

**EFFECTS OF GAS FLARING ON THE LUNG
HEALTH OF IBENO COMMUNITY RESIDENTS,
IBENO LGA, AKWA IBOM STATE,
SOUTH-SOUTH NIGERIA**

BY

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A DISSERTATION

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(PULMONOLOGY)**

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DECLARATION

It is hereby declared that this work is original, unless otherwise acknowledged. It has not been presented to any other college for a fellowship requirement and has not been submitted for any publication.

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CERTIFICATION

We certify that Dr. Mfon Etop Ekwere of the Department of Internal Medicine, University of Uyo Teaching Hospital, Uyo, carried out this work under our supervision.

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DEDICATION

This work is dedicated to Almighty God for preserving my life and for His inspiration.

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LIST OF ABBREVIATIONS

No ₂	Nitrogen Dioxide
CO	Carbon Monoxide
SO ₂	Sulphur Dioxide
PEFR	Peak Expiratory flow rate
FEV ₁	Forced Expiratory volume in one second
FVC	Forced Vital Capacity
FEF ₂₅₋₇₅	Forced Expiratory flow between 25-75%
PM ₁₀	Particulate matter < 10µm
PM _{2.5}	Particulate matter > 2.5 µm
WHO	World Health Organization
BMI	Body Mass Index
EPA	Environmental Protection Agency
NAAQS	National Ambient Air Quality Standards
AQI	Air Quality Index
FEPA	Federal Environmental Protection Agency
NNPC	Nigerian National Petroleum Co-operation
LPG	Liquified Petroleum Gas
PIC	Products of Incomplete Combustion
BTEX	Benzene, Toluene, ethybenzene and xylene
GGFR	Global Gas Flaring Reduction

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ABSTRACT

Background: Epidemiological studies indicate that exposure to air pollution caused by gas flaring may have an association with an increased risk of adverse respiratory effects. This study investigates the effect(s) of gas flaring on the lung health of Ibeno community residents, Akwa Ibom State focusing on respiratory symptoms and lung function indices.

Methods: A total of 386 adults resident in Ibeno for at least two years and 386 age, sex and height matched controls resident in Etinan, also for at least two years were recruited to a cross-sectional survey comparing their respiratory symptoms and lung function indices. The study was conducted between March and May 2014 using spirometer and questionnaire as investigative tools. Both descriptive and inferential statistics were used to analyze the data.

Results: Most of the respondents in both exposed and control communities were aged 18-30 years, with a height range of 161-170cm. Both exposed and controls subjects experienced similar symptoms suggestive of respiratory disorder, however the prevalence was higher among exposed subjects than controls: cough- 57(14.8%) vs. 39(10.1%); breathlessness 58(15%) vs. 28(7.3); wheezing 22(5.7) vs. 12(3.1). The respondents from the exposed community were mostly traders and fishermen while the controls were predominantly farmers. Most of the respondents from both communities had lived there for at least 30 years. The PEFR, FVC and FEV1 (mean±SD) for the subjects were 300.6±2.15 l/min, 2.58±8.43 and 2.01±0.76 respectively; while the PEFR, FVC and FEV1 for the controls were 342±2.16l/min, 2.27±0.82 and 2.13±0.75 respectively. The mean PEFR and FEV1 was relatively lower among exposed subjects than controls (p= 0.000 and 0.027 respectively) while the difference in FVC between the subjects and controls was not statistically significant. Possibly this was masked by the

negative effects of biomass exposure from cooking among both exposed subjects and controls. The anthropometric variables (height, weight,) were positively correlated with the lung function indices; however the exposed subjects had more overweight and obese individuals: 128(33.2%) vs. 62(16.1%) than the controls: 92(23.8%) vs.56 (14.5%)

Conclusion: In view of these findings, there is evidence that prolonged exposure to air pollution from gas flaring impacts negatively on lung function reflected as reduced PEFR, FEV1, and worsening respiratory symptoms among residents in the exposed community. There is need for the government to speed up the implementation of policies regarding the reduction of flaring of natural gas associated with oil production. There is also a need for additional research to be carried out on the long term effects of chronic exposure to gas flaring on lung health and methods of interventions to minimize or possibly eliminate these effects.

CHAPTER ONE

INTRODUCTION

Air pollution caused by gas flaring is a global problem associated with negative impacts on the environment as well as respiratory morbidity and mortality. It has been estimated that air pollution contributes to 6% of total mortality. (1) Gas flaring generates smoke which is a recognized source of toxic exposure. Smoke is a vaporous colloidal system formed when a material undergoes combustion, and is composed of a collection of emitted noxious gases, airborne solid and liquid particles whose distribution is determined by size, breathing mechanics and total volume. (2) The exact composition of smoke depends on the nature of the burning fuel and the conditions of combustion. (2-3) Combustion can be complete or incomplete depending on the amount of oxygen available. For most fuels such as diesel oil, coal or wood, pyrolysis occurs before combustion. (4) During incomplete combustion, products of pyrolysis remain unburnt and contaminate the smoke with noxious particulate matter and gases. Pyrolysis is a thermo-chemical decomposition of organic materials at elevated temperature without the participation of oxygen. (5)

The entire respiratory system is continuously exposed to particulate and infective agents and is protected by a well-developed physiological barrier. However, these protective barriers can be overwhelmed in adverse situations leading to untoward effects on the respiratory system.

Gas flaring is widely used for the disposal of natural gas in petroleum producing areas where there is no infrastructure to make use of the gas. (6) It is the burning of natural gas that is associated with crude oil when it is pumped from the ground, and is a cheap way to separate the identified product, crude oil from the associated natural gas. Gas

flaring can also be performed as part of the completion and testing of natural gas well to assess a well's capability. (6-7) It is the usual option where insufficient investment was made in infrastructure to utilize the associated gas. Gas flaring in Nigeria has been practiced since oil exploration began in the sixties. (8) After Russia, Nigeria flares more gas than any other country in the world in terms of total volume of gas flared. Nigeria flares over 75% of the associated gas it produces and this represents a pollution equivalent to 45 million tons of carbon dioxide per day. Currently there are over 123 flaring sites in the Niger Delta region and Nigeria has been regarded as one of the highest emitter of greenhouse gases in Africa. (8-9) Gas flaring not only wastes a potentially valuable source of energy; the carbon emissions it adds to the atmosphere leads to global warming and its concomitant effects.

The efficiency of flares can be dependent on several factors like composition of the flare stream, flow rate of gases, wind velocity, ambient turbulence, presence of hydrocarbon droplets in the flare stream and presence of water droplets in the flare stream. (10) Flaring combustion is typically incomplete releasing substantial amount of soot and carbon monoxide which contribute to air pollution problems. Gas flaring is generally discouraged as it releases toxic components into the atmosphere and contributes to climate change. (11) In Western Europe, 99% of associated gas is used or re-injected into the ground. If properly stored, associated gas can be used as an energy source for community projects. Flaring in Nigeria has grown proportionally with oil production. Gas flares also have potentially harmful effects on the health and livelihood of the communities in their vicinity as they release a variety of poisonous chemicals including nitrogen dioxides, particulate matter, sulphur dioxide, volatile organic compounds like benzene, toluene, xylene and hydrogen sulphide as well as carcinogens like

benzopyrene and dioxin. (12-14) Humans who are exposed to such substances can suffer from a variety of respiratory problems. These chemicals can aggravate asthma, chronic obstructive pulmonary disease (COPD), hay fever and also lead to increased mortality both in children and adults. Benzene is a recognized cause of leukemia and bronchial carcinoma. As outdoor air pollution became a well-known public health problem, efforts have been geared towards reducing air pollution over the years. In the United States, the Federal Government enacted a series of Clean Air Acts, which required the Environmental Protection Agency (EPA) to set National Ambient Air Quality Standards (NAAQS) for pollutants considered harmful to both the public health and the environment. The EPA has set standards for six of the most common air pollutants called criteria pollutants, and they include ground level ozone, particulate matter, lead, Nitrogen dioxide (NO₂), Carbon monoxide (CO) and Sulphur dioxide (SO₂). (15) This standardized index known as Air Quality Index (AQI) uses concentrations of these criteria pollutants to calculate an index value from 0-500. Air quality is particularly important for subpopulations that are more susceptible (i.e. children, the elderly, subjects with cardiorespiratory diseases) or at a higher risk of specific exposures (occupational exposure). Values greater than 100 are generally considered unhealthy for sensitive groups and values greater than 150 are considered unhealthy for everyone.

(2) A prior study on Air Quality Index pattern around petroleum production facilities in the Niger Delta area of Nigeria, 2010 reported that AQI from measured Carbon monoxide in the study area ranged between 1 and 44 (between distances 50-500 metres of petroleum flow stations) an indication of good AQI, however at the 60 metre distance, AQI was 210 (unhealthy). (16) This report is not in keeping with findings from a comparative study of emission levels in Lagos and the Niger Delta area in 2000. (17)

Two major cities, Port Harcourt and Warri were considered for the Niger Delta Area. The results obtained showed that carbon monoxide (CO) levels in the selected Niger Delta communities were 5.0-61.0ppm and 1.0-52ppm; levels of other critical pollutants were above the Federal Environmental Protection Agency (FEPA) recommended limits. This finding was also in line with the findings of an earlier study in 1996 in a typical air quality assessment of the Niger Delta which showed that levels of volatile oxides of carbon, nitrogen and sulphur and total particulates exceed existing FEPA standard. (18) Despite these efforts, epidemiological studies continue to show associations between adverse health effects and air pollutants, even at concentrations near or below the current national standards. (15) Another study in Delta State(in the Niger Delta) revealed adverse effects of exposure to gas flaring in the study population as evidenced by a significant decline in lung function using peak expiratory flow rate measurement across all age groups. (19)

The use of asbestos based materials for roofing by residents of these gas flaring communities to repel acid-rain deterioration has its attendant risk as asbestos exposure is known to increase the risk of developing lung cancer, pleural and peritoneal mesothelioma and asbestosis. (20-23). Ibeno, host community to an oil producing company in Akwa Ibom State is located in the South-south zone of Nigeria, lies on the eastern side of the Qua Iboe River about 3kilometers from the River mouth and is one of the largest fishing settlements on the Nigerian coast. It occupies the largest Atlantic coastline of more than 129km in Akwa Ibom State, with a population of 74,840. (24) Residents of this community have been exposed to gas flares for over thirty years. The latest figure on gas flaring released by Nigeria National Petroleum Co-operation (NNPC) in its monthly publication "Petroleum Information Bulletin" reports that Mobil

which operates in the locality had flared 9.85 billion standard cubic feet of gas in January 2012, the highest in the industry. (25) The investigator is not aware of any scientific work documenting the effect of gas flaring on the lung health of the residents of this community.

Aim and Objectives

General Aim

To determine the effects of gas flaring on the lung health of Ibeno community residents.

Specific Objectives

The specific objectives of the study would include:-

1. To identify the socioeconomic characteristics of the respondents.
2. To compare the pattern of respiratory symptoms seen among Ibeno residents and those of a similar community devoid of gas flaring.
3. To compare the lung function indices (peak expiratory flow rate-PEFR, forced vital capacity-FVC, forced expiratory volume in one second-FEV1, FEV1/FVC) of Ibeno residents and those of controls from a similar community not exposed to gas flaring.
4. To determine the relationship between age, height, weight, body mass index (BMI), duration of residency and lung function indices in residents of both communities.

Justification of the Study

The World Health Organization (WHO) reports that air pollution is now the world's largest single environmental health risk, making breathing of clean air an important public health priority. A variety of air contaminants are emitted throughout the oil and gas development process, yet little is known about associated exposures and potential public health consequences particularly in developing countries. Lung function decline has been shown to occur long before the onset of respiratory symptoms. The ability to objectively measure pulmonary function provides a tool that can identify and quantify defects and abnormalities in function of the respiratory system, follow disease progression as well as monitor response to therapy. (26-27) The information from this study will hopefully encourage early detection of lung function impairment and impact positively policies regarding environmental pollution from gas flaring.

