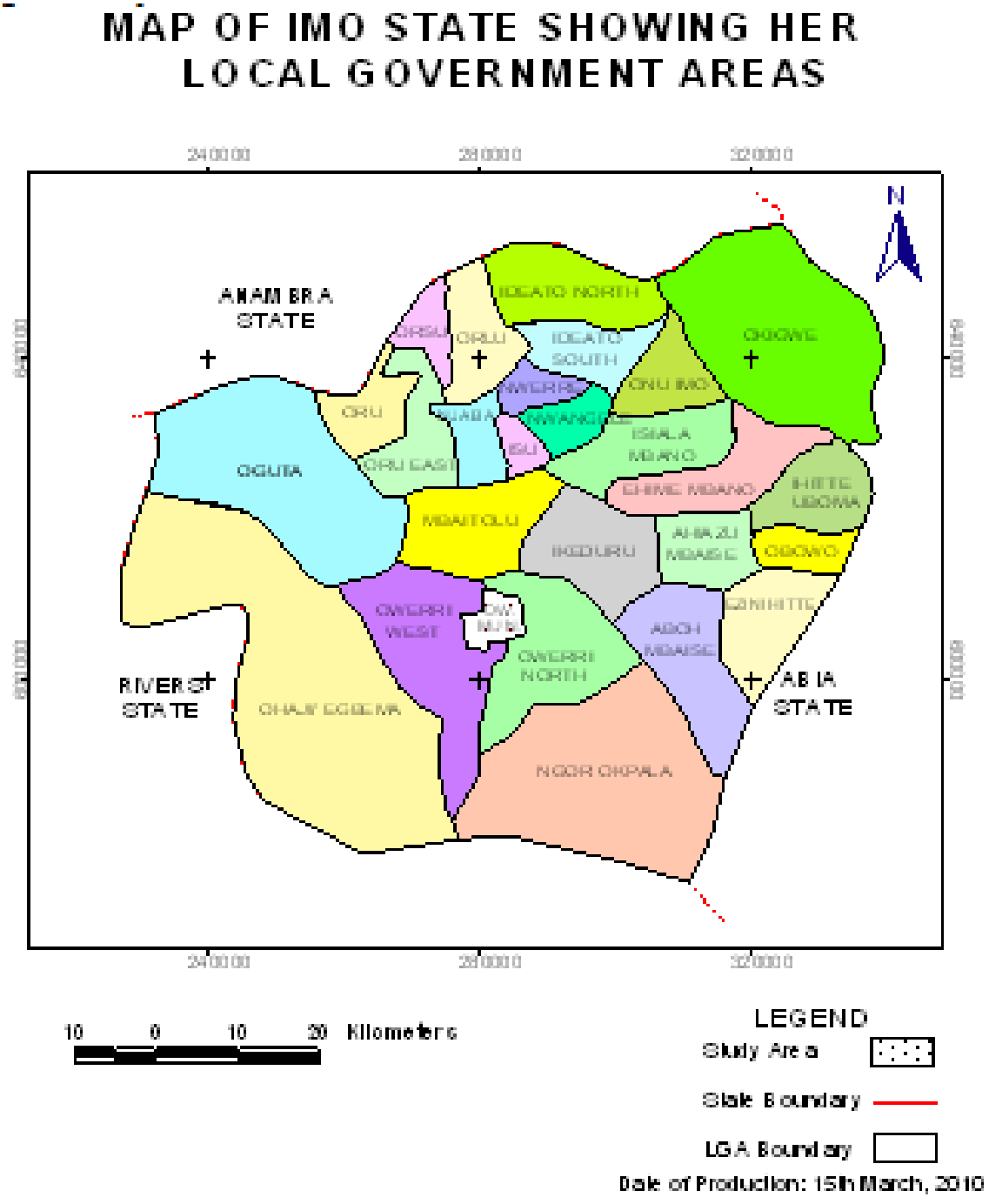

Figure 3.1: Map of Imo state

3.2 STUDY DESIGN

This research was a cross-sectional study of Imo state, Nigeria. It was both an observational study and a clinical study involving the measurement of outdoor gaseous pollutants, interview of participants with a questionnaire and the health examination of participants.

3.3 POPULATION OF STUDY

All workers at road construction sites in Imo State, Nigeria made up the population of study.

3.3.1 Inclusion Criteria

All workers from the ages of 18 years and above who work at road construction sites in Imo State, Nigeria was part of the study.

3.3.2 Exclusion Criteria

i) People who did not give an informed consent to be part of the study

ii) People who have a debilitating illness

iii) People who have a mental illness

3.4 SAMPLE SIZE DETERMINATION

Fisher's formula (Ogbeibu, 2014) for cross-sectional studies was used to determine the minimum sample size of participants to be interviewed.

$$N = \frac{Z^2 Pq}{d^2}$$

Where N = Desired sample size for a population greater than 10,000

Z = 1.96 (The standard normal deviate at 95% confidence interval)

p = proportion of the total population required for the study

q = 1 – p

d = sampling error to be tolerated which is 0.05

p = 0.20

$$N = \frac{1.96^2 \ X \ 0.20 \ X \ 0.80}{0.05^2} = \quad 245.86 = 246$$

The minimum sample size calculated was 246. However, a total of 353 subjects were used in this study.

3.5 SAMPLING TECHNIQUE

The multistage sample technique was adopted starting with large cluster samples at the first stage which comprised the three senatorial zones in Imo State. In the second stage, strategic construction sites were randomly selected from local government areas in the three zones in Imo State. The simple random sampling technique was adopted to obtain the total sample size.

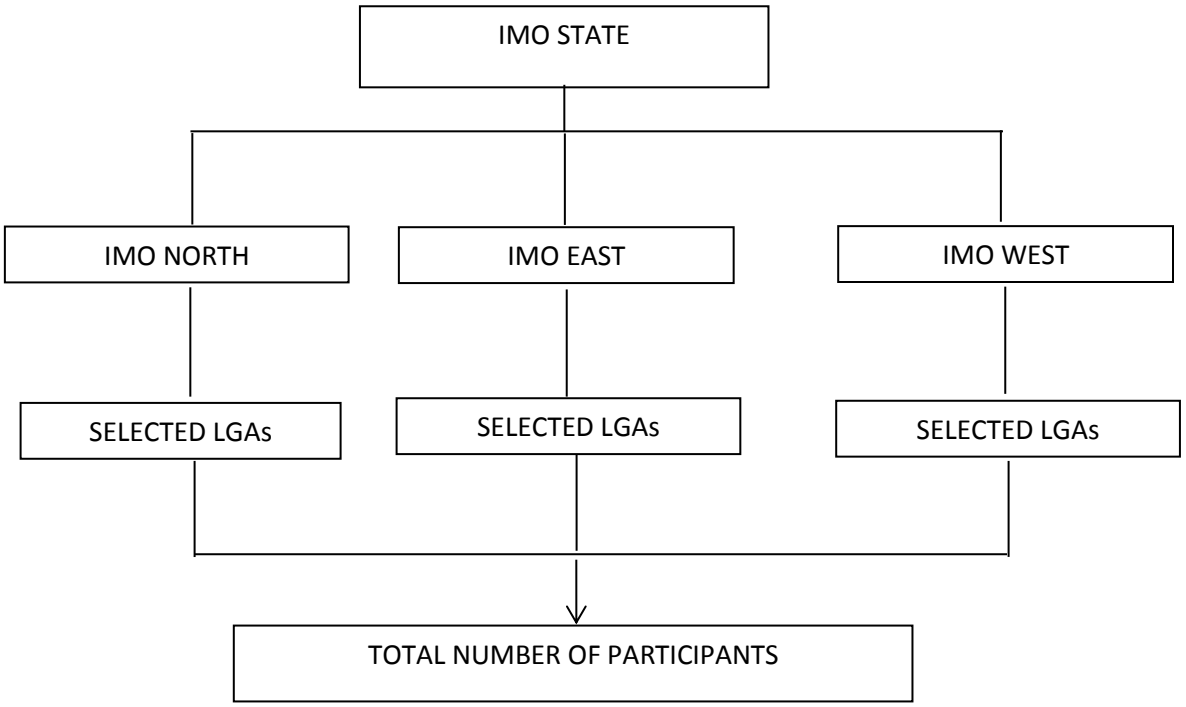

Figure 3.2: Flow chart for multistage sampling

3.6 INSTRUMENTS FOR DATA COLLECTION

(i) A well-structured questionnaire

(ii) Sphygmomanometer

(iii) Spirometer

(iv) Carbon monoxide meter

(v) Sulfur dioxide meter

(vi) Particulate matter meter

3.7 PROCEDURE FOR DATA COLLECTION

At the construction sites, the various gas meters were used to measure gas levels at strategic points. The construction workers were assembled at a hall and measurements of their blood pressure, pulse rate and forced vital capacity readings were taken by well-qualified health personnel.

Air Quality Measurements for concentrations of air pollutants (SO_2, CO_2, NO_2, $PM_{2.5}$, and PM_{10})

Sulphur Dioxide (SO_2). To measure SO2, an SO_2 monitor was placed at the road construction sites where the measurement was taken, while taken into account, some of the influencing factors such as wind direction, sources of emissions, The samples were taken at different road construction locations and the concentration of SO_2 was recorded at regular intervals. The monitor was calibrated before and after each sampling session to maintain accuracy. This reading was taken six times, twice daily (morning and evening) for three days, and the mean of the readings of was estimated and noted as concentration levels for the gas in the different locations.

Carbon Dioxide (CO), A non-dispersive Infrared (NDIR) CO sensor was used to measure the CO concentrations in the air at the road construction sites. While taking into account, the other influencing factors such as wind speed, wind direction, time of the day and industrial activities in the environment the analyzer was calibrated before and after each reading, The reading was taken six times (twice daily for three days) and the mean reading was estimated and recorded as the CO concentration level for that study location.

Suspended Particulate Matter

While taken into account, the location, time, and meteorological conditions, high-volume PM sampler was used to measure the $PM_{/}$ The reading was taken six times and the mean reading was estimated and recorded for use. Also, the instrument was calibrated before and after sampling to ensure accurate measurements. For the $PM_{2.5}$, a size-selective inlet was used to ensure that only particles with a diameter of 2.5 micrometers or smaller are collected. While for PM_{10}, an inlet that captures particles with a diameter of 10 micrometers or smaller was used on the instrument.

Measurement for Blood Pressure

The participants were examined of their blood pressure level using manual sphygmomanometer. The systolic blood pressure (SBP) measurement was recorded in mmHg. The measure or check for blood pressure was taken 3 times at 2 minutes interval gap each and the average of the last two measurements was computed and used in data analysis.

Hypertension was established based on systolic blood pressure (SBP). of 140mmHg and above and diastolic blood pressure of 90mmHg and above and above (Chobanian, 2003)

Forced Expiratory Flow (FEF) measurement:

The Forced Expiratory Flow (FEF) measurement was taken to measure the respiratory conditions of the study participants. To measure the PEF, a handheld spirometer was used. The meter was set to zero. The subjects were asked to stand up straight and take a deep breath, and also seal the lips tightly around the mouthpiece. A clip was placed over their nose, and they were given a tube to breathe into The participants were then requested to you are asked to inhale as deeply as

possible and then exhale as forcefully as they can. The value was noted. The test was repeated three times, and the average value was recorded.

