## KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY

## **COLLEGE OF HEALTH SCIENCES SCHOOL**



## ASSESSING THE LEVEL OF PARTICULATE MATTER AND CARBON

MONOXIDE EXPOSURE IN THE KAASE COMMUNITY OF KUMASI-

**METROPOLIS, GHANA.** 

BY

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**SEPTEMBER 2019** 

## KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY, KUMASI

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DEPARTMENT OF OCCUPATIONAL AND ENVIRONMENTAL HEALTH

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A THESIS SUBMITTED TO THE DEPARTMENT OF OCCUPATIONAL AND ENVIRONMENTAL HEALTH, SCHOOL OF PUBLIC HEALTH, COLLEGE OF HEALTH SCIENCES, IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF DEGREE OF MASTER OF PUBLIC HEALTH IN

**OCCUPATIONAL AND ENVIRONMENTAL HEALTH AND SAFETY** 

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**SEPTEMBER 2019** 

## **DECLARATION**

I hereby do declare that except for references to other people's work, which have been duly acknowledged, this piece of work is my own composition and neither in whole nor in part has this work been presented for the award of a degree in this university or elsewhere.

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## **DEDICATION**

This thesis is dedicated to the honour of Jehovah God for his gracious love and mercy that kept me throughout this thesis work.



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## LIST OF ACCRONYMS AND ABBREVIATIONS

AB	-	Abattoir Area
BMF	-	Biomass Fuel
CHRPE	-	Committee of Human Research Publications and Ethics
СО	-	Carbon Monoxide
COHb	-	Carboxyhemoglobin
COPD	-	Chronic Obstructive Pulmonary Disease
СМ	-	Community Members
EPAM		Environmental Particulate Air Monitor
G-EPA	¢	Ghana Environmental Protection Agency
HAS	12	Health and Safety Authority
OSHA	(- )	Occupational Safety and Health Administration
PM	1	Particulate Matter
USA-EPA	ale an	United State Environmental Protection Agency
USAID	N	United States Agency for International Development
WHO	-	World Health Organization

### ABSTRACT

Particulate matter (PM) is made of solid and liquid particles from industry, traffic, domestic heating and various natural sources while carbon monoxide (CO) is scentless and profoundly poisonous gas, virtually undetectable by an individual during exposure. Both PM and CO pose some health problems. This study looked at the occupational and environmental health risks of exposure to CO and PM from biomass (car tyre and firewood smoke). The PM and CO monitoring and sampling took place in two locations (the abattoir area and 100 meters into the community) purposively selected from Kaasi Community, in the Asokwa Municipality of Kumasi- Ghana. The meat preparation technologies identified was the use of car tyre and firewood. Measurement were made on a minute basis using Aeroqual Series 500 (S500) gas monitor for CO and Environmental Particulate Air Monitor (EPAM-7500) for PM. It was discovered that, CO and PM concentrations in the abattoir area were higher than that which was recorded in the community by a large margin of (an average five times) with a mean of CO 5.78ppm and 1.31 ppm respectively. PM levels in abattoir area were higher than the community. With PM 10, an average mean of 2.35 mg/m3 and .19 mg/m3 was seen in the abattoir area and community, respectively. PM 2.5 was also higher in the abattoir area than the community at an average of 1.50 mg/m3 and .14mg/m3 respectively. A sample t-test was carried out to compare concentration levels with local and international standards and the finding revealed that, it was within the acceptable rage for CO at (p<0.001) for USA-EPA but statistically higher to WHO. PM10 and 2.5 were both statistically higher to WHO, USA-EPA and G-EPA. Due to the higher concentration levels of PM to local and international standard, both workers and community members exposed reported some health symptoms and diseases (blurred vision, eye irritation, Hypertension, headache, nausea, asthma, respiratory tract infection, muscular weakness, chest pain etc) associated with the exposure to CO and PM. A regression analysis was carried out to check the duration of exposure and associated health effects, and the finding showed that blurred vision was statistically significant with CO exposure. Asthma and Hypertension were also statistically significant with PM exposure for a longer exposure duration. It is recommended that, charcoal,

Liquefied Petroleum Gas and Biogas usage are the best in addressing CO and PM exposure.

#### **CHAPTER ONE**

#### INTRODUCTION

#### 1.1 Background of the Study

Air is a worldwide gas that is odorless, colorless and vital for the survival of lifespan on earth. Air contaminants are a diverse mix of gassy and particulate matter (PM) (Zala & Jure, 2011). The key gassy constituents of air contamination comprises of Sulphur dioxide (SO<sub>2</sub>), carbon monoxide (CO), nitrogen oxide (NO<sub>2</sub>), ozone (O<sub>3</sub>), nitrate (NH<sub>3</sub>), carbonyl compounds, and organic solvents (Monn & Shaeppi 1993; Matsumoto & Tanaka 1996; Zala & Jure, 2011). PM is in the form of solid and liquid particles. These are gotten from industrial activities, traffic flow, native activities and several natural sources (Zala & Jure, 2011). Indoor besides outdoor air pollution pose a health risk to the human population and the environment. Air pollution exposure is a public health issue associated with various health effects, including respiratory disease, cardiovascular disease, cancer, pregnancy complications, and adverse birth outcomes (Health Effects Institute, 2010).World Health Organization (2009), predicted air pollution contributes to 3.1 million early mortality globally each year. According to Jimoda, 2012, as cited by Njoku et al., 2016, Particulate matter has historically been a concern due to its presence in a variety of occupational environments). PM is a well-known air pollutant, consisting of a mixture of liquid and solid particles suspended in the air, Also the indicators for describing PM that is relevant to health refer to the mass concentration of particles with a diameter of less than 10 µm (PM10) and of particles with a diameter of less than 2.5 µm (PM2.5). PM2.5, often called fine PM Some Common chemical constituents of PM include nitrates, ammonium, sulfates, and other inorganic ions such potassium, magnesium, ions of sodium, calcium, and chloride, organic and elemental carbon, crustal material, particle-bound water, metals (WHO, 2013). It is estimated that about 5% of lung cancer and 3% of cardiopulmonary deaths are attributable to PM globally (WHO, 2013).

A study by WHO, 2016 reviewed that in 2010, ambient air pollution, as annual PM2.5 accounted for 3.1 million deaths and around 3.1% of global disability-adjusted life years. Fireplaces, industrial-scale boilers, diesel trucks and meat cooking operations, cars with and without catalytic converters, all emit particles primarily in the range 0.1–0.2 µm which is capable of causing health effects (Ho et al.; 2002). According to WHO (2013), Recent studies suggest that short-term differences in particulate matter exposure are associated with health effects even at low levels of exposure (below 100  $\mu$ g/m<sup>3</sup> Long-term PM2.5 exposure is associated with a 6–13 percent rise in the long-term danger of cardiopulmonary death per 10  $\mu$ g / m<sup>3</sup> of PM2.5 (Pope et al., 2002 ; Beelen R et al., 2008 ; Krewski et al., 2009 ; WHO, 2013). Firewood has been the prime energy type that has been consumed in Ghana from 2003 to 2008 and contributing about 70%, 77.7% and 76.4% in 2000, 2004 and 2008 respectively (Arthur et al., 2011). It is predicted that Smoke from cooking fires will release about 7 billion tons of carbon in the form of greenhouse gases to the environment by 2050 in Africa alone (Arthur et al., 2011). There are numerous health risks associated with the use of biomass which has been documented by several studies (Ezzati and Kammen, 2002; Goldstein, 2008). These smoke from the biomass exposes families to destructive quantities of gases such as carbon monoxide, benzene, etc. (Mukherjee et al., 2014). Global statistics according to Costa et al. (2014) estimated air pollution to be related to an unexpected 17% prolonged obstructive pulmonary ailment, over 30% ischemic stroke, 9% of lung cancer deaths, and 9% respiratory disease. Exposure to carbon monoxide both acute and chronic is very serious health concern. In Ghana, human activities such as the use of scrap tyres in roasting meat instead of firewood and gas stove have been documented (Obiri-Danso et al., 2008). Locally singeing of meant ensues in open fire were supposed to use Gas fuel however due to high cost Gas fuel recently has resulted in local butchers using scrap tyres and firewood as an alternative source of fuel to singe slaughtered livestock (Obiri-Danso et al., 2008).). This practice has increased the volume

of smoke produced, therefore, contributing intensely to the health implications from the release of carbon monoxide and particulate matter. Due to the increasing demand for these activities vis-à-vis the negative health implications associated with the use of scrap tires, it will not be wrong to associate the many respiratory, cardiovascular disease and deaths in Kumasi metropolis, Ghana to this practice.

#### **1.2 Statement of Problem**

Disregarding practices that negatively affect human health within communities is a key component of public health. The Environmental Protection Agency does not consider the scrap tyres as hazardous materials, but it is the burning of these tyres which emits toxic gases and have major adverse health effects is the problem of concern (EPA, 2010). The burning of car tyres improperly is mostly a problem in developing countries in which Ghana is a victim. Ambient air pollution is one of the most crucial environmental concerns in cities throughout the world – especially in developing countries (Faiz & Sturm, 2002). The sustenance of life of an individual is based on the quality of air around. Air pollution due to human activities is an abuse to the environment thus degrading air quality. PM is in the form of solid and liquid particles. These are gotten from industrial activities, traffic flow, native activities and several natural sources. WHO predicted air pollution contributes to 3.1 million early mortality globally each year. (WHO, 2009). The health effects of inhalable PM are well documented (WHO, 2013). Due to the increasing demand for meat production, several unhygienic means are used in processing meat for consumption which poses serious health implications on consumers. A research by Obiri-Danso et al. (2008) examined this in Ghana that, one of the means of meat processing is through the activities of the use of scrap tyres as a substitute for firewood which poses a lot of heavy metal contamination to the meat. Even though the use of scrap tyres pose heavy metal contamination, there is also another serious effect from the smoke emission from this burning tyres which releases particulate matter and carbon monoxide into the atmosphere.

Emissions like Oxides of Nitrogen (NO<sub>3</sub>) and others like sulphur (IV) Oxides (SO<sub>2</sub>) from open tyre fires are known to have serious health impact on humans and the environment (Cont *et al.*, 2016). A study by Cont *et al.* (2016) reviewed that, due to limited data, it was uncertain to know exactly what was being emitted, how much was being emitted, and how dangerous these emissions are, especially to sensitive individuals such as the children and the elderly. Even though several studies have shown the health effects of particulate matter to our health (Goldberg *et al.*, 2001; Kim, *et al.*, 2015), there is limited available data on the effect of smoke released from such local butchers in Ghana who use biomass fuel and scrap tyres for meat processing, Hence this study sought to assess the level of particulate matter and carbon monoxide exposure at Kaase community, Kumasimetropolis.

#### **1.3 Research Questions**

- 1. What is the level of PM and CO within the Kaase Community and in the Kumasi Abattoir Area?
- 2. Is there any statistical difference between concentrations of PM and CO in the abattoir station against standards and within?
- 3. What are the health effects experienced by local butchers and community members due to CO exposure?
- 4. Are there any health effects experienced by residents in community and local butches because of PM emission?

## **1.4 Statement of Objectives**

**1.4.1 Principal Objective:** The principal objective of the study was to assess the level of particulate matter and carbon monoxide exposure in the Kaase Community of Kumasimetropolis, Ghana.

## **1.4.2 Specific objectives**

- 1. To conduct air sampling for particulate matter and carbon monoxide levels within the working space of local abattoir workers and in the community.
- 2. Determine if a statistical difference exists between the concentrations of particulate

Matter and carbon monoxide levels in the abattoir station and the community as well as compare with standards and within.

- 3. To ascertain health effects and clinical signs experienced by local butchers and community members due to CO exposure.
- To identify health effects and clinical symptoms experienced by local butchers residents in the Community due to PM exposure.

## **1.5 Justification of the Study**

The study provides insight into the biomass fuel usage around the Kaase abattoir station in the Kumasi metropolis. Public health and environmental officials were made aware of the effects of the activities on the health of both workers and the community to inform the effectiveness of their monitoring roles in the community through the findings of this study. The level of risk which this activity poses to the community, was highlighted by the findings of this study. The study contributed to the academic literature on air pollution practices. Finally, it instigated further interest and research into general practices and policy issues concerning car tyre activities in the meat siege in Ghana.

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## **CONCEPTUAL FRAMEWORK**

## Figure 1.1 Effects of Biomass Fuel burning Activity and its Effect on Human Health.

## 1.6 Summary of Conceptual Framework

Figure 1 above describes the impact of air pollution in Ghana; emanating from innumerable sources varying from Industrial sector to mining operations, but significantly impacted upon by a human through domestic activities. The activities of humans such as the use of biomass fuel lead to the release of emissions into the atmosphere such as sulphur dioxide, carbon monoxide particulate matter, nitrogen dioxide, etc. This release substance into the atmosphere exposes people to various health effects such as premature deaths, irritations, cardiovascular diseases, etc which goes a long way to also affect the environment and cost of medical bills.

## **CHAPTER TWO**

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#### **REVIEW OF RELATED LITERATURE**

#### **2.1 Introduction**

Air is one of its most important natural resources on earth surface for all living things. The quality of air for human consumption has become a global health concern. Air pollution was formally associated to industrial pollution but recently the attention is now on human activities such as scrap tyre burning, the use of biomass for cooking, the uses of scrap tyre for meat singe, vehicular emissions and improper burning of refuse. According to Fiahagbe (2008) as cited by (Shadow, 2016) conducted a study in Accra and it reviews air pollution has been mainly attributed to vehicular exhaust emissions. Other activities such as domestic uses of biomass and scrap tyre activities also contribute largely to air pollution. They normally release chemical pollutions emissions into the atmosphere are, sulphur dioxide, carbon monoxide particulate matter, nitrogen dioxide etc. Literature related to this study is reviewed under major subheadings which advances in-depth understanding of air pollution due to human activities especially the use of biomass fuel and scrap tyre in meat singe. The two major pollutants that will be discussed in detail in this Session is Particulate Matter (PM) And Carbon Monoxide (CO).

#### **2.2 Pollution**

According to (Seyyednejad *et al.*, 2011) air pollution is defined as the human introduction of chemicals into the atmosphere, biological materials or particulate matter, that cause destruction or discomfort to humans, other living organisms, or damage the environment. Any substance in a sufficient amount when introduced into the environment will cause adverse health effects on human beings, animals or plants is known as a pollutant. Pollutants are in two forms, namely primary and secondary pollutants. Primary pollutants are known to exert the harmful effects in the original form in which they enter the atmosphere examples include  $CO^2$ , CO,  $NO^2$ ,  $SO^2$  and particulate matter. Also, secondary pollutants are dangerous substances formed in the

atmosphere when a primary air pollutant reacts with a substance usually found in the atmosphere or with other pollutants. Secondary pollutants include ozone ( $O_3$ ), hydrogen peroxide, peroxyacetylnitrate (PAN) and peroxybenzoyl nitrate (PBN) (Agbaire and Esiefarienrhe, 2009; Seyyendnjad *et al.*, 2011). Air pollution is essentially made up of three components and these are the source of pollutants, the transporting medium, which is air and target or receptor which could be human, animal, plant and materials (Shadow, 2016).

