UNIVERSITY OF CAPE COAST

INDOOR AIR POLLUTION MONITORING FOR URINARY POLYCYCLIC AROMATIC HYDROCARBONS, PHENOLS, PHTHALATES AND SOME METABOLITES OF FISH SMOKERS FROM THREE COASTAL REGIONS OF GHANA

CHARITY KUSI

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BY

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Thesis submitted to the Department of Chemistry of the College of Agriculture and Natural Sciences, School of Physical Sciences, University of Cape Coast, in partial fulfilment of the requirements for the award of Doctor of Philosophy degree in Chemistry

JULY 2020

DECLARATION

Candidate's Declaration

I hereby declare that this thesis is the result of my own original research and that no part of it has been presented for another degree in this university or elsewhere.

Candidate's Signature……  …… Date……

Name: Charity Kusi

Supervisors' Declaration

We hereby declare that the preparation and presentation of the thesis were supervised in accordance with the guidelines on supervision of thesis laid down by the University of Cape Coast.

Principal Supervisor's Signature……  ……Date…….

Name: Prof. David Kofi Essumang

Co-Supervisor's Signature……  …… Date……..…

Name: Dr. Reginald Quansah

ABSTRACT

Exposure to biomass combustion has been linked to increased risk for a suite of negative health outcomes such as acute and chronic respiratory diseases. The objective of this study was to assess the levels and risk of polycyclic aromatic hydrocarbons, phenols, phthalates, 1-hydroxypyrene and 9-phenanthrenol from urine samples of fish smokers in three coastal regions (Western, Central and Volta regions) of Ghana due to their exposure to indoor air pollution. Urine samples of 155 fish smokers were analysed using QuEChERS method and gas chromatography coupled with mass spectrometer for analysis. The micro-environment indoor levels of particulate matter ($PM_{2.5}$), carbon monoxide and ozone were also measured using low-cost air quality monitors. In addition, personal $PM_{2.5}$ and CO were measured using MicroPEM (ECM) and Lascar EL-USB-CO respectively. The eyes of the fish smokers were also screened by qualified and practicing optometrists from Optometry Department, University of Cape Coast. Risk assessment on urinary contaminants showed high health risk values in the study areas. Fish smokers suffered from cataract and other eye diseases. Self-reported health symptoms were headache, cough, wheezing breathlessness, asthma and pneumonia. Personal CO was strongly correlated with benz[a]anthracene and benzo[a]pyrene. Also, personal $PM_{2.5}$ was strongly correlated with benz[a]anthracene, benzo[a]pyrene, 9-phenanthrenol and 1-hydroxypyrene. In conclusion, biomass fuel usage by the fish smokers could affect their health greatly. It is recommended that the fish smokers should be encouraged to use personal protective equipment such as respirators, googles and face masks to minimize the harmful effect of their work on their health.

KEY WORDS

Fish Smokers

Indoor Air Pollution

Metabolites

Phenols

Phthalates

Polyclic Aromatic Hydrocarbons

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DEDICATION

To my children: Daniel Kwame Gyamfi Owusu and Franklina Akosua Nyamekye Owusu and Board of Directors, Amansie West Rural Bank.

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LIST OF ABBREVIATIONS

µg/L	Microgram per litre
ACGIH	American Conference of Governmental Industrial Hygienists
ACL	Acenaphthalene
ACN	Acenaphthene
ANT	Anthracene
ASHRAE	American Society of Heating, Refrigerating and Air-Conditioning Engineers
ATSDR	The Agency for Toxic Substances and Disease Registry
B(2IPE)	Bis(2-chloroisopropyl) ether
B[A]A	Benzo[a]anthracene
B[A]F	Benzo[a]flouranthene
B[A]P	Benzo[a]pyrene
B[B] F	Benzo[b]flouranthene
B[K]F	Benzo[k]flouranthene
B2-CEE	Bis(2-Chroethyl) ether
BBP	Benzylbutyl phthalate
BEP	Bis(2-ethylhexyl) phthalate
BPE	Bromophenyl ether
CHP	Chlorophenol
CHR	Chrysene
CMPH	Chloromethylphenol
CO	Carbon monoxide
COPD	Chronic obstructive pulmonary disease
CRA	Comparative Risk Assessment

DBP	Dibutyl phthalate
DCB	Dichlorobenzene
DCPH	Dichlorophenol
DEP	Diethyl phthalate
DMP	Dimethyl phthalate
Dn-OP	Di-n-octylphthalate
DPH	Dimethylphenol
DSPE	Dispersive solid phase extraction
FAO	The Food and Agriculture Organisation
FLR	Flourene
FRT	Flouranthene
GBD	Global Burden for Disease
GCB	Graphitized carbon black
GC-MS	Gas chromatography-mass spectrometry
GLSS5	The 5th round of the Ghana living standards survey
HCB	Hexachlorobenzene
HDE	Hexanoic acid dioctylester
HOOAC	Acetic acid
HPA	Health Protection Agency
IEA	International Energy Agency
IHME	Institute for Health Metrics and Evaluation
LLE	Liquid -liquid extraction
LOD	Limit of detection
MDB	Methyldinitrobenzene
MeCN	Acetonitrile

MNBA	Methylnitrobenzenamine
MPH	Methylphenol
NAP	Naphthalene
NP	Nitrophenol

LIST OF FORMULAS

NaCl	Sodium Chloride
O_3	Ozone
$MgSO_4$	Magnesium Sulphate

CHAPTER ONE

INTRODUCTION

Chapter one provides background to the study; fish smoking and pollutants that are released during fish smoking. It also highlights problem statement, overall objective, specific objectives and significance of the study.

Background to the Study

Fish smoking, drying, salting, frying, fermenting and various combinations of these are traditional methods that are employed in fish preservation. However, fish smoking is the most popular post-harvest preservation method of fish preservation in Ghana and it is predominantly carried out by women in fishing communities.

According to (Douben, 2003), the absorption, dissolution and diffusion of smoke by fish is a physical process. Fish smoking's chemical processes are supposed to aid in the smoke's contact with the fish (Zakaria, Takada, Tsutsumi, Ohno, Yamada, Kouno and Kumata, 2002).

Over the years, several studies have established a few fundamental concepts of smoke formation and application (Cypel, Yeung, Machuca, Chen, Singer, Yasufuku, de Perrot, Pierre, Waddell and Keshavjee, 2012).

Wood used for fish smoking usually contains three basic polymers: cellulose, hemicellulose and lignin (Douben, 2003). Chemical bonds bind the three polymer groups together (Amos-Tautua, Inengite, Abasi and Amirize, 2013). The breakdown of these chemical bonds, which is triggered by vibrational energy, results in the generation of smoke, which is dependent on the variety, temperature and degree of degradation of the wood (Cypel *et al*., 2012).

During the burning of wood's constituents, a variety of chemicals are emitted (Yusuf, Ezechukwu, Fakoya, Akintola, Agboola and Omoleye, 2015). According to some research, wood contains over 300 chemicals, some of which are deposited on smoked foods (Kleter, 2004). Phenols, alcohols, lactones, carbonyls, furans, esters and polycyclic aromatic hydrocarbons are the most important chemicals found in wood, depending on the temperature of burning (Zakaria et al., 2002).

Food gains several good properties from smoking, such as color, preservation and flavor. Some volatile chemicals, such as formaldehyde, phenols and cresols have been shown to have antibacterial and antifungal properties, making them useful in the prevention of infections (Karam, Kadhim, and Alkaim, 2015). Formaldehyde has been linked to antibacterial, antifungal and antiviral activity. Despite the fact that smoke components are ineffective against spore-forming bacteria, the lingering effect has been observed to be stronger against bacteria than molds (Zakaria et al., 2002).

Smoke, on the other hand, has certain negative consequences, such as the breakdown of important amino acids in proteins. This contaminates food with the smoke's harmful components (Nkpaa, Wegwu and Essien, 2013). The most poisonous of all the components of wood smoke is PAH especially benzo[a]pyrene. Benzo[a]pyrene has been found to be carcinogenic in several investigations (Opel, Palm, Steffen and Ruck, 2011).

Indoor air is the air within an indoor environment. Most people in industrialised countries spend roughly 22 hours a day indoors, with 72.7 percent of their time spent at home, according to time activity diaries (Szalai, 1972). In most developing nations, exposure to indoor air pollutants such as

particulate matter, carbon monoxide, sulfur oxides and nitrogen dioxide is a major environmental and health concern (Yang and Holgate, 2013). Biomass combustion, according to the US Environmental Protection Agency (2004), causes significant indoor air pollution, particularly particulate matter (PM).

PM has been related to an increased risk of a variety of health problems, including acute respiratory infection, chronic respiratory disease and mortality. The range of affects induced by particulate matter exposure as a result of burning biofuels, according to Balakrishnan and Nigel (2006), is broad, impacting the respiratory and cardiovascular systems, extending to children and adults, as well as a number of large, sensitive populations.

Particulate pollution levels have been linked to an increase in the occurrence of respiratory symptoms and disorders, such as acute lower respiratory infections (ALRI) in children (Smith, Samet, Romieu, and Bruce, 2000), lung cancer (Mu, Liu, Niu, Zhao, Shi and Li, 2013), bronchial asthma, and chronic obstructive pulmonary disease exacerbations (Perez-Padilla, Regalado, Vedal, Paré, Chapela and Sansores, 1996)

Furthermore, epidemiological studies have connected indoor air pollution exposure to impaired pulmonary function, increased respiratory hospitalization and increased mortality (Sood, 2012).

The combustion of biomass fuels in poorly ventilated kitchens with poorly running stoves produces large amounts of respirable particles, gases such as carbon monoxide, sulfur dioxide and nitrogen oxides and toxic compounds such as benzene and formaldehyde (Ezzati, Saleh and Kammen, 2000).

According to Adeyeye and Oyewole (2016), fish smoking is the most extensively used method of fish preservation in most African countries, including Benin, Senegal, Sierra Leone, Liberia, Kenya, Uganda, Tanzania, Nigeria, Ghana, Cote d'Ivoire, Togo and others. According to Adeyeye and Oyewole (2016), almost any kind of fish collected can be smoked and it is believed that 70–80 percent of the domestic marine and freshwater catch is consumed in smoked form.

Chukwu and Shaba (2009) explained that smoked or dry fish forms part of traditional diet taken by a large section of the world's population. Fish smoking supports many families and households in the coastal communities of Africa with income and also as a source of food. Smoked fish has between three and six months' shelf-life and is able to preserve the nutritional value of the fish (Akande and Diei-Ouadi, 2010).

About 10 percent of Ghana's population engage in different aspects of the fishing industry with the fisheries sector having about 5.8% growth rate of GDP at 2006 constant prices (Ghana Statistical Service, 2014). The fisheries sector in Ghana includes aquaculture fishery, inland (fresh water) fishery and marine fishery, storage, preservation, distribution and marketing. Marine fish is a very important source of employment for the people living along the coast, while inland water in Ghana is mainly for domestic consumption (Fisheries Commission, 2010). The Food and Agriculture Organisation (FAO) (2010), adds that the fisheries sector is an important sector in Ghana's socio-economic development. Traditionally, most Ghanaians like smoked fish as it is perceived to have nice flavor and taste because of the smoke which gives it a competitive

pricing advantage over other perishable protein diets such as eggs, fresh fish and milk.

The fishery industry in Ghana with its processes such as; harvesting, handling, processing and distribution however, faces one major challenge; fish smokers in Ghana inhale a lot of smoke particles during fish smoking as a result of indoor biomass fuel usage (Obeng, 2018: Flintwood-Brace, 2016).

When indoor biomass fuel is burned, approximately 200 different chemical compounds are produced, with over 90% of them being inhalable. Gaseous air pollutants such as carbon monoxide, sulfur dioxide and nitrogen dioxide, as well as particulate matter air pollutants with aerodynamic diameters of less than 2.5 microns ($PM_{2.5}$) and less than 10 microns (PM_{10}), polycyclic aromatic hydrocarbons (PAHs), chlorinated dioxins, arsenic, lead, fluorine and vanadium, are all toxic to the human body.

The usage of biomass fuel for cooking and heating is projected to be used by half of the world's population (about 3 billion people). Smoke from the combustion of biomass fuels has been linked to a number of respiratory illnesses, including asthma, chronic obstructive pulmonary disease (COPD), respiratory tract infections, particularly in youngsters and the elderly and lung cancer.

In the rural fishing town of Obaka in Nigeria, Salvi and Barnes (2009) and Umoh and Peters (2014) found that exposure to biomass smoke from the fish smoking sector resulted in lung function declines and an increase in respiratory symptoms. In comparison to women who did not work in the fish smoking industry, peak expiratory flow rates (PEFRs), forced expiratory

volume in 1 second (FEV1)'+ and forced vital capacity (FVC) values were nearly 1.5 times lower in women who worked in the fish smoking sector.

Also, the prevalence of symptoms of chronic bronchitis was 68% among women who worked in the fish smoking industry, compared to 8% among those who did not work in the fish smoking industry. The mean FEV_1/FVC ratio in these women was 68%, indicating that a sufficiently large proportion of women in the fish smoking industry had significant airways obstruction.

The women who smoke fish use biomass fuel for cooking, indicating that additional exposure to biomass smoke in the fish smoking industry has a further worsening effect on lung health. This is likely because exposure to biomass smoke during cooking lasts for 2-4 h every day, while exposures to biomass smoke at fish smoking industries will be continuous for 24 h (Sundeep and Bill, 2014). Thus exposure to smoke could affect the health of fish smokers in Ghana.

Although fish smokers have a significantly increased risk of worsening of lung health and the risk of developing chronic obstructive pulmonary disease (COPD), it is important to realize that fish smoking may be the only source of livelihood for these people living in the coastal region or along river beds and it may therefore be difficult to give up this occupation for economic reasons.

Notwithstanding the health effects associated with fish smoking using biomass, the industry is small and does not come under the jurisdiction of regional occupational norms or even get recognition (Sundeep and Bill, 2014).

Fueled with poor access to healthcare facilities, health hazards related to indoor air pollution are likely to remain unnoticed in this population until later stages. It is worth noting that air pollution, including those from fish smoking is one of the environmental problems that scientists, policy makers and citizens are concerned about globally.

Many "researchers have recently focused on air quality and different studies on air quality and its effects on human health and ecological integrity have been conducted. Most of the studies are carried out in urban areas to assess the quality of air due to urbanization and population growth and their association with increased domestic and vehicular fuel usage as well as industrial growth."

When reporting, it is common to make a distinction between interior and outdoor air quality to avoid mixing up the results. For some pollutants, such as nitrogen dioxide (NO_2), sulfur dioxide (SO_2), chlorine gas (Cl_2) and carbon dioxide (CO_2). Taneja, Saini and Masih (2008) proposed that outdoor concentrations of air pollutants may be used to estimate indoor contamination (CO_2). They did, however, suggest that interior air pollution may be worse than outside air pollution due to a variety of reasons, including pollutant sources and the wind's dispersive effect.

In "Ghana, toxic gases from old and rickety cars, waste fires, road dust, and soot from biomass-fueled cook stoves continue to be a major cause of premature death, with hundreds dying each year (WHO, 2012). Every year, air pollution causes roughly 7 million premature deaths worldwide, with Africa and Asia bearing the brunt of the burden (http://web.unep.org/environmentassembly/air, 2018). Ghana's yearly urban outdoor air (31.1 g/m3) exceeds

WHO recommendations for PM $_{2.5}$, which are ultra-fine particles with a diameter of 2.5 or less that can clog human lungs (the WHO's recommended yearly PM $_{2.5}$ guidelines is 10 g/m^3). In Ghana, air quality monitoring is confined to only 15 places, all of which are inside the Greater Accra Region. None for the remaining 15 regions of the country (https://asic.aqrc.ucdavis.edu/8, 2018). This lack of sufficient air quality monitoring networks could be contributing to Ghana's poor air" quality.

Indoor air pollution as a result of biomass fuel usage is a challenge among fish smokers, their children and could affect their health over a long exposure time.

When the physical, chemical and biological properties of air are altered, air pollution occurs. Air pollution has negative consequences for humans and other living things. Air pollutants are the compounds that contribute to air pollution (Sinha and Nag, 2011).

The risk of stroke, heart disease, lung cancer and chronic and acute respiratory disorders such as asthma are all health implications of air pollution (WHO Global Urban Ambient Air Pollution, 2016b). The majority of these health impacts are linked to the combustion of solid fuels, which produces hazardous particles known as solid fuel "smoke." Smoke is the most common cause of indoor air pollution, contributing to the burden of disease. Indoor air pollution from biomass has been designated as a possible human carcinogen by some organizations, such as the International Agency for Research on Cancer (IARC). Indoor smoke from solid fuel combustion has also been identified by the World Health Organization (WHO) as one of the top 10 risks for global burden of disease, accounting for 2.7 percent of the global burden of

disease and 2 million premature deaths each year from acute lower respiratory infections, chronic obstructive pulmonary disease (COPD) and lung cancer.

Furthermore, most solid-fuel-using households in the developing world are from lower socioeconomic groups and are primarily located in rural and isolated communities, where basic health-care facilities are severely lacking. Cooking using biomass fuels including wood, crop residues, dung, charcoal and coal over open flames or in rudimentary stoves exposes family members to daily pollutant concentrations that fall in between secondhand smoke and active smoking (Pope, Brook, Burnett and Dockery, 2011: Smith and Peel, 2010).

Over 2 million people were expected to die prematurely from pneumonia, chronic obstructive pulmonary disease and lung cancer as a result of cooking with biomass fuel on open flames (Smith and Tager, 2004; WHO 2009).

The Global Burden of Disease, GBD (2010) research, published in 2012, estimated that household air pollution is responsible for 3.5 million premature deaths worldwide, as well as other health consequences like as cataracts and cardio vascular disease, based on household fuel estimates (Lim, Vos, Flaxman, Danaei and Shibuya, 2012). Household cooking fuels, according to the GBD (2010), contributed significantly to outdoor air pollution in numerous regions, resulting in around half a million more premature deaths (Lim et al., 2012).

Additional health effects of home solid fuel consumption include poorer pregnancy outcomes (Pope, Mishra, and Thompson, 2010), the danger of burns and scalds. There is also the danger of injury and aggression during

fuel collection (WHO, 2006), as well as the contribution to ambient (outdoor) air pollution. Many solid fuel users are forced to spend time obtaining fuel that could otherwise be spent earning money, caring for their children, or attending school (WHO, 2006).

Furthermore, inefficient solid fuel usage in households has significant environmental and global climate change consequences (Edwards, Smith, Zhang and Ma, 2004: Smith, Uma, Kishore, Zhang and Khalil, 2000). Given the public health, social and environmental ramifications of household solid fuel consumption, policymakers in a range of sectors must keep track of present rates and developments (e.g., energy, environment, health).

The indicator "solid fuel utilization" (SFU) is utilized as an input for calculating health consequences in the GBD 2010's Comparative Risk Assessment (CRA) (Institute for Health Metrics and Evaluation (IHME), 2012).

In the World Health Statistics series, the use of solid fuels is documented (WHS).

Smoke exposure is also influenced by the types of ovens used for smoking fish. For centuries, solid fuel cook stoves have been used as the major energy source for home cooking and heating, and the practice is still prevalent, particularly in underdeveloped nations (Dang, 2016).

The majority of rural homes burn biomass fuels in inefficient clay or metal stoves, or cook over open fires in poorly ventilated kitchens, resulting in extremely high levels of indoor air pollution. It is estimated that using these fuels to light open fires exposes almost 2 billion people throughout the world to elevated particulate matter and gas concentrations that are up to 10–20

times higher than health-based guideline levels for typical urban outdoor concentrations (WHO, 1999).

According to Bonjour, Adair-Rohani and Wolf (2013), it is estimated to be 46 percent and 35 percent, respectively. In the Caribbean and Latin America, roughly 16% of dwellings use solid fuels, with significant variances between nations. According to a study conducted in Guatemala, solid fuels are consumed by 62 percent of the population. This means that 88 percent and 29 percent of the population lives in rural and urban areas, respectively. In Mexico, solid fuels are used by 15% of the population, with 5% in urban regions and 45 percent in rural regions (World Health Organization, 2012).

Solid fuel utilization, on the other hand, is below 5% in most developed countries. Poverty is caused by increased usage of solid fuels in countries, communities within countries and households within communities (Perez-Padilla, Schilmann and Riojas-Rodriguez, 2010: Lim and Seow, 2012).

In Nepal, it is estimated that roughly 80% of families utilize biomass as their primary source of domestic energy (Central Bureau of Statistics, 2011), with cooking taking place on open fires with minimal ventilation. Domestic PM levels exceeded World Health Organization (WHO) and National Ambient Air Quality Standards, according to Devakumar, Semple, Osrin, Yadav, Kurmi, Saville, Shrestha, Manandhar, Costello and Ayres (2014a) in a study in the Himalaya valley region and Dhanusha District. Domestic PM levels also exceeded the US EPA (Environmental Protection Agency) outdoor air quality index, according to Kurmi, Semple, Steiner, Henderson and Ayres (2008).

Air Pollution as a Result of Biomass Usage

In Athens, air pollution is an important environmental concern due to combustion processes. Air pollution in Athens occurred as a result of over population and the confinement of commercial and industrial activities (Kalogridis, Vratolis, Liakakou, Gerasopoulos, Mihalopoulos and Eleftheriadis, 2018). Domestic wood burning in Athens suffers from low burning efficiency. The extensive use of wood burning has resulted in considerable emissions from incomplete combustion, such as CO, hydrocarbons and soot particles.

For domestic cooking, heating and lighting, biomass fuels such as wood, crop leftovers and animal dung are used (Bruce, Perez-Padilla and Albalak, 2000; Rehfuess, Mehta and Prüss-Üstün, 2006).

Furthermore, research by Fullerton et al. (2008) and Smith (2002) found that solid fuel burning emits dangerous airborne pollutants like particulate matter (PM), carbon monoxide (CO) and other toxic organic compounds. Wood is the most common solid fuel. There are also agricultural residues, animal waste (dung) and charcoal, as well as leaves and grass. These fuels are typically derived from the environment in rural areas, while they are acquired from marketplaces in metropolitan areas.

Traditional biomass accounts for about 10% of all primary energy usage on the planet. About 2.7 billion people utilize biomass fuels to cover their residential energy needs around the world. By 2030, around 2.8 billion people are expected to consume biomass. This indicates that solid fuel consumption is expected to stay stable in the future (International Energy Agency (IEA), 2010). The percentage of people who cook at home using solid

fuels varies substantially between countries (urban and rural areas) and regions. According to Perez-Padilla et al. (2010), household usage of solid biomass fuels is the most prevalent source of indoor air pollution worldwide. Solid fuels are commonly used for cooking and domestic heating in developing countries, particularly in rural areas.

The consequences of using biomass as a fuel are numerous. When solid fuels are burned inefficiently, a complex mixture of particulate and gaseous species is produced. The gases released by solid fuel-burning stoves contain over 60 hydrocarbons, as well as 17 aldehydes and ketones. Compounds that are carcinogenic are among the gas-phase contaminants (benzene, formaldehyde, 1, 3-butadiene and styrene). Incomplete combustion of wood produces two- to four-membered aromatic rings, which are released in gaseous form when solid fuels are burned (Sinha and Nag, 2011).

"Burning biomass in traditional stoves, open-fire three-stone stoves or other low-efficiency stoves, according to Balakrishnan et al. (2004), emits smoke containing large quantities of harmful pollutants, with serious health consequences for those exposed, particularly women involved in cooking and young children spending time around their mothers."

In Africa, air pollution from biomass fuels is a major source of mortality and morbidity in both adults and children.

In Ghana, biomass is the major source of fuel used by fish smokers and often in environments without chimneys where the smoke is uncontrolled. Fish smokers spend on average six hours a day going about their activities which expose them to pollutants from firewood smoke. There is a direct health risk as they are exposed continuously to indoor air pollution due to biomass

smoke during fish processing of which women and children are the most affected (http://ugspace.ug.edu.gh).

According to von Schirnding, Bruce, Smith, Ballard-Tremmeer, Ezzati and Lvovsky (2002) fish smoking, a non-farm income generating activity contribute immensely to Ghana's labour force as many people in the country are not engaged in formal employment.

For instance, the 5th round of the Ghana living standards survey (GLSS5) shows that about 46.4% of the households in Ghana practice some form of non-farm enterprise (Ghana Statistical Service, 2014), including fish smoking. This percentage translates into almost about 3.2 million people, with seventy-two percent being women (Ghana Statistical Service, 2014). Women constitute about 95% of labour force to the fishery industry as well as other post-harvest processing activities like fish smoking (Torell, Owusu and Okyere Nyako, 2015).

Statement of the Problem

Indoor "air pollution cause between 1.6 and 2 million deaths each year in developing countries (Smith, 2000). In developing countries, about 50% of people, rely on coal and biomass in the form of wood, dung and crop residues for domestic energy. These materials are typically burnt in simple stoves with very incomplete combustion. The resulting exposure to indoor air pollution (IAP) accounts for a significant portion of the global burden of death and disease and disproportionately affects women and children in developing" regions.

Even though the contributions of women to household and national income is huge, they normally work under very bad conditions which put their

health at risk (Torell et al., 2015). Fish smokers in Ghana are exposed to smoke on average six hours a day when the peak harvest season is on (United Nations Development programme (UNDP), 2008). Most fish smokers work within indoor smokehouses in concentrated smoke environments which could expose them to toxic compounds and particulate matter from incomplete combustion of solid fuels which can contribute to respiratory diseases.

Smoking fish with traditional methods require a lot of firewood as fuel, with the production of large volumes of smoke during fish smoking in commercial quantities. This leads to the production of harmful pollutants and gases like particulate matter (PM) of different diameters and polycyclic aromatic hydrocarbons (PAHs) that could cause cancers and defects to the human respiratory system. A lot of health concerns could be attributed to smoke that come out as a result of using traditional kilns or ovens. It could also lead to eye diseases if fish smokers are exposed to smoke for a long time.

Motorykin, Schrlau, Jia, Harper, Harris and Harding (2015) determined Parent and Hydroxy PAHs in Personal PM $_{2.5}$ and urine samples collected during Native American fish smoking activities. Essumang et al. (2013) also worked on the effect of smoke generation sources and smoke curing duration on the level of PAHs in different suite of fish. Furthermore, Nwaichi and Ntorgbo (2016) assessed the levels of PAHs in commonly consumed and commercially viable and sea food species.

However, limited amount of studies has been done on effect of smoke exposure on fish smokers in Ghana through inhalation during their fish smoking activities. It is against this background that the research topic ‘indoor air pollution monitoring for urinary polycyclic aromatic hydrocarbons,

phenols, phthalates and some metabolites of fish smokers from three coastal regions of Ghana was done to determine the impact of smoke on health of fish smokers.

Purpose of the Study

The purpose of the study is to determine indoor air pollution and urinary PAHs, phenols, phthalates and some metabolites of fish smokers from three coastal regions communities of Ghana.

Research Questions

1. Does exposure duration to smoke affect the health of fish smokers?
2. Does the levels of parent PAHs, metabolites and phenols of fish smoking pose any health risk to fish smokers?
3. Are the levels of personal CO and personal $PM_{2.5}$ among fish smokers within recommended standards?

Objectives for the Study

The objectives are to:

1. quantify levels of parent PAHs, metabolites, phenol and phthalates in urine samples of fish smokers.
2. measure micro-environment indoor levels of $PM_{2.5}$, CO and ozone in the Western region.
3. determine the link between the length of time fish smokers are exposed to smoke and the development of eye illnesses and other health problems.
4. measure 48-hr personal levels of $PM_{2.5}$ and CO for fish smokers in Central and the Volta regions

5. determine the link between personal CO and PM $_{2.5}$ exposure and urine PAHs and metabolites

Hypothesis

The study seeks to test the following hypothesis in attempt to achieve the specific objectives stated:

H1: The levels of parent PAHs, metabolites, phenols and phthalates in the urine samples of fish smokers do differ significantly from EPA or WHO values.

H2: The levels of CO, PM $_{2.5}$ and ozone in the smokehouses differ significantly from EPA or WHO values.

Significance of Study

This study is significant in that it contributes to understanding the effects of exposure to smoke particles by fish smokers in Ghana due to inhalation resulting from their fish smoking activities thereby adding to scientific knowledge. The study could be useful to researchers to develop technologies to improve ovens that could filter smoke effectively as biomass fuel is being used in fish smoking communities. Also the study could be useful to non-governmental organisations (NGOs) who engage the fish smokers to provide personal protective equipment, workshops on proper and hygienic ways of handling fish and safety practices in the smokehouses. In addition, the study would provide the Fisheries sector with information to enable it to assist fish smokers with various institutional interventions. Furthermore, the study would encourage the leaders of the fish smokers to collaborate with occupational health safety experts in capacity building of their members on health and safety issues. The leaders of the fish smokers could also encourage

the fish smokers to buy modern ovens which has the potential of reducing their exposure to smoke.

Delimitations

Urine sample were collected from women in Central, Volta and Western Regions.

Limitation

Due to some constraints, personal CO and $PM_{2.5}$ could not be measured for fish smokers in Abuesi.

Organisation of the Study

This thesis is divided into five chapters. The methodology employed in this study is quantitative method. Chapter One covers the background of the study and explains the health effects associated with biomass fuel usage and some traditional ovens used by fish smokers. The problem statement in Chapter One states the actual problem of this study; effect of smoke exposure on fish smokers in Ghana through inhalation during their fish smoking activities.

Other sections such as general objective, specific objectives and significance of the study are also discussed in chapter one.

Chapter Two is the literature review. Concepts such as sources, health effects and mechanism of PAHs, CO and $PM_{2.5}$ in humans are reviewed in Chapter Two. Method of analysis (QuECHERS approach) and instrument used (GC-MS) were also reviewed.

Chapter Three of this study covers research methods. Cross-sectional design was used for Abuesi in the Western Region and purposive sampling was also used for both Central and Volta Regions. The study areas,

population, sampling procedure, data collection instrument, data collection procedure, data processing and analysis were discussed.

Chapter Four in this study highlights results and discussion. The levels of urinary (PAHs, metabolites, substituted benzenes, phenol and phthalates) in samples fish smokers in Western, Volta and Central regions were discussed in Chapter Four. The demography of fish smokers in Abuesi, association between exposure duration and self-reported health outcomes were also discussed. Results obtained after eye screening among fish smokers at Abuesi were discussed. Furthermore, results obtained from indoor air pollution monitoring in Abuesi, risk assessment and personal $PM_{2.5}$ and personal CO for both Central and Volta Regions have been discussed.

Chapter Five talks about conclusion, recommendation and summary. Conclusions based on specific objectives of this study were made and summarized in a general conclusion based on the general objective of this study. Some further studies and institutional interventions were finally recommended.

Chapter Summary

Indoor air pollution as a result of biomass fuel usage is a challenge in Ghana particularly among fish smokers. The health effects of smoke that could affect fish smokers and the need to assess urinary PAHs and monitor indoor air pollution in smokehouses have been captured under Chapter One.

CHAPTER TWO

LITERATURE REVIEW

Introduction

Chapter Two will look at biomass fuel usage which is a challenge to fish smokers in some coastal regions of Ghana. Some pollutants that are released in smokehouses such as PAHs, particulate matter, carbon monoxide, ozone and benzene would also be explained. In addition, analytical method (QUECHERS approach) and GC-MS instrument also would be reviewed.

The ways in which fish is being smoked pose a challenge. For different traditions, the science and manner of smoking varies, but the concept stays the same. In the past, smoking was done on a commercial basis in a smoke house. The smoke is produced by burning biomass wood on an iron device with a protective roof at a depth of around 180 cm underneath the oven. This is to prevent the oils from igniting when they drip onto the wood fire.

The purpose of the protection roof is to reduce the creation of PAHs, which are known to have carcinogenic qualities and are associated to partial fat burning. This technique has the advantage of changing the smoke's flow pattern as it rises, therefore covering a large region. Because the smoke does not come into direct contact with the product, the temperature and content of the smoke is lowered (Dennis, Massey, McWeeny, Knowles and Watson, 1983).

Traditional ovens, which were rectangular or cylindrical in shape and built of metal or mud in the 1960s, were mostly used for smoking fish products (Essumang, Dodoo and Adjei, 2013). This system encouraged too much hand-holding of fish throughout the smoking process, which was a

serious flaw. Traditional ovens have limited capacity and they could not handle the massive quantities of fresh fish harvested during peak seasons (Nkpaa et al., 2013). Increased post-harvest losses resulted as a result of this phenomena.

Steiner et al. (1992) also brought to light the inefficiency of these ancient ovens in terms of fuel consumption, as well as several health implications such as preventable eye and lung infections that the operators were exposed to as a result of the high levels of smoke.

The cylindrical metal oven is made by welding two 44-gallon metal oil drums that have been opened at the basal segment with a stokehole. This design has a circumference of approximately 115 cm and a height of approximately 90 cm. It has a stokehole that is 40 x 40 cm in size. Unlike the mud oven, which relied on sticks for support, the cylindrical metal oven has 60 cm long built-in iron rods. It has a stokehole that is 40 x 40 cm in size. Unlike the mud oven, which relied on sticks for support, the cylindrical metal oven supports the fish with built-in iron rods that extend 60 cm from the drum's bottom (Nti, Plahar and Larweh, 2002).

In Ghana and other African countries, cylindrical ovens are commonly utilized (Agbozu, 2014). It has the benefit of being lightweight and portable. The cylindrical oven has the problem of being easily corroded.

One of the most popular ovens used by fish smokers is the rectangular mud oven, which is similar to the original cylindrical mud oven. Despite the fact that the rectangular mud oven is made of mud, it is supported by heavy iron bars that support the layers of smoked fish (Steiner, Burtscher and Gross ,1992). It has a stokehole all the way down the middle. Iron rods hold the fish

in place on wire mesh. In Ghana's Greater Accra and Volta regions, the rectangular mud oven is the most common.

Some of the drawbacks of using a rectangular mud oven include the difficulty in controlling the hot wire mesh during the smoking process, as well as the low intake capacity and rapid heat loss during operation (Opel et al., 2011).

The rectangular/square metal oven is made out of 44-gallon steel oil drums that have been split open and welded together to form a rectangular and in some cases square oven (Perry, Campiglia and Winefordner, 1989). To support the fish, which are arranged on wire mesh, metal rods are positioned at the bottom of the structure. Due to radiation, this oven loses a lot of heat (Kleter, 2004).

Fish smokers in Ghana's central and western regions mostly use cylindrical mud ovens. A typical Fante oven has an internal diameter of about 105 cm, an external diameter of 132 cm, and a height of about 80 cm (Amuna, 2014). A ledge 50 cm from the oven's base serves as a holder for the sticks that hold the fish layers in place (Amuna, 2014). Two disadvantages of cylindrical mud ovens are reshuffling during smoking and fish destruction by the sticks that support them. It quickly deteriorates, lasting only two fishing seasons (Steiner-Asiedu et al., 1991).

Chorkor smoker ovens are used in most of the fish smokers in this work. The chorkor smoker, according to Nunoo, Etornam Tornyeviadzi, Asamoah, and Addo (2018), is an improvement over the traditional fish smoking oven that is currently in use in Ghana. The Chorkor smoker appealed to traditional fish smokers. It has a combustion chamber and trays for

smoking. The combustion chamber is rectangular in shape, twice as long as it is wide, with two stokeholes in front and a wall running the length of it.

A height of 60 cm, a wall thickness of 12.5 cm, a breadth of 12.5 cm, a length of 225 cm, a breadth of 112.5 cm, a height of 37.5 cm for the stoke hole and a depth of 15 cm for the fire pit are all requirements for the combustion chamber. The base of the smoker is where the combustion chamber is. It is usually made of mud, but it can also be made of cement blocks or burned bricks. The cost of cement and burned bricks is higher than that of mud. Because it cannot withstand the high temperatures of smoking, cement is not recommended. The top of the wall should be flat to ensure that the trays fit flush against the wall and that no smoke or heat escapes through gaps.

The trays' wooden frame runs parallel to the midline of the smoker's base walls, ensuring that they are well supported and don't catch fire. The smoking unit is comprised of 5 to 15 smoking trays. The size and quality of the smoked fish play a role in this (Adeyeye and Oyewole, 2016). For small to medium-sized fish, ten trays in a single oven are used on average.

The Chorker smoker, on the other hand, has some drawbacks: the smoking process necessitates a large amount of fuel wood, which may result in deforestation. It is also a bit of a splurge. The fact that they must work close to the smoker and turn the fish on a regular basis puts the fish processors' health at risk. For example, Nunoo et al. (2018) discovered higher levels of PAHs in wood smoked fish samples after using a Chorkor oven and a gas oven to smoke fish.

According to Akpambang, Purcaro, Lajide, Amoo, Conte and Moret (2009), smoking fish directly with fuel wood at high heating temperatures may be a contributing factor to higher PAH levels in processed meals.

Essumang, Dodoo and Adjei (2012) discovered higher levels of benzo [a]pyrene (BAP) in wood smoked fish samples. Smokers of fish may be exposed to smoke particulates because they use Chorkor ovens in indoor smoke houses.

Biomass Fuel Production and Effects of PAHs

Biomass fuel is any material derived from plants or animals that is burned for fuel by humans. The most prominent example is wood, but animal excrement and crop waste are also prevalent (Bruce et al., 2000). Biomass fuel provides for more than half of home energy in many developing countries and it can account for as much as 95 percent in low-income countries (Smith et al., 2004). Around 2.4 billion people utilize it as their primary source of domestic energy for cooking, heating and lighting (Reddy et al., 1996: Smith et al., 2004).

It is estimated that 1.9 million individuals die prematurely as a result of solid fuel combustion smoke (Kurmi, Lam and Ayres, 2012). According to the World Health Organization, exposure to smoke from solid fuel burning is the leading environmental risk factor in the world, accounting for 3.3 percent of all deaths and 2.7 percent of all disability-adjusted life years per year.

Chronic obstructive pulmonary disease (COPD), higher risk of acute respiratory infections/pneumonia, lung cancer, tuberculosis (TB) and cataracts are all linked to long-term exposure to solid fuel smoke (Kurmi, Semple, Simkhada, Smith and Ayres, 2010).

Modernization in developed countries has been accompanied by a shift away from biomass fuels like wood and toward petroleum products and electricity, whereas in developing countries, even where cleaner and more sophisticated fuels are available, households frequently continue to use simple biomass fuels (Nti et al., 2000).

The negative health consequences of indoor air pollution from biomass fuel consumption are sometimes exacerbated by a lack of ventilation in such homes, as well as the poor design of stoves that lack flues or hoods to remove smoke from the living space. The smoke produced may contain PAHs, which are directly ingested by the occupants of the residence. In cultured human cells, biomass smoke can cause DNA damage as well as inflammatory and oxidative stress response gene expression.

The inhalation of biomass smoke has been recognized as a probable factor for COPD development. Pandey, Bajpayee, and Parmar (2005) found DNA damage in lymphocytes of women exposed to biomass smoke, as well as decreased macrophage function, in an Indian study.

Biomass Fuel and Air Pollutants

Incomplete biomass fuel combustion results in the release of hazardous particles such as particulate matter, carbon monoxide and nitrogen dioxide, all of which are damaging to human health (Bruce, Perez-Padilla and Albalak, 2000: Naeher, Brauer, Lipsett, Zelikoff, Simpson and Koenig, 2007).

The World Health Organization (WHO) has identified certain biomass combustion gases that are harmful to human health. Some of the categories include particulate matter, polycyclic aromatic hydrocarbons (PAH), carbon monoxide (CO), aldehydes, organic acids, semi-volatile and volatile organic

chemicals, nitrogen and sulphur-based chemicals, ozone and photochemical oxidants, inorganic portion of particles and free radicals. Indoor air pollution caused by cooking is recognized to be linked to a combination of fuel types, food components and cooking practices. They, like many other hazardous chemicals generated from food items by fire-based cooking, are released in the form of smoke and particles from cooking fuel (Kim, Jahan and Kabir, 2011). Among the contaminants found were volatile organic compounds (VOCs), carbonyls, carbon monoxide (CO), nitrous oxides, sulfur oxides (mainly from coal), formaldehydes and polycyclic organic matter (e.g., carcinogenic benzo[a] pyrene) (Kim et al., 2011).

Coal is also commonly utilized in China, Korea, South Africa and a few other countries for domestic reasons. Indoors, these fuels are burned over open flames in inefficient stoves, which could be a primary channel for delivering large amounts of indoor pollutants to chefs and their families because most of these stoves' combustion is incomplete.

Biomass and PAHs

The mechanisms of PAH creation during the combustion of organic matter are poorly understood, particularly the fundamentals of PAH creation, which occurs when the radicals created by high-temperature combustion recombine to create PAH at lower temperatures (Sumpter and Chandramohan, 2013). PAHs such as acenaphtene, dibenz [a.h]anthracene, fluoranthene, naphthalene, phenanthrene and pyrene have been linked to major health problems in humans (Kurmi, Lam and Ayres, 2012).

Benzo[a]pyrene is one of the PAHs contributing to cancer development in human cells. PAH compounds were found in wood burning

(Diette, Accinelli, Balmes, Buist, Checkley, Garbe and Yip, 2012). More than 30 PAHs were discovered by Zhang and Smith (2007) when different types of wood were burned. Obstructive lung disease has also been associated with exposure to burning biomass fuel in the indoor environment.

Several of these PAHs, such as benzo[a]anthracene, benzo[a]pyrene, and cyclopenta[c,d]pyrene, have been discovered as mutagenic and genotoxic (Clark, Cooper, Stapleton and Di Giulio, 2013). Fluorene, phenanthrene, anthracene, fluoranthene and pyrene accounted for more than 70% of the mass of PAHs in birch wood burning, according to Diette et al. (2012). Another study revealed PAH and genotoxic PAH values of 11,508 g/kg and 953 g/kg, respectively, in wood burning emissions (Clark et al., 2013). A study also discovered total PAHs at 110,200 g/kg and genotoxic PAHs at 13,400 g/kg (Kurmi et al., 2012).

The characteristics of PAH emission from biomass combustion as a function of wood type and combustion appliances were investigated (Li, Sjoedin, Romanoff, Horton Fitzgerald and Eppler, 2011) and moisture (Shen, Huang, Wang, Zhu, Li, W., Shen, Wang, Zhang, Chen, Lu, Chen, Li, Sun, Li, Liu, Liu and Tao, 2013). The results showed that these factors influenced PAH emissions from biomass burning.

In summary, fish smokers could be exposed to a lot of respiratory and eye disease due to the use of biomass fuel, poor ventilation in enclosed or indoor smokehouses, lack of chimneys in smokehouses, lack of protective apparel and closeness of smokehouses to bedrooms or houses apart from smokehouses.

Effects of Traditional Fish Smoking and Toxicity of PAHs

Traditional fish smoking has been practiced in numerous countries since antiquity, according to (Dore, 1993). According to Basak, Engör, and Karakoç (2010), this was done to preserve the fish in part by drying it and in part by adding naturally occurring anti-microbiological elements such as phenols, acetic acids and a variety of polycyclic aromatic hydrocarbons created by wood combustion. This method is still frequently employed in both regulated (modern) and uncontrolled (traditional) kilns.

According to Nti, Plahar and Larweh (2002), the traditional kiln operates at a wood burning temperature of 300-700 0C and an oven temperature of usually over 80 degree Celsius. Smokers of fish may be subjected to extreme temperatures as well as harmful chemicals like PAHs. As a result, research into the effects of smoke inhaled by fish smokers who use biomass fuel is required. Wood smoke produced during fish smoking is an example of incomplete combustion, according to Stolyhwo and Sikorski (2005) and polycyclic aromatic hydrocarbons (PAHs) are formed and released into the various smoked products. PAHs are a well-known class of pervasive ecotoxicants that are hazardous to human health, with some of them being known to be highly carcinogenic and mutagenic (Tfouni and Toledo, 2007).

PAHs could also be released into the environment via vehicle exhaust gases (Adetona, Sjodin, Zheng, Romanoff, Aguilar-Villalobos and Needham, 2012) and cigarette smoke (Adetona, Sjodin, Zheng, Romanoff, Aguilar-Villalobos and Needham, 2012: Zhong, Carmella, Upadhyaya, Hochalter, Rauch and Oliver, 2011).

Many studies, such as Veyrand, Sirot, Durand, Pollono, Marchand, Dervilly-Pinel, Tard, Leblanc and Le Bizec, 2013; Essumang et al., 2012; Alomirah, Al-Zenki, Al-Hooti, Zaghloul, Sawaya and Ahmed (2011), found that human exposure to PAHs occurs primarily through ingestion of food containing high PAH concentrations, such as mollusks, crustaceans, oil, margarine smoked or grilled meat. The majority of the studies mentioned above were done on food samples.

To add to the body of knowledge on PAHs, this study looked at how fish smokers working on food samples are impacted by PAHs by examining their urine samples and a questionnaire based on self-reported health complaints, which would provide more clarity and aid in making educated judgments.

The findings will also assist policymakers in developing appropriate practices, clothes, stoves and procedures to assist fish smokers and improve the livelihood of some Ghanaians who smoke fish.

Biomass Fuel Usage in Ghana

There are many factors that influence biomass fuel choice but cost and socioeconomic position appear to be the most important.

Kurmi, Lam and Ayres (2012) point to the lack of clean fuels in rural locations as another key factor. This could be due to a lack of a long-term supply chain as well as the infrastructure needed to bring clean fuels to rural communities. Industry suppliers practically bring wood and other biomass fuels to their clients' doorsteps in the case of biomass fuel.

As a result, clean fuel is either unavailable or cannot meet demand on a regular basis. Rural dwellers, as well as women who smoke fish, will have to rely on biomass fuels permanently as a result of this.

According to the World Resources Institute, the United Nations Environment Programme, the United Nations Development Programme and the World Bank (1998), approximately 2.5 to 3 billion people or about 90% of rural families in developing countries, rely on biomass fuels for their household energy needs in traditional ways. As a result, solid fuels are largely used in developing countries, where households choose to cook and heat using wood and crop leftovers (Balakrishnan et al., 2004).

According to WHO (2005), biomass is used as a fuel in various countries, including India, Nepal, Pakistan and Sri Lanka (72 percent, 88 percent and 67 percent, respectively) for everyday family cooking. Because biomass fuel has a low combustion efficiency, it produces a high amount of incomplete combustion products, which are more hazardous to human health.

The following four factors are usually the most essential in a household's fuel selection: (a) fuel cost, stove type and fuel accessibility; (b) technical aspects of stoves and cooking methods; (c) cultural preferences; and (d) potential health consequences, if applicable (Masera, Saatkamp and Kammen, 2000).

Association between Biomass Fuel Usage and Health Effects

Primary air pollutants are created by direct activities such as carbon monoxide from automobiles, sulfur dioxide from factories or volcanic eruptions, whereas secondary pollutants are created when primary pollutants interact with the air.

Solid biomass fuel is one example of a secondary air pollution. Wood, agricultural wastes, animal manure, bushes, coal, charcoal, grass, straw and other materials fall into this category. More than 80% of individuals living in cities are exposed to air that is of low quality and exceeds WHO guidelines (Sinha and Nag, 2011).