CHAPTER TWO

LITERATURE REVIEW

Historical Background

Attempts at lung function testing dates back as far as 129-200AD when Galen did a volumetric experiment on human ventilation. He had a boy breath in and out of a bladder and found that the volume of the gas was unchanged after sometime though he did no absolute measurement of lung volumes. (28) Several others made attempts at measuring lung volumes until John Hutchinson (a London surgeon) in 1844 invented a water spirometer with which he recorded the vital capacities of over 4000 persons. The vital capacity is measured by having the subject inhale maximally and then exhale slowly and completely. He showed the linear relationship between vital capacity and height and also showed that vital capacity does not relate with weight at any given height. (29) Various studies (30) tried to disprove Hutchinson's work by relating vital capacity to body weight, body surface area, stem length (sitting height) and chest circumference. (30) Hutchinson's data was re-examined for suspected errors thought to have accounting for Hutchinson's failure to find these relationships. These studies were eventually discredited by the mid-1920s. (31)

With increasing work being done on lung function, it became obvious that there was a racial difference in lung function. The first study of record to document a difference in lung function between blacks and whites was reported (32) over a century ago. Black Civil War soldiers were found to have a vital capacity about 11% less than their white counterparts. Further studies in 1927, (33) on 160 adult male prisoners showed that vital capacity was lower in blacks of all ages.

Spirometry as a Tool for Lung Function Assessment

Spirometers are user-friendly, available and can measure the rate at which the lung changes volume during forced breathing maneuvers (dynamic lung volumes). The most commonly performed test procedure uses the forced vital capacity (FVC) maneuver, in which the subject inhales maximally and then exhales as rapidly and completely as possible. The forced expiratory volume in one second (FEV_1) is the volume of air forcefully expired from full inspiration in the first second. It is usually 75% of FVC in healthy individuals. (34) The ratio of FEV_1/FVC gives information about the degree of airflow obstruction. A ratio of less than about 70-75% indicates airflow obstruction. This ratio is very useful because it is hardly affected by age, sex, height, ethnic origin, etc; it is self normalizing. (34)

Peak expiratory flow rate (PEFR) is the maximal expiratory flow rate sustained by a subject for at least 10 milliseconds expressed in litres per minute; it can be assessed using hand-held devices. (35)

Determinants of Lung Function

Correct interpretation of lung function requires an appreciation of normal values. There are tables and equations that are used to predict the normal values of the measurements in pulmonary function tests. The best values have been obtained from nonsmoking, normal subjects. The important prediction variables are the size, sex, and age of the subject. Therefore, the individual measures of FEV_1 and FVC do need corrections for the above factors and are usually quoted as % predicted. The range of normality is considerable and it may not be clear if results are simply at the bottom end of normal, or considerably reduced from the patient's normally much higher figures. Serial measurements indicating continuing deterioration may be the first clue. If the

FEV1/FVC ratio is normal, then the FVC can be interpreted as to whether there is a reduced total lung volume. This is known as a restrictive pattern. (34)

A variety of individual, behavioural and environmental factors affect lung function development in childhood and adolescence and the subsequent lung function decline with age. (36) Genetic factors may control body habitus and lung function development. It has been well documented (37) that different lung function values and reference values for lung function tests should not be applied across different ethnic groups without thorough evaluation. Exogenous factors such as smoking, nutrition, exercise, air quality and occupational exposure may affect both lung function development and decline.(38) Therefore, spirometry values need to be interpreted in the context of the patient, their symptoms, and other tests for the highest yield in diagnosis and management. (39)

Race

Race has been shown to be an important determinant of lung function. Various studies have reported higher values for Caucasians than Africans. (41-42) A difference of 10-15% in spirometric measurements has been established, and this difference is attributed to the variation in trunk to leg length ratio, which is lower in blacks compared to Caucasians. (42) Certain races, African American, African and Asian, for example, require race-specific values. (43) A study at Meharry Medical College, Tennessee, United States (44) contributed to the development of spirometric standards for healthy nonsmoking black adults. In 512 subjects ranging from 20 to 92 years, forced vital capacity was measured, as well as ratio of forced expiratory volume in one second to forced vital capacity expressed as percent and forced expiratory flow between 25 and 75 percent of forced vital capacity expired. Regression equations based on standing

height and age; along with prediction tables were also provided. Since sitting height (trunk length) was the same as one half the standing height among the subjects used, they discounted the effect of anthropometric differences. Another study (45) also noted a reduction in vital capacity, FEV₁ and total lung capacity in Nigerians compared with findings from American and British studies which they attributed to possible differences in body habitus.

Height

Size is best estimated with body height. The taller the subject, the larger the lung and its airways, and thus maximal flows are higher. Several studies (46-47) have shown a stronger positive correlation of height with various lung function tests, hence, height has been incorporated as a variable for prediction formulae and tables. It is recognized as one of the strongest predictors of lung function.

Sex

Studies have shown that at the same height and age, pulmonary function is higher in males than females. (46-47) This has been attributed to the fact that women have smaller lungs than men of a given height. Some authors have also argued that anthropometric measurements such as height and weight alone could not completely explain the differences in lung volumes in the sexes, and stressed that other factors such as differences in muscle strength, size and shape of thoracic cage may play a part. (45)

Age

Age has been shown to have a major influence on lung function. In children there is a positive correlation of age with lung function. (49) After the age of about 35 years, lung function begins to decline; lung elasticity is lost, and thus airways are smaller and flows lower. (50)

Lung function among Africans

The first work in literature in Africa was done by in 1968 using 220 healthy Bantu men and women (120 males and 100 females). (51) Total lung capacity and all lung compartments were found to be significantly lower than in whites. FEV1 were also significantly lower in the Bantu when compared to earlier studies done on whites. (52) The subjects however, were not representative of the general population as they were all hospital workers; either nurses or manual labourers.

In Nigeria, earlier works reported PEF values of 482.1L/min (+/-183.3) for males and 385.6L/min (+/-65.7) for females. (45) These values were significantly lower than that of Americans (35) which showed values of 578.5 L/min (+/-39.9) for males and 436.9L/min (+/-23.5) for females. The values were similar to those of normal Bantu men and women. (51) The major limitation in this study was the sample size selection which was not representative of the general population as it was mainly drawn from hospital workers or other white collar workers. It was however comparable with the existing literature of that time.

Air Pollution and Lung Function

Air pollution which can be indoor or outdoor is a major factor that can lead to derangements in lung function over time. Ambient air contains a range of pollutants, the exact combination of which varies from one microenvironment to the next. Many of the individual pollutants that make up the ambient mix are free radicals (for example, nitrogen dioxide which is present in gas flares) or have the ability to drive free radical reactions (for example ozone and particulates). As a consequence, exposure to a wide range of air pollutants give rise to oxidative stress within the lungs and this appears to

initiate responses that are dangerous to susceptible members of the population. Free radicals attack and oxidize other cell components such as lipids (particularly polyunsaturated lipids) proteins, and nucleic acids. This leads to tissue injury and in some cases, the influx of inflammatory cells to the site. (35) However, studies have also shown that there is a large variation between individuals in their response to air pollutants. This is thought to be related in part to their pulmonary antioxidant defenses.

(1) Previous studies have focused on toxic inhalational effects of biomass fuel which is well documented internationally and locally. A recent study in rural Nepal in 2013 reported a reduction in lung function due to biomass smoke exposure in young adults. (54) Ventilatory function (FEV₁, FVC and forced expiratory flow at 25-75% of FVC) was significantly reduced in the population using biomass across all age groups compared to the non-biomass-using population, even in the youngest (16-25 years) age group (mean FEV₁ (95% confidence interval) 2.65 (2.57-2.73) versus 2.83 (2.74-2.91) L; $\mu p=0.004$) Another study in Ibaka (a fishing settlement in South-south Nigeria) among adults chronically exposed (at least five years) to burning firewood for fish drying showed a higher prevalence of respiratory and other symptoms among the subjects than the controls. (55) These findings were corroborated by a recent study in Ile-Ife (56) which demonstrated the negative impact of pollution from different domestic energy sources using randomly selected residents from three communities according to the predominant type of fuel used for household cooking which were firewood, kerosene and liquefied petroleum gas (LPG). The investigators also assessed indoor particulate matter (PM)₁₀ levels by filtration using the Gent stacked filter unit sample for collection of atmospheric aerosol in two size fractions (PM_{2.5} and PM₁₀) in participants; a methodological issue that earlier local studies did not address. The mean

PM₁₀ concentration in participants using LPG, kerosene and firewood was 80.8 +/- 9.52 µg/m³, 236.9 +/- 26.5 µg /m³ and 269 +/- 93.7 µg/m³ respectively, showing that users of firewood had significantly lower FEV₁ and FVC compared with LPG users (p= 0.05). The study was limited by the small sample size (90 participants) which potentially limits the extent to which the findings can be generalized.

Gas Flaring and Lung Function

Lung diseases following occupational exposure among Nigerians have been extensively studied, (57-61) however, there is paucity of data on lung health of residents of gas flaring communities. A recent study in 2012 looked at the effects of gas flaring on lung function among residents in a gas flaring community in Delta State using peak expiratory flow rate (PEFR) measurements which showed a statistically significant reduction in PEFR across all age groups studied compared with age and sex matched controls obtained from a non-exposed population with similar socio-demographic factors. (19) Interestingly, the findings of another study (62) on the correlation between body mass index (BMI) and PEFR in an oil exploration environment in Bayelsa, South-south Nigeria in 2012 did not quite support this finding as the study results showed PEFR values for the overall population fell within the lower limits of normal ranges for the general normal adult population. It also showed a positive relationship between PEFR and BMI for the overall population (males and females). The reduction in PEFR was not as marked as those earlier reported considering that the participants had lived in the community for more than half their lives. The males also had higher BMI and PEFR values than their female counterparts; a finding the investigators thought could be related to genetic and environmental influences on eating and sedentary behaviours. The limitation of this study was inability to assess air pollution objectively using

appropriate instruments. Another possible explanation for the apparently normal PEFR could be the effect of land and sea breezes which could help to disperse air pollutants. The increasing temperature difference between the land and water creates a pressure minimum over the land due to its relative warmth and force high pressure, cooler air from the sea to move inland. In another study, 384 plant workers were interviewed in a chemical fertilizer industry at Onne in the Niger Delta, (70.5% spent eight hours per day at work), 66.1% reported respiratory disorders, 24.4% reported skin disorders and 22.6% reported eye disorders. There was a strong association between respiratory disorders and the industrial activities carried out by the workers, although it can be argued that the sources of air pollution in this location were multiple (industrial emissions, biomass combustion, traffic emissions, etc) and cannot be attributed to activities in the oil and gas industry alone. (63)

If the efficiency of the 'burn' during flaring is very high (>99%), the main products of combustion are carbon dioxide and water. However, rarely are such conditions met during the routine flaring of natural gas. Instead, the combustion efficiency typically ranges from 65 – 95%. (9) Under these conditions, carbon dioxide remains the major product of combustion; however, a number of minor chemicals also may be formed which are collectively referred to as 'products of incomplete combustion' (PICs). (9-10) These PICs can include unburned hydrocarbon, particulate matter (i.e. soot and ash), volatile organic compounds (e.g. benzene, toluene, ethylbenzene, and xylene – also known as BTEX compounds) polycyclic aromatic hydrocarbons as well as chemicals known as aldehydes and ketones.(11) If the natural gas is "sour" and contains hydrogen sulphide, some sulphur – containing chemicals e.g. sulphur dioxide may be formed during flaring, which contribute to the unpleasant odour associated with "sour" gas

flaring. Sour gas is heavier than air and tends to collect in low-lying areas. It is also corrosive and requires special handling in pipelines and processing facilities, where the sulphur is stripped out to turn it into “sweet” gas- natural gas that does not contain significant amounts of hydrogen sulphide. Long term studies on the health effects of sour gas are difficult. Most research works in developed countries have focused on cattle probably because gas flaring has been checked to a large extent in these climes. A study in Alberta, Canada found no measureable impact of plant emissions on the animal herd risk of mortality but could not rule out localized effects. (64) For several years public concerns have been expressed over perceived negative health effects relating to the oil and gas industry both locally and internationally. A more recent study in 2006 from the Western Interprovincial Scientific Studies Association, found no association between low-level exposure and animal health – with one exception. Calf death rates increased slightly, also the frequency of veterinary care, with exposure to both hydrogen sulphide and sulphur dioxide (65); none of the studies was definitive and conclusions were only valid for large herds.