#### 2.3 Sources of Air Pollution

In order to improve monitoring and controlling air pollution, it is essential to accurately identify the emission sources and determine their emissions. Air pollution can come from various sources such as; industrial activities and the effects of natural occurrences such as volcanoes eruption. These sources release both primary and secondary pollutants into the atmosphere (Pooley & Mille, 1999). According to Chen et al. (2014), human activities such as the use of biomass fuel is a cardinal contributing factor for indoor air pollution internationally. Also, a study was done by Akunne et al., 2006, reviewed that, in many developing countries use biomass fuel in the open for smoking meat and other activities which generate more smoke. common sources of CO production include; open fires, exhaust from internal combustion engines, malfunctioning heating and conditioning systems (cooper, 1980). According to Yadav et al., (2018) stated in their report that, vapour contaminants, for instance, CO, CO2 and O3 are emitted into the atmosphere during biomass combustion which was also supported by the findings of Andreae and Merlet (2001). Another source of concern is the presence of PM in the environment as a result of human activities such as; biomass combustion, bush burning, refuse burning, generator emission, industrial emissions and indiscriminate refuse disposal. Soil and dust re-suspension can also be contributing source of PM, mostly in arid areas or during episodes of long-range transport of dust (WHO, 2013).

#### 2.4 Overview of Atmospheric Air Pollution in Ghana

In African and especially Ghana, and most of the other Sub-Saharan Africa (SSA) countries, there is inadequate consistent and systematic air quality-monitoring for air quality assessment there led to inadequate data on the concentrations as well as the characteristics of atmospheric air pollutants. Environmental Protection Authority of Ghana (EPA) has routine monitoring programs in Accra to measure only particulate matter 10 (PM10) (Shadow, 2016). As at 2016 according to Shadow, there was no systematic record of PM 2.5 concentrations in Accra, partially because the air quality standard for PM2.5 was not then established in Ghana. Results from a study by Ghana EPA and other international bodies such as; United States Agency for International Development (USAID), the United States Environmental Protection Agency (USEPA) and the United Nations Environment Programme (UNEP) between March 2005 and December 2008 on an air quality monitoring program in Accra reviewed vehicular exhaust emissions were the leading contributing factor for air quality monitoring. Even though, a little monitoring has been established in Accra there is not a significant data of Kumasi concerning environmental monitoring for air quality by the use of biomass fuel. The Environmental report in 2004 as was stated by EPA listed the following as the main issues related to air quality in the country; Inefficient utilization of fuels, poorly planned modes of transport, poorly serviced motor vehicles, Inefficient cook-stoves, and fireplaces, Rudimentary kilns and stoves in industries, Charcoal production, and Widespread bush burning. as was cited by (Shadow, 2016)

12	10		-	50	
Table 2.1 Standar	ds for some Princip	al Pollutants.	E Br		
Pollution	Averaging time	Safe	WHO	Ghana	EPA
		concentration	Guideline	Voluo in u	a/m <sup>3</sup>
		zone (US)	Guideline	v alue in u	ıg/m
			Value		

Table 2.1 Standarus IVI Some I Incipar I Onutants	Table	2.1	<b>Standards</b>	for some	Principal	<b>Pollutants.</b>
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Carbon monoxide	8 hours	9ppm(10mg/m <sup>3</sup> )		
	24 hours		0.09ppm	35 ug/m <sup>3</sup>
Particulate matter	Daily			10-100 ug/ m <sup>3</sup>
PM(10)	24 hours	150 ug/m <sup>3</sup>	50 ug/m <sup>3</sup>	$70 \ \mu/g/m^3$
Particulate PM	24 hours	35 ug/m <sup>3</sup>	25 ug/m <sup>3</sup>	
(2.5)				

Source: US- EPA,2004; Ghana- EPA, 2015; WHO, 2005)

Table 2.2: Occupational Exposure Limit Value (OELV) for Carbon Monoxide and PM

<b>Reference Period</b>	Parts Per Mill	ion (ppm)	mg/m <sup>3</sup>	
	HAS	OSHA	HAS	OSHA/EPA
8 hour	20	50	23	
15 minute	100		115	
8 hours (Daily)	PM 10			10–100 μg/m <sup>3</sup>
8 hours (Daily)	PM 2.5	- X-P		

Source:(HSA, 2007; OSHA,2003, Ghana EPA, 2015)

## 2.5 Pollutants from Burning Biomass Fuel and Scrap Tyre.

Domestic and commercial uses of biomass fuel and scrap tyre burning pose serious health effects to both attendants and people living in the communities. There are a lot of chemical reactions that take place during the burning process which emits poisonous pollutants such as CO, PM, nitrogen dioxide, Sulphur dioxide, etc. Respiratory and cardiovascular disease form these activities has been established (Amigun *et al.*, 2008; Nagar *et al.*, 2014; Kim *et al.*, 2015; Shadow, 2016).

#### 2.5.1 Carbon Monoxide

Carbon-monoxide is a potentially fatal, unscented, tasteless, and no color gas that results from inadequate burning of carbon -containing fuel. The prime source of atmospheric carbon monoxide accounting for virtually 90% is exhaust from gasoline engines, whiles bonfires, forest fires and waste treatment and disposal processes are also the remaining 10% contributing a large part (Urbanski et al., 2008). Carbon monoxide is nonpolar and therefore lipophilic diffuses easily when inhaled across pulmonary epithelium and are dangerous to human health because it causes a reduction in the oxygen carrying capacity of the blood, resulting in fatigue, headaches, respiratory problems, and in some cases even death (Lipfert et al., 2008; Shadow, 2016). Acute and chronic carbon monoxide (CO) exposure can produce a varied diversity of non-specific clinical features, all of which mimic other pathologies (Clarke et al., 2012). Evidence suggests that CO toxicity is normally missed by healthcare professionals (Wright ,2002; Clarke et al., 2012; Hanley and Patel, 2019). The emergency departments (EDs), under the Department of Health in England and Wales, has recently estimated 4000 people/year as diagnosed with CO poisoning but this figure does not include those whose diagnosis is missed (Department of Health, 2011). The annual global emissions of CO into the atmosphere is estimated to be as high as 2,600 million tones, of which about 60% are from human activities and about 40% from natural processes (Paul, 2008). CO is such poisonous that, People may sometimes die from exposure —low levels of carbon monoxide (CO) without implicating other toxic agents (Nelson, 1998). The noxious effects of CO become evident in organs and tissues with high oxygen depletion such as the brain, the heart, exercising skeletal muscle and the developing fetus. Despite the proof that burning biomass fuel and scrap tyre causes negative health impacts, many individuals in developing nations are still using them in view in their daily life with Ghana.

#### 2.5.2 Mechanism of Action of Carbon Monoxide

Carbon monoxides get into the body through the lungs and are successively engrossed by the blood. It then bonds with hemoglobin located in red blood cells. CO absorption in the human system is very swift such that, the capillaries' actions remain low (Prockop and Chichkova, 2007). CO and oxygen compete for a similar binding site on the hemoglobin molecule. CO inhibits the quantity of oxygen reaching these sites, and ends up binding to the hemoglobin molecule located at this site. This action results in the formation of carboxyhemoglobin (COHb). At the cellular level, carbon monoxides bind with heme-proteins such as myoglobin, cytochrome oxidase, mixed-function oxidase (cytochrome p- 45), tryptophan oxygenase, and dopamine (Nelson *et al.*,1998). The protein, mostly likely to be inhibited at a relevant level of COHb is myoglobin, which abounds in skeletal muscle and the myocardium. By this action, CO weakens the capacity, in the process known as allosteric binding.CO intoxication impairs the capacity of hemoglobin to release oxygen at tissue delivery sites (Nelson et al., 1998). Therefore, with a high concentration of CO ends –up disrupting cell actions specifically (Nelson et al., 1998). Despite the fact that all tissues are defenseless to carbon monoxide, through hypoxic or potentially non-hypoxic mechanisms (Carratu et al., 1993, 2000a, 2000b; De salvia et al., 1995; Lopez et al, 2003, 2008).

#### **2.5.3 Particulate Matter**

Particulate matter (PM) is well-defined as the sum of all solid and liquid particles suspended in air, of which many are hazardous. This compound mixture contains for instance pollen, dust, smoke, soot, and liquid droplets (; Vallero, 2014). Currently ambient air particulate pollutants are under intensive epidemiological and toxicological investigation (Shadow, 2016). Particulate matter (PM) pollution is the most health a relevant pointer of urban air quality and is widely used in setting air quality guidelines globally (Petkova *et al., 2013*; Cohen *et al.* 2000). particulate matter is usually categorized and studied according to particle size (Petkova *et al., 2013*; Cohen *et al.*, *al., 2000*).

2013). Particulate matter can be seen in two constituent's inhalable particles with diameters less than 10  $\mu$ m (PM10), —finel particles with diameter less than 2.5  $\mu$ m (PM2.5), and later on —ultrafinel particles with a diameter of 0.1  $\mu$ m or less (Petkova *et al.*, 2013; Shadow, 2016). The health effect of Particulate Matter is indeed huge and therefore harmful to human health, but more emphasis is given to the exposure of PM2,5 in particular, as increasing evidence connects PM2,5 with multiple cardiovascular, respiratory and cardiopulmonary effects. (Goldberg *et al.*, 2001; Janssen *et al.*, 2002). Anthropogenic and natural sources, such as soil or road dust, vehicle exhaust, biomass combustion, sea salt, forest fires, windblown dust, harmattan dust, industrial emissions, cooking, heating, agriculture, wood burning, power plants, heavy-duty diesel engine, construction, and demolition, etc are associated with particulate matter. They mostly arise from the combustion of fossil fuels, tyre and brake wear, and re-suspended dust (Catalano, & Kelsh, 1996; Buzorius *et al.*, 1999; EPA, 2004). A significant body of evidence from both developed and developing countries have proven that particulate matter causes a wide range of short- and long-term adverse health effects ( HEI 2000; Krewski *et al.*, 2004; Dominici *et al.*, 2006; Roger *et al.*, 2011).

## 2.5.3.1 Mechanism of Action of Particulate Matter

Particulate matter is Smaller particles that are deposited more in the lungs, where they can penetrate tissue or be absorbed directly into bloodstream (Pektova *et al.*, 2013). According to Salvi and Holgate, 1999, the human lung with a surface area of  $40\pm120m2$  is continually exposed to between 10 000 and 20 000 L of ambient air daily. The lungs have its mechanism of dealing with larger and smaller particles that may find its way into the surfaces such as mechanical removal and biochemical neutralization. Larger particles deposited on the conducting areas, for instance, the trachea and bronchi are propelled upwards by the mucociliary clearance mechanisms into the throat and are swallowed. Also smaller particles which reach the oxygen-absorbing area of the lung are removed by scavenging cells called macrophages, which carry the phagocytosed particles up to the airways towards the mucociliary clearance system (Salvi *et al.*, 1999). Despite the ability of the lungs to deal with particles, some amount still finds its way in the lungs due to the overwhelming of these defense mechanisms by either particle number overload or by the inherent toxicity of the particle. For instance, a person with compromised lung functions such as defective mucociliary clearance or abnormal immune function, even a small particle load may be adequate to produce harmful effects on the lungs. Several studies have proven the health effects of particulate matter exposure on individuals with health outcome indices, including mortality, chronic bronchitis, respiratory tract infections, exacerbations of asthma, ischaemic heart diseases and strokes (Dockery *et al.*,1993; Pope, 1995; Kaiser, 1997; Rehfuess *et al.*, 2011).

## 2.6.1 Health Effects of Exposure to Carbon Monoxide

The venomous and destructive properties of carbon monoxide (CO) have been known for an extensive length of time; old Greeks and Romans used the gas for execution purposes (Ganong, 1995). It was named as the silent killer because of its capacity of not having color, scent, or taste, formed from fragmented ignition of hydrocarbons and other carboncontaining compounds. A research undertaken by Lin, Krishnaswamy and Chi (2008) revealed that, if inhaled at low levels, CO can lead to dizziness, nausea, headaches, weakness and can also be harmful in elevated levels. Epidemiological investigations have proven that biomass fuel causes the following health effects asthma, lung cancer, tuberculosis, cardiovascular disease, cataracts, visual deficiency, low birth babies and mortality as a results of CO emissions during burning (Albalak *et al.*,1999; Bruce *et al.*,200; Warwick and Doig, 2004). An investigation by Goldstein (1997) established that, CO was among the main source of death through poisoning in the united states (US) from 1979-1988. Also according to Rathore and Rein (2016), a total of 438 lives were claimed annually as of late 11999-2012l in the US alone. A study was done by Rathor *et al.* (2016) reviewed that, Solid fuel (wood, scrap tyre, charcoal, and coal) cause

about 3.5 million unexpected losses in a year with a further 0.5 million outdoor air pollution deaths being ascribed to emissions from household cooking.

#### 2.6.2 Carbon Monoxide and Cardiovascular Diseases

Many types of research have been conducted to determine the negative impacts of CO poisoning to cardiovascular diseases, prior to exposure from the use of biomass fuel. A research conducted by Liu et al (2018, p. e12) found that "strong proof exists of the connection between short-term carbon monoxide exposure and enhanced cardiovascular disease mortality, particularly coronary heart disease mortality." A comparable study by Milojevic and colleagues on the Short-term effects of air pollution on a range of cardiovascular events in England and Wales and discovered that there were no associations between carbon monoxide and cardiovascular disease (Milojevic et al., 2014). The difference in the outcome of the study may perhaps be as a result of the limited size of the population and different subtypes of a cardiovascular disease considered. Lee et al (2015), envisaged the part carbon monoxide played regarding heart failure among old people in 1995. The group indicated that an intensification in surrounding CO levels prompted an upsurge in admission to health facilities with regards to heart failure. They suggested that surrounding CO could fuel existing wellbeing conditions. A comparable study by Morris et al (1995) reported positive and significant associations between increased risk of hospital admissions for cerebrovascular diseases and carbon monoxide exposure in the USA. Conversely, another study in Hong Kong by Tian et al (2015) observed negative associations between emergency hospital admissions for stroke and ambient carbon monoxide exposure. possibly because of the limited sample size of the study, the study population and different subtypes of a cardiovascular disease considered. Also, the differences in the study outcome may also be as a result of the difference in the year in which the studies were conducted and the availability of knowledge and technology at that time. A scientific report on a study by Reboul et al (2017) on risk of hospital readmission and cardiac mortality

increases with atmospheric pollution for patients with heart failurel discovered that, there is a high sensibility and vulnerability of individuals with cardiac disease to daily exposure to CO. A similar study by Lifespan (2008) reviewed that, I carbon monoxide also causes direct damage to the heart muscle, separate from the effects of oxygen deprivation, which reduces the heart's pumping capacity and permanently impairs cardiac functionl. Another study by Sveinung et al (2006) on CO exposure from smokers also reviewed that, that low dose CO exposure effects on the cardiovascular system seem to involve myocardial hypertrophy, but not atherogenesis. Rose et al (2017), commented that the World Health Organization recommends that levels greater than 6 ppm are hypothetically toxic over a longer period of time. Clinical manifestations of acute CO poisoning may be imprecise and may imitate multiple non-specific viral diseases carefully (Rose et al., 2019). Acute CO poisoning symptoms are disclosed in brain and heart susceptibility. IPatients can complain primarily about dizziness, headache, nausea, confusion, emotional responsibility, impaired judgement, clumsiness and syncope l(Burney, 1982; Myers and Synder, 1985; Ely et al., 1995). The table below indicates the level of concentrations and clinical manifestation in relation to CO exposure according to Burney,

1982; Myers and Synder, 1985; Ely et al., 1995.