3.8 DATA ANALYSIS METHOD

Tables were used for presentation of data. Data was uploaded into the Statistical Package for Social Sciences (SPSS) software (version 23). Descriptive technique was used to compute the mean of the measured variables which frequency distribution table was constructed for class variables. The one-way ANOVA was used to test the hypotheses at 95% confidence interval and 0.05 level of significance.

3.9 ETHICAL CONSENT

An ethical approval was obtained from the ethical committee of the School of Health Technology, Federal University of Technology, Owerri, Imo State, Nigeria. An informed consent was gotten from all subjects who participated in the study.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1.1 Age Demographic Profile of Subjects

A total of 353 subjects from the 3 senatorial zones of Imo State participated in the study. All the subjects were males. Data collected is presented below in tables. For the Tables, "n" represents the number while "%" represents percentage value.

Table 4.1: Age distribution of all study Participants

Age group	Imo West n(%)	Imo East n(%)	Imo North n(%)	Total n(%)
11 – 20	2(0.57%	3(0.85)	4(1.13)	9(2.55)
21 – 30	16(4.53)	19(5.38)	19(5.38)	54(15.30)
31 – 40	43(12.18)	38(10.76)	37(10.48)	118(33.43)
41 – 50	35(9.92)	31(8.78)	32(9.07)	98(27.76)
Above 50	25(7.08)	31(8.78)	18(5.10)	74(20.96)
Total	121(34.28)	122(34.56)	110(31.16)	353(100.00)

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From the table, there were 121 (34.28%) subjects in Imo West; 2 (0.57%) subjects were aged between 11 – 20 years; for 21 - 30, 16 (4.53%); 31 – 40, 43 (12.18%); 41 – 50, 35 (9.92%); above 50, 25 (7.08%). There were 122 (34.56%) subjects in Imo East; 3 (0.85%) subjects were aged between 11 – 20 years; for 21 - 30, 19 (5.38%); 31 – 40, 38 (10.76%); 41 – 50, 31 (8.78%); above 50, 31 (8.78%). There were 110 (34.56%) subjects in Imo North; 4 (0.85%) subjects were aged between 11 – 20 years; for 21 - 30, 19 (5.38%); 31 – 40, 38 (10.76%); 41 – 50, 31 (8.78%); above 50, 31 (8.78%). In total, 9 (2.55%) subjects were aged between 11 – 20 years; for 21 - 30, 54 (15.30%); 31 – 40, 118 (33.43%); 41 – 50, 98 (27.76%); above 50, 74 (20.96%).

4.1.2 Awareness and Knowledge on Health Effects of Air Pollution

From table 4.2, 274(70.21%) of the subjects responded “strongly agree” to road construction being the main source of dust in the village; 303(93.01%) to dust causing respiratory health problems; 322(87.81%) to road construction materials containing harmful chemicals; 311(87.85%) to workers wearing their PPEs; 278(64.06%) to rain and cold weather reducing dust; 229(65.31%) to sunny weather promoting distance of air movement.

Table 4.2: Response of all subjects in Imo state on Air Pollution Awareness

Information on Air Pollution	SA (%)	A (%)	D (%)	SD (%)
General (All the three zones)				
Road construction is a major source of dust	274(70.21)	27(7.03)	38(5.33)	0(0.00)
Dust can cause respiratory health problems	303(93.01)	15(3.90)	0(0.00)	0(0.00)
Road construction materials contain harmful chemicals	322(87.81)	38(9.64)	4(0.53)	0(0.00)
Workers should always wear their PPE	311(87.85)	35(9.89)	0(0.00)	0(0.00)
Rain and cold weather reduce dust	278(64.06)	76(33.45)	0(0.00)	0(0.00)
Sunny weather promotes distant air movement	229(65.31)	40(11.30)	0(0.00)	0(0.00)
Imo West				
Road construction is a major source of dust	97(82.45)	7(8.50)	4(1.50)	0(0.00)
Dust can cause respiratory health problems	82(73.30)	5(5.00)	0(0.00)	0(0.00)
Road construction materials contain harmful chemicals	95(85.57)	8(7.30)	0(0.00)	0(0.00)
Workers should always wear their PPE	106(91.24)	10(9.17)	0(0.00)	0(0.00)
Rain and cold weather reduce dust	85(77.55)	10(8.86)	0(0.00)	0(0.00)
Sunny weather promotes distant air movement	94(86.90)	15(10.56)	0(0.00)	0(0.00)
Imo East				
Road construction is a major source of dust	90(80.45)	7(9.50)	4(1.50)	0(0.00)
Dust can cause respiratory health problems	93(88.30)	9(6.00)	0(0.00)	0(0.00)
Road construction materials contain harmful chemicals	99(85.44)	7(6.35)	0(0.00)	0(0.00)
Workers should always wear their PPE	91(84.24)	18(13.18)	0(0.00)	0(0.00)
Rain and cold weather reduce dust	90(82.50)	15(12.86)	0(0.00)	0(0.00)
Sunny weather promotes distant air movement	98(89.90)	13(10.02)	0(0.00)	0(0.00)
Imo North				
Road construction is a major source of dust	87(78.45)	10(9.11)	1(1.90)	0(0.00)
Dust can cause respiratory health problems	96(90.28)	12(9.60)	0(0.00)	0(0.00)
Road construction materials contain harmful chemicals	102(95.44)	4(6.35)	0(0.00)	0(0.00)
Workers should always wear their PPE	91(85.29)	16(12.10)	0(0.00)	0(0.00)
Rain and cold weather reduce dust	100(92.92)	11(8.30)	0(0.00)	0(0.00)
Sunny weather promotes distant air movement	88(79.43)	11(9.02)	0(0.00)	0(0.00)

SA- Strongly Agree; A- Agree; D- Disagree; SD- Strongly Disagree; PPE- Personal Protective Equipment

In Imo west, , 97(82.45%) of the subjects responded “strongly agree” to road construction being the main source of dust in the village; 82(73.30%) to dust causing respiratory health problems; 95(85.57%) to road construction materials containing harmful chemicals; 106(91.24%) to workers wearing their PPEs; 85(77.55%) to rain and cold weather reducing dust; 94(86.90%) to sunny weather promoting distance of air movement.

In Imo East, 90(80.45%) of the subjects responded “strongly agree” to road construction being the main source of dust in the village; 93(88.30%) to dust causing respiratory health problems; 99(85.44%) to road construction materials containing harmful chemicals; 91(84.24%) to workers wearing their PPEs; 90(82.50%) to rain and cold weather reducing dust; 98(89.90%) to sunny weather promoting distance of air movement.

In Imo North, 87(78.45%) of the subjects responded “strongly agree” to road construction being the main source of dust in the village; 96(90.28%) to dust causing respiratory health problems; 102(95.44%) to road construction materials containing harmful chemicals; 91(85.29%) to workers wearing their PPEs; 100(92.92%) to rain and cold weather reducing dust; 88(79.43%) to sunny weather promoting distance of air movement.

4.1.3 Particulate Matter (SPM) levels at construction sites in Imo State

Table 4.3 showed that in Imo West, the SPM level in $\mu g/m^3$ was 1930 at GPS location 5.4882^oN and 7.0175^oE; 1906 at 5.4859^oN and 7.0171^oE; 1934 at 5.4840^oN and 7.0180^oE; 1940 at 5.4833^oN and 7.0169^oE; 1977 at 5.4851^oN and 7.0173^oE; 1956 at 5.4889^oN and 7.0182^oE. The mean SPM levels (1940.5 ± 24.15) was significantly higher than normal levels at 5% level of significance ($P<0.0001$).

Table 4.3: Distribution of Suspended particulate Matter (SPM) levels at construction sites in each of the three senatorial zones of Imo State

GPS Location North	GPS Location East	SPM (µg/m^3)
IMO WEST		
5.4882^o	7.0175^o	1930
5.4859^o	7.0171^o	1906
5.4840^o	7.0180^o	1934
5.4833^o	7.0169^o	1940
5.4851^o	7.0173^o	1977
5.4889^o	7.0182^o	1956
Mean ± Standard deviations		1940.5 ± 24.15
P-value		<0.0001
5.7822^o	7.0390^o	1942
5.7835^o	7.0388^o	1956
5.7830^o	7.0398^o	1923
5.7846^o	7.0383^o	1959
5.7892^o	7.0381^o	1960
5.7851^o	7.0398^o	1941
Mean ± Standard deviations		1960.17 ± 30.79
P-value	0.01	
IMO NORTH		
5.8191^o	7.3411^o	1945
5.8170^o	7.3406^o	1933
5.8186^o	7.3424^o	1909
5.8198^o	7.3439^o	1962
5.8182^o	7.3401^o	1981
5.8169^o	7.3453^o	1963
Mean ± Standard deviations		1956.68 ± 30.25
P-value	<0.001	

In Imo East, the SPM level in μg/m^3 was 1942 at GPS location 5.7822^{o}N and 7.0390^{o}E; 1956 at 5.7835^{o}N and 7.0388^{o}E; 1923 at 5.7830^{o}N and 7.0398^{o}E; 1959 at 5.7846^{o}N and 7.0383^{o}E; 1960 at 5.7892^{o}N and 7.0381^{o}E; 1941 at 5.7851^{o}N and 7.0398^{o}E. The SPM levels was significantly higher ($P<0.05$) than normal levels.

In Imo North the SPM level in μg/m^3 was 1945 at GPS location 5.8191^{o}N and 7.3411^{o}E; 1933 at 5.8191^{o}N and 7.3411^{o}E; 1909 at 5.8191^{o}N and 7.3411^{o}E; 1962 at 5.8191^{o}N and 7.3411^{o}E; 1981 at 5.8191^{o}N and 7.3411^{o}E; 1963 at 5.8191^{o}N and 7.3411^{o}E. The SPM levels was significantly higher ($P<0.05$) than normal levels.

4.1.4 Carbon Monoxide levels at construction sites in Imo State

Table 4.4 showed that the CO level in ppm was 183 at GPS location 5.4882^{o}N and 7.0175^{o}E; 181 at 5.4859^{o}N and 7.0171^{o}E; 183 at 5.4840^{o}N and 7.0180^{o}E; 182 at 5.4833^{o}N and 7.0169^{o}E; 187 at 5.4851^{o}N and 7.0173^{o}E; 185 at 5.4889^{o}N and 7.0182^{o}E. The mean CO levels (183.5 ± 2.17) was found to be significantly higher than normal levels at 5% level of significance ($P<0.0001$).

In Imo East, the CO level in ppm was 185 at GPS location 5.7822^{o}N and 7.0390^{o}E; 186 at 5.7835^{o}N and 7.0388^{o}E; 184 at 5.7830^{o}N and 7.0398^{o}E; 180 at 5.7846^{o}N and 7.0383^{o}E; 185 at 5.7892^{o}N and 7.0381^{o}E; 189 at 5.7851^{o}N and 7.0398^{o}E. The CO levels was significantly higher ($P<0.05$) than normal levels.