Other pollutants that may be associated with oil and gas production include dust, ozone, particulate matter, natural gas, nitrogen oxides, carbon monoxide.

It has been documented that flares contain as many as 250 toxins; however, the exact chemical makeup of flaring emissions is not constant and is difficult to predict. (8)

Appendix 1 shows on-site characterization of chemical Emissions from the sweet gas flares. (66)

Appendix 2 also shows the Emissions in the downwind plume of a Waste gas Flare from a Sour Oilfield battery”. (66)

Some of these pollutants are described below:-

Btex Compounds: Otherwise known as volatile, monocyclic aromatic compounds, present in coal tar, petroleum products and various organic chemical product formulations. These are the most soluble of the major gasoline compounds. Exposure to these substances has been linked with impaired hearing, lung function and reproductive function. (11)

Carbon Monoxide: Colourless, odourless, flammable gas produced by incomplete burning of carbon-based fuels such as oil, natural gas, coal and even wood. It can cause dizziness, seizures, unconsciousness and even death by interfering with oxygen delivery which results in asphyxiation. (67-70)

Particulate Matter(PM) : This refers to a wide range of particles, organic and nonorganic, but predominantly the products of combustion processes that are traditionally subdivided into size categories for the purpose of research and regulation :- thoracic particles PM_{10} , coarse fraction $PM_{10-2.5}$, fine particles $PM_{2.5}$, ultrafine particles $PM_{0.1}$ ². The American Cancer Society study, documented increased cardiopulmonary mortality risk associated with fine PM and, in addition, demonstrated increased lung cancer risk. For specific disorders such as chronic obstructive pulmonary disease (COPD), others have argued for increased toxicity of coarse particles². A recent Chinese study further supports these findings; showing that long-term exposure to $PM_{2.5}$ increases the risk of mortality from lung cancer by 15-21% per $10\mu g/m^3$ increase. (71)

The United Nations and World Bank have assessed PM_{10} concentration as a surrogate for overall ambient air pollution in 1,100 cities from 91 countries. The highest concentrations of particulate matter are found in Africa and the Eastern Mediterranean Region where PM concentrations averaged over 90 and $140\mu g/m^3$, respectively. (72)

Natural Gas: This is released during venting operations or when there are leakages in equipment used during oil and gas development. The primary component of natural gas is methane, which is odourless when it comes directly out of the gas wells. (12)

Ozone: Ozone itself is not released during oil and gas development. However, some of the main compounds that combine to form ozone (e.g. volatile organic compounds and nitrogen oxides) are released from oil and gas operations. Ozone, when found at ground level can cause or aggravate respiratory ailments such as asthma following inhalation. (67) Because ozone has limited solubility in water, the upper respiratory tract is not very effective in getting rid of it. Consequently, the majority of inhaled ozone reaches the lower respiratory tract where it dissolves in the thin layer of epithelial lining fluid. It then reacts rapidly in the lung with a number of biomolecules ultimately leading to the formation of toxic free radicals. (53)

Hydrogen Sulphide: This is both a respiratory irritant and asphyxiant. It is a colourless, naturally occurring gas, known for its typical “rotten-egg” odour. It is heavier than air and therefore accumulates in low-lying areas, and can cause poisoning during oil drilling or following natural gas field leaks. (73-75) The hydrogen sulphide reaction with metalloenzymes such as cytochrome oxidase, accounts for much of its toxicity in humans. At concentrations of 50ppm, hydrogen sulphide is a mucous membrane irritant. Above 100ppm, the gas fatigues the sense of olfaction which makes individuals insensitive to its continued presence. The respiratory passages (nasal cavities and pharynx) conduct, warm and moisten air as it moves into the lungs. (20) It is protected by a well-developed lymphoid barrier and more superficially a mucous barrier. Any

level of the respiratory tract can be the target for toxins depending on the size of the particles, solubility and concentration of toxins as well as duration and frequency of exposure.

A variety of chemicals when liberated into the atmosphere as gases, fumes or mist can cause irritant lung injury or asphyxiation. The effects can be immediate:-pharyngitis, burning of the eyes, nose and throat, laryngotracheitis, acute asthma, acute bronchitis, or long term: -carcinoma of the bronchus and other cancers particularly skin. Studies done on firefighters and other rescue workers and volunteers following the collapse of World Trade Center on September 11th 2001 showed pulmonary function decline, reactive airways dysfunction syndrome (RADs), asthma reactive upper airways dysfunction syndrome (RUADS), sinus complaints, gastroesophageal reflux disease (GERD) and cases of inflammatory pulmonary parenchymal disease such as sarcoidosis. Specifically, among rescue workers with a high level of exposure, 8% experienced new onset cough, 95% had symptoms of dyspnoea, 87% had GERD, 54% had nasal congestion and 23% of the workers were identified as having bronchial hyperactivity at 6 months after the collapse.

Furthermore, 16% of the rescue workers met the diagnostic criteria for RADs one year after the collapse. In a longitudinal study of pulmonary function in rescue workers before and after exposure, the average adjusted FEV₁, fell 372mls during the year after the World Trade Centre collapse. (76-78)

Gas flaring, incidentally is being curbed in the developed world, unlike Nigeria which flares about 70% (about 2.7 billion cubic feet daily) from more than 100 flare sites spread across the oil rich Niger Delta area.

In 2002 six major oil companies joined the World Bank in creating the GGFR (Global Gas Flaring Reduction), a public-private partnership to encourage the reduction of flaring of natural gas associated with oil production in October, 2012 at the 10th anniversary of the GGFR Partnership in London a Russian Oil Company (Rosneft) received an award for its Associated Gas Recovery Project. The installation of a booster compression station by this company though expensive (costing 60 million US dollars) has significantly reduced gas flaring and impacted positively on the environment. It now reduces flaring of 1.3 million cubic meters of associated gas annually, translating into a reduction of one million tons of carbon yearly. Russia still tops the world's flaring countries followed by Nigeria, Iran and Iraq. (6, 11) The situation in Nigeria is not that encouraging, although some progress has been made in flaring reduction; it needs to be sustained.

It is important to have an understanding of the adverse impact of chronic exposure from multiple flaring discharges on the health of people who live and work in proximity to the industry. Proximity has been defined as any distance between 0.2 – 35km from the flare stack, (79) although in the Alberta study, a distance of 70 km was used as the reference; this was empirically determined to be the distance at which sulphur dioxide concentrations approached zero under the Gaussian atmospheric dispersion model. (64) Some investigators have argued that the emissions design(example stack heights, diameter, exit speeds and temperature) of flare stacks makes its pollutant effect to be felt in greater dimension farther away from the actual flare site depending on the wind speed, stability and direction. (64)

The adverse effects of gas flaring on animals was also documented in a study that conducted interviews with animal owners in six states in the United States (Colorado,

Louisiana, New York, Ohio, Pennsylvania and Texas) affected by gas drilling. They also interviewed the owners' veterinarians and examined the results of water, soil and air testing as well as the laboratory tests on affected animals and their owners, thus highlighting the possible links between gas drilling and negative health effects. (80) In another study, the human health risk assessment of air emissions from development of unconventional natural gas resources was also examined in a population in Colorado. Their findings demonstrated that residents living less than or equal to half a mile (about 804 meters or 0.804 km) from wells are at greater risk for health effects from natural gas development than residents living more than half a mile away. (81)

Despite extensive studies on lung diseases following environmental exposure, there is paucity of data on lung health of gas flaring community residents, particularly in Ibeno. This study will provide information on the effect of exposure to gas flaring on the lung function of adult residents in this community.

It is hoped that this study will also help unfold the respiratory challenges faced by residents of Ibeno community and improve awareness of practicing physicians in the community and state at large on the epidemiology of respiratory conditions other than chest infections that are not regularly seen in the outpatient clinics. The findings of this study will hopefully help expedite the required interventions necessary to reduce the incidence and complications associated with exposure to gas flaring among the study population and in the Niger Delta generally.

CHAPTER THREE

MATERIALS AND METHODS

Study Design

This was a comparative cross-sectional study.

Study Area

This study was carried out in Ibeno community (Ibeno LGA), a riverine community in Akwa Ibom State. It is bounded to the West by Eastern Obolo LGA, to the North by Onna, Esit Eket and Eket LGAs and to the south by the Atlantic Ocean. It has a population of 74,840 inhabitants. (24) Ibeno occupies the largest coastline of more than 129km in Akwa Ibom State located in the mangrove swamp forest; much of the area is not habitable.

The prime occupation of the people is fishing; however petty trading and minimal farming are also carried out by the people. The women are actively involved in fish smoking. For the purpose of this study, three village were selected by multistage sampling :- Mkpanak, Inua Eket and Upenekang.

Controls were selected from three villages also selected by multistage sampling from Etinan :- Ikot Abasi, Ekom Iman and Etinan. The inhabitants of these communities are mainly farmers involved in oil palm processing and cassava cultivation.

Population of the Study

Adult males and females who met the inclusion criteria were recruited for the study.

Inclusion Criteria

1. Age above 18 years
2. Individuals should have been resident in the study community for at least two years. This position was based on qualitative research methodology using focus group discussion and in-depth interview with key informants, who included landlords that have had experiences with new comers to the community. Based on an outcome of the qualitative research, it was observed that new comers to the community usually start complaining of respiratory symptoms within two years of residency, and the research team so adopted two years as a criterion. However, the investigator did not come across any supportive literature to strengthen this view.

Exclusion Criteria

1. Age less than 18 years
2. Pregnancy
3. Individuals who have not been resident in the study community for up to two years
4. History of cigarette smoking
5. History of heart disease
6. Individuals with respiratory disease
7. Individuals with thoracic cage abnormalities such as kyphoscoliosis
8. Subjects with history suggestive of active tuberculosis
9. Individuals with severe uncontrolled hypertension(BP>180/100mmHg)
10. Individuals who refuse to consent

Sampling Procedures and Sample Size

The subjects were all adults living in Ibeno community for at least two years. The control group was also comprised of adults resident in a community not exposed to gas flaring (Etinan) estimated to be at least 100km from the flare stack. Multistage sampling technique was adopted for the study. Ibeno local government is comprised of ten wards. Three villages were selected by simple random sampling from this stratum, while the participants from each village were also stratified into family units and selected by simple random sampling using the ballot method. To ensure that the participants sampled were representative of the sample population, the number of subjects selected from each family unit was relatively proportional to the total population of the settlements. This sampling technique was replicated in selecting the control group which comprised of similar age, height and gender - matched individuals resident in Etinan for at least two years. This was achieved with the co-operation of the village heads and elders who informed the villagers about the study and reminded them on the scheduled days using the town crier. A total of 396 subjects and 414 controls who met the inclusion, criteria had their lung function (PEFR, FVC and FEV₁) tested.

Sample Size Estimation

Sample size was determined using the formula for sample size estimation in an infinite population for comparing means below (82):-

$$N = (u + v)^2 \times 2 \times (SD)^2 / (m_1 - m_2)^2$$

Where:

N = The minimum desired sample size

u = one sided percentage of the normal distribution, corresponding to 100% the power.

The power is the probability of finding significant result. Power of 90% = 1.28

v =Percentage part of the normal distribution corresponding to the (two sided) significance level. If significance level is 5%, $v = 1.96$

SD= Standard deviation of the primary outcome variable which is 25.1 (60)

m_1, m_2 = Means ($m_1=112.1, m_2=77.8$). (60)

Applying the formula above, the desired sample size is:-

$$N = \frac{(1.28 + 1.96)^2 \times 2 \times (25.1)^2}{112.1 - 77.8}$$

$$\text{Minimum sample size} = (3.24)^2 \times 2 \times 630.01/34.3 = 10.5 \times 1260.02/34.3 = 385$$

Minimum sample size is 385.