CONCENTRATION	SYMPTOMS
35 ppm (0.0035)	Headache and dizziness within 6 to 8 hours of continual exposure
100 ppm	Slight headache in 2 to 3 h.
200 ppm	Slight headache within 2 to 3 hours; loss of judgment
400 ppm	Frontal headache within one to 2 h.

800 ppm	Dizziness, nausea, and convulsions within 45 min; insensible within 2 h.
1,600 ppm	Headache, tachycardia, dizziness, and
	nausea within 20 min; death in less than 2 h
3,200 ppm	Headache, dizziness and nausea in five to ten minutes. Death within 30 min
6,400 ppm	Headache and dizziness in one to two minutes. Convulsions, respiratory arrest, and death in less than 20 min
12,800 ppm	Unconsciousness after 2 to 3 breaths. Death in less than 3 min

# Table 2.4. Clinical Signs and Symptoms Associated with Carbon Monoxide Poisoning and Specific Percentage Carboxyheamoglobin (COHb).

MILD	COHb %
Mild Headache	0-30
Nausea/ Vomiting	A CONTRACT
Shortness of breath	
Dizziness	22/13
Blurred vision	
MODERATE	5 BADY
CWJ	SANE NO



*Source of information; (Kao et al., 2005; Dydek,2009)* Despite that some studies disapprove that CO has negative or no association with cardiovascular diseases, several studies have proven that chronic breathing of biomass smoke can result in irritation and oxidative pressure which will result in arterial blood pressure. (Goa a Uzoigwe 2013).

#### 2.6.3 Carbon Monoxide and Respiratory Diseases

Several studies have revealed that CO exposure poses serious health effects to respiratory systems such as pulmonary edema, aspiration pneumonia due to decreased oxygen saturation during CO intoxication (Kao *et al*, 2006). A study by Malvalankar *et al.*,1991 and Albalak *et al.*,1999,2001 reported that biomass combustion increases the dangers of acute lower respiratory infection in children and chronic obstructive pulmonary disease in adults.

According to Tian et al., 2014 short-term exposure to ambient carbon monoxide was associated with a decreased risk of hospitalization for Chronic Obstructive Pulmonary Disease (COPD) and further went on to conclude that, carbon monoxide exposure offers some acute protection against exacerbation of OCPD. This implies that although CO has a health effect on the respiratory system, it is not a direct danger of certain respiratory diseases being hospitalized. The United Nations (1998) under the United Nations International Children Emergency Fund discovered that Central Region of Kenya had a predominant of acute respiratory infection due to overcrowding of families in confined spaces who uses biomass fuel as their source of energy for cooking and warming foods especially in the cold season. A comparable study in Nigeria have also shown that smoke from biomass activities in communities is associated with pneumonia and bronchiolitis especially in children (Sofoluwe, 1968). The above reviews clearly show that the activities of biomass pose a significant effect on the respiratory systems in comparing to electricity and gas in terms of CO poisoning. Research by Dasgupta et al. (2004) exhibited indoor air contamination is linked to an increase in the danger of acute respiratory infection, OCPD and eye infection, for instance, cataract. WHO (2008), reported that more than one-third of all child demises causes via air pollution happens in the African Continent. This, therefore, makes it critical that local studies should be taken up to prove the hypothesis made by WHO.

## 2.6.4 Carbon Monoxide and the Eye

Studies have well documented that both acute and chronic exposure to CO gas poses serious health effects on the human eye (WHO, 2004; Peabody, Furr, and Ditmetaroj 2013; Dias *et al.*, 2016). According to Peabody, Furr, and Ditmetaroj (2013), the brain then eyes are the part mostly at-risk upon exposure CO due to its larger Oxygen demands for its structures? According to a study done on Carbon Monoxide Exposures by United State (2000-2009) recorded that, nearly 70,000 Cases of CO poisoning have been reported to the Eye Clinic with

the most prevalent symptoms of headaches and nausea. Comparable studies also showed that blurred vision, photophobia, and diplopia can also be associated with CO exposure (Hudnell and Benignus, 1989; Fielding *et al.*,2010; Hampson and Weaver, 2011). Other studies have shown that delayed symptoms of CO exposure in chronic patients may lead to focal edema or demyelination within the cerebral white matter (Chu *et al.*,2004; Lo *et al.*,2007). Despite the numerous research supporting the fact that CO has a significant effect on the eye with some specific diseases of the eye, Vision loss and other adverse visual effects due to CO poisoning are considered rare (Peabody, Furr, and Ditmetaroj ., 2013). Also, an experiment was demonstrated to determine whether CO functions as "an significant vascular paracrine factor and plays a part in the regulation of blood flow in several tissues" and the findings demonstrate that retinal and choroidal blood flow during CO inhalation increases.

(Dias et al., 2016).

#### 2.7.1 Particulate Matter and its Associated Health Effects

The significance of air, a mixture of gases and small solids and liquids for preferment of metabolism, good health and hence sustenance of plants and animals' life cannot be underrated (Owusu-Boateng *et al*, 2016). Most particles in the atmosphere including particulate matter (PM) (a mixture of solids particles and liquid droplets suspended in the air) is one of the air pollutants enlightened at different stages of industrial and local activities in most communities in developing countries (Dotse et al., 2012). The lifetime of PM10 is from minutes to hours, and its travel distance varies from < 1 km to 10 km (Owusu-Boateng et al, 2016). The health effects of PM exposure have been documented on both short and long term exposure (WHO, 2004; Dotse et al., 2012). Due to the prevalence of PM health effects recorded in developing countries PM10 and PM2.5 have been recorded by the World Health Organization (WHO) in addition to interim target concentrations for use by developing countries in measuring
development towards the guideline concentrations (WHO, 2006). According to a study carried out in Ashaiman Accra by Dotse et al (2012) revealed that PM

10 and PM 2.5 levels were higher than the Ghana Environmental Protection Agency (Ghana EPA) guideline value (70.0:g/m3 for 24 hour average and 50: g/m3 yearly average) even though Ghana EPA is yet to set a guideline value for fine particulates (PM2.5). All these guidelines there to regulates the levels of PM to reduce the health the effects of PM on our health. According to Owusu-Boateng (2016),I Historically, in many episodes of air pollution, the connection between PM10 and mortality has been created, such as in Belgium (1930), Pennsylvania (1948), London (1952), New York (1953) and London (1962), where the amount of fatalities attributed to air pollution was 63, 20, 4000, 200 and 700, respectively. Another study has also demonstrated the association between PM10 air pollution and cardiopulmonary and lung cancer mortality (Pope et al., 1995). From the above review, it can be concluded that Several studies have demonstrated the relationship between low or high concentrations of PM10 and PM2.5 increases in health effects and mortality.

#### 2.7.2 Particulate Matter and Cardiovascular System

Many research has been conducted to determine the negative impact of PM to cardiovascular diseases, prior to exposure from the use of biomass fuel and other human and industrial activities (Brook *et al.*, 2010; Pražnikar *et al.*, 2012; Chiang *et al.*, 2015). A study was done by Pope *et al* (1996) to test for association between PM10 air pollution and cardiopulmonary and lung cancer mortality and discovered that, PM10. PM2.5 was associated with a 36% increase in death from lung cancer and 26% in cardiopulmonary deaths. A comparable study by Ostro (2004) confirmed the association between PM10 levels with Rapid Infant Disease Syndrome (SIDS). Another recent study done by Loxham and Nieuwenhuijsen (2019), also documented that, exposure to PM is associated with cardiovascular disease (eg. heart disease, myocardial infarction, and stroke) also with asthma, lung cancer, recently type 2 diabetes, dementia and

loss of cognitive function and a major risk factor for mortality and morbidity. Newby *et al* (2014) also emphasized the abundance of evidence that air pollution contributes to CVD and associated mortality. Comparable studies prove that Chronic exposure levels of fine particle matter impair vascular function, which can lead to arterial hypertension, myocardial infarction, stroke, and heart failure (Brook *et al.*,2010; Münzel *et al.*,2017). Further studies by Chen *et al.*,2015 discovered that, —exposure to PM2.5 by 10mg/m3 leads to an increase of systolic and diastolic blood pressure by 1–3mmHg and is associated with a hazard ratio of 1.13 for the development of arterial hypertensionl. The average life expectancy of the European Union is 8.6 months lower due to exposure to PM produced by human activities (WHO, 2009). Clinical studies established that PM exposure is a key cause of the upsurge in hospitalization for Heart diseases (Poloniecki *et al.*,1997). Wellenius *et al* (2005); von Klot *et al* (2004) and Chang *et al.* (2015) have also documented that, upsurge in PM2.5 and PM 10 results in high risk of hospitalization for heart failure and cardiac arrest.

#### 2.7.3 Particulate Matter and Respiratory System

Several studies have well established the link concerning upsurge levels of air pollution and mortality rates from respiratory diseases (Dockery *et al.*, 1993; van der Wal and Janssen, 200; McCreano *et al.*, 2007). Exposure to air pollution is connected with substantial deficits in respiratory growth over a period, leading to deficits in lung function at younger age. (Gauderman *et al.*,2004; Gauderman *et al.*,2007). Comparable research also established that PM to be significantly related with emergency hospital visits due to asthma and respiratory tract infection (Schwartz, 1992; Sugiri *et al.*, 2006; D'amato *et al.*,2010). Other studies in addition revealed PM exposure to causes an exacerbation of existing lung conditions subsequent in acceleration of disease and death (Ling and Eeden, 2009). A mixed-method study also reviewed that, both indoor and outdoor studies show the frequent occurrence of wheezing and deterioration of lung function (Liu, 2018). A study carried out by Dunea *et al.*, (2016) in

two Romanian cities demonstrated that the prevalence of asthma in children is increasing at a rate of 8–11% per year. A comparable study by Zanobetti *et al.*, (2006) and Dominici *et al.*, (2006) documented that the incidence rate of respiratory illnesses increased by 2.07%, while hospitalization rates increased by 8%, with daily PM2.5 rising by 10  $\mu$ g / m3. From the above reviews, it can be said that about 90% of the literature support that indeed PM 10 and 2.5 causes a serious respiratory disease.

#### 2.7.4 Particulate Matter and the Human Eye.

Eye is the most vulnerable organ to atmospheric and environmental abuses. Though, naturally, eyes are organized to protect themselves from foreign objects such as wind, dust and very bright light. The purpose of the eye is for vision, therefore, it needs to remain open always. Chronic exposures to noxious pollutants present in the air, water as well as in soil can damage the eye in various ways (Gupta and Muthukumar, 2018). Some studies have proven that exposure to PM causes several eye effects (Gupta and Muthukumar ,2017; Tan et al., 2018). A study by (Tan et al., (2018) show that, the symptoms of dry eye include redness, eye dryness, severe pain, and itching. It can further develop into corneal ulcers, decreased eyesight and even blindness. A comparable study by Mimura et al (2014) surveyed patients with acute conjunctivitis from May to October 2012 and found that the number of patients with acute conjunctivitis was increased with a higher level of PM2.5. Another study by Torricelli et al (2013) also supported that tear secretion and epithelial obstruction function of the eye are less influenced by PM 2.5. Even though there have not been many studies carried out on the impact of PM on the few available data supports that, PM is associated with eye irritations and eye WJSANE problems.

#### **CHAPTER THREE**

#### **METERIALS AND METHODS**

#### **3.1 Introduction**

This chapter describes the research methodology which comprises the research design, study area, population, sampling technique, sample size, data collection, data analysis, instrumentation and ethical consideration.

#### 3.2 Study Area and Location

This research was carried out in the metropolis of Kumasi, Ghana. Kumasi is a Ghana metropolis and the Ashanti region's capital city. It is located in a rainforest region 30 kilometers north-east of a Crater Lake, Lake Bosomtwe. It lies 6° 41' 0" North, 1° 37' 0" West.

Kumasi metropolis is known to have a lot of development ongoing in the area of infrastructure development, tertiary institutions, secondary schools, junior high schools, estate, hospitals, and industries. Kaase community is a suburb of Kumasi metropolis which is 10 kilometers westwards from the center of the regional capital. It is both a residential and industrial area under Asokwa Municipal Assembly with many commercial activities such as Ash foam production and wholesale, abattoir activities, quarrying site, and Finance company making it a place suitable for the study. Due to the many industrial and local activities in the community, therefore, making it the best study site in terms of air pollution.

#### **3.3 Study Population and Sample Size**

According to Tetteh of the department of the Kumasi Metropolitan Assembly, the population as of 2015 was 1,730,249 which comprises of 826,479 males and 903,770 females representing 36.2% of the Ashanti region total population making it the second-largest city in Ghana. Kaase community is a suburb of the Kumasi metropolis which is 10 kilometers westwards from the center of the regional capital. Itis both a residential and industrial area under Asokwa Municipal Assembly. Kaasi Abbatoire has about 30 local butchers who work at the abattoir extension using the biomass fuel.d Kaasi Community takes about 50 market sellers and vendors as well as residents within are the defined 100 meters yard away from the source of pollution. There are about 15 houses within the catchment area. Their main occupation is butchering work, buying, and selling of food and animal products. Sample size of 95 took part in the study of which 74 were clustery sampled from the community and 21 form the local butchers and assistant .

#### 3.4 Study Design

Cross-sectional analytical study design was used to investigate air pollution in Kaase- Kumasi.

#### 3.5 Inclusion Criteria

The following were the criteria for inclusion;

- All Local butchers and assistants available at the time of study and who works at the open space
- Butchers and assistants above 18 years of age and worked at the open space
- Butchers and assistants who worked at least four times in a week and were exposed to the burning activities
- Butchers and assistants who used biomass fuel in meat singe
- Community members above 18 years staying within 100 meters away from the source of pollution at Kaase.

#### 3.5.1 Exclusion Criteria

The following was the criteria for exclusion;

- Customers who visited to buy meat
- Butchers and Assisting butchers less than 18 years
- Butchers and assisting butchers who absented themselves 3 times in a week

- The immigrant who has not stayed in the area for not more than one year
- Butchers and assistant who worked less than 8 hours each day

#### **3.6 Participants Recruitment**

A letter of introduction from the Department of Occupational and Environmental Health and Safety, College of Health Science, Kwame Nkrumah University of Science and Technology (KNUST) was send to the head of the abattoir where the study was conducted. This was to seek approval for the study to be conducted under their jurisdiction. Ethical clearance was sought from the Committee of Human Research Publications and Ethics (CHRPE), KNUST. Consent for participation in the study was sought from participants after explaining the rationale of the study to them and recruited after giving consent.

#### **3.7 Sampling Method**

Multistage sampling technique was employed and purposive sampling was used to select Kaase abattoir station, and Kaasi community. Cluster sampling was also used to select butchers and assistance involved in meat smoking activities as well as individuals who sells and stay within the 100 meters away from the source of pollution.

#### **3.8 Instrumentation**

Instrumentation is the set of instruments used to carry out research work. All data that were sued, was done using standardized instruments. The following are the instruments that were used;

Aeroqual Series 500 (S500) gas monitor, Aeroqual limited, 109 Valley Road, Mount Eden Auckland, New Zealand. This instrument was used to measure Carbon monoxide levels within the workspace and 100 meters away from the source into the community (Kaasi Community market).

- Environmental Particulate Air Monitor (EPAM), Model -7500, Environmental Device Corporation, 4 Wilder Drive Bldg.#15, Plaistow, NH03867, USA. This instrument was used for measuring fine particulate matter levels (PM 2.5,10) within the working space and 100 meters away from the source into the community (Kaasi Community Market).
- Questionnaire, was administered to capture participant information on; Demographic characteristics and health impacts (knowledge hazards on smoke, self -reported respiratory and cardiovascular signs and symptoms, hospital admissions and eye irritations).