In Imo north the CO level in ppm was 188 at GPS location 5.8191^{o}N and 7.3411^{o}E; 185 at 5.8191^{o}N and 7.3411^{o}E; 181 at 5.8191^{o}N and 7.3411^{o}E; 180 at 5.8191^{o}N and 7.3411^{o}E; 185 at 5.8191^{o}N and 7.3411^{o}E; 187 at 5.8191^{o}N and 7.3411^{o}E. The CO levels was significantly higher ($P<0.05$) than normal levels.

Table 4.4: Distribution of Carbon Monoxide levels at construction sites in Imo State

GPS Location North	GPS Location East	CO (ppm)
5.4882^{o}	7.0175^{o}	183
5.4859^{o}	7.0171^{o}	181
5.4840^{o}	7.0180^{o}	183
5.4833^{o}	7.0169^{o}	182
5.4851^{o}	7.0173^{o}	187
5.4889^{o}	7.0182^{o}	185
Mean ± Standard deviations		183.5 ± 2.17
P-value	<0.0001	
IMO East		
5.7822^{o}	7.0390^{o}	185
5.7835^{o}	7.0388^{o}	186
5.7830^{o}	7.0398^{o}	184
5.7846^{o}	7.0383^{o}	180
5.7892^{o}	7.0381^{o}	185
5.7851^{o}	7.0398^{o}	189
Mean ± Standard deviations		185.34 ± 6.49
P-value	<0.0001	
IMO North		
5.8191^{o}	7.3411^{o}	188
5.8170^{o}	7.3406^{o}	185
5.8186^{o}	7.3424^{o}	181
5.8198^{o}	7.3439^{o}	180
5.8182^{o}	7.3401^{o}	185
5.8169^{o}	7.3453^{o}	187
Mean ± Standard deviations		187.51 ± 6.49
P-value	0.02	

4.1.5 Sulphur dioxide levels at construction sites in Imo West

In table 4.5, the SO_2 level in $\mu g/m^3$ was 460 at GPS location 5.4882^oN and 7.0175^oE; 559 at 5.4859^oN and 7.0171^oE; 453 at 5.4840^oN and 7.0180^oE; 477 at 5.4833^oN and 7.0169^oE; 510 at 5.4851^oN and 7.0173^oE; 536 at 5.4889^oN and 7.0182^oE. The mean SO_2 levels (499.17 ± 42.92) was found to be significantly higher than normal levels at 5% level of significance ($P<0.0001$).

In Imo East SO_2 level in $\mu g/m^3$ was 466 at GPS location 5.7822^oN and 7.0390^oE; 557 at 5.7835^oN and 7.0388^oE; 470 at 5.7830^oN and 7.0398^oE; 449 at 5.7846^oN and 7.0383^oE; 590 at 5.7892^oN and 7.0381^oE; 544 at 5.7851^oN and 7.0398^oE. The SO_2 levels was significantly higher ($P<0.05$) than normal levels.

In Imo North SO_2 level in $\mu g/m^3$ was 449 at GPS location 5.8191^oN and 7.3411^oE; 578 at 5.8191^oN and 7.3411^oE; 461 at 5.8191^oN and 7.3411^oE; 480 at 5.8191^oN and 7.3411^oE; 503 at 5.8191^oN and 7.3411^oE; 522 at 5.8191^oN and 7.3411^oE. The SO_2 levels was significantly higher ($P<0.05$) than normal levels.

Table 4.5: Distribution of Sulphur dioxide levels at construction sites in Imo State

GPS Location North	GPS Location East	SO_2 (µg/m^3)
IMO WEST		
5.4882^o	7.0175^o	460
5.4859^o	7.0171^o	559
5.4840^o	7.0180^o	453
5.4833^o	7.0169^o	477
5.4851^o	7.0173^o	510
5.4889^o	7.0182^o	536
Mean ± Standard deviations		499.17 ± 42.92
P-value	<0.0001	
IMO EAST		
5.7822^o	7.0390^o	466
5.7835^o	7.0388^o	557
5.7830^o	7.0398^o	470
5.7846^o	7.0383^o	449
5.7892^o	7.0381^o	590
5.7851^o	7.0398^o	544
Mean ± Standard deviations		518.26 ± 55.12
P-value	<0.0001	
Imo North		
5.8191^o	7.3411^o	449
5.8170^o	7.3406^o	578
5.8186^o	7.3424^o	461
5.8198^o	7.3439^o	480
5.8182^o	7.3401^o	503
5.8169^o	7.3453^o	522
Mean ± Standard deviations		520.14 ± 55.34
P-value	**<0.0001**	

4.1.6 Blood pressure and pulse rate of the road construction sites Workers in Imo state

4.1.6.1 Systolic Blood Pressure and Diastolic Blood Pressure of the road construction sites Workers

From the table 4.6, a total of 15 (4.25%) subjects had a systolic pressure between 81 – 99; for 100 - 119, 85 (24.08%); 120 – 139, 171 (48.44%); 140 – 159, 62 (17.56%); 160 and above, 20 (5.67%). The systolic blood pressure of the study group differs significantly than normal levels (P< 0.005). A total of, 23 (6.52%) subjects had a diastolic pressure between 41 – 59; for 60 - 79, 225 (63.74%); 80 – 99, 95 (26.91%); 100 – 120, 10 (2.83%).

Table 4.6: Summary Distribution of systolic and Diastolic Blood pressure classes of all subjects in Imo state

	Systolic Blood Pressure (SBP)			**Diastolic Blood Pressure (DBP)**		
BP Class	SBP class (mmHg)	n	%	DBP class (mmHg)	n	%
Low BP	81 – 99	15	4.25	41 – 59	23	6.52
Normal BP	100 – 119	85	24.08	60 – 79	225	63.74
Elevated BP	120 – 139	171	48.44	80 – 99	95	26.91
HBP stage 1	140 – 159	62	17.56	100 and above	10	2.83
HBP stage 2	160 and above	20	5.67			
Total		353	100.0		353	100.00
Min (Max)		90 (178)			50 (113)	
Mean ± SD		128.85 ± 17.44			75.38± 11.21	
P-value		< 0.0001			< 0.0001	

4.1.6.2 Comparison of systolic pressure values at different Senatorial zones

In table 4.7, up to 6 (4.96%) subjects had a systolic pressure between 81 – 100 at Imo West, 5 (4.10%) in Imo East; 4 (3.64%) in Imo North; 101 – 120, 25 (20.66%) at Imo West, 30 (24.59%) at Imo East, 30 (27.27%) at Imo North; 121 – 140, 58 (47.93%) at Imo West, 56 (45.90%) at Imo East, 57 (51.82%) at Imo North; 141 – 160, 22 (18.18%) at Imo West, 24 (19.67%) at Imo East, 16 (14.55%) at Imo North; 161 – 180, 10 (8.26%) at Imo West, 7 (5.74%) at Imo East, 3 (2.73%) at Imo North. There was no significant difference ($P<0.05$) in systolic blood pressure between the 3 Senatorial zones.

From the table, the minimum value was 90 at Imo West, 102 at Imo East, 98 at Imo North; maximum value was 173 Imo West, 178 at Imo East, 177 at Imo North; mean value was 127.31 at Imo West, 128.98 at Imo East, 129.20 at Imo North; standard deviation was 17.47 at Imo West, 17.11 at Imo East, 17.56 at Imo North. In Imo State, the minimum value was 90; maximum 178; mean 128.85; standard deviation 17.44.

Table 4.7: Comparison of systolic pressure values at different Senatorial zones

Systolic Pressure	Imo West		Imo East		Imo North	
(mmHg)	**n**	**%**	**n**	**%**	**n**	**%**
81 – 99	6	4.96	5	4.10	4	3.64
(Low BP)						
100 – 119	25	20.66	30	24.59	30	27.27
(Normal BP)						
120 – 139	58	47.93	56	45.90	57	51.82
(Elevated BP)						
140 – 159	22	18.18	24	19.67	16	14.55
(HBP stage 1)						
160 and above	10	8.26	7	5.74	3	2.73
(HBP stage 2)						
Total	121	100.00	122	100.00	110	100.00
Min (max)	90	173	102	178	98	177
Mean ± SD	127.31	17.47	128.98	17.11	129.20	17.56
P-value			0.348			

n- Number; Min- Minimum value; Max- Maximum value; S.D - Standard Deviation

4.1.6.3 Comparison of diastolic pressure values at different Senatorial zones

From the table 4.8, we have 8 (6.61%) subjects had a diastolic pressure between 41 – 60 at Imo West, 8 (6.56%) at Imo East; 7 (6.36%) at Imo North; 61 – 80, 78 (64.46%) at Imo West, 76 (62.30%) at Imo East, 71 (64.55%) at Imo North; 81 – 100, 32 (26.45%) at Imo West, 35 (28.69%) at Imo East, 28 (25.45%) at Imo North; 101 – 120, 3 (2.48%) at Imo West, 3 (2,46%) at Imo East, 4 (3.64%) at Imo North. The table above, the minimum value was 50 at Imo West, 52 at Imo East, 53 at Imo North; maximum value was 110 Imo West, 113 at Imo East, 105 at Imo North; mean value was 74.66 at Imo West, 76.14 at Imo East, 75.89 at Imo North; standard deviation was 11.60 at Imo West, 10.04 at Imo East, 11.67 at Imo North. In Imo state, the minimum value was 50; maximum 113; mean 75.38; standard deviation 11.21mmHg.

There was no significant difference (P= 0.11) in diastolic blood pressure between the 3 senatorial zones.

Table 4.8: Comparison of diastolic pressure values at different Senatorial zones

Diastolic Pressure	Imo West		Imo East		Imo North	
(mmHg)	**n**	**%**	**n**	**%**	**n**	**%**
40 – 59	8	6.61	8	6.56	7	6.36
60 – 79	78	64.46	76	62.30	71	64.55
80 – 99	32	26.45	35	28.69	28	25.45
100 and above	3	2.48	3	2.46	4	3.64
Total	121	100.00	122	100.00	110	100.00
Min (max)	50 (110)		52	113	53	105
Mean ± SD	74.66 ± 11.60		76.14 ± 10.04		75.89 ± 11.67	
P-value	0.11					

n- Number; Min- Minimum value; Max- Maximum value; S.D - Standard Deviation

4.1.6.4 Pulse rate

Data in figure 4.1 is the pulse rate measurements of the study participants. It shows that , 29 (8.22%) subjects had a pulse rate between 41 – 60; for 61 – 80, 181 (51.24%); for 81 - 100, 121 (34.28%); 101 – 100, 22 (6.23%). The pulse rate of the subjects did not differ significantly from the normal range (P = 0.22).