Instruments for Data Collection

These consisted of a questionnaire (interviewer administered), a Hanson bathroom weighing scale model H89 RED manufactured by Hanson fitness products United Kingdom, spirometer (Spirolab III, medical international research (MIR)), and a calibrated ruler. The choice of the above height and weight scales were convenient since the study involved travelling to remote areas and the instruments could be easily moved from one location to another. The prohibitive cost of more sophisticated scales also encouraged their choice. Please see appendix IV to view the interviewer administered questionnaire (which seeks to evaluate for respiratory symptoms; it contains some items adopted from the Medical Research Council chronic respiratory questionnaire). (830)

The items used were validated by the supervisors and also the statistician. The socio-demographic data of the participants and medical history were obtained focusing on history suggestive of evidence of current respiratory tract infection or symptoms of respiratory disorder as well as past history of chronic pulmonary diseases. Family history of asthma was also enquired for. The investigator was available to respond to

any enquiries by participants during the completion of the questionnaires. The questionnaire was translated from English into Ibibio language by the investigator with the help of a linguist and retranslated back to ensure standardization. Four research assistants (including some health workers in the community health centre to gain the trust of the people) were trained for a day by the investigator on the administration of the questionnaire and on proper anthropometry. Weight was measured in kilograms using a portable bathroom weighing scale; height was measured using a calibrated ruler and Body Mass Index (BMI) was calculated as weight in kilograms (kg) divided by the square of the height in metres (i.e. kg/m^2).

Physical Examination

A general physical examination and a thorough clinical examination of the cardiopulmonary system were performed by the investigator in a sequestered area provided by the community leaders to exclude any significant disease that would influence lung function.

Weight

The weight was taken using a Hanson bathroom scale model H89 RED with the individual standing erect, in light clothing, without shoes and all head gears removed. Weight was read with precautions against errors due to parallax by reading at right angle to the pointer. The scale's pointer was adjusted to the zero point before commencement of each measurement. The accuracy was to the nearest 0.5kg (kg)

Height

The standing height (centimeters) without shoes was taken against the calibrated ruler. The subject stood erect against the ruler and was positioned such that the feet were placed together on the floor with the heels, buttocks and occiput touching the ruler. The

height was the distance from the floor to the ruler at the level of the vertex. This was read off against the calibrated ruler (using a smaller ruler placed horizontally on the vertex) to the nearest centimeter (cm).

Lung Function Test

Lung function was assessed using spirometric measures of lung function capacities specifically forced vital capacity (FVC), defined as the volume of air in litres that can be forcefully and maximally exhaled. Forced expiratory volume in 1 second (FEV₁) defined as the volume of air (in litres) that can be forcefully exhaled in 1 second. (34, 84) Ratios of FEV₁ to FVC (FEV₁/FVC) were subsequently calculated to assess the pattern of lung function impairment if present. Peak expiratory flow rate was measured by a pneumotacometer in the spirometer. Unlike older machines that need to be calibrated daily, on the day of use and/or whenever relocated, Spirolab III does not require calibration and has an internal temperature sensor for automatic temperature conversion.

The measurements were performed according to the methods recommended by the American Thoracic Society. (85, 86) Each participant performed at least three forced expiratory manoeuvres (maximum of eight) while sitting with the nostrils closed with a nose clip to prevent leakage of air through the nose, to ensure reproducibility of the results. The patient's best was taken. Spirograms were accepted if they (1) did not show artifacts such as cough or glottis closure during the first second of exhalation, early termination, variable effort, leakage, and obstructed mouthpiece (tongue or dentures); (2) had good starts with back-extrapolated volume not exceeding 5% of FVC or 150mL (whichever was larger), (3) had satisfactory exhalation length (at least 6 seconds and/or

a plateau in the volume/time curve); and (4) spirometric measurements were considered reproducible if the best and second-best FVC or FEV₁ measurements were within 200mL of each other.

Data Analysis Techniques

The specific objectives were analyzed using a combination of descriptive statistical tools such as means, frequency counts and percentages. Statistical Package for Social Sciences (SPSS) version 17 computer software was used. A p-value of <0.05 was considered significant, 95% confidence interval was also reported. Prediction equations for FEV₁ and FVC derived by linear regression analysis using healthy Nigerian adults was used to determine the predicted values for lung function indices of the participants to ensure proper interpretation of results. (87, 88) The hypothesized relationships were analyzed using multiple regression.

Consent/ Ethical considerations

Ethical clearance was obtained from the University of Uyo Teaching Hospital before embarking on the study (Appendix VII). Approval was obtained from Ibeno local government council and the community leaders were also informed on the details of the study and verbal consent obtained. An informed consent was also obtained from the participants. (Appendix III)

CHAPTER FOUR

RESULTS

4.1 Socioeconomic Characteristics of the Respondents

Based on result of the analysis as shown on Table 1, item 1 shows the sex distribution of both the subjects and control group of the respondents. The subjects comprised 226 females (58.5%) and 160 males (41.5%); while 215 (55.7 %) of the controls were

females and 171(44.3%) were males giving a female: male ratio of 1: 1.4 and 1: 1.3 for subjects and controls respectively. With regard to distribution of the respondents in terms of BMI, majority of the respondents were those within the normal BMI however the exposed subject had more over weight and obese individuals than the control group (33.2% and 16.1% vs.23.8% and 14.5%), while the control group had more underweight individuals than the exposed group (13.7% vs.3.1%). BMI was categorized according to the World Health Organization (WHO) classification in 1997(published 2000).With reference to item 3, most of the respondents among both subjects and controls were in the 18 – 30 year age bracket. With regard to item 4, majority of the respondents in both groups were within the 171-180 cm. Table 1, item 6 shows that most of the respondents had formal education (at least primary level) while few had no formal education among both controls and subjects. According to item 7, three villages each from the exposed and control groups were used for the study. With regards to item 8, most of the respondents among both controls and subjects were born and raised in the community and had spent not less than 30 years.

Table 1: Distribution of Respondents based on Socioeconomic Characteristics

Item	Variable	Subject	Control	Total
1	Sex			
	Female	226(58.5)	215(55.7)	441(57.1)
	Male	160(41.5)	171(44.3)	331(42.9)
	Total	386(100.0)	386(100.0)	772(100.0)
2	BMI			
	Under Weight(<18.5kg/m ²)	12(3.1)	53(13.7)	65(8.4)
	Normal(18.5-24.9kg/m ²)	184(47.7)	185(47.9)	369(47.8)
	Overweight(25-29.9kg/m ²)	128(33.2)	92(23.8)	220(28.5)
	Obesity(≥30kg/m ²)	62(16.1)	56(14.5)	118(15.3)
	Total	386(100.0)	386(100.0)	772(100.0)
3	Age (in years)			

	18-31	153(39.6)	122(31.6)	275(35.6)
	32-44	110(28.5)	118(30.6)	228(29.5)
	45-57	76(19.7)	108(28.0)	184(23.8)
	58-70	38(9.8)	28(7.3)	66(8.5)
	71-83	9(2.3)	10(2.6)	19(2.5)
	Total	386(100.0)	386(100.0)	772(100.0)
4	Height (in cm)			
	140-150	34(8.8)	35(9.1)	69(8.9)
	151-160	137(35.5)	123(31.9)	260(33.7)
	161-170	158(40.9)	133(34.5)	291(37.7)
	171-180	49(12.7)	87(22.5)	136(17.6)
	181-190	8(2.1)	8(2.1)	16(2.1)
	Total	386(100.0)	386(100.0)	772(100.0)
5	Education			
	Nil	13(3.4)	11(2.8)	24(3.1)
	Primary	177(45.9)	112(29.0)	289(37.4)
	Secondary	151(39.1)	176(45.6)	327(42.4)
	Tertiary	45(11.7)	87(22.5)	132(17.1)
	Total	386(100.0)	386(100.0)	772(100.0)
6	Years of Residency			
	2-29 years	220(57.0)	180(46.6)	400(51.8)
	30-56 years	127(32.9)	172(44.6)	299(38.7)
	57-83 years	39(10.1)	34(8.8)	73(9.5)
	Total	386(100.0)	386(100.0)	772(100.0)

Source: field survey 2014

BMI categories established by WHO in 1997

4.2 The Pattern of Respiratory Symptoms among Ibeno Residents and Those of Similar Community Devoid Of Gas Flaring

The study (Table 2) also showed that more of the exposed subjects had symptoms of cough, chest pain, breathlessness and wheezing (14.8, 9.6, 15.0 and 5.7 respectively) compared with the controls (10.1, 1.6, 7.3 and 3.1 respectively). More of the exposed subjects also chronic symptoms of cough (lasting more than two months) and chest pain.

Interestingly, more of the subjects had a family history of asthma (2.6) compared with the controls (1.0) as shown on table 2, items 1-6.

Table 2: Distribution of Respondents according to Pattern of Respiratory Symptoms among Ibeno Residents and those of Similar Community Devoid Of Gas Flaring

Item	Variable	Subject	Control	Total
1	Cough			
	Absent	329(85.2)	347(89.9)	676(87.6)
	Present	57(14.8)	39(10.1)	96(12.4)
2	Cough Duration			
	Absent	329(85.2)	347 (89.9)	676(87.6)
	Acute	39(10.1)	30(7.8)	69(8.9)
	Chronic	18(4.7)	9(2.3)	27(3.5)
3	Chest pain			
	Absent	349(90.4)	380(98.4)	729(94.4)
	Present	37(9.6)	6(1.6)	43(5.6)
4	Chest pain duration			
	Absent	350(90.7)	380(98.4)	730(94.6)
	1 month	17(4.4)	5(1.3)	22(2.8)
	≥6 months	19(4.9)	1(0.3)	20(2.6)
5	Breathlessness			
	Absent	328(85.0)	358(92.7)	686(88.9)
	Present	58(15.0)	28(7.3)	86(11.1)
6	Wheezing			
	Absent	364(94.3)	374(96.9)	738(95.6)
	Present	22(5.7)	12(3.1)	34(4.4)
7	Asthma			
	Absent	376(97.4)	382(99.0)	758(98.2)
	Present	10(2.6)	4(1.0)	14(1.8)

Source: field survey 2014

4.3 Comparative Assessment of Lung Function Indices of Residents and Non-Residents of Ibeno Community

The independent T-test was used to compare the mean of the ventilatory parameters of the subjects and controls (Table 3). There was a statistically significant difference in PEFR among subjects and controls (p-value= 0.000). Mean FEV₁ was also significantly lower between the exposed subjects (2.01± 0.76) compared with that of the controls (2.13±0.75), p=0.027. Both obstructive and restrictive pattern of ventilatory defects

were seen more among respondents from the exposed community, however the difference was not statistically significant.

i. Comparative Analysis of lung function index of Residents and Non-Residents of Ibeno Community

Table 3a: Distribution of Respondents based on Comparative Analysis of lung function index of Residents and Non-Residents of Ibeno Community

Item	Lung Function Indices	Mean of Exposed	Mean of Control	T-test value	Sig. (2-tailed)	Remark
1	PEFR (l/min)	300.6 ± 2.15	342± 2.16	4.446	0.000	Significant
2	FVC (l)	2.58 ± 8.43	2.27 ± 0.82	-0.721	0.471	Not Significant
3	FVC% p	88.25±302.43	75.84±23.64	-0.697	0.422	Not Significant
4	FEV ₁ (l)	2.01±0.76	2.13±0.75	2.221	0.027	Significant
6	FEV ₁ _FVC	93.64 ± 11.89	94.21 ± 8.39	0.763	0.446	Not Significant
7	FEV ₃ (l)	2.37 ± 5.05	2.36 ± 2.80	-0.006	0.995	Not Significant
8	FEV ₃ _FVC	100.34 ± 45.58	98.36 ± 7.12	-0.844	0.399	Not Significant
9	FEF _{25_75}	3.18 ± 1.47	3.23 ± 1.23	0.531	0.595	Not Significant

Source: field survey 2014

FVC% p= FVC percentage predicted,

Df=770, Sig. (2-tailed) ≤ P value=0.05 is significant

Table 3b: Distribution of Respondents based on Comparative Analysis of Ventilatory defects of Residents and Non- Residents of Ibeno Community

Functional classification	Exposed			Control			Pooled Total
	Male	Female	Total	Male	Female	Total	
Normal	66	52	118	70	68	138	256
Restrictive	89	161	250	98	140	238	488
Obstructive	2	6	8	2	5	7	15
Mixed pattern	3	7	10	1	2	3	13

Source: field survey 2014

4.4 Effect of Gas Flaring on the Lung Function Indices of the Residents of the study Community.

The primary focus of this subtheme was to evaluate the hypothesized conceptualization that there could be effects of gas flaring on the lung function indices of the respondents. A multivariate statistical modeling was required to understand the relative complexity of diverse sources of factors that can influence the indices of lung function in an individual as he or she interacts daily with natural and man-made adverse drivers of respiratory function in any particular environment. Therefore, an econometric modeling technique was adopted to simulate the influence of the air quality which informed the selection of study areas as dummy variable. The prominent weight of 1 was assigned to the exposed communities to gas flaring sites while 0 was assigned to the communities that served as control. This technique was inevitable so as to provide the needed statistically processed information in terms of its significance and the relative coefficient value as the effect of the gas flaring on the lung function indices.