#### 3.8.1 Procedure for Measuring Carbon Monoxide

The level of CO was determined by placing Aeroqual Series 500 (S500) gas monitor close by the fire to determine the gas level. The instrument was placed 1.5 meters away from the stove, placed on a height 1 meter above ground level and at a breathing statue (Padhi *et al.*, 2010). The monitor was set to capture exposure trends for every minute, for an 8 hours' period. The data was then transferred to a computer to give the average gas level for the 8hours period.

#### **3.8.2 Procedure for Measuring Particulate Matter**

The EPAM, Model -7500 is suitable for measuring fine particles. The gadget was set to Pm 2.5 and 10 independently for 8 hours' occupational exposure on two different days, each day recording a different parameter within the working space and the community. The gauge was set vertically with the rim of the funnel horizontally oriented. The whole gauge was placed on height 2 meters above ground level to keep it secure and upright with the rim 304.8 mm above the surrounding grass level a height chosen so that no rain splashes from the surroundings into the funnel. The geographic location within the working space and the community relative to the pollution sources was determined. A special function was selected from the main menu and the inlet inserted in the sensor head of the monitor. The filter cassette holder will be attached

to the sensor and the manual zeroed. The monitor was set to run and set to either continue the previous sampling or to overwrite the previous data. The date and time was set and the sampling rate selected to sample at every minute for 8 hours' occupational exposure limit.

#### 3.9 Data Analysis

The data collected was cleaned, imputed into software and then analyzed using the STATA version 15.0. Statistical tools include frequency and percentage. Descriptive statistics of means, standard deviation. A significant test for sample test, was done to compare mean values to both local and international stands concerning levels of CO and PM in the community and abattoir area. A regression analysis was conducted to predict which symptom is a significant predictor of CO and PM owing to extended exposure years. The significant test was set at 0.05 alpha level. Results were presented in tables.

#### **3. 9.1 Ethical Consideration**

The committee of the human research, publications and ethics and ethical approval at the Konfo Anokye Teaching Hospital, Kwame Nkrumah University of Science and Technology provided ethical approval of all procedures regarding the study after fulfilling the necessary requirements.

#### 3.9.2 Entry and Approval of Study Area

An introductory letter was taken from the Department of Occupational and Environmental Health, School of Public Health, Kwame Nkrumah University of Science and Technology and sent to the director Asokwa municipal to seek approval for the study to be conducted at the Kaase community. A letter from the Municipal was sent to the municipal environmental health department which was further sent to the abattoir area for approval. Subsequently, a copy of the approval letter from the committee of human research, publications and ethics board was send to them after the approval from the study site.

#### 3.9.3 Privacy and Confidentiality

The questionnaires were coded and respondent names were not required to complete the questionnaire. The questionnaire was conducted in separate locations with individual participants to ensure their privacy. The names of the participants were not mentioned in the study report and the information collected about the participants was kept strictly confidential between the researcher, the board of ethics, the supervisor, and the participants in the study.

#### **3.9.4** Compensation

Study participants were given free education about dangers of carbon monoxide and particulate matter exposure, free sachets of water and hand washing soap. This was made known to participants before they chose to take part in the study.

#### 3.9.5 Risk and Benefits

Aside from the time that was lost by study participant in answering the questionnaires, there was no risk or cost associated with participating in the study. Participants were not given any direct benefits.

#### 3.9.6 Voluntary Withdrawal

Participation in this study was entirely voluntary and participants chose not to answer any individual question or all the questions. Participants was given the opportunity to withdraw from the study if they wished. In the event of any withdrawal by a participant, all data gathered on the participant was discarded.

#### **3.7 Consenting Process**

Participants in this study were approached individually to explain the objectives of the study to them and their consent was sought. The decision to take part in this study was completely voluntary and refusal to take part did not affect the relationship between the participant(s) and the researcher. Furthermore, participants were made to sign a written consent form after a thorough explanation to them before they took part in the study.

#### 3.9.8 Data Storage and Usage

Data collected in this study was strictly for research purposes. The data was stored with passwords on electronic media and in safely locked boxes. Anonymity was ensured in dissemination of findings from this study since participants were not identified by their names.

#### **3.1 IMAGES FROM STUDY SITE**



**CHAPTER FOUR** 

#### RESULTS

#### 4.1 Socio- Demographic Characteristics of Respondents

#### **4.1.1 Demographic Characteristics**

From the survey conducted, 52.6% of the sample population were females while 47.4 % were males. The results further show that the majority of the respondents were within the ages group of 21-30 years, representing 31.6 % of the total sample size. This was followed by those within the age bracket of above 50 years, which accounted for 28.4%, next group 31-40 years, which accounted for 18.9% of the sample size and 41-50 years group 15.8% of the sample. Those below 21 years group accounted for the least sample size of 5.3% of the total sample size. The marital status analysis showed that the majority of the respondents were married which accounted for 50.5% of the sample population while the singles were 29.5%. Those with others were 14.7 % and the divorced was just 5.3 % of the total sample. Majority of them had Primary/Junior High school education, which accounted for 47.4% of the sampled population, not surprisingly; the Kaasi Community is an industrialized region and a municipal assembly with a majority of the sample population in the 12-30-year age group.

In addition, Non –formal education level accounted for 23.2% due to the higher percentage sample for those above 50 years of age group and were mostly market women, Senior High/ O-Level /A level, which also accounted for 21.1% and the least had Tertiary education which accounted for 8.4% of the sample population. Out of the total sample population of 95, 77.9% of the sample lives and work in the Kaasi Community whiles the remaining 22.1% of the sample work at the Abattoir area and are engaged in the use of biomass fuel eg. Car tyre and firewood for their activities. The hazards identified at the abattoir area. The most common hazards identified were Slips and falls, burns, Cuts, and pricks. Even though these hazards were present, workers were not much concern about it because its occurrences were low. Table 4.1 below the demographic details of respondents understudy

Characteristics	Number	Percent	Mean $\pm$ SD
Gender			
Male	45	47.4	
Females	50	52.6	
Age	5	5.3	
<21	30	31.6	
21-30	18	18.9	
31-40	15	15.8	$41.54 \pm 16.95$ 31-40
41-50	27	28.4	
>50			
Marital status	N. 11	12	
Single	28	29.5	
Married	48	50.5	
Divorced	5	5.3	
Others	14	14.7	
	50	2	L
Level of Education			+F3
No formal education	22	23.2	Z
Primary/Junior High school	45	47.4	2
Senior High /O-Level /A level	20	21.1	
Tertiary	8	8.4	
	177	3	
Place of work /resident	~~~		
Abattoir workers	21	22.1	15
Community workers/resident	74	77.9	12

#### Table 4.1: Demographic Characteristics of Respondents

#### 4.2 Duration of Potential Exposure to CO and PM in Years.

Results represented that in Table 4.2. Provides a summary of the duration of potential exposure to CO and PM. The results showed that a larger proportion of the respondents spent less than 5 years, which is 30.5 %, followed by, 5-10 years of 29.5%. The next group is 11-20 years

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making 20%, 21-30 years of 20% and the last group with a lower percentage of 9.5% yet spent more than 30 years of their lives in the community respectively. Table 4.2 below shows the duration of exposure to CO and PM in years in details of respondents understudy **Table 4.2**:

Duration of potential exposure to CC	O and	PM		
Duration		Number	Percentage	Mean ± SD
<5 years		29	30.5	
5-10 years		28	29.5	13.20±11.92
11-20 years		19	20.0	
21-30 years		10	10.5	
>30 years		9	9.5	

#### Duration of Exposure to CO and PM in Years

#### 4.3. General Knowledge about Exposure to CO and PM.

Table 4.3 below shows the level of knowledge the study populations know about the health effects of exposure to CO poisoning and PM. From the results, out of the 95 participants that were involved in the study, 82.1% know that the activities of car tyre burning and firewood used in smoking meat are hazardous to their health and 17.9% said they had no knowledge about the hazardous nature of their activities. Also, 67.3% of the sample said no health personnel from the community or municipal assembly has ever come to educate them on the dangers associated with the use of car tyres and firewood for meat preparation. Only 32.9% said there has been someone from the Municipal assembly who came round to educate them. The differences could be attributed to the individuals not present at the day, hour or week in which the exercise was carried out. Since the majority of the community members are market women, they would have traveled on that particular day. In addition, it is not easy to access the Abattoir due to the perception of the workers in that particular area. They think that your

coming to educate them is a treat to their job and place of revenue to cater for their families. Table 4.3 below shows the general knowledge of exposure to CO and PM in details of respondents understudy.

Table 4.3: General Knowledge about Exposure to CO and PM	И.	
Question	Response % (n)	
	Yes	No
General Knowledge about Exposure to CO and		
PM	2	
Do you know that smoke from the burning	82.1 (78)	17.9 (17)
activity is hazardous to your health?		
Any health personnel in this community come and educate you about the dangers of exposure to air pollution?	32.9(31)	67.3(64)
EIK &	77	3

#### 4.4 Daily Exposure Duration and Hospital Attendance

From the study, it was reported that 42.1%, thus (40) respondent work for maximum duration of fewer than 9 hours per day as their daily exposure duration, followed by 9-12 hours exposure representing 37.9% (36) of respondents and the last group exposed for greater than 12 hours accounting for 20.0% (19) of respondents. Concerning hospital admission, about 61.1% (58) of the respondent visited the hospital less than 3 times for the past 6 months, followed by 30.6%(30) of respondents visited 3-4 times and few of the respondent visited more than 5 times accounting for 7.4%(7).

Table 4.4 below show the daily exposure and Hospital attendance in details of respondents understudy.

#### Table

#### 4.4 Daily Exposure Duration and Hospital Attendance

Exposure duration and hospital attendance

	to this	air	Response %	(n)	
How long are you exposed	Z	Ν	<9 hours	9-12 hours	>12 hours
pollution in a day?	visited	the	42.1 (40)	37.9(36)	20.0 (19)
How frequently have you			<3 times	3-4 times	>5 times
hospital for the past 6 months?	2		61.1 (58)	30.6(30)	7.4(7)

### 4.5 The concentration of CO and PM within the Abattoir working space and Kaasi

#### Community.

In the Abattoir segment, the outcome details for PM 10 were 2.35 mg / m3 as the average concentration at 59.24 mg / m3 as the largest concentration followed by 4.82 mg / m3 as the minimum concentration and standard deviation. Also the level of community concentration was 1.19 mg / m3, with.11 mg / m3 being the largest concentration followed by.91 mg / m3 and the standard deviation of 0.08. The concentration of PM 2.5 in the Abattoir region was a mean of 1.50 mg / m3 with a peak concentration of 26.44 mg / m3 accompanied by a standard deviation of 1.08 mg / m3 and 2.91 respectively. The community also reported a mean concentration of 0.14 mg / m3 with a peak concentration of 0.68 mg / m3 accompanied by standard deviation of 0.91 mg / m3 and 0.06 Table 4.5 below shows the details for CO and PM levels in both the Abattoir area and the Community.

4.5 Details for CO and PM levels in both the Abatoir Area and the Comm
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	CO <mark>Con (Mean)</mark>	Std.	Min value	Maximum
	ppm	Deviation		Value
Abattoir area	5.78	5.32	.00	40.32

Table							
Kaasi Community	1.31		1.79	.00		11.05	
PM 10Con(mean)	µg/m3	Mg/m <sup>3</sup>		µg/m3	Mg/m <sup>3</sup>	µg/m3	Mg/m <sup>3</sup>
Abattoir area	2350	2.35	4.82	70	.07	59240	59.24
Kaasi Community	190	.19	.08	910	.91	110	.11
PM2.5Con(mean)	µg/m3	Mg/m <sup>3</sup>	V U	µg/m3	Mg/m <sup>3</sup>	µg/m3	Mg/m <sup>3</sup>
Abattoir area	1500	1.5	2.91	80	.08	26440	26.44
Kaasi Community	140	.14	.06	90	.09	680	6.8

\*CO= carbon monoxide and \*PM= particulate matter

## 4.6 Statistical Difference in Means between CO and PM levels in the Abattoir Against

#### the Community

Area.

One sample t-test was conducted to examine the extent to which difference in means was significant. The results of the test showed that the difference in means in terms of CO concentration of the Community and the Abattoir area was statistically significant (p<0.001). Thus, carbon monoxide levels in the Abattoir area was greater than that of the community. Table 4.6 below shows the statistical test of significance for the level of concentration between the Abattoir area and the community.

## 4.6 One-Sample Test for CO in the Community against the Abattoir working

and the second se			and the second s
Tested value 1.31pp	m AB	SH	
Variable	Mean Difference	(95% Conf. Interval)	P value
CO CM	4.465	4.846- 4.085	0.000

\*CO= carbon monoxide and \*p<0.05

#### Table

The results of the test showed that, the difference in means in terms of PM 10 concentration of the Community and the Abattoir area was statistically significant (p<0.001). Thus, PM 10 levels in the Abattoir area was greater than that of the community. For PM 2.5, the concentration in the community and the Abattoir area was also statistically significant (p<0.001). Thus PM 2.5 was also higher in the abattoir area than the community. Table 4.6 .2 below shows the statistical test of significance for level of concentration between the Abattoir area and the community respectively.

 Table 4.6.1 One-Sample Test for PM 10 and 2.5 in the Community Against the Abattoir

 Working Area.

Test Value $= 2.35 \text{ m}$	ng/m <sup>3</sup> AB		
Variable	Mean Difference	(95% Conf. Interval)	P value
PM 10 CM	-2.160	-2.1522.168	0.000
Test Value = 1.50 m	ng/m <sup>3</sup> AB	THE	3
PM2.5 CM	-1.361	-1.3561.367	0.000

\*PM= Particulate Matter \*p<0.05

#### 4.7 Statistical Difference in Means between CO and PM levels in the Abattoir Area, the

#### Community and compared with International standards.

One sample t-test was conducted to examine the extent to which difference in means was

significant. The results of the test showed that, the difference in means in terms of CO

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concentration of the Community, the Abattoir area compared with US-EPA 8 hours was statistically significant (p<0.001). Thus the concentration from this study was lower than that of USA-EPA daily exposure limit but was higher than WHO 24hours standard value . Table 4.7 below gives details of concentration of CO between community, the Abattoir area, and US-EPA.

Table 4.7 One-Sample Test for CO levels in the Abattoir area and Community against US-EPA and WHO Standard respectively.

Variable	Mean ± SD	(95% Conf. Interval)	P value
Tested value 9ppm US	S-EPA	1 m	
CO AB	5.775±5.320	5.394 - 6.155	0.000
CO CM	1.306± 1.791	1.164 - 1.449	0.000
Tested value 0.09ppm	WHO		
CO AB	5.775 ± 5.320	5.395 - 6.155	0.000
COCM	1.306±. 1.791	1.164- 1.449	0.000

\*CO= carbon monoxide and \*p<0.05

In addition, the results of the test for PM showed that, the difference in means in terms of PM 10 concentration of the Community and the Abattoir area against WHO standards were statistically significant (p<0.001). Thus, PM 10 levels in the Abattoir area, the community was far greater than that of the WHO 24 hour standard and Ghana EPA. For PM 2.5, the concentration in the community, the Abattoir area against WHO was also statistically significant (p<0.001). Thus PM 2.5 was also greater in the abattoir area and the community than the WHO concentration. Table 4.7.1 below shows the statistical test of significance for level of concentration between the Abattoir area, the community, and the WHO respectively.