Air pollution is a global issue because it affects every country on the planet. Air pollution, on the other hand, has a significant influence in low-income developing countries. According to the most recent Urban quality database, 98 percent of cities or 3000 cities in 103 poor and medium income countries with more than 100,000 people, do not fulfill WHO air quality recommendations. In high-income countries, however, it is only 56% (Sinha and Nag, 2011).

Indoor air pollution, according to (De Maeseneer and Maeseneer, 2009), may be responsible for around 2 million extra deaths in underdeveloped countries and 4% of the worldwide burden of disease. Because their metabolic mechanisms are undeveloped and immature, children are particularly vulnerable to indoor biomass (Bruce et al., 2000).

Non-respiratory disorders such as low birth weight in neonates, nutritional deficiencies, cardiac events, stroke, eye disease and nasopharyngeal and laryngeal malignancies may be linked to these activities (Kim et al., 2011).

The health effects of both acute and long-term exposure to air pollution are considerable, but those from long-term exposure are substantially bigger than those from short-term exposure, implying that damage is caused not merely by exacerbated diseases but also by their progression.

Long-term exposure to fine airborne particulates is linked to an increased incidence of premature mortality due to cardiovascular disease, chronic obstructive pulmonary disease and lung cancer, according to numerous follow-up studies that account for other factors such as tobacco smoking, diet and physical activity.

The strongest evidence for public health concern comes from fine particulate matter and gases (mainly carbon monoxide, ozone, nitrogen oxides, sulfur dioxide and volatile organic compounds) (WHO, 2014). Due to particle deposition in smaller airways and alveoli in the lungs, as well as their penetration into the bloodstream, fine particulate matter, which can be found both indoors and outdoors, causes more harm to people's health than any other air pollutant (Dust and pollen, for example, are limited to the thoracic cavity and are unable to travel via the narrower airway passageways.)

Fine particulate matter being examined includes carbon and organic molecules, transition metals and metal compounds, inorganic sulfates and nitrates, ammonia, sodium chloride and mineral dust. Absorbing particles can affect lung function and the cardiovascular system by causing endothelial cell activation and malfunction, as well as heart failure and ischemia, due to oxidative stress, changes in the electrical processes of the heart and systemic inflammation (WHO, 2013).

In Papua New Guinea, patients with chronic lung disease were found to have lived in communities substantially polluted by indoor biomass smoke. Despite smoking less, women were shown to have a higher risk of chronic bronchitis in a Pakistani study (Ranabhat, Kim, Kim, Jha, Deepak and Connel, 2015).

Women who cook with biomass fuel have a greater prevalence of COPD-related respiratory symptoms and a substantially steeper decline in lung function than women who do not, according to study conducted in Nepal, India, Saudi Arabia, Turkey and Mexico.

According to a study conducted in Nepal, biomass smoke caused much more respiratory illnesses than cleaner fuels. Cleaner fuels were also linked to fewer respiratory symptoms among women in a number of randomized control trials conducted in rural Mexico (Romieu, Riojas-Rodriguez, Marron-Mares, Schilmann, Perez-Padilla and Masera, 2009).

Furthermore, research has revealed that biomass fuel smoke causes COPD in non-smoking women in rural settings. After controlling for any confounding factors, the share of COPD related to biomass smoke has been estimated to be 23.1 percent in women from rural Turkey (Ekici, Ekici, Kurtipek, Akin, Arslan, Kara, Apaydin and Demir, 2005).

In epidemiological studies from India and Nepal, indoor cooking with biomass fuel has been associated to blindness or cataracts (Saha, Kulkarni, Shah, Patel and Saiyed, 2005).

Smoking promotes oxidative stress, which depletes plasma ascorbate, carotenoids and glutathione, all of which serve as antioxidants and help to prevent cataract formation. Women who cook with biomass fuel have a higher frequency of COPD-related respiratory symptoms and a much greater loss of lung function than women who do not, according to studies conducted in India, Saudi Arabia, Turkey, Mexico and Nepal.

Po and Co-workers found that exposure to biomass smoke was substantially related with COPD, ARTIs, and wheeze in a meta-analysis of 36 trials (Fullerton, Gordon and Calverley, 2009).

Respiratory tract infections, exacerbations of inflammatory lung diseases, cardiac events, stroke, eye illness, tuberculosis (TB) and even cancer have all been associated to cooking emissions. Despite the fact that IAP is not a direct cause of tuberculosis and/or related disorders, pollution from biomass fuels has been associated to a higher prevalence of infection.

A relationship between biomass smoke exposure and chronic bronchitis and/or chronic obstructive lung disease has been discovered in numerous investigations. Energy technology, fuels used, home and transportation system energy efficiency and energy transmission and distribution patterns all have a direct impact on the majority of sources of both ambient and household air pollutions.

As a result, avoiding diseases connected to air pollution necessitates the implementation of sector-specific policies aimed at reducing pollution at its source (for example, in energy and power generation, transportation, urban planning and construction).

According to several studies (Gao and Mann, 2009; Lee, Li, Johnson, and Stein, 2005; Mann and Niehueser-Saran, 2007), chronic inhalation of biomass smoke can cause inflammation and oxidative stress, which can lead to an increase in arterial blood pressure. Because most sources of air pollution coexist, determining the relative importance or proportional contribution of specific sources in a given location is required in order to properly target mitigation actions for the greatest health benefits.

Characteristics of Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs can be explained as a class of chemicals created during the incomplete combustion of organic substances such as waste, charbroiled meat, coal, oil, gas, wood and fish (Karam et al., 2015). Because of their lipophilic nature, PAHs have a low water solubility.

The majority of PAHs have a low vapour pressure and can adsorb on surfaces (Opel, Palm, Steffen and Ruck, 2011). PAHs can undergo disintegration of a compound in the presence of light or radiation during adsorption or dissolution on surfaces.

Photodecomposition of PAHs in the environment occurs when they are degraded utilizing UV light from the sun (Goulas, Louvel and Waterlot, 2015). Warshawsky (1999) showed that PAHs trigger a lot of health hazards such as immunological, carcinogenic, genotoxic, haematological and hepatic effects.

Polycyclic aromatic hydrocarbons (PAHs) occur in nature as aromatic compounds such as single ringed compounds such as xylene, benzene and toluene (Gallego, Loredo, Llamas, Vázquez and Sánchez, 2001). “Polycyclic” for PAHs means they are composed of two or more fused aromatic rings which makes them possess different makeup.

Gallego *et al*. (2001) further explains that the single unit of PAHs is a benzene that possesses delocalized electrons which makes PAHs very hydrophobic in nature. Delocalized electrons cause carcinogenicity in PAHs because they can easily produce radicals. PAHs can produce weak Van der Waal forces with other organic molecules.

PAHs are made up of more than just hydrogen and carbon. They are also composed of other elements like nitrogen and sulphur. PAHs can be formed as a result of natural aromatization at temperatures of 100-150 degree Celsius. PAHs can form spontaneously in nature without the need for human intervention. Organic matter abounds in nature, from plants to animals to soil flora and fauna (Liu et al., 2009).

Many activities that involve incomplete burning of organic matter, on the other hand, result in their artificial synthesis. As a result of human activity, organic matter can also be formed as garbage. Organic matter accumulates in hot sections of the planet, such as the crust, where it is subjected to extreme temperatures and pressure (Cejpek *et al*., 1995). This causes them to decompose into small, unstable molecules, a process known as pyrolysis. In a process known as pyrosynthesis, the unstable chemicals mix to generate far more stable PAHs (Schuetzle, Lee, Prater and Tejada, 1981).

PAHs can also form at lower temperatures (100–1500 degree Celsius) over longer periods of time, albeit this may result in high levels of alkylated PAHs (Liu, Chen, Huang, Li, Tang and Zhao, 2009).

Humans can induce aromatization which occurs faster due to human interference. This happens during the application of heat to organic materials for example biomass fuel burning. During biomass fuel burning, there is partial combustion of wood as the source of organic matter.

Classes of PAHs

Polycyclic aromatic hydrocarbons may be characterized by their melting, boiling point, vapor and water solubility according to their structures. Increase in molecular weight results in increase in their solubility in non-polar

organic solvent and soluble in polar water. They are insoluble in aqueous systems.

Their solubility is important characteristic for their distribution in foods (Lerario, Giandomenico Lopez and Cardellicchio, 2003). The water solubility of PAHs increases with temperature and decreases with increasing molecular weight (Yu, Huang, Lou, Chang, Dong Wang and Liu, 2015). Relatively high solubility is likely to have a higher environmental mobility and is likely to be found in higher concentration in aquatic samples (Gilbert and Şenyuva, 2008).

They are lipophilic and they adhere to particles of food (Lawley, Curtis and Davis, 2012). They can easily traverse lipid membranes and accumulate in aquatic organisms since they are lipophilic substances (Yu et al., 2015) and also accumulates in the soils by binding to the organic water. The transportation of PAHs in the atmosphere is influenced by their volatility.

PAHs are also classified to their size and genotoxicity. Light PAHs have a maximum of four benzene rings fused together and are hence referred to as low molecular PAHs. Heavy PAHs are defined as those with more than four benzene rings. In comparison to heavy PAHs, light PAHs are more volatile, water soluble and less lipophilic (Ferrarese, Andreottola and Oprea, 2008). Heavy PAHs are both more hazardous and stable than light PAHs.

Both light and heavy PAHs are mostly treated as being similar during risk assessments. This is due to the fact that they are frequently encountered in mixes. Their toxicity and structure are also connected. Individual compounds are segregated during risk characterization, despite the fact that PAHs are

generally considered as a group. There are more than 100 chemicals in the PAHs family.

Genotoxicity of PAHs

PAHs can also be classed according to their genotoxicity. PAHs are classified into four groups: B2, N/A, D and E. Group B2 has carcinogenic activity. The USEPA has named seven compounds from the B2 group (EPA, 1993). This categorization was made using the International Agency for Research on Cancer (IARC) approach and in accordance with the findings of several studies.

Other PAHs that are not classified, however, may be carcinogenic (Nisbet and LaGoy, 1992), therefore they should not be considered to be non-carcinogenic (USEPA class E).

Benzo[a]Pyrene was the first compound to be published under the class B2 heading (B[a]P). According to the USEPA, it was the only cancer slope factor until more study added around six chemicals to the B2 categorization. The first PAH with carcinogenic potential was Benzo[a]Pyrene (B[a]P), which is a module of a typical PAH with a fused aromatic ring system.

Seven PAHs that have been identified to be carcinogenic are: Benzo (a)anthracene, Benzo(a) pyrene, Benzo(b) fluoranthene, Benzo(k) fluoranthene, Chrysene, Dibenz(a,h)anthracene and Indeno(1,2,3-cd)pyrene.

Health Effects of PAHs

Carcinogenic chemicals are those that can cause mutations and as a result, cancer. Because of their carcinogenicity, PAHs have become a problem.

Carcinogens easily produce free radicals, which can set off a chain reaction of radical generation in the body. As a result of the breakdown of the cells' regulatory mechanisms, metastasis occurs, resulting in uncontrolled cell proliferation and the formation of malignant tumors and lesions.

A handful of cancer cases have been linked to Class B2 PAHs. As a result, they have risen to the top of the food safety and quality management agenda. Cancer is difficult to treat, especially in its advanced stages, which can be quite costly. Chemotherapy and irradiation surgery are two types of cancer treatment that are prohibitively expensive for the typical person in a developing nation like Ghana.

As a result, it is critical to identify all of the elements that contribute to this illness so that they can be avoided (WHO, 1993). This is why fish smokers who are exposed to smoke during their work should be monitored in order to help them make informed decisions.

Sources of PAHs in the Environment

There two sources of PAHs; natural and man activities which prompt their creation. Forest fires, for example, are known to be a source of PAH in the environment. Forests are made up of natural materials (trees). There is a chance that PAHs will be produced after partial combustion of natural matter by flames.

Petroleum (fossil fuels) is one of the natural sources of PAHs, which are produced as a result of urbanization and industrialization, which necessitate the provision of energy. The plastic industry also uses fossil fuel (petroleum) as a basic material.

Many of the plastics floating about in the environment are made from petroleum, which could be a source of PAH. When plastics are burned in the presence of high temperatures or ionizing radiation, for example, PAHs may be produced.

Pyrolytically generated PAHs, which are manufactured as a result of human activity, are another major source of PAHs. PAHs created by pyrolysis may also be produced by industries that generate heat and particulate matter, for example.

Contamination of air by dust and particulate matter containing pyrolytically produced PAHs may also have an impact on plants or raw materials due to the phenomena of PAHs dropping from the atmosphere during the growing period. PAHs can make their way into the finished product. PAH contamination levels from vehicle exhaust were extremely high (Ikins, 1994).

Furthermore, dust from roadways, which includes gasoline, vehicle exhaust, oil spills, tire, diesel and pavement, is a source of PAHs (asphalt or bitumen). Based on enrichment factor, vehicle exhaust was shown to be the primary contributor to PAHs in dust collected from highways with considerable traffic. In Tokyo's residential districts, atmospheric fallout also had a significant influence (Takada, Onda, Harada and Ogura, 1991). Used crankcase oil is a major component of PAH found in Malaysian road dust (Zakaria *et al*., 2002).

PAHs have their source from air particulates and may be carried in the atmosphere and deposited through dry and wet deposition (Wang, Tian, Yang,

Liu and Li, 2009). Once deposited, high molecular PAHs may leach into soil or sediment for a long time.

There are around 500 distinct PAHs that have been found in air, according to IARC scientific journal – 161, 2013, although most measurements are focused on benzo[a]pyrene as a representative of the entire PAH family.

The majority of PAHs in ambient air are carcinogenic (IARC, 2010) and B[a]P has been elevated to a Group 1 recognized human carcinogen following a recent evaluation of its carcinogenic potential (IARC, 2010). As a result, there is a need for worry regarding the link between PAHs exposure in the environment and their potential to cause cancer in humans.

"Because of health concerns, the US Environmental Protection Agency (EPA) monitors sixteen (16) priority PAHs in the air: naphthalene, acenaphthylene, acenaphthene, fluorene, anthracene, phenanthrene, fluoranthene, pyrene, chrysene, benz[*a*] anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, B[*a*]P, indeno[1,2,3-cd] pyrene, benzo[*g,h,i*]-perylene and dibenz[*a,h*]anthracene (in order of number of aromatic rings per structure)."

Several "PAHs have been proven to be carcinogenic in experimental animals after intratracheal consumption or inhalation: fluoranthene, benzo[*k*]fluoranthene, naphthalene, chrysene, benzo[*b*] B[*a*]P, dibenz[*a,h*]anthracene, dibenzo[*a,e*]pyrene and dibenzo[*a,l*]pyrene This raises concerns about the amounts of carcinogens in the air we breathe." These USEPA 16 common PAHs are presented in Figure 1.

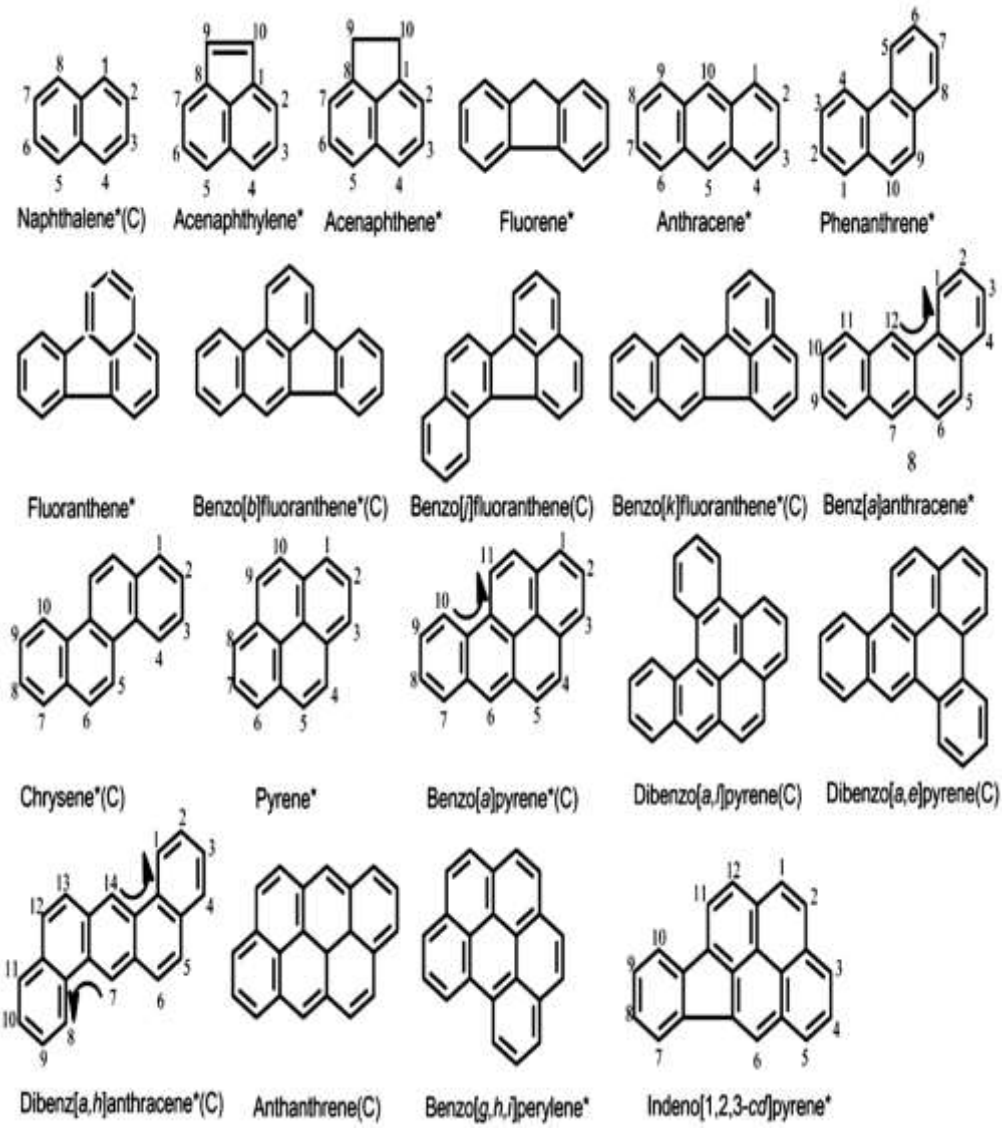


Figure 1: PAHs in Ambient Air
Reference: (Park and Penning (2008); reprinted with John Wiley and Sons' permission).

An asterisk represents a USEPA priority pollutant. (C) shows that the compound is carcinogenic by intratracheal administration or inhalation in experimental animals. They may indicate pollution sources due to their source profile in addition to their degradation (Bzdusek, Christensen, Li and Zou, 2004).

Chronic "exposure to PAHs has been linked to lung cancer (Motorykin, Matzke, Waters and Simonich, 2013) and peripheral arterial

disease (Xu, Hu, Kearney, Kan and Sheps, 2013). The U.S. Environmental Protection Agency Integrated Risk Information System (1992) and the International Agency for Research on Cancer (IARC) (1983) identify some PAHs as probable human carcinogens."

Health Effects of PAHs and Metabolites in Humans

Although the acute health effects of PAHs in humans are unknown, there have been some reports of occupational exposure to high levels of pollutant mixes containing these compounds.

Diarrhea, eye discomfort, nausea, vomiting and confusion are just a few of the symptoms. It has not been determined which of the mixture's components was responsible for the effects. The fact that PAH combinations can induce inflammation and skin irritation in people and animals has been proven. Benzo(a)pyrene, naphthalene and anthracene all induce skin irritation, whereas benzo(a)pyrene and anthracene also produce allergic reactions in people (skin sensitizers).

PAHs have been linked to kidney and liver damage, as well as jaundice, in those who have been exposed to them for a long time. Inflammation and reddening of the skin may result from prolonged skin contact with PAHs. Inhaling or ingesting excessive amounts of naphthalene can cause haemolysis (Nisbet *et al*., 1992).

Animals have developed stomach cancer and lung cancer as a result of long-term exposure through ingesting and inhalation, respectively.

Exposure to PAH mixes and other industrial pollutants has been linked to chronic bronchitis, a decreased immunological response and lung function disorders in a long-term study of employees. Lung, skin, gastrointestinal and

bladder cancers are all on the rise (Ramesh, Walker, Hood, Guillén, Schneider and Weyand, 2004).

Some people are known to be vulnerable to the effects of PAH in a population. The people are those who have a low immune system; those whose organs are failing, particularly the elderly and children with undeveloped and immature organs. Susceptibility to the circumstances listed above applies to all substances, not simply PAH.

In addition, activities that expose a person to PAH, such as excessive sun exposure and smoking, have been shown to enhance vulnerability to PAH's effects. The consequences of PAH are exacerbated by skin and liver problems.

PAHs have an effect on women of childbearing age as well. PAH has been demonstrated in several studies to be passed from pregnant mothers to their offspring through the placenta in the womb.

Metabolic Activation Pathways for PAHs

Parent PAHs are characterized by low chemical reactivity. Parent PAHs must be metabolically activated to electrophilic intermediates in order to exert their carcinogenic effects (Thakker, Yagi and Levin, 1985).

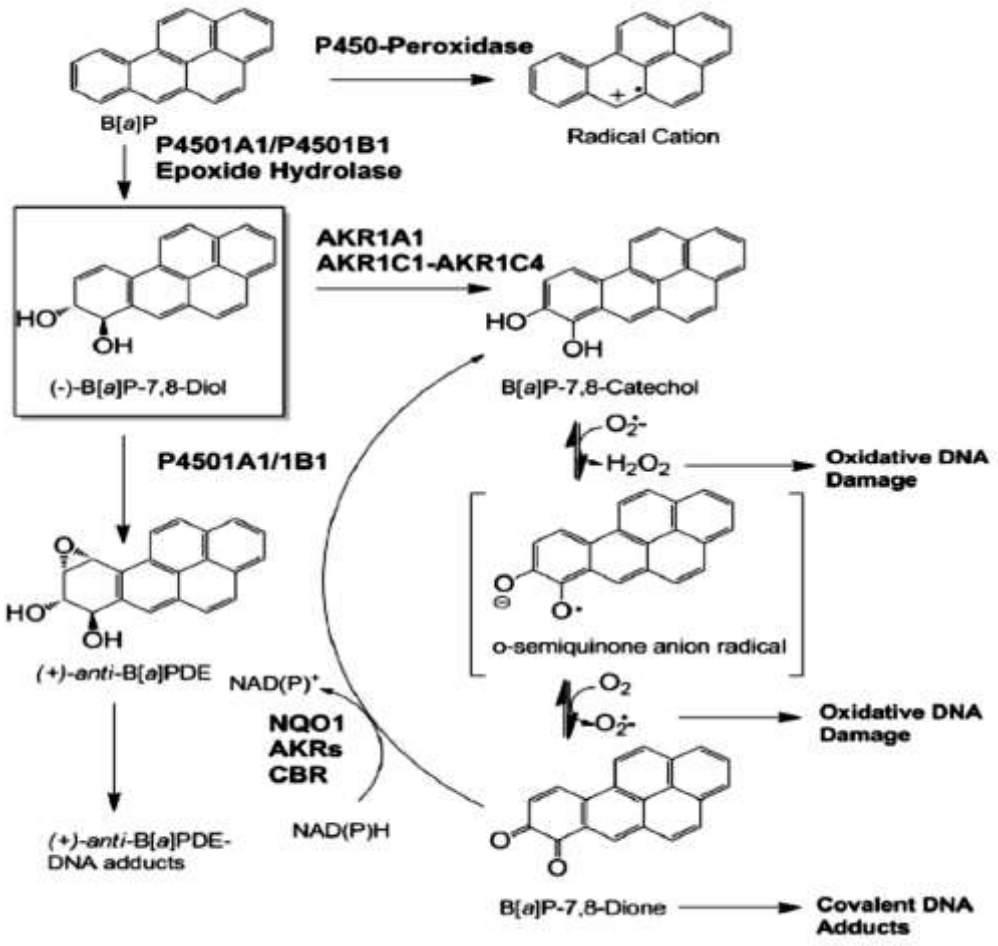


Figure 2: PAHs Activation Mechanism using Benzo[a]pyrene
Reference: Park and Penning (2008); reprinted by John Wiley and Sons with permission.

Three "PAH activation pathways are depicted in Figure 2. B[a]P is metabolically activated by either P450 peroxidase or another peroxidase in the first pathway by acting as a co-reductant of complex-1 (FeV). This produces a radical cation on the most electron-deficient C6 atom. The C6 atom is very reactive, creating unstable C8-guanine [8-(benzo[a]pyren-6-yl) guanine], N7-guanine [7-benzo[a]pyren-6-yl) guanine] and N7-adenine [7-benzo[a]pyren-6-yl) adenine, which all depurinate DNA adducts" (Cavalieri and Rogan, 1995).

In "vitro reactions using B[a]P, a peroxide substrate and microsomes support the first pathway. This results in the trapping of DNA adducts, as well

as experiments on mouse skin (Cavalieri, Higginbotham and RamaKrishna ,1991). Thus, there is evidence that dibenzo[a,l]pyrene and B[a]P can cause tumors in mouse skin and rat mammary" glands by this method (Cavalieri, Rogan and Li, 2005).

"Additionally, trace levels of B[a]P-depurinating DNA adducts have been found in the urine of smokers and women exposed to household smoke" (Casale, Singhal & Bhattacharya, 2001).

The following is the second path: Through a three-step process including oxidation and hydrolysis processes, B[a]P is metabolically activated to vicinal diol-epoxides (Jerina, Chadha and Cheh, 1991). In the second step, epoxide hydrolase is primarily responsible for the formation of 7R,8R-trans-dihydrodiol. Another oxidation pathway, including P4501B1 and other P450 enzymes, generates diol-epoxide diastereomers in the third step (Shimada, Oda and Gillam, 2001).

Routes of Exposure of Polycyclic Aromatic Hydrocarbons

Ingestion: These substances enter the body through drinking water and swallowing food, soil or dust particles.

Food contamination by PAHs occurs as a result of food processing methods. PAHs were discovered in cereals, grains and flour after drying processes such as heating after smoking were applied to preserve them.

PAHs in the atmosphere and soil could contaminate fruits and vegetables.

Humans may be exposed by processed meats or pickled meals, as well as infected cow's milk or human breast milk. PAHs can also be found in food that has been grown in contaminated soil or exposed to contaminated air.

PAHs can also be released when meat or other foods are cooked at high temperatures.

Human Exposure Routes of Exposure Routes of PAHs

Inhalation: By inhaling contaminated air, PAHs can enter the body through the lungs. PAHs vapours or PAHs adhering to dust and other particles in the air are most likely to be inhaled by humans.

PAHs can be found in household smoke from wood fires and tobacco smoke. Tobacco smoke contains PAHs such as dibenzo(a, g)anthracene, anthracene, benzo(a) pyrene, benzo(a)fluorene and chrysene in both main stream and side stream smoke (ATSDR., 1995).

The concentrations of PAHs in main stream smoke range from 11 ng to 199 ng cigarette-1, whereas PAHs in side streams smoke from tobacco vary from 39 ng kg-1 to 1224 ng cigarette-1 (Nelson., 2001).

Skin contact: PAHs can enter the body through skin contact if they come into touch with high-PAH soil or other substances. People may be exposed to PAHs when they enter areas where products containing PAHs are burned, such as coal, wood, gasoline and other sites where products containing PAHs are burned or when they visit a hazardous waste site, such as a former gas factory site or wood-preserving facilities, such as creosote-treated wood goods.

Coal tar creosote is a by-product of coal tar, which is created when bitumen is carbonized during the manufacture of coke or natural gas. About 85 percent of PAHs such as anthracene, naphthalene, and phenanthrene, as well as 2 percent to 17 percent phenolic compounds, make up coal tar creosote.

Creosote has been used to preserve wood in the construction of wooden houses and rail road tiles (Bogan and Sullivan, 2003).

Eye Infection or Visual Impairment

Visual impairment can lead to functional limits in the visual system, such as irreversible vision loss, a narrower visual field, lower contrast sensitivity, greater susceptibility to glare and a reduced capacity to conduct daily activities like reading and writing (Kavitha, Manumali, Praveen and Heralgi, 2015).

"Individuals with visual impairment have quantifiable vision, but even with refractive correction. Corn and Lusk (2010) describe that they have difficulty doing visual tasks. Corn and Lusk (2010) go on to say that with the help of compensating low vision aids and/or environmental changes, these people can occasionally improve their ability to do visual tasks."

Occupational eye injuries, according to Alvi, Hassan, Sia, Qidwai, Aurangzeb and Rehman (2011) and Ho, Yen, Chang, Chiang, Sheen and Chang (2008), can cause severe morbidity and economic loss, with nearly half a million people worldwide suffering from monocular blindness as a result of ocular injuries (Aghadoost, 2014).

According to Rosenstock, Cullen and Fingerhut (2006), employees in developing nations are frequently affected by occupational health issues. According to Négrel et al. (1998), the adult working population in developing nations is highly exposed to job dangers and up to 5% of all blindness in these nations may be due to work-related accidents (Thylefors, 1992).

Increased incidence of ocular injuries and diseases in developing countries can be caused by a variety of causes such as poor working

conditions, longer hours at work and insufficient or poor safety procedures (Addisu, 2011)

In India, investigations by Mohan et al., 1989 found that those who use biomass fuel have a higher incidence of cataracts, with an odds ratio of 1.6. According to Zodpey and Ughade (1999), the adjusted odds ratio is 2.4.

Indoor air pollution resulting from the burning of biomass fuel has also been associated with blindness through trachoma (Prüss and Mariotti, 2000). Exposure to smoke is associated to eye infections or irritation such as growth on the eye, redness of the eye and eye watery.

Indoor air pollution resulting from the use of biomass fuels can also cause eye irritation, cataracts or blindness (McCarthy, Nanjan and Taylor, 2000).

Other Nepalese and Indian studies have indicated a correlation between biomass fuel smoke and cataract (Pokhrel, Smith, Khalakdina, Deuja, Bates, 2005: Saha et al., 2005). Smoking can cause oxidative stress by depleting antioxidants including plasma ascorbate, carotenoids and gluthathione, which protect against cataract formation.

People in India who use biomass as their primary source of energy have a higher rate of partial or total blindness (Mishra, Retherford and Smith, 1999).

Again, it was discovered in some animal experiments that wood smoke condenses and destroys the lens of rats, resulting in discoloration, opacities and debris particles. Absorption and accumulation of toxins that aid oxidation are thought to be part of the process.

Radicals found in biomass fuel smoke will directly attack the eye, causing damage to lens proteins and the fiber cell membrane (McCarty, Nanjan and Taylor, 2000).

High levels of smoke exposure have also been linked to severe headaches and eye infections, according to studies conducted in Ghana (Odoi-Agyarko, 2009). According to Kyei, Owusu-Ansah, Boadi-Kusi, Nii and Abu (2016), there were 1126 answers (due to numerous replies) to ocular symptoms after exposure to risks during fishmongers' smoking of fish, with irritation of the eyes being the most common and redness being the least common. Photophobia was the most common symptom among fishmongers, with 222 (62.2%), 217 (60.8%) complaints of blurry distance vision, 201 (56.3%) symptoms of tearing, 154 (43.1%) symptoms of ocular pain and 143 (51.3%) symptoms of burning feeling.

Biomonitoring of PAHs

Personalized air monitoring systems, in which PM is caught on filters and then evaluated for PAH material, can be used to compute external exposure during biomonitoring.

Calculating exposure indicators in the urine and blood can also be used to track internal dose. Two examples of analytes utilized as biomarkers of PAH exposure are measuring PAH metabolites in urine and intermediate biomarkers of impact (e.g. DNA and haemoglobin adducts).

Urinary metabolites are used to achieve the most precise results. Particulate pyrene and full PAH have a close interaction in the respiratory zone. Individual variability in PAH metabolism can also be seen in urinary 1-hydroxypyrene levels.

Occupational exposure has been associated to a 10–100-fold increase in urine 1-hydroxypyrene (IARC scientific paper – 161). Danish bus drivers excreted more 1-hydroxypyrene than mail carriers, although outside mail carriers had more PAH metabolites in their urine than inside mail carriers, implying that outdoor air pollution had an influence (Hansen et al., 2004).

Suwan-ampai et al., 2009 used the National Health and Nutrition Examination Survey to look at the influence of involuntary tobacco smoke exposure on urine levels of 23 monohydroxylated PAH compounds in 5060 participants aged > 6 years (NHANES). After adjusting for other confounders, they discovered significant increases in urinary 9-hydroxyflourene, 1-hydroxypyrene, 1-hydroxypyerene, 2-hydroxyfluorene, 3-hydroxyfluorene and 1-2-hydroxyphenanthrene. Involuntary exposure caused a 1.1–1.4-fold increase, which climbed to 1.6–6.9-fold when youngsters were intentionally exposed.

The use of 1-hydroxypyrene as a biomarker of PAH exposure has been questioned because pyrene is not a carcinogenic PAH. As a result, 3-hydroxy-B[a]P has been used as a substitute, but alternative sensitive testing methods have been difficult to come across. Because 3-hydroxy-B[a]P is not derived from any of the known B[a]P activation routes, its value as a biomarker has been questioned (Dybing, Nafstad, Victorin and Penning, 2013).

Due to evidence that PAHs can be transformed to diol-epoxides as an activation mechanism, there have been breakthroughs in assessing their equivalent tetraol hydrolysis products in people. To identify phenanthrene tetraols, stable isotope dilution liquid chromatographic mass spectrometric technologies have progressed (Hecht et al., 2010; Zhong et al., 2011).

Phenanthrene has a bay area and goes through a similar metabolic transformation to B[a]P, generating diol-epoxides that hydrolyze to tetraols. According to (Hecht et al., 2010), the finding of phenanthrene tetraols has also been criticized because it is not a carcinogenic PAH.

Some femtomole sensitivity methods for quantifying urinary B[a]P tetraols have recently been developed, and these techniques can now be employed in biomonitoring research.

The levels of nine urine monohydroxylated PAH metabolites and 8-oxo-2′-deoxyguanosine in Chinese children from polluted and non-polluted locations were compared by Fan, Wang and Mao (2012). (8-oxo-dG). According to the findings, children from polluted areas had a higher PAH load than children from non-polluted areas. The levels of 8-oxo-dG, on the other hand, showed no obvious variation. Urinary 2-naphthol, 1-hydroxypyrene-glucuronide and malondialdehyde are tested by synchronous fluorescence spectroscopy or high-performance liquid chromatography. The goal of this study was to see how PAH exposure and oxidative stress in Korean adults and women altered seasonally and regionally (Yoon, Lee, Lee, Kim, Choi and Kang, 2012). Individuals from industrialized countries had greater levels in the winter as well.

1-hydroxypyrene-glucuronide levels were greater in children exposed to ambient cigarette smoke.

Carbon Monoxide

Carbon monoxide (CO) is a poisonous gas that is created by incomplete biomass combustion, such as wood burning (Smith et al., 2011), agricultural and grassland burnings (Li et al., 2011).

Carbon monoxide is a major compound generated by biomass burning, according to (Naeher et al., 2007), with emission factors of around 130 g/kg wood burned. Carbon monoxide emission factors from peat combustion were estimated to be 37 g/kg (Po, Fitzgerald and Carlsten, 2011). Carbon monoxide was released when cereal was burned, with an emission factor of 35 g/kg per kilogram (Sumpter Chandramohan, 2013).

Carbon monoxide derived from biomass burning is said to account for 32 percent of all carbon monoxide produced from other sources (Kurmi et al., 2012). It has been proposed as a less expensive but more proximate indicator of indoor air pollution caused by biomass fuel combustion (Kurmi, et al., 2012).

Effects of CO in Humans

When a person is exposed to high levels of carbon monoxide, the body substitutes carbon monoxide for oxygen in the red blood cells. Carboxy haemoglobin is formed when carbon monoxide reacts with hemoglobin. As a result, oxygen supply to tissues and the developing fetus is decreased. This may result in low birth weight babies as well as a rise in perinatal deaths.

It can also increase bronchial reactivity, leading to asthma flare-ups, wheezing and recurrent respiratory tract infections. Drowsiness, nausea, vomiting, headaches, dizziness and chest tightness are all symptoms of carbon monoxide poisoning. Carbon monoxide poisoning may result in a coma, brain injury or death (Study and Reed, 2011).

Particulate Matter ($PM_{2.5}$) Exposure

Particulate matter, also known as particle contamination, has been related to a variety of serious health consequences.

Particles with a diameter of less than or equal to 10 micrometers are very small and can enter the lungs, causing severe health problems. $PM_{2.5}$ (fine particles) have a diameter of 2.5 micrometers or less and are only visible under an electron microscope.

Sources of Particulate Matter ($PM_{2.5}$)

$PM_{2.5}$ "is produced from all types of combustion, including motor vehicles, power plants, residential wood burning, forest fires, agricultural burning and some industrial processes."

Health Effects of $PM_{2.5}$ due to Biomass Fuel Usage

It is "thought to pose the greatest health danger. Owing to physiological variations, children and older adults are more vulnerable to PM-induced effects.

Children are more vulnerable to the effects of PM than adults because they spend more time indoors than outside, have higher activity levels and have a lower minute volume per unit body weight, both of which contribute to higher PM dose per unit body weight of the subpopulation that led to increases in PM dose per lung" surface area.

As a result, the vulnerability of developing lungs to negative effects increases. Since physiological processes gradually deteriorate with age, the elderly are considered a vulnerable group. "An elderly person has a higher prevalence of pre-existing cardiovascular and respiratory disorders than children or younger people, which may make them more susceptible" to PM.

Environmental epidemiology research has shown that short- and "long-term exposure to ambient PM raises mortality and morbidity, decreases life

expectancy and increases the risk of respiratory and cardiovascular diseases in the last decade."

Short-term $PM_{2.5}$ exposure has been linked to increased respiratory symptoms, increased drug usage and decreased pulmonary function in asthmatic children, according to research.

A "case-crossover research conducted in Taipei discovered that admissions for chronic obstructive pulmonary disease (COPD) are significantly and positively correlated with higher $PM_{2.5}$ levels on hot and cold days" (Hu, Zhong and Ran, 2015).

Furthermore, poisonous "substances such as gases, organic compounds and heavy metals bind to the surface of $PM_{2.5}$, increasing toxicity and interfering with genes, DNA and other genetic material. $PM_{2.5}$ and toxic compounds have also been linked to cancer" growth.

Long-term exposure to fine particulate air pollution is related to a slight but significant rise in lung cancer mortality (Turner et al., 2011).

Short "and long-term exposure to PM, specifically $PM_{2.5}$, is linked to cardiovascular morbidity and mortality, according to evidence from epidemiological studies" (Dockery, 2001).

Myocardial "infarction, heart failure and arrhythmia have all been related to high PM levels. The elderly, diabetics and those with a history of coronary artery disease tend to be particularly vulnerable to the negative effects of PM exposure. Long-term exposure to small particulate air pollution increases the risk of cardiovascular disease death by mechanisms such as pulmonary and systemic inflammation, increased atherosclerosis and altered cardiac autonomic control" (Fuller et al., 2013).

In addition, studies from Nigeria found a clear connection between anxiety and biomass exposure in women with chronic bronchitis (Umoh, Ibok, Edet, Essien and Abasiubong, 2013).

Benzene

Benzene is found in abundance in the world. Long periods of time subject the general population to low-level inhalation.

This "is because the general public is exposed to benzene primarily through inhalation of polluted air, especially in congested areas, near gas stations and through tobacco smoke inhalation from both active and passive smoking." Benzene may also be inhaled through biomass fuel combustion smoke.

Sources of Benzene

Human and anthropogenic sources of benzene in the atmosphere are possible sources. Carbon pollution from volcanoes and forest fires are two natural sources of benzene in the atmosphere.

"Benzene (an anthropogenic source) is emitted into the air via automobile exhaust, gasoline, tobacco smoke, bushfires and industries. It has a high mobility in soils and is water soluble." Domestic sources of benzene contribute the greatest proportion of benzene emissions, particularly through the combustion of fuels for cooking and" heating (Office for National Statistics, 2006).

Distribution of Benzene in the Environment

The amount of benzene in the air varies significantly based on the activities that occur in a given area. Benzene levels in the air could be raised

by emissions from biomass fuel, coal, oil, benzene, waste, storage operations, motor vehicle exhaust and evaporation from gasoline service stations.

Tobacco smoking, especially indoors, is another source of benzene in the air. As a result of industrial discharge, the disposal of benzene-containing goods and fuel leakage from underground storage tanks, benzene is discharged into water and soil (Wilbur et al., 2007).

Health Effects of Benzene

The effects benzene exposure "on human health depend on the concentration of benzene, the type and extent of exposure. The effects of benzene" on humans also depends on the health and age of the affected individual (Public Health Fact Sheet, 2011).

Acute exposure to benzene could resemble solvent intoxication. Some of the symptoms could be drowsiness, dizziness, delirium, loss of consciousness and respiratory arrest. Chronic health effects associated with benzene exposure are anaemia and leukaemia. Benzene is a known human carcinogen.

Route of Exposure of Benzene

Inhalation is the most common way to be exposed to benzene. Breathing in benzene-containing air exposes you to the chemical. Benzene evaporates fast into the air and is swiftly absorbed into the body through the lungs. In addition, benzene-contaminated water can be absorbed by the body through skin contact or eating. Reactive metabolites such as benzene oxide have been implicated in the mechanism of benzene toxicity.

In most cases, indoor benzene levels are higher than outside benzene levels. People who live near hazardous waste sites, petroleum refining

operations, petrochemical production facilities or gas stations may be exposed to greater quantities of benzene in the air. This shows that fish smokers who smoke fish indoors could be exposed to some level of benzene through smoke inhalation.

Ozone

"Ozone is a gas that exists in both the upper and lower atmospheres of the Earth. Ozone at ground level is a significant air contaminant. It is created by the reaction of contaminants in the atmosphere with sunlight. The country's surface ozone demonstrates temporal and geographic" changes.

"Karthik, Sujith, Rizwan and Sehgal (2017) found surface ozone levels above the acceptable 8-hour average of 100 g/m^3 for air quality monitoring at various stations in" India.

Distribution of Ozone in the Environment

The ozone layer is found 6 to 30 miles above the Earth's surface in the high atmosphere (the stratosphere). One of the most important roles of the ozone layer is to protect the Earth from the sun's UV rays. On the other hand, human-made pollution are steadily depleting this layer, resulting in an ozone hole above the north and south poles.

In the Earth's lower atmosphere, ground-level ozone (troposphere) is a significant photochemical pollutant. Surface ozone (O_3) is created when pollutants such as volatile organic compounds (VOCs) and nitrogen oxides chemically react in the presence of sunlight.

According to WHO Air Quality Guidelines, the US Environmental Protection Agency (EPA) proposed introducing an ozone level of 80 ppb based on the daily 8-hour maximum concentration in 1997.

Despite the fact that the WHO Air Quality Guidelines for Europe (World Health Organization AQG, 2000) set the ozone guideline value for an 8-hour daily average at 120 g/m^3, research have indicated that concentrations below 120 g/m^3 have negative health impacts.

As a result, the WHO Air Quality Guidelines AQG cut-off was dropped from 120 g/m^3 to 100 g/m^3 in 2005 (daily maximum 8-hour mean). As a result, when the weather is sunny, the maximum levels of ozone emissions occur. Automobiles, power plants, industrial boilers, refineries, chemical factories and other sources all contribute to these pollutants.

NO scavenges ozone once developed, leading in a "picture stationary state" with NO, NO_2, and O_3 concentrations all interlaced. The presence of CO and VOCs, on the other hand, might disturb this steady state relationship by inducing the formation of peroxy radicals, resulting in a rise in ozone levels (Tang, 2009).

CO_2 concentrations in the atmosphere are rising at a pace of roughly 0.5 percent per year, while surface ozone levels are rising at a pace of 0.32 percent per year, due to an increase in the burning of fossil fuels around the world (Akram, Ibrahim, Hassan and Hanaa, 2002). Even rural places may experience high ozone levels due to the capacity of wind to transport ozone over vast distances.

Strong ozone concentrations, as well as significant levels of local VOC and NO pollution, have been detected this winter.

Ground-level ozone is the major component of smog, which is combined with other gases and particle emissions. On the surface, ozone is a greenhouse gas that contributes to global warming (Karthik et al., 2017).

Health Effects of Ozone Exposure

When people are exposed to high levels of surface ozone, their lung function deteriorates. The mucociliary role of the lungs is also affected by high levels of surface ozone, increasing vulnerability to bacterial infections.

There could be "an increase in hospital admissions for Chronic Obstructive Pulmonary Diseases (COPD) as well as the number of cardiovascular and respiratory deaths" as a result of this.

Increased ozone concentrations in children are linked to further hospitalizations and unscheduled asthma treatment.

Phenol

Food curing with smoke, such as ham, sausages and fish, is an ancient flavoring and preservation method that is now widely used in industrialized versions.

Importance of Phenol in Fish Smoking

The "impact on health of wood smoke is currently an issue of great concern particularly small-scale burning of wood as a renewable fuel. Particle-related health hazards due to polycyclic organic compounds have been assessed (Cupitt, Glen and Lewtas, 1994). The carcinogens benzene and 1,3-butadiene are prominent among the emitted volatile hydrocarbons (Barrefors and Petersson, 1995)."

The majority of the organic compounds emitted from incomplete combustion are structurally related to the biomass's lignin and carbohydrates (Alen, Kuoppala and Oesch, 1996).

As a result, these compounds vary significantly from the more well-known "hazardous compounds found in combustion emissions from stationary

and mobile sources. For lignin-specific methoxyphenols in smoke from forest biomass burning," analytical data has previously been provided (Kjallstrand, Ramnas and Petersson, 2000).

Methoxyphenols were first known as important wood thermal degradation products (Faix, Meier and Fortmann, "1990), as semi-volatile components of wood smoke particles (Faix, Meier and Fortmann, 1990) and as semi-volatile components of wood smoke particles (Hawthorne," Krieger, Miller and Mathiason, 1989).

Basic antioxidant properties are possessed by phenols (Simic, 1992). Because of their critical significance in living organisms, the "antioxidant activities of phenolic tocopherols and ubiquinols have been extensively studied (Barclay," Baskin, Dakin, Locke and Vinqvist, 1990).

Because of their antioxidant properties, flavonoids and other plant phenols are gaining popularity (Ogata, Hoshi, Shimotohno, Urano and Endo, 1997). "Antioxidant properties have also recently been demonstrated for several lignin-related methoxyphenols" (Ogata et al., 1997).

"Toth and Potthast (1984) studied the chemical characteristics of meat smoking and numerous investigations looked at the chemical composition of smoke condensates and liquid smoke. Methoxyphenols are important components for the smoke flavor as well as the smoke's maintaining antioxidant function."

In "model experiments for many particular methoxyphenols related to those found in wood smoke, high antioxidant effects on lipid peroxidation have recently been established. Phenols and other smoke components can be

found in two forms: gaseous and condensed on particles or both (Hawthorne et al., 1989)."

Sources of Phenol

The methoxy phenol formed from lignin, constitute a large part of smoke from incomplete combustion of biomass. The cellulose and the hemicelluloses mainly decompose to anhydro sugars, which constitute another large part.