I. Relationship between Gas flaring and PEFR

With regard to the information on the summary of the model and diagnostic statistics, it shows that correlation coefficient (R) is 0.629, meaning that the interactions between the dependent and independent variables was good and the sig. (P-value) = 0.000 implies that the model estimation was statistically significant at 99% probability level. The tests of significant relationship between the predictors and dependent variable showed that the relationship was statistically significant at $P < 0.05$ (95%). The value of coefficient of determination (R^2) showed the proportion of the total variation in the dependent variable Y that was explained by the independent variables (X_1 - X_6). This implies that the independent variables were able to explain the variability in the respondents' performance of PEFR by 39.5 %. Although the result shows that all the variables (X_1 to X_6) have a significant relationship with PEFR, the standardized coefficient indicated the relative magnitude of effects of predictor variables on the PEFR ability of the respondents. Across the six predictor variables, sex of the respondents had relatively the highest influence on the PEFR index of lung and was followed by age and thirdly by the status of exposure to site of gas flaring. Statistically, the interaction between the status of exposure to gas flaring shows a significant negative relationship with the PEFR. This suggests that the exposed had relatively lower PEFR than the control factor.

The predicted equation is presented as follows

$$\ln \text{PEFR} = 1.088 - 0.008 (\text{age}) - 0.002 (\text{Duration of residency}) + 0.005 (\text{Weight}) + 0.005 (\text{Height}) - 0.356 (\text{sex}) - 0.176(\text{Gas flaring exposure status}) + e$$

Table 4: Shows the predicted function of PEFR and its determinants

Predictors Variables	Unstandardized Coefficients		Standardized Coefficients		
	B	Std. Error	Beta	T	Sig.
(Constant)	0.912	0.334		2.728	0.007
Age	-0.008	0.001	-0.252	-6.919	0.000
Duration of residency	-0.002	0.001	-0.088	-2.432	0.015
Weight	0.005	0.001	0.144	4.650	0.000
Height	0.005	0.002	0.100	2.635	0.009
Sex	-0.356	0.033	-0.369	-10.690	0.000
Gas flaring exposure status	-0.176	0.027	0.184	6.426	0.000

a. Dependent Variable: lnPEFR

Sig.= .000

Source: computed based on data from field survey, 2014

II. Relationship between Gas flaring and FVC

With regard to the information on the summary of the model and diagnostic statistics shows that correlation coefficient (R) is 0.655, meaning that the interactions between the dependent and independent variables was good and the sig. (P-value) = 0.000 implies that the model estimation was statistically significant at 99% probability level. The tests of significant relationship between the predictors and dependent variable showed that the relationship was statistically significant at $P < 0.01$ (99%). The value of coefficient of determination (R^2) implies that the independent variables were able to explain the variability in the respondents' performance of FVC by 42.9%. Although the result shows that all the variables (X_1 to X_6) have a significant relationship with FVC, the standardized coefficient indicated the relative magnitude of effects of predictor variables on the FVC ability of the respondents. Across the six predictor variables, a similar pattern of relative influence of the predictors variables were found on sex and age but the effect of exposure to gas flaring on FVC statistically appeared the least

among determinants within the mix. Statistically, the interaction between the status of exposure to gas flaring showed a negative relationship with the FVC and it was statistically significant.

The predicted equation is presented as follows

$$\ln FVC = -0.858 - 0.008 (\text{age}) - 0.003 (\text{Duration of residency}) + 0.004 (\text{Weight}) + 0.012 (\text{Height}) - 0.246 (\text{sex}) - 0.066 (\text{Gas flaring exposure status}) + e$$

Table 5: Shows the Predicted Function of FVC and Its Determinants

Predictors Variables	Unstandardized Coefficients		Standardized Coefficients		
	B	Std. Error	Beta	T	Sig.
(Constant)	-0.924	0.296		-3.120	0.002
Age	-0.008	0.001	-0.251	-7.074	0.000
Duration of residency	-0.003	0.001	-0.113	-3.195	0.001
Weight	0.004	0.001	0.127	4.192	0.000
Height	0.012	0.002	0.239	6.451	0.000
Sex	-0.246	0.030	-0.279	-8.339	0.000
Gas flaring exposure status	-0.066	0.024	0.075	2.696	0.007

a. Dependent Variable: lnFVC

Source: computed based on data from field survey, 2014

III. Relationship between Gas flaring and FEV₁

With regard to the information on the summary of the model and diagnostic statistics shows that correlation coefficient (R) is 0.712, meaning that the interactions between the dependent and independent variables was sufficiently good and the sig. (p-value) = 0.000 implies that the model estimation was statistically significant at 99% probability level. The tests of significant relationship between the predictors and dependent variable showed that the relationship was statistically significant at $p < 0.01$ (99%). The value of coefficient of determination (R^2) implies that the independent variables were able to explain the variability in the respondents' performance of FEV₁ by 50.7%. Similar pattern of FVC result was also observed on the result of FEV₁; all the variables have a significant relationship with FVC, the standardized coefficient indicated the relative magnitude of effects of predictor variables on the FEV₁ capacity of the respondents. Statistically, the interaction between the status of exposure to gas flaring showed a negative relationship with the FEV₁ and that relationship was statistically significant.

. The predicted equation is presented as follows

$$\ln\text{FEV}_1 = -0.083 - 0.009 (\text{age}) - 0.002 (\text{Duration of residency}) + 0.003 (\text{Weight}) + 0.012 (\text{Height}) - 0.264 (\text{sex}) - 0.264 (\text{Gas flaring exposure status}) + e$$

Table 6 : Showing multiple regression result on the Predicted Function of FEV₁ and Its Determinants

Predictors Variables	Unstandardized Coefficients		Standardized Coefficients		
	B	Std. Error	Beta	T	Sig.
(Constant)	-1.010	0.264		-3.827	0.000
Age	-0.009	0.001	-0.309	-9.375	0.000
Duration of residency	-0.002	0.001	-0.085	-2.576	0.010
Weight	0.003	0.001	0.120	4.261	0.000
Height	0.012	0.002	0.258	7.499	0.000
Sex	-0.264	0.026	-0.312	-10.021	0.000
Gas flaring exposure status	-0.083	0.022	0.099	3.828	0.000

a. Dependent Variable: lnFEV₁

Source: computed based on data from field survey, 2014.

4.7 Relative effects of Biodata on the Lung function Indices in the Exposed and Non-Exposed Communities

I: Relationship between PEFr and its Determinants in both Exposed and Non-exposed Communities

This subsection attempts to comparatively analyze the effect of sex, age, height, weight and duration of residency on the lung functions of the residents in both control and the communities with regard to Table 7. For the exposed communities, all the biodata variables had significant relationships with PEFr, but the situation within the control communities was different, where age and sex of the residents displayed a statistically significant relationship with PEFr. Between the two communities their model statistically were relatively adequate as their R value was high and above 0.600.

The predicted equation is presented as follows

$$\text{PEFR} = 1.353 - 0.317 (\text{age}) - 0.053 (\text{Duration of residency}) + 0.180 (\text{Weight}) + 0.080 (\text{Height}) - 0.388 (\text{sex}) + e$$

Table 7: Showing multiple regression result on the Relationship between PEFr and its Determinants

Predictors Variables	Standardized Coefficients			
	Control		Exposed	
	Beta	Std Error	Beta	Std Error
Age	-.317***	.001	-.214***	.002
Duration of residency	-.053	.001	-.105**	.001
Weight	.180***	.001	.125***	.002
Height	.080	.002	.127**	.004
Sex	-.388***	.033	-.358***	.054
(Constant)	1.353***	.403	.526	.561

a. Dependent Variable: ln PEFr

Source: Computed based on Data from field survey, 2014

Note: ***= statistically significance at 99% probability level

**= statistically significance at 95% probability level

II. Relationship between FVC and its Determinants

This subsection attempts to comparatively analyze the effects of sex, age, height, weight and duration of residency on the lung functions of the residents in both control and the communities with regard to Table 8. For the control communities, all the biodata variables had significant relationships with FVC, while age and sex of the residents displayed a statistically significant relationship with FVC in the exposed group.

The predicted equation is presented as follows

$$\text{FVC} = -0.120 - 0.336 (\text{age}) - 0.134 (\text{Duration of residency}) + 0.168 (\text{Weight}) + 0.182 (\text{Height}) - 0.362 (\text{sex}) + e$$

Table 8: Showing multiple regression result on the Relationship between FVC and its Determinants

Predictors Variables	Standardized Coefficients			
	Control		Exposed	
	Beta	Std Error	Beta	Std Error
Age	-0.336***	0.001	-0.189***	0.002
Duration of residency	-0.134***	0.001	-0.093***	0.001
Weight	0.168***	0.001	0.080	0.002
Height	0.182***	0.002	0.304	0.003
Sex	-0.362***	0.033	-0.213***	0.050
(Constant)	-0.120	0.317	-1.857***	0.529

a. Dependent Variable: lnFVC

Source: Computed based on Data from field survey, 2014

Note: ***= statistically significance at 99% probability level

****= statistically significance at 95% probability level**

III. Relationship between FEV₁ and its Determinants

This subsection attempts to comparatively analyze the effect of age, sex, height, weight and duration of residency on the lung functions of the residents in both control and the communities with regards to Table 9. For the control communities, all the biodata variables had significant relationships with FEV₁, while age and sex of the residents displayed a statistically significant relationship with FEV₁.

The predicted equation is presented as follows

$$FVC = -0.182 - 0.368 (\text{age}) - 0.137 (\text{Duration of residency}) + 0.119 (\text{Weight}) + 0.199 (\text{Height}) - 0.374 (\text{sex}) + e$$

Table 9: Showing multiple regression result on the Relationship between FEV₁ and its Determinants

Predictors Variables	Standardized Coefficients			
	Control		Exposed	
	Beta	Std Error	Beta	Std Error
Age	-.368***	.001	-.251***	.001
Duration of residency	-.137***	.001	-.045	.001
Weight	.119***	.001	.111***	.001
Height	.199***	.002	.317***	.003
Sex	-.374***	.032	-.270***	.043
(Constant)	-0.182	.309	-1.853***	.447

a. Dependent Variable: lnFEV₁

Source: Computed based on Data from field survey, 2014

Note: *= statistically significance at 99% probability level**

****= statistically significance at 95% probability level**

CHAPTER FIVE

DISCUSSION

5.1 Socioeconomic Characteristics of the Respondents

The study has shown that the mean age, height and sex distribution of both subjects and controls were comparable. However, the subjects were observed to have more overweight and obese individuals than the controls 33.2% and 16.1% vs. 23.8% and 14.5% respectively. There were also more underweight individuals in the control group than the exposed group (13.7% vs. 3.1%). This could be as result of improved nutrition as the exposed subjects have increased access to sea foods and unlike the controls are not involved in strenuous farming activities. Most of the exposed subjects and controls had at least primary education and very few had no formal education, this was not surprising as both communities had long standing educational institutions. Most participants among both exposed subjects and controls had spent most of their lives in the community, a few were married from other locations and some others were transferred from the workplace. The respondents were selected from three villages each from both the exposed and control communities. The villages selected for Ibeno community were Inua eyet Ikot, which is estimated to be about 25 km from the flare stack, Mkpanak (about 30km from the flare stack) and Upenekang which is about 40 km from the flare stack, while those selected from Etinan community were Ekom Iman (about 120km from the flare stack), Ikot Abasi (about 100km from the flare stack) and Etinan which is about 90km from the flare stack. Most participants among both exposed subjects and controls were born and raised in the community and had spent most of their lives in the community, a few were married from other locations and some others were transferred from the workplace. This suggests that they were chronically exposed to noxious air pollutants from childhood. In this scenario, even relatively small exposures

could have a cumulative effect. Therefore, this could have a negative impact on lung growth and development, as well as accelerate the physiologic lung function decline with age.