# Table 4.7.1 One-Sample Test for PM10 and 2.5 levels in the Abattoir Area and Community against WHO Standard respectively.

Variable	Mean ± SD	(95% Conf. Interval)	P value	
Tested value 0.05 mg/n	n <sup>3</sup>			
PM10 AB	2.353± 4.815	1.922 - 2.785	0.000	
PM10 CM	.189 ± .085	.182198	0.000	
Tested value 0.025 mg/m <sup>3</sup>				
PM2.5 AB	1.499 ±2.914	1.234 - 1.765	0.000	
PM 2.5 CM	.139 ±.057	.134144	0.000	

\*PM= Particulate Matter and \*p<0.05

#### 4.8. Health Effects /Signs and Symptoms of CO poisoning and PM exposure among

#### Respondents

Research has shown that exposure to CO and PM can have a certain health effect on the person. The respondents of the study were asked to outline some of the health problems they experience from a list of some health conditions due to exposure to smoke over the years. The results showed that some of the major symptoms among the lot for CO were Headache

83.2% representing 79 respondent, followed by Muscular Weakness 74.7%, Dizziness 72.6%, Blurred vision 69.5%, and Chest pain 62.1% while the following conditions were not reported by respondents begins with Cardiac arrest, Seizures, and Coma. The following conditions were also less reported by respondents, Nausea /vomiting, shortness of breath, confusion, palpitation, hypertension, Respiratory arrest, and myocardial ischemia. Under the groupings, none of the severe symptoms was among the major reported conditions. For PM exposure, the major condition reported was Eye Irritations accounting for 91.6% making up 87 of the sample of the respondent. It was followed by Respiratory tract Infection 71.6% and the less reported condition was Hypertension, Type two diabetes, Myocardial infarction, Loss of cognitive Function, Asthma. None of the respondents reported anything about stroke. Table 4.8 below gives details of the sign and symptoms reported by respondents due to exposure to CO and P

### 

	Response %	) (n)
Signs and Symptoms of CO Exposure	_	
	Yes	No
Mild		
Nausea/ Vomiting	49.5(47)	50.5(48)
Shortness of breath	43.2(41)	56.8(54)
Dizziness	72.6(69)	27.4(26)
Headache	83.2(79)	16.8(16)
Moderate		
Blurred vision	69.5(66)	<u>30.50(</u> 29)
Confusion	15.8(15)	<u>84.</u> 2(80)
Chest pain	62.1 (59)	37.9(36)
Muscular Weakness	74.7(71)	25.3(24)
Respiratory arrest Seizures	2.1(2)	97.9(93)
/ Coma	0.00(0)	100(05)
/ Coma	0.00(0)	100(95)
/ Coma Signs and Symptoms of PM Exposure	0.00(0) Response	100(95) %(n)
/ Coma Signs and Symptoms of PM Exposure	0.00(0) Response Yes	100(95) %(n) No
/ Coma Signs and Symptoms of PM Exposure Hypertension	0.00(0) Response Yes 20.0(19)	100(95) %(n) No 80.0(76)
/ Coma Signs and Symptoms of PM Exposure Hypertension Type two diabetes	0.00(0) Response Yes 20.0(19) 5.3(5)	100(95) %(n) No 80.0(76) 94.7(90) 96.8(92)
/ Coma Signs and Symptoms of PM Exposure Hypertension Type two diabetes Myocardial infarction	0.00(0) Response Yes 20.0(19) 5.3(5) 3.2(3)	100(95) %(n) No 80.0(76) 94.7(90) 96.8(92) 94.7(91)
/ Coma Signs and Symptoms of PM Exposure Hypertension Type two diabetes Myocardial infarction Loss of cognitive Function	0.00(0) Response Yes 20.0(19) 5.3(5) 3.2(3) 4.2(4)	100(95) %(n) No 80.0(76) 94.7(90) 96.8(92) 94.7(91) 100(95)
/ Coma Signs and Symptoms of PM Exposure Hypertension Type two diabetes Myocardial infarction Loss of cognitive Function Stroke	0.00(0) Response Yes 20.0(19) 5.3(5) 3.2(3) 4.2(4) 0.0(0)	100(95) %(n) No 80.0(76) 94.7(90) 96.8(92) 94.7(91) 100(95) 85.3(81)
/ Coma Signs and Symptoms of PM Exposure Hypertension Type two diabetes Myocardial infarction Loss of cognitive Function Stroke Asthma	0.00(0) Response Yes 20.0(19) 5.3(5) 3.2(3) 4.2(4) 0.0(0) 14.7(14)	100(95) %(n) No 80.0(76) 94.7(90) 96.8(92) 94.7(91) 100(95) 85.3(81) 28.4(27)
/ Coma Signs and Symptoms of PM Exposure Hypertension Type two diabetes Myocardial infarction Loss of cognitive Function Stroke Asthma Respiratory tract Infection	0.00(0) Response Yes 20.0(19) 5.3(5) 3.2(3) 4.2(4) 0.0(0) 14.7(14) 71.6(68)	100(95) %(n) No 80.0(76) 94.7(90) 96.8(92) 94.7(91) 100(95) 85.3(81) 28.4(27) 8.4(8)

4.9 Health Effects Experienced by both the Community and the Abattoir workers based				
Cardiac arrest	0.0(0)	100(95)		
Myocardial Ischemia	1.1(1)	98.9(94)		
Hypertension	20.0(19)	80.0(76)		
Palpitations	45.3(43)	54.7(52)		
SEVERE				

#### on CO exposure with Respect to Years of Exposure.

A Linear regression analysis was further conducted to assess the duration of exposure (work experience) to CO exposure for the understudied respondent. The regression model, which uses Work experience to predict the level of CO was found to be statistically significant. From the test, it was determined that Blurred vision was the major indicator for CO posing, and this was statistically true (p-value <0.049). The R-squared value of 25 percent means that Blurred vision is a 25 percent predictor of CO posing in the exposed population. Table 4.9 gives details of which symptom is associated with CO exposure.

A similar procedure using linear regression analysis was also done for the PM to assess the duration of exposure to PM. The regression model, which uses Work experience to predict the level of PM was found to be statistically significant. The outcome of the test indicated that both Asthma and Hypertension were the major indicators for PM exposure and this was statistically true (p-value <.014 and .037) respectively. Table 4.9 gives details of which symptom is associated with PM exposure.

# Table 4.8.1 Linear Regression showing which symptom is a Major Predictor of CO poisoning.

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Coefficients	2	5 BA			
Model	Unstandardized Coefficients		Standardized	Т	Sig.
			Coefficients		
	В	Std. Error	Beta		

1	(Constant)	.608	3.044		.200	.842
	Nausea/ vomiting	330	.296	129	-1.116	.268
	Shortness of breath	442	.277	171	-1.594	.115
	Dizziness	.198	.341	.069	.581	.563
	Headache	.828	.422	.243	1.962	.053
	Blurred vision	606	.304	219	-1.997	.049
	Confusion	.146	.410	.043	.355	.723
	Chest pain	.693	.364	.264	1.903	.061
	Muscular Weakness	155	.321	056	483	.630
	Hypotension	328	.347	128	944	.348
E	Myocardial Ischemia	521	.359	163	-1.452	.150
	Cardiac arrest	1.067	1.290	.085	.828	.410
	Respiratory arrest	.421	.939	.047	.448	.655

\*\* Significant at the 0.005 level

Model Utility

 $R^2 = .251$ 

Adjusted  $R^2 = .141$  Standard

error =1.1886

a. Dependent Variable: Work experience

 b. Predictors: (constant), Nausea/ vomiting, Shortness of breath, Dizziness, Headache, Blurred vision, Confusion, Chest pain, Muscular Weakness, Hypotension, Myocardial Ischemia, Cardiac arrest, Respiratory arrest.

Table 4.8.2 Linear	<b>Regression showing</b>	which symptom i	s a Major predict	tor of PM
Exposure.				

Coefficients						
Model		Unstandardized		Standardized	Т	Sig.
L L		Coefficients		Coefficients		
		В	Std. Error	Beta	_	
1	(Constant)	3.844	2.369		1.623	.108
	Hypertension	687	.324	215	-2.119	.037
	Type two diabetes	364	.569	064	640	.524
ç	Myocardial infarction	-1.403	.723	192	-1.941	.056
	Loss of cognitive Function	.781	.629	.123	1.243	.217
	Asthma	.900	.359	.250	2.508	.014
	Respiratory tract Infection	.106	.317	.037	.335	.738
	Eye Irritations	071	.495	016	144	.886

\*\* Significant at the 0.005 level

Model Utility

 $R^2 = .180$ 

Adjusted  $R^2 = .114$  Standard

error =1.2133

a. Dependent Variable: Work experience

b. Predictors: (constant), Hypertension ,Type two diabetes, Myocardial infarction, Loss of cognitive Function, Asthma, Respiratory tract Infection, Eye Irritations.

#### **CHAPTER FIVE**

#### DISCUSSION

#### 5.1 Scio-demographic Characteristics of Respondents to CO and PM Exposure

Gender plays a vital role when it comes to our traditions especially to household choices such as cooking, washing and some other professions such as trading and minor retail activities. This makes such a group of gender mostly present at home in Ghana. Generally, women are engaged in these activities more than men. The descriptive statistics from the research revealed that 52.6% of the sample population were females while the remaining 47.4% were males. Even though the values are, very close it was because the workers at the Abattoir area were only men, which is about one-third of the sample, and the remaining were from the community. Women ended up leading because of the role women play at homes and within the community. Thus, the issue of CO and PM exposure may be more problematic for women than men. This is in agreement with early research by (Ezzati, and Kammen, 2002). This study also focused on women and children being exposed due to their responsibilities at home.

Duration of potential exposure has a serious impact on health. How long one is exposed to CO and PM has a consequence on their health. The longer your exposure duration, the higher your chances of developing a health problem. From the survey, it was discovered that 30.5% of the sample population was exposed less than 5 years representing the majority of the population. Followed by, 5-10 years of 29.5%. The next group is 11-20 years making 20%, 21-30 years of 20% and the last group with a lower percentage of 9.5 spent more than 30 years of their lives in the community respectively. Few populations of the study had been exposed for more than 30 years. Even though few percentages were exposed for longer years, research have shown that both acute and chronic exposure have health effects (Tian et al., 2014; Clarke et al., 2012; Uzoigwe et al., 2013). All these studies spoke about both the health effects associated with both acute and chronic effect of being exposed to CO and PM which is also in agreement with this study.

A further statistical test was run to determine which condition or symptoms are the major predictor for CO and PM exposure concerning years of exposure. A regression model was used to test and the findings were Blurred vision was the major indicator for CO posing, and this was statistically true (p-value <0.049). Concerning PM exposure, Asthma and Hypertension were the major indicators for PM exposure and this was statistically true (pvalue <.014 and .037) respectively. Thus the longer you are exposed to CO and PM, the higher your risk of developing blurred vision, asthma and Hypertension.

#### 5.2 Hospital Admission on Potential Exposure to CO and PM among Respondents

Another point to consider is hospital admission and rate of the visit. The rate of once visits hospital and admission rate has a consequence on exposure to CO and PM aware or not aware. From the survey, it was discovered that 58 of respondents visited and were admitted or visited the hospital less than 3 times for the past 6 months, followed by 30 of respondents visited 3-4 times and few of the respondents visited more than 5 times accounting for 7 respondents. Natural in Ghana, the majority of the populace do not visit the hospital rather prefer over the counter medication so for this respondent to visit the hospital more than twice and upward in six months is of greater interest and some even visited or admitted more than five times with the symptoms and condition as highlighted in this study. This outcome is also in agreement with research work by (Morris et al 1995; Poloniecki et al., 1997; Reboul et al., 2017). All these studies spoke about the association between hospital admission/ visit due to potential exposure to CO and PM. Also under daily exposure rate, it was revealed that, 42.1% thus (40) respondent work for maximum duration of less than 9 hours per day as their daily exposure duration, followed by 9-12 hours exposure representing 37.9% (36) of respondents and the last group exposed for greater than 12 hours accounting for 20.0% (19) of respondents. Some studies have confirmed that daily exposure to Co and PM thus have serious health implication and this is not different from this study where the majority of the sample population are exposed

more than 9 -12 hours upward a day. (Zanobetti *et al.*, 2006; Dominici *et al.*,2006). These studies emphasize that daily exposure really has health consequences.

#### 5.3 Level of CO and PM in the Environment of both the Abattoir Area and the

#### Community.

The first objective of this study was to evaluate the level of CO and PM in the community environment as well as local butchers working space within the Abattoir area. The finding showed that most of the sample population stay and worked in the community. Almost all the sample from the local abattoir area were exposed directly to the smoke for a maximum of 8 hours a day. Thus to say they are not residents in the community they only come to work, close and leave as well as some workers in the community who are not residents. The findings from this study revealed that the amount of PM 10 and PM 2.5 concentrations were 2350 µg/m3 in the Abattoir area and 190  $\mu$ g/m3 in the community which shows that there is the presence of PM 10 which is capable of causing a health effect either by acute or chronic exposure for PM 10. In addition, the level of PM 2.5 for both Abattoir area and community were 1500 µg/m3 and 140 µg/m3 respectively showing the presence of PM in these areas. Concerning PM 2.5, any little amount of concentration is very dangerous to our health. This study is in agreement with a study by Ho et al. (2002). His study revealed that any activity such as car tyre burning, cooking, and industrial work, which emit particles primarily in the range 0.1–0.2 µm, is capable of causing health effects. This finding was not different because local butchers at this place use both car tyre sand firewood in meat preparation. In addition, Firewood has been the prime energy type that has been consumed in Ghana from 2003 to

2008 and contributing about 70%, 77.7%, and 76.4% in 2000, 2004 and 2008 respectively (Arthur et al., 2011). This study is also in agreement with the current study where firewood is among the fuels used in meat preparation, which is capable of polluting our environment as

well as causing health effects. It has also been confirmed in another study that, smoke from the biomass (car tyre and firewood) exposes families to destructive quantities of gases such as carbon monoxide, benzene, etc. (Mukherjee et al.,2014). This study finding is also in agreement with this study which revealed there is the presence of CO in both the abattoir area and the community at a concentration of 5.78ppm and 1.31ppm respectively. A comparable study by Obiri-Danso et al. (2008) also revealed that, in Ghana, human activities such as the use of scrap tyres in roasting meat instead of, the gas stove were identified, and it was because of the high cost in Gas fuel and electrical stove. This practice has increased the volume of smoke produce, therefore, contributing intensely to the health implications from the release of carbon monoxide and particulate matter into the environment. This clearly shows that the use of biomass fuel (car tyres and firewood) is of great concern to the local abattoir butchers and the community.