Biomass smoke also contains smaller amounts of hazardous organic compounds. Too often mentioned groups are volatile hydrocarbons and polycyclic aromatic compounds (PAC). Inorganic compounds, formed lead to all kinds of combustion of carbon-containing fuels, for example carbon oxides and nitrogen oxides.

Phenol is one of the pollutants present in biomass smoke (IARC, 2010). Individuals could be exposed to phenol through skin contact in the workplace or breathing contaminated air. "Other exposures to phenol may occur through smoking tobacco or the use of phenol-containing medicinal products (including mouthwashes, toothache drops, throat lozenges, analgesic rubs and antiseptic lotions)."

Route of Exposure to Phenol

Phenol is mostly absorbed by humans through dermal, inhalation and oral routes. Sulfate and glucuronide conjugation oxidation are involved in the point of entry for metabolism in the inhalation and oral routes. Catechol and hydroquinone are the main oxidative metabolites and they are also conjugation substrates.

Trihydroxybenzene and benzoquinone are examples of secondary products of catechol or hydroquinone metabolism. As phenol is consumed, it is generally dispersed across the body, though it is often stated that levels in the liver, kidney and lung are "higher than in other tissues (on a per-gram-tissue basis). Regardless of the route of administration, the drug is quickly eliminated from the body, mainly as sulfate and glucuronide conjugates in the urine. Phenol does not seem to accumulate in the body in large amounts" (Toxicological Review of Phenol, 2002).

Metabolism of Phenol in Humans

For starters, phenol is immediately conjugated to glucuronic acid or sulfate. Non-conjugated phenol can be utilized as an oxidation substrate as well. The cytochrome P450 2E1 isozyme (CYP2E1) then catalyzes the addition of one oxygen atom to a range of low-molecular-weight substrates, such as chloroform and benzene and is thought to be the major P450 isozyme for phenol oxidation, though other P450 enzymes may play a modest role.

The oxidation products of phenol generated by CYP2E1 activity appear to be primarily catechols and hydroquinone, which can be further peroxided to benzoquinone or oxidized to trihydroxybenzene by CYP2E1.

Conjugation reactions can also occur in the hydroquinone or catechol metabolites. Other studies have suggested that phenol can undergo peroxidative metabolism, resulting in biphenols and diphenoquinones in addition to P450-mediated oxidation.

The detoxifying pathways that make up the majority of phenol metabolism are direct sulfate and glucuronic acid conjugations. The metabolic profiles found in humans and laboratory animals support this theory. Sulfation

predominates at lower doses in humans and most other animals studied (Toxicological Review of Phenol, 2002).

Health Effects of Phenols

"After acute (short-term) inhalation or dermal exposures, phenol is highly irritating to the skin, eyes and mucous membranes in humans. Oral exposure to phenol is thought to be quite harmful to humans. Chronically (long-term) exposed humans have experienced anorexia, increasing weight loss, diarrhea, vertigo, salivation, a black coloring of the urine, blood and liver damage."

"The negative effect; the biological consequence that causes changes in morphology, physiology, growth, development or life span results in a reduction in the body's ability to cope with increased stress. Furthermore, an increase in bodily vulnerability makes the body more vulnerable to additional dangerous or hazardous chemicals (Barlow, Johnson, Belcher and Fenton, 2007)."

Phthalates

Phthalates are a family of "man-made chemical compounds used in the manufacture of plastics, solvents and personal care products. Phthalates are used as plasticizers (Australian Government, 2008a)."

PVC Poly (vinyl chloride) uses plasticizers to add durability "and other attractive properties. Phthalate-containing PVC products covered a range of industrial and consumer products, as well as specialist medical and dental applications."

The phthalate or mixture of phthalates used in a product's formulation is determined by the qualities of the phthalates used, as well as their cost.

Wickson (1993) reports that BBP is the most widely used stain-resistant plasticizer in PVC, with vinyl tile being the most prevalent use (NTP, 2003e).

DBP is used in a variety of products, including varnishes, plastics, paints, wood and lacquers. They are used in textiles, propellants, cosmetics, medical supplies, food packaging, dental items and paper (OSHA, 2009).

Distribution of Phthalates in the Environment

"Refining, agricultural wastes, municipal solid waste, industrial releases, manufacturing waste disposal, sewage sludge land application and the release of phthalate-containing commodities are all sources of phthalates in the atmosphere. The EPA's Toxics Release Inventory (TRI) list of dangerous chemicals lists just two of the eight phthalates (DBP and DEHP)."

According to the EPA, cumulative on-site and off-site DBP and DEHP discharges totaled more than 336,000 pounds from 134 sites and 1,229,000 pounds from 251 sites, respectively, totaling more than 336,000 pounds and 1,229,000 pounds. However, some facilities do not report a specified level of discharges under TRI."

The "number of releases to various media is commonly listed in the following order (from highest to lowest): ground, air and water, according to these data. Some facilities record excessive phthalate discharges, potentially resulting in high phthalate concentrations in the" atmosphere.

Production "and import levels show that the great majority of phthalates would be absorbed into plastics and other commodities, based on a comparison of TRI releases to IUR results (likely between 95 and 99.9 percent). Phthalates can be released from a product due to prolonged storage,

heat or agitation. The product can be released at any time during its existence, from manufacture to usage to disposal."

Route of Exposure of Phthalates

Exposure can occur through ingestion, inhalation, cutaneous absorption or intravenous injection. The widespread usage of phthalates as a flexible ingredient to PVC (polyvinyl chloride) products and as a scent extender in personal care items during the last 50 years has resulted in extensive general population exposure.

Phthalates are easily absorbed by the human body and swiftly transformed to their metabolites. They tend to travel out of the body swiftly via urine and feces, unlike certain substances. Phthalates can interact with one another, increasing the effect of exposure.

Health Effects of Phthalates

Despite the fact that "no causative link has been shown," phthalate exposure has been related to unfavorable developmental and reproductive outcomes in rats (Swan et al., 2005; Huang et al., 2009). Some of the reproductive developmental impacts reported in humans include "shorter" pregnancy, lower sex, shortened anogenital distance reported in newborn males, thyroid hormones and decreased sperm production observed in adults (NAS, 2008). Because the process for sexual differentiation in the fetus is substantially conserved in other animals, the reproductive developmental impacts observed in the rat experiments may be relevant to humans. According to statistics from the National Health and Nutrition Examination Survey (NHANES), phthalates are commonly utilized in the general population (CDC, 2009). According to biomonitoring evidence from amniotic

fluid and urine, humans are exposed to phthalates in pregnancy, as babies, during puberty and in adulthood. Multiple phthalates are frequently present in people's bodies at the same time.

In addition, recent animal experiments examining the combined effects of several active phthalate combinations on male and female reproductive growth, testosterone production, fetal and adult mortality discovered that all mixes were cumulative for all endpoints (Rider, Wilson, Howdeshell, Hotchkiss, Furr, Lambright and Gray, 2009). The findings on human biomonitoring, the reproductive consequences seen in animal studies and in people and the evidence on cumulative effects of mixtures all corroborate the EPA's concern about phthalate exposure posing a health risk to humans.

Human Health Risk Assessment

"Human health risk assessment is described as the characterization of the potential adverse health impacts of humans as a result of environmental risks, according to the USEPA (2013). It is also known as the process of estimating the nature and likelihood of adverse health consequences in individuals who may be exposed to chemicals in polluted environmental media in the present or future (USEPA, 2004)."

This "method combines science, engineering and statistics to identify and quantify a hazard, discover various routes of exposure and finally produce a numerical number to represent the potential risk (Lushenko, 2010)."

Hazard Identification

"The process of hazard detection decides whether a chemical is causally related to specific health effects or not. It purposely evaluates the weight of evidence for adverse effects in humans based on assessment of all available

data on toxicity and mode of action. In hazard identification, two questions are addressed:"

1. "whether an agent may pose a health hazard to humans"
2. "under what circumstance an identified hazard may be expressed. Often, multiple end-points are observed following exposure to a given chemical. The critical effect, which is usually the first significant adverse effect that occurs with increasing dose, is determined (Sipter, 2008)."

Dose-Response

Dose-response analysis determines the relationship between the amount of exposure and the likelihood of the health outcomes under question. It is a method of determining the link between the dose of an agent given or received and the occurrence of a negative health consequence.

Most toxic effects, such as organ-specific, neurological/behavioral immunological, non-genotoxic, carcinogenesis, reproductive or developmental, are thought to have a level or concentration below which no deleterious effects occur (i.e. a threshold). Other sorts of hazardous effects are thought to have a chance of causing harm at any degree of exposure (i.e. non-threshold). For mutagenesis and genotoxic carcinogenesis, the non-threshold assumption is commonly used (Sipter, 2008).

Exposure Assessment

Exposure assessment is a similar phase in the hazard detection and dose-response assessment process, with the aim of assessing the type and degree of chemical substance interaction encountered or expected under various conditions.

The risk of a toxic agent is quantified in an exposure assessment by the cumulative volume of the toxic agent taken into the body, which may include any combination of oral, inhalation and dermal routes of exposure. Exposure can be expressed as an environmental concentration in certain evaluations that are unique to a single exposure path.

To estimate the amounts to which human populations or environmental spheres (water, soil and air) can be exposed, exposure assessment involves determining the emissions, routes and rates of movement of a material, as well as its transformation or degradation (Sipter, 2008).

The numerical performance of an exposure assessment may be an estimation of the severity, rate, length, or frequency of contact exposure or dosage, depending on the intent of the assessment.

“Three main exposure routes are determined in exposure assessment namely; dermal, oral and respiratory. For risk assessments based on dose-response relationships, the output usually includes an estimate of dose. It is important to note that the internal dose, not the external exposure level, determines the toxicological outcome of a given exposure.”

The term "worst case exposure” means “the maximum possible exposure, or where everything that can plausibly happen to maximize exposure happens. The worst case scenario depicts a fictional person under extreme circumstances; this is unlikely to occur in a real population (US EPA, 1992) and for that matter, in most risk assessments the "weakest link" is the exposure assessment (Bridges, 2003). The use of biomarkers may provide the information about the pollution (Kakkar and Jaffery, 2005).”

Liquid-Liquid Extraction and Dispersive Solid Phase Extraction

"The method of separating a liquid mixture of components using liquid solvents followed by dilution of one or more components of the initial mixture is known as liquid-liquid extraction (LLE). The addition of a sorbent directly into the analytical solution, accompanied by dispersion that favors interaction between the sorbent and the analytes, is the basis for dispersive solid phase extraction (Lehotay, 2011: Anumol, Lehotay, Stevens and Zweigenbaum, 2017)."

"Following the dispersion phase, the sorbent is isolated from the analytes on its surface using a mechanical process such as centrifugation or filtration. One of the benefits of DSPE is that it reduces sample treatment time, allowing more samples to be tested in less time. Dispersive solid phase extraction has other advantages such as simplicity, adaptability and easy handling in comparison with the traditional techniques (Han, Sapozhnikova and Lehotay, 2014)."

History and Context of Dispersive Solid Phase Extraction (DSPE)

Dispersive solid phase extraction (DSPE) has been successfully implemented as a method of extraction, separation and cleaning in the analytic treatment of a wide variety of veterinary drugs used in the livestock industry since its discovery around 2000 (Anastassiades, Lehotay, Stajnbaher and Schenck, 2003).

DSPE simplifies SPE clean-up, permits more samples to be examined at once, is relatively quick and uses less solvent. The addition of a solid sorbent, usually silica or polymer based, directly into the sample solution is what DSPE is all about (Lehotay 2011; Anumol et al., 2017; Han et al., 2014).

During the dispersion process, the contact area between the sorbent and the analyte increases. The sorbents used in DSPE to determine antibiotic residues are solids whose affinities have been altered by the addition of a variety of chemicals. These modifications ensure analyte selectivity, allowing for maximum retention while minimizing interferences in the analytical matrix (Xiong et al., 2015).

Centrifugation or filtration are used to remove the sorbent from the dispersion. The analytes or interferences adsorbed on the surface of the sorbent could be easily eluted or removed with the addition of appropriate organic solvents until the solid phase was separated.

DSPE is a micro- and macroscale extraction and cleaning method that is used in a variety of analytical methodologies to remove potential interferences (clean-up) that could interfere with the subsequent determination of analytes (Deme, Azmeera, Prabhavathi Devi, Jonnalagadda, Prasad, Vijaya and Sarathi, 2014).

However, choosing the sorbent is an important step in DSPE and it is important to think about chemical and physical properties that allow the sorbent and analytes to interact as much as possible, ensuring selectivity extraction, removal6/, or preconcentration of analytes in analytical matrices (Fagerquist, Lightfield and Lehotay, 2005).

The DSPE method achieves adequate limits of detection (LOD) for antibiotic analysis while consuming minimal solvent during sample treatment.

As a result, when compared to classic procedures like LLE and SPE, it is considered a low-cost methodology (Silva, Haesen and Camara, 2012). The QuEChERS (quick, easy, cheap, effective, rugged and safe) sample

preparation method uses acetonitrile to partition liquids and dispersive solid-phase extraction to purify the extract (d-SPE).

The QuEChERS was designed to analyze pesticide residues in high moisture fruits and vegetables, but it can now be used to analyze a wide range of analytes in a wide range of samples. Various changes based on the use of alternative extraction solvents and salt formulations, as well as buffer additions for the salting-out partitioning step and the application of various d-SPE sorbents for the clean-up stage, allowed for a wide range of technique applications.

Pesticides, veterinary medications, pharmaceuticals, mycotoxins, polycyclic aromatic hydrocarbons (PAHs), dyes and acrylamide can all be analyzed with the QuEChERS method. Synthetic musks and ultra violet (UV) filters, bisphenols, polybrominated diphenyl ethers and other flame retardants, endocrine disruptors and other chemical substances could all be analyzed with it (Rejczak and Tuzimsk, 2015).

Anastassiades, Lehotay, Stajnbaher and Schenck first presented the QuEChERS method at the 4th European Pesticide Residue Workshop in Rome in 2002. (Anastassiades et al., 2002). The detailed method was later published in 2003, according to Anastassiades and Lehotay (2003).

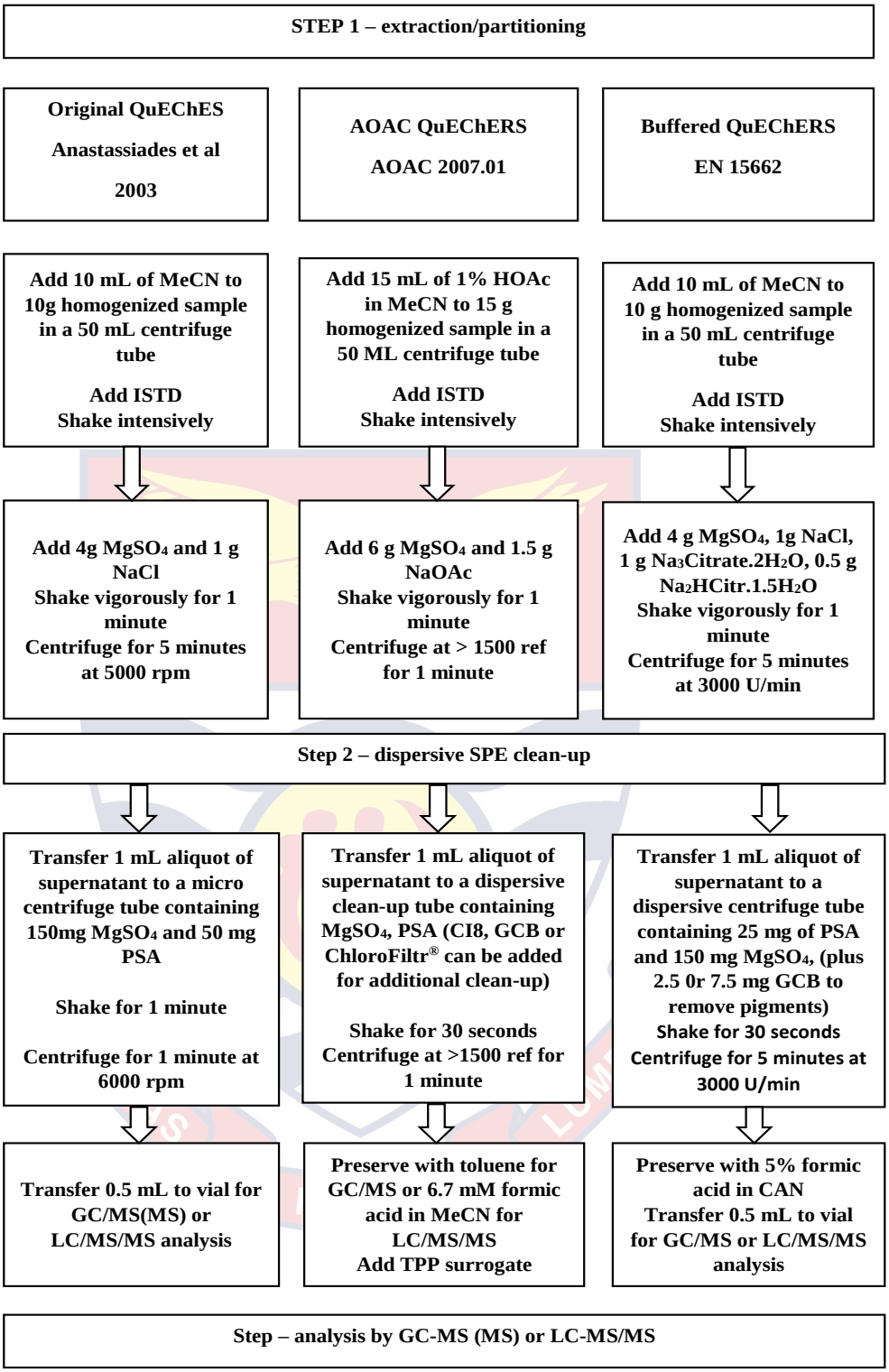


Figure 3: Schematic Flow Chart for Main Steps of Three Primary QuEChERS Methods

Reference: (Rejczak and Tuzimsk, 2015)

Original QuEChERS Method (Anastassiades et al., 2003) AOAC 2007.01 Official Method (AOAC Official Method 2007.01(2007)) EN 15662 The

European Official Method (EN 15662:2008 (2008)) (abbreviations used: GCB – graphitized carbon black; MgSO4 – magnesium sulfate anhydrous; MeCN – acetonitrile; HOAc – acetic acid; NaOA

The QuEChERS method is a type of high-throughput multi-residue technology that is used to detect contaminants in food, feed and environmental samples on a regular basis. The extract is purified by dispersive solid-phase extraction (d-SPE) after liquid-liquid partitioning with acetonitrile (MeCN) (Anastassiades and Lehotay, 2002).

The QuEChERS method offers several advantages over traditional extraction methods, including high recovery rates for a wide range of analytes, extremely accurate (true and precise) findings and the use of an internal standard (IS) to eliminate significant commodity discrepancies.

Internal standard addition is also vital in the QuEChERS strategy for minimizing error production in the numerous steps of the QuEChERS.

In addition, the QuEChERS technology has a fast character and a high sample throughput. A single analyst could extract a batch of 1020 samples in 30-40 minutes using this procedure (Lehotay, 2004).

Due to reduced solvent consumption, the lack of chlorinated solvents and minimal waste creation, the QuEChERS methodology is thus in line with so-called green chemistry (Schenck and Hobbs, 2004).

In compared to most standard extraction methods, the QuEChERS methodology requires only basic laboratory instruments, making this sample preparation process extremely affordable (Lehotay, 2004).

Summary of QUECHERS Method

"QUECHERS method is a simple, fast and inexpensive method. The procedure involves initial single-phase extraction of 10 g sample with10 mL acetonitrile, followed by liquid–liquid partitioning formed by addition of 4 g anhydrous MgSO4 plus 1 g NaCl."

"Removal of residual water and clean-up are performed simultaneously by the use of a rapid procedure known as dispersive solid-phase extraction (dispersive-SPE), in which 150 mg anhydrous $MgSO_4$ and 25 mg primary secondary amine (PSA) sorbent are simply mixed with 1 mL acetonitrile extract."

"The dispersive-SPE with PSA effectively removes many polar matrix components, such as organic acids, certain polar pigments and sugars, to some extent from the extracts (Anastassiades and Lehotay, 2000)."

GC/MS (Gas Chromatography/Mass Spectrometry) (GC-MS)

GC-MS is very useful in the analysis of sample since its development in the mid- 1950. It is used in the determination of drugs and metabolites in pharmaceutical industries. It can also be used to determine elemental composition and molecular weights of complex mixtures.

Most importantly, GC-MS is used to measure the amount of semi-volatile organic and volatile compounds in some complex mixtures including water, soil and other environmental samples. GC-MS serves as the basis of many Environmental Protection Agency, EPA methods.

According to Hites (1997), GC-MS can be used in "structural determination of unknown organic compounds in complex mixtures both by

matching their spectra with reference spectra and by a priori spectral interpretation."

Basic Principles and Instrumentation of GC-MS

Gas "chromatography coupled with mass spectrometer is an important technique for quantitative and qualitative determination of semi-volatile and volatile organic compounds in a variety of samples. The limit of detection is within sub-ng. Samples are prepared in solution prior to injection into the gas chromatography. A dissolution solvent such as dichloromethane can be used (Sneddon, Masuram and Richert, 2007)."

When the two techniques of gas chromatography (GC) and mass spectrometry (MS) are combined to make gas chromatography mass spectrometry, there are numerous benefits (GC-MS).

Although the GC is capable of separating a wide range of volatile and semi-volatile chemicals, it does not always do so selectively. The MS can also identify several chemicals selectively, but it is not always able to separate them. One of the difficulties with GC-MS was the substantial pressure variations; for example, the GC gas departing the system is roughly one atmosphere (760 torr), but the MS runs at roughly 1025–1026 torr.

Many suggestions were made, such as separating the GC effluent, with a small portion going to the MS and the remainder being vented out. It was also suggested that the interfaces between the GC and the MS be used to reduce the pressure to that of the MS.

A jet separator was frequently used for packed column GC, according to Sneddon et al., 2007, however it is readily obstructed and requires a lot of frequent maintenance (Sneddon et al., 2007).

"When the GC's gas flow is low (a few ml/min) and the MS vacuum system's pumping speed is high (a few hundred liters per second), the GS effluent can enter the MS more easily. For capillary column GC, this is the most used approach. For capillary GC-MS, the capillary column is currently put directly into the ion source. The GC effluent is at a specific temperature (Sneddon et al., 2007). For capillary column GC, this is the most used approach. For capillary GC-MS, the capillary column is currently put directly into the ion source. The effluent temperature leaving the GC is roughly 3008C (as high as 4008C possible)."

The effluent (individual chemicals) from the GC enters the MS, which includes the electron ionization detector as an example. To create ions, an ion trap or a time-of-flight system could be utilized. A stream of electrons bombards them, causing them to fragment. The mass charge ratio (M/Z) is calculated by dividing the fragment's mass by its charge. Almost always, the amount is one. The molecular weight of the fragment is represented by the M/Z ratio.

Each piece is focused via a slit into the detector by a quadrupole (four electromagnets). A computer programs the quadrupoles to direct just particular fragments and to cycle the fragments one by one (scan) until the M/Z range is retrieved.

The mass spectrum is then generated, which is a graph of signal intensity (relative abundance) vs M/Z ratios (essentially molecular weight). Each substance has its own fingerprint and software to generate a library of spectra for unknown substances is easily available.

Chapter Summary

Smoke exposure in indoor smokehouses is a challenge among fish smokers. Biomass fuel usage is a major source of pollution which releases pollutants in the smokehouses. The pollutants such as PAHs, particulate matter, CO, $O_{3,}$ phenol and phthalates could cause respiratory effects such as asthma and non-respiratory effect such as eye diseases. The need to look at these challenges and assess them have been captured under this chapter. Risk assessment was also captured under this chapter.

CHAPTER THREE

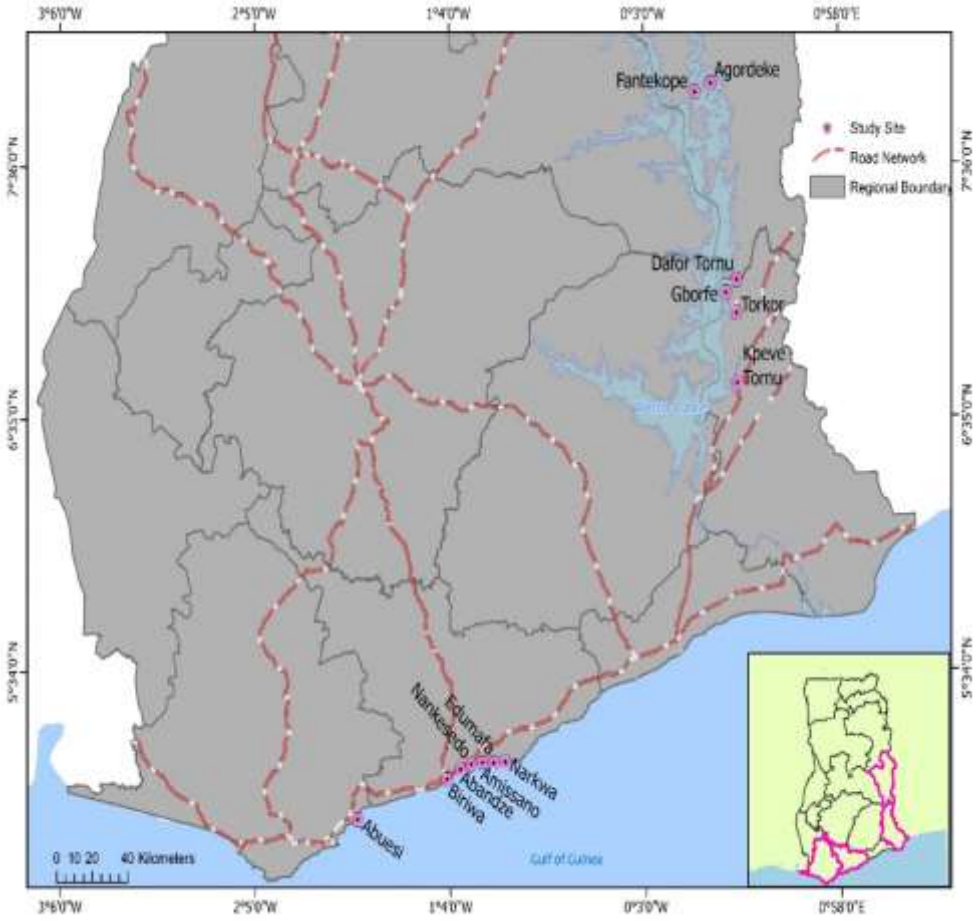
METHODOLOGY

Introduction

In Chapter Three, research design, method and analytical procedure used for analysis such as QuEChERS and GC-MS are discussed. Also, questionnaire and eye screening are highlighted. Air pollution measurements using low-cost air quality monitors, MicroPem and Lascar are discussed. In addition, statistical tools used for data analysis are also discussed.

Research Design

In the first study, six communities in the Central region (coastal; Atlantic, Ocean, marine fishing) and six communities in the Volta region (inland Lake Volta, freshwater fishing) were conveniently selected as study sites. The second study was carried out at Abuesi, in the Western region. The map of the study areas can be seen on Figure 4. The study areas include some fish smoking communities in Central, Volta and Western Regions of Ghana.



Study Areas

Figure 4: Map of Study Areas
Source: Geography Department, University of Cape Coast

Abuesi

Abuesi is a fishing community in the Western region. It occupies an area of about 3,190 km^2 (Ghana Statistical Services, 2014). The town is situated in the Sharma District and it is about 20 km from Takoradi, the Western Regional capital. Abuesi shares a border on the east by Sharma, on the west by Injreisia, on the north by Inchaban and the south by the Atlantic Ocean. The town is located at 4.98333 or 4°58'60" N, 1°37'60" W in DMS (Degrees Minutes Seconds) and -1.63333 in DMS (Degrees Minutes Seconds) according to (Ghana Statistical Services, 2014). Abuesi lies within the tropical

climate zone and experiences two rainy seasons (Ghana Statistical Services, 2014).

According Obeng (2018), about 75% percent of the houses in Abuesi are built with cement block/bricks with few been constructed with mud. The people of Abuesi have access to portable roads, public toilet facility and electricity. About 70% of the female population are engaged in fish smoking.

There are four first cycle schools in Abuesi, two of them are private schools and the other two are public schools. There are about six religious centers like churches and mosques in Abuesi. The males are mostly fishermen and the females smoke fish as their major occupation. However, in the lean season, most females are engaged in petty trading. Small-scale fish smokers may smoke fish alone or engaged relatives or friends for assistance. Fish smokers do the smoking work alone or employ the services of other fish smokers to assist them. They mostly use old smoking stoves that are made of mud with the smoking done either in an enclosed smoke house or in the open. Most of the women who are involved in the fish smoking business learnt immediately they turn ten (10) years from their parents. Herrings, macquerel and red grouper are the types of fish smoked. Most of them start the smoking business with a limited capital and with the help of few smoke helpers.

In Abuesi, fish is normally smoked in traditional smokehouses which is built normally with wood and/or bricks. Some of the firewood used by fish smokers include: mangrove, esa, cocoa, bolambo, rubber, etc. Smokehouses are usually located few kilometers away from the homes of fish smokers while others are located inside the community. Fish smokers mostly buy the fish at the bank of the sea or buy them from fish sellers who make it available to

them at their smoke houses. Fish are washed and arranged on a metal wiring nets with holes inside to help get rid of water by drying it under the sun for 20-30 minutes then after smoked on mud ovens with fuel wood mostly burnt inside.

Mfantseman District

Mfantseman Municipal is located on Ghana's Atlantic coast, in the Central Region. It covers an area of 300.662 square kilometers and spans for around 21 kilometers along the shore and 13 kilometers inland. The municipality's land area makes up 3.1 percent of the region's total.

Saltpond is Mfantseman's administrative capital. “Abura-Asebu-Kwamankese District borders Mfantseman on the west, Ajumako-Enyan-Essiam District on the north, Ekumfi District on the east and the Gulf of Guinea on the south (Atlantic Ocean). It runs from Eguase (at the far western end) to Mankessim (at the far eastern end) (Ghana Statistical Service, 2014).”

Mfantseman has an average temperature of 24°C and a relative humidity of around 70%, with double-digit maximum rainfall in May-June and October. In the coastal savannah sections, annual total rainfall ranges between 90cm and 110cm and between 110cm and 160cm in the interior along the forest zone's edge. The dry seasons are normally December to February and July to September.

According to the 2010 Population and Housing Census, the municipality has a total population of 196,563, accounting for 9.6% of the total population in the Central Region. Males account for 45.9% of the population, while females account for 54.1 percent.

The Annual Growth rate within the Municipality is 2.8%. There are also 95 settlements within the Municipality. There are four urban centres consisting of Saltpond, Anomabo, Mankessim and Yamoransa. About 27.9% of the population lives in areas classified as urban leaving 72.1% of the population residing in the rural areas. This indicates that majority of the population do not have access to adequate social amenities.

The municipality boasts of abundant fishing grounds along the coast, and fishing has been a popular pastime in coastal towns and villages such as Biriwa, Anomabo, Abandze, Ankaful and Kormantse. Farming is possible due to effective interactions between climate, soils and rivers/streams, particularly in inland places. Crop cultivation is practiced in nearly every portion of the municipality, particularly in the inland sections. Cocoa, oil palm, pineapples, oranges, plantains, maize, cassava, cocoyam and coconut are among the crops grown. Oil palm is planted in Akobima and coffee and cocoa are farmed in Dominase and Kyeakor. These cash crops can be exported or sold domestically at significant profit margins.

"Trading activities, which is an important economic activity is carried out virtually in every area in the Municipality with Mankessim as a major focal point and involves agriculture and fishing products and other merchandise. Other significant trading centres are Saltpond, Anomabo and Yamoransa."

Ekumfi District

The Ekumfi District is located in Ghana's Central Region, along the Atlantic coast. The Mfantsiman Municipality borders the District on the west,

the Ajumako–Enyan–Essiam District on the north, the Gomoa West District on the east, and the Gulf of Guinea on the south.

It has a total land area of 276.65 square kilometers, or 0.12% of Ghana's total land area, making it the fifth smallest of the Central Region's twenty districts. Cretaceous—Eocene marine sands with thin pebbly sands and occasional limestone are found along the beaches.

"Upper and lower Birimian rocks, as well as intrusive Tarkwaian rocks, make up the District. Metalogenetic materials (metals) found in these rocks include valuable metals, light metals and base metals like talc and diamonds. The soil in the District is primarily sandy loam on the lower slopes, with clay loam on the top slopes. Because the soils are richer in plant nutrients, they can support tree crops such as citrus and oil palm, which thrive in the area."

"Also, vegetables such as garden eggs, okro and tomatoes thrive well while other crops like cassava, plantain and maize also thrive in the soils within the District. Due to the nearness to the sea, the soil is predominantly saline. The Ekumfi District is basically a low-lying area with loose quaternary sands. The area has an elevation lower than 60m above sea level. The rivers Narkwa and Emissa drain into the sea via the Narkwa and Emissa lagoons at Narkwa and Emissano respectively (Ghana Statistical Service, 2014)."

"The Ekumfi District with its proximity to the Atlantic Ocean has mild temperatures, which range between 22^{o}C and 34^{o}C. It has a relative humidity of about 70 per cent (Dickson and Benneh, 2001)."

Rainfall peaks twice a year in the District, with peaks in May—June and October. In coastal savanna areas, annual total rainfall ranges between

90cm and 110cm and between 110cm and 160cm in the interior along the forest zone's edge. The months of December through February, as well as July through early September, are significantly drier than the rest of the year.

The district is expected to have a total population of 76,528 people in 2017, with 35,203 men and 41,325 women. The district's annual population growth rate is 3.8 percent. The district has 55 settlements, with Narkwa being the most heavily populated.

The principal farming activity in the District is pineapple production. In the District, other agricultural goods such as vegetables and fruits are also produced in significant quantities.

Fishing is another economic activity that people engage in, particularly along the coast. Suprodo and Narkwa have large-scale salt mining operations. Trading, an important economic activity that incorporates agricultural products and other commodities, is carried out nearly in every part of the District, with Essuehyia serving as a key focus point.

South Dayie District

"The South Dayi District is located between 3020- and 3.5005-degrees north latitude and 0017- and 0027-degrees east longitude. It is bordered on the north-by-North Dayi and Afadzato South Districts, on the east by Ho West District and on the south by Asougyaman District, with the Volta Lake forming the western boundary. The District has a total land size of 358.3 square kilometers, accounting for 1.7 percent of the Volta Region's total land area, with the Volta Lake covering nearly 20% of it (Ghana Statistical Service, 2014). According to the 2010 Population and Housing Census, the population of South Dayi District is 46,661, accounting for 2.2 percent of the region's

total population. Males make up 47.4 percent of the population, while females make up 52.6 percent."

"The District is largely rural, with 61.2 percent of the people living in rural areas vs 38.8% in urban areas. The sex ratio in the district is 90.2. More than one-third (39.0 percent) of the population in the district is under the age of 15. As a result, there is a broad base population pyramid that tapers off with a limited number of old people (those aged 60 and up). The district has an age dependency rate of 86.5 percent (Ghana Statistical Service, 2014)."

"Fish farming was first launched in 2006 and it is quickly gaining popularity. For example, at Kpeve Tornu, there are ten (10) cages, as well as several others at Kayira Tornu. The majority of fish caught in the district are caught in lakes. Migrant fisherman, who supply around 20% of the district's daily fish requirements, do the majority of the interior fishing in the Volta Lake, which runs for 80 kilometers along the district's lake shore."

"Dzemeni is a significant market that relies heavily on the fishing sector. Given the existence of the Volta Lake, Dzemeni has a competitive edge in fishing. Dzemeni's fish catch has steadily increased throughout the years (Ghana Statistical Service, 2014)."

Study Participants

The study participants were selected from 12 communities in the Central Region: Abandze, Nankesedo, Briwa (Mfantseman District), Ammisano, Ekumfi Narkwa, Edumafa (Ekumfi District) and the Volta Region: Torkor, Dafortornu, Gborfe (Kpando District), Kpevetornu, Fantikorfe, Agordeke (South Dayie District). In all, 10 participants were

selected from each of the 12 communities, making up a total of 120 participants.

All 120 participants were considered for this study. In addition, 33 indoor smokehouses were selected at Abuesi in Western Region to measure particulate matter, carbon monoxide and ozone in the smokehouses.

Eligibility Criteria

Inclusion criteria in the "Invisible Fishers" project were applied here and these include women who must be 15-49 years of age, not pregnant, not planning to move from the community during the study period and engaged in fish processing as a primary livelihood activity.

Similar criteria were applied in the cross-sectional study at Abuesi, except for the criterion on pregnancy.

Sample Size

The sample size was calculated using a single population proportion formula (Cochran 1977). The parameters considered were: 95% confidence level (1.96), margin of error (0.05), prevalence of wheezing reported among farmers at Akumadan (27%) (Abdul-Rahaman, 2015).

After calculation three hundred and twenty was obtained. Finally, an estimated of three hundred and twenty people were selected in Abuesi for the study.

Ethical Clearance

Community entry: The leaders in the Abuesi were contacted as well as the leader of the fish smokers to seek permission to go into the community. A meeting was also held with some fish smokers in the community to explain the procedure to them.

Ethical clearance was obtained from the University of Michigan Health Sciences and Behavioral Sciences Institutional Review Board (HUM00138934) and the University of Ghana Institutional Review Board (ECBAS 0033/17-18).

Questionnaires in Abuesi

In Volta and Central regions, communities were purposefully selected because they are characterized by similar activities; small-scale fisheries system (marine for Central region and fresh water for Volta region). Also, majority of community members are engaged in fishing and fish smoking activities. Ten women in each community were selected for participation.

A cross-sectional study was used for the work at Abuesi whiles purposive sampling was employed for fish smokers in Volta and Central regions. The study was conducted from September 2018 to March 2019.

Quantitative approach was used in a form of field survey with the use of structured questionnaire to investigate the knowledge of fish smoking on human health risk associated with the use of biomass fuel as the main source of energy for the smoking of fish.

The questionnaire also found out whether fish smokers observe any safety measures and use any protective apparel during fish smoking. The distance from smokehouses to the homes of fish smokers was also asked to check if it had any effect on the parameters being measured. The relationship between the number of years spent smoking fish and experience of health effects such as headache, cough, phlegm/ wheezing, breathlessness, respiratory (chest cold, chest illness) eye disease (redness), asthma and

pneumonia was also assessed. About 292 fish smokers were interviewed at Abuesi.

Personal CO and PM $_{2.5}$ Exposure Measurement and Indoor Air Monitoring

Personal CO and PM $_{2.5}$ was measured among fish smokers in the Central and Western regions. From May through July 2018, each participant was asked to wear a Lascar EL-USB-CO monitor (Lascar Electronics, Erie, PA, USA) and enhanced children's MicroPEM Monitor (ECM) (ECM, RTI International, Raleigh, NC) and for a period of 48 hours. All Lascar EL-USB-CO monitors were calibrated before use. Each Lascar EL-USB-CO was programmed to measure and record CO concentration (parts per million [ppm]) once every five minutes. The monitor was secured in a breast pocket of a specially designed apron and was proximate to the breathing space of the participant, allowing for estimation of the amount of inhaled CO.

The level of particulate matter (PM $_{2.5}$), carbon monoxide, ozone, humidity and temperature from biomass combustion from fish smoking was measured using low-cost monitors in 33 smokehouses. Low- cost air quality monitors were mounted in the various smokehouses to measure temperature, CO and PM $_{2.5}$ in the smoke houses for 24 hours. The monitors were checked from time to time if they are working effectively to ensure quality data collection.

Eye Screening

Fish smokers who work in the selected smokehouses in Abuesi were selected for eye screening. Eye screening was done with the help of some optometry doctors from Department of Optometry, University of Cape Coast

to check for some ocular disorders faced by fish smokers using Logmar visual acuity chart, opthalmoscope, retinoscope and handheld slit lamp biomicospe.

Seventy-seven participants were screened with the help of doctors from optometry Department University of Cape Coast. A standardized questionnaire on demographic data, employment history and ocular health history was completed by all participants. Participants in the study had a full eye examination that included visual assessment with the logMAR, an external eye examination with a handheld slit lamp, funduscopy, objective refraction with a hand held retinoscope and subjective refraction

Urine Sample Collection

One hundred and fifty-five (155) urine samples (60, 59 and 36 from Central, Volta and Western respectively) were collected from fish smokers. Sterile, metal-free plastic urine containers were used to collect urine samples from study participants. They were instructed to void out the first portion of the urine stream before collecting 15-20 mL midstream urine into the plastic containers. Urine samples were kept into cool containers containing ice packs at 4-8º C and transported to laboratory at the Department of Chemistry, University of Cape Coast for analysis.

Urine Sample Preparation

The method that was employed was AOAC method 2007, QUECHERS Q110 En (Restek, www.restek.com). A 4 mL urine sample was pipetted into 10 mL centrifuge tube and 4 mL acetonitrile added followed by adding 1.625g of QUECHERS salt and vortexed for 5 minutes. The resulting mixture was centrifuged for 15 minutes at 3500 rounds per minutes. The

supernatant was decanted into another tube containing primary secondary amine (PSA) and $MgSO_4$ for cleanup and drying respectively.

The cleaned-up sample was then centrifuged for 15 minutes at 3500 rpm and collected into 1.5mL glass sample vials for GC-MS analysis (AOAC method 2007).

Chemicals and Standards

High-purity reagents and standards were utilized.

"Restek provided the 8270 Mega mix standards (#31850), the SV internal standards (6 components; 31206), the B/N Surrogates mix (4/89 SOW, #31062) and the GCMS tuning mixture (benzidine; DFTPP; 4,4′-DDT and pentachlorophenol, #31615). Millipore Corporation, Germany provided GC grade hexane (99.8%) and dichloromethane (99.8%, # K4799165633) solvents; BDH Chemicals Limited, Poole, England provided silica gel (60120 mesh) and DAEJUNG chemical and metals supplied anhydrous Na_2SO_4 (99.0%, #7630-4405), acetonitrile 99 percent analytical grade and acetone 99 percent analytical grade. QUECHERS salt containing 1g NaCl, 4g $MgSO_4$, 1g trisodium citrate dihydrate, 0.5g disodium hydrogencitrate for extraction was also obtained from Restek."

QUECHERS pouch containing PSA (primary secondary amine) and magnesium sulphate for clean-up and drying respectively (4mL packed in 10ml centrifuge tube) was obtained from Restek.

Preparation of Standard and Operational Conditions for GC-MS

From a 1000 ppm standard, standard solution analytes of 10, 20, 50, 100 and 500 ppb were generated. The analytes in the urine samples were analyzed using a Shumazu GC-MS model 2020.

The GC/MS analysis was performed using "EPA method 8270 (SIM) with minor modifications to improve selectivity and sensitivity. The analysis was performed using a Shimadzu GCMS QP2020 system with an AOC 20i auto injector. The capillary column utilized was a Rtx-5 ms fused capillary column with dimensions of 30.0m (length), 0.25mm (ID) and 0.25m (thickness). As the carrier gas, helium (purity: 99.9995) was used."

"The injection port temperature was initially set at 265.0 °C, whereas the column oven temperature was set at 70.0 °C. For GC activities, temperature programming was used. The temperature was set to 70 degrees Celsius and kept for 2.0 minutes. It was then ramped to 90°C at a rate of 20°C/min, then to 250°C at a rate of 10°C/min."

"The temperature was then increased at a rate of 5.0 °C/min to 300 °C and maintained for 3.0 minutes. A total of 32.00 minutes of programming was used. The injection volume was set at 1.0 liters. The flow control mode was linear velocity, with a linear velocity of 42.3 cm/sec for a column flow of 1.33 mL/min and a total flow of 8.7 mL/min."

Operational Conditions of Mass Spectrometer

"The electron impact ionization source was used and quantitative data were collected using Selected Ion Monitoring (SIM) mode with ≥ 2 ions monitored for each compound. The temperatures of the ion source and the interface were set at 230 °C and 280 °C respectively."

Controlling Quality

"This investigation used an internal standard quantitative procedure. For quantification, a five-point calibration curve for 8270 standards ranging

from 0.01 to 0.5 mg/L was employed, with 50.0 L of 5.0 mg/L internal standard (ISTD) added to each."

To "verify for method recovery, surrogate standards (S) were added to each standard (0.30–3.0 mg/L of S) and sample (EPA method 8270). For each of the 10 continuous sample runs, initial calibration standards (ICVs) at 0.2 mg/L and CCVs at 0.5 mg/L were used to validate the GCMS method."

"For each batch of sample analysis, a method reagent blank spiked with ISTD and surrogates were examined first. Manual tuning was performed every 12 hours using the GCMS tuning mixture, in accordance with the criteria for method 8270 E/D."

Data Analysis Methods

Data obtained from questionnaire was checked in order to correct for discrepancies. Data analysis was done using the Statistical Package for Social Sciences version 22 (SPSS, Chicago, IL, USA). The tests of independence between the main explanatory variable and other variables were done using the Pearson Chi square test.

The test for variance in means of variables in the different regions was also done using ANOVA. On statistical analysis on personal $PM_{2.5}$ and personal CO, SPSS version 25 was used for descriptive statistics and Graph Pad Prism version 8 was used for the correlational analysis.

Risk Assessment

To clarify the effects of changes in urinary creatinine [UCr] alone on urinary biomarker determinations, we first assumed that there is no change in urinary biomarker excretion; we assumed an arbitrary biomarker excretion rate of 60 ng/h, a urinary flow rate of 0.6 dl/h and an UCr excretion rate of 60

mg/h. The baseline concentration of the urinary biomarker is 60 ng/dl (absolute) and 1 ng/mg creatinine (normalized).

"A survey published by Unwin, Cocker and Scobbie (2006) which involved an occupational hygiene study of 25 sites using both airborne monitoring of 17 individual PAHs and biological monitoring set the relationship between airborne BaP and urinary 1-OHP" to be almost linear therefore making it reasonable to use biomonitoring of urinary 1-OHP to assess extent exposure to PAH.

Exposure levels in $\mu g/m^3$ can be back-calculated from urinary 1-OHP as follows: the concentration of airborne BaP is in $\mu g/m^3$ and the concentration of urinary 1-OHP in µmol/mol creatinine.

$$\text{Concentration of airborne B[a]P} = \frac{(\text{Concentration}_{1-\text{OHP}} - 1.13)}{11.1} \quad (1)$$

Urinary biomonitoring data may be expressed as µg 1-OHP/g creatinine. To convert µg 1-OHP/g creatinine into µmol 1-OHP/mol creatinine, a factor of 1.93 can be used. "Creatinine is excreted from the human body in urine at a fairly constant rate per day and is dependent on the sex of the individual (1.642 g/day for men and 1.041g/day for women, on average (James, Sealey, Alderman, Ljungman, Mueller and Pecker, 1988). Because the total daily volume of urine excreted was not measured in this study, these creatinine excretion rates were used in calculation."

"Biomonitoring of 1-OHP is based on the excretion of pyrene metabolites in urine and therefore reflects exposure to PAH but only indirectly reflects the risk posed by systemic exposure to BaP."