5.2 Pattern of Respiratory Symptoms of the Respondents

The symptoms indicative of respiratory disorder were similar in both exposed subjects and controls, though a higher prevalence was noted among the exposed subjects when compared with the controls: cough (14.8% vs.10.1%), chest pain (9.6% vs. 1.6%), and breathlessness (15% vs.7.3%), and wheezing (5.7% vs.3.1%). The most prevalent respiratory symptoms were breathlessness and cough. There were also more respondents with a positive family history of asthma among the exposed subjects than controls (2.6% vs.1%). This finding suggests that the environment in the exposed community impacted more negatively on the lung health of the residents than that of the control community. Although cough chest pain, breathlessness and wheezing can occur in other conditions other than respiratory, an attempt was made to exclude participants with history or examination features suggestive of heart disease.

5.3 Comparative Assessment of Lung Function Indices among Respondents

This study showed that the PEF_R and FEV₁ of the exposed subjects were significantly lower than that of the controls. This finding was in keeping with the findings of Ovuakporaye in a similar study done in the Niger Delta region, which noted a significant decrease in PEF_R across all age groups in the study population.¹⁹ This could be related to the negative effect of exposure to air pollutants, most likely from gas flaring. Although some studies have previously showed a decline in PEF_R with increasing BMI, the current study did not demonstrate a statistically significant association between both parameters. There was no statistically significant difference

between FVC values among both exposed subjects and controls; this may not be unrelated to the negative impact of chronic exposure to biomass fuels in both exposed and control communities. Lung function decline worsened with increasing age; the greatest decline was noted among the age group 58-70years and 71-83 years, which suggests increased vulnerability to lung damage with age. Both obstructive and restrictive ventilatory defects were seen among respondents from exposed and non-exposed communities (mostly among females), but the difference was not statistically significant. Air pollution accelerates the already existing physiologic lung function decline with age as a result of decreasing lung elasticity. This is further complicated by the fact that most of the exposed population were born and raised in the same community and have probably suffered from the deleterious effects of air pollution right from childhood during lung growth and development.

5.4 Effect of BMI on lung function of Residents of Ibeno Community

The results of this study had shown that there was a significant variation in the distribution of respondents across different BMI categories (underweight, normal BMI, overweight and obese) with respondents from the exposed community having more overweight and obese individuals than the controls who equally had more underweight respondents. Although this finding could possibly introduce bias as some studies have shown a negative relationship between increasing BMI and lung function indices; particularly PEF_R(89); the current study showed in a result of a one way analysis of variance which was conducted for different BMI categories: normal weight, overweight and obese that, the mean group comparison of PEF_R, FEV₁, FEV₁ /FVC and FEF₂₅₋₇₅ for the underweight category was significantly different from all other BMI categories but other BMI categories (normal, overweight and obesity) were not significantly

different from each other in the study area. Therefore, this implies that although there was a significant difference in the PEF_R, FEV₁ and FEV₁/FVC performance based on BMI categories, respondents who were overweight or obese did not differ in the performance of their lung function indices. This finding can also be explained by the fact that most of the study participants among both exposed subjects and controls had normal BMI (47.7% vs. 47.9%) rendering the negative effect of increasing BMI statistically insignificant. Several other studies have examined the association between body mass index (BMI in kg/m²) or weight change and pulmonary function testing variables, and the associations vary in different subpopulations. (90-93) A Canadian cross-sectional survey of 738 men and 936 women aged 18-79 years by in 2007 which determined the predictability of waist circumference and BMI for pulmonary function in adults with or without excess body weight showed that BMI was positively associated with FVC and FEV₁; while the association between waist circumference and pulmonary function was consistently negative in subjects with normal weight, overweight and obesity. (94) This suggests that BMI is not consistently negatively associated with pulmonary function in normal weight, overweight and obese subjects. These findings were contrary to the findings of another Canadian study (95) in 2006 on the effects of BMI on lung volumes which showed that BMI had significant effects on all of the lung volumes and the greatest effects were on functional residual capacity (FRC) and expiratory reserve volume (ERV) and occurred at BMI values < 30kg/m² The limitation of this study however, was that the entire population was white and the findings may not be reproducible in other ethnic groups.

Body weight and BMI can be easily measured and therefore are frequently used in large scale epidemiologic studies. A major limitation of these measures is that they do not

distinguish between fat mass and muscle (lean) mass, which have opposite effects on pulmonary function. (90-91, 96) Another important limitation is that both weight and height are surrogate measures of body size and are important predictors for pulmonary fat distribution, both of which play an important role on the association between obesity and pulmonary function.

5.5 Relationship between age, height, weight, BMI, duration of residency and the Lung function Indices in the Exposed and non- Exposed Communities

The results of this study showed that between the two communities, sex of respondents showed the highest relative magnitude of influence on the PEFR, FVC and FEV₁ and was followed by age and weight of the respondents. The significant predictors of PEFR in the current study were age, gender, height, and duration of residency. Across the predictor variables, a similar pattern of relative influence of the predictor variables was also found on sex and age. These findings are in keeping with the findings of previous studies. (46-50)The association between duration of residency and FVC and FEV₁ showed a negative relationship which was statistically significant.

The study also showed a negative relationship between the status of exposure to gas flaring and other lung function indices measured which was statistically significant. These findings are consistent with previous studies that looked at PEFR and exposure to gas flaring in the Niger Delta. (19) Although this finding has not been consistently documented by all investigators in the Niger Delta (a previous study reported no significant difference between lung function indices of exposed respondents and controls), (62) a possible explanation for this could be the result of involvement of small airways in early exposure to toxic pollutants which regular spirometric measurements may not detect. The respiratory effects of air pollution from gas flaring depend on the

type and mix of pollutants, the concentration in the air, the amount of time that an individual is exposed to the pollutant, how much of the pollutant the individual breathes in and how much of the pollutant penetrates the lungs. The air flow pattern and approximate distance of deposition of the gas flaring by products need robust investigation as that would suggest the likely points of severe impact among the distant communities other than the immediate Ibeno communities.

CHAPTER SIX

SUMMARY

This study focused on the effects of gas flaring on the lung function of Ibeno community residents, compared with the lung function of residents of a similar community devoid of gas flaring (Etinan). Previous studies on lung function in other areas of the Niger Delta region which are relatively few, had reported findings which were inconsistent. There is also no documented evidence of a similar study in the study location. Therefore the study's specific objectives were to identify the socioeconomic characteristics of

respondents in both exposed and control communities. Also, to compare the pattern of respiratory symptoms seen among residents in both communities, as well as ascertain the variation in status of the lung function indices (PEFR, FVC, FEV₁, FEV₁/FVC) of Ibeno residents and those of the non-exposed community(Etinan). Finally, the study sought to determine the relationship between age, height, weight, BMI, duration of residency and lung function indices in residents of both communities.

A total of 386 adults resident in Ibeno for at least two years who met the inclusion criteria and 386 age, sex and height- matched controls resident in Etinan, also for at least two years were recruited to a cross-sectional survey comparing respiratory symptoms and lung function indices. The instruments for data collection were a questionnaire, weighing scale, spirometer, and a calibrated ruler. The questionnaire enquired about the socio-demographic data of the respondents, as well as medical history focusing on history suggestive of respiratory disorder, past history of chronic pulmonary disorder and family history of asthma. With the help of four research assistants who worked throughout the study in both locations the study was carried out after obtaining ethical approval from the University of Uyo Teaching Hospital, Uyo.

The results of the study showed that the mean age, height and sex distribution of both subjects and controls were comparable, although more of the respondents in the exposed community were overweight or obese compared with respondents from the control community. The respondents from the exposed community (a riverine area) were mostly involved in trading of sea foods, fishing or fish smoking, while the respondents from the control community were predominantly farmers, and had at least primary education. Respondents were sampled from six villages across both communities. More of the respondents from the exposed community experienced symptoms of cough, chest

pain, breathlessness and wheezing. They also had more respondents with a positive family history of asthma. A comparative assessment of the lung function indices of residents of Ibeno and the control community showed statistically significant differences in PEFR, and FEV₁. In this study, sex of respondents showed the highest relative magnitude of influence on the PEFR, FEV₁ and FVC followed by age and weight of the respondents. The association between duration of residency and lung function indices also showed a negative relationship which was statistically significant.

CONCLUSION

Based on the findings of this study, Ibeno residents had more respiratory symptoms than the controls and had lower PEFR and FEV₁ values than the controls which were statistically significant. The deficit in PEFR and FEV₁ were observed to be more with longer duration of stay, and status of gas flaring was also negatively associated with lung function indices. Important predictors of lung function include age, sex and height.

RECOMMENDATIONS

The study has shown that chronic exposure to gas flaring is associated with reduction in lung function among residents of Ibeno where this practice has been ongoing for decades. As a result, the following recommendations are made:

- 1) Government should expedite actions to stop gas flaring in the oil producing communities or reduce it to the barest minimum.
- 2) Residents of affected communities should be provided with adequate health care facilities and regular lung function assessments done to ensure early detection of lung function impairment.
- 3) Healthcare professionals should intensify efforts to educate the populace and increase awareness about air pollution and lung health.

Limitations of the Study

Lung function decline can occur following exposure to pollutants other than those contained in gas flares. Although cigarette smoking was included in the exclusion criteria, another important confounder in this study was exposure to biomass fuel. Inability to assess Air quality was also a limitation.

Future Research

Future studies should incorporate measurements of personal exposure to gas flaring possibly using exhaled breath assessments of toxic components of gas flaring or sputum analysis. It may also be useful to follow up potentially susceptible groups for a longer time to identify specific disorders.

REFERENCES

1. Yang IA, Fong KM, Zimmerman PV, Holgate ST, Holloway JW. Genetic susceptibility to the respiratory effects of air pollution. *Thorax*. 2008;63(6):555-563.
2. Levin BC. Combustion toxicology. In: Wexler P, editor. *Encyclopedia of Toxicology*. vol. 1. San Diego: Academic Press; 1998. p. 360-374.
3. Balmes JR, Eisner MD. Indoor and outdoor air pollution. In : Mason RJ, Broaddus VC, Martin TR, King TE, Schraufnagel DE, Murray JF, et al., editors. *Murray and Nadel's Textbook of Respiratory Medicine*. 5th ed. Philadelphia Pa: Saunders Elsevier; 2010. p.1601-1618.
4. Lee AS, Mellins RB. Lung injury from smoke inhalation. *Paediatr Resp Rev*. 2006 Jun;7(2):123-128.
5. Alarie Y. Toxicity of fire smoke. *Crit Rev Toxicol*. 2002 Jul;32(4): 259-289.
6. Ajugwo AO. Negative effects of Gas Flaring: The Nigerian Experience. *Journal of Environment Pollution and Human Health* 2013;1(1):6-8.
7. Ayoola TJ. Gas flaring and its implications for environmental accounting in Nigeria. *Journal of Sustainable Development*.2011; 4(5):244-250.
8. Ite AE, Udo JI. Gas Flaring and Venting Associated with Petroleum Exploration and Production in the Nigeria's Niger Delta. *American Journal of Environmental Protection*.2013;1(4):70-77.
9. Osuola A, Roderick P. Gas Flaring in Nigeria: A human rights environmental and economic monstrosity [Internet]. Amsterdam: Environmental Rights Action/Friends of the Earth and the Climate Justice Programme;2005[cited 2013Feb10]. Available from:

http://www.foe.co.uk/sites/default/files/downloads/gas_flaring_nigeria.pdf

10. Ismail O, Umuokoro G. Global Impact of Gas Flaring. *Energy and Power Engineering*. 2012; 1(4):290-302.
11. Oni SI, Oyewo MA. Gas Flaring, Transportation and Sustainable Energy Development in the Niger-Delta, Nigeria. *Journal of Human Ecology*. 2011; 33(1):21-28.
12. Summer W, Haponik E. Inhalation of irritant gases. *Clin Chest Med*. 1981; (2):273-287.
13. Blanc PD. Chemical inhalation injury and its sequelae. *West J Med*. 1994; 160(6):563.
14. Kinsella J, Carter R, Reid WH, Campbell D, Clark CJ. Increased airways reactivity after smoke inhalation. *Lancet*. 1991; 337: 595-597.
15. Huang YT, Ghio AJ, Maier LA. *A Clinical Guide to Occupational and Environmental Lung Diseases*. Humana Press; 2012:217-30.
16. Sonibare JA, Adebisi FM, Obanijesu EO, Okelana OA. Air Quality Index pattern around petroleum production facilities. *Management of Environmental Quality: An International Journal*. 2010; 21(3): 379-392.
17. Jerome A, "Use of Economic Instruments for Environmental Management in Nigeria". A Paper presented at Workshop on Environmental Management in Nigeria and Administration (NCEMA) 2000. In: Tawai CC, Abowei JFN. Air pollution in the Niger Delta Area of Nigeria. *International Journal of Fisheries and Aquatic Sciences*. 2012;1(2):94-117.