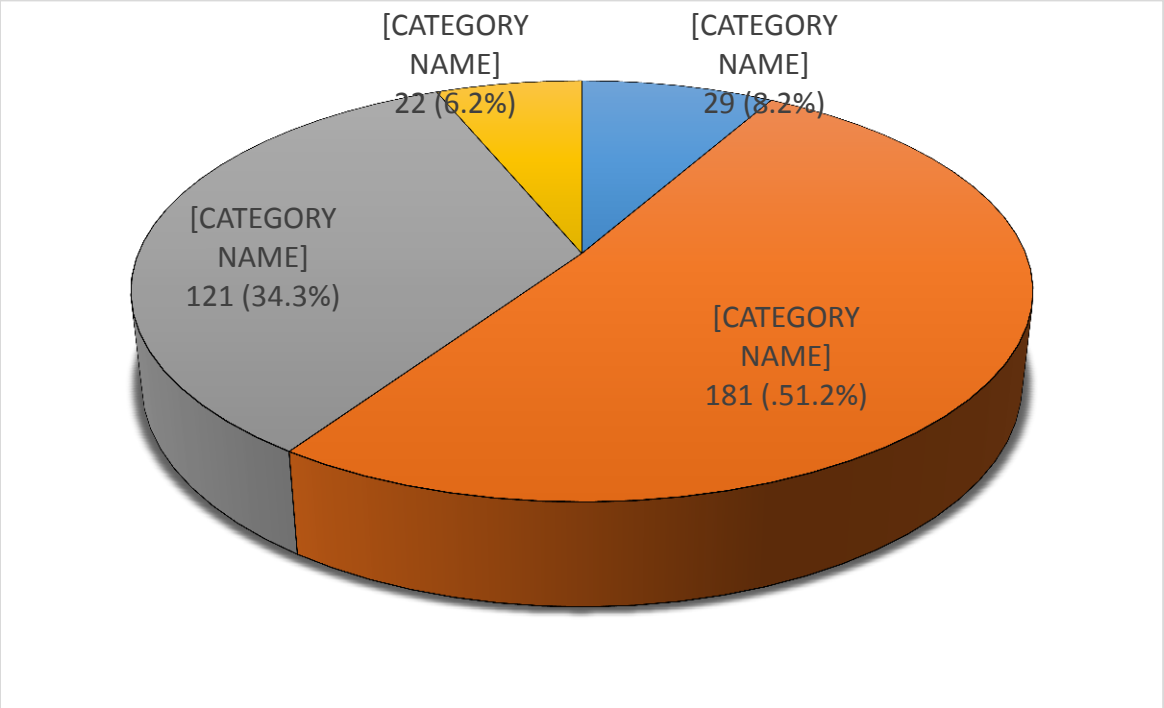

Figure 4.1: Distribution of pulse rate of all subjects in Imo state (P = 0.22)

4.1.6.5 Comparison of pulse rate at different Senatorial zones

In table.4.9, 10 (8.26%) subjects had a pulse rate between 41 – 60 at Imo North, 11 (9.02%) at Imo East; 8 (7.27%) at Imo North; 61 – 80, 61 (50.41%) at Imo North, 62 (50.82%) at Imo East, 58 (52.73%) at Imo North; 81 – 100, 40 (33.06%) at Imo North, 43 (35.25%) at Imo East, 38 (34.55%) at Imo North; 101 – 120, 10 (8.26%) at Imo North, 6 (4.92%) at Imo East, 6 (5.45%) at Imo North. There was no significant difference (P<0.05) in the pulse rate between the 3 senatorial zones.

From the table, the minimum value was 53 at Imo West, 52 at Imo East, 55 at Imo North; maximum value was 115 Imo West, 109 at Imo East, 112 at Imo North; mean value was 79.45 at Imo West, 76.68 at Imo East, 78.02 at Imo North; standard deviation was 12.40 at Imo West, 12.63 at Imo East, 12.46 at Imo North. In Imo state, the minimum value was 52; maximum 115; mean 78.50; standard deviation 12.55

Table 4.9: Comparison of pulse rate at different Senatorial zones

Systolic Pressure (mmHg)	Imo North		Imo East		Imo North	
	n	%	N	%	n	%
41 – 60	10	8.26	11	9.02	8	7.27
61 – 80	61	50.41	62	50.82	58	52.73
81 – 100	40	33.06	43	35.25	38	34.55
101 – 120	10	8.26	6	4.92	6	5.45
Total	121	100.00	122	100.00	110	100.00
Min (max)	53 (115)		52 (109)		55 (112)	
Mean ± SD	79.45 ± 12.40		76.68 ± 12.63		78.02 ± 12.46	
P-value	0.45					

n- Number; Min- Minimum value; Max- Maximum value; S.D - Standard Deviation

4.1.7 Forced Vital Capacity (FVC) of road construction sites Workers in Imo

4.1.7.1 Distribution for FVC among the Study Group

From Figure 4.2, it can be observed that 17 (4.82%) subjects had a Forced Vital Capacity between 0 – 1000; for 1001 – 2000, 63 (17.85%); for 2001 – 3000, 200 (56.66%); 3001 – 4000, 63 (17.85%); 4001 – 5000, 10 (2.83%).

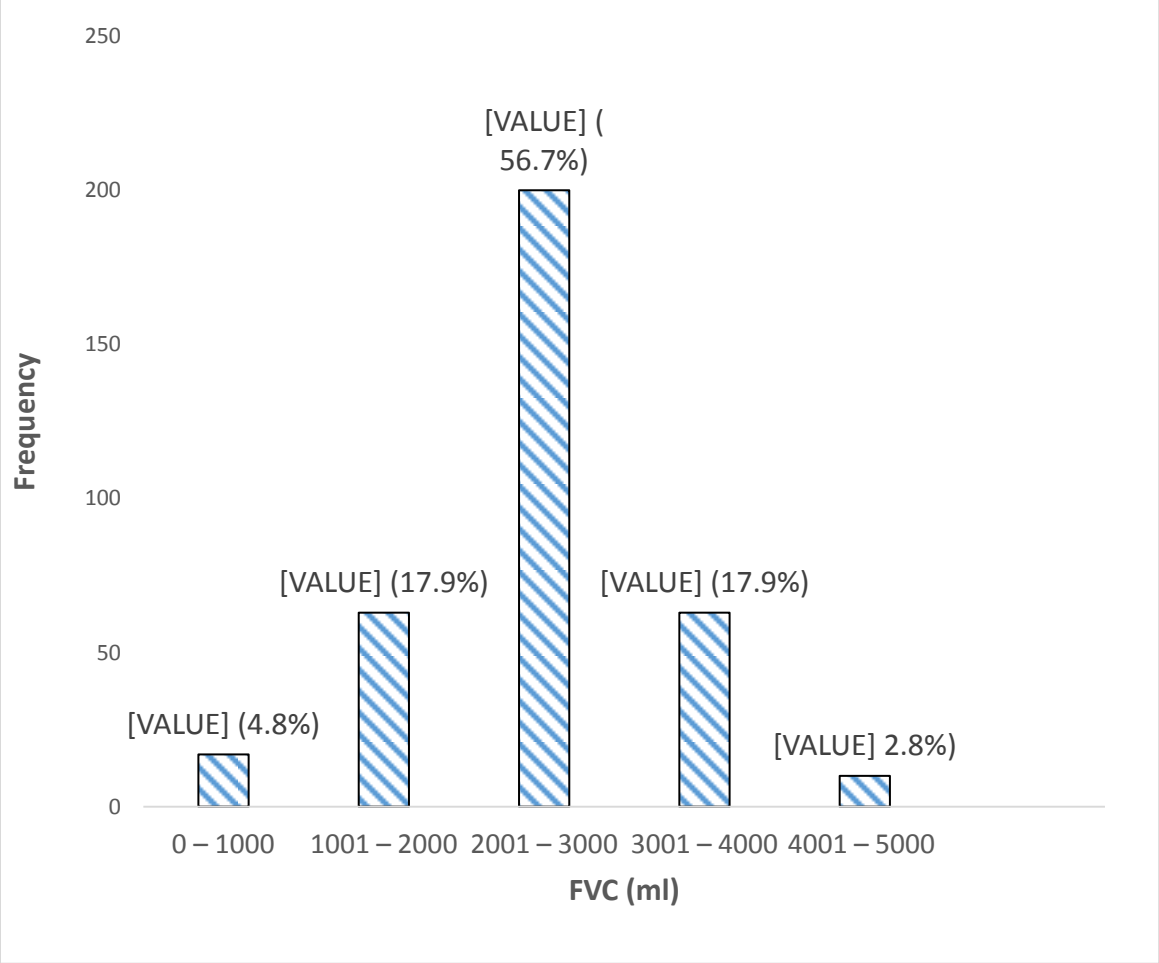

Figure 4.2: Distribution for FVC among Road Site Construction Workers Studied

4.1.7.2 Comparison of Forced Vital Capacity values at different Senatorial zones

At different senatorial zones (Table 4.10), 6 (4.96%) subjects had a Forced Vital Capacity between 0 – 1000 at Imo West, 6 (4.92%) at Imo East; 3 (2.73%) at Imo North; 1001 – 2000, 25 (20.66%) at Imo West, 20 (16.39%) at Imo East, 21 (19.09%) at Imo North; 2001 – 3000, 67 (55.37%) at Imo West, 70 (57.38%) at Imo East, 67 (60.91%) at Imo North; 3001 – 4000, 19 (15.70%) at Imo West, 22 (18.03%) at Imo East, 19 (18.92%) at Imo North; 4001 – 5000, 4 (3.31%) at Imo West, 4 (3.28%) at Imo East, 0 (0.00%) at Imo North;. There was no significant difference ($P<0.05$) in the Forced Vital Capacity between the 3 Senatorial zones.

Table 4.10: Comparison of Forced Vital Capacity values at different Senatorial zones

		Imo West		**Imo East**		**Imo North**	
FVC (ml)	**Total**	**N**	**%**	**n**	**%**	**n**	**%**
0 – 1000	**17**	**6**	5.0	7	5.7	4	3.6
1001 – 2000	63	**24**	19.8	19	15.6	20	18.2
2001 – 3000	200	**66**	55.0	68	55.7	66	60.0
3001 – 4000	63	**20**	16.7	23	18.9	20	18.2
4001 – 5000	10	**5**	4.2	5	4.1	0	0.0
Total	353	121	100.00	122	100.00	110	100.00
Min (Max)		1250 (3270)		1190 (4118)		1885 (4491)	
Mean ± std dev		2263.3 ± 482.3		2578.8 ± 751.80		2382.8 ± 610.4	
P-value				**P=0.17**			

n- Number; Min- Minimum value; Max- Maximum value; S.D - Standard Deviation

From the table above, the minimum value was 1250 at Imo West, 1190 at Imo East, 1885 at Imo North; maximum value was 3270 Imo West, 4118 at Imo East, 4491 at Imo North; mean value was 2263.34 at Imo West, 2578.80 at Imo East, 2382.78 at Imo North; standard deviation was 482.33 at Imo West, 751.80 at Imo East, 610.42 at Imo North. In Imo State, the minimum value was 1190; maximum 4491; mean 2369.22; standard deviation 562.87.

4.1.7.3 Distribution of Respiratory Health problems among all subjects

Figure 4.3 represents the distribution of respiratory health problems among the study road construction workers in Imo state. It showed that 211(59.77%) of the subjects complained of cough; sneezing, 290(82.15%); catarrh, 79(22.38%); sore throat, 72(20.39%); asthma, 29(8.22%); short breadth, 121(34.28%); pneumonia, 66(18.70%); headache, 133(37.68%), wheezing, 170(48.16%).