"As a result, occupational biomonitoring of urinary 1-OHP has been used extensively as a biological monitoring indicator of exposure to PAHs (Unwin et al., 2006)."

"Estimated daily intake doses of pyrene for adult women from the respective study areas was calculated using the following equation (Lakind and Naiman, 2008)."

$$\mathrm{DD} = \frac{C \times P \times EF \times ED}{BW \times AT \times F} \qquad (2)$$

where DD is the daily intake dose of pyrene (mg/kg-day),

"C is the urinary 1-OHP concentration (mg/mL) obtained in this study (for each studied region), P = expected urinary output (mL/day), W = bodyweight (kg), and F= fraction of the ingested dose of pyrene eliminated as 1-OHP in urine."

"The recommended urinary output value of 576–2124 mL/day, even distribution for 24 hours (Pérez-Maldonado, Ochoa-Martínez, López-Ramírez and Varela-Silva, 2019) was used in this study for adult females since urine volumes were not recorded in this study (Brendle, 2007)."

"Research has indicated that humans are exposed to pyrene particularly through dietary ingestion in non-occupational settings (Li, Mulholland and Romanoff, 2010). According to Viau, Hakizimana and Bouchard (2000), 4% of 1-OHP in urine is eliminated after the ingestion of pyrene."

After estimating the daily intake dose of the 1-OHP metabolite in urine, the hazard quotient (HQ) was calculated. Hazard Quotient (HQ) is calculated as the ratio of the daily intake dose of 1-OHP (DD) to its corresponding reference dose (RfD).

$$HQ = \frac{DD}{RfD} \quad (3)$$

where DD is the daily intake dose of pyrene and RfD the reference dose for pyrene compound, given as 30 µg/kg/day.

The reference dosage is the estimated daily dose of a chemical over the course of a lifetime that is unlikely to cause damage (Barnes and Dourson,1988). If the daily dose is less than the reference dose (HQ1), no adverse effects are likely, however if the daily dose is greater than the reference dose (HQ>1), the factor in issue is more likely to cause a morbid response (USEPA, 1993).

Chapter Summary

Fish smokers in some communities in Western, Central and Volta regions are exposed to smoke during their work in indoor smokehouses. Chapter Three has described the study design, study areas, sampling procedures and method of analysis. Also, data collection, data analysis and risk assessment procedure have been captured under this chapter.

CHAPTER FOUR

RESULTS AND DISCUSSION

Introduction

This chapter examines the results obtained after urine sample analysis, indoor air pollution monitoring and personal $PM_{2.5}$ and CO measurements.

The chapter would also discuss results on demographics data on fish smokers as well as data collected during eye screening. Finally, results from indoor air pollution monitoring would be discussed.

PAHs in Urine Samples Among Fish Smokers

The mean concentration of PAHs, are presented in Table 1. In Volta Region, mean PAHs levels ranged from 17.71 ± 38.85 µg/ L for Dibenz [a, h] anthracene to 31519.74 ± 31557.29 µg/ L for pyrene. Mean concentration of benzo[a] pyrene which is a known carcinogen was 2654.63± 2506.02 µg/ L. The mean concentration of pyrene is 6.57% of the total mean PAHs concentration.

This supports the observation made by McClean, Osborn, Snawder, Olsen, Kriech and Sjödin (2012), that pyrene is often 2-10% of total PAHs and that pyrene is present in all PAH mixtures at relatively high concentrations.

From Table 1, the higher molecular PAHs range from 17.71 ± 38.85 µg/ L for dibenz [a, h] anthracene to 2654.63 ± 2506.02µg/ L for benzo(a)pyrene. With the exception of benzo(a)pyrene, the concentration of the higher molecular weight PAHs that is benzo(b)flouranthene, benzo(k)flouranthene, indo[1,2,3cd]pyrene, dibenz(a)anthracene and benzo(ghi)perylene were low compared to the lower molecular weight PAHs.

This is due to the fact that lower molecular PAHs are vapourised at low temperatures as compared to heavy weight PAHs. This shows that the light weight PAHs are vapourise faster and it is susceptible to be inhaled by fish smokers as compared to higher molecular PAHs.

This observation similar to observation made by Zheng et al., 2008; where higher molecular weight urinary hydroxylated metabolite had lower concentration.

Generally, the urinary PAHs concentrations were inversely related to the size of compounds. The higher the molecular weight, the lower the urinary hydroxylated metabolites concentrations.

For substituted naphthalene compounds; 2-Chloronaphthalene had the highest concentration. The mean concentration of 1-Hydroxypyrene; the most commonly used biomarker for PAH exposure was 548.16 ± 22.23 µg/L.

Fish smokers in the three regions could suffer chronic effects such as respiratory diseases asthma and non-respiratory diseases such as eye diseases due to inhalation of pollutants from smoke.

In Central Region, mean PAHs concentration varied widely from 2.57± 6.61 µg/L for dibenz [a,h]anthracene and 2.57 ± 6.57 µg/L for pyrene. Also, the mean concentration of 2-Chloro-naphthalene was the highest that is 4207.66 µg/L. Low molecular weight PAHs had mean concentrations higher than high molecular weight PAHs. The mean concentration of benzo[a] pyrene 2.91 ± 7.69 µg/L.

In Western Region, mean PAHs concentration ranges from 12.41± 13.97 µg/L for benzo(a)anthracene to 1598.50± 4741.82 µg/L for 2-Chloronapthalene. The mean concentration of benzo(a) pyrene was 14.29±

5.88 µg/L. For substituted PAHs, 2-Chloronaphthalene had the highest concentration.

Again, low molecular weight PAHs had mean concentrations higher than high molecular weight PAHs.

There are similar concentration levels of PAHs in the three regions; high PAHs concentration was found for lower molecular weight PAHs and substituted naphthalene.

The results obtained for 1-Hydroxypyrene is higher than those found by Aquino (2016) though that work was done on firefighters and asphalt pavers.

In this study, the concentration level of 1-Hydroxypyrene is also more than those obtained by (Bortey-Sam et al., 2017: Ratelle, Khoury, Adlard and Laird, 2020).

The levels of PAHs in urine of fish smokers could be attributed to exposure through inhalation and skin contact as they go about their activities in the smokehouses (ATSDR, 1995).

The fish smokers used ovens (Chorkor ovens) that are not able to filter the smoke efficiently leading to high levels of PAHs (Nunoo et al. (2018). They could therefore be prone to chronic diseases that are attributed to occupational exposure to PAHs such as eye irritation. Exposure to naphthalene, anthracene and benzo (a) pyrene could lead to irritation of the skin.

Also, exposure to benzo (a) pyrene could lead allergic response in the fish smokers. The fish smokers who had pre-existing health conditions could

also suffer from other chronic health effect such as impaired lung function in asthmatics.

The purpose for analysing urine samples for PAHs levels was to compare them to WHO accepted levels and give necessary recommendations.

Table 1: Mean Concentration of PAHs in Urine Samples (µg/L) in Central, Volta and Western Region

	Volta			Central			Western	(n=155)	
	Mean	Std. Deviation	cv volta	Mean	Std. Deviation	cv central	Mean	Std. Deviation	cv western
Naphthalene	130.75	157.23	120.25	1262.52	2693.26	213.32	150.62	88.69	58.88
Acenaphthylene	86.8	200.63	231.14	69.13	260.93	377.45	1051.3	3291.05	313.05
Acenaphthene	1347.17	5192.86	385.46	374.01	1742	465.76	648.02	2271.09	350.47
Fluorene	1932.7	6243.32	323.04	152.49	520.56	341.37	185.78	568.04	305.76
Anthracene	48.61	86.8	178.56	15.61	18.24	116.85	45.81	21.93	47.87
Phenanthrene	29.57	35.17	118.94	4.9	14.21	290.00	42.65	49.6	116.30
Fluoranthene	148.97	357.47	239.96	2.87	6.24	217.42	18.86	24.24	128.53
Pyrene	31519.7	31557.3	100.12	2.57	6.57	255.64	16.45	13.82	84.01
Benz[a]anthracene	2069.11	2470.53	119.40	2.79	6.59	236.20	12.41	13.97	112.57
Chrysene	127.39	242.47	190.34	5.69	14.23	250.09	26.79	33.73	125.91
Benzo[b]fluoranthene	25.09	53.8	214.43	4.16	11.24	270.19	27.28	45.02	165.03
Benzo[k]fluoranthene	32.79	57.13	174.23	2.8	6.82	243.57	22.28	52.81	237.03
Benzo[a]pyrene	2654.63	2506.02	94.40	2.91	7.69	264.26	14.29	5.88	41.15
Indeno[1,2,3-cd]pyrene	18.2	45.97	252.58	2.96	6.38	215.54	30.24	56.7	187.5
Dibenz[a,h]anthracene	17.71	38.85	219.37	2.57	6.61	257.20	30.83	49.52	160.63
Benzo[ghi]perylene	27.58	59.81	216.86	4.17	8.78	210.55	36.4	87.17	239.48
1-Methylnaphthalene	75.18	75.43	100.33	75.87	334.03	440.27	115.62	88.54	76.58
2-Methylnaphthalene,	134.35	544.36	405.18	1869.84	6751.17	361.06	78.97	53.41	67.63
2-Chloro naphthalene	6989.06	42296.5	605.18	4207.66	1882.66	44.74	1598.5	4741.82	296.64
1-Hydroxypyrene	548.16	22.23	4.06	1294.7	1034.41	79.90	589.72	90.3	15.32

Source: Laboratory work (2018-2019)

The concentration of PAHs in three regions is presented in Figure 5. The PAHs concentration ranges respectively from 17.71-31519.74 µg/ L; 2.57-4394.70 µg/ L and12.41-1598.80 µg/ L for Volta, Central and Western regions respectively. Comparing the PAHs in the samples from the three regions, it could be seen that PAHs concentration in Volta Region were higher than the two other regions.

This could be due to the type of firewood that were used in the various regions.

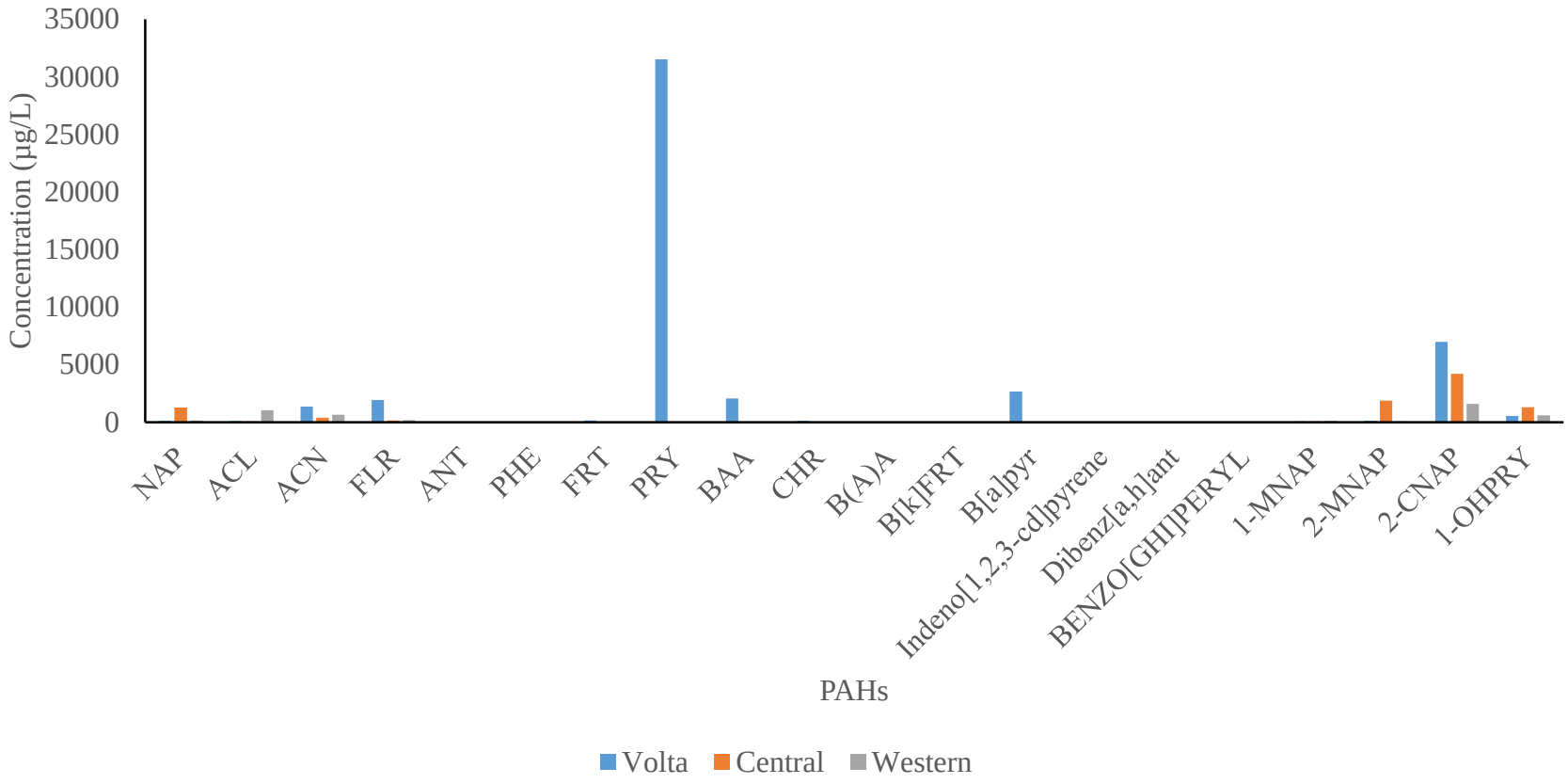


Figure 5: Concentration of PAHs in Urine Sample in Three Regions

Variation in PAHs in urine samples is presented in Figure 6. There was similar variation in all three regions except 1-Hydroxypyrene which was high.

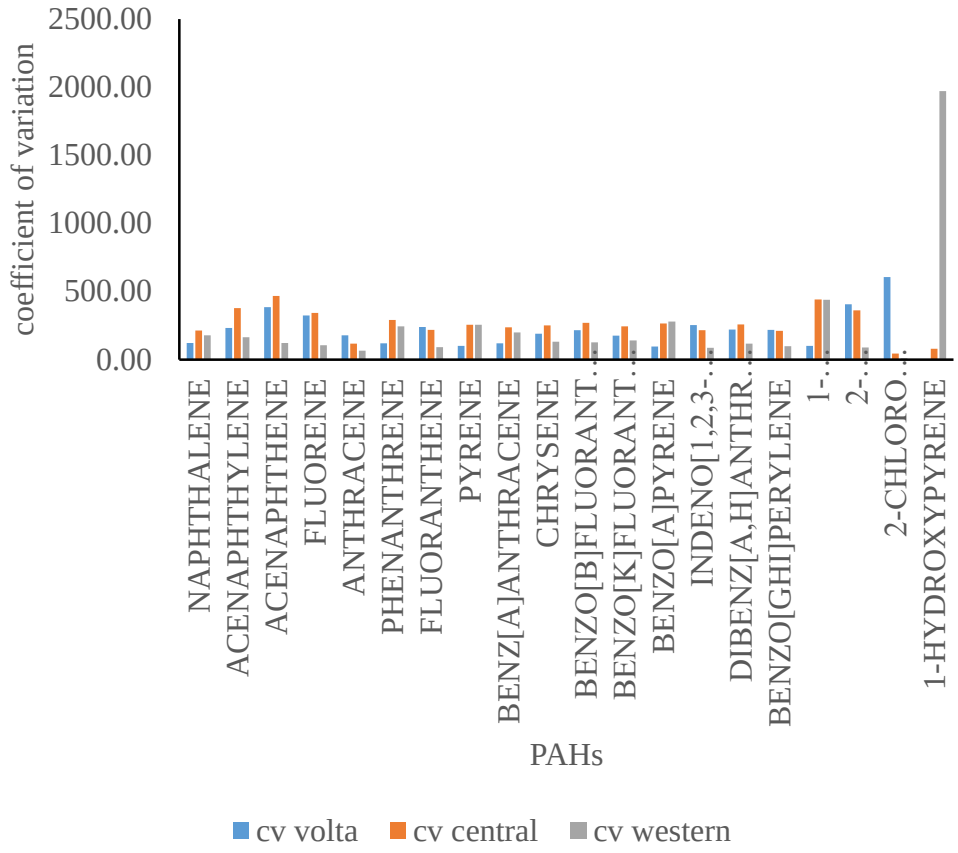


Figure 6: Variation in PAHs Concentration in Three Regions

The concentration of substituted benzene and other substituted compounds are presented in Table 2. "The contribution of environmental indoor biomass smoke is a major cause of indoor benzene exposure. Benzene is a known carcinogen and causes hematotoxicity at exposure levels below 1 ppm (3.25 mg/m^3) (Fustinoni, et al., 2010) which is the recommended occupational limit value (Recommendation from the Scientific Committee on Occupational Exposure Limits for Benzene, 1991)."

Mean substituted benzene in urine sample from Volta Region ranged from 161.98 ± 192.80 µg/L for 4-chloroaniline to 152814.37 ± 474952.20 µg/L for nitrobenzene.

The concentration of substituted benzene in urine sample from Central Region ranged from 21.49 ± 42.01 for 1,4-Dichlorobnenzene and 378833.37± 1775009.46 µg/L for isophorone. Finally, mean substituted benzene in Western Region ranged from 18.15 ± 22.61 µg/L for 1,2,4-Trichlorobenzene to 209725.67 ± 337984.91 µg/L for nitrobenzene.

The concentration level of benzene in smoke particles due to biomass fuel usage could cause some illness among fish as they work over a long number of years smoking fish.

Table 2: Mean Concentration of Substituted Compounds in Urine Samples (µg/L) in Central, Volta and Western Region

	Volta			Central			Western (n=155)		
	Mean	Std. Deviation	CV (VOLTA)	Mean	Std. Deviation	CV (CENTRAL)	Mean	Std. Deviation	CV (WESTERN)
Bis(2-Chloroethyl) ether	4777.26	22237.89	465.49	198.66	842.97	424.33	28255.64	123431.84	436.84
1,2-Dichlorobenzene	11776.58	49353.68	419.08	54.98	171.61	312.13	90.95	284.30	312.59
1,3-Dichlorobenzene	165.23	664.86	402.38	49.83	73.91	148.32	70.85	217.11	306.44
1,4-Dichlorobenzene	298.66	1149.92	385.03	21.49	42.01	195.49	142.63	558.17	391.34
Benzyl alcohol	967.05	4457.66	460.95	90.9	360.85	396.97	1522.33	3560.56	233.89
Bis(2-Chloroisopropyl) ether	21650	156840.25	724.44	2580.4	7271.47	281.80	958.67	1958.28	204.27
Hexachloroethane	6237.48	22480.51	360.41	1010.42	5844.7	578.44	2885.13	11377.47	394.35
Nitrobenzene	152814.37	474952.2	310.80	61698.95	175574.72	284.57	209725.7	337984.91	161.16
Isophorone	274.11	1028.1	375.07	378833.4	1775009.5	468.55	93.21	123.63	132.64
Bis(2-Chloroethoxy)methane	130.9	387.4	295.95	45523.57	87917.94	193.13	357.69	767.35	214.53
1,2,4-Trichlorobenzene	2009.76	2459.17	122.36	4795.57	10228.68	213.29	18.15	22.61	124.57
1,1,2,3,4,4-Hexachloro1,3-butadiene	253.16	470.13	185.70	81195.68	201862.27	248.61	463.29	468.10	101.04
4-Chloroaniline	161.98	192.8	119.03	78178.95	175524.41	224.52	274.18	186.93	68.18
1,4-Dinitrobenzene	423.62	2146.53	506.71	206.95	565.55	273.28	470.59	676.12	143.67
2-Methyl-1,3-dinitrobenzene	121576.78	466133.59	383.41	4762.51	18009	378.14	6678.84	13417.72	200.90
1,2-Dinitrobenzene	9329.28	13419.72	143.85	2204	6541.2	296.79	3893.99	7546.98	193.81
Dibenzofuran	965.92	2640.13	273.33	283.3	958.11	338.20	281.69	600.95	213.34
1-Methyl-2,4-dinitro-benzene	1009.69	1536.79	152.20	4127.71	1762.28	42.69	493.02	999.07	202.64
4-Methyl-3-nitrobenzenamine	2719.7	4713.51	173.31	2482.27	10322.05	415.83	3749.72	4859.78	129.60
4-Bromophenyl ether	25.21	76.42	303.13	3860.11	2124.69	55.04	13.13	5.95	45.32
Hexachlorobenzene	3419.98	2347.66	68.65	27.35	59.6	217.92	22.4	42.64	190.36

Source: Laboratory work (2018-2019)

The concentration of substituted compounds in urine samples in three regions is presented in Figure 7. The level of substituted compounds is generally higher in Central Region as compared to the other regions which could be as a result of types of firewood used in the various regions.

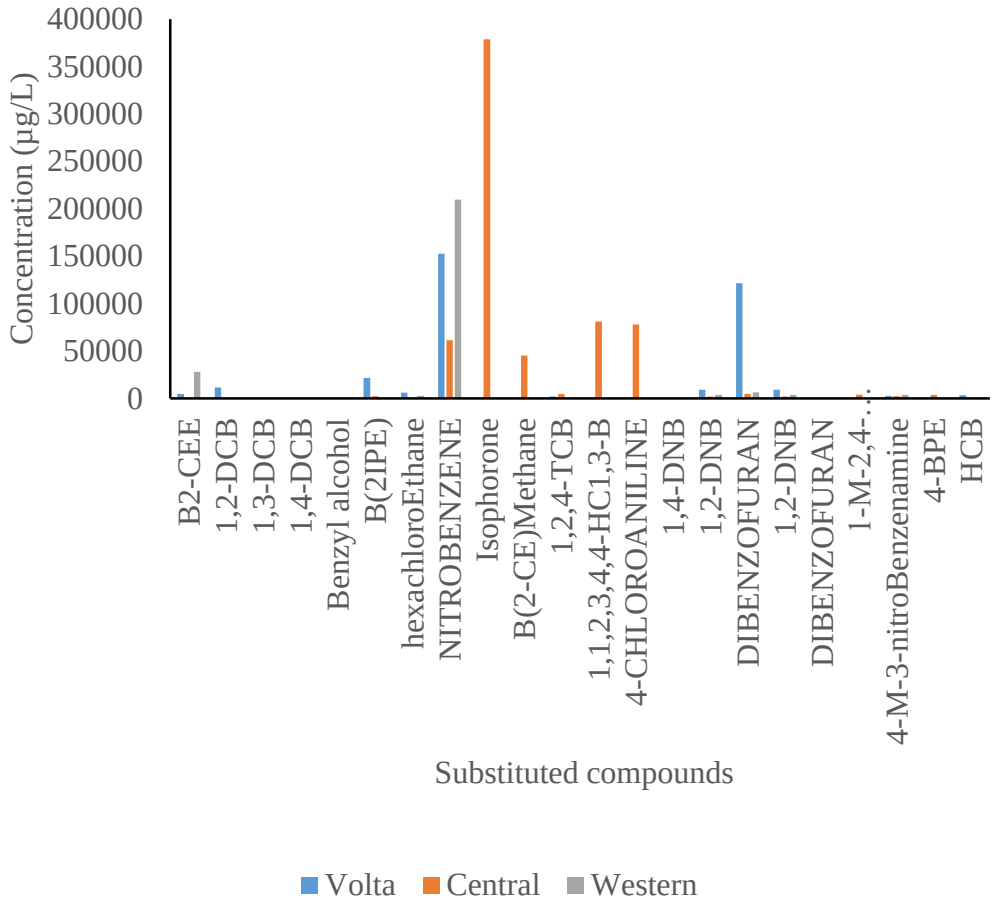


Figure 7: Concentration of Substituted Compounds in Urine Samples in Three Regions

The coefficient of variation in levels of substituted PAHs is presented in Figure 8. There was higher variation for both Volta and Central Regions. In western Region, the variation was not high.

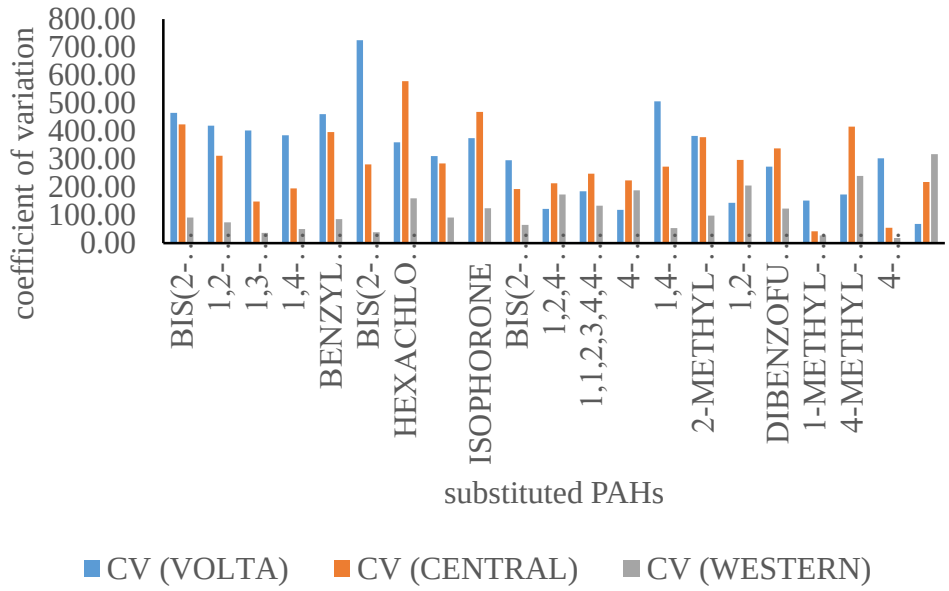


Figure 8: Variations in Substituted PAHs in Three Regions

Phenol compounds play an important role during fish smoking by the addition of taste to smoked fish. The phenol compounds are generated as biomass fuel is being burnt.

The fish smokers used ovens that do not filter the smoke effectively. The smoking activities are also done indoors. Though phenols from biomass burning give taste to smoked fish, fish smokers inhale smoke particles which could affect their health.

The mean concentration of phenols in urine samples are presented on Table 3. The mean concentration of phenols in Volta Region are high with 2, 4, 5-trichlorophenol being the lowest and 2-Methylphenol having the highest concentration.

In Central Region, 2, 4, 5-Trichlorophenol has the lowest mean concentration and 2-Nitrophenol had the highest concentration. In Western Region, 2-Chlorophenol has the lowest mean concentration and 2-Methylphenol has the highest mean concentration.

Table 3: Mean Concentration of Phenol in Urine Samples (µg/L) in Central, Volta and Western Regions

	Volta			Central			Western	(n=155)	
	Mean	Std. Deviation	cv volta	Mean	Std. Deviation	cv central	Mean	Std. Deviation	cv western
2-Chlorophenol	1220.46	7609.41	623.49	1233.55	6468.27	524.36	188.14	661.11	351.39
Phenol	1151443.37	6675863.89	579.78	6203.64	15211.52	245.20	1965496.06	8013515.19	407.71
2-Methylphenol	9474864.25	46677935.09	492.65	221060.18	834833.85	377.65	4309008.28	14021076.01	325.39
3-Methylphenol	328885.71	990526.73	301.18	77468.4	231073.15	298.28	357355.30	588032.40	164.55
2-Nitrophenol	239.75	835.48	348.48	783850.32	2391709.4	305.12	225.32	183.21	81.31
2,4-Dimethylphenol	239.44	439.58	183.59	4107.53	5355.43	130.38	537.81	615.75	114.49
2,4-Dichlorophenol	2551.31	4811.88	188.60	8468.56	27456.51	324.22	2108.68	3599.72	170.71
4-Chloro-3-methylphenol	13309.55	10273.02	77.19	109484.25	404851.82	369.78	1826.62	6594.38	361.02
2,4,6-Trichloro phenol	1143.54	2340.76	204.69	9660.29	37647.09	389.71	13712.82	31350.88	228.62
2,4,5-Trichloro phenol	196.46	528.04	268.78	1595.49	7185.74	450.38	2463.15	9233.17	374.85
2,3,5,6-Tetrachloro phenol	39539.05	177159.25	448.06	44718.76	199597.65	446.34	75201.73	254843.67	338.88
2,3,4,5-Tetrachloro phenol	21755.78	56358.63	259.05	23662.08	94758	400.46	28939.35	107278.29	370.70
9-Phenanthrenol	829916.67	178825.12	21.55	1426110.67	1275356.6	89.43	797178.24	285131.62	35.77

Source: Laboratory work (2018-2019)

The concentration level of phenols for the three regions is presented in Figure 9. The highest concentration of phenol was found in Volta Region.

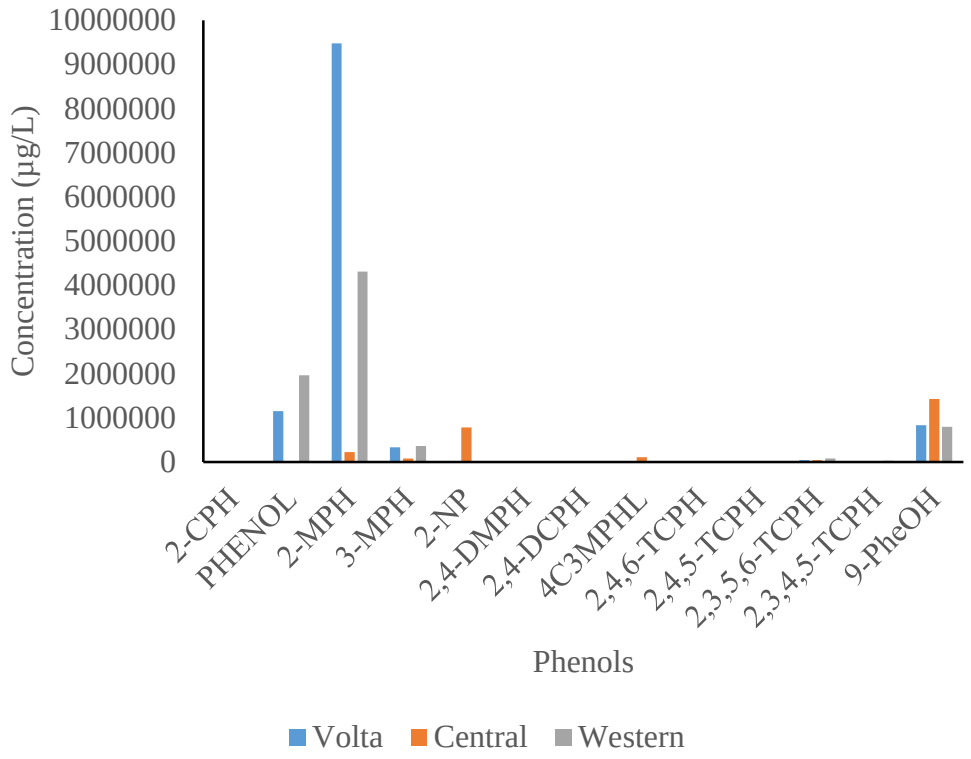


Figure 9: Concentration of Phenols in Urine Samples in Three Regions

The variations in phenols in urine sample from three regions is presented in Figure 10. There were variations in phenols in the urine samples in the three regions.

This could be due to some factors like time spent by fish smokers in smoke houses being different, using different types of biomass fuel and using different amount of biomass fuel.

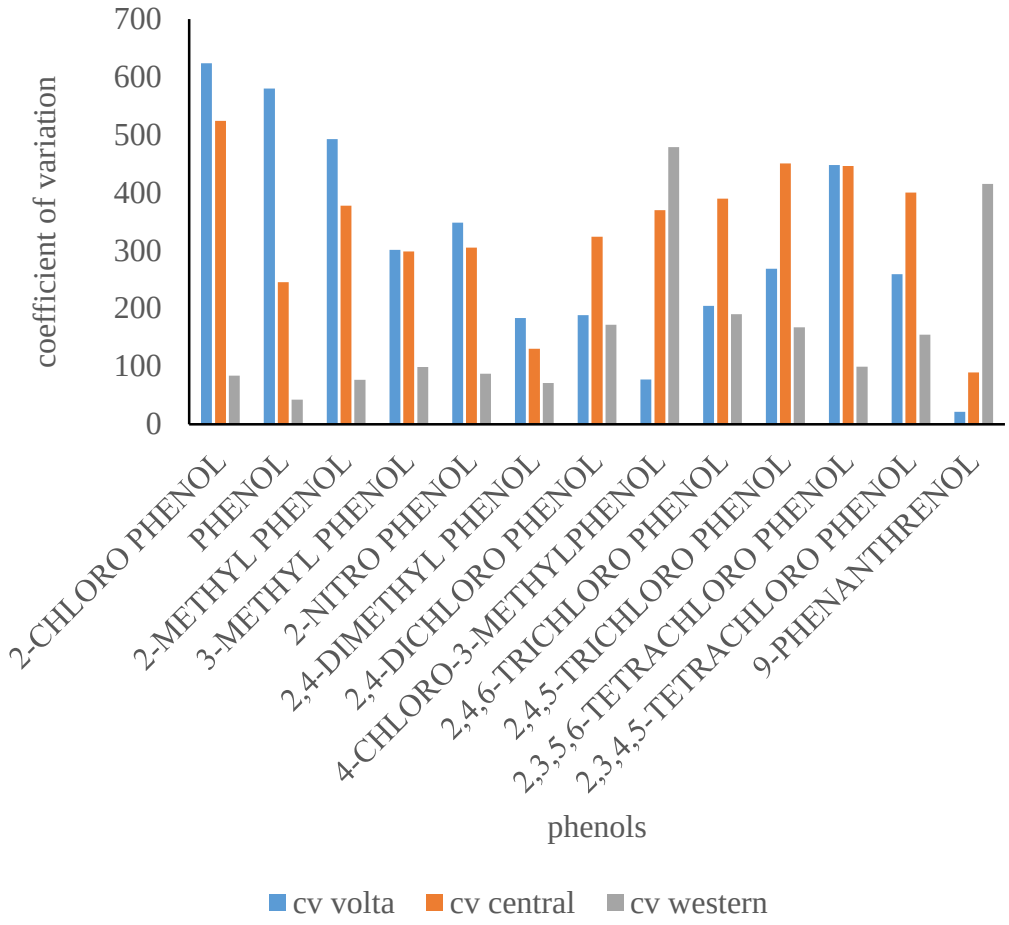


Figure 10: Variation in Phenols in Three Regions

The mean concentration of phthalates is presented in Table 4. The diesters of 1,2-Benzenedicarboxylic acid (phthalic acid), commonly known as phthalates, are a group of man-made chemicals with a wide spectrum of industrial applications.

Although their toxicity is regarded to be low, phthalates are potential endocrine disruptors. Ingestion, inhalation and cutaneous exposure can all result in combined exposure. Exposure to phthalates during fish smoking could be as a result of polyethylene bags and plastics that are used to set fire by fish smokers.

Table 4: Mean Concentration of Phthalates in Urine Samples (µg/L) in Central, Volta and Western Regions

	Volta			Central			Western (n=155)		
	Mean	Std. Deviation	cv volta	Mean	Std. Deviation	cv central	Mean	Std. Deviation	cv western
Dimethylphthalate	4088.85	11188.6	273.64	1459.92	5914.47	405.12	554.87	1065.48	192.02
Diethylphthalate	971.06	2593.96	267.13	20948.9	115503	551.36	36260.3	150180.00	414.17
Dibutylphthalate	303.83	717.85	236.27	105.35	240.68	228.46	198.21	112.96	56.99
Benzylbutyl phthalate	212.86	297.63	139.82	36.65	93.93	256.29	102.65	131.33	127.94
Hexanedioic acid dioctylester	209.2	534.61	255.55	77.03	411.56	534.29	465.05	633.67	136.26
Bis(2-Ethylhexyl)phthalate	496.56	916.77	184.62	60.93	139.46	228.89	282.67	401.69	142.11
Di-n-octylphthalate	29.09	40.41	138.91	11.97	53.76	449.12	32.79	67.52	205.92

Source: Laboratory work (2018-2019)

Mean concentration of phthalates in urine sample from Central Region ranged from 29.09± 40.41 µg/L for Di-n-octyl phthalate and the highest concentration was found in Dimethyl phthalate (DMP); 4088.85± 11188.64 µg/L.

In Volta Region, Di-n-octyl phthalate had the lowest concentration and dimethyl phthalate had highest concentration.

In Western Region, Di-n-octyl phthalate had the lowest concentration and diethylphthalate had the highest concentration.

The concentration of Diethylpthalate was the highest all the three regions. Mean concentration of Diethyl Phthalate was highest for both Central and Western Regions.

The concentration level of phthalates is presented in Figure 11 where the highest concentration was found in Western Region.

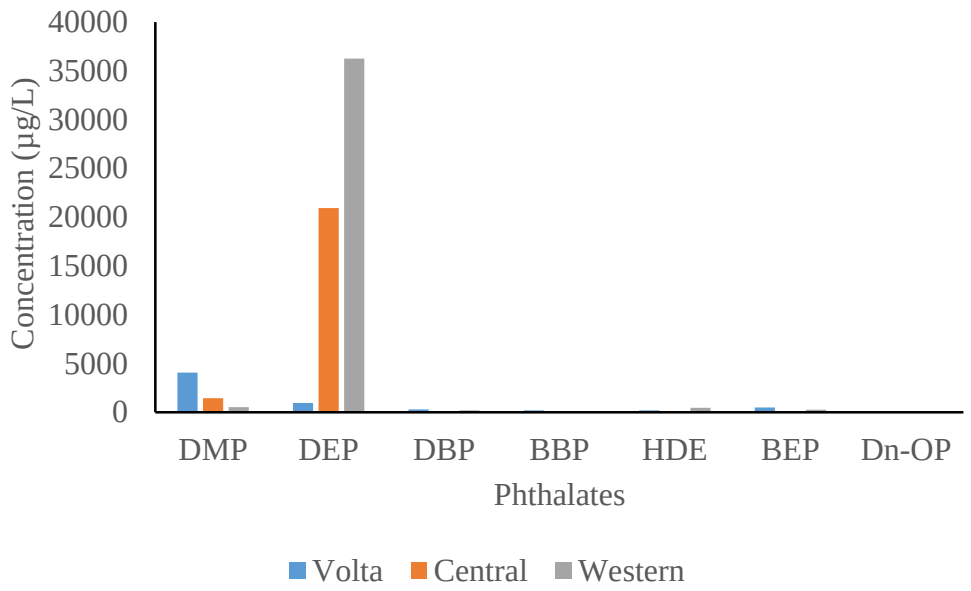


Figure 11: Concentration of Phthalates in Urine Samples in Three Regions

Variations in phthalates in urine sample is presented in Figure 12. In Volta region, there was similar variations in Dimethyl phthalate,

Dethylphthalate, Dibutylphthalate and Hexanedioic acid dioctyl ester were similar but bis(2-Ethylhexyl) phthalate and Di-n-octyl phthalate had different variations.

For Central and Western Regions, there were variation in all the phthalates. Sometimes, fish smokers use polyethylene bags, take away pack, tyres and cartons to set fires. These could have led to phthalate being released as part of the smoke.

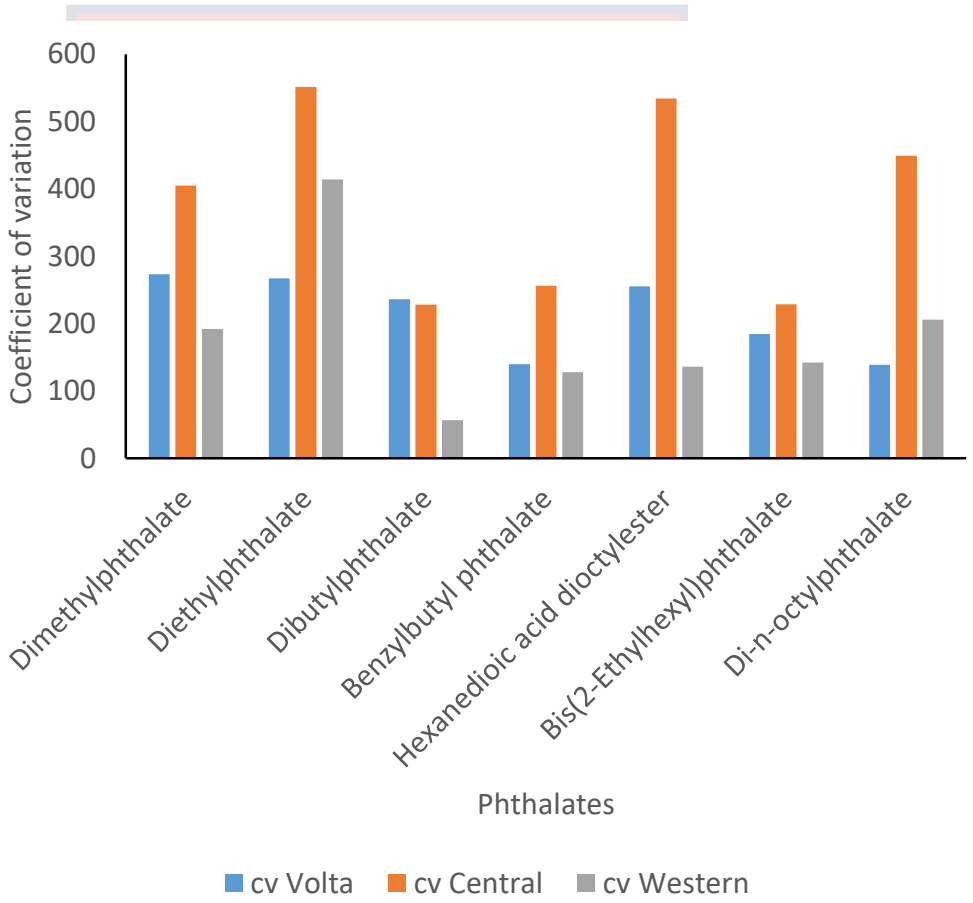


Figure 12: Variations in Phthalates in Three Regions

Analysis of variance was conducted for the parameters measured for urine sample in the three region in order to check for differences in the means and is shown in Table 5. It was realized that most of the mean of parameters

were not significantly different. Some of the parameters however, had significant difference among the three region which are presented in Table 5.

Table 5: p- values for Analysis of Variance for Urine Sample Parameters in the Three Regions

Analyte	p-value
1,2-Dichloro-benzene	0.000
Bis(2-chloroethoxy)methane	0.000
2,4-Dimethylphenol	0.000
Naphthalene	0.001
4-Chloroaniline	0.001
Acenaphthylene	0.029
1-Methyl-2,4-dinitrobenzene	0.000
4-Bromophenylether	0.000
Hexachlorobenzene	0.000
Anthracene	0.050
Phenanthrene	0.000
Pyrene	0.000
Benzyl butyl phthalate	0.002
Dioctyl esterhexanedioic acid	0.031
Benz[a]anthracene	0.001
Chrysene	0.003
Bis(2-Ethylhexyl) phthalate	0.014
Benzo[a]pyrene	0.000
Indeno[1,2,3-cd]pyrene	0.047
Dibenz[a,h]anthracene	0.014

Source: Data Analysis

Health Implications of PAHs, Metabolites, Substituted Compounds, Phenol and Phthalates

The Agency for Toxic Substances and Disease Registry ATSDR (2009) sets maximum contaminant level of benzo (a) pyrene as 0.2ppb that is 0.2 µg/L. The levels of PAHs, metabolites, substituted compounds, phenol and phthalates obtained for some of urine samples were higher than 0.2 µg/L.

This implies that fish smokers could be affected if they work for a number of years. Fish smokers could show health symptoms such as coughing, headache, wheezing, breathlessness, asthma and pneumonia. They could also be affected by eye diseases such as cataract and dry eye syndrome.

The demographic characteristics of fish smokers in Abuesi are presented on Table 6. In Abuesi, 4(1.3%) men and 77(25.5) representing 26.8%; fish smokers were less than 30 years. Again, 10(3.3%) men and 193(63.9%) females representing 67.2% were between 30 and 65 years. Finally, 2(0.7) men and 16(5.3%) totaling 6 % were above 65 years. This means that most of the youth in Abuesi; some few men and a lot of women are engaged in fish smoking.

Table 6: Demographic Characteristics of the Study Population

Demographics	Gender	Male n(%)	female n(%)	Total n (%)	X^2 / p-value
Age	less than 30	4(1.3)	77(25.5)	81(26.8)	2.492 / 0.646
	30-65	10(3.3)	193(63.9)	203(67.2)	
	>65	2(0.7)	16(5.3)	18(6.0)	
	Total	16(5.3)	286(94.7)	302(100.0)	
Highest level of education	non formal	7(2.4)	87(29.6)	94(32.0)	
	Primary	4(1.4)	82(27.9)	86(29.3)	6.423/.779
	junior high	2(0.7)	86(29.3)	88(29.9)	
	senior high	1(0.3)	15(5.1)	16(5.4)	
	vocational/technical	0(0.0)	4(1.4)	4(1.4)	
	Tertiary	0(0.0)	6(2.0)	6(2.0)	
	Total	14(4.8)	280(95.2)	294(100.0)	
Marital Status	Unmarried	1(0.3)	51(17.4)	52(17.7)	3.552/.895
	Married	8(2.7)	152(51.9)	160(54.6)	
	Divorced	3(1.0)	37(12.6)	40(13.7)	
	Cohabitation	1(0.3)	11(3.8)	12(4.1)	
	Widow	1(0.3)	28(9.6)	29(9.9)	
	Total	14(4.8)	279(95.2)	293(100.0)	
Religion	Christian	14(4.8)	230(78.5)	244()	3.588/.892
	Muslim	0(0.0)	39(13.3)	39(13.3)	
	Traditional	0(0.0)	10(3.4)	10(3.4)	
	Total	14(4.8)	279(95.2)	293(100.0)	

Source: Field work (2018-2019)

From Table 6, looking at the level of education, 7(2.4%), 87(29.6) totaling 32% of the fish smokers had non- formal education.

There were 4(1.4%) and 82(27.9); amounting to 29.3% who had primary school education. In total 29.9% had Junior High education with 2(0.7%) being men and 86(29.3%) being women. With Senior High education, there were 5.4% in all; with 1(0.3%) man and 15(5.1%) women. A total of 1.4% had Vocational/Technical and 2% had attained tertiary level.

The above shows that the majority of the people surveyed had non-formal education, followed by primary school education, Junior High education and Senior High education. Per the survey, the percentage of people who attended Vocational/Technical and tertiary level were low.

"Educational attainment is widely believed to be a very important component of human capital. It is also an effective avenue for improving income-earning capacity."

"Higher education, particularly for women, is usually associated with greater knowledge and use of good cooking practices as well as providing alternatives to drudge work."

The results obtained in this work for level of education is similar to the work done by Obeng (2018) and Flintwood -Brace (2016)) where most of the people interviewed had no formal education.

Also, the result on level of education in this work corroborates Odoi –Agyarko (2009) which showed that the level of education among women who used biomass as fuel in the Bongo district was low.

This work's finding on level of education again agrees with Ghana Statistical Service, 2010 that formal education among most people living in coastal areas is low.