# 5.4 Comparing levels of CO and PM in the Abattoir area, Community with Standards, and within.

The second objective of the study compared the concentration levels of CO and PM against Standards of United States Environmental Protection Agency (US-EPA), World Health Organization (WHO) and the Ghana Environmental Protection Agency (G-EPA) set up to monitor and control the levels produced in the environment and within. The findings of this study identified the concentration level for CO to be of 5.78ppm and 1.31ppm respectively in both Abattoir and community, which is in the acceptable values of USA-EPA and G-EPA standards. According to US-EPA, allowable exposure for daily 8 hours should be less than 9ppm (10mg/m3) and 0.09ppm for WHO 24 hours exposure . This study was also in agreement with US-EPA values and fell within the acceptable range therefore thus not cause any serious alarm. Even though levels of 5.78 ppm was identified, it's still within the acceptable range. In addition, WHO has an average level for 24 hours watch instead of 8 hours with an acceptable value of 0.09ppm. Even though the levels seen in this study is higher than that of WHO but since its 24 hours, it cannot be directly compared with. Furthermore, the G-EPA value for CO is 35 ug/m3. This value is 24 hours just like that of WHO. This difference in values in this study may be subjected to several factors Such as the open-air burning is influenced by wind dispersion, rainfall and the volume produced per time this is because maximum ppm was 40.32 at a one-minute interval which shows a greater concern. Even though the levels are very low and fall within the acceptable range for USEPA and G-EPA standards, studies have reported on chronic and acute exposure thus have health effects. (US- EPA,2004; Ghana- EPA, 2015; WHO, 2005).

Concerning levels of PM 10 and 2.5, the findings in this study reported values of PM 10 to be 2350  $\mu$ g/m3 in the abattoir area and 190  $\mu$ g/m3 in the community. Concerning PM2.5, 1500  $\mu$ g/m3 was recorded in the abattoir and 140  $\mu$ g/m3 also for the community. All the recordings were done for 8 hours due to occupational exposure hours. Workers work for only 8 hours in a day so data was taken for 8 hours a day. When the values were compared with both international and local values, it was way higher than the acceptable range. This calls for serious action to be taken.( US- EPA,2004; Ghana- EPA, 2015; WHO, 2005). For US-EPA PM values are 150  $\mu$ g/m3, WHO value is 50  $\mu$ g/m3 and G-EPA is 10-100  $\mu$ /g/m3. Concerning PM 2.5, the US-EPA value is 35  $\mu$ g/m3, WHO is 25  $\mu$ g/m3 but there is no standard value as at yet for G-EPA. The concentrations obtained in this study is much higher than both international and local standards, which call for immediate attention. Both the community and the workers are at higher risk of developing serous health effects.

# 5.4.2 A Statistical Test to compare Concentrations in the Abattoir Area and the Community

The results from the study showed that when a one-sample t-test was conducted, the level for both CO, PM 10 and 2.5 was much higher at the abattoir area than that of the community. The

concentration level for CO was 5.78ppm and 1.31ppm with respect to the abattoir area and the community. The significant test, however, showed that the level of CO in Abattoir to the community was significant at (p < 0.001). Thus, it can be concluded that one who works at the Abattoir area is more susceptible to CO poisoning than those residents in the community. This result is in agreement with the study of Nelson et al. (1998). His study reported that one who is exposed to a high concentration of CO ends -up disrupting cell actions specifically to impairs the capacity of hemoglobin to release oxygen at tissue delivery sites. Concerning PM 10, the value in the Abattoir area was statistically significant to the community at (p<0.001) with average values of 2.35mg/m<sup>3</sup> and .19mg/m<sup>3</sup> respectively. In addition, PM 2.5 was also significant at (p<0.001) with average values of 1.50 mg/m3 and .14 mg/m3 respectively. This means that both PM 10 and 2.5 were higher in the Abattoir area than the community and those closer to the higher volume exposure are at a higher risk than those in the community. This study agrees to the findings of Ho et al. (2002) study which indicated that any little amount if PM especially 2.5 is capable of causing health effects. According to WHO (2013), low levels of exposure (below 100  $\mu$ g/m<sup>3</sup>) are associated with health effects which this study is in agreement with.

# 5.5 Health Effects Experienced and Clinical Symptoms Reported by the Respondent under study due to CO exposure.

The objective of the study was assessing the health effects experienced by the respondent due to CO exposure. The uses of biomass (scrap tyres, firewood) have been clearly associated with CO posing as reported early in this study. We went further to ask the respondent to selfreport symptoms and conditions encountered and were recorded. Form the study as reported by respondents showed that, majority of them experience Headache giving a percentage of 83.2 representing 79 of the sample population. Muscular Weakness was the next higher symptoms that were reported with a percentage of 74.7 representing 71 of respondent followed by

Dizziness with a percentage of 72.6 making a sample of 69This finding is consistent with the earlier research by Lin et al. (2008), which found that CO causes dizziness, nausea, headaches and weakness when inhaled at small levels. This study finding is further in agreement with several research, which reported clinical symptoms associated with CO exposure are a headache, dizziness, nausea and muscular weakness (Burney, 1982; Myers and Synder, 1985; Ely et al., 1995). There was also a reported symptom about cardiovascular diseases such as Palpitations with the percentage of 45.3 by 43 respondent and Hypertension with percentage of 20.0 representing 19 of the sample population. Even though the percentages were low, yet people are experiencing it. This outcome is also in agreement with prior research which found that some cardiovascular disease is correlated with CO exposure (Milojevic et al., 2014; Lifespan, 2008; Sveinung et al., 2006). Concerning CO exposure and respiratory disease, the study findings recorded Shortness of breath with a percentage of 43.2 making a sample size of 41 out of the total sample. Even though the percentage is less, this finding is in agreement with Kao et al, (2006) which revealed that CO exposure poses serious health effects to respiratory systems such as pulmonary edema, aspiration pneumonia due to decreased oxygen saturation. In addition, CO exposure has been associated with eye issues on which the finding of this study identified Blurred vision with a percentage of 69.5 of a respondent of 66 from the sample population. This percentage is very high and it is supported by findings of several researchers (Hudnell and Benignus, 1989; Fielding et al., 2010; Hampson and Weaver, 2011). All of these research found that CO exposure has severe impacts on human eyes, including blurred vision, photophobia, and diplopia. From the above, it can be clearly seen that CO exposure has some health implications in the cardiovascular system, respiratory system and the Human eye.

#### 5.6 Health Effects Experience by Respondents due to PM Exposure.

The objective of the study was assessing the health effects experienced by the respondent due to PM exposure. The usages of biomass (scrap tyres, firewood) have been clearly linked with PM posing as reported early in this study. The outcome from the finding of the study showed that PM 10 was present in both the abbatoir area and the community at a concentration of 2.35 mg/m3 and .19 mg/m3 respectively. Based on this we went on further to asked them some of the clinical symptoms are disease associated with it and the following were the response from study participants. Eye Irritations was the major symptom reported from respondent at a percentage of 91.6 representing 87 samples out of a total 94-sample population. This means that PM was clearly present and the effects were clearly seen. This study is in agreement with a study by Tan et al., (2018) which reported that the symptoms of dry eye include redness, eye dryness, severe pain, and itching. It can further develop into corneal ulcers, decreased eyesight and even blindness. This study finding was further supported by the study of Gupta and Muthukumar, (2018) also revealed that chronic exposure to PM 10 is associated with developing serious eye problems such as blindness, eye irritations, and cataract. The next reported symptom was Respiratory tract Infection with a percentage of 71.6 representing 68 of the sample respondent. This finding is also in agreement with (Schwartz, 1992; Sugiri et al., 2006; D'amato et al., 2010). The research results showed some symptoms connected with PM exposure such as asthma, wheezing, bronchitis and symptoms of lower respiratory tract infections. A cardiovascular condition such as Hypertension was also reported with a lower percentage of 20.0 representing 19 respondent. This means that PM is not associated with hypertension, which is also supported by (Brook et al., 2010; Pražnikar et al., 2012; Chiang et al., 2015). Their studies revealed that there is a negative impact of PM to cardiovascular diseases, prior to exposure from the use of biomass fuel and other human and industrial activities, which contains PM.

Concerning PM 2.5, which has been documented in studies to be the recent most dangerous form of PM and is capable of causing a serious health effect even with a small amount of exposure. The findings of the study showed that PM 2.5 was both present in the Abbatoir area and Community at concentrations of 1.50 mg/m3 and .14 mg/m3 respectively. The study recorded some symptoms reported from participants such as Respiratory tract Infection and Eye Irritations. This is in agreement with studies of Pope et al (1996) who reported that PM2.5 was associated with a 36% increase in death from lung cancer and 26% in cardiopulmonary deaths. In addition, Loxham and Nieuwenhuijsen (2019), also documented that, exposure to PM is associated with cardiovascular disease (eg. heart disease, myocardial infarction, and asthma and Type two diabetes) which was reported in this study. Myocardial infarction was reported by 3 respondents, Type two diabetes was reported by 5 respondent and asthma 14 respondent. Even though the figures were small, it is of greater concern due to the serious nature of such health issues. Further studies by Chen et al., 2015 discovered that, -exposure to PM2.5 by 10mg/m3 leads to an increase of systolic and diastolic blood pressure by 1–3mmHg and is associated with a hazard ratio of 1.13 for the development of arterial hypertension which this study is in agreement with by the reported presence of Hypertension among respondents. In the nutshell, PM 2.5 has also been associated with eye irritations by Mimura et al (2014) surveyed patients with acute conjunctivitis from May to October 2012 and found that the number of patients with acute conjunctivitis was increased with a higher level of PM2.5. It is now clear, evident that the findings of the study is supported with previous research, and all concluded that both PM 10 and 2.5 have serious health effects regardless of the amount of concentration W J SANE NO present in an environment.

#### CHAPTER SIX

#### CONCLUSION. AND RECOMMENDATION

#### 6.1 Conclusion

The study has established that exposure to carbon monoxide (CO) and particulate matter (PM) indeed increased the risk of developing some health effects in the study participants. In Ghana, many people use biomass fuel for both commercial and local activities such as cooking, meat preparation, waste management and baking due to expensive use of gas fuel, electrical appliances, and even charcoal. In this study, it was seen that local butchers who prepare meat at the Abattoir area rely on the use of both car tyre and firewood. It was proven that these biomass fuel used contain some poison substances such as carbon monoxide and particulate matter. The finding of the study revealed that the level of CO and PM in the Abattoir area was statistically significantly higher than the levels in the community Even though the concentration levels were in the acceptable limit of both local and international standard for CO, both workers and community members exposed reported some health symptoms and diseases (blurred vision, eye irritation, Hypertension, headache, nausea, asthma, respiratory tract infection, muscular weakness, chest pain etc) associated with the exposure to CO and PM. A further analysis was carried out to check the duration of exposure and associated health effects and the finding as that blurred vision statistically significant with CO exposure. Asthma and Hypertension were also statistically significant with PM exposure for a longer exposure duration. In the nutshell, frequent hospital visit and admission was also associated with CO and PM exposure. There is, therefore, the need to create better options to reduce the health effects due the fact that the concentration levels for PM 10 and 2.5 were statistically higher than the acceptable concentration values for both international and local standards. Thus, both abattoir and community members are at higher risk for developing serous health effects from the exposure.

#### **6.2 Recommendation**

Based on the outcome of this research, the following are the recommendations proposed to address the issue of CO and PM poisoning.

- The National Commission for Civic Education (N.C.C.E) in the Municipality together with the environmental Health Unit must carry out an intensive education on awareness on the dangers associated with CO and PM exposure, since its dangers go covertly.
- Ideally, LPG and Biogas usage are the best in addressing CO and PM exposure. Even though this form of energy is the best, yet comes with a high cost, of which workers at the abattoir area could not afford it. Based on this, it sis recommended that the usage of charcoal is encouraged to reduced CO emission and PM release into the atmosphere and to minimize the health effects experienced by community members and themselves.
- It is further recommended that, Ghana EPA together with its regulatory body should act accordingly to stop or regulate the activities of using car tyres in meat preparation due to the higher presence of PM 10 and 2.5 with the abattoir area and the community.

#### REFERENCES

- Agbaire, P.O. and Esiefarienrhe, E., 2009. Air Pollution tolerance indices (apti) of some plants around Otorogun Gas Plant in Delta State, Nigeria. *Journal of Applied Sciences and Environmental Management*, 13(1). Pp.5-15
- Akunne, A.F., Louis, V.R., Sanon, M. and Sauerborn, R., 2006. Biomass solid fuel and acute respiratory infections: the ventilation factor. *International journal of hygiene and environmental health*, 209(5), pp.445-450.

- Albalak R, Frisancho AR, Keeler GJ. 1999. Domestic biomass fuel combustion and chronic bronchitis in two rural Bolivian villages. *Thorax* 54:1004–1008
- Albalak, R., Frisancho, A.R. and Keeler, G.J., 1999. Domestic biomass fuel combustion and chronic bronchitis in two rural Bolivian villages. *Thorax*, *54*(11), pp.1004-1008.
- Amigun, B., Sigamoney, R. and von Blottnitz, H., 2008. Commercialisation of biofuel industry in Africa: a review. *Renewable and sustainable energy reviews*, *12*(3), pp.690-711.
- Amigun, B., Sigamoney, R. and von Blottnitz, H., 2008. Commercialisation of biofuel industry in Africa: a review. *Renewable and sustainable energy reviews*, *12*(3), pp.690-711.
- Amini, H., Cont, R. and Minca, A., 2016. Resilience to contagion in financial networks. *Mathematical finance*, 26(2), pp.329-365.
- Andreae, M.O. and Merlet, P., 2001. Emission of trace gases and aerosols from biomass burning. *Global biogeochemical cycles*, 15(4), pp.955-966.
- Arthur, R., Baidoo, M.F. and Antwi, E., 2011. Biogas as a potential renewable energy source: A Ghanaian case study. *Renewable Energy*, *36*(5), pp.1510-1516.
- Brook, R.D., Rajagopalan, S., Pope III, C.A., Brook, J.R., Bhatnagar, A., Diez-Roux, A.V.,
  Holguin, F., Hong, Y., Luepker, R.V., Mittleman, M.A. and Peters, A., 2010.
  Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation*, 121(21), pp.2331-2378.
- Burney, R.E., Wu, S.C. and Nemiroff, M.J., 1982. Mass carbon monoxide poisoning: clinical effects and results of treatment in 184 victims. *Annals of emergency medicine*, *11*(8), pp.394-399.