18. Oluwole AF, et al. "Impact of the Petroleum Industry on Air Quality in Nigeria." 8th Biennial International Seminar on the Petroleum Industry and the Nigerian Environment, Port Harcourt 1996; pp 17-21.
19. Ovuakporaye SI, Aloamaka CP, Ojieh AE, Ejebe DE, Mordi JC. Effects of Gas Flaring on Lung Function among Residents in a Gas Flaring community in Delta State. *Research Journal of Environmental and Earth Science*.2012; 4(5):523-528.
20. Chapman S, Robinson G, Stradling J, West S. *Oxford Handbook of Respiratory Medicine*. 2nd Ed. Oxford University press; 2009:107-112
21. BTS Statement on malignant mesothelioma in the United Kingdom. *Thorax*. 2007; 62: ii1-ii19.
22. Robinson BW, Lake RA. Advances in malignant mesothelioma, *N Engl J Med*. 2005; 353(15): 1591-1603.
23. American Thoracic Society. Diagnosis and initial management of non-malignant diseases related to asbestos. *Am J Respir Crit Care Med*. 2004; 170: 691-715.
24. Report of the 2006 census final results. Federal Republic of Nigeria Official Gazette 2009; 96:B22.
25. Corporate Planning and Strategy Division. Annual Statistical Bulletin [Internet]. Abuja: NNPC; 2012.1st Ed. [Feb 2013]. Available from:
<http://www.nnpcgoup.com/Portals/o/monthly%20performance/2012%/20ASB%201st%20edition.pdf>
26. Ranu H, Wilde M, Madden B. Pulmonary Function Tests. *Ulster Med J* 2011; 80(2):84-90.

27. Hegewald MJ, Crapo RO. Pulmonary Function Testing. In: Mason RJ, Broaddus VC, Martin TR, et al, eds Murray and Nadel's Textbook of Respiratory Medicine 5th ed. Philadelphia Pa: Saunders Elsevier; 2010:522-553.
28. Kirally A. History of Spirometry. Journal of Pre-health Affiliated Students, JPHAS. 2005; 4(1):21-25.
29. Hutchinson J. On capacity of lungs and on respiratory functions with view of establishing a precise and easy method of detecting disease by spirometer. Tr Med-Chir Soc London.1846; 29:137. In Kory RC, Callahan R, Boren HG, Syner MJC. The Veterans administration army cooperative study of pulmonary function. Clinical spirometry in normal men. Am J Med. 1961; 30:243-258.
30. Dreyer G. Investigations on the normal vital capacity in man and its relation to the size of the body. Lancet 1919; 2: 227. In Kory RC, Callahan R, Boren HG, Syner MJC. The Veterans administration army cooperative study of pulmonary function. Clinical spirometry in normal men. Am J Med. 1961; 30:243-258.
31. Smith D, Harrocks S. Defining Perfect and Not-So-Perfect Bodies, The Rise and Fall of the 'Dreyer method' for the Assessment of Physical Fitness. 1918-1926, in Sobal J, Maurer D, Aldine de Gruyter (eds). Weighty Issues: Fatness and Thinness as social problems. 1999:75-94.
32. Gould BA. Investigations in the military and anthropological statistics of American soldiers. New York: Hurd and Houghton 1869. In Young RC, Rachael RE. Pulmonary function tests: a good measure? J Natl Med Assoc. 1982; 74(5):415-417.

33. Smillie WG, Augustine DL. Vital capacity of the Negro race. JAMA 1926; 87: 2055-2058. In Young RC, Rachael RE. Pulmonary function test: a good measure? J Natl Med Assoc. 1982; 74(5):415-417.
34. Hyatt RE, Scanlon PD, Nakamura M. Interpretation of Pulmonary Function Tests, A Practical Guide. 2nd Ed. Philadelphia: Maple press; 2003:5-25.
35. Leiner GC, Abramowitz's, Small MJ, Stuby UB, Lewis WA. Expiratory peak flow rate. Standard values for normal subjects. Use as a clinical test of ventilatory function. Am Rev Resp Dis. 1963; 88:644-651.
36. Scanlon, PD, Connett JE, Waller LA, Altose MO, Bailey WC, Buist AS, Tashkin DP. Smoking cessation and lung function in mild to moderate chronic obstructive pulmonary disease: the Lung Health Study/Research Group. Am J Respir Crit Care Med. 2000; 161,381-390.
37. Golshan M, Nematbakhsh M, Amra B, Crapo RO. Spirometric reference values in a large Middle Eastern population. Eur Respir J. 2003; 22:529-534.
38. Morgan WK, Reger RB. Rise and fall of the FEV(1). Chest. 2000; 118(6):1639-1644.
39. Nancy EL, Mary M, Mary EK. Spirometry: Don't Blow it! Chest. 2009; 136(2):608-614.
40. Salisu AI. Reference Population Equations using Peak Expiratory Flow Meters: An overview. Bayero Journal of Pure and Applied Sciences. 2009; 2(2):16-18.
41. Chin NK, Ng TP, Hui KP, Tan WC. Population based standards for pulmonary function in non-smoking adults in Singapore. Respirology. 1997; 2:143-9.
42. Le Souef PN. Paediatric prediction equations for PEF (growth, ageing, gender, race and health). Eur Resir J. 1997; 10 Suppl 24:75-79s.

43. Mary SP, Fanny WK, Arthur CL et al. Updated Spirometric Reference Values for Adult Chinese in Hong Kong and Implications on Clinical Utilization. *Chest*. 2006; 129(2):384-392.
44. Stinson JM, McPherson GL, Hicks K, et al. Spirometric standards for healthy black adults. *JNMA*. 1981; 73:733-739.
45. Elebute EA, Femi-Pearse D. Peak Flow Rate in Nigeria. Anthropometric determinants and usefulness in assessment of ventilatory function. *Thorax*. 1971; 26:597-601.
46. Shamssain MH. Forced expiratory indices in normal black southern African children aged 6-19 years. *Thorax*. 1991; 46:175-79.
47. Wang X, Dockery DW, Wypij D, Fay M, Ferris BG. Pulmonary function between 6 and 18 years of age. *Paed Pulmonol*. 1993; 15(2):75-88.
48. Vijayan VK, Reetha AM, Kupuro KV, Venkatsen P, Navakavathy S. Pulmonary function in normal south Indian children aged 7-19 years. *Indian J Chest Dis Allied Sci*. 2000; 42(13):147-56.
49. Edemeka DBU, Udoma MG, Ibrahim M. Peak expiratory flow rate in rural Nigerian children. *Sahel Med J*. 2000; 3:37-39.
50. Njoku CH, Anah CO. Reference values for peak expiratory flow rate in adults of African descent. *Trop Doc*. 2004; 34:135-140.
51. Johannsen ZM, Erasmus LD. Clinical spirometry in normal Bantu. *Am Rev Resp. Dis*. 1968; 97:585-597.
52. Kory RC, Callahan R, Boren HG, Syner MJC. The Veterans Administration Army Cooperative Study of Pulmonary Function. Clinical spirometry in normal men. *Am J Med*. 1961; 30:243-258.

53. Kelly FJ. Oxidative stress: Its role in air pollution and adverse health effects. *Occup Environ Med.* 2003; 60:612-616.
54. Kurmi OP, Devereux G, Smith WC, Semple S, Steiner MF, Simkhada P, Lam KB, Ayres JG. Reduced lung function due to biomass smoke exposure in young adults in rural Nepal. *Eur Respir J.* 2013; 41(1):25-30.
55. Peters EJ, Esin RA, Immananagha KK, Siziya S, Osim EE. Lung function status of some Nigerian men and women chronically exposed to fish drying using burning firewood. *Cent Afr J Med.* 1999; 45:119-124.
56. Ibhafidon LI, Obaseki DO, Erhabor GE, Akor AA, Irhabor I, Obioh JB. Respiratory symptoms, lung function and particulate matter pollution in residential indoor environment in Ile-Ife, Nigeria. *Niger Med J.* 2014; 55:48-53.
57. Alakija W, Iyawe VI, Jakire LN, Chiwuzie JC. Ventilatory function of workers at Okpella cement factory in Nigeria. *West African Journal of Medicine* 1990; 9(3):187-192.
58. Nagoda M, Okpapi JU, Babashani M. Assessment of respiratory symptoms and lung function among textile workers at Kano Textile Mills, Kano, Nigeria. *Niger J Clin Pract.* 2012; 15:373-379.
59. Afolabi BM, Akintowa A. Pulmonary ventilatory function of petrochemical workers in Warri, Nigeria. *Niger Med J.* 1994; 27:41-46.
60. Umoh VA, Peters EJ. The relationship between lung function and indoor air pollution among rural women in the Niger Delta region of Nigeria. *Lung India.* 2014; 31(2):110-115.
61. Jain BL, Patrick M. Ventilatory function in Nigerian coal miners. *Br J Ind Med.* 1981; 38(3):275-280.

62. Joffa PK, Nwafor A, Adienbo OM. Correlation between Body Mass Index and Peak Expiratory Flow Rate of an Indigenous Nigerian Population in the Niger Delta Region. *Research Journal of Recent Sciences*. 2013; 2:28-32.
63. Ana G, Sridhar MK, Bamgboye EA. Environmental risk factors and health outcomes in selected communities of the Niger Delta area, Nigeria. *Perspect Public Health*. 2009; 129:183-91.
64. Scott HM, Soskolne CL, Lissemore KL, Martin SW, Shoukri MM, Coppock RW, Guidotti TL. Association between air emissions from sour gas processing plants and indices of cow retainment and survival in dairy herds in Alberta. *Can J Vet Res*. 2003; 67(1):1-11.
65. Western Canada Study of Animal Health Effects Associated with Exposure to Emissions from Oil and Natural Gas Field Facilities: Interpretive Overview by the Scientific Advisory Panel: a Study of 33,000 Cattle in British Columbia and Saskatchewan. *Western Interprovincial Scientific Studies Association*. 2006:1-15.
66. Stroscher M. Investigation of Flare Gas Emissions in Alberta. Final Report, Environment Canada, Conservation and Protection, The Alberta Energy and Utilities Board and the Canadian Association of Petroleum Products, Environmental Technologies, Ottawa, 1996.
67. Miller K, Chang A. Acute Inhalational Injury. *Emerg Med Clin North Am*. 2003; 21:533-557.
68. Blanc PD, Glabo M, Hiatt P, Olson KR, Balmes JR. Symptoms, lung function and airway responsiveness following irritant inhalation. *Chest*. 1993; 103:1699-1705.