In Pakistan, work done by Qasim, Ghani, Anees, and Bashir, (2013). on 'Indoor Particulate Pollutant (Biomass Fuel) Epidemiology and Socio Environmental Impact and Assessment of Awareness Level among Women' concluded that majority of respondents were illiterate in the Sabour village.

From Table 6, majority of the fish smokers were married representing 54.6%, 17.7% unmarried and 13.7% divorced, 4.1 % cohabitation and 9.9% widows. From Table 6, about 83.3% of the fish smokers were Christians, 13.3% Muslims and 3.4% belonging to the Traditional religion.

The results obtained is similar to work done by Obeng (2018) though there were some slight disparities due to the fact that sample sizes were different from this work. Work done by Flintwood (2016) is also similar.

The Mean number of years spent smoking is presented on Table 7. The mean age for males was 45.250± 17.9833 years and mean age of women was 40.420 ±14.6313. Mean number of years spent smoking was 17.429 years ±17.7014 years for males and 13.843±11.8783 for females.

The mean number of years spent smoking shows that a fish smoker might spend most of time in the fish smoking business in order to cater for themselves and their dependents.

They therefore need the necessary environment that is conducive for their health. They also need to be well informed about effects of exposure to smoke that would enable to embark on safety measures in order to reduce biomass smoke exposure.

Table 7: Mean Number of Years Spent Smoking

Gender		Number of years spent smoking	Age
Male	Mean	17.429	45.250
	N	14	16
	Std. Deviation	17.7014	17.9833
Female	Mean	13.843	40.420
	N	280	286
	Std. Deviation	11.8783	14.6313
Total	Mean	14.014	40.675
	N	294	302
	Std. Deviation	12.2000	14.8320

Source: Field work (2018-2019)

Some health effects that were self- reported by the fish smokers are presented in Table 8. Most of the fish smokers did not report any symptoms for the different health effects.

Those who reported headache had worked for a mean of 13.72 ± 12.67years but there was no significant difference between exposure duration and headache. The result obtained in this study is different from Obeng (2018) where there was significant difference between exposure duration and headache.

Those who reported of coughing had spent an average of 13.08 ± 11.95 years in fish smoking but there was no significant difference between exposure duration and a fish smoker suffering from coughing. This results obtained for coughing is also different from Obeng (2018) where exposure

duration had significant difference with a fish smoker suffering from coughing.

For those who reported on phlegm and wheezing, the mean years spent smoking fish was 12.32 ±12.85 years but there was no significant difference between exposure duration and a fish smoker suffering from phlegm and wheezing.

Breathlessness was also reported with mean years spent smoking being 10.26 ± 11.15. There was a significant difference between exposure duration and a fish smoker suffering from breathlessness. The finding for breathlessness in this study is similar to Flintwood-Brace (2016) where there was association between exposure duration and breathlessness for fish smokers.

With effect of respiratory symptoms, the mean number of years spent by the fish smokers was 13.38 ± 12.44 but there was no significant difference between exposure duration to smoke and respiratory symptoms.

This study does not corroborate with finding by Umoh and Peters (2014) though their work was done on the relationship between lung function and indoor air pollution among rural women in the Niger Delta region of Nigeria.

The finding in this work for respiratory symptoms does not also corroborate Sanbata, Asfaw and Kumie (2014) on their work about biomass fuel use with acute respiratory infections among children in a slum urban of Addis Ababa, Ethiopia.

Fish smokers also reported eye diseases. This was done with the help of Doctors from Optometry Department University of Cape Coast. The mean

number of years spent smoking for those reported eye disease was 13.38 ±11.62. There was significant difference between exposure duration and a fish smoker suffering from eye disease.

This shows that the fish smokers could be affected by eye diseases if they work for long period of time. Response on eye symptoms in this study is similar to that of Obeng (2018) where exposure duration had significant difference with a fish smoker suffering from eye disease.

The finding on eye disease in this study also corroborates Odoi-Agyarko (2009) in which eye irritation, in the form of the tearing was prevalent among women who used biomass fuel for cooking.

There were fish smoker who had worked for a mean of six years in which there was significant difference between and asthma. Smoking fish over a long period of time could expose the fish smoker to prolong asthma. Asthma continues to be a health effect associated with biomass fuel exposure to smoke particles.

This study is in agreement with Oluwole, Ganiyu, Dezheng and Christopher (2016) though their work was done among school children suffering from asthma in Nigeria.

Barry et al. (2010) also explains that current or former asthma was reported by 21.3% of an adult population. A history of using biomass fuels when cooking indoors significantly increased the risk of reporting current asthma in this population in their study.

Fish smokers could be exposed to smoke as they go about their duties. As a result, they could inhale smoke particles such as particulate matter,

carbon monoxide, PAHs which could affect fish smokers with or without history of asthma.

Pneumonia was also reported where mean number of years spent smoking was 6.14± 8.59 years. There was significant difference between exposure duration and pneumonia among the fish smokers.

Other health effects that were reported by those who had worked for a mean of 5.25± 3.59 years. There was significance between exposure duration and being affected by those other diseases.

Table 8: Association among Duration of Exposure to Smoke and Self-Reported Health Outcomes

		Number of years spent smoking			
		Mean	Standard Deviation	F	p-value
Headache	No	14.56	11.32	.361	.548
	Yes	13.72	12.67		
Cough	No	14.76	12.38	1.279	.259
	Yes	13.08	11.95		
Phlegm/wheezing	No	14.66	11.91	2.417	.121
	Yes	12.32	12.85		
Breathlessness	No	14.84	12.29	6.099	.014
	Yes	10.26	11.15		
Respiratory (chest cold, chest illness)	No	14.61	11.98	.829	.363
	Yes	13.38	12.44		
Eye disease (redness)	No	17.02	14.41	3.868	.050
	Yes	13.38	11.62		
Asthma	No	15.13	12.23	18.595	.000
	Yes	6.00	8.50		
Pneumonia	No	15.08	12.24	17.297	.000
	Yes	6.14	8.59		
Others	No	14.13	12.23	2.082	.150
	Yes	5.25	3.59		

Source: Field work (2018-2019)

Table 9 below shows number of children below 5 years who come to the smokehouses. Some children followed their parents to the smokehouses during fish smoking. The mean number of children below 5 years who go to the smokehouses with their parents and guardians was 2.7±2.2 and the total mean for number of respondents working in a particular smokehouse is 3.3±2.0.

This study was not about children under 5 per say; it was necessary finding out about children who come to the smoke house as this could lead to further study.

Children below five years spending time at the smokehouse is a concern as this could predispose them to some health effects because children could easily be prone to diseases such as asthma, eye diseases and other respiratory symptoms due to their weak immune system.

Pravallika, Sharvani, Nandini and MurikiPudi (2018) explains that long duration of exposure to biomass fuel mostly affects women, young girls and children.

During fish smoking biomass fuel is being used and a lot of smoke pollutants are being released particularly indoor biomass usage. Such high levels of pollutants are due to the high proportion of unprocessed biomass fuels which have high pollutant emission factors.

Also, fish smoking is carried out in an enclosed space with poor ventilation and inefficient stoves.

Rumchev, Win, Bertolatti and Dhaliwal (2016) found that $PM_{2.5}$ and CO were significant contributors for the prevalence of acute respiratory infections and trouble breathing among young children.

Another study by Hasan et al. (2019) found the use of in-house biomass fuel as a significant risk factor associated with respiratory symptoms of children under 5 years of age.

Table 9: Number of Children below 5 Years who Come to the Smokehouse

	Gender					
	Male		Female		Total	
	Mean	Standard Deviation	Mean	Standard Deviation	Mean	Standard Deviation
Number of children below 5 years who come to the smokehouse	2.3	1.7	2.8	2.2	2.7	2.2
Number of people working in a smokehouse	3.6	2.3	3.3	1.9	3.3	2.0

Source: Field work (2018-2019)

Fish smokers self-reported of distance from smokehouses, number of people who work in a smokehouse and the work they do aside fish smoking which is presented in Table 10. Some had their residence closer to their smokehouses and some of them also lived far away from the smokehouses.

There were quite a number of people working in one smokehouse. Some were assisting their parents while others were also hired to do the work.

Some fish smokers engaged in other jobs such as petty trading, dress making when fish is not in bumper season to earn a living. Some also engaged in fish smoking almost all the time.

Table 10: Self- Reported Distance from Residence to the Smokehouse, Number of People in a Smokehouse and Work Aside Fish Smoking

		Count	Table N %
Distance from residence to the smokehouse	.0	3	1.0%
	1.0	240	81.9%
	2.0	48	16.4%
	18.0	1	0.3%
	32.0	1	0.3%
	Total	293	100.0%
Number of people working in a smokehouse	.0	7	2.4%
	1.0	38	12.9%
	2.0	70	23.8%
	3.0	61	20.7%
	4.0	47	16.0%
	5.0	38	12.9%
	6.0	16	5.4%
	7.0	4	1.4%
	8.0	6	2.0%
	9.0	3	1.0%
	10.0	3	1.0%
	11.0	1	0.3%
	Total	294	100.0%
Do you do any work aside fish smoking	13.0	1	0.3%
	No	183	62.2%
	Yes	100	34.0%
	2	5	1.7%
	3	1	0.3%
	5	4	1.4%
	Total	294	100.0%

Source: Field work (2018-2019)

The health hazard encountered by fish smokers is presented in Table 11. Fish smokers had become aware of effect of smoke and heat exposure to their health.

For awareness to smoke, 4.1% responded NO whiles 94.6% responded YES which shows that most them are concerned about their health as they go about their work in the smokehouses.

On awareness to heat, 3.1% responded NO and 96.9% responded YES.

Table 11: Health Hazards Encountered as a Result of Fish Smoking and Awareness of Health Hazards

		Have you ever encountered any health hazards as a result of fish smoking							
				No		yes		Total	
		Count	Table N %	Count	Table N %	Count	Table N %	Count	Table N %
Are you aware high exposure to smoke could affect your health	Yes	2	0.7%	10	3.4%	266	90.5%	278	95.9%
	No	0	0.0%	0	0.0%	1	0.3%	12	4.1%
Are you aware high exposure to heat could affect your health	Yes	2	0.7%	8	2.7%	275	93.5%	285	96.9%
	No	0	0.0%	7	2.4%	2	0.7%	9	3.1%

Source: Field work (2018-2019)

Table 12 below shows safety measures practiced by fish smokers during fish smoking. Some of the fish smokers (66.7%) do not practice any safety measures going about activities in the smoke house.

Some fish smokers (31.3%) fish smokers self-reported that they practice safety measures such as leaving the smoke house for a while when the smoke becomes too much at the smokehouse.

Usage of safety apparel, routine safety measures, using clean stoves and keeping hygiene at the smokehouses could protect the fish smokers from inhaling harmful smoke particles.

Table 12: Safety Measures Practiced during Fish Smoking

		Count	Table N %
Do you practice any safety measures during smoking	No	196	66.7%
	Yes	92	31.3%

Source: Field work (2018-2019)

The types of wood that were used by fish smokers were sugar cane 9.5%, palm kernel 3.4% cocoa 15.3% and acacia10.2% which are presented in Table 13.

There were other types of firewood that were mentioned by the fish smokers apart from those asked in the questionnaire (61.2%) such esa, odum, rubber, yaya, light pole, aboduea, edua bolambo, okanto, abodua, denta, kumkum ban, pepe, duaawon, danwoma, apam, duany, gyama, wawa, nyinadzen, tanta, edua awor, emire, charcoal (using oven), ember and wobinbo.

Table 13: Types and Choice of Firewood Used by Fish Smokers

		Count	Table N %
Type of firewood			
	sugarcane	28	9.5%
	Palm kernel	10	3.4%
	cocoa	45	15.3%
	acacia	30	10.2%
	others	180	61.2%
Which type of firewood do you prefer	sugarcane	8	2.7%
	Palm kernel	2	0.7%
	cocoa	25	8.5%
	acacia	21	7.1%
	others	234	79.6%
What is the reason for choice			
	0	8	2.7%
	1	282	95.9%
	2	1	0.3%
	5	1	0.3%
Type of material used for stove construction	mud	246	83.7%
	cement	24	8.2%
	wood	12	4.1%
	stone	4	1.4%
	others	7	2.4%

Source: Field work (2018-2019)

In this work the reasons why the fish smokers chose the types of firewood include; hardness, better smoke production, nice looking of fish, fast drying of fish, makes fish stronger and better flame. Other reasons were: availability of firewood, easy working, less harmful to eyes, prevents fish from insects, hygienic, adds value and attraction of customers. Other responses were; firewood last long, saves money.

A lot of the fish smokers preferred the firewood called esa since it had most of the qualities they have mentioned.

Some of the types of firewood (acacia, sugar cane) used by fish smokers in this study were corroborated by (Essumang et al., 2013) on the

effect of smoke generation sources and smoke curing duration on the study of the level of PAHs in different suite of fish.

Also, types of firewood that were used by fish smokers in this study is similar to works done by Flintwood- Brace (2016) and Obeng (2018). This work also agrees with Odoi-Agyarko (2009) where most women in the Bongo District of Ghana used firewood as their source of fuel for cooking.

Furthermore, the type of fuel used by fish smokers is comparable to that employed by (Sanbata et al., 2014) in their study of the link between biomass fuel usage and childhood respiratory illnesses in Addis Ababa, Ethiopia.

The type of fuel used in this study corroborates with Oluwole et al. (2016) where biomass fuel was used in some households in Nigeria which had a prevalence for asthma among school children.

The fish smokers use a lot of firewood which is an indication that most of them and their families especially children could be exposed to health effects associated with biomass fuel usage.

Most of the women self–reported of headache cough, phlegm/wheezing, breathlessness, respiratory (chest cold, chest illness), eye disease (redness), asthma and pneumonia. The smokehouses are enclosed and they spend a lot time there.

Again most of the smokehouses are closer to the main houses (bedrooms) which shows people who do not even visit the smokehouse could still be exposed to smoke which have a chronic effect over the years.

The types of material used for construction of stove were mud 83.7%, cement 8.2%, wood 4.1%, stone 1.4% and others 2.4%. Most of the ovens do not work efficiently since they are unable to filter the smoke efficiently.

This could release harmful substance like particulate matter, carbon monoxide and nitrogen dioxide which are detrimental to human health (Bruce, Perez-Padilla and Albalak, 2000; Naeher et al., 2007).

It could also release other harmful substances such as PAHs according to (Nti, et al., 2002). Studies conducted by (Philips, 1999; Stolyhwo and Sikorski, 2005) showed that wood smoke that is produced during fish smoking is an example of incomplete combustion and polycyclic aromatic hydrocarbons (PAHs) are generated and released into the various smoked products.

Essumang et al., (2013) worked on the effect of smoke generation sources and smoke curing duration on the level of PAHs in different suite of fish and recommended the need for improved stoves for fish smoking.

With reference to presenting visual acuity in the better-seeing eye, the prevalence of visual impairment was 11.7% in fish smokers (Table 14). The result showed most of fish smokers that were screened had no vision impairment, some having moderate impairment and the rest being blind.

Table 14: Distance Visual Impairment

Distance Visual Impairment				
		Frequency	Percent	Valid Percent
Valid	normal /mild visual impairment	63	20.8	81.8
	Moderate visual impairment	5	1.7	6.5
	Blind	9	3.0	11.7
	Total	77	25.4	100.0

Source: Field work (2018-2019)

Causes of visual impairment are presented in Table 15. These were cataract (28.6%), ineffective conjunctivitis (5.4%), allergic conjunctivitis (5.4%), dry eye syndrome 5(8.9%) glaucoma suspect (12.5%), pterygium (10.7%), pinguecula, (3.6%) retinopathies (7.1%) and refractive error (17.9%).

There were 11 posterior segment abnormalities which comprised 7 glaucoma suspect and 4 retinopathies. One of oculo-visual disorders were refractive errors which was experienced by 10 fish smokers. Anterior segment ocular disorders (28 problems) were present which included 16 cataracts, 6 pterygium, 3 allergic conjunctivitis and 3 conjunctivitis. Proper visual functioning among fish smokers is important for task performance.

In the present study, the prevalence of visual impairment was 11.7%. Comparing the results in this work to other occupational exposures, Abu et al. (2016) had (2.1%) prevalence of visual impairment (2.1%) among mechanics in Central Region of Ghana.

Abu et al. (2015) in epidemiology of ocular disorders and visual impairment among school pupils in the Cape Coast Metropolis, Ghana could also be compared to this study where the visual impairment was 1.1%.

The findings in this study is also comparable to Ohumwangho et al. (2010) where occupational eye injury among sawmill workers in Nigeria were assed (4.3%).

Budenz et al. (2012) on the topic blindness and visual impairment in an urban West African population: The Tema Eye Survey corroborates the present study with 3.7 % visual impairment among general population aged at least 40 years.

One research done among cocoa farmers by Boadi- Kusi on Ocular Health Assessment of Cocoa Farmers in a Rural Community in Ghana also showed visual impairment of 1.1%.

Ovenseri-Ogbomo et al. (2012) also concluded (Oculo-Visual Findings among Industrial Mine Workers at Goldfields Ghana Limited, Tarkwa) that 3% visual impairment was found among industrial mine workers.

Table 15: Eye Diseases that Causes Blindness

Disease	Frequency	Percentage
Cataract	16	28.6
Infective Conjunctivitis	3	5.4
Allergic Conjunctivitis	3	5.4
Dry Eye Syndrome	5	8.9
glaucoma Suspect	7	12.5
Pterygium	6	10.7
pinguecula	2	3.6
Retinopathies	4	7.1
Refractive error	10	17.9
Total	56	100.0

Source: Field work (2018-2019)

Table 16: Self- Reported Eye Discomfort

Symptoms	Frequency	%
blurred vision	17	28.3
Itching	13	21.7
Pain	10	16.7
Tearing	4	6.7
foreign body sensation	3	5.0
photophobia	3	5.0
Discharge	3	5.0
burning sensation	2	3.3
headaches	2	3.3
Glare	1	1.7
intermittent diplopia	1	1.7
blepharospasm	1	1.7
	60	100.0

Source: Field work (2018-2019)

Blurred vision was the most eye discomfort experienced by fish smokers (28.3%). A considerable number of people also complained of itching (21.7%) and pain (16.7%) in their eyes. Fish smokers are exposed to a lot of smoke as they perform their activities.

Result for eye disease symptoms in this could be corroborated with Kyei et al. (2016) where eye disease symptoms were also found among fish smokers.

From Table 17 below, the mean temperature ranges from 23.09 ±0.49 to 46.29 ±6.29 °C. In addition to temperature, mean humidity ranged from 0.1± 0.00 to 81.59 ±0.69 %.

Mean $PM_{2.5}$ ranged 0.16± 0.04 to 630.37±549.33 µg/m^3. Mean CO ranged from 2.37±2.84 to 36.43±5.27 ppm. Also, mean ozone level ranged from 2.55±0.30 to 74.69±2.12 ppb.

Table 17: Means of Parameters for Air Pollution Measured in Smokehouse

Smoke house	Temperature(°C)	Humidity (%)	$PM_{2.5}$(µg/m^3)	CO(ppm)	ozone (ppb)
1	29.76±2.41	51±6.65	93.70±399.09	6.99±4.95	9.16±1.97
2	32.88±3.11	41.50±7.23	119.53±662.38	4.68±10.09	6.76±1.15
3	46.29±6.29	11.18±9.04	31.29±170.49	16.00±6.82	16.33±8.00
4	36.68±5.27	29.16±10.88	89.64±315.89	9.72±6.18	21.28±6.12
5	23.09±0.49	71.37±.95	26.43±14.91	28.11±5.42	3.39±0.49
6	32.65±2.15	57.16±3.22	204.61±203.75	36.43±5.27	3.13±0.57
7	34.85±3.97	51.75±7.86	246.35±297.38	27.96±16.55	2.7±0.65
8	29.78±2.88	64.50±6.85	137.30±227.56	13.79±8.12	3.25±0.51
9	36.93±6.07	46.40±11.70	410.36±314.72	15.24±11.75	2.60±0.87
10	31.14±2.36	64.83±5.00	0.17±0.1	27.31±12.04	11.92±3.75
11	29.22±1.36	68.67±3.86	0.16±0.04	14.37±2.06	58.77±2.35
12	26.41±0.11	81.59±0.69	0.16±0.04	8.27±1.09	74.69±2.12
13	34.61±2.38	67.31±5.67	0.16±0.04	36.23±8.07	33.93±6.16
14	40.14±2.74	50.65±7.38	0.16±0.04	36.19±6.00	36.43±4.71
15	36.97±3.98	53.56±7.55	0.16±0.04	25.83±7.83	46.03±8.20
16	36.89±0.36	49.98±2.68	0.16±0.04	26.41±7.17	44.17±5.35
17	31.0±1.82	71.34±5.10	0.17±00	12.32±4.75	59.30±6.40

Table 17 continued

18	40.21±2.47	48.82±6.17	0.16±0.4	17.88±5.94	51.08±9.21
19	29.78±2.77	73.87±11.57	251.68±358.82	17.54±8.18	10.60±1.26
20	32.35±3.16	56.65±1064	410.61±585.76	15.74±1378	8.86±0.70
21	31.12±1.74	66.39±8.48	31.62±93.25	2.63±3.99	9.49±0.69
22	30.89±2.31	70.49±0.63	30.24±33.06	4.17±1.99	8.89±0.45
23	31.56±0.15	62.73±8.61	326.33±36.13	4.16±4.06	10.74±1.22
24	32.62±4.89	57.53±19.80	145.70±280.50	5.16±7.85	11.66±2.17
25	33.67±4.06	1.00±00	283.21±281.69	20.70±12.61	18.16±4.70
26	37.52±5.30	1.00±01	134.99±222.54	17.40±16.78	13.52±3.05
27	30.54±2.51	1.00±00	89.67±150.09	2.37±2.84	23.18±6.60
28	34.60±4.68	1.00±00	174.03±207.18	15.04±10.06	19.40±6.47
29	36.61±0.86	.1±00	19.89±14.50	16.53±12.34	15.04±3.37
30	33.73±3.32	56.73±6.74	630.37±549.33	6.96±5.81	2.55±0.30
31	35.97±3.81	51.03±8.33	549.33±576.89	35.29±7.09	4.27±.78
32	34.27±5.57	54.02±12.11	502.89±572.34	25.88±7.13	5.37±1.48
33	32.55±2.85	58.47±6.82	234.41±520.17	17.41±7.05	8.26±1.38

Source: Field work (2018-2019)

From Table 18 mean temperature before working hours was 31.35± 4.28 °C which was lower than the mean temperature during working hours. This shows that during working hours the smokehouses became warm, that is 47.07±28.18 °C and 39.65±4.99 °C respectively.

The mean humidity recorded before working hours was higher than during working hours. Also there was an increase in the mean $PM_{2.5}$ from 169. 23± 437.16 to 265.92±392.01. Level of CO before working hours also increased from 11.89±12 to 19.70±13.46. Again mean ozone level increased from 11.76±7.87 before working hours to 13.31±10.88.

Table 18: Smoke Particles Measured in the Smokehouses

	Before working hours Mean	Working hours Mean	After working hours Mean (n=33)
Temperature (°C)	31.35±4.28	35.15±5.08	33.84±6.11
Humidity(%)	47.07±28.18	39.65±24.99	42.12±26.15
PM 2.5(µg/m^3)	169.23±437.16	265.92±392.01	183.15±380.76
CO(ppm)	11.89±12	19.70±13.46	14.8±13.65
ozone(ppm)	11.76±7.87	13.31±10.88	10.21±7.06

Source: Field work (2018-2019)

From Table 19, the total mean temperature inside the smokehouses was 33.63± 5.45 °C, relative humidity had mean total of 42.51± 26.49%. There was total mean of 212.30±404.93 µg/m^3 for $PM_{2.5}$.

Carbon monoxide and ozone had mean concentration of 15.85± 13.49 ppm and 11.44± 8.96 ppb respectively. Most of the smokehouses had high temperatures.

High temperatures coupled with long term exposure to heat could predispose the fish smokers to health effects such as heat stress, heat stroke morbidity and mortality.

Total mean temperature observed in this work could be compared to Obeng (2018) which recorded total mean of 31.85°C.

Total mean relative humidity in this study was lower than the standard set by ASHRAE 1989, with lower limit of 60% and upper limit of 80%.

Most smokehouses had relative humidity within the standard set by ASHRAE (1989A). Appropriate level of humidity is needed in the smokehouses in order to achieve conditions that are conducive for human health and comfort.

Also, from Table 19, the total mean for $PM_{2.5}$ (212.30 µg/m^3) was higher than WHO standard for 24 hours of 25µg/m^3. Total mean for $PM_{2.5}$ was also higher than the Ghana EPA standard of 35µg/m^3.

Most of the smokehouses had levels of $PM_{2.5}$ higher than WHO and Ghana EPA standards. The levels of $PM_{2.5}$ in the smokehouses suggests that fish smokers could be exposed to health impacts such as respiratory tract infection, asthma and chronic obstructive pulmonary disease.

Concerning CO, total mean concentration was 15.85 ppm which was higher than WHO 24-hour of 7mg /m^3. CO can combine with haemoglobin to form carboxy -haemoglobin in humans which could be dangerous to health.

In their assessment of diseases linked to household air pollution owing to the use of biomass fuels, Kim et al. (2011) determined that incomplete biomass combustion causes significant levels of indoor pollutants such as carbon monoxide.

This could have negative consequences for humans, including respiratory infections, chronic obstructive pulmonary disease and asthma. The situation could also result in some cardiovascular diseases such high blood pressure and stroke.

Concentration level of carbon monoxide in this study is corroborated by Fatmi et. al. (2020) where substantially higher average of CO and particularly PM $_{2.5}$ were found in the kitchen of biomass users.

In addition, Bartington, Bakolis, Devakumar, Kurmi, Gulliver, Chaube, Manandhar, Saville, Costello, Osrin, Hansell and Ayres (2016) indicated that domestic CO and $PM_{2.5}$ levels in households that use biomass fuel in an area of Nepal frequently exceed WHO Air Quality Standards. The levels of CO and $PM_{2.5}$ are likely to contribute to increased morbidity, mortality and adverse birth outcomes.

Haven et al., 2018 reported a high CO concentration and concluded that children in settings like those in Malawi are likely to be exposed to potentially harmful levels of CO as a result of biomass usage.

The result in this work could also be compared to North et al., 2019 in a rural setting in Uganda, where personal CO exposure frequently exceeded international thresholds, correlated with biomass exposure and also associated with respiratory symptom among women.

In a study by Kalpana et al. (2015) results from baseline phase of the study showed mean 24-hour kitchen area concentrations for PM $_{2.5}$ that are comparable to this study. The concentration of CO in Kalpana et al. (2015) in base line phase for mean 24-hour kitchen area is rather lower than the CO concentration in smokehouses in this study.

According to Fullerton et al., 2009, biomass fuel consumption and indoor air pollution in Malawian houses were likewise high.

Table 19: Total Mean of Indoor Air Pollution Parameters

	Mean	Std. Dev.
Temperature (°C)	33.63	5.45
Humidity (%)	42.51	26.49
$PM_{2.5}$	212.30	404.93
CO (ppm)	15.85	13.49
Ozone (ppm)	11.44	8.96

Source: Field work (2018-2019)

From Figure 13, most of the smokehouses had CO concentration levels higher than the WHO recommended standard for 24-hour CO.

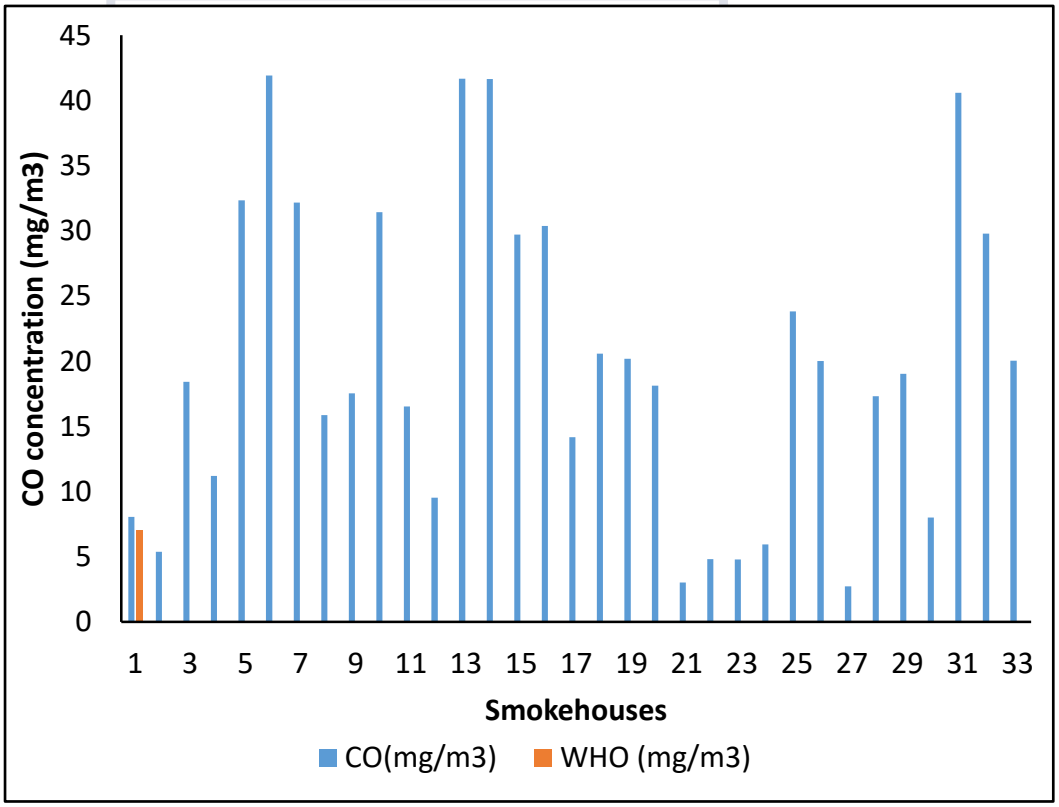


Figure 13: Comparing CO Concentration to WHO 24-Hour Concentration

NB: CO concentration in ppm was multiplied by molar mass 28 g/mol and divided by a factor, 24.45 to convert to (mg/m^3) to conform to WHO value.

Table 20: Correlation among Parameter of Indoor Air Pollution

	Temperature	Humidity	$PM_{2.5}$	Carbon monoxide	Ozone
Temperature	1	-.498**	.203**	.438**	-.087**
Humidity	-.498**	1	-0.004	-.020**	-.357**
$PM_{2.5}$	.203**	-0.004	1	.338**	-.201**
Carbon monoxide	.438**	-.020**	.338**	1	-.272**
Ozone	-.087**	-.357**	-.201**	-.272**	1

** Correlation is significant at the 0.01 level (2-tailed).
Source: Data analysis

There was significant correlation between temperature and humidity. Whiles temperatures increased, there was a decrease in relative humidity. There was also significant correlation among temperature and $PM_{2.5}$, CO, O_3. There was increase in both $PM_{2.5}$, CO as temperature increased.

This also show that $PM_{2.5}$ and CO could come from the same source that is the smoke particles as biomass fuel was being burnt during fish smoking. There was a decrease in O_3 concentration as temperature increased.

There was no significant correlation between humidity and $PM_{2.5}$ but there was significant correlation between CO as well as O_3. Again there was correlation between $PM_{2.5}$ and CO. There was correlation between $PM_{2.5}$ and O_3. There was correlation between CO and O_3.

Assessment of Health Risk

Table 21 below shows the estimated daily doses of pyrene among adult women. From the measured urinary 1-OHP levels, the estimated mean daily intake doses were 252706.96 µg/kg for samples from the Central region.

Also, the estimated mean daily intake doses were 596873.55µg/kg for samples from the Volta region and 271867.89 µg/kg for samples from the

Western region. The results represent the doses for the women that participated in the studied areas.

Table 21: Estimated daily intake dose of pyrene (Dose) and hazard quotient (HQ)

Regions	Central	Volta	Western
Concentrations (µg/L)	548.16	1294.70	589.720
Dose (µg/Kg/day)	252706.96	596873.55	271867.89
Hazard Quotient	8.42E+00	1.99E+01	9.06E+00

Source: Data analysis

Considering the HQ of the samples, the mean estimated HQ values of samples from Central, Volta and Western regions were 8.42E+00, 1.99E+01 and 9.06E+00 respectively. When HQ is less than one, it is unlikely that a negative health effect will occur. When HQ is greater than 1, however, a harmful health effect is likely (USEPA, 1993).

According to the findings of this work, women exposed to PAHs in the studied areas are likely to suffer serious health repercussions. High health risk values were noted from the study and will pose serious adverse health problems for the women who live in the study areas, since all the mean HQs calculated were far more than 1.

Moreover, the highest HQ value (approximately mean HQ=20) was obtained for samples from the Volta region (Table 21). Although high HQs values were obtained, that is greater than 1.0. This indicates that some harmful consequences are likely, but additional evidence may be needed to confirm the true impact of PAHs exposure on women's health.

This is because a wide range of nutritional, environmental, socioeconomic and demographic factors influence human health (Huang, Wang, Morello-Frosch, Lam, Sirota, Padula and Woodruff, 2018).

Judging the levels of HQ values found in the study, it will be recommendable to implement health programs for the people in the study areas. This would help reduce the impact of PAHs thereby reducing the health risk in the women who engage in the smoking activities.

Previous research has found that lower educational levels are connected to a higher risk of mortality as a result of PAH exposure (Romero-lankao, Qin and Borbor-cordova, 2013).

Assessment of the study areas with regards to this study show that all women participating in our research had similar ethnic and socioeconomic backgrounds (such as poverty, low income, education level, lifestyle etc). It was also observed from the study areas that all the women basically use wood combustion as the main energy source for the smoking of the fishes.

Therefore, the high HQs values obtained is due to ingestion, inhalation or dermal contact of PAHs in wood smoke which massive use can be related to low socioeconomic status of the women (Pruneda-Álvarez, Pérez-Vázquez, Ruíz-Vera, Ochoa-Martínez, Orta-García, Jiménez-Avalos and Pérez-Maldonado, 2016). The difference in HQ values from the regions may be due to the type of wood used in the combustion process.

The evaluation performed in this work assessed urinary 1-OHP (pyrene metabolite) levels. However, simultaneous exposure to mixtures of PAHs was possible due to the fact that a complex mixture of different PAHs congeners is generated in the combustion process.

Exposure monitoring of persons through early toxic effects checks and the measurement of biomarkers of exposure will help in early prevention in order to limit the adverse health impacts of PAHs on people living in these

areas. This would help to better understand the dangers involved in PAHs exposure to people living in the study areas (Pruneda-Álvarez et al., 2016).

Personal PM $_{2.5}$ and Personal CO among Fish smokers in Central and Volta Regions

Fish smoking is a source of livelihood for most people in Ghana. Biomass fuel (firewood) is mostly used by fish smokers to smoke fish. In addition to biomass usage for fish smoking, fish smokers use biomass fuel (firewood, charcoal) and LPG to cook their food. Some of them cook in the house, in a separate building or outdoors.

Real time personal particulate matter with an aerodynamic diameter ≤2.5 μm ($PM_{2.5}$) and personal carbon monoxide (CO) samples were measured among fish smokers for 48 hours.

Table 22 provides general characterization of the time of exposure during fish smoking, age and cooking location (where fish smokers cook their meals) of the participants according to the type of fuel they use. A total of 120 participants were recruited. Out of this 50, 69 and 1 participant used wood, charcoal and LPG respectively.

The average time spent on working with wood was 7.6±2.6 hours, charcoal was 6.7±3.1 hours and LPG was 6.0±0.0 hours. Majority of the participants were between the ages of 31-40 years, (48.3%). Ages ≤ 30 years were 12.5% being the least age group to participate.

Participants in the ages below 30 mostly preferred charcoal 11(15.9) to wood 3(6.0), those between 31-40 preferred wood 30(60.0) to charcoal 28(40.6) whiles those above 40 years used more charcoal 30(43.5) to wood 17(34.0). Most of the cooking was done in separate buildings 70(58.3).

The choice of fuel used for cooking by fish smokers depends upon the availability of fuel, financial status, knowledge about the health implications of the gases that are release during the biomass usage (if any).

Using firewood and charcoal to cook in addition to fish smoking could also add to inhalation of smoke particles especially cooking in the house where there is poor ventilation.

Even those who cook in a separate building could also be affected since they spend time there, sometimes with their children. Only one fish smoker among the participants used LPG to cook.

This should motivate focus on policies that specifically address biomass use and create incentives and conditions for transition to cleaner fuels such as LPG. This is because using cleaners for cooking would reduce the amount of smoke inhaled by the fish smokers.

Table 22: General Characterization of the Time of Exposure, Age and Cooking Location of the Participants according to the Type of Fuel They Use

Variable	Wood (n=50)	Charcoal (n=69)	LPG (n=1)	Total (n=120)
Time in Hours (n ± SD)	7.6±2.6	6.7±3.1	6.0±0.0	7.2±2.9
Age (n ± SD)	38.9±6.1	39.1±7.0	28.0±0.0	34.0±6.7
Age				
≤30 years	3(6.0)	11(15.9)	1(100.0)	15(12.5)
31-40 years	30(60.0)	28(40.6)	0(0.0)	58(48.3)
≥41 years	17(34.0)	30(43.5)	0(0.0)	47(39.2)
Cooking Location				
In a Separate Building	39(78.0)	30(43.5)	1(100.0)	70(58.3)
Outdoor	8(16.0)	22(31.9)	0(0.0)	30(25.0)
In the House	3(6.0)	17(24.0)	0(0.0)	20(16.7)

n±SD = mean ± the standard deviation
Source: Field work (2018-2019)

Table 23 shows the characterization of the time of exposure, age and cooking location of the participants in the Volta Region according to the type of fuel they use.

In the Volta Region, 29, 31, and 0 participant used wood, charcoal and LPG respectively. A total number of 60 participants were recruited. Most of the participants were between the ages of 31-40 years 38(63.3). Those ≤ 30 years used more charcoal 6(19.4) than wood 1(3.4), participants between 31-40 years used more wood 22(75.9) than charcoal 16(51.6). Whiles those above 40 years also used more charcoal 9(29.0) than wood 6(20.7).

The Kpando and South Dayi District both recruited 30 participants. In order of descending 36, 18 and 6 participants cooked in separate buildings, outdoor and in the house respectively. Fuel usage and location of cooking in addition to fish smoking in the Volta Region by fish smokers shows that some of them could be affected by smoke particles especially using biomass fuel (firewood and charcoal) in the house.

Fuel usage by age group in Volta Region is similar to the general trend in Table 13 (Types and choice of firewood used by fish smokers), most people used charcoal to cook followed by firewood.

Looking at the average time spent by fish smokers to smoke fish, they could be exposed to particulates or gases that could affect their health and their dependents. This could lead to chronic respiratory disease such chronic asthma among fish smokers and young children.

Table 23: Characterization of the Time of Exposure, Age and Cooking Location of the Participants in the Volta Region according to the Type of Fuel They Use

Variable	Wood (n=29)	Charcoal (n=31)	LPG (n=0)	Total (n=60)
Time in Hours (n ± SD)	7.4±2.8	6.6±2.9	0±0.0	7.0±2.8
Age (n ± SD)	37.7±4.7	37.4±6.3	0±0.0	37.6±5.5
Age				
≤30 years	1(3.4)	6(19.4)	0(0.0)	7(11.7)
31-40 years	22(75.9)	16(51.6)	0(0.0)	38(63.3)
≥41 years	6(20.7)	9(29.0)	0(0.0)	15(25.0)
District				
Kpando	16(55.2)	14(45.2)	0(0.0)	30(50.0)
South Dayi	13(44.8)	17(54.8)	0(0.0)	30(50.0)
Cooking Location				
In a Separate Building	23(79.3)	13(41.9)	0(0.0)	36(60.0)
Outdoor	6(20.7)	12(38.7)	0(0.0)	18(30.0)
In the House	0(0.0)	6(19.4)	0(0.0)	6(10.0)

n±SD = mean ± the standard deviation
Source: Field work (2018-2019)

Table 24 is the characterization of the time of exposure, age and cooking location of the participants in the Central Region according to the type of fuel they use.

A total of 60 participants were recruited in the Central region from the Ekumfi and Mfansteman districts, 30 from each district. Most of the participants in the two districts used charcoal as a source of fuel 38, followed by the usage of wood 21 and then to LPG 1.

The average time of exposure in the Central Region was 7.2±3.0 hours. Age-wise, the participants were mostly above 40 years 32(52.3) and very few were ≤30 years 8(13.3).

In terms of cooking location, most of the participants that used wood 16(76.2) did their cooking in separate buildings whiles 2(9.3) did it outdoor

and 3(14.3) did it in the house. The choice of fuel for cooking by fish smokers in the Central Region is similar to that of the participants in the Volta Region.

Table 24: Characterization of the Time of Exposure, Age and Cooking Location of the Participants in the Central Region according to the Type of Fuel They Use

Variable	Wood (n=21)	Charcoal (n=38)	LPG (n=1)	Total (n=60)
Time in Hours (n ± SD)	7.8±2.4	6.9±3.4	6.0±0.0	7.2±3.0
Age (n ± SD)	405±7.5	40.6±7.3	28.0±0.0	40.4±7.4
Age				
≤30 years	2(9.5)	5(13.2)	1(100.0)	8(13.3)
31-40 years	8(38.1)	12(31.6)	0(0.0)	20(33.3)
≥41 years	11(52.4)	21(55.3)	0(0.0)	32(53.3)
District				
Ekumfi	17(81.0)	12(31.6)	1(100.0)	30(50.0)
Mfantseman	4(19.0)	26(68.4)	0(0.0)	30(50.0)
Cooking Location				
In a Separate Building	16(76.2)	17(44.7)	1(100.0)	34(56.7)
Outdoor	2(9.5)	10(26.3)	0(0.0)	12(20.0)
In the House	3(14.3)	11(28.9)	0(0.0)	14(23.3)

n±SD = mean ± the standard deviation
Source: Field work (2018-2019)

Figure 14 shows the correlations between CO, $PM_{2.5}$ present and the general time of exposure. None of the correlations above were statistically significant.

However, it can be noted that there was a negative correlation between the time of exposure and carbon monoxide (r= -0.2, P = 0.008), a positive correlation between the time of exposure and the $PM_{2.5}$ (r= 0.05, P= 0.539) and a negative correlation between carbon monoxide and the particulate matter $_{2.5}$ (r= -0.08, P= 0.148).

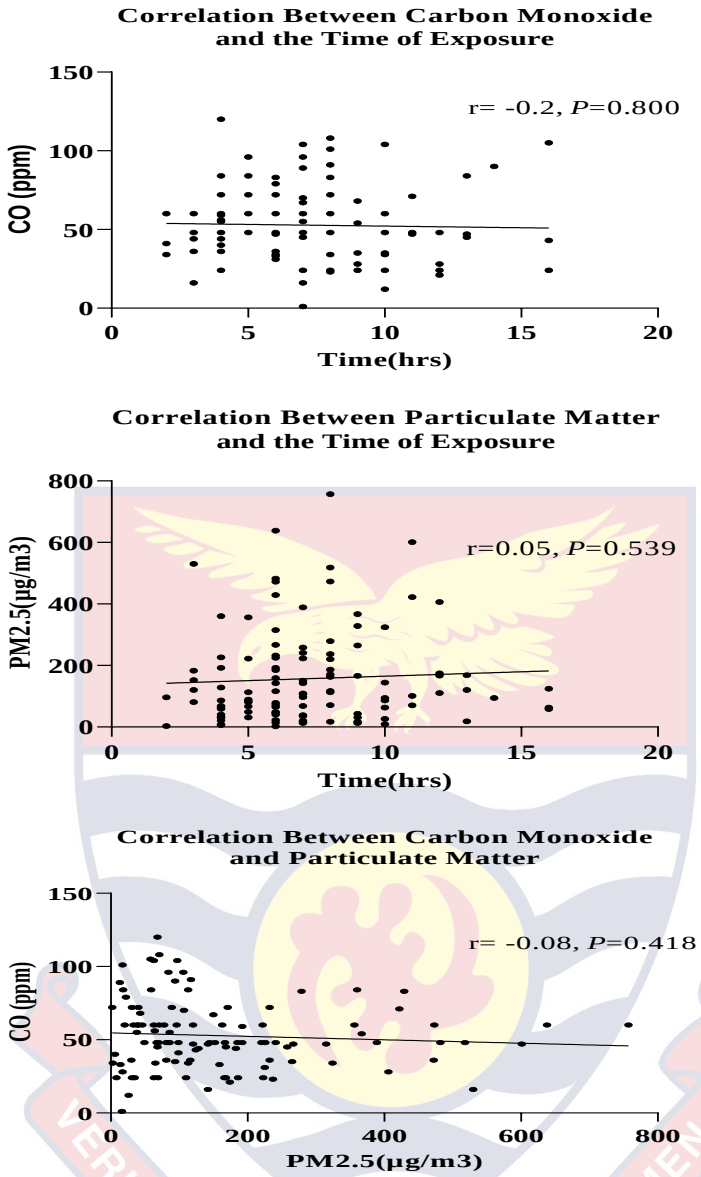


Figure 14: Correlations between Carbon Monoxide, Particulate Matter $_{2.5}$ Present and the General Time of Exposure
r= correlation coefficient, *P*= P-value (<0.05 implies it was statistically significant)

In the Central Region, Figure 15 demonstrates connections between carbon monoxide, particulate matter $_{2.5}$ present and exposure time.

In the Central Region, there was a positive correlation (r=0.02) between time of exposure and particulate matter $_{2.5}$, a negative correlation (r= -0.20) between time and carbon monoxide, and a positive correlation (r=0.02)

between carbon monoxide and particulate matter $_{2.5}$. Yet none of the correlations were statistically significant.