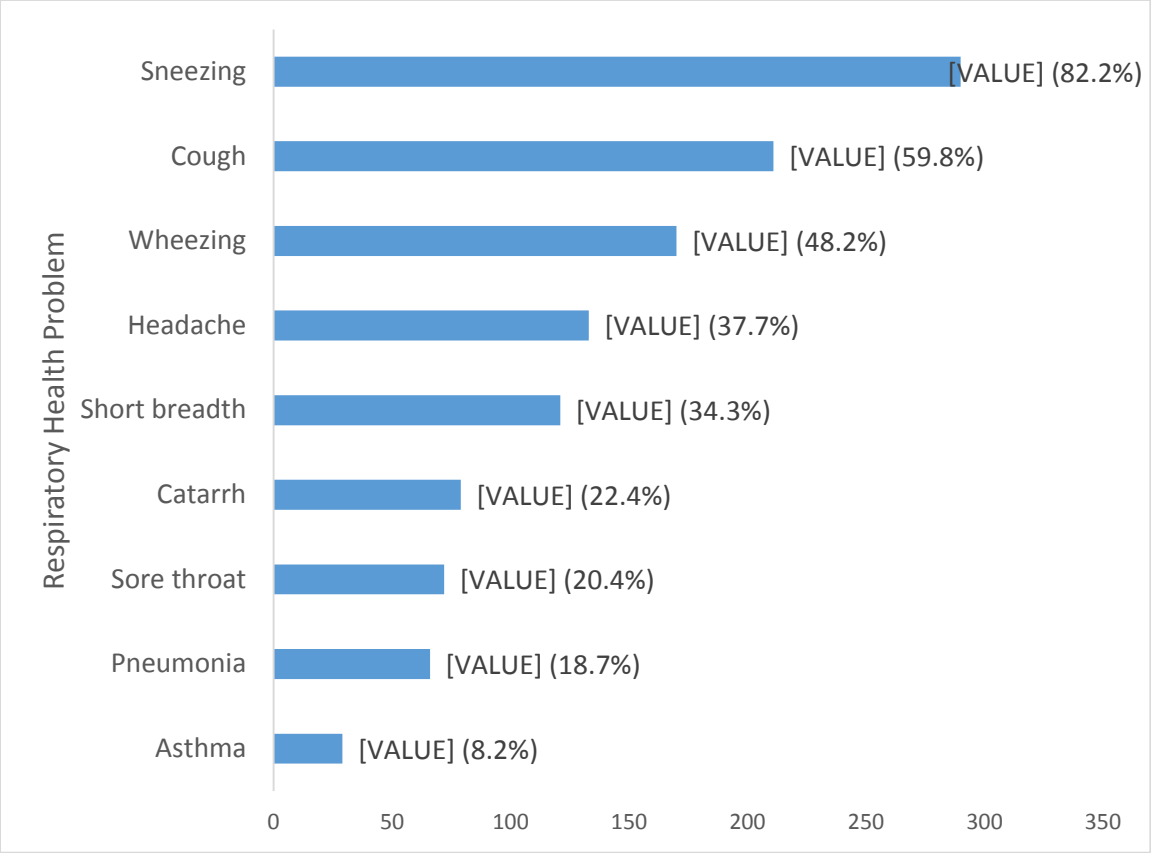

Figure 4.3: Distribution of Respiratory Health problems among Road site Construction Workers in Imo state

4.1.7.4 Comparison of Respiratory Health problems among all subjects in the 3 Senatorial zones of Imo State

The table above showed that 69(56.50%) of the subjects in Imo West complained of cough, 75(58.50%) in Imo East, 62(56.40%) in Imo North; sneezing, 90(76.45%) in Imo West, 88(75.11%) in Imo East, 85(80.10%) in Imo North; catarrh, 24(21.47%) in Imo West, 26(20.32%) in Imo East, 28(20.72%) in Imo North; headache, 45(40.03%) in Imo West, 44(36.83%) in Imo East, 46(42.14%) in Imo North; wheezing, 54(49.12%) in Imo West, 52(44.15%) in Imo East, 55(53.45%) in Imo North.

Table 4.11: Comparison of Respiratory Health problems among all subjects in the 3 Senatorial zones

Health Problem	Imo West		Imo East		Imo North	
	n	%	n	%	n	%
Cough	69	56.50	75	58.50	62	56.40
Sneezing	90	76.45	88	75.11	85	80.10
Catarrh	24	21.47	26	20.32	28	20.42
Sore throat	23	20.05	24	19.50	20	19.87
Asthma	8	6.49	10	7.32	7	5.45
Short breadth	40	35.80	42	37.59	33	29.23
Pneumonia	26	18.43	27	20.36	24	18.90
Headache	45	40.03	44	36.83	46	42.14
Wheezing	54	49.12	52	44.15	55	53.45

4.2 DISCUSSION OF FINDINGS

Road construction involves intensive labor with the use of heavy machinery which emit gases and dust into the atmosphere and thus exposing the workers to inhalation of poisonous gases and dust particles that can cause respiratory health problems. It is a male-dominated profession due to its labor intensive nature. Most of the equipment involved in road construction include tractors, cement mixers, bull dozers, caterpillars, etc., all of which are operated by men. In this study, all the road construction workers interviewed were males. They engage in the removal of earth and rock by digging or blasting, construction of embankments, removal of vegetation and the laying of pavement material. Storm drainages, erosion and sediment controls are constructed to prevent detrimental effects. The most common types of pollution found on a construction site are dust and diesel emissions. The dust types include soil dust, gravel dust, wood dust, silica dust, non-silica mineral dust and demolition dust (Passchier-Vermeer and Passchier, 2000). These dust particles are called particulate matter. Diesel emissions can be produced on a construction site when an engine combusts diesel fuel for energy, which results in the creation of diesel exhaust. Heavy duty vehicles (dump trucks, cement mixers, transport trucks), road-building machines (excavators, cranes, bulldozers), and stationary engines (generators, pumps, compressors) are all sources of diesel exhaust emissions. Diesel exhaust gas-based pollutants include carbon dioxide, sulphur and nitrogen compounds, carbon monoxide, and hydrocarbons (Geldenhuys, et al., 2022).

Measurements of gaseous levels were taken at the construction sites. Carbon monoxide levels were found to be significantly higher ($P<0.05$) than normal levels. The implication of this is that continuous inhalation and exposure of carbon monoxide gas to the construction workers could result to adverse health effects. Hemoglobin in the blood has a higher affinity for carbon

monoxide than oxygen and it combines with carbon monoxide to form carboxy-hemoglobin. Mild acute CO poisoning can cause light-headedness, confusion, headaches, vertigo, and flu-like effects (Henry, et al., 2006).

Larger exposures can lead to significant toxicity of the central nervous system and heart, and death. Following acute poisoning, long-term sequelae often occur. Chronic exposure to low levels of carbon monoxide can lead to depression, confusion, and memory loss. Carbon monoxide mainly causes adverse effects in humans by combining with hemoglobin to form carboxyhemoglobin (HbCO) in the blood. This prevents hemoglobin from carrying oxygen to the tissues, effectively reducing the oxygen-carrying capacity of the blood, leading to hypoxia. Additionally, myoglobin and mitochondrial cytochrome oxidase are thought to be adversely affected. Carboxyhemoglobin can revert to hemoglobin, but the recovery takes time because the HbCO complex is fairly stable. Inhaling CO gas can lead to hypoxic injury, nervous system damage, and even death. Different people and populations may have different carbon monoxide tolerance levels. On average, exposures at 100 ppm or greater is dangerous to human health (Prockop and Chichkova, 2007).

Carbon monoxide exposure may lead to a significantly shorter life span due to heart damage. The carbon monoxide tolerance level for any person is altered by several factors, including activity level, rate of ventilation, a pre-existing cerebral or cardiovascular disease, cardiac output, anemia, sickle cell disease and other hematological disorders, barometric pressure, and metabolic rate (Lipman, 2006).

Sulfur dioxide (SO_2) was another gas emitted at the construction sites and was found to significantly higher ($P<0.05$) than normal levels. Because SO_2 is highly soluble in water, most

inhaled SO_2 is absorbed by the mucous membranes of the upper airways with little reaching the lung; however, increased ventilation and oral breathing, such as from exercise, can raise the dose delivered to the lung. SO_2 exposure has been associated with reduced lung function, bronchoconstriction (increased airway resistance), respiratory symptoms, hospitalizations from cardiovascular and respiratory causes, eye irritation, adverse pregnancy outcomes, and mortality. However, it is difficult to attribute these reported associations to SO_2 itself, because it is a precursor to particulate matter and generally exists as a component of a complex, combustion-related pollutant mixture. Experimental studies (Mustapha, et al., 2011; Thacher, et al., 2013) suggest short-term exposures to high levels of sulfur dioxide can be life-threatening. Exposure to 100 parts of sulfur dioxide per million parts of air (ppm) is considered immediately dangerous to life and health. Previously healthy nonsmoking miners who breathed sulfur dioxide released as a result of an explosion in an underground copper mine developed burning of the nose and throat, breathing difficulties, and severe airway obstructions. Long-term exposure to persistent levels of sulfur dioxide can also affect your health.

Lung function changes have been observed in some workers exposed to 0.4–3.0 ppm sulfur dioxide for 20 years or more. However, these workers were also exposed to other chemicals, making it difficult to attribute their health effects to sulfur dioxide exposure alone. Long-term studies on surveying large numbers of children have indicated possible associations between sulfur dioxide pollution and respiratory symptoms or reduced breathing ability (Desalu, 2011). Road construction workers who have breathed sulfur dioxide pollution may develop more breathing problems as they get older, may make more emergency room visits for treatment of wheezing fits, and may get more respiratory illnesses than is typical for children.

The level of suspended particulate matter as a result of road construction activities was also significantly higher than normal levels in our area of study. When these particles are inhaled, the lungs produce mucous to trap the particles and tiny hairs wiggle to move the mucous and particles out of the lung. The mucous leaves the airway by coughing or swallowing. If the particle is small and it gets very far into the lungs, special cells in the lung trap the particles and then they can't get out and this can result in lung disease, emphysema, lung cancer. Because the $PM_{2.5}$ travels deeper into the lungs and because the $PM_{2.5}$ is made up things that are more toxic (like heavy metals and cancer causing organic compounds), it can have worse health effects than the bigger PM_{10}. The effects of inhaling particulate matter include asthma, lung cancer, cardiovascular disease, respiratory diseases, premature delivery, birth defects, and premature death. A study (Pope, 2002) indicated that $PM_{2.5}$ leads to high plaque deposits in arteries, causing vascular inflammation and atherosclerosis which can lead to heart attacks and other cardiovascular problems. The World Health Organization (WHO) estimated in 2005 that fine particulate air pollution ($PM_{2.5}$), causes about 3% of mortality from cardiopulmonary disease, about 5% of mortality from cancer of the trachea, bronchus, and lung, and about 1% of mortality from acute respiratory infections in children under 5 years, worldwide (Cohen, et al., 2005). Short-term exposure at elevated concentrations can significantly contribute to heart disease.