- Buzorius, G., Hämeri, K., Pekkanen, J. and Kulmala, M., 1999. Spatial variation of aerosol number concentration in Helsinki city. *Atmospheric Environment*, *33*(4), pp.553-565.
- Carratu MR, Cagiano R, Desantis S, et al.2000a Prenatal exposure to low levels of carbon monoxide alters sciatic nerve myelination in rat offspring. *Life Sci*; 67:1759–1772.
- Carratu MR, Cagiano R, Tattoli M, et al.2002. Prenatal exposure model simulating CO inhalation in human cigarette smokers: Sphingomyelin alterations in the rat sciatic nerve. *Toxicol Lett* ;117(1–2):101–106.
- Carratu MR, Renna G, Giustino A, et al. 1993. Changes in peripheral nervous system activity produced in rats by prenatal exposure to carbon monoxide. *Arch Toxicol.*;67:297–301.
- Chan, S.H., Van Hee, V.C., Bergen, S., Szpiro, A.A., DeRoo, L.A., London, S.J., Marshall, J.D., Kaufman, J.D. and Sandler, D.P., 2015. Long-term air pollution exposure and blood pressure in the sister study. *Environmental health perspectives*, *123*(10), pp.951-958.
- Chang CC, Chen PS, Yang CY. Short-term effects of fine particulate air pollution on hospital admissions for cardiovascular diseases: a case-crossover study in a tropical city. J Toxicol Environ Health A 2015;78:267-77. [PubMed] [Google Scholar]
- Charlson, R.J., Schwartz, S.E., Hales, J.M., Cess, R.D., Coakley, J.J., Hansen, J.E. and Hofmann, D.J., 1992. Climate forcing by anthropogenic aerosols. *Science*, 255(5043), pp.423-430.
- Chen, J., Liu, G., Kang, Y., Wu, B., Sun, R., Zhou, C. and Wu, D., 2014. Coal utilization in China: environmental impacts and human health. *Environmental geochemistry and health*, 36(4), pp.735-753.
- Chu K, Jung K, Kim H, et al.2004. Diffusion-weighted MRI and 99mTcHMPAO SPECT in delayed relapsing type of carbon monoxide poisoning: evidence of delayed cytotoxic edema. *European Neurology* ;51:98-103.
- Clarke, S., Keshishian, C., Murray, V., Kafatos, G., Ruggles, R., Coultrip, E., Oetterli, S., Earle, D., Ward, P., Bush, S. and Porter, C., 2012. Screening for carbon monoxide exposure in selected patient groups attending rural and urban emergency departments in England: a prospective observational study. *BMJ open*, 2(6), p. e000877.
- COHEN, A.J.2000. Outdoor air pollution and lung cancer. *Environmental health perspectives*, 108: 7
- Cooper, J.A., 1980. Environmental impact of residential wood combustion emissions and its implications. *Journal of the Air Pollution Control Association*, *30*(8), pp.855-861.
- Costa, S., Ferreira, J., Silveira, C., Costa, C., Lopes, D., Relvas, H., Borrego, C., Roebeling, P., Miranda, A.I. and Paulo Teixeira, J., 2014. Integrating health on air quality assessment—Review report on health risks of two major European outdoor air pollutants: PM and NO2. *Journal of Toxicology and Environmental Health, Part B*, *17*(6), pp.307-340.
- D'Amato, G., Cecchi, L., D'amato, M. and Liccardi, G., 2010. Urban air pollution and climate change as environmental risk factors of respiratory allergy: an update. *Journal of Investigational Allergology and Clinical Immunology*, 20(2), pp.95-102.
- De Salvia MA, Cagiano R, Carratu MR, et al 1995. Irreversible impairment of active avoidance behavior in rats prenatally exposed to mild concentrations of carbon monoxide. *Psychopharmacology*; 122:66–71

- Department of Health. carbon-monoxide-poisoning-alert http://gp.dh.gov.uk/2011/09/27// (accessed August, 2019).
- Dias, N.V., Billberg, H., Sonesson, B., Törnqvist, P., Resch, T. and Kristmundsson, T., 2016. The effects of combining fusion imaging, low-frequency pulsed fluoroscopy, and lowconcentration contrast agent during endovascular aneurysm repair. *Journal of vascular surgery*, 63(5), pp.1147-1155.
- Dockery, D.W., Pope, C.A., Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris Jr, B.G. and Speizer, F.E., 1993. An association between air pollution and mortality in six US cities. *New England journal of medicine*, *329*(24), pp.1753-1759.
- Dominici, F., Peng, R.D., Bell, M.L., Pham, L., McDermott, A., Zeger, S.L. and Samet, J.M., 2006. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *Jama*, 295(10), pp.1127-1134.
- Dominici, F., Peng, R.D., Bell, M.L., Pham, L., McDermott, A., Zeger, S.L. and Samet, J.M., 2006. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. *Jama*, 295(10), pp.1127-1134.
- Dotse, S.Q., Asane, J.K., Ofosu, F.G. and Aboh, I.J.K., 2012. Particulate Matter and Black Carbon Concentration Levels in Ashaiman, a Semi-Urban Area of Ghana, 2008. *Research Journal of Environmental and Earth Sciences*, 4(1), pp.20-25.
- Dunea, D., Iordache, S. and Pohoata, A., 2016. Fine particulate matter in urban environments: a trigger of respiratory symptoms in sensitive children. *International journal of environmental research and public health*, *13*(12), p.1246.

- Ely EW, Moorehead B, Haponik EF 1995. Warehouse workers' headache: emergency evaluation and management of 30 patients with carbon monoxide poisoning. Am. J. Med., 98:145-155.
- EPA (2010) Science and technology, scrap tyres. Innovative uses forscrap tyres. http://www.epa.gov/epawaste/conserve/materials/tyres/science.htm air burning of waste vehicle tyres and their possible impacts on the environment. Atmos Environ 42:6555–6559.

Sandow, B., 2016. Diurnal Rhythms of Ambient Air Pollution Due to Vehicular Traffic in Accra (*Doctoral dissertation*, University of Ghana).

EPA, 2000. Air quality index: A guide to air quality and your health. Environmental Protection Agency (EPA). http://www.njaqinow.net/App\_AQI/AQI.en-US.pdf.

accessed August, 2019

- EPA, 2015, Air quality standards for Ghana, *Environmental Protection Agency* (EPA). pdf https://wedocs.unep.org/bitstream/handle/20.500.11822/17202/Ghana.pdf?sequence= 1&isAllowed=y. Accessed on 24, August, 2019.
- EPA, U. 2004. Air quality criteria for particulate matter. *National Center for Environmental* Assessment-RTP Office.
- Ezzati, M. and Kammen, D.M., 2002. The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps, and data needs. *Environmental health perspectives*, 110(11), pp.1057-1068.
- Faiz, A., & Sturm, P. (2002). New directions: air pollution and road traffic in developing countries. *Developments in Environmental Science*, 1, 241-243.

Fiahagbe, E. (2008) < Air\_ Monitoring \_Accra.Pdf>

- Fielding J, Lang W, White OB 2010. Carbon monoxide poisoning: impact on ocular motility. *Cognitive and Behavioral Neurology*;23(4):256-261.
- Gauderman, W.J., Avol, E., Gilliland, F., Vora, H., Thomas, D., Berhane, K., McConnell, R., Kuenzli, N., Lurmann, F., Rappaport, E. and Margolis, H., 2004. The effect of air pollution on lung development from 10 to 18 years of age. *New England Journal of Medicine*, 351(11), pp.1057-1067.
- Gauderman, W.J., Vora, H., McConnell, R., Berhane, K., Gilliland, F., Thomas, D., Lurmann, F., Avol, E., Kunzli, N., Jerrett, M. and Peters, J., 2007. Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *The Lancet*, 369(9561), pp.571-577.
- Gehring, U., Heinrich, J., Krämer, U., Grote, V., Hochadel, M., Sugiri, D., Kraft, M., Rauchfuss, K., Eberwein, H.G. and Wichmann, H.E., 2006. Long-term exposure to ambient air pollution and cardiopulmonary mortality in women. *Epidemiology*, pp.545-551.
- Goldberg M S, Burnett R T, Stieb D M, et al. Associations between ambient air pollution and daily mortality among elderly persons in Montreal, Quebec [J]. *Science of the Total Environment*, 2013,464(463-464c):931-942.

Goldstein, J.1977 Police Discretion Not to Invoke the Criminal Process. Hartford: Yale Press

Goldstein, N.J., Cialdini, R.B. and Griskevicius, V., 2008. A room with a viewpoint: Using social norms to motivate environmental conservation in hotels. *Journal of consumer Research*, *35*(3), pp.472-482.

Gupta PD, Muthukumar A (2017) Why environmental pollutants makes our eye sick? J Clin

Opthalmol Eye Discord 1: 1010.

- Gupta, P.D. and Muthukumar, A., 2018. Minor to Chronic Eye Disorders Due to Environmental Pollution: A Review. *J Ocul Infect Inflamm*, 2(108), p.2.
- Hampson NB, Weaver LK.2011. Residential carbon monoxide alarm use: opportunities for poisoning prevention. *Journal of Environmental Health*;73(6):30-33
- Hanley, M.E. and Patel, P.H., 2019. Carbon Monoxide Toxicity. In *StatPearls [Internet]*. StatPearls Publishing.
- HAS (2007) Carbon Monoxide. Dublin. Available at: www.carbonmonoxide.ie. (Accessed August, 2019)
- HEALTH EFFECTS INSTITUTE (HEI) 1999. Diesel Emissions and Lung Cancer: *Epidemiology* and Quantitative Risk Assessment. Available on:

http://healtheffects.org/pubs-special.htm, Health Effects Institute (Accessed on August, 2019).

- Health Effects Institute. Panel on the Health Effects of Traffic-Related Air Pollution, 2010. *Traffic-related air pollution: a critical review of the literature on emissions, exposure, and health effects* (No. 17). Health Effects Institute.
- Ho, K.F., Lee, S.C., Jimmy, C.Y., Zou, S.C. and Fung, K., 2002. Carbonaceous characteristics of atmospheric particulate matter in Hong Kong. *Science of the Total Environment*, *300*(1-3), pp.59-67.
- Hoek, G., Beelen, R., De Hoogh, K., Vienneau, D., Gulliver, J., Fischer, P. and Briggs, D., 2008. A review of land-use regression models to assess spatial variation of outdoor air pollution. *Atmospheric environment*, 42(33), pp.7561-7578.

- Hudnell, H.K. and Benignus, V.A., 1989. Carbon monoxide exposure and human visual detection thresholds. *Neurotoxicology and teratology*, *11*(4), pp.363-371.
- Huq, M., Dasgupta, S., Khaliquzzaman, V., Pandey, K. and Wheeler, D., 2004. *Indoor air quality for poor families: new evidence from Bangladesh*. The World Bank.
- Jerrett, M., Burnett, R.T., Pope III, C.A., Ito, K., Thurston, G., Krewski, D., Shi, Y., Calle, E. and Thun, M., 2009. Long-term ozone exposure and mortality. *New England Journal of Medicine*, 360(11), pp.1085-1095.
- Johnson, M.A. and Chiang, R.A., 2015. Quantitative guidance for stove usage and performance to achieve health and environmental targets. *Environmental health perspectives*, *123*(8), pp.820-826.
- Kaiser, J., 1997. Showdown over clean air science.
- Kao LW, Nanagas KA 2005. Carbon monoxide poisoning. *Med Clin North Am.*, 89: 1161-1194.
- Kim, K.H., Kabir, E. and Kabir, S., 2015. A review on the human health impact of airborne particulate matter. *Environment international*, *74*, pp.136-143.
- Kumar, M., Singh, R.S. and Banerjee, T., 2015. Associating airborne particulates and human health: Exploring possibilities: Comment on: Kim, Ki-Hyun, Kabir, E. and Kabir, S.
  2015. A review on the human health impact of airborne particulate matter. Environment International 74 (2015) 136-143. *Environment international*, 84, p.201.
- Lee, F.Y., Chen, W.K., Lin, C.L. and Kao, C.H., 2015. Carbon monoxide poisoning and subsequent cardiovascular disease risk: a nationwide population-based cohort study. *Medicine*, 94(10).

- Lifespan. 2008"Carbon Monoxide May Cause Long-lasting Heart Damage." *ScienceDaily* <www.sciencedaily.com/releases/2008/01/080129125412.htm>. (Accessed on August 2019).
- Lin, T.C., Krishnaswamy, G. and Chi, D.S., 2008. Incense smoke: clinical, structural and molecular effects on airway disease. *Clinical and Molecular Allergy*, 6(1), p.3.
- Ling, S.H. and van Eeden, S.F., 2009. Particulate matter air pollution exposure: role in the development and exacerbation of chronic obstructive pulmonary disease. *International journal of chronic obstructive pulmonary disease*, *4*, p.233.
- Lipfert, F. W., & Wyzga, R. E. (2008). On exposure and response relationships for health effects associated with exposure to vehicular traffic. *J Expo Sci Environ Epidemiol*, 18(6), 588-599. doi: 10.1038/jes.2008.4
- Liu, H., Tian, Y., Xiang, X., Li, M., Wu, Y., Cao, Y., Juan, J., Song, J., Wu, T. and Hu, Y., 2018. Association of short-term exposure to ambient carbon monoxide with hospital admissions in China. *Scientific reports*, 8(1), p.13336.
- Liu, H.Y., Dunea, D., Iordache, S. and Pohoata, A., 2018. A review of airborne particulate matter effects on young children's respiratory symptoms and diseases. *Atmosphere*, 9(4), p.150.
- Lo C, Chen S, Lee K, et al. 2007.Brain injury after acute carbon monoxide poisoning: early and late complications. *American Journal of Roentgenology*.189:205-211.

ANF

Lopez I, Acuna D, Webber DS, et al. 2003.Mild carbon monoxide exposure diminishes selectively the integrity of the cochlea of the developing rat. *J Neurosci Res*.74(5):666– 675.

- Loxham, M. and Nieuwenhuijsen, M.J., 2019. Health effects of particulate matter air pollution in underground railway systems-a critical review of the evidence. *Particle and fibre toxicology*, *16*(1), pp.1-24.
- Machin, D., Campbell, M.J., Tan, S.B. and Tan, S.H., 2018. Sample Sizes for Clinical, Laboratory and Epidemiology Studies. Wiley-Blackwell. Chapter 1.pp.1-28
- Mavalankar, D.V., Trivedi, C.R. and Gray, R.H., 1991. Levels and risk factors for perinatal mortality in Ahmedabad, India. *Bulletin of the World Health Organization*, 69(4), p.435.
- McCreanor, J., Cullinan, P., Nieuwenhuijsen, M.J., Stewart-Evans, J., Malliarou, E., Jarup,
   L., Harrington, R., Svartengren, M., Han, I.K., Ohman-Strickland, P. and Chung, K.F.,
   2007. Respiratory effects of exposure to diesel traffic in persons with asthma. *New England Journal of Medicine*, 357(23), pp.2348-2358.
- Medina-Ramon, M., Zanobetti, A. and Schwartz, J., 2006. The effect of ozone and PM10 on hospital admissions for pneumonia and chronic obstructive pulmonary disease: a national multicity study. *American journal of epidemiology*, 163(6), pp.579-588.
- Milojevic, A., Wilkinson, P., Armstrong, B., Bhaskaran, K., Smeeth, L. and Hajat, S., 2014.
   Short-term effects of air pollution on a range of cardiovascular events in England and Wales: case-crossover analysis of the MINAP database, hospital admissions and mortality. *Heart*, *100*(14), pp.1093-1098.
- Mimura, T., Ichinose, T., Yamagami, S., Fujishima, H., Kamei, Y., Goto, M., Takada, S. and Matsubara, M., 2014. Airborne particulate matter (PM2. 5) and the prevalence of allergic conjunctivitis in Japan. *Science of the Total Environment*, 487, pp.493-499.

- Monn, C. and Schaeppi, G., 1993. Concentrations of total suspended particulates, fine particles and their anionic compounds in ambient air and indoor air. *Environmental technology*, *14*(9), pp.869-875.
- Morris, R.D., Naumova, E.N. and Munasinghe, R.L., 1995. Ambient air pollution and hospitalization for congestive heart failure among elderly people in seven large US cities. *American journal of public health*, 85(10), pp.1361-1365.
- Mukherjee, S., Roychoudhury, S., Siddique, S., Banerjee, M., Bhattacharya, P., Lahiri, T. and Ray, M.R., 2014. Respiratory symptoms, lung function decrement and chronic obstructive pulmonary disease in pre-menopausal Indian women exposed to biomass smoke. *Inhalation toxicology*, *26*(14), pp.866-872.
- Münzel, T., Sørensen, M., Gori, T., Schmidt, F.P., Rao, X., Brook, J., Chen, L.C., Brook, R.D. and Rajagopalan, S., 2017. Environmental stressors and cardio-metabolic disease: part I–epidemiologic evidence supporting a role for noise and air pollution and effects of mitigation strategies. *European heart journal*, *38*(8), pp.550-556.
- Myers RA, Snyder SK, Emhoff TA 1985. Subacute sequelae of carbon monoxide poisoning. *Ann. Emerg. Med.*, 14: 1163-1167.
- Nagar, J.K., Akolkar, A.B. and Kumar, R., 2014. A review on airborne particulate matter and its sources, chemical composition and impact on human respiratory system. *Int J Environ Sci*, *5*(2), pp.447-463.
- Nelson, G. 2006. Carbon monoxide determination in human blood. Pp. 175-180 in Carbon Monoxide and Human Lethality: Fire and Non-fire Studies, *M.M. Hinschler, editor.*, ed. New York: Taylor and Francis.