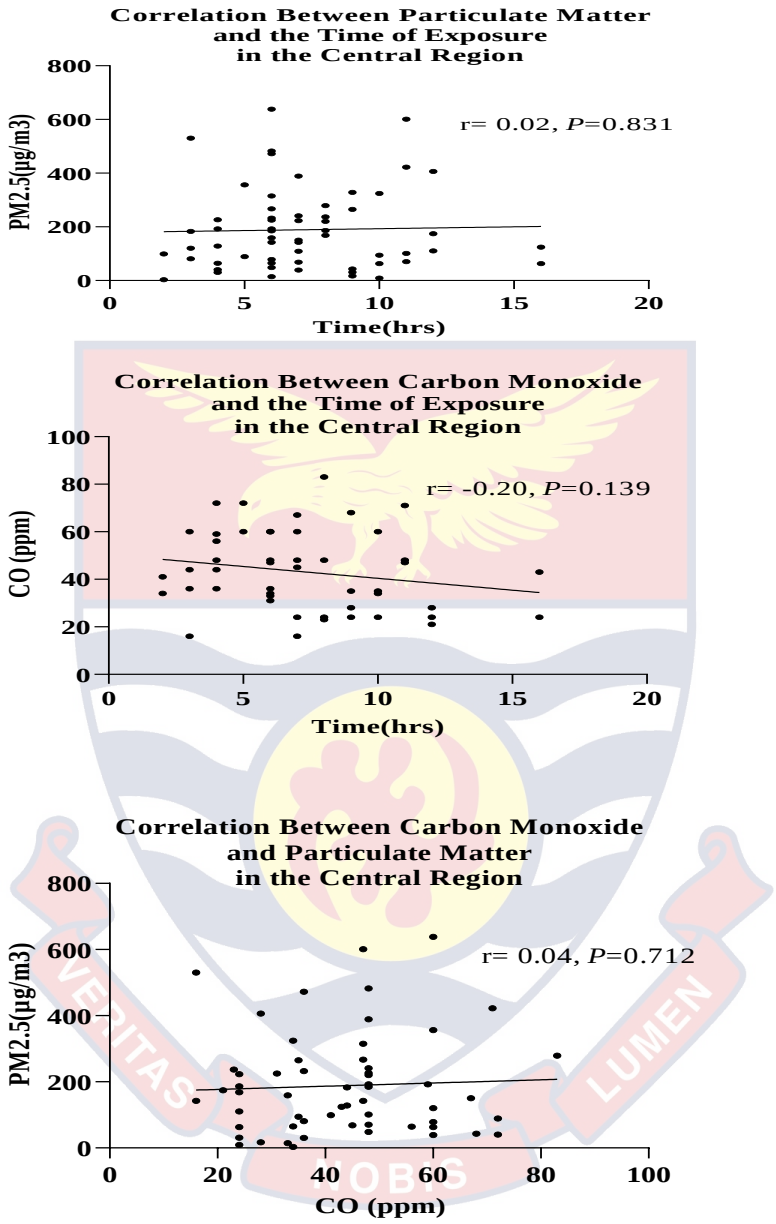


Figure 15: Correlations between Carbon Monoxide, Particulate Matter $_{2.5}$ Present and the Time of Exposure in the Central Region
r= correlation coefficient, *P*= P-value (<0.05 implies it was statistically significant)

Figure 16 provides correlations between carbon monoxide, particulate matter $_{2.5}$ present and the time of exposure in the Volta Region.

Correlations between particulate matter $_{2.5}$, time of exposure and carbon monoxide in the Volta Region were all statistically insignificant. Notably, positive correlations were recorded between time of exposure and particulate matter $_{2.5}$ and time of exposure and carbon monoxide, 0.08 and 0.10 correlations respectively.

A negative correlation of -0.01 was recorded between the particulate matter $_{2.5}$ and carbon monoxide.

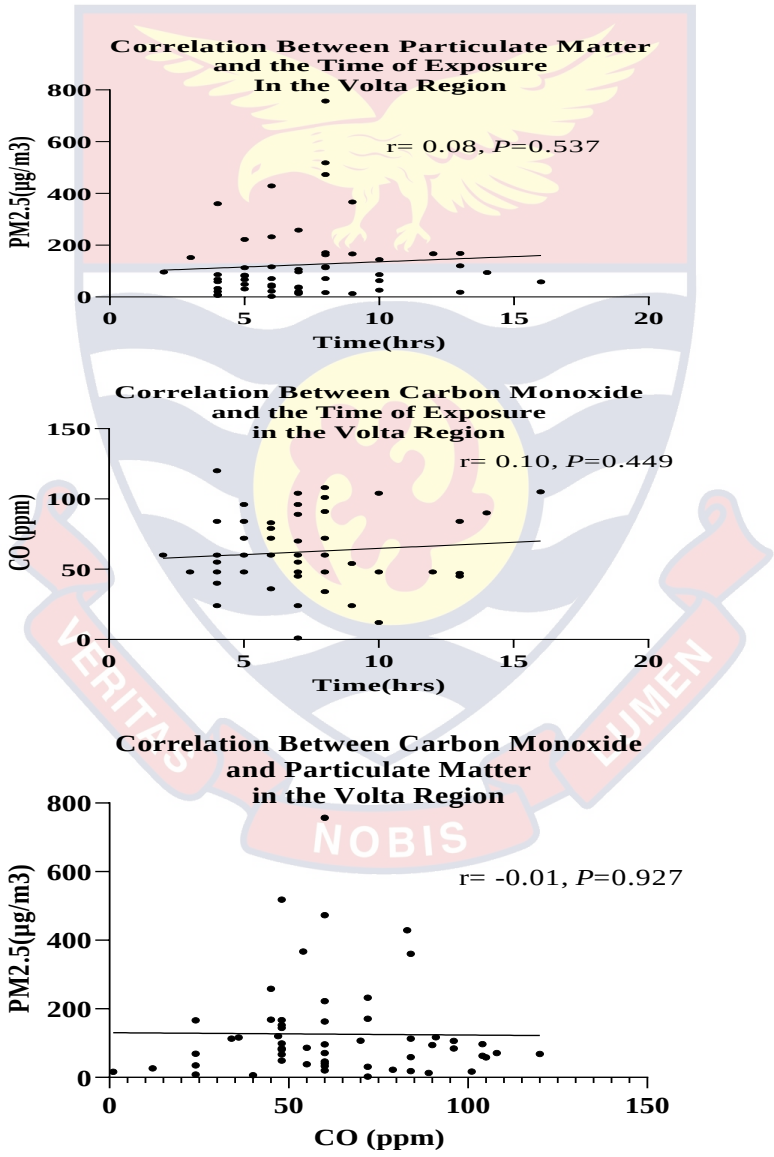


Figure 16: Correlations between Carbon Monoxide, Particulate Matter $_{2.5}$ Present and the Time of Exposure in the Volta Region
r= correlation coefficient, *P*= P-value (<0.05 implies it was statistically significant)

From Table 25, the average PM $_{2.5}$ concentration recorded when using wood was 148±189.2 µg/m^3, charcoal was 161.8±151.5 µg/m^3 and LPG was 267.0 µg/m^3 with no deviation.

Charcoal usage had the highest record of particulate matter $_{2.5}$ whiles wood hard the record, 757.0 µg/m^3 and 2.0 µg/m^3 respectively. Participants who cooked in a separate building recorded both the lowest and the highest level of particulate matter $_{2.5}$, 2.0 µg/m^3 and 757.0 µg/m^3 correspondingly. An average of 102.6±90.0 µg/m^3 PM $_{2.5}$ was recorded in outdoor cooking.

Also from Table 25, on personal PM $_{2.5}$, mean concentration for this study is higher than WHO air quality standard of 25 ug/m^3.

Mean concentration personal PM $_{2.5}$ in this study was higher than that observed by Raphael et al, 2014 though their study was conducted among students. Similar to observation in this study, Raphael et al, 2014, also observed that biomass fuel usage was one of the determinants of air pollution.

Furthermore, mean level of personal PM $_{2.5}$ in this study was higher Nti et al, where person particulate matter was assessed among e-waste workers.

The lowest and highest carbon monoxide concentration were both recorded when using wood, 1.0 ppm and 120.0 ppm respectively. Averagely, 47.0 ppm carbon monoxide were recorded when LPG was used. In cooking location, those outdoor recorded the least carbon monoxide, 1.0 ppm and those that worked in a separate building recorded the highest 120.0 ppm.

Personal CO in this study were higher than WHO standard (8.7ppm for 8-hours) and USEPA limit (9ppm for 8- hours) for most of the participants though personal CO was monitored for 48- hours in this study.

In this study, biomass fuel usage has been shown to contribute to air pollution which could lead diverse respiratory diseases among fish smokers.

The mean concentration of personal CO in this study was higher than that found in Flintwood-Brace,2016, despite the fact that Flintwood-Brace,2016 measured personal CO for six hours.

Result for personal CO in this study was also higher than Yip, Christensen, Sircar, Naeher, Bruce, Pennise, Lozier, Pilishvili, Farrar, Stanistreet, Nyagol, Muoki, Beer, Sage and Kapil (2017) for 48-hour CO.

Table 25: Mean ± Standard Deviation, Minimum, Maximum and Standard Error of Mean of Particulate Matter $_{2.5}$ and Carbon Monoxide Recorded according to the General Fuel Type used and Cooking Location

Variable		Mean ± SD	Min	Max	Standard Error of Mean
$PM_{2.5}$ ($\mu g/m^3$)					
	Fuel				
	Wood	148.0±149.2	2.0	638.0	21.3
	Charcoal	161.8±151.5	3.0	757.0	18.3
	LPG	267.0±0.0	267.0	267.0	0.0
	Cooking Location				
	In a Separate Building	182.6±166.5	2.0	757.0	20.0
	Outdoor	102.6±90.0	8.0	482.0	16.4
	In the House	149.8±143.5	3.0	530.0	32.0
CO(ppm)					
	Fuel				
	Wood	52.6±23.7	1.0	120.0	3.4
	Charcoal	53.0±23.0	16.0	108.0	2.7
	LPG	47.0±0.0	47.0	47.0	0.0
	Cooking Location				
	In a Separate Building	54.7±22.1	21.0	120.0	2.6
	outdoor	49.4±24.1	1.0	108.0	4.5
	In the House	50.6±25.4	16.0	101.0	5.8

Mean ± SD= Mean ± Standard Deviation, Min= Minimum number, Max= Maximum Number
Source: Field work (2018-2019)

Table 26 provides information on mean ± standard deviation, minimum, maximum and standard error of mean of particulate matter $_{2.5}$ and carbon monoxide recorded according to the fuel type used and cooking location in the Central Region.

In the Central Region, the average particulate matter $_{2.5}$ recorded when using wood was 193±162.9 µg/m^3, charcoal was 184.5±138.3 µg/m^3 and LPG was 267.0 µg/m^3 with no deviation.

Wood usage had the highest record of particulate matter $_{2.5}$ whiles charcoal had the record, 638.0 µg/m^3 and 3.0 µg/m^3 respectively.

Participants who cooked in a separate building recorded the highest level of particulate matter $_{2.5}$, 638.0 µg/m^3 and those who cooked in the house recorded the least, 3.0 µg/m^3. An average of 159.7±112.8 µg/m^3 particulate matter $_{2.5}$ was recorded in outdoor cooking which the lowest among cooking location.

The lowest and highest carbon monoxide concentration were both recorded when using charcoal, 16.0 ppm and 83.0 ppm respectively. Averagely, 47.0 ppm carbon monoxide were recorded when LPG was used.

In cooking location, those outdoor and in the house, both recorded the least carbon monoxide, 16 ppm. However, the highest carbon monoxide recorded was from outdoor cooking, 83.0. In terms of cooking location, the highest CO concentration was recorded for cooking in a separate building, followed by cooking in the house. Cooking outdoor had the lowest CO concentration.

Table 26: Mean ± Standard Deviation, Minimum, Maximum and Standard Error of Mean of Particulate Matter $_{2.5}$ and Carbon Monoxide Recorded according to the Fuel Type Used and the Cooking Location in the Central Region

Variable		Mean ± SD	Min	Max	Standard Error of Mean
$PM_{2.5}$ ($\mu g/m^3$)					
	Fuel				
	Wood	193.8±162.9	9.0	638.0	39.0
	Charcoal	184.5±138.3	3.0	601.0	22.4
	LPG	267.0±0.0	267.0	267.0	0.0
	Cooking Location				
	In a Separate Building	200.7±162.9	9.0	638.0	27.9
	outdoor	159.7±112.8	63.0	482.0	32.5
	In the House	186.3±157.9	3.0	530.0	42.2
CO					
	Fuel				
	Wood	45.3±17.9	21.0	72.0	4.0
	Charcoal	41.9±14.6	16.0	83.0	2.4
	LPG	47.0±0.0	47.0	47.0	0.0
	Cooking Location				
	In a Separate Building	44.2±14.8	21.0	72.0	2.5
	outdoor	43.3±14.5	16.0	60.0	4.3
	In the House	40.4±19.20	16.0	83.0	5.3

Mean ± SD= Mean ± Standard Deviation, Min= Minimum number, Max= Maximum Number
Source: Field work (2018-2019)

In the Volta Region, as can be seen on Table 27, the least particulate matter $_{2.5}$ recorded was from wood usage, 2.0 $\mu g/m^3$ and the highest was from charcoal usage, 757.0 $\mu g/m^3$. None of the participants used LPG.

The average particulate matter $_{2.5}$ emitted when cooking in a separate building was 165.1± 170.5 $\mu g/m^3$ and the average particulate matter $_{2.5}$ for outdoor usage was 64.5±41.9 $\mu g/m^3$ in the house was 64.5±32.4 $\mu g/m^3$. The lowest and the highest particulate matter $_{2.5}$ was recorded from using in a separate building, 2.0 $\mu g/m^3$ and 757.0 $\mu g/m^3$ respectively.

Carbon monoxide recorded the least emission in wood usage, 1.0 ppm and also the highest emission of 120.0. The average carbon monoxide concentration when using charcoal was 66.1±24.3 ppm. The average carbon monoxide concentration when cooking in a separate building, outdoor and in the house are 64.6±24.0 ppm, 53.3±28.3ppm, and 72.8±23.8 ppm respectively. The highest recorded carbon monoxide was in a separate building, 120.0 and the lowest was outdoor, 1.0.

The results obtained in Volta Region is similar to that of the Central Region with slight variations. In Volta Region, mean personal $PM_{2.5}$ concentrations for participants using wood was lower than those using charcoal.

In terms of cooking location, fish smokers who cooked in a separate building had highest mean personal $PM_{2.5}$ concentrations.

For personal CO in Volta Region, participants using charcoal had higher personal CO concentrations than those who cooked with wood. For cooking location, those cooking outdoor had the lowest CO concentrations.

Table 27: Mean ± Standard Deviation, Minimum, Maximum and Standard Error of Mean of Particulate Matter $_{2.5}$ and Carbon Monoxide Recorded according to the Fuel Type used and Cooking Location in the Volta Region

Variable		Mean ± SD	Min	Max	Standard Error of Mean
$PM_{2.5}$ ($\mu g/m^3$)					
	Fuel				
	Wood	113.2±114.1	2.0	473.0	21.5
	Charcoal	134.0±164.3	13.0	757.0	29.5
	LPG				
	Cooking Location				
	In a Separate Building	165.1±170.5	2.0	757.0	28.8
	Outdoor	64.5±41.9	8.0	152.0	9.8
	In the House	64.5±32.4	17.0	106.0	13.2
CO					
	Fuel				
	Wood	57.8±26.1	1.0	120.0	4.9
	Charcoal	66.1±24.3	24.0	108.0	4.37
	LPG				
	Cooking Location				
	In a Separate Building	64.6±23.4	24.0	120.0	3.9
	Outdoor	53.3±28.3	1.0	108.0	6.9
	In the House	72.8±23.8	48.0	101.0	9.7

Mean ± SD= Mean ± Standard Deviation, Min= Minimum number, Max= Maximum Number
Source: Field work (2018-2019)

Figure 17 shows correlation between personal CO and 1, 2-Dimethylbenzene, Bis(2-chloroethyl) ether, 1,2-Dichlorobenzene, 2-Chlorophenol, 1,2-Dichlorobenzene and phenol.

There was significant correlation between personal CO (ppm) and 2-chlorophenol (r= 0.032, *P*= 0.004). None of the compounds left showed a significant association with CO (ppm).

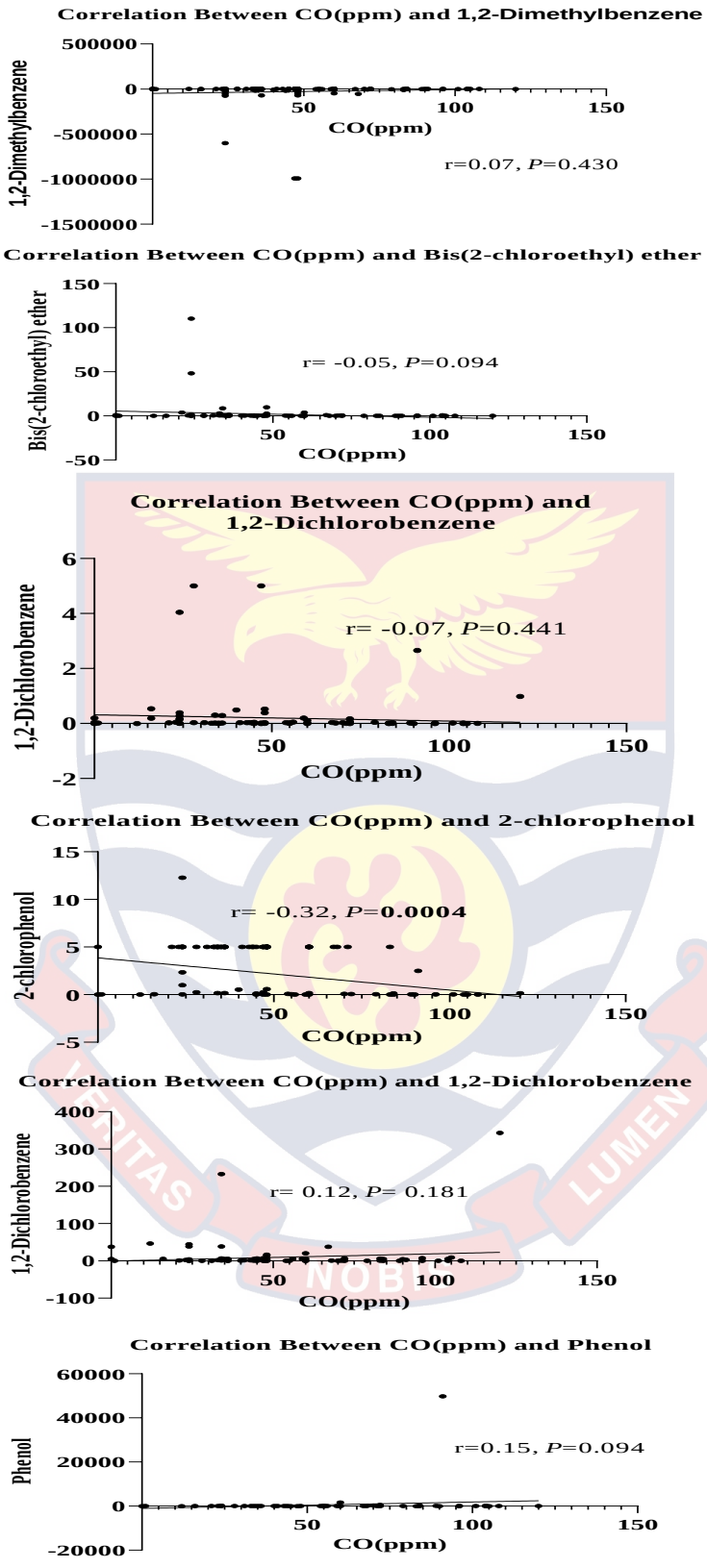
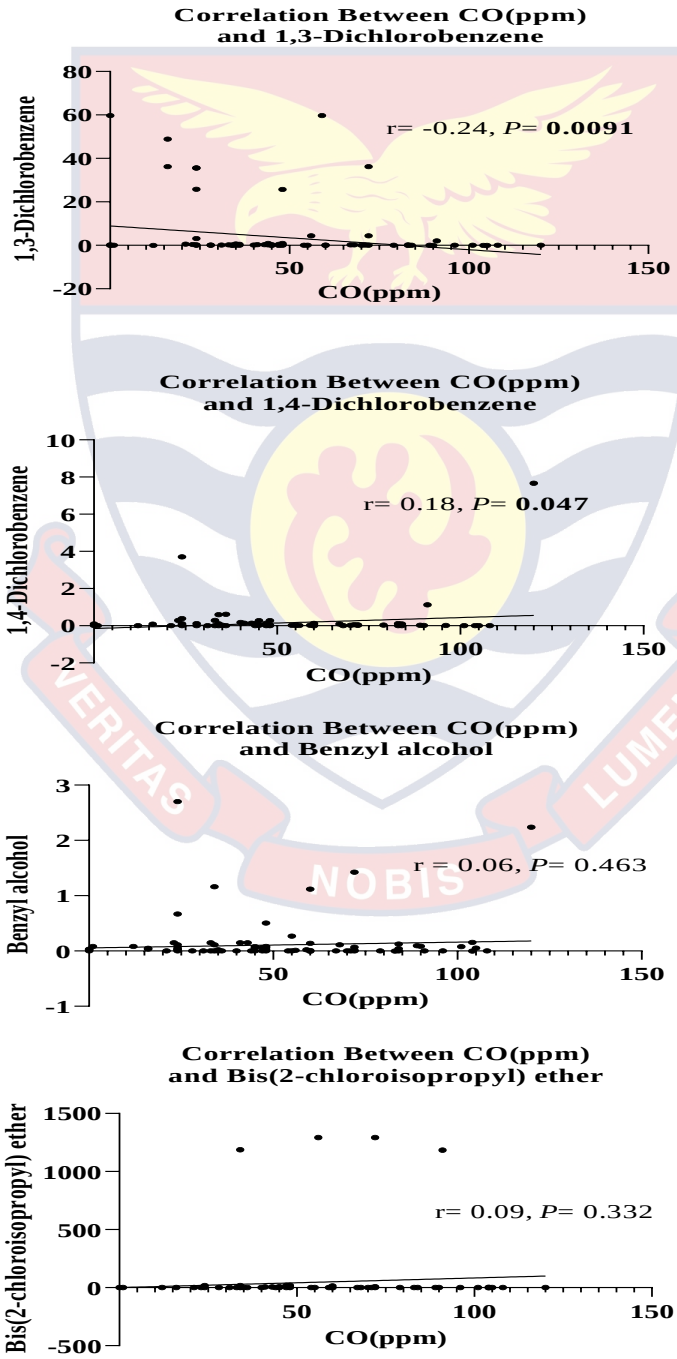


Figure 17: Correlation between personal CO (ppm) and 1, 2-Dimethylbenzene, Bis(2-chloroethyl) ether, 1,2-Dichlorobenzene, 2-Chlorophenol, 1,2-Dichlorobenzene and Phenol

r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Correlations between personal CO and 1,3-Dichlorobenzene, 1,4-Dichlorobenzene, Benzyl alcohol, Bis(2-chloroisopropyl) ether, Hexachloroethane and 2-Methylphenol are presented in Figure 18. Correlation between CO and 1,3-Dichlorobenzene (r= -0.24, P= 0.0091), and 1,4-Dichlorobenzene (r= 0.18, P= 0.047) were statistically, significant. However, the other compounds showed no significant correlation.



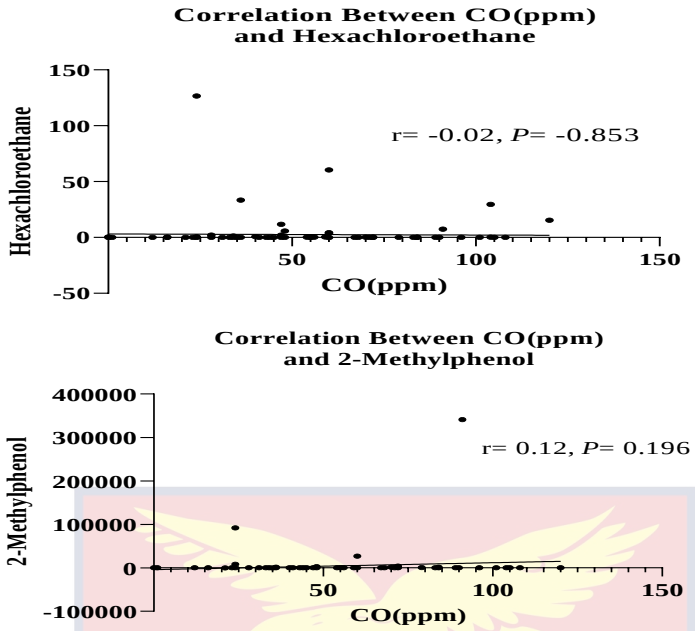
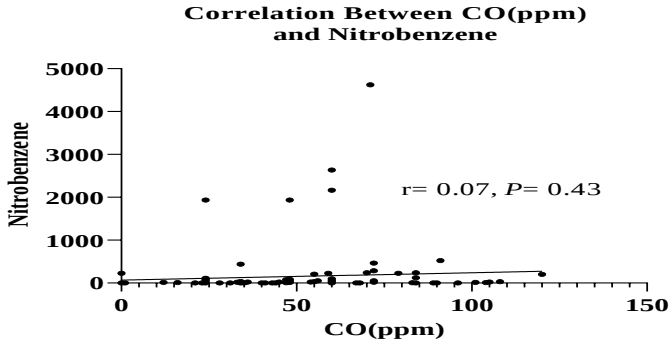
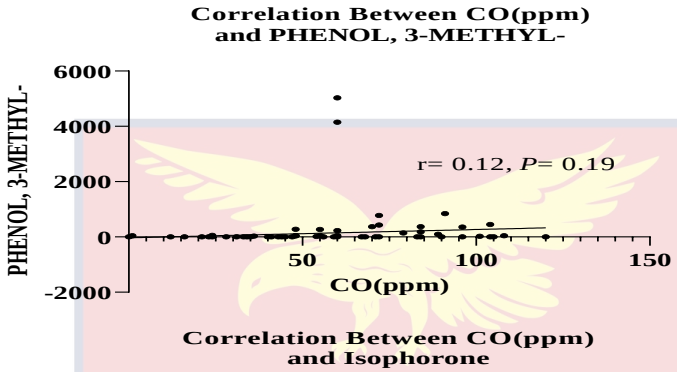


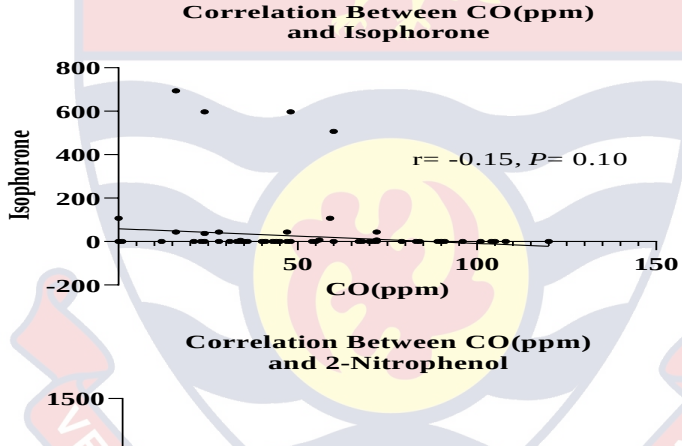
Figure 18: Correlation between Personal CO (ppm) and 1,3-Dichlorobenzene, 1,4-Dichlorobenzene, Benzyl alcohol, Bis(2-chloroisopropyl) ether, Hexachloroethane and 2-Methylphenol

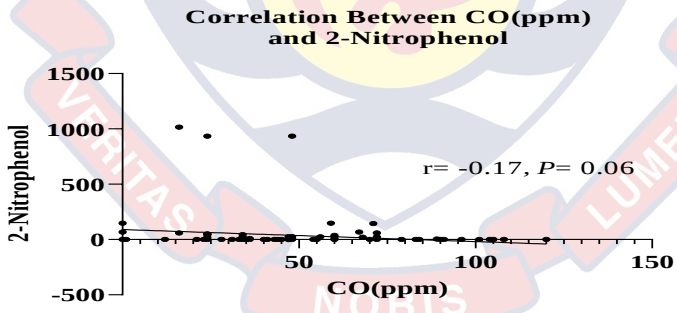
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

In Figure 19 correlation between CO and the compounds such as Nitrobenzene, 3-Methylphenol, Isophorone, 2-Nitrophenol, Bis(2-chloroethoxy) methane and 2,4-Dimethylphenol showed no statistically significant association.









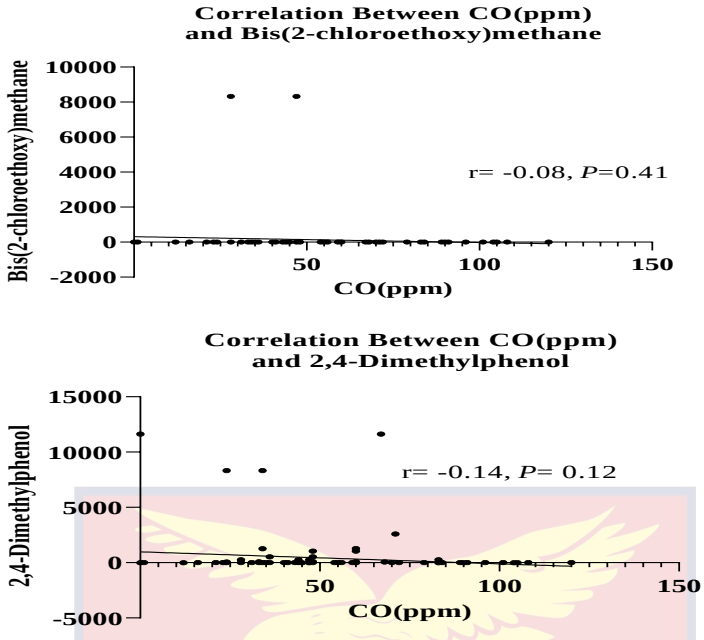
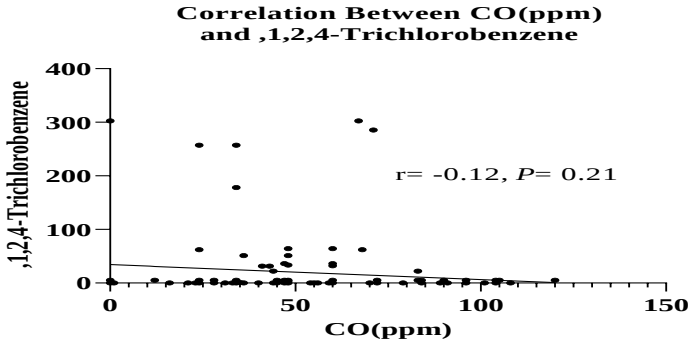
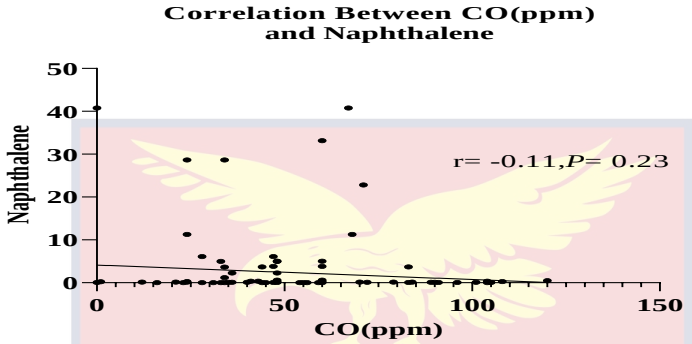


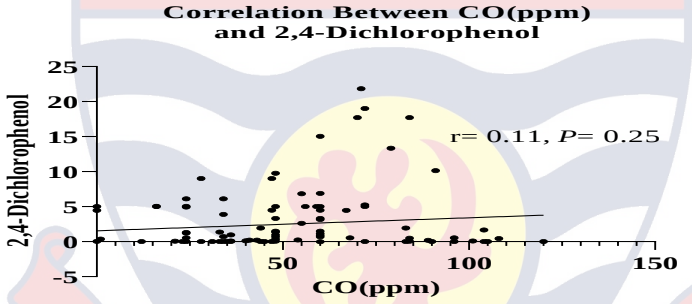
Figure 19: Correlation between CO (ppm) and Nitrobenzene, 3-Methylphenol, Isophorone, 2-Nitrophenol, Bis(2-chloroethoxy) methane and 2,4-Dimethylphenol

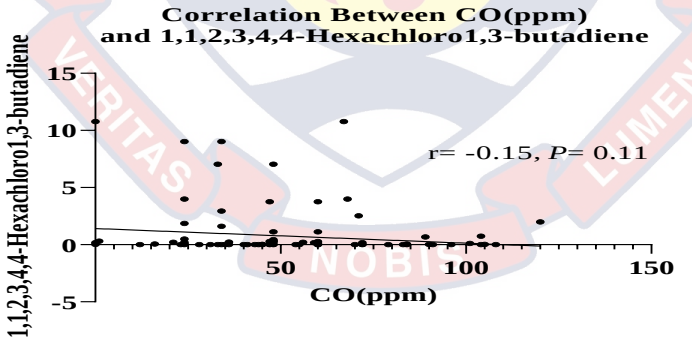
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

From Figure 20, correlation between personal CO and 1,2,4-Trichlorobenzene, Naphthalene, 2,4-Dichlorophenol, 1,1,2,3,4,4-Hexachloro1,3-butadiene, 4-Chloroaniline, and 1-Methylnaphthalene showed no statistically significant association.









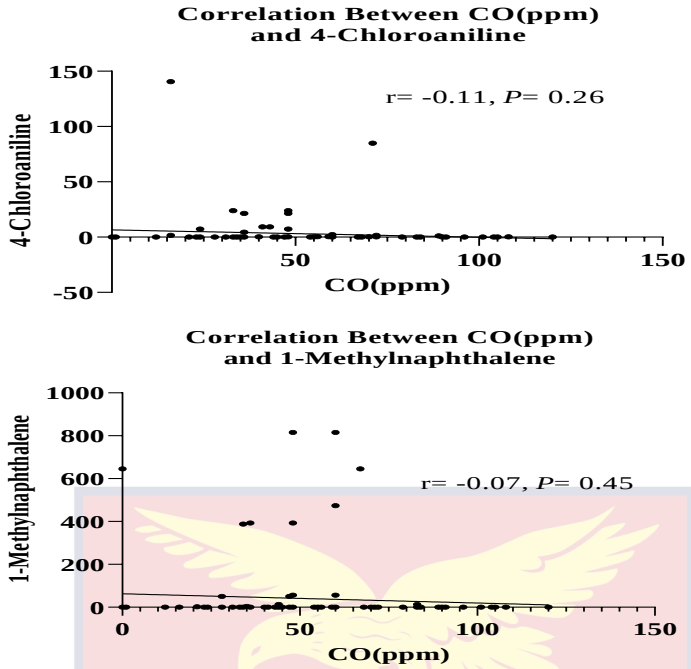
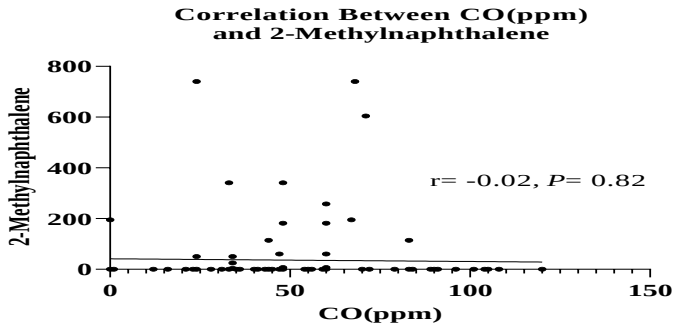
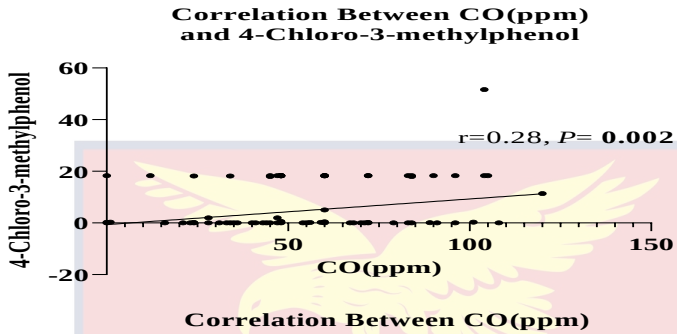


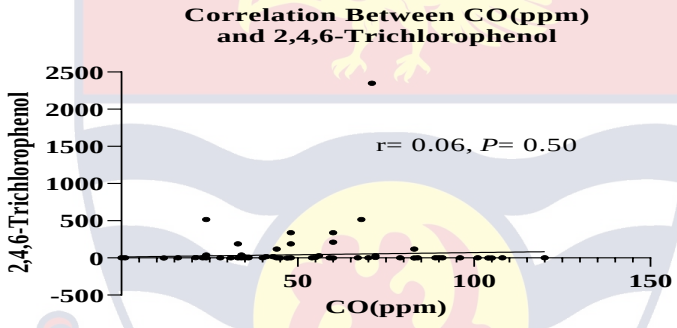
Figure 20: Correlation between Personal CO(ppm) and 1,2,4-Trichlorobenzene, Naphthalene, 2,4-Dichlorophenol, 1,1,2,3,4,4-Hexachloro1,3-Butadiene, 4-Chloroaniline and 1-Methylnaphthalene
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

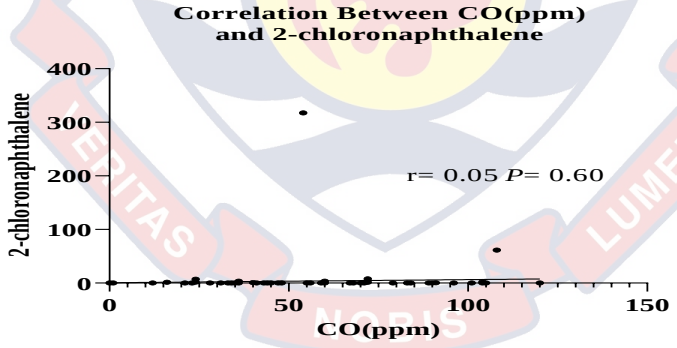
Figure 21 shows correlation between personal CO and 2-Methylnaphthalene, 4-Chloro-3-methylphenol, 2, 4,6-Trichlorophenol, 2-chloronaphthalene, 2,4,5-Trichlorophenol and 1,4-Dinitro-benzene. Correlation between Co (ppm) and the compounds above showed no statistically significant association.

From Figure 21, correlation between CO and 4-Chloro-3-methylphenol, 2, 4,5-Trichlorophenol and 1,4-Dinitro-benzene were significant statistically, r= 0.28, *P*= 0.002, r= 0.20, *P*=0.03, and r=0.19, *P*=0.03, respectively. The rest of the compounds did not correlate significantly with CO.









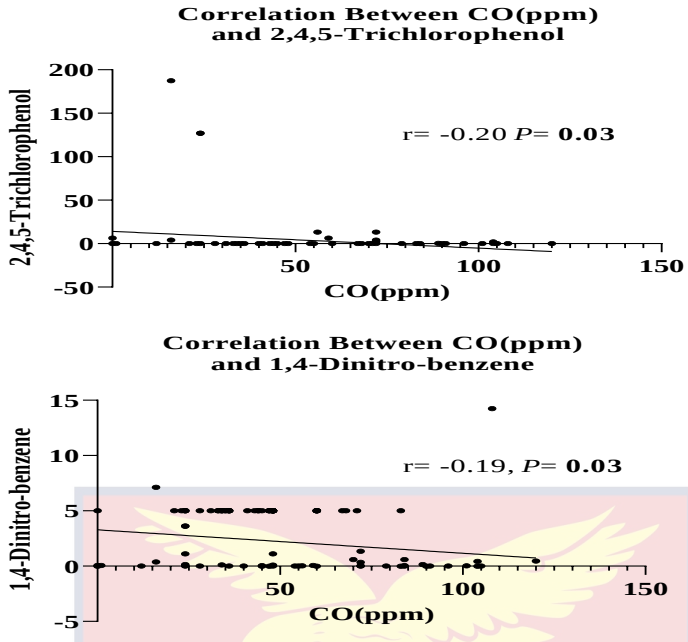
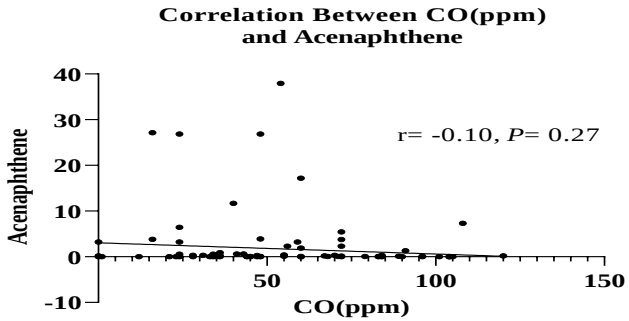
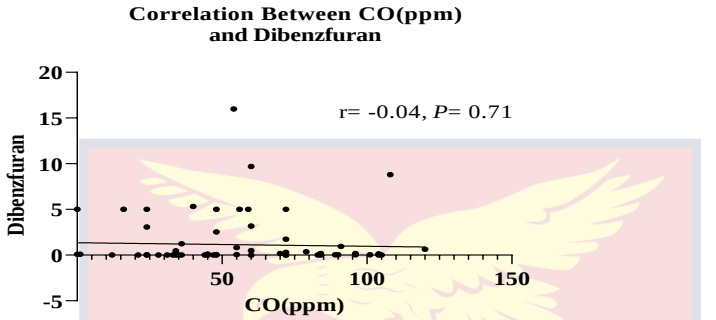
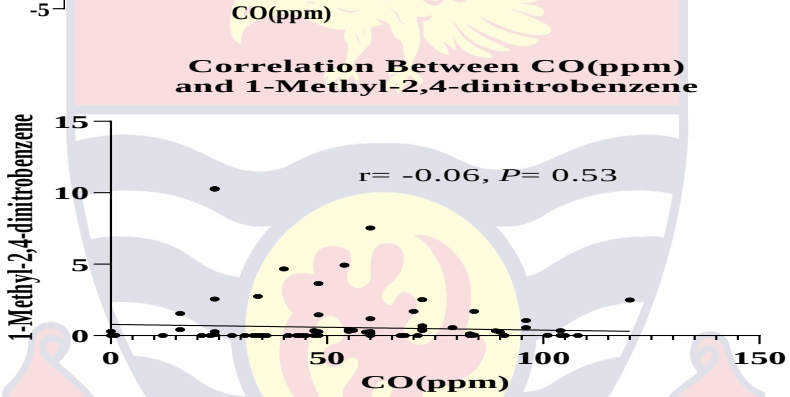


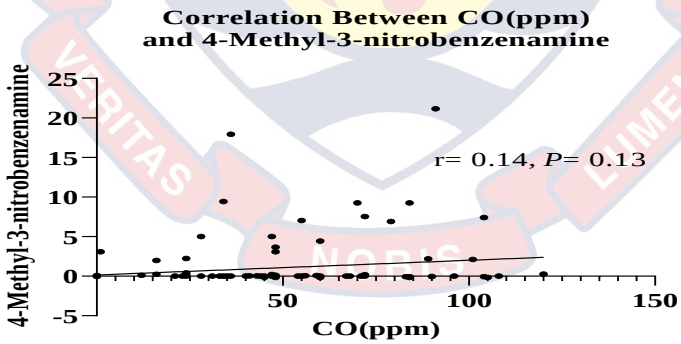
Figure 21: Correlation between Personal CO (ppm) and 2-Methylnaphthalene, 4-Chloro-3-methylphenol, 2, 4,6-Trichlorophenol, 2-Chloronaphthalene, 2,4,5-Trichlorophenol and 1,4-Dinitro-benzene
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Figure 22 shows correlation between CO and acenaphthene, dibenzfuran, 1-Methyl-2, 4-Dinitrobenzene, 4-Methyl-3-Nitrobenzenamine, 2,3,5,6-Tetrachlorophenol and 2,3,4,5-Tetrachlorophenol. From Figure 22, none of the compounds significantly correlated with CO.









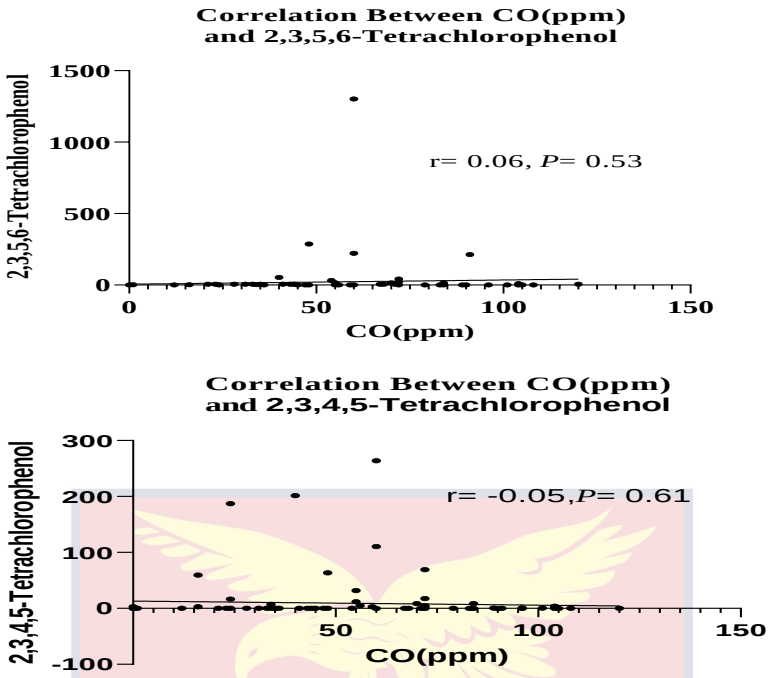
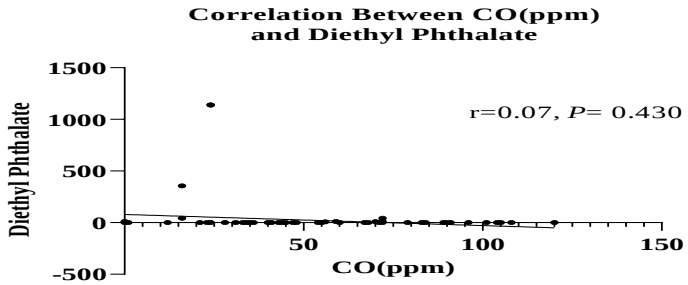
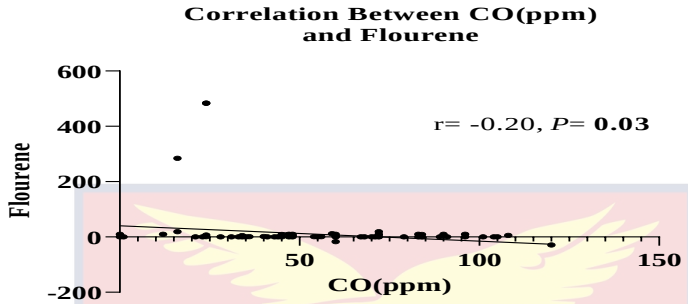


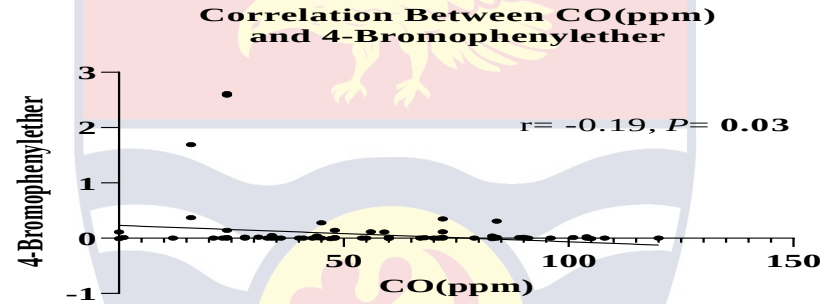
Figure 22: Correlation Between CO (ppm) and Acenaphthene, Dibenzfuran, 1-Methyl-2,4-Dinitrobenzene, 4-Methyl-3-Nitrobenzenamine, 2,3,5,6-Tetrachlorophenol and 2,3,4,5-Tetrachlorophenol

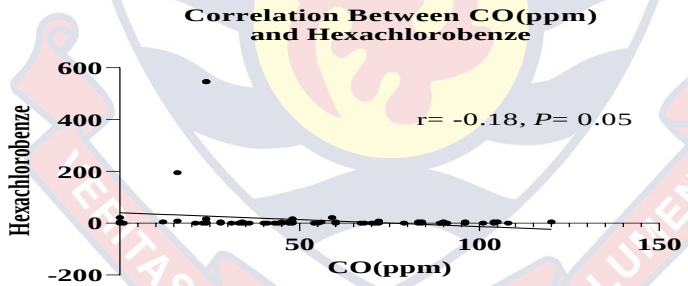
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant).