Spirometry measures the forced vital capacity (FVC), which is the greatest volume of air that can be breathed out in a single large breath. In our study, values were low for some subjects and this could be an indicator for respiratory problems (Mason, et al., 2010). Subjects interviewed in this study complained of asthma, sneezing, and cough. Most of the workers and community members have never checked their forced vital capacity in a clinic before and therefore are not aware of the gradual reduction in normal level over the years of which they are exposed to dust and other

gases in the atmosphere. As they breathe in this polluted air, particulate matter blocks the air spaces in their lungs and respiratory problems gradually develop. Ekpenyong, et al. (2012) carried out to assess the respiratory health effect of city ambient air pollutants on transit and non-transit workers and reported respiratory function impairment due to their forced vital capacity levels. It is reasonable to perform spirometry every one or two years to follow how well a person's asthma is controlled. Similar studies (Ana et al. 2009a; Thacher et al. 2013; Mustapha et al. 2013) have reported symptoms of coughing, wheezing, sneezing, etc. upon inhalation of air pollutants.

Road construction workers spend hours at the construction sites and are constantly exposed to wind, dust, debris and varying temperature changes. The continuous inhalation of dust particles will lead to several repertory health problems. In this study, we observed that the workers complained of short breadths, headaches, sore throat, wheezing, catarrh, asthma, coughing and pneumonia. These symptoms were also reported by similar studies (Odeshi et al. 2014; Ibhafidon, 2014). Other studies (Isawumi et al., 2011; Erdogan et al., 2011) have identified pterygium and cataract as the two major ocular problems among construction workers. Mantyjarvi (2000) reported that cataract reduced the visual acuity and contrast sensitivity of industrial workers. In another study (Azuamah, et al. 2014), long-term exposure to ultraviolet rays of the sun was found to cause cataracts as well as pterygium. These ocular signs were seen upon external examination of the construction workers. Though they do not cause any symptoms, but can cause severe reduction in vision if left unmanaged. Azuamah, et al. (2013) reported that cataract and pterygium were among the major causes of low vision in Southeast Nigeria. By wearing their personal protective equipment such as face masks, these respiratory health problems can be mitigated.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 CONCLUSION

This study evaluated ambient air pollutant levels at road construction sites in Imo State, Nigeria, and their impact on workers' respiratory health. The key findings were that road construction workers are exposed to high levels of ambient air pollutants (CO, SO_2 , SPM) exceeding WHO safe limits. The respiratory symptoms, such as wheeze, sneezing, coughing, and shortness of breath, were highly prevalent, with FVC values indicating compromised lung function in some workers. A proportion of employees were ignorant of the dust and chemical hazards associated with road building. These findings highlight the urgent need for protective measures, including mandatory high-quality PPE, routine health monitoring with spirometry, and environmental controls like cleaner fuels and dust suppression, strict enforcement of air quality regulations, and worker education awareness campaigns. Future research should include studies to investigate long-term health problems from exposure to air pollution among road construction workers and comparative geographic comparisons in other regions to support national policy development. Immediate action is critical to safeguard workers' respiratory health and ensure compliance with environmental and occupational safety standards.

(i) The mean levels of CO, SO_2 and SPM of road construction workers in Imo State were found to be higher than safe levels.

(ii) The Forced Vital Capacity of road construction workers in Imo State were found to be higher than normal levels for many of the workers, however compromised in those with respiratory symptoms.

(iii) The mean systolic blood pressure and pulse rate of road construction workers in Imo State were within normal levels.

(iv) Some of the road construction workers were not aware of the respiratory health problems that can result from exposure to air pollutants related to road construction.

(v) The major respiratory health problems found among road construction workers in Imo State are coughing, sneezing, catarrh, wheezing and shortness of breadth.

5.2 RECOMMENDATIONS

(i) Mandatory use of protective equipment: Employers should supply and mandate the use of high-quality PPE, such as respirators that filter harmful gases and fine particles.

(ii) Pre-start and Periodic health monitoring: To track employees' respiratory health and identify early indicators of illnesses linked to pollution, routine medical examinations, including spirometry, should be instituted. The management of the road Construction company should start applying training, induction, pre-employment health screening, warning signs, etc.

(iii) Environmental controls: It is recommended to employ environmental strategies such as the use of cleaner fuel alternatives, regular equipment maintenance, and dust suppression techniques like water spraying.

(iv) Regulatory enforcement: The Federal Ministry of Environment and Occupational Health departments should enforce the adherence to air quality regulations at construction sites.

(v) Awareness campaigns: Workshops and training courses should inform employees about the dangers of air pollution, safe work procedures, and the significance of wearing personal protective equipment (PPE) consistently. Public education and awareness on

respiratory health problems related to air pollution from road construction work should be intensified by health professionals.

(vi) Incentives can be used to motivate the road construction workers to comply to the use of PPEs.

(vii) Further studies are recommended on health problems of air pollution. Areas of further studies include:

- Taking blood samples of the workers to the laboratory for blood test of health problems.
- Investigation into cardiovascular, dermatological, and neurological problems.
- Long term studies to investigate long-term health problems from exposure to air pollution among road construction workers.

5.3 CONTRIBUTION TO KNOWLEDGE

This study added to the existing data that the level of gases (CO, SO_2) and suspended particulate matter at road construction sites are higher than safe levels. This has therefore predisposed road construction workers in Imo State to potential respiratory health problems.

REFERENCES

Abrar, A., Cheema, K. J., Saif, S., and Mahmood, A. (2017). Health status assessment of workers during construction phase of highway rehabilitation projects around lahore, Pakistan. *Journal of occupational health*, *59*(1), 74–80. https://doi.org/10.1539/joh.16-0123-FS

Agency for Toxic Substances and Disease Registry (2008). Health effects of sulfur dioxide. Available at: http://www.eoearth.org/view/article/ Retrieved 15th August 2015.

Aguwa, E.N., Okeke, T.A. and Asuzu, M.C. (2007). The prevalence of occupational asthma and rhinitis among woodworkers in south-eastern Nigeria. *Tanzan Health Res Bull*, 9(1):52-5.

Albert, R.H. (2010). Diagnosis and treatment of acute bronchitis. *Am fam physician*, 82 (11): 1345–50.

Ana, G., Sridhar, M.K. and Bamgboye, E.A. (2009b). Environmental risk factors and health outcomes in selected communities of the Niger delta area, Nigeria. *Perspective Public Health*, 129(4):183-91.

Ana, G.R., Odeshi, T.A., Sridhar, M.K. and Ige, M.O. (2014). Outdoor respirable particulate matter and the lung function status of residents of selected communities in Ibadan, Nigeria. *Perspective Public Health*, 134(3):169-75.

Ana, G.R., Shendell, D.G., Odeshi, T.A. and Sridhar, M.K. (2009a). Identification and initial characterization of prominent air pollution sources and respiratory health at secondary schools in Ibadan, Nigeria. *Journal of Asthma*. 46(7):670-6.

Ashby, B. and Turkington, C. (2007). *The encyclopedia of infectious diseases.* 3rd ed. New York: Facts on File.

Awosusi, A.O. and Akinduitire, I.O. (2014). Perceived health effects of Environmental Noise Pollution on the inhabitants of Ado-Ekiti Metropolis, Ekiti State, Nigeria. *Journal of Biology Agric & Healthcare*, 4(26): 106-113.

Azuamah, Y.C., Amadi, A.N., Esenwah, E.C. and Iloh, G.U. (2013): Major Causes of Low Vision and Blindness in Southeast Nigeria, *International Journal of Health Science Research*, 3(12): 48-52.

Azuamah, Y.C., Obeme-Ndukwe, O., Esenwah, E.C., Bazuaye, K.N., Agu, G.C. and Nwala, O.R. (2014): Major ocular complaints among commercial vehicle drivers in Southern Nigeria. *International Journal of Health Science Research*,, 1(10): 685-688.

Barbosa, F., Tanus-Santos, J.E., Gerlach, R.F. and Parsons, P.J. (2005). A Critical Review of Biomarkers Used for Monitoring Human Exposure to Lead: Advantages, Limitations, and Future Needs. *Environmental Health Perspectives*, 113 (12): 1669 –74.

Bateman, D.N. (2003). Carbon Monoxide. *Medicine*, 31 (10): 233.

Baxi, S.N. and Phipatanakul, W. (2010). The role of allergen exposure and avoidance in asthma. *Adolesc Med State Art Rev*,21 (1): 57–71.

Bell, M. L. (2007). Spatial and Temporal Variation in PM 2.5 Chemical Composition in the United States for Health Effects Studies. *Environmental Health Perspectives*, 115: 989 – 995.

Bell, M. L., Davis, D. L. and Fletcher, T. (2004). A Retrospective Assessment of Mortality from the London Smog Episode of 1952: The Role of Influenza and Pollution. *Environmental Health Perspectives*, 112: 6- 8.

Blumenthal, I. (2001). Carbon monoxide poisoning. *Journal of Royal Society of Medicine*, 94 (6): 270–272.

Boadu, E. F., Wang, C. C., & Sunindijo, R. Y. (2020). Characteristics of the Construction Industry in Developing Countries and Its Implications for Health and Safety: An Exploratory Study in Ghana. *International journal of environmental research and public health*, *17*(11), 4110. https://doi.org/10.3390/ijerph17114110

Boadu, E. F., Okeke, S. R., Boadi, C., Bonsu, E. O., & Addo, I. Y. (2023). Work-related respiratory health conditions among construction workers: a systematic narrative review. *BMJ Open Respiratory Research*, *10*(1), e001736. https://doi.org/10.1136/bmjresp-2023-001736

Boederman, J. (2016). *The Cambridge Ancient History*. Cambridge University Press, 178.

Boulet, L.P. (2009). Influence of comorbid conditions on asthma. *Euro Resp J*, 33 (4): 897–906.

Brook, R. D. (2004). Air Pollution and Cardiovascular Disease. A Statement for Healthcare Professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*, 109: 2655 – 2671.

Chen, H., Goldberg, M.S. and Villeneuve, P.J. (2008). A systematic review of the relation between long-term exposure to ambient air pollution and chronic diseases. *Rev Environ Health*, 23 (4): 243–297.

Choi, I.S. (2001). Carbon monoxide poisoning: systemic manifestations and complications. *J Korean Med Sci*, 16 (3): 253–261.

Cohen, A. J., Ross Anderson, H., Ostro, B., Pandey, K. D., Krzyzanowski, M., Künzli, N., Gutschmidt, K. and Smith, K. (2005). The global burden of disease due to outdoor air pollution. *J. Toxicol. Environ. Health*, 68 (13–14): 1301–7.

Cohen, J. and Powderly, W. (2004). *Infectious Diseases*. 2nd ed. London: Mosby.

da-Silva-Filho, P. L., Botelho, C., Castro, H. A., Ferreira, M. J. M., & Silva, A. M. C. (2020). Prevalence and factors associated with respiratory symptoms among civil construction workers: an occupational health surveillance proposal. *Revista brasileira de medicina do trabalho : publicacao oficial da Associacao Nacional de Medicina do Trabalho-ANAMT*, *17*(1), 119–129. https://doi.org/10.5327/Z1679443520190263

Decramer, M., Janssens, W. and Miravitlles, M. (2012). Chronic obstructive pulmonary disease. *Lancet*, 379 (9823): 1341–51.

Desalu, O.O. (2011). Prevalence of chronic bronchitis and tobacco smoking in some rural communities in Ekiti state, Nigeria. *Niger Postgrad Med J*, 18(2):91-7.

Devereux, G. (2006). ABC of chronic obstructive pulmonary disease. Definition, epidemiology and risk factors. *BMJ*, 332 (7550): 1142–4.