69. Chia KS, Jeyaratnam J, Chan TB, Lim TK. Airway responsiveness of firefighters after smoke exposure. *Br J Ind Med.* 1990; 37:524-527.
70. Carlsten C, Kaufman JD. Air Pollution. In: Albert RK, Spiro SG, Jett JR, eds *Clinical Respiratory Medicine* 3rd ed. Mosby Elsevier; 2008:843-851.
71. Palange P, Simonds A. *ERS Handbook, Respiratory Medicine*, European Respiratory Society; 2010
72. Wheeler DR, Deichmann U, Pandey KD, Hamilton KE. Ambient Particulate Concentrations in Residential and Pollution Hotspot areas of World Cities: New Estimates based on the Global Model of Ambient Particulates (GMAPS), The World Bank Development Economics Research Group and the Environment Department Working Paper (forthcoming 2006), The World Bank, Washington DC.
73. Jappinen P, Vilkkka V, Martila O, et al: Exposure to hydrogen sulphide and respiratory function. *Br J Ind Med*1990;47:824-828.
74. Parra O, Monso E, Gallego M, Morera. Inhalation of hydrogen sulphide: A case of subacute manifestations and long term sequelae. *Br J Ind Med.*1991; 48:286-287.
75. Guidotti TL. Hydrogen sulphide. *Occup Med.*1996; 46: 367-3711.
76. Herbstman JB, Frank R, Schwab M, Williams DL, Samet JM, Breyse PN, et al. Respiratory effects of inhalation exposure among workers during the clean-up effort at the World Trade Centre disaster site. *Envir Res.*2005; 99:85-92.
77. Banauch GL, Hall C, Weiden M, Cohen HW, Aldrich TK, Christodoulou V, et al. Pulmonary function after exposure to the World Trade Centre collapse in the New York Fire Department. *Am J Respir Crit Care Med.* 2006; 174:312-319.

78. Park GY, Park JW, Jeong DH, Jeong SH. Prolonged airway and systemic inflammatory reactions after smoke inhalation. *Chest*.2003; 123:475-480.
79. Argo J: Unhealthy effects of upstream oil and gas flaring. A report prepared for save our seashores. 2002:1-28.
80. Bamberger M, Oswald RE. Impacts of gas drilling on human and animal health. *New Solut*. 2012; 22(1):51-77.
81. Mckenzie LM, Witter RZ, Newman LS, Adgate JL. Human health assessment of air emissions from development of unconventional natural gas resources. *Sci Total Envir*. 2012; 424:79-87.
82. Kirkwood B. Essentials of medical statistics. Oxford Blackwell scientific populations. In: Olawuyi JF. Choosing the study subjects and sampling. In *Biostatistics: A foundation course in health sciences*. Yotson consult communications (publishers) 1996:110-117.
83. British Medical Research Council (BMRC). Standardized questionnaire on respiratory symptoms. *Br Med J*. 1960; 2:1665.
84. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Crapo P, et al: Standardization of spirometry. *Eur Resp J*. 2005; 26(2): 319-338.
85. Miller MR, Crapo R, Hankinson J, Brusasco V, Burgos F, Casaburi R et al: General consideration for lung function testing. *Eur Res J*. 2005; 26(1):153-161.
86. Ferguson GT, Enright PL, Buist AS, Higgins MW. Office spirometry for lung health assessment in adults: A consensus statement from the National Lung Health Education Program. *Chest*. 2000; 117 (4): 1146-1161.

87. Erhabor GE, Ojo JO, Oluwole AF, Fatusi AO. Reference Values for spirometric indices in native Nigerian men from Ile-Ife, Yoruba land, Nigeria. *Nigerian Journal of Health Sciences*. 2002; 2:7-10.
88. Patrick JM, Femi-Pearse D. Reference Values for FEV₁ and FVC in Nigerian men and women, a graphical summary. *Niger Med J*. 1976; 6:380-385.
89. Veale D, Rabec C, Labaan JP. Respiratory complications of obesity. *Breathe*. 2008; 43:210-223.
90. Chen Y, Hone SL, Dosman JA, Body weight and weight gain related to pulmonary function decline in adults: a six year follow up study. *Thorax*. 1993; 48:375-80.
91. Wise RA, Enright PL, Connett JE, Anthonisen NR, Kanner RE, Lindgren P et al. Effect of weight gain on pulmonary function after smoking cessation in the Lung Health Study. *Am J Respir Crit Care Med*. 1998; 157:866-72.
92. McKay RT, Levin LS, Lockey JE, Lemasters GK, Medvedovic M, Papes DM et al. Weight change and lung function: implications for workplace surveillance studies. *J Occup Environ Med*. 1999; 41:596-604.
93. Chinn DJ, Coles JE, Reed JW. Longitudinal effects of change in body mass on measurements of ventilatory capacity. *Thorax*. 1996; 51(7):699-704.
94. Yue C, Donna R, Yvon FC, James D. Waist circumference is associated with pulmonary function in normal-weight, overweight and obese subjects. *Am J Clin Nutr*. 2007; 85(1)35-39.
95. Jones RL, Nzekwu MM. The effects of body mass index on lung volumes. *Chest*. 2006; 1303:827-833.

96. Santana H, Zoico E, Turcato E, Tosoni P, Bissoli L, Olivieri M, et al. Relation between body composition, fat distribution and lung function in elderly men. *Am J Clin Nutr.* 2001; 73:827-31.
97. Ana G, Adeniyi B, Ige O, Oluwole O, Olopade C. Exposure to emissions from firewood cooking stove and the pulmonary health of women in Olorunda community, Ibadan, Nigeria. *Air quality, Atmosphere and Health.* 2013; 6(2):465-471.

APPENDIX 1

Constituents of Flares

Table 1: On-Site Characterization of chemical emissions from the sweet gas flares by Strosher, M. in 1996(51)

On site characterization	Mg/m ³	Thermal Absorption >10mg/m ³	Mg/m ³	Solvent Extraction >10mg/m ³	Mg/m
Hydrogen	20.0	Pentane	12.8	Subst benzene	9.83
CO	15.7	3-penten-1-yne	19.3	Azulene	21.2
CO ²	4890	Benzene	144.5	Subst benzene	11.47
Carbon	54.2	1, 5-hexadiyne	48.2	Naphthalene	99.39
Methane	103.8	Methyl benzene	27.5	2-methyl naphthalene	9.25
Ethylene	29.0	Ethyl benzene	13.7	1-methyl Naphthalene	6.18
Acetylene	53.7	Ethynyl benzene	94.8	1, 1-biphenyl	58.7
Ethane	9.9	Ethenyl benzene	82.1	Biphenylene	42.81
C3 HC's	6.4	Benzldehyde	18.7	1H phenalene	21.01
C4 HC's	116.5	Phenol	26.4	9H fluorine	41.09
Benzene	18.2	Napthalene	88.7	Phenenthrene	10
Toluene	29.8	1, 1'-biphenyl	16.1	Anthracene	42.11
Xylenes	75.5	Biphenylene	19.1	Fluoranthene	51.35
Styrene	79.6	Acenapthalene	23.2	Pyrene	32.37
Ethylene benzene	77.2			4-methyl;pyrene	9.1
Napthalene	128.5			1 methyl pyrene	8.4
Other HC's	65.0%	38 Others HC's	132.8	Benzo(ghi) fluoranthene	10.18
CE				Cyclopenta (cd)-pyrene	29.77
				Benz(a)-anthracene	17.33
				48 Others HC's	94.47

The analysis of the sour gas flare is included as Table 2. All amounts are in units of mg/m³

APPENDIX II

Table 2: Emissions in the downwind plume of a Waste gas Flare from a Sour Oilfield battery” By Strosher, M. in 1996(51)

On Site	Thermal Desorption	Mg/m ³	Solvent extraction	Mg/m	
Characterization	Mg/m ³	>~1mg/m ³ n= 36	>1 mg/m ³ n=54		
Hydrogen	150	Carbon Disulphide	453.3	Hexanoic acid, 2-ethyl	5.04
CO	8	Thiophene	79.2	Naphthalene	77.1
CO ₂	6870	Benzene	64.3	Benzo[b]thiophene	46.7
Carbon	18.2	Methylbenzene	20.5	Benzoic acid	6.4
Methane	83.1	3-Methyl Thiophene	2.7	Benzo[b] thiophene, 4 methyl	8.9
Ethylene	6	Benzene-ethyl	7.1	Naphthalene, 2-methyl	14.3
Acetylene	36.4	Benzene, 1-3 dimethyl	6.5	Naphthalene, 1-methyl	10.8
Ethane	4.9	Benzene, ethynyl	41.9	Phthalic anhydride	2.3
C ₃ HC's	5.7	Benzene, ethynyl	34.4	1-1' Biphenyl	78.0
C ₄ HC's	2.9	Benzene, methoxy	1.4	Naphthalene, 1-ethyl	6.7
Benzene	24.4	2(511)-thiophene	31.1	Thiophene, 2-phenyl	7.0
Toluene	12.4	2-Thiazolamine	0.9	Thiophene, 3-phenyl	12.0
Xylenes	6.7	Benzaldehyde	12.6	Naphthalene, 2,3-dimethyl	5.4
Styrene	22.7	Benzonitrile	1.3	Biphenylene	13.2
Ethynyl benzene	18.4	Benzonitrile	3.6	Dibenzofuran	7.1
Napthalene	31.2	Decane	1.2	9II-fluorene	54.2
Others 'HC's	111	Phenol	12.2	Dibenzothiophene	82.2
Efficiency	84%	Ethanone, 1-phenyl	61.9	Phenanthrene	34.1
Carbon CP		Naphthalene	61.5	Sulphur (S ₈)	157.4
Sulphur Dioxide	6910	Azulene	34.4	Fluoranthene	14.1
Hydrogen Sulphide	126	Benzo[b]thiophene	156.6	Pyrene	83.3
Carbonyl Sulphide	64	Naphthalene, 2-methyl	1.5	Chrysene	2.4
Carbon Disulphide	482	Naphthalene, 1-methyl	0.9	Benzo[a]pyrene	0.5
Other S	210	1,1' Biphenyl	8.0		
Efficiency	82.4%	Dibenzothiophene	6.6		
sulphur CP					

APPENDIX III

INFORMED CONSENT FORM

To be read out to patients before consultation starts

I amof the Department of Internal
Medicine, University of Uyo Teaching Hospital.I am conducting a study on “Effects of
Gas Flaring on the Lung Health of Ibeno Community Residents”

You are hereby invited to take part in the study.

You will be required to answer questions from a questionnaire. Please note that any
information we receive from you will be kept secret and your name will not appear
directly in the record. After which your weight and height will be measured and physical
examination done using a stethoscope while you lie on a comfortable couch with a
screen. Your lung function will be assessed after proper demonstration by blowing into
a simple device which carries no negative health consequences.

Should you have any problems please call the following number – 08029196846

I have read the description of the research or have had it translated into the language I
understand. I have also talked it over with the doctor to my satisfaction. I know enough
about the purpose, methods, risks and benefits of the research study to judge that I want
to take part in it. I understand that my participation is voluntary and that I may freely
stop being part of this study at any time.

.....
Subject’s signature /thumb print

.....
Name/Signature of Investigator

.....
Date

.....
Date

APPENDIX IV

Effects of Gas Flaring on the Lung Health of Ibeno Community Residents

STUDY IDENTIFICATION NUMBER:

A. SOCIODEMOGRAPHIC CHARACTERISTICS

Patient's initials

Age (in years)

Sex (a) Male (b) Female

Occupation

Marital Status (a) Single (b) Married (c) Widow / Widower

Educational Status (a) No formal education (b) Primary (c) Secondary (d)

Tertiary

Ethnicity

B. HISTORY

- How long have you lived in this community?
- Are you coughing?
- If yes, for how long?
- Is the cough productive of sputum?
- If yes what is the colour of the sputum?
- Are there any blood stains in the sputum?
- Do you have chest pain?
- If yes for how long?
- Do you have breathing difficulties?
- If yes, for how long?

• MRC Grade	Description
1	Not troubled by breathlessness except with strenuous exercise
2	Troubled by shortness of breath when hurrying on the level or walking up a slight hill
3	Walks slower than people of the same age on the level because of breathlessness or has to stop for breath when walking at own pace on the level
4	Stops for breath after walking about 90 m or after a few minutes on the level
5	Too breathless to leave the house or breathlessness when dressing or undressing

- Do you experience wheezing (noisy breathing)?
- If so, when is it worse?
- Have you been to the hospital for any of these complaints?
- If yes, WHEN?
- Are you on medications? If yes, list them.
- What do you use to cook your food?
- Do you have a separate cooking room?
- Are any of your family members or friends having the same symptoms?

PHYSICAL EXAMINATION

1. Weight (kg)
2. Height (metres)
3. Body Mass Index (kg/m²)
4. Respiratory Rate

5. Pulse rate.....
6. Blood Pressure.....
7. FEV₁
8. FVC
9. FEV₁/FVC
10. PEFR.....