Figure 23 shows correlation between personal CO and diethylphthalate, flourene, 4-Bromophenylether, hexachlorobenze, anthracene and phenanthrene. Correlation between CO and flourene and 4-Bromophenylether were statistically significant, r= 0.20, P=0.03, and r= 0.19, P=0.03, respectively. All other compounds showed no significant correlation.









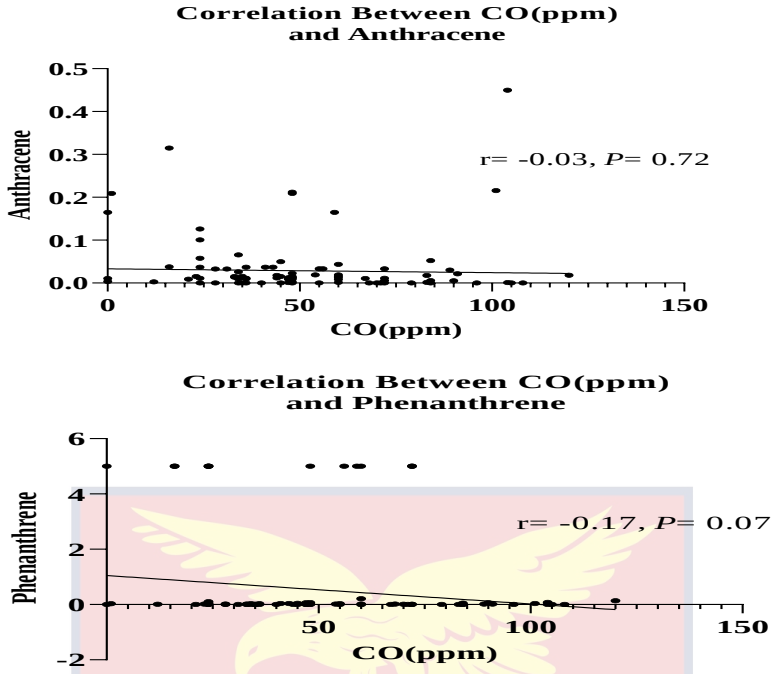
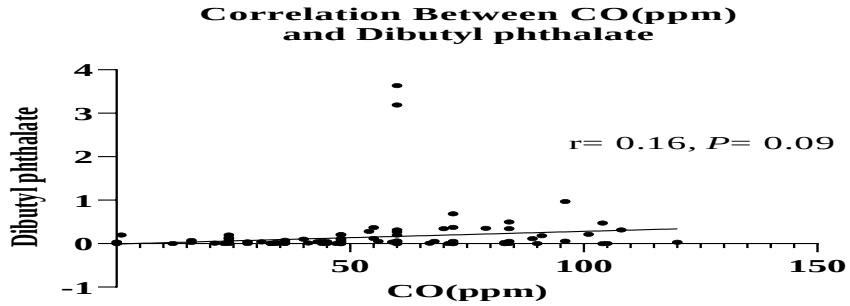
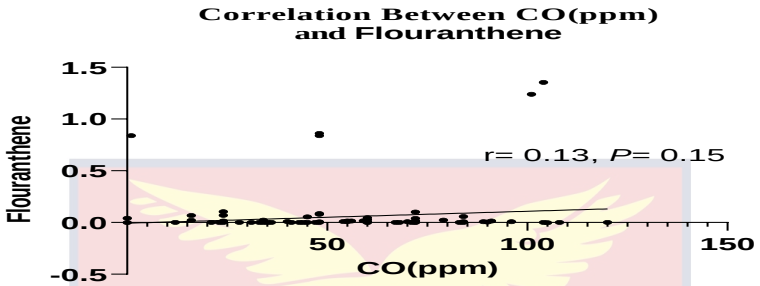


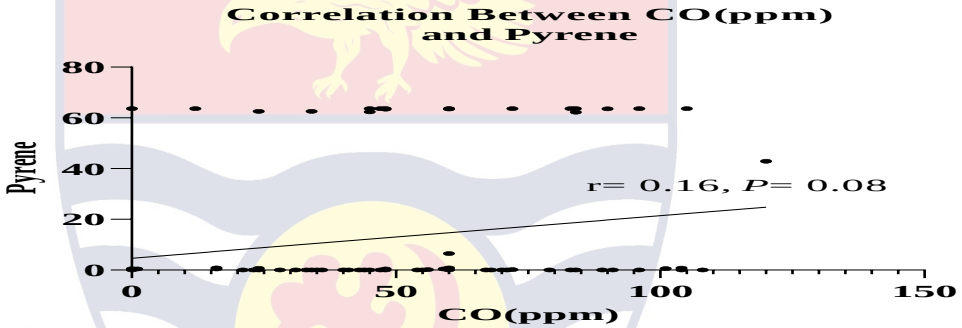
Figure 23: Correlation between Personal CO (ppm) and Diethylphthalate, Flourene, 4-Bromophenylether, Hexachlorobenze, Anthracene and Phenanthrene.

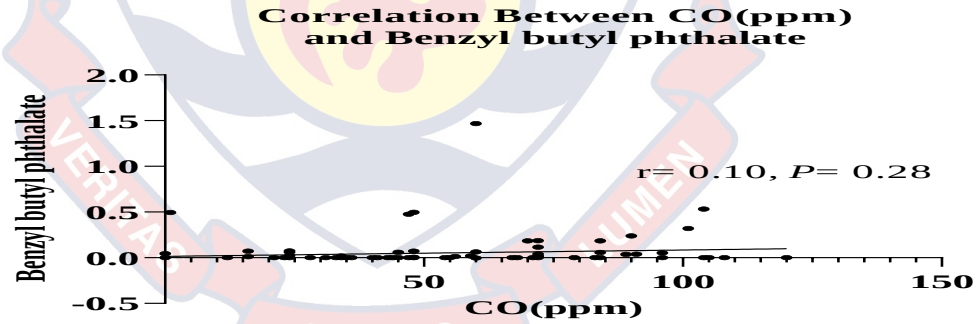
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Figure 24 shows correlation between CO and dibutylphthalate, flouranthene, pyrene, benzylbutylphthalate, dioctylesterhexanedioic acid and benzo[a]anthracene. Correlation between CO (ppm) and Benz[a]anthracene showed a significant positive association (r= 0.22, P= 0.02). However, the other compounds showed no statistically significant correlation.









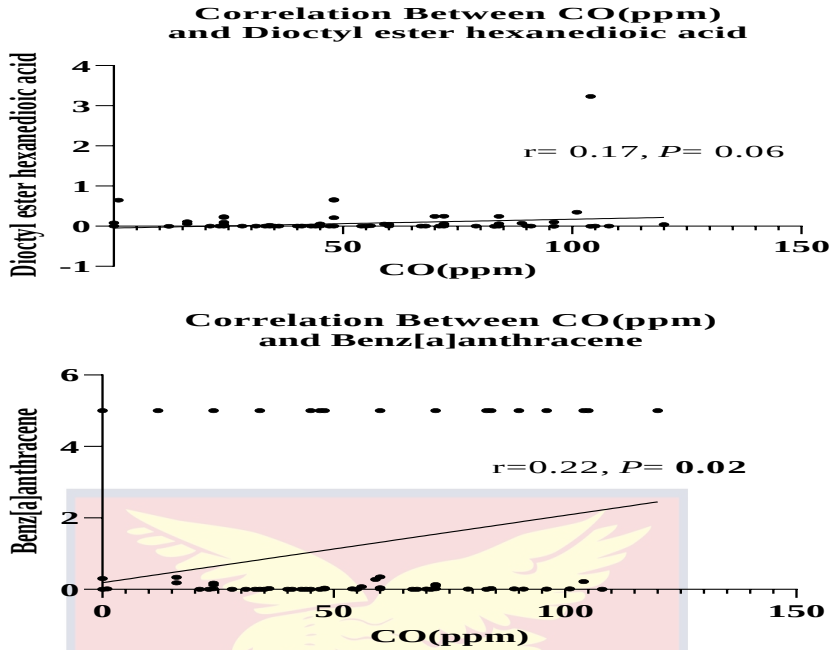
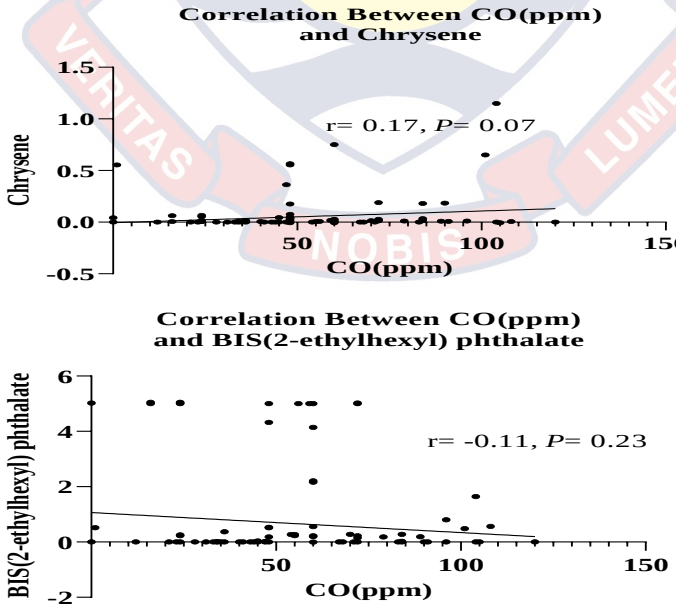


Figure 24: Correlation between CO (ppm) and Dibutylphthalate, Flouranthene, Pyrene, Benzyl butyl phthalate, Dioctyl ester hexanedioic acid and Benz[a]anthracene

r= correlation coefficient, P= P-value (<0.05 implies statistically significant)

Figure 25 shows correlation between CO and chrysene, Bis(2-ethylhexyl) phthalate, Di-n-octyl phthalate, benzo[b]fluoranthene, benzo[k]fluoranthene and benzo[a]pyrene. Correlation between personal CO and benzo[a]pyrene was significantly positive (r= 0.22, P= 0.02). All the other correlations were statistically insignificant.



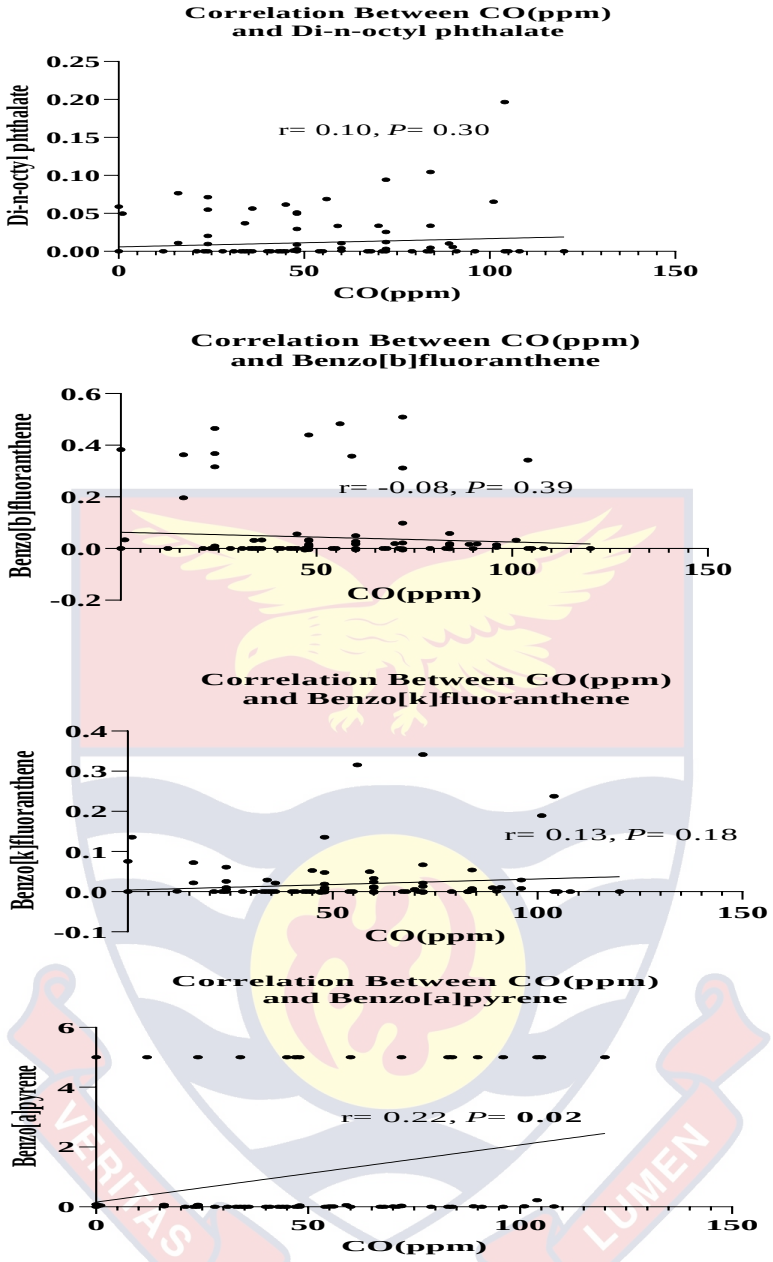
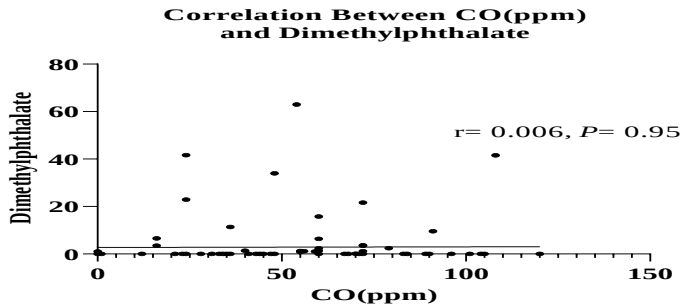
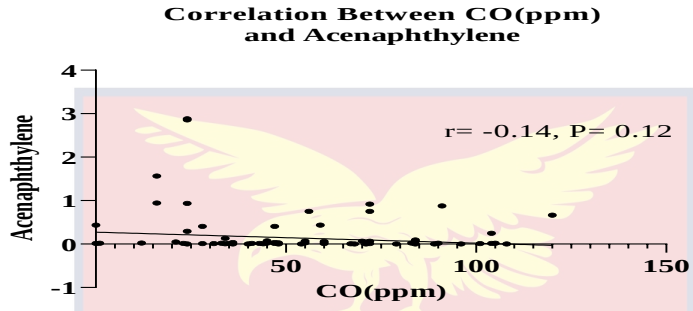
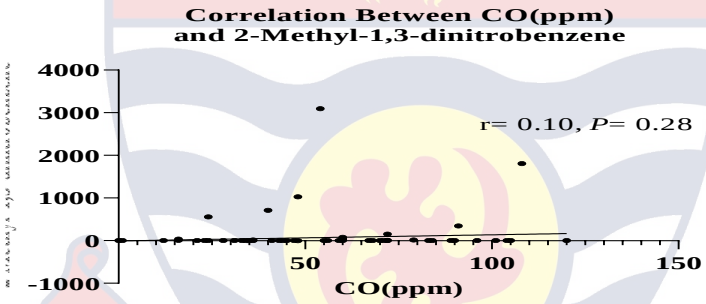


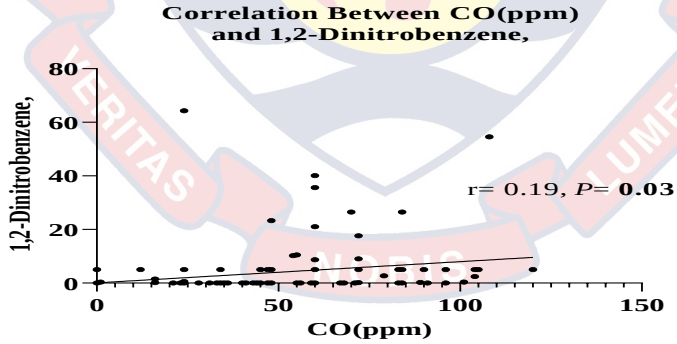
Figure 25: Correlation between CO (ppm) and Chrysene, Bis(2-Ethylhexyl) phthalate, Di-n-octylphthalate, Benzo[b]fluoranthene, Benzo[k]fluoranthene and Benzo[a]pyrene

Figure 26 shows correlation between personal CO and dimethylphthalate, acenaphthylene, 2-Methyl-1,3-dinitrobenzene, 1,2-dinitrobenzene, indeno[1,2,3-cd] pyrene and dibenz[a,h]anthracene. None of the above correlations was significant except personal CO(ppm) and 1,2-Dinitrobenzene which exhibited a weak positive correlation (r= 0.19, *P*= 0.03).









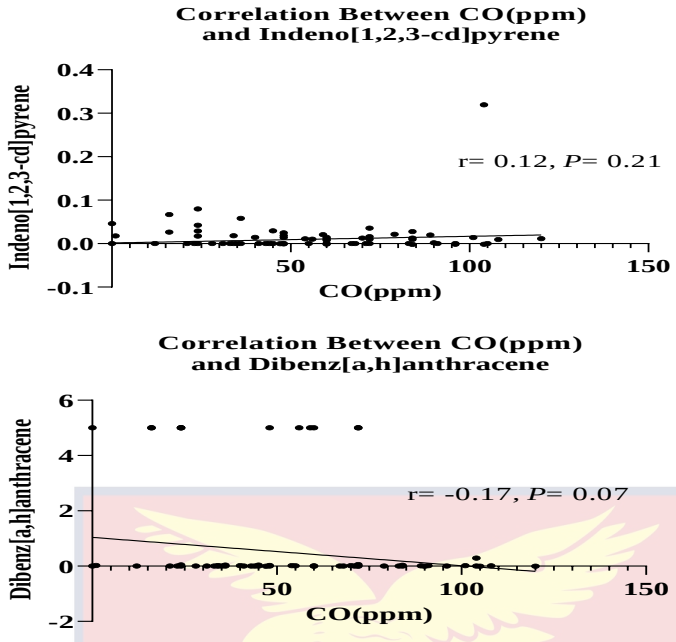


Figure 26: Correlation between Personal CO(ppm) and Dimethylphthalate, Acenaphthylene, 2-Methyl-1,3-Dinitrobenzene, 1,2-Dinitrobenzene, Indeno[1,2,3-cd]pyrene and Dibenz[a,h]anthracene

r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Figure 27 shows correlation between CO and benzo [ghi] perylene

There was no significant correlation between CO (ppm) and Benzo [ghi] perylene.

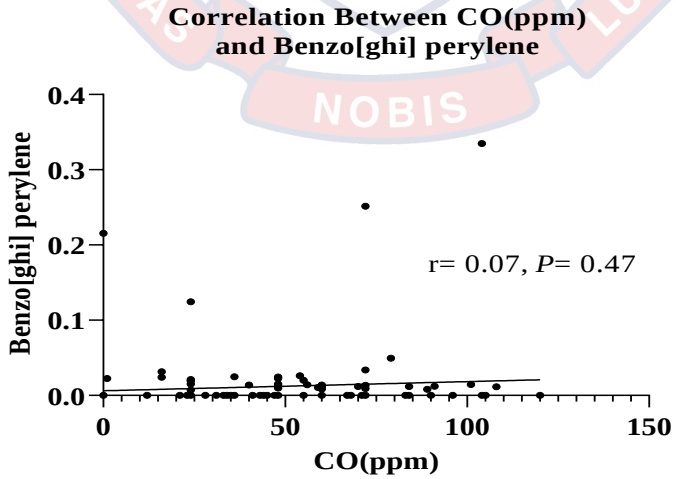
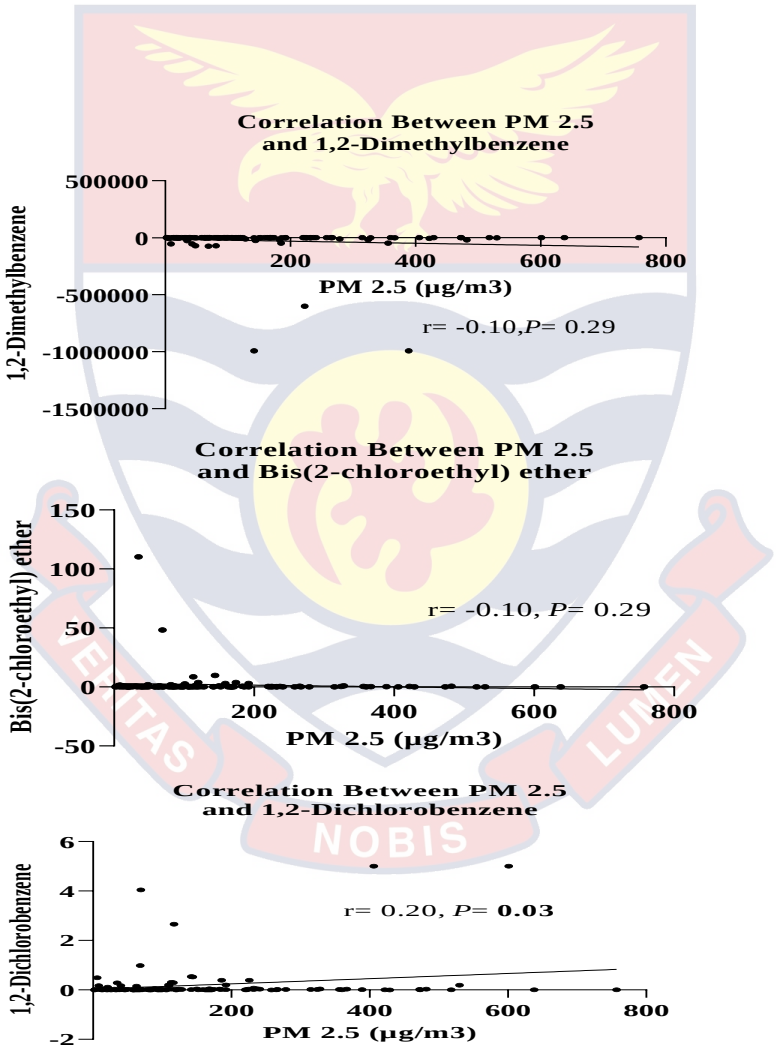


Figure 27: Correlation between CO(ppm) and Benzo [ghi] perylene

r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant) Figure 28 shows correlation between personal PM $_{2.5}$ and 1,2-Dimethylbenzene, Bis(2-Chloroethyl) ether, 1,2-Dichlorobenzene, 2-Chlorophenol, 1,2-Dichlorobenzene and phenol. A significant positive correlation was observed between personal PM $_{2.5}$ and 1,2-Dimethylbenzene, r= 0.20, P= 0.03. All other compounds showed no statistically significant correlation.



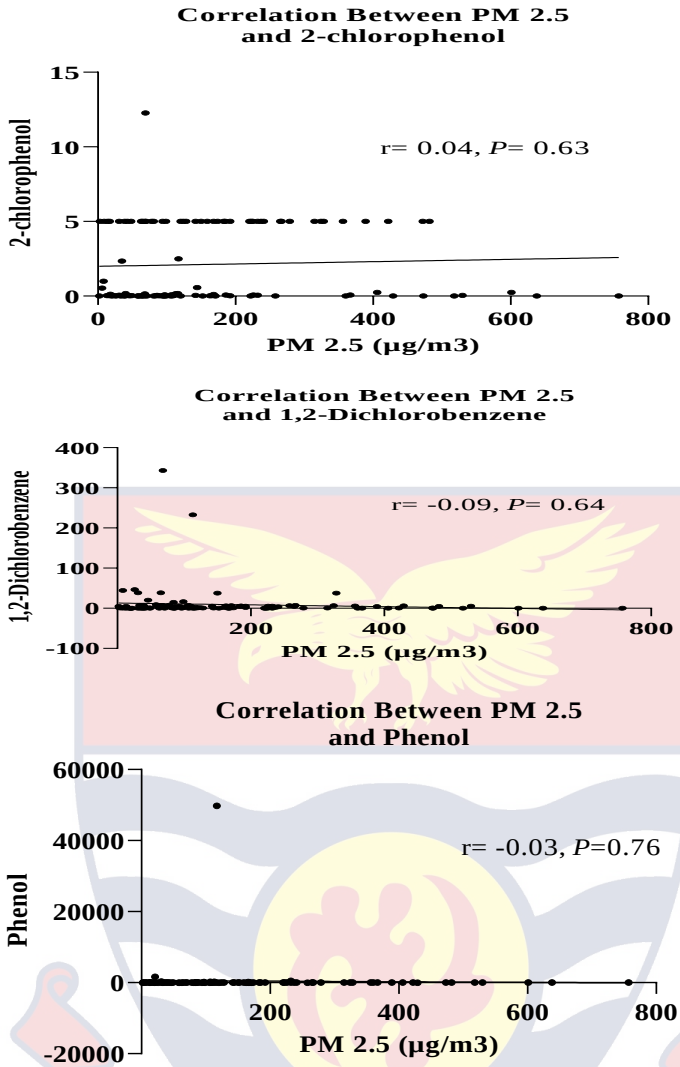
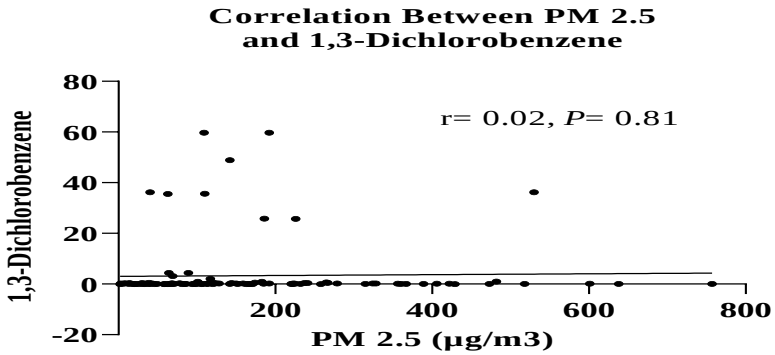


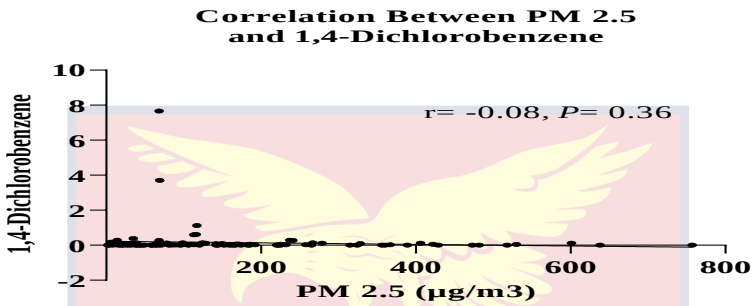
Figure 28: Correlation between Personal PM $_{2.5}$ and 1,2-Dimethylbenzene, Bis(2-chloroethyl) ether, 1,2-Dichlorobenzene, 2-Chlorophenol, 1,2-Dichlorobenzene and Phenol

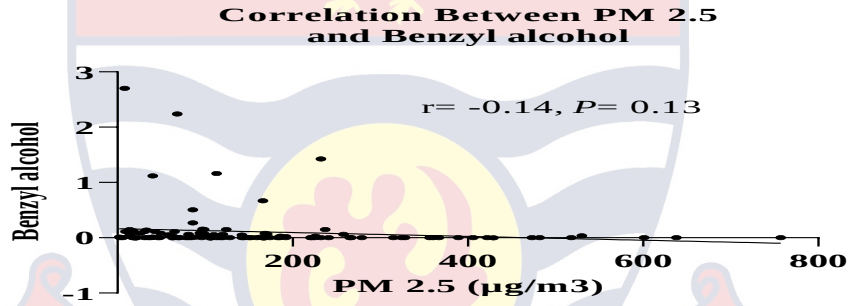
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

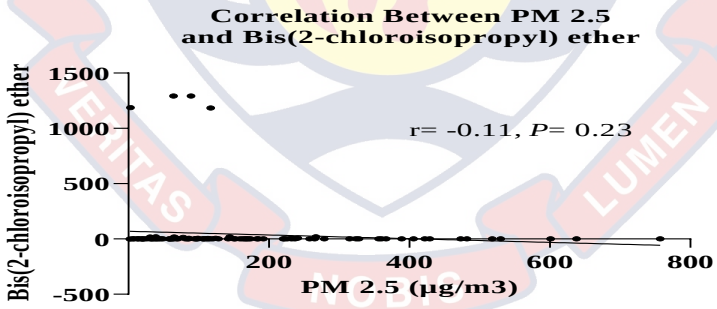
Figure 29 shows correlation between personal PM $_{2.5}$ and 1,3-Dichlorobenzene, 1,4-Dichlorobenzene, Benzyl alcohol, Bis(2-chloroisopropyl) ether, Hexachloroethane and 2-Methylphenol

There were no significant correlations between personal PM $_{2.5}$ and 1,3-Dichlorobenzene, 1,4-Dichlorobenzene, Benzyl alcohol, bis(2-Chloroisopropyl) ether, hexachloroethane and 2-Methylphenol.









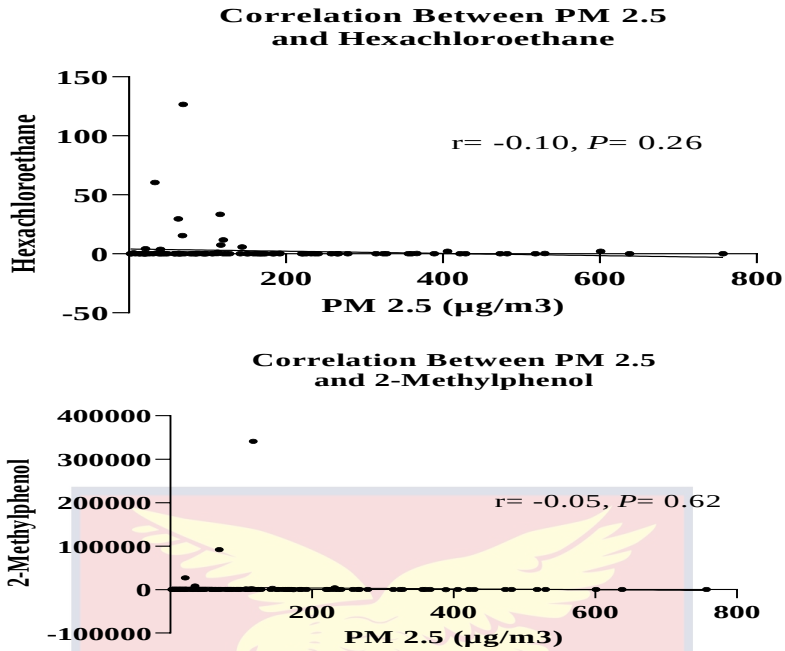
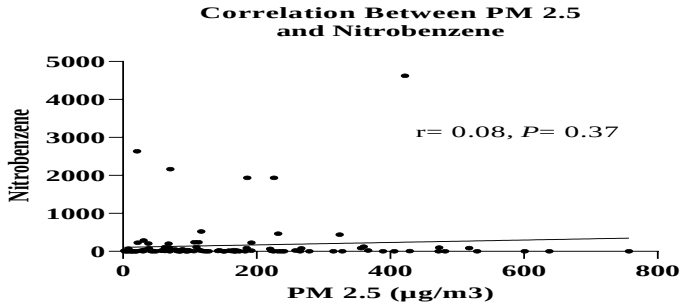
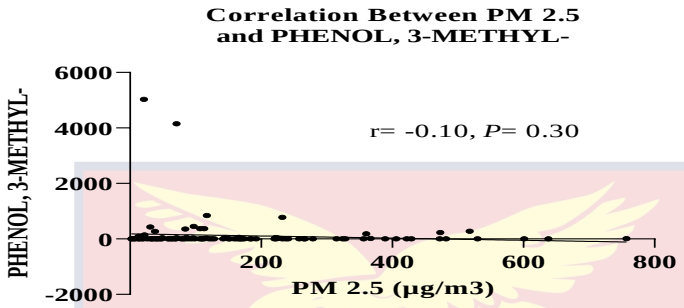


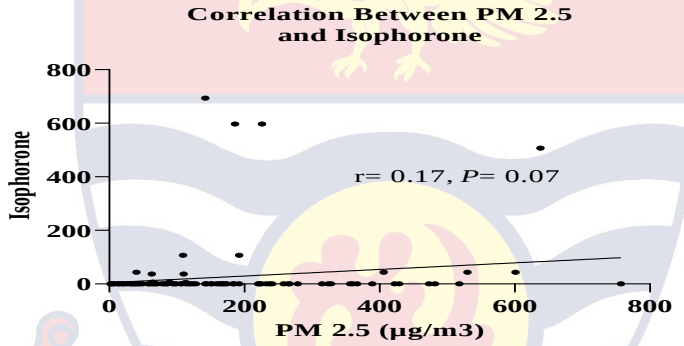
Figure 29: Correlation between Personal PM $_{2.5}$ and 1,3-Dichlorobenzene, 1,4-Dichlorobenzene, Benzyl alcohol, Bis(2-chloroisopropyl) ether, Hexachloroethane and 2-Methylphenol

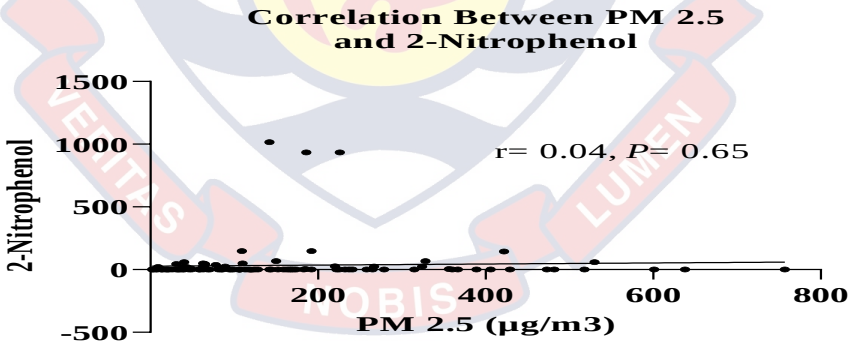
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Figure 30 shows correlation between personal PM $_{2.5}$ and Nitrobenzene, Phenol, 3-Methyl-, Isophorone, 2-Nitrophenol, Bis(2-Chloroethoxy) methane and 2,4-Dimethylphenol. There were no significant correlations between personal PM $_{2.5}$ and Nitrobenzene, Phenol, 3-Methyl-, Isophorone, 2-Nitrophenol and 2,4-Dimethylphenol. Except correlation between PM $_{2.5}$ and Bis(2-chloroethoxy) methane (r= 0.30, P<0.001) which showed a positive correlation.









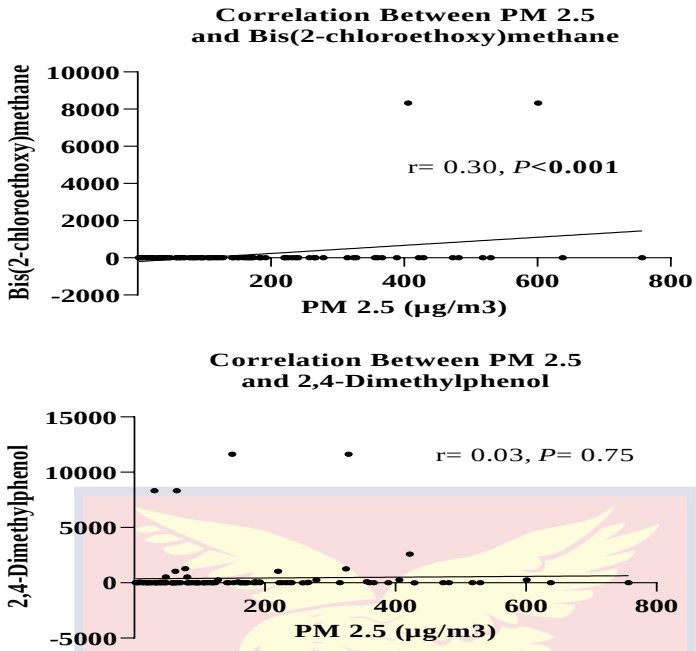
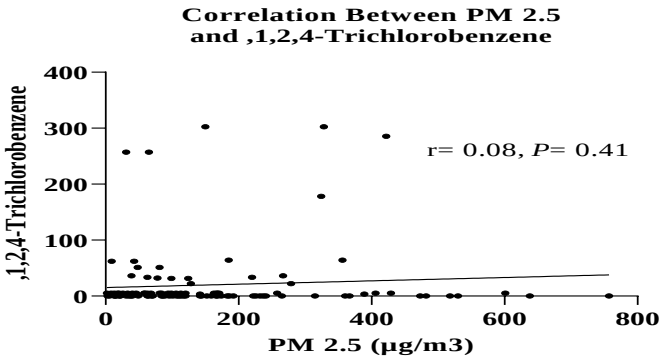


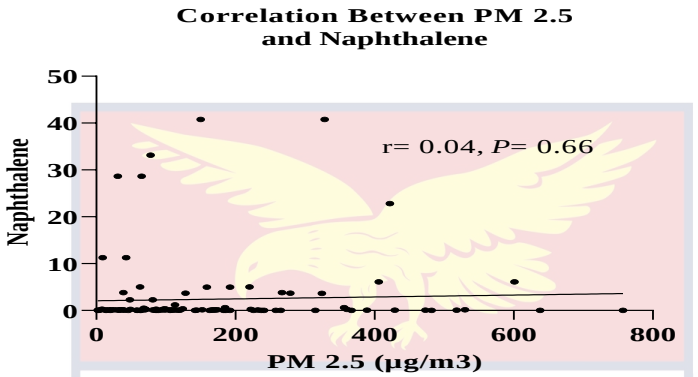
Figure 30: Correlation between Personal PM $_{2.5}$ and Nitrobenzene, 3-Methylphenol, Isophorone, 2-Nitrophenol, Bis(2-chloroethoxy) methane and 2,4-Dimethylphenol

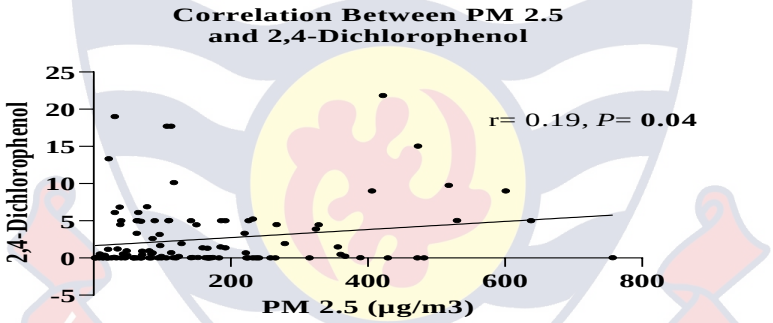
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

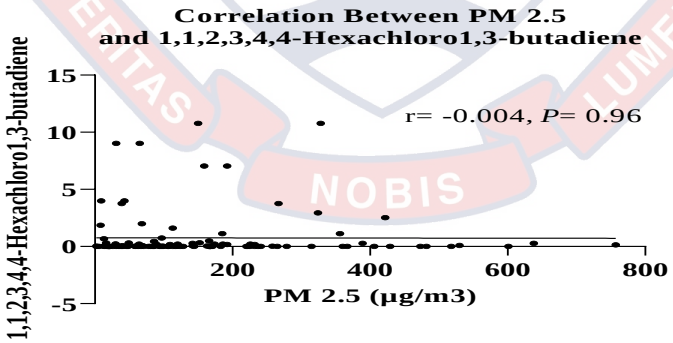
Figure 31 shows correlation between personal PM $_{2.5}$ and 1,2,4-Trichlorobenzene, naphthalene, 2,4-Dichlorophenol, 1,1,2,3,4,4-Hexachloro1,3-butadiene, 4-Chloroaniline and 1-Methylnaphthalene.

Correlation between PM $_{2.5}$ and, 1, 2, 4-Trichlorobenzene, Naphthalene, 1,1,2,3,4,5-Hexachloro1,3-butadiene, 4-Chloroaniline and 1-Methylnaphthalene all showed no significant relationship statistically. However, correlation between PM $_{2.5}$ and, 2,4-Dichlorophenol was significant statistically (r= 0.19, *P*= 0.04).









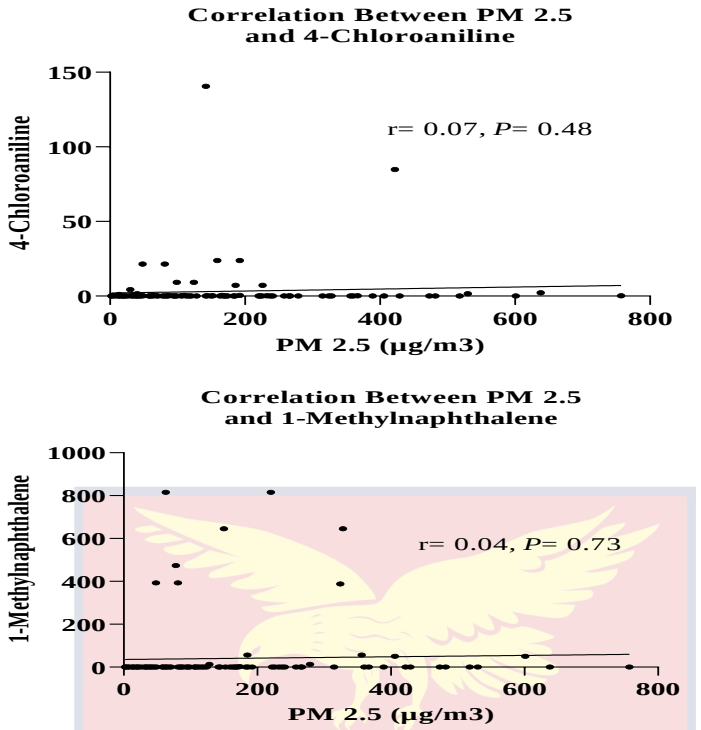
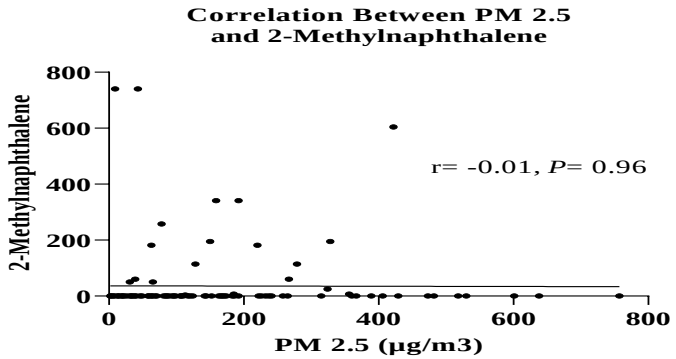


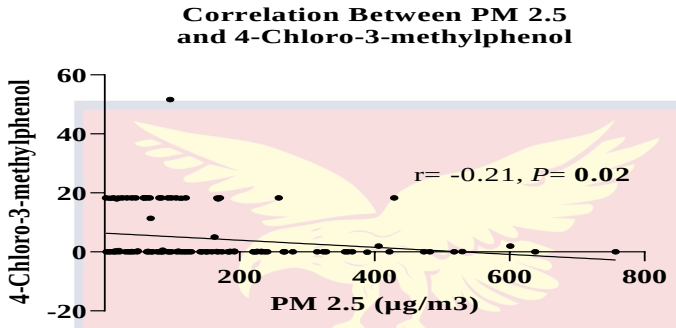
Figure 31: Correlation between Personal PM $_{2.5}$ and ,1,2,4-Trichlorobenzene, Naphthalene, 2,4-Dichlorophenol, 1,1,2,3,4,4-Hexachloro1,3-Butadiene, 4-Chloroaniline and 1-Methylnaphthalene

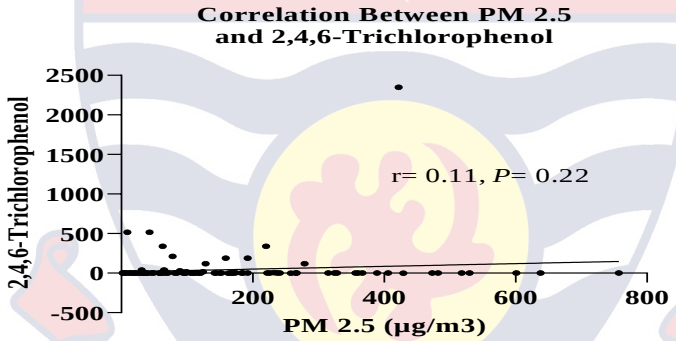
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

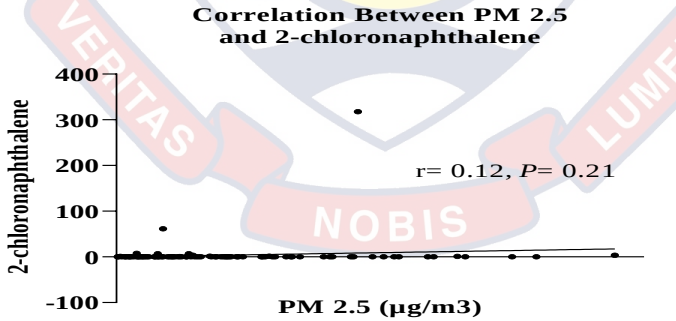
Figure 32 shows correlation between personal PM $_{2.5}$ and 2-Methylnaphthalene, 4-Chloro-3-methylphenol, 2,4,6-Trichlorophenol, 2-chloronaphthalene, 2,4,5-Trichlorophenol and 1,4-Dinitro-benzene.

Correlation between personal PM $_{2.5}$ and 4-Chloro-3-methylphenol was negatively significant (r= -0.21, *P*= 0.002). None of the compounds left showed a significant association.