Dietert, R.R. (2011). Maternal and childhood asthma: risk factors, interactions, and ramifications. *Reproductive toxicology*, 32 (2): 198–204.

Dye, J. A. (2001). Acute Pulmonary Toxicity of Particulate Matter Filter Extracts in Rats: Coherence with Epidemiological Studies in Utah Valley Residents. *Environmental Health Perspectives*, 109 (3): 395 – 403.

Ekpenyong, C.E., Ettebong, E.O., Akpan, E.E. and Samson, T.K. (2012). Urban city transportation mode and respiratory health effect of air pollution: a cross-sectional study among transit and non-transit workers in Nigeria. *BMJ*, 2(5): 126-131.

ENVIRONMENTAL PROTECTION AGENCY 40 CFR Part 51 Revisions to the Guideline on Air Quality Models: Enhancements to the AERMOD Dispersion Modeling System and Incorporation of Approaches To Address Ozone and Fine Particulate Matter. (n.d.). https://www.epa.gov/sites/default/files/2020-09/documents/appw_17.pdf

Falk, S. and Williams, C. (2010). *Lung Cancer - the facts*. 3rd ed. London: Oxford University Press.

Fareed, Mohammad & Mahmoud, Mahmoud & Al-Dubai, Sami & Khoja, Abdullah. (2018). Adverse Respiratory Health and Decline in Lung Functions among Workers of Riyadh Metro Railway Tunnel. *International Journal of Medical Research & Health Sciences*, 7. 127-131.

Fawcett, T.A., Moon, R.E., Fracica, P.J, Mebane, G.Y., Theil, D.R. and Piantadosi, C.A. (1992). Warehouse workers' headache. Carbon monoxide poisoning from propane-fueled forklifts. *Journal of Occupational Medicine*, 34 (1): 12–15.

Frumkin, H. (2010). *Environmental Health: From Global to Local*. 2nd ed. San Francisco: John Wiley and Sons.

Fukuhara, M., Abe, I., Matsumura, K., Kaseda, S., Yamashita, Y., Shida, K., Kawashima. H. and Fujishima, M. (1996). Circadian variations of blood pressure in patients with sequelae of carbon monoxide poisoning. *American Journal of Hypertension*, 9 (4): 300–305.

Gandini, C., Castoldi, A.F., Candura, S.M., Locatelli, C., Priori, S. and Manzo, L. (2001). Carbon monoxide cardiotoxicity. *Journal of Toxicology*, 39(1): 35–44.

Geldenhuys, G., Wattrus, M., & Forbes, P. B. C. (2022). Gas and particle phase polycyclic aromatic hydrocarbon emission factors from a diesel vehicle engine: Effect of operating modes in a developing country context. *Atmospheric Environment: X*, *13*, 100158. https://doi.org/10.1016/j.aeaoa.2022.100158

Gerald, L. M., John, E. and Bennett, R. (2010). *Principles and Practice of Infectious Diseases*. 7th ed. Philadelphia: Elsevier.

Gold, D.R. and Wright, R. (2005). Population disparities in asthma. *Annu Rev Public Health*, 26: 89–113.

Goldfrank, L., Neal, F., Nea,l L. and Lewis, N. (2002). *Carbon Monoxide. Goldfrank's toxicologic emergencies.* 7th ed. New York: McGraw-Hill.

Gorman, D., Drewry, A., Huang, Y.L., Sames, C. (2003). The clinical toxicology of carbon monoxide. *Toxicology* 187 (1): 25–38.

Guidotti, T.L. and Ragain, L. (2007). Protecting children from toxic exposure: three strategies. *Pediatr Clin North Am*, 54 (2): 227–35.

Hardy, K.R. and Thom, S.R. (1994). Pathophysiology and treatment of carbon monoxide poisoning. *Journal of Toxicology*, 32 (6): 613–629.

Health Effects Institute. (2002). *Understanding the Health Effects of Components of the Particulate Matter Mix: Progress and Next Steps*.

Hecht, S.S. (2012). Lung carcinogenesis by tobacco smoke. *International Journal of Cancer*, 131 (12): 2724–2732.

Heidari, B., and Marr, L.C. (2015). Real-time emissions from construction equipment compared with model predictions. *Journal of Air Waste Management Association*. 65:115–25.

Helbing, D., Molnar, P., Farkas, I.J. & Bolay, K. (2017). Environment and planning. *Journal of Environmental Health*, 24(3): 376-381.

Henry, C.R., Satran, D., Lindgren, B., Adkinson, C., (2006). Myocardial Injury and Long-term Mortality Following Moderate to Severe Carbon Monoxide Poisoning. *JAMA*, 295 (4): 398–402.

Hoare, Z. and Lim, W.S. (2006). Pneumonia: update on diagnosis and management. *BMJ* 332 (7549): 1077–9.

Hoiberg, D.H. (2010). Abia. Encyclopedia Britannica. 15th ed. Chicago: Encyclopedia Britannica Inc.

Horn, L., Lovly, C.M. and Johnson, D.H. (2015). *Harrison's Principles of Internal Medicine*. 19th ed. McGraw-Hill.

Ibhafidon, L.I., Obaseki, D.O., Erhabor, G.E. and Akor, A.A. (2014). Respiratory symptoms, lung function and particulate matter pollution in residential indoor environment in Ile-Ife, Nigeria. *Niger Medical Journal*, 55(1):48-53.

Ige, O.M. and Awoyemi, O.B. (2002). Respiratory symptoms and ventilatory function of the bakery workers in Ibadan, Nigeria. *West African Journal of Medicine*, 21(4):316-8.

Ige, O.M. and Onadeko, O.B. (2000). Respiratory symptoms and ventilatory function of the sawmillers in Ibadan, Nigeria. *African Journal of Medical Sciences*, 29(2):101-4.

Integrated Urban Drainage (2021**)**. *Modelling Guide*. Available at: https://www.ciwem.org/assets/uploads/IUD_1.pdf

Isawumi, M.A., Adeoti, C.O., Ubah, I.N., Oluwatimlelin, I.O. and Raji, R.A. (2011). Ocular status of commercial vehicle drivers in osun state, Nigeria. *African Journal of Medical Sciences*. 40(4): 405-411.

Jeffrey, C.P. (2010). *Alcamo's Fundamentals of Microbiology*. 9th ed. Sudbury MA: Jones & Bartlett.

Jindal, S.K. (2011). *Textbook of pulmonary and critical care medicine*. New Delhi: Jaypee Brothers Medical Publishers.

Kagawa, J. (2002). Health Effects of Diesel Exhaust Emissions: A Mixture of Air Pollutants of Worldwide Concern. *Toxicology*, 27, 349 – 353.

Karri, S.K., Saper, R.B. and Kales, S.N. (2008). Lead Encephalopathy Due to Traditional Medicines. *Cur Drug Safety,* 3 (1): 54–9.

Kasper, D.L., Hauser, S.L., Jameson, J.L., Fauci, A.S. and Longo, D.L. (2015). *Harrison's Principles of Internal Medicine*. 19th ed. London: McGraw-Hill.

Kattan, M. (2007). Health effects of indoor nitrogen dioxide and passive smoking on urban asthmatic children. *Environ Health Perspect,* 120 (3): 618–24.

Kelly, F.J. and Fussell, J.C. (2011). Air pollution and airway disease. Clinical and experimental allergy. *J Brit Soc Allergy Clin Immunol,* 41(8): 1059–71.

Kendall, G.M. and Smith, T.J. (2002). Doses to organs and tissues from radon and its decay products. *J Radiol Prot,* 22(4):389–406.

Kennedy, S.M., Chambers, R., Du, W. and Dimich-Ward, H. (2007). Environmental and occupational exposures: do they affect chronic obstructive pulmonary disease differently in women and men? *Pro Am Thoracic Soc,* 4 (8): 692–4.

Konstantinos, A. (2010). Testing for tuberculosis. *Aus Prescriber,* 33 (1): 12–18.

Kryter, K.D. (2014). *The handbook of hearing and the effects of noise: physiology, psychology, and public health*. Boston: Academic Press, 45.

Kumar, K., Abbas, A.K. and Fausto, N. (2008). *Robbins and Coltran Pathologic Basis of Disease*. 7th ed. Elsevier.

Kumar, V., Abbas, A.K. and Aster, J.C. (2013). *Robbins Basic Pathology*. 9th ed. Elsevier Saunders.

Landrigan, P.J., Schechter, C.B, Lipton, J.M. (2002). Environmental pollutants and disease in American children. *Environmental Health Perspectives,* 110 (7): 721–8.

Lawn, S.D. and Zumla, A.I. (2011). Tuberculosis. *Lancet,* 378 (9785): 57–72.

Lay, B. & Maxwell, G. (2012). *Ways of the World: A History of the World's Roads and of the Vehicles that used them*. New Brunswick: Rutgers University Press, 66.

Lemanske, R.F. and Busse, W.W. (2010). Asthma: clinical expression and molecular mechanisms. *Journal of Allergy and Clinical Immunology*. 125 (2): S95–102.

Lim, W.Y. and Seow, A. (2012). Biomass fuels and lung cancer. *Respirology,* 17 (1): 20–31.

Linster, C.L. and Van Schaftingen, E. (2007). Vitamin C: Biosynthesis, recycling and degradation in mammals. *FEBS J,* 274 (1): 1–22.

Lipman, G.S. (2006). Carbon monoxide toxicity at high altitude. *Wilderness Environ Med,* 17 (2): 144–145.

Maduka, S.O., Osim, E.E., Nneli, R.O. and Anyabolu, A.E. (2009). Effect of occupational exposure to local powdered tobacco (snuff) on pulmonary function in south eastern Nigerians. *Niger Journal of Physiologic Sciences,* 24(2):195-202.

Mantyjarvi, J. (2000). The Effect of cataract in traffic. *American Journal of Optomology*.122: 101-111.

Martinez, F.D. (2007). Genes, environments, development and asthma: a reappraisal. *Euro Respiratory Journal*, 29 (1): 179–84.

Mason, R.J., Murray, J.F., Broaddus, V.C. and Nadel, J.A. (2010). *Murray and Nadel's textbook of respiratory medicine*. 5th ed. Elsevier.

McGwin, G., Lienert, J. and Kennedy, J.I. (2010). Formaldehyde exposure and asthma in children: a systematic review. *Environmental Health Perspectives,* 118 (3): 313–7.

McLuckie, A. (2009). *Respiratory disease and its management*. New York: Springer.

Mendell, M. J. (2007). Indoor residential chemical emissions as risk factors for respiratory and allergic effects in children: A review. *Indoor Air,* 17 (4): 259–77.

Miller, R.L. and Ho, S.M. (2008). Environmental epigenetics and asthma: current concepts and call for studies. *American Journal Respiration & Critical Care Medicine*, 177 (6): 567–573.

Michie, S., van Stralen, M.M. and West, R. (2011). The behaviour change wheel: A new method for characterising and designing behaviour change interventions. *Implementation Science*, [online] 6(42). doi:https://doi.org/10.1186/1748-5908-6-42.