- Newby, D.E., Mannucci, P.M., Tell, G.S., Baccarelli, A.A., Brook, R.D., Donaldson, K., Forastiere, F., Franchini, M., Franco, O.H., Graham, I. and Hoek, G., 2014. Expert position paper on air pollution and cardiovascular disease. *European heart journal*, *36*(2), pp.83-93.
- Njoku, D.I., Ukaga, I., Ikenna, O.B., Oguzie, E.E., Oguzie, K.L. and Ibisi, N., 2016. Natural products for materials protection: corrosion protection of aluminium in hydrochloric acid by Kola nitida extract. *Journal of Molecular Liquids*, *219*, pp.417-424.
- Ntim, M., Owusu-Boateng, G. and Plange-Rhule, J., 2013. Air quality and the lung function of communities in the concessional area of the Chirano Gold Mines Limited, BibianiGhana.
- Obiri-Danso, K., Hogarh, J.N. and Antwi-Agyei, P., 2008. Assessment of contamination of singed hides from cattle and goats by heavy metals in Ghana. *African Journal of Environmental Science and Technology*, 2(8), pp.217-221.
- Ostro B 2004. Outdoor air pollution: Assessing the environmental burden of disease at national and local levels. Geneva, World Health Organization (*Environmental Burden of Disease Series*, No. 5; http://www.who.int/quantifying\_ehimpacts/publications/ ebd5.pdf). (Accessed on August 2019)
- Paul, G. H. J. N. 2008. Estimating Vehicle Emissions and Air Pollution Calming. Urban Transport Systems.
- Peabody, T., Furr, A. and Ditmetaroj, N., 2013. Carbon Monoxide and the Eye: A Teaching Case Report. *Optometric Education*, *38*(3).

- Petkova, E.P., Jack, D.W., Volavka-Close, N.H. and Kinney, P.L., 2013. Particulate matter pollution in African cities. *Air Quality, Atmosphere & Health*, 6(3), pp.603-614.43– 750.
- Poloniecki JD, Atkinson RW, de Leon AP, et al 1997. Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occup Environ Med* ;54:535-40.
- Pooley, F.D. and Mille, M., 1999. Composition of air pollution particles. In *Air pollution and health* (pp. 619-634). Academic Press.
- Pope III, C.A. and Dockery, D.W., 2006. Health effects of fine particulate air pollution: lines that connect. *Journal of the air & waste management association*, *56*(6), pp.709-742.
- Pope III, C.A., Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., Ito, K. and Thurston, G.D., 2002. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Jama*, 287(9), pp.1132-1141.
- Pope III, C.A., Burnett, R.T., Thurston, G.D., Thun, M.J., Calle, E.E., Krewski, D. and Godleski, J.J., 2004. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation*, 109(1), pp.71-77.
- Pope, C. A. et al.1995. Particulate air pollution as predictor of mortality in a prospective study of U.S. adults. *American journal of respiratory and critical care medicine*, 151: 669– 674

- Pražnikar, Z. and Pražnikar, J., 2012. The effects of particulate matter air pollution on respiratory health and on the cardiovascular system. *Slovenian Journal of Public Health*, *51*(3), pp.190-199.
- Pražnikar, Z. and Pražnikar, J., 2012. The effects of particulate matter air pollution on respiratory health and on the cardiovascular system. *Slovenian Journal of Public Health*, 51(3), pp.190-199.

Prockop, L.D. and Chichkova, R.I., 2007. Carbon monoxide intoxication: an updated review.

*Journal of the neurological sciences*, 262(1-2), pp.122-130. Rathore, O. and R Ruiz-Mercado, I.M., Omar; Zamora, Hilda; Smith, Kirk R 2016., Adoption

and sustained use of improved cookstoves. Energy Policy, 2011. 39: p. 7557-

7566.ein,

- Reboul, C., Boissière, J., André, L., Meyer, G., Bideaux, P., Fouret, G., Feillet-Coudray, C., Obert, P., Lacampagne, A., Thireau, J. and Cazorla, O., 2017. Carbon monoxide pollution aggravates ischemic heart failure through oxidative stress pathway. *Scientific reports*, 7, p.39715.
- Rehfuess, E.A., Bruce, N.G. and Smith, K.R., 2011. Solid Fuel Use: Health Effect. In: Nriagu JO (ed.) Encyclopedia of Environmental Health, v 5, pp. 150161 Burlington: Elsevier, 2011. Environmental Health, 5, p.150161.
- Ricci, P.F., Catalano, J.A. and Kelsh, M.D., 1996. Time series (1963-1991) of mortality and ambient air pollution in California: An assessment with annual data. *Inhalation toxicology*, 8(1), pp.95-106.

Roger, Véronique L., Alan S. Go, Donald M. Lloyd-Jones, Robert J. Adams, Jarett D. Berry,

Todd M. Brown, Mercedes R. Carnethon et al. "Heart disease and stroke statistics— 2011 update: a report from the American Heart Association." *Circulation* 123, no. 4 (2011): e18-e209.

Ross, M.H., Zick, B.L. and Tsalik, E.L., 2019. Host-Based Diagnostics for Acute Respiratory Infections. *Clinical therapeutics*.

Salvi, S.S., Sampson, A.P. and Holgate, S.T., 2001. Asthma. e LS.

- Seyyednejad, S.M., Niknejad, M. and Koochak, H., 2011. A review of some different effects of air pollution on plants. *Research Journal of Environmental Sciences*, 5(4), pp.302309.
- Sofoluwe, G.O., 1968. Smoke pollution in dwellings of infants with bronchopneumonia. *Archives* of Environmental Health: An International Journal, 16(5), pp.670-672.
- Sørhaug, S., Steinshamn, S., Nilsen, O.G. and Waldum, H.L., 2006. Chronic inhalation of carbon monoxide: effects on the respiratory and cardiovascular system at doses corresponding to tobacco smoking. *Toxicology*, 228(2-3), pp.280-290.
- Srikanth, M.B., Dydek, Z.T., Annaswamy, A.M. and Lavretsky, E., 2009, June. A robust environment for simulation and testing of adaptive control for mini-UAVs. In 2009 American Control Conference (pp. 5398-5403). IEEE.
- Takahashi, N., Shinjoh, H., Iijima, T., Suzuki, T., Yamazaki, K., Yokota, K., Suzuki, H.,
  Miyoshi, N., Matsumoto, S.I., Tanizawa, T. and Tanaka, T., 1996. The new concept
  3-way catalyst for automotive lean-burn engine: NOx storage and reduction catalyst. *Catalysis Today*, 27(1-2), pp.63-69.
- Tian, L., Qiu, H., Pun, V.C., Ho, K.F., Chan, C.S. and Ignatius, T.S., 2015. Carbon monoxide and stroke: a time series study of ambient air pollution and emergency

hospitalizations. International journal of cardiology, 201, pp.4-9.

- Urbanski, S.P., Hao, W.M. and Baker, S., 2008. Chemical composition of wildland fire emissions. Developments in environmental science, 8, pp.79-107.
- Uzoigwe, J.C., Prum, T., Bresnahan, E. and Garelnabi, M., 2013. The emerging role of outdoor and indoor air pollution in cardiovascular disease. *North American journal of medical sciences*, 5(8), p.445.

Vallero, D.A., 2014. Fundamentals of air pollution. Academic press.

- Van der Wal, J.T. and Janssen, L.H.J.M., 2000. Analysis of spatial and temporal variations of PM 10 concentrations in the Netherlands using Kalman filtering. *Atmospheric Environment*, 34(22), pp.3675-3687.
- Warwick, H. and Doig, A., 2004. Smoke-the Killer in the Kitchen. In Smoke-the Killer in the Kitchen: Indoor Air Pollution in Developing Countries (pp. 941-961). Practical Action Publishing.
- Wellenius, G.A., Schwartz, J. and Mittleman, M.A., 2005. Air pollution and hospital admissions for ischemic and hemorrhagic stroke among medicare beneficiaries. *Stroke*, *36*(12), pp.2549-25 Peters, A., Von Klot, S., Heier, M., Trentinaglia, I., Hörmann, A., Wichmann, H.E. and Löwel, H., 2004. Exposure to traffic and the onset of myocardial infarction. *New England Journal of Medicine*, *351*(17), pp.1721-1730. 53.
- WHO (2005). Indoor air pollution and health. Geneva, World Health Organization (WHO Fact Sheet No. 292; *http://www.who.int/mediacentre/factsheets/fs292/en/index. html*).(Accessed on ... August 2019)

- WHO (2009). Global health risks: Mortality and burden of diseases attributable to selected major risks. *Geneva, World Health Organization* ( http://www.who.int/healthinfo/ global\_burden\_disease/GlobalHealthRisks\_report\_full.pdf). (Accessed on *August* 2019)
- WHO 2009. WHO handbook on indoor radon, a public health perspective. Geneva, World Health Organization (http://whqlibdoc.who.int/publications/2009/9789241547673 eng.pdf). (Accessed on August 2019)

World Health Organization (WHO) 2008 World report on child injury prevention. WHO

World Health Organization (WHO), 2013World Health Organization (WHO) Health effects of particulate matter. Policy implications for countries in eastern Europe, Caucasus and central AsiaWHO Regional Office for Europe, Copenhagen (2013)

World Health Organization, 2009. World health statistics 2009. World Health Organization.

- World Health Organization, 2016. Ambient air pollution: A global assessment of exposure and burden of disease.
- Wright J.2002. Chronic and occult carbon monoxide poisoning: we don't know what we're missing. *Emerg Med J*; 19:386–90.

Yadav, I.C. and Devi, N.L., 2018. Biomass Burning, Regional Air Quality, and Climate Change. Earth Systems and Environmental Sciences. Edition: Encyclopedia of Environmental org/10.1016/B978-0-12-409548-9.11022-X. (Accessed on August 2019)

#### APPENDICES

### **APPENDIX I**

### **QUSETIONNAIRE**

This questionnaire is designed purposefully for collecting information as part of the partial fulfillment of the ward of a Master of Public Health degree in Occupational and Environmental Health and safety. This will also assist in policy formulation regarding occupational exposures that may affect the health indigenous workers and local district assemblies. This information that will be gathered will be treated strictly and confidential as well as identity to respondents will not be disclose under any circumstance. Pleases kindly feel free and give appropriate answers as possible.

## **SECTION A**

- 1. Code of participant.....
- 2. Age of participant (years) .....
- 3. Marital status
- A. Married [ ] B. Single [ ] C. Devoice D. Others [ ]
- 4. Level of Education
- A. Non formal education [] B. Primary / Junior High school [] C. Senior High/ O-Level /A level [] D. Tertiary level [].
- 5. Number of years of work : .....
- 6. Work within Abattoir area [] Live in Kaase community closer to 100m []

# SECTION

B

## HAZARDS AND ACCIDENTS IDENTIFICATION

- 7. Physical hazards
- A. Slips and falls [] B. Noise [] C. burns [] D. Cuts and pricks [] E.

## **SECTION C**

## HEALTH IMPACTS OF CARBON MONOXIDE AND PARTICULATE MATTER

## EXPOSURE

- GENERAL KNOWLEDGE
- 8. Do you know that smoke from the burning activity is hazardous to your health?
- A. Yes [ ] B. No [ ]
- 9. How long are you exposed to this smoke or air pollution in a day?
- A. Less than 8 hours [] B. 12 hours[] C. Above 12 hours []
- 10. Has there been any health personnel in this community come and educate you about the dangers of exposed to CO and PM through the smoke from the open burning and the air pollution? A. Yes [] B. No []
- 11. How frequently have you been admitted to the hospital for the past 6 months?

## SECTION

A. 1-2 times [] B. 3-4 times [] c. Above 5 times []

D

# CARBON MONOXIDE EXPOSURE SIGNS AND SYMPTOMS

12. Have you experienced any of the following signs and symptoms recently or for the past one

year?

Please thick appropriate box

MILD	YES	NO	MODERATE	YES	NO	SEVERE	YES	NO
SYMPTOMS			SYMPTOMS			SYMPTOMS		
		4		1		1	7	
Nausea/ Vomiting		3	Blurred vision	8	1	Palpitations		
Shortness of breath	À	X	Confusion	万		Hypotension		
Dizziness		3	Chest pain	2		Myocardial Ischemia		
Headache Yes			Muscular	Y	12	Cardiac arrest		
ARK .	1	×	Weakness		5	Jan Jan		
~	2	E Z		7	5	Respiratory arrest		
		1	J SANE	14				

### SECTION

		Seizures / Coma
		ICT
E	KIN	USI

## PARTICULATE MATTER IMPCT ON HEALTH

13. Have you ever experienced or been diagnosed of any of the following ailment for the past

one year or three months?

Please tick appropriate box

Yes	No
1	
15	F
137	7
5	
and	N
1-1	
	1
	5
/	2
13	
BA	
5	
	Yes





### **APPENDIX II**

#### **COPY OF ETHICAL CLEARANCE**



KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY **COLLEGE OF HEALTH SCIENCES** 

SCHOOL OF MEDICAL SCIENCES / KOMFO ANOKYE TEACHING HOSPITAL COMMITTEE ON HUMAN RESEARCH, PUBLICATION AND ETHICS

Our Ref: CHRPE/AP/538/19

4th September, 2019.

Mr. Isaac Kwaku Acheampong Department of Occupational and Environmental Health KNUST-KUMASI.

Dear Sir,

### LETTER OF APPROVAL

Protocol Title:

"Assessing the Level of Particulate Matter and Carbon Monoxide Exposure in the Kaase Community of Kumasi-Metropolis, Ghana"

Proposed Site:

Sponsor:

#### Principal Investigator.

Your submission to the Committee on Human Research, Publications and Ethics on the above-named protocol refers.

The Committee reviewed the following documents:

- A notification letter of 29th August, 2019 from the Kaase Abattoir Station, Kumasi •
  - (study site) indicating approval for the conduct of the study at the Facility.

Kaase Abattoir Station, Kumasi Metropolis.

- A Completed CHRPE Application Form.
- Participant Information Leaflet and Consent Form.
- Research Protocol.
- Questionnaire. .

The Committee has considered the ethical merit of your submission and approved the protocol. The approval is for a fixed period of one year, beginning 4th September, 2019 to 3rd September, 2020 renewable thereafter. The Committee may however, suspend or withdraw ethical approval at any time if your study is found to contravene the approved protocol.

Data gathered for the study should be used for the approved purposes only. Permission should be sought from the Committee if any amendment to the protocol or use, other than submitted, is made of your research data.

The Committee should be notified of the actual start date of the project and would expect a report on your study, annually or at the close of the project, whichever one comes first. It should also be informed of any publication arising from the study.

Thank you, Sir, for your application.

Yours faithfully,

ng MD, FWACP Osomfo Pro Chairman

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