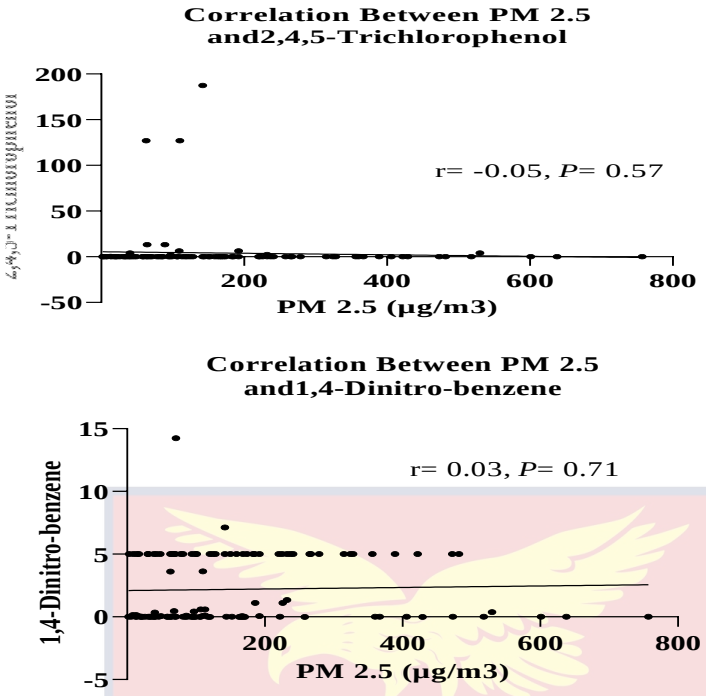
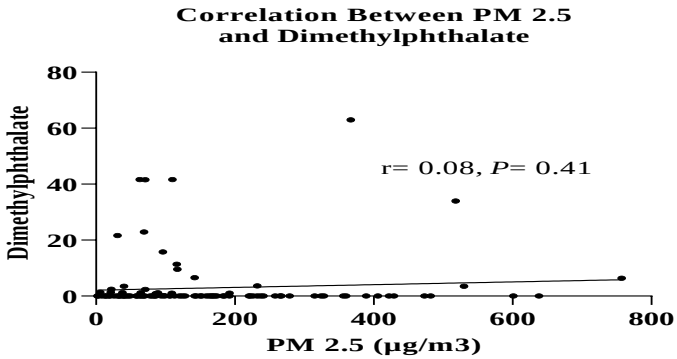
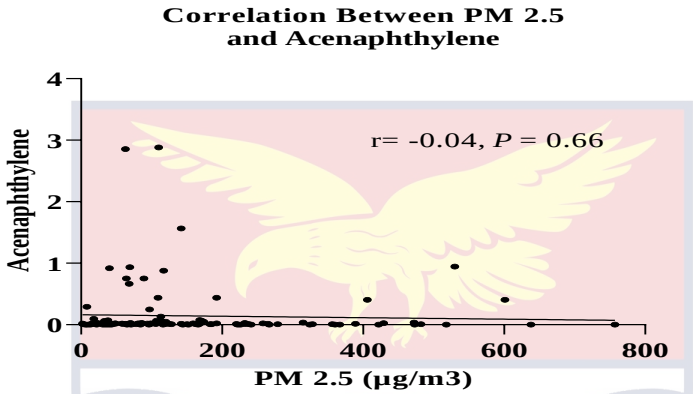
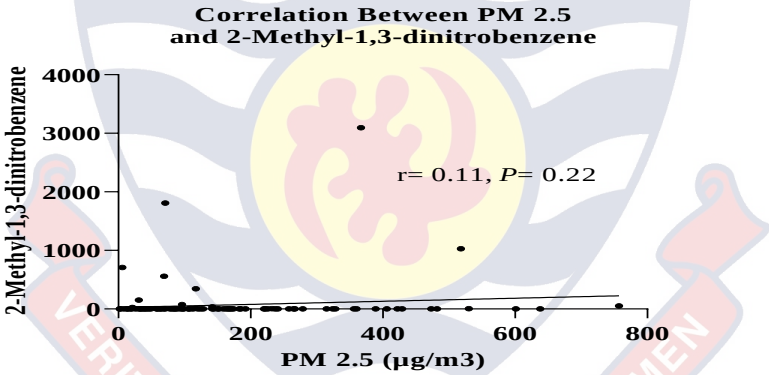


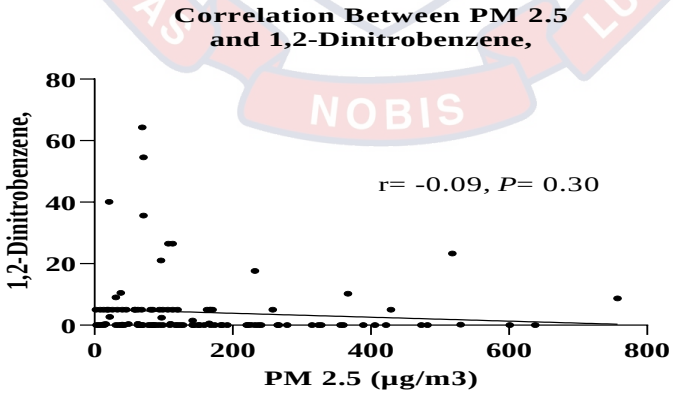
Figure 32: Correlation Between Personal PM $_{2.5}$ and 2-Methylnaphthalene, 4-Chloro-3-Methylphenol, 2,4,6-Trichlorophenol, 2-Chloronaphthalene, 2,4,5-Trichlorophenol and 1,4-Dinitrobenzene
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Figure 33 shows correlation between PM $_{2.5}$ and dimethylphthalate, acenaphthylene, 2-Methyl-1,3-dinitrobenzene, 1,2-Dinitrobenzene, acenaphthene and dibenzfuran. Correlation between personal PM $_{2.5}$ and Dimethylphthalate, Acenaphthylene, 2-Methyl-1,3-dinitrobenzene and 1,2-Dinitrobenzene all showed no significant association. However, positive correlations were found between PM $_{2.5}$ and Acenaphthene (r= 0.19, P= 0.04), and Dibenzfuran (r= 0.22, P= 0.03).









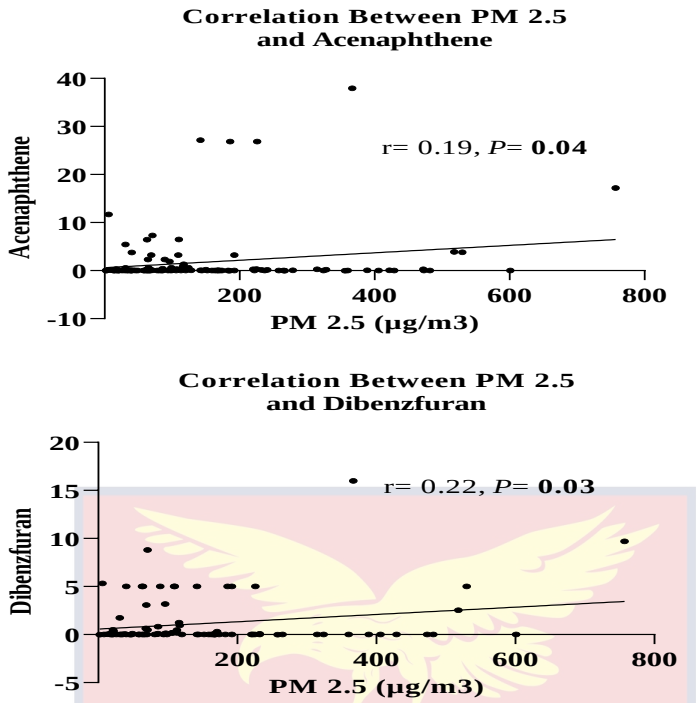
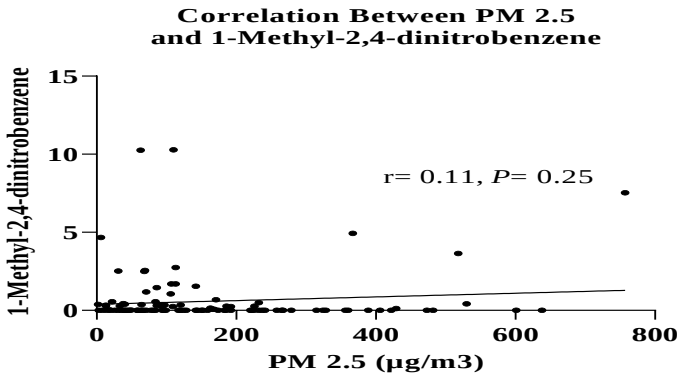
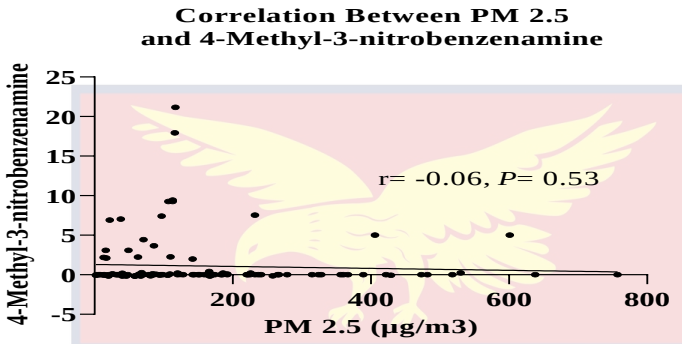
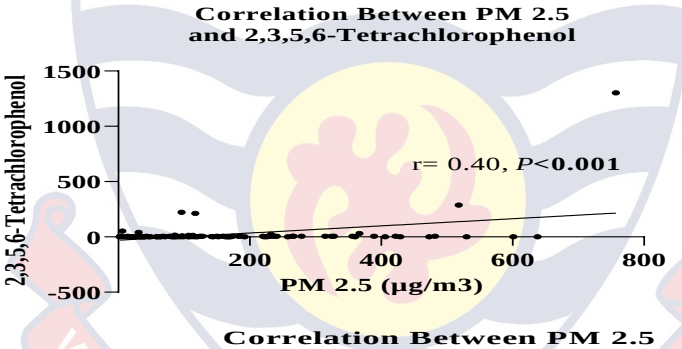


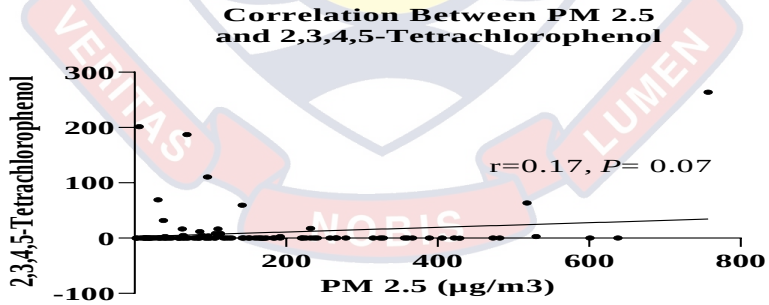
Figure 33: Correlation between PM $_{2.5}$ and Dimethylphthalate, Acenaphthylene, 2-Methyl-1,3-Dinitrobenzene, 1,2-Dinitrobenzene, Acenaphthene and Dibenzfuran
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Correlations between personal PM $_{2.5}$ and 1-Methyl-2,4-Dinitrobenzene, 4-Methyl-3-Nitrobenzenamine, 2,3,5,6-Tetrachlorophenol, 2,3,4,5-Tetrachlorophenol, diethylphthalate and flourene are presented in Figure 34. Correlation between PM $_{2.5}$ and 2,3,4,5-Tetrachlorophenol was statistically significant positively (r=0.40, P= 0.001). The other compounds showed no significant correlation with personal PM $_{2.5.}$









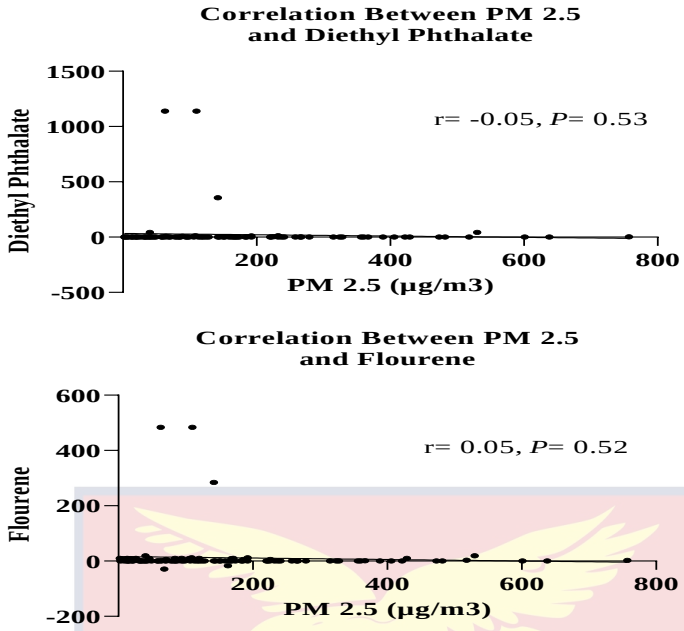
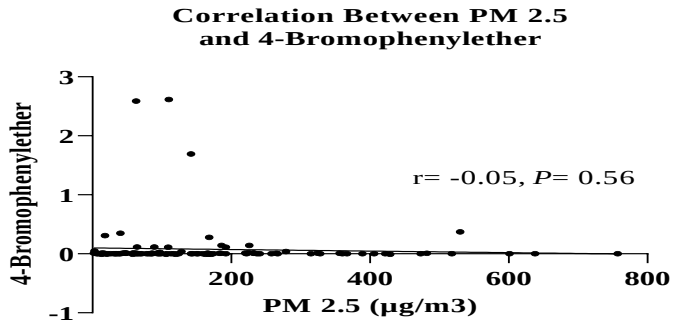


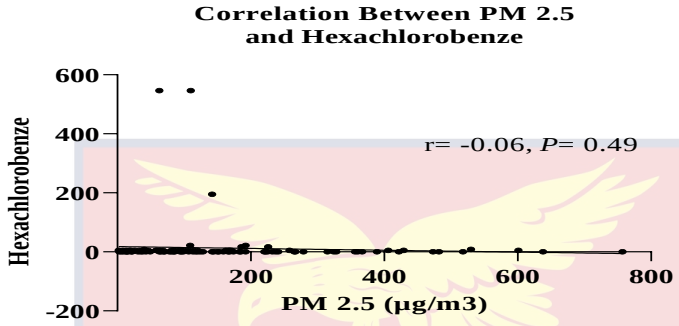
Figure 34: Correlation between personal PM $_{2.5}$ and 1-Methyl-2,4-Dinitrobenzene, 4-Methyl-3-Nitrobenzenamine, 2,3,5,6-Tetrachlorophenol, 2,3,4,5-Tetrachlorophenol, Diethyl Phthalate and Flourene

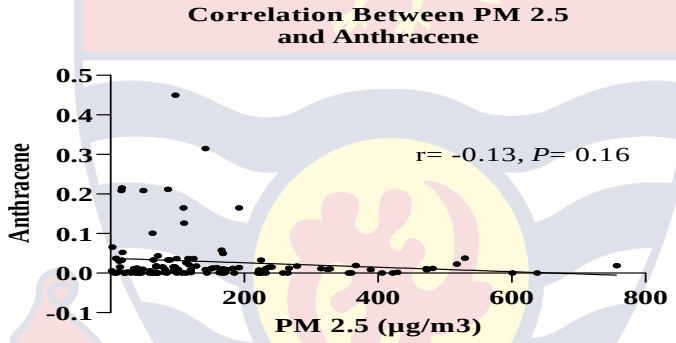
r= correlation coefficient, P= P-value (<0.05 implies statistically significant)

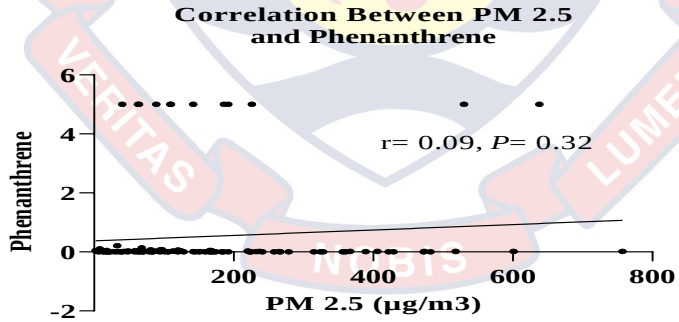
Correlation between PM $_{2.5}$ and 2,3,4,5-Tetrachlorophenol was statistically significant positively (r=0.40, *P*= 0.001). The other compounds showed no significant correlation with personal PM $_{2.5.}$

Correlations between personal PM $_{2.5}$ and 4-Bromophenylether, hexachlorobenze, anthracene, phenanthrene, dibutyl phthalate and flouranthene are shown in Figure 35. There were no significant correlations between personal PM $_{2.5}$ and 4-Bromophenylether, hexachlorobenze, anthracene, phenanthrene, dibutyl phthalate and flouranthene.









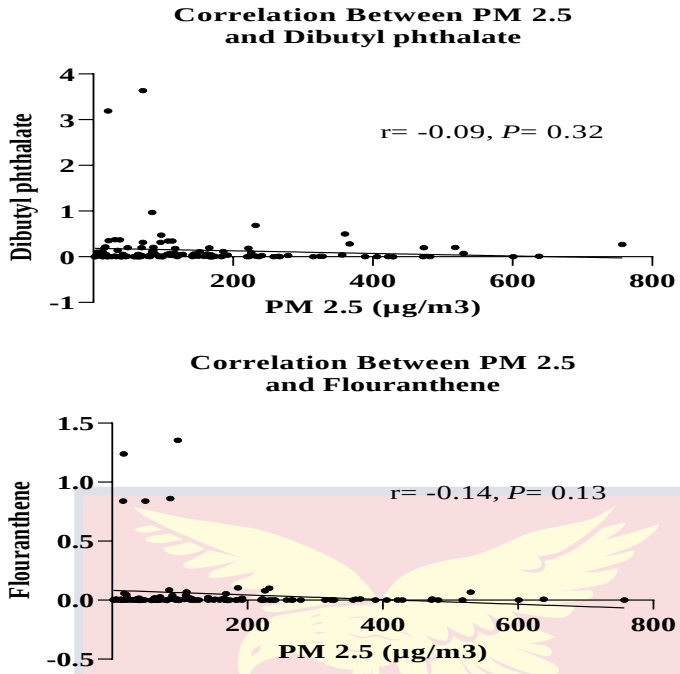
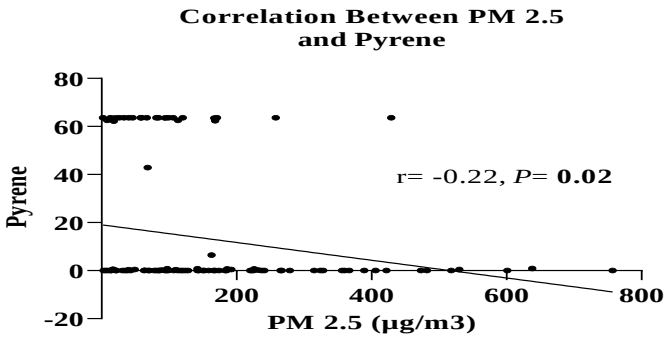
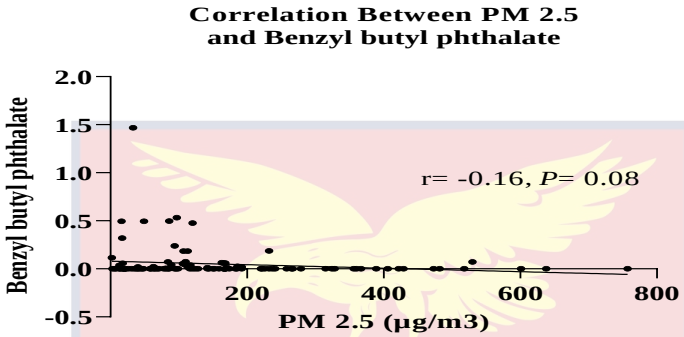


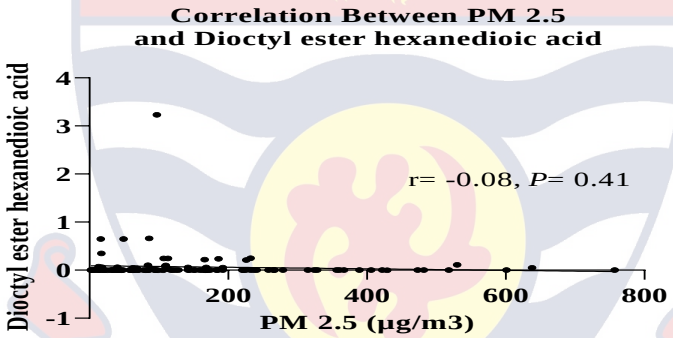
Figure 35: Correlation between Personal PM $_{2.5}$ and 4-Bromophenylether, Hexachlorobenze, Anthracene, Phenanthrene, Dibutyl phthalate and Flouranthene

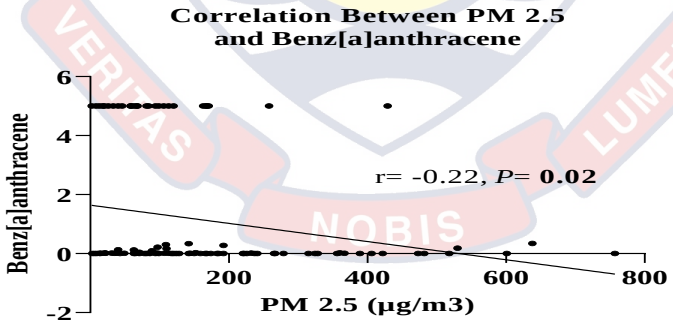
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Correlations between personal PM $_{2.5}$ and pyrene, benzylbutylphthalate, dioctylesterhexanedioic acid, benzo[a]anthracene, chrysene and bis(2-Ethylhexyl) phthalate are shown in Figure 36. Correlation between PM $_{2.5}$ and pyrene, and benz[a]anthracene was negatively significantly correlated, r= -0.22, P= 0.02 in each case. However, correlation between PM $_{2.5}$ and benzylbutylphthalate, dioctylesterhexanedioic acid, chrysene, and bis(2-Ethylhexyl) phthalate all had no significant correlation statistically.









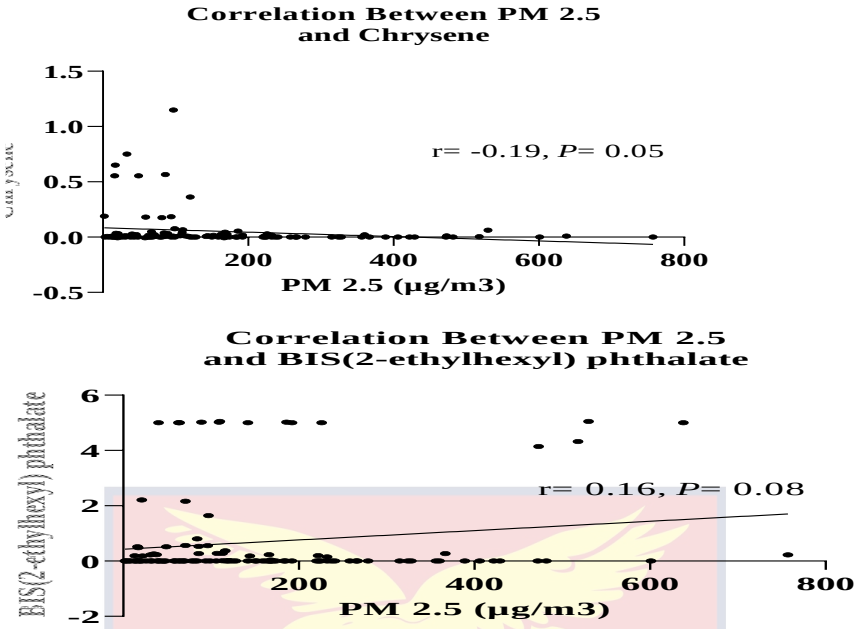


Figure 36: Correlation between Personal PM $_{2.5}$ and Pyrene, Benzylbutylphthalate, Dioctylesterhexanedioic acid, benzo[a]anthracene, Chrysene and bis(2-Ethylhexyl) phthalate
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Figure 37 shows correlation between personal PM $_{2.5}$ and Di-n-octyl phthalate, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, indeno [1,2,3-cd] pyrene and dibenz [a, h] anthracene. Correlation between PM 2.5 and Di-n-octyl phthalate, benzo[b]fluoranthene, benzo[k]fluoranthene, indeno [1, 2,3-cd] pyrene, and dibenz [a, h] anthracene was all insignificant statistically. Yet correlation between PM $_{2.5}$ and benzo[a]pyrene was negatively significant statistically, r= -0.23, P= 0.01.

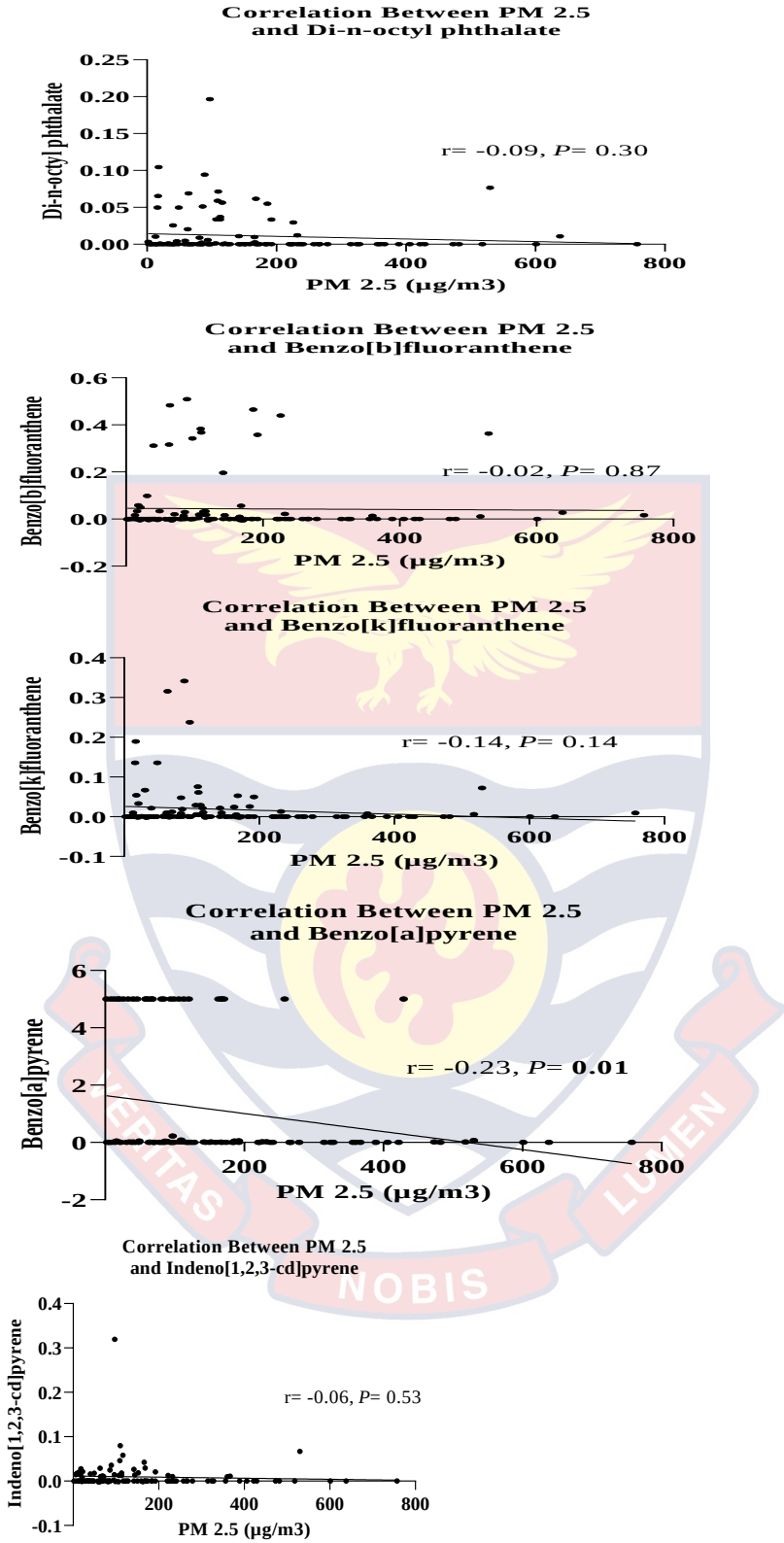


Figure 37: Correlation between Personal PM $_{2.5}$ and Di-n-octyl phthalate, Benzo[b]fluoranthene, Benzo[k]fluoranthene, Benzo[a]pyrene, Indeno [1,2,3-cd] pyrene and Dibenz [a, h] anthracene

Figure 38 shows correlation between PM $_{2.5}$ and benzo[ghi] perylene. Correlation between PM $_{2.5}$ and Benzo[ghi] perylene was not significant statistically.

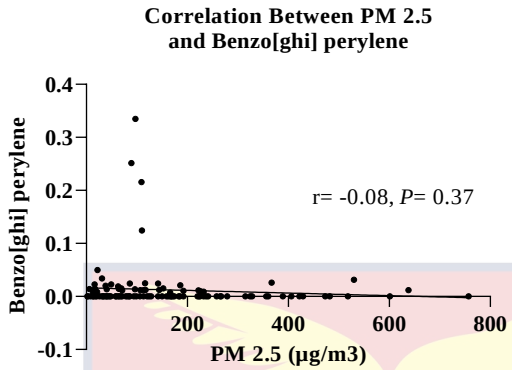


Figure 38: Correlation between PM $_{2.5}$ and Benzo[ghi] perylene
Figure 39 shows correlation between PM $_{2.5}$ and 9-Phenanthrenol

A significant positive correlation was observed between personal PM $_{2.5}$ and 9-Phenanthrenol (r= 0.24, P= 0.007).

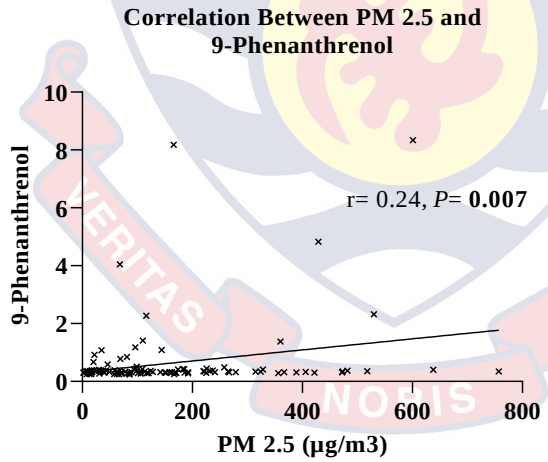


Figure 39: Correlation between PM $_{2.5}$ and 9-Phenanthrenol
Correlation between personal PM $_{2.5}$ and 1-Hydroxypyrene is shown in Figure 40. A significantly positive correlation was observed between personal PM $_{2.5}$ and 1-Hydroxypyrene, r= 0.23, P= 0.01.

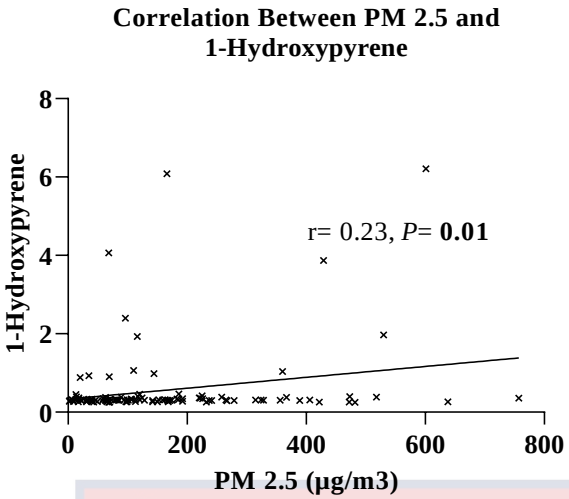


Figure 40: Correlation between Personal PM $_{2.5}$ and 1-Hydroxypyrene

In Figure 41 there was no significant relationship between the correlation of personal CO and 9-Phenanthrenol.

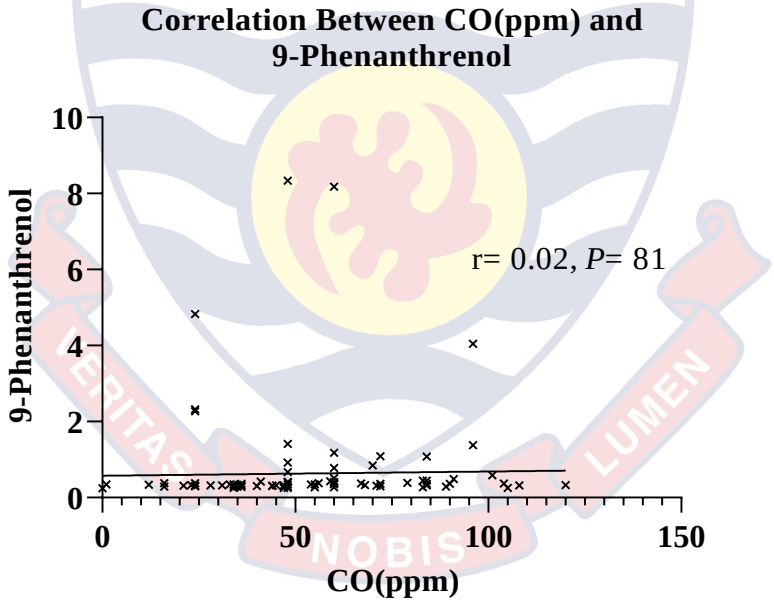


Figure 41: Correlation between CO (ppm) and 9-Phenanthrenol

No significant relationship was observed between the correlation of CO and

1-Hydroxypyrene as shown in Figure 42.

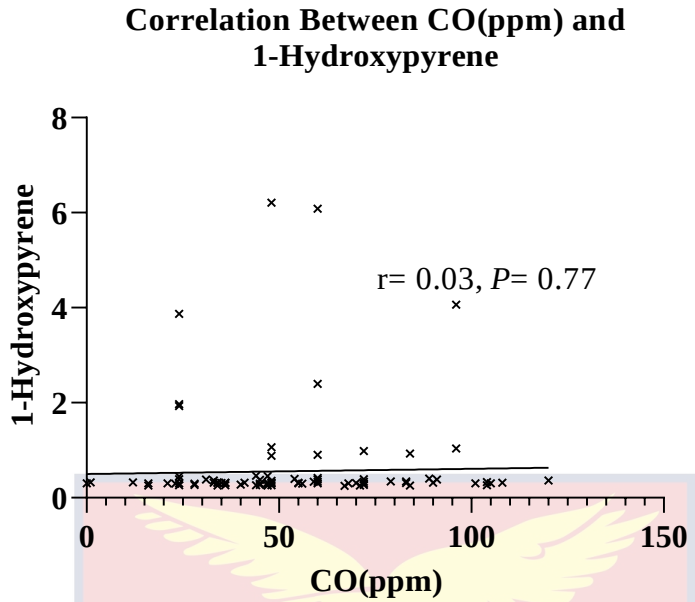


Figure 42: Correlation between Personal CO (ppm) and 1-Hydroxypyrene
r= correlation coefficient, *P*= P-value (<0.05 implies statistically significant)

Chapter Summary

Levels of PAHs, phenols, substituted benzene and phthalates that were assessed in urine samples have been discussed. Health outcomes after eye screening such as cataracts have also been discussed after eye screening. Also, self-reported health symptoms such as asthma, headache and cough have been discussed. Furthermore, levels of particulate matter, carbon monoxide and ozone have been discussed. Finally, concentration of personal $PM_{2.5}$ and personal CO as well as association between exposure to $PM_{2.5}$ and personal CO and urinary PAHs and metabolites have been discussed. Fish smoking serves as a source of livelihood for fish smokers and their dependents but they are exposed to a lot of smoke particles. This work has explained the need for stakeholders and policy makers to device interventions that would help them to work in a conducive environment.

CHAPTER FIVE

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

In this study biomass fuel usage has been shown to be a source of indoor air pollution among fish smokers. Factors such as type of fuel, type of stove, ventilation and location of cooking have also been shown to affect the concentration of personal $PM_{2.5}$ and personal CO. Fish smokers could be exposed to smoke particles such as PAHs, $PM_{2.5}$ and CO especially in indoor smokehouses.

Conclusions

Urinary PAHs, metabolites, substituted benzenes, phenol and phthalates among fish smokers were above the standard set by The Agency for Toxic Substances and Disease Registry, ATSDR.

Most of the smokehouses had high temperatures and could expose fish smokers to heat which could affect their health over long period of time. Relative humidity in the smokehouses were within standards set by American Society of Heating, Refrigerating and Air-Conditioning Engineers, ASHRAE 1989. Most smokehouses had $PM_{2.5}$ higher than WHO standard and Ghana EPA standard.

Pearson's correlation analysis showed no correlation between humidity and $PM_{2.5}$ but there was significant correlation between CO and O_3. Also, there was significant correlation between $PM_{2.5}$ and CO; correlation between $PM_{2.5}$ and $O_{3;}$ correlation between CO and $O_{3.}$

Another objective was to assess the relationship between exposure duration to smoke by fish smokers and eye diseases and other health

outcomes. On self- reported eye discomforts, fish smokers reported suffering from blurred vision, itching, pain, tearing, foreign body sensation, photophobia, discharge, burning sensation, headache, glare, intermittent diplopia, blepharopasm.

After eye screening it was also realized that some fish smokers suffered disease such as cataract, ineffective conjunctivitis, allergic ineffective conjunctivitis, dry eye syndrome, glaucoma suspect, pterygium, pinguecula, retinopathies, refractive error. The prevalence of visual impairment in this study was 11.7%.

On the exposure duration and self-reported health outcomes, it could be concluded that exposure duration had no association with headache, cough, wheezing. There was association between exposure duration and breathlessness, eye disease, asthma and pneumonia.

Risk assessment on urinary contaminants showed high health risk values in the study areas and could pose adverse health problems for the fish smokers living in the studied communities.

Personal PM $_{2.5}$ and personal CO were assessed among fish smokers in Central and Volta regions of Ghana. Most of the participants were between the 30-40 years. Biomass fuel (firewood) was used by fish smokers to smoke fish.

In addition, fish smokers used biomass fuel (firewood, charcoal) and one participant used LPG to cook their meals. Personal PM $_{2.5}$ and personal CO among fish smokers were higher as compared to WHO recommended standards. Fish smokers who cooked outdoors had lower concentrations for both personal PM $_{2.5}$ and personal CO compared to those who cooked in the house and in a separate building.

There were no correlations between most of the urinary PAHs and personal CO as well as personal PM $_{2.5}$. However, there were correlations between personal CO and the following compounds in urine samples of fish smokers; 2-chlorophenol (r= 0.032, *P*= 0.004), 1,3-dichlorobenzene (r= -0.24, P= 0.0091), and 1,4-dichlorobenzene (r= 0.18, P= 0.047) 4-chloro-3-methylphenol (r= 0.28, *P*= 0.002), 2,4,5-trichlorophenol (r= 0.20, *P*=0.03) and 1,4-Dinitro-benzene (r=0.19, *P*=0.03), Flourene (r= 0.20, *P*=0.03), 4-bromophenylether (r= 0.19, *P*=0.03), benz[a]anthracene (r= 0.22, *P*= 0.02), Benzo[a]pyrene (r= 0.22, *P*= 0.02), 1,2-Dinitrobenzene (r= 0.19, *P*= 0.03).

Also, there were correlations between personal PM $_{2.5}$ and the following compounds in urine of fish smokers; 1,2-dimethylbenzene (r= 0.20, *P*= 0.03, bis(2-chloroethoxy) methane (r= 0.30, *P*<0.001), 2,4-dichlorophenol, (r= 0.19, *P*= 0.04)., 4-chloro-3-methylphenol r= -0.21, *P*= 0.002), acenaphthene (r= 0.19, *P*= 0.04), and dibenzfuran (r= 0.22, *P*= 0.03), 2,3,4,5-tetrachlorophenol (r=0.40, *P*= 0.001), Pyrene (r= -0.22, P= 0.02) and Benz[a]anthracene (r= -0.22, P= 0.02), benzo[a]pyrene (r= -0.23, P= 0.01). In addition, there were correlations between PM $_{2.5}$ and urinary 9-phenanthrenol (r= 0.24, P= 0.007) urinary 1-hydroxypyrene (r= 0.23, *P*= 0.01). However, no significant relationship was observed between the correlation of personal CO and urinary 1-hydroxypyrene and urinary 9-phenanthrenol.

Recommendations

The findings in this work have led to the identification of some key areas that require further research and some recommendations which include the following:

1. Most fish smokers bring their younger children to the indoor smokehouses; the effect of smoke exposure on children should be assessed.
2. The types of ovens used by fish smokers could not filter smoke effectively. There should be research into production of affordable but efficient ovens for fish smokers.
3. The government of Ghana through Ministry of Fisheries should support the gas smokehouse at Abuesi.
4. Non-governmental organisations should engage the fish smokers to provide personal protective equipment, workshops on proper and hygienic ways of handling fish and safety practices in the smokehouses.
5. Most of the fish smokers used biomass fuel to cook in addition to using biomass to smoke fish, policy makers and nongovernmental organisations should assist fish smokers with cleaner fuels and stoves that could filter smoke effectively.
6. Policy makers should support researchers with funding to extend the research to a larger number of fish smokers. This would give a broader picture of the findings to inform policies among fish smokers in Ghana.

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APPENDICES

APPENDIX A

QUESTIONNAIRE FOR FISH SMOKERS IN ABUESI

QUESTIONNAIRE

SCHOOL OF PHYSICAL SCIENCES, UNIVERSITY OF CAPE COAST

DEPARTMENT OF CHEMISTRY

AIR POLLUTION MONITORING AMONG FISH SMOKERS IN ABUESI, WESTERN REGION.

Interviewer

This an academic research on air pollution monitoring among fish smokers in Abuesi, Western Region being undertaken by student of the above institution. This exercise is strictly for academic purpose and respondent is fully assured that any information given will be treated as confidential.

Thanks for your cooperation.

Section A: Socio demographic characteristics of fishsmoker

1.	Age		
2.	Marital status	Unmarried Married Divorce Co-habitation Widow	1 2 3 4 5
3.	Highest level of education	Non-formal Primary Junior high/Middle School Senior High/ Secondary Vocational/Technical Tertiary	0 1 2 3 4 5
4.	Religion	Christian Moslem Traditional Other (specify)	1 2 3
5.	Number of years spent		

	Smoking		
6.	Distance from residence to the smokehouse		
8.	Number of children below 5 years who come to the smokehouse		
9.	How often do the children come to the smokehouse	Sometimes Very often Always	1 2 3
10.	Number of people working in a smoke house		
11.	Do you do any work aside fish smoking		

Section B: Type of biomass fuel used by fish smoker

12.	Type of fuel used for smoking	Firewood Charcoal LPG Others (Specify)	1 2 3
13.	Type of firewood	Sugar cane Palm kernel Cocoa Acacia Others (specify)	1 2 3 4
14.	Type of material used for stove Construction	Mud Cement Wood Stone Others (specify)	1 2 3 4
15.	Which type of firewood do you prefer?		
16.	What is the reason for your choice?		

Section C: Awareness, perception and knowledge of fish smokers about effect of smoke on their health

17.	Are you aware high exposure to smoke could affect your health?	No Yes	0 1
18.	Are you aware high exposure to heat could affect your health	No Yes	0 1
19.	Have you ever encountered any health hazards as a result of fish smoking?	No Yes	0 1
20.	If yes, what was the symptom?	Headache Cough Phlegm/ wheezing Breathlessness Respiratory (chest cold, chest illness) Eye disease (redness) Asthma Pneumonia Others (specify)	1 2 3 4 5 6 7 8

Section D: Safety precautions practiced by fish smokers

21	Do you use any safety clothing during fish smoking?	No Yes	0 1
22	Which of these safety materials do you use during smoking? Safety googles Safety boots Hand gloves Protective clothing Others (specify)	 No Yes No Yes No Yes No Yes	 0 1 0 1 0 1 0 1
23	Do you practice any safety measures during smoking?	No Yes	0 1
24	If yes (specify)		

APPENDIX B

PARAMETERS OF AIR SAMPLERS

Sensors ID: MICS_2614_O3_1-0034, MICS_5524_CO_1-0044, Shinyie_1-0054, Temp/RH_DHT22_1-0074, SD Card_1-0084) for monitor 2, Sensors ID: MICS_2614_O3_1-0035, MICS_5524_CO_1-0045, Shinyie_1-0055, Temp/RH_DHT22_1-0075, SD Card_1-0085) for monitor 3, Sensors ID: MICS_2614_O3_1-0036, MICS_5524_CO_1-0046, Shinyie_1-0056, Temp/RH_DHT22_1-0076, SD Card_1-0086) for monitor 5, Sensors ID: MICS_2614_O3_1-00040, MICS_5524_CO_1-00050, Shinyie_1-00060, Temp/RH_DHT22_1-00080, SD Card_1-00090) for monitor 6, Sensors ID: MICS_2614_O3_1-0038, MICS_5524_CO_1-0048, Shinyie_1-0058, Temp/RH_DHT22_1-0078, SD Card_1-0088) for monitor 7 and Sensors ID: MICS_2614_O3_1-0033, MICS_5524_CO_1-0043, Shinyie_1-0053, Temp/RH_DHT22_1-0073, SD Card_1-0083).

APPENDIX C

ASSOCIATION BETWEEN TYPE OF FUEL AND SELF-REPORTED HEALTH OUTCOME

			Type of firewood					
			sugarcane	Palm kernel	cocoa	Acacia	others	Total
Headache	No	Count	1_a	0_a	14_b	21_c	69_b	105
		Expected Count	10.3	3.6	16.0	11.0	64.1	105.0
	Yes	Count	28_a	10_a	31_b	10_c	111_b	190
		Expected Count	18.7	6.4	29.0	20.0	115.9	190.0
Total		Count	29	10	45	31	180	295
		Expected Count	29.0	10.0	45.0	31.0	180.0	295.0

Each subscript letter denotes a subset of type of firewood categories whose column proportions do not differ significantly from each other at the .05 level.

APPENDIX D

ASSOCIATION BETWEEN DISTANCE OF SMOKEHOUSES AND SELF- REPORTED HEALTH OUTCOME

	Cases					
	Valid		Missing		Total	
	N	Percent	N	Percent	N	Percent
Headache * distance from residence to the smokehouse	295	97.4%	8	2.6%	303	100.0%
Cough * distance from residence to the smokehouse	295	97.4%	8	2.6%	303	100.0%
Phlegm/wheezing * distance from residence to the smokehouse	295	97.4%	8	2.6%	303	100.0%
Breathlessness * distance from residence to the smokehouse	295	97.4%	8	2.6%	303	100.0%
Respiratory(chest cold, chest illness) * distance from residence to the smokehouse	295	97.4%	8	2.6%	303	100.0%
Asthma * distance from residence to the smokehouse	295	97.4%	8	2.6%	303	100.0%
Pneumonia * distance from residence to the smokehouse	295	97.4%	8	2.6%	303	100.0%

Headache * distance from residence to the smokehouse

			Distance from residence to the smokehouse				
			.0	close	far	18.0	Total
Headache	No	Count	$0_{a, b}$	75_{b}	30_{a}	$0_{a, b}$	105
		Expected Count	.4	87.2	17.1	.4	105.0
	Yes	Count	$1_{a, b}$	170_{b}	18_{a}	$1_{a, b}$	190
		Expected Count	.6	157.8	30.9	.6	190.0
Total		Count	1	245	48	1	295
		Expected Count	1.0	245.0	48.0	1.0	295.0

Each subscript letter denotes a subset of distance from residence to the smokehouse categories whose column proportions do not differ significantly from each other at the .05 level.

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	18.916[a]	3	.000	.000
Likelihood Ratio	18.783	3	.000	.000
Fisher's Exact Test	18.011			.000
N of Valid Cases	295			

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .36.

Cough * Distance from residence to the smokehouse

			