Mo, S., Wang, Y., & Xiong, F. (2022). Identification and prioritization of key health hazards to workers in roadway construction. *Transportation Safety and Environment*, *4*(2). https://doi.org/10.1093/tse/tdac009

Mustapha, B.A., Blangiardo, M., Briggs, D.J. and Hansell, A.L. (2011). Traffic air pollution and other risk factors for respiratory illness in school children in the Niger-Delta region of Nigeria. *Environmental Health Perspectives*, 119(10):1478-82.

Nair, G.B. and Niederman, M.S. (2011). Community-acquired pneumonia: an unfinished battle. *Med Clin North Am*, 95 (6): 1143–61.

Nathan, E. G. and Sean, R. (2013). *Evidence-based practice of palliative medicine.* Philadelphia: Elsevier/Saunders.

Nathell, L., Nathell, M., Malmberg, P. and Larsson, K. (2007). COPD diagnosis related to different guidelines and spirometry techniques. *Resp Res*. 8 (1): 89.

Odeshi, T.A., Ana, G.R., Sridhar, M.K., Olatunji, A.O. and Abimbola, A.F. (2014). Outdoor air particle-bound trace metals in four selected communities in Ibadan, Nigeria. *Environ Geochem Health*, 36(4):755-64.

Ogbeibu, A.E. (2014). *Biostatistics: A practical approach to research and data handling*. 2nd ed. Benin City: Mindex Publishing.

Okereke, C.D. (2006). *Environmental Pollution Control*, Barloz Publishers.

Okwari, O.O., Antai, A.B., Owu, D.U., Peters, E.J. and Osim, E.E. (2005). Lung function status of workers exposed to wood dust in timber markets in Calabar, Nigeria. *Afr J Med Med Sci*, 34(2):141-5.

Oluwole, O., Arinola, G.O., Ana, G.R., Wiskel, T. and Huo, D. (2013). Relationship between household air pollution from biomass smoke exposure, and pulmonary dysfunction, oxidant-antioxidant imbalance and systemic inflammation in rural women and children in Nigeria. *Global Journal Health Sciences*, 5(4):28-38.

Omaye, S.T. (2002). Metabolic modulation of carbon monoxide toxicity. *Toxicology*, 180 (2): 139–50.

Pacyna, E.G., Pacyna, J.M., Steenhuisen, F., Wilson, S. (2006). Global anthropogenic mercury emission inventory for 2000. *Atmos Environ,* 40 (22): 4048–63.

Parekh, K. (2024). *Road Divider ReportL*. Scribd. https://www.scribd.com/document/266652754/Road-Divider-ReportL

Park, S.K., O'Neill, M.S., Vokonas, P.S., Sparrow, D. and Hu, H. (2008). Air Pollution and Heart Rate Variability: Effect Modification by Chronic Lead Exposure. *Epidemiology*, 19(1): 111–20.

Pope, C.A. (2002). Cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of American. Medical. Association*. 287 (9): 1132–1141.

Prockop, L.D. and Chichkova, R.I. (2007). Carbon monoxide intoxication: an updated review. *Journal of Neurological Sciences*, 262(1): 122-130.

Potter, T. and Schaefer, T.J. (2020). *Hypertensive Encephalopathy*. [online] PubMed. Available at: https://www.ncbi.nlm.nih.gov/books/NBK554499/.

Powers, K. A., & Dhamoon, A. S. (2023, January 23). *Physiology, Pulmonary, Ventilation and Perfusion*. The National Center for Biotechnology Information; StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK539907/

Ruuskanen, O., Lahti, E., Jennings, LC. and Murdoch, D.R. (2011). Viral pneumonia. *Lancet*, 377 (9773): 1264–75.

Schick, S. and Glantz, S. (2005). Philip Morris toxicological experiments with fresh sidestream smoke: more toxic than mainstream smoke. *Tobacco Control*, 14(6): 396–404.

Schiffman, G. (2016). Emphysema. Available at www.emedicinehealth.com [Retrieved 10th June 2016]

Sood, A. (2012). Indoor fuel exposure and the lung in both developing and developed countries: an update. *Clin Chest Med*, 33 (4): 649–65.

Southwick, F. (2007). *Infectious Diseases: A Clinical Short Course*. 2nd ed. McGraw-Hill Medical Publishing Division.

Spengler, J.D., Samet, J.M. (1991). *Indoor air pollution: A health perspective*. Baltimore: Johns Hopkins University Press.

Sulaiman, N. N. M., Awang, N., & Kamaludin, N. F. (2020). Association between respirable dust exposure and lung function deterioration among construction site workers. *Current Science*, *119*(11), 1789–1796. https://www.jstor.org/stable/27229926

Sylla, F. K., Faye, A., Fall, M., & TAL-DIA, A. (2017). Air Pollution Related to Traffic and Chronic Respiratory Diseases (Asthma and COPD) in Africa. *Health*, *09*(10), 1378–1389. https://doi.org/10.4236/health.2017.910101

Tackett, K.L. and Atkins, A. (2012). Evidence-based acute bronchitis therapy. *J Pharm Prac*, 25 (6): 586–90.

Thacher, J.D., Emmelin, A., Madaki, A.J. and Thacher, T.D. (2013). Biomass fuel use and the risk of asthma in Nigerian children. *Respir Med*, 107(12):1845-51.

Thomas, M., & Bomar, P. A. (2023). *Upper respiratory tract infection*. National Library of Medicine; StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK532961/

Thun, M.J., Hannan L.M. and Adams-Campbell, L.L. (2008). Lung cancer occurrence in never-smokers: an analysis of 13 cohorts and 22 cancer registry studies. *PLoS Med*, 5(9):185-89.

Tobias, J. and Hochhauser, D. (2010). *Cancer and its Management*. 6th ed. Wiley-Blackwell.

Ugheoke, A.J., Ebomoyi, M.I. and Iyawe, V.I. (2006). Influence of smoking on respiratory symptoms and lung function indices in sawmill workers in Benin City, Nigeria. *Niger Journal of Physiological Sciences*, 21(1-2):49-54.

United States Department of Labor (2024). *Silica, Crystalline - Overview | Occupational Safety and Health Administration*. [online] www.osha.gov. Available at: https://www.osha.gov/silica-crystalline.

US EPA,OAR. (2019, July). *Air Quality Models | US EPA*. US EPA. https://www.epa.gov/scram/air-quality-models

van Zyl, R.N., Yew, W.W., Leung, C.C. and Zumla, A. (2010). Global lung health: the colliding epidemics of tuberculosis, tobacco smoking, HIV and COPD. *European Respiratory Journal*, 35 (1): 27–33.

Vestbo, J. (2013). *Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease*. Global Initiative for Chronic Obstructive Lung Disease. pp. 1–7.

Weaver, L.K. (2009). Clinical practice. Carbon monoxide poisoning. *England Journal Medicine*, 360 (12): 1217–1225.

Welding Fume. (2020, January 21). Health and Safety Executive Northern Ireland. https://www.hseni.gov.uk/articles/welding-fume

Weschler, C.J. (2006). Workgroup report: indoor chemistry and health. *Environmental Health Perspectives*, 11 (3): 442–6.

Wikipedia Contributors. (2019, September 30). *Respiratory disease*. Wikipedia; Wikimedia Foundation. https://en.wikipedia.org/wiki/Respiratory_disease

Wolkoff, P., Wilkins, C.K., Clausen, P.A.; Nielsen, G.D. (2006). Organic compounds in office environments - sensory irritation, odor, measurements and the role of reactive chemistry. *Ind Air*, 16(1): 7–19.

World Health Organization (2011). The sixteenth global report on tuberculosis. Available at: www.who.org [Retrieved 3rd June 2016]

WHO. (2020). Air quality guidelines for Europe. European Series. No. 91. 2nd ed. WHO Regional Publications; Geneva WHO.

World Health Organization. (2021). *Types of pollutants*. Www.who.int. https://www.who.int/teams/environment-climate-change-and-health/air-quality-and-health/health-impacts/types-of-pollutants

Xu, J., Yan, H.C., Yang, B., Tong, L.S. and Zou, Y.X. (2009). Effects of lead exposure on hippocampal metabotropic glutamate receptor subtype 3 and 7 in developmental rats. *J Neg Res Biomed*, 8: 5-11.

Yankson, I. K., Okyere, S., Wireko-Manu, F., Dugan, E., Ampomah, A. F., Acquah, E., Yeboah-Adjei, E., Okyere, P., Yalley, A. K., Asenso-Gyambibi, D., Donkor, P., Mock, C., & Afukaar, F. K. (2024). Assessment of particulate matter concentrations and air quality index during road construction-related activities in Ghana. *Discover Environment*, *2*(1). https://doi.org/10.1007/s44274-024-00172-9

Yawn, B.P. (2008). Factors accounting for asthma variability: achieving optimal symptom control for individual patients. *Pri Care Resp J*, 17 (3): 138–147.

APPENDICES

APPENDIX A: WHO Air Quality Guideline

Table 0.1Recommended AQG levels and interim targets

Pollutant	Averaging time	Interim target				AQG level
		1	2	3	4	
$PM_{2.5}$, μg/m³	Annual	35	25	15	10	5
	24-hour[a]	75	50	37.5	25	15
PM_{10}, μg/m³	Annual	70	50	30	20	15
	24-hour[a]	150	100	75	50	45
O_3, μg/m³	Peak season[b]	100	70	–	–	60
	8-hour[a]	160	120	–	–	100
NO_2, μg/m³	Annual	40	30	20	–	10
	24-hour[a]	120	50	–	–	25
SO_2, μg/m³	24-hour[a]	125	50	–	–	40
CO, mg/m³	24-hour[a]	7	–	–	–	4

[a] 99th percentile (i.e. 3–4 exceedance days per year).

b Average of daily maximum 8-hour mean O3 concentration in the six consecutive months with the highest six-month running-average O3 concentration.

APPENDIX B: NIGERIA Federal Ministry of Environment Air Quality Guideline

AMBIENT AIR QUALITY STANDARDS FOR CRITERIA POLLUTANT AND AIR TOXICS

S/N	*Pollutants*	*Time Weighted Average*	*Concentration in Ambient Air*
1.	Sulphur dioxide (SO_2)	Annual	80 µg/m³
		24 hours	120 µg/m³
		1hour	350 µg/m³
2.	Nitrogen dioxide (NO_2)	Annual	80 µg/m³
		24 hours	120 µg/m³
		1hour	200 µg/m³
3.	Carbon monoxide (CO)	8 hours	5.0 mg/m, 10.000ᵇ
		1 hour	10 mg/m³ 25.000ᵇ
4.	Particulate Matter (PM_{10})	Annual	60 µg/m³
		24 hours	150 µg/m³
5.	Particular Matter ($PM_{2.5}$)	Annual	20 µg/m³
		24 hours	40 µg/m³
6.	Ozone (O_3)	8 hours	100 µg/m³
		1 hour	180 µg/m³
7.	Lead (Pb)	Annual	1.0 µg/m³ 0.5
		24 hours	1.4 µg/m³
8.	Arsenic (As)	Annual	6.000 µg/m
9.	Nickel (Ni)	Annual	20.000 µg/m³
10.	Cadmium (Cd)	Annual	5,000 µg/m³
11.	Ammonia (NH_3)	Annual	0.2 mg/m³
		24 hours	0.6 mg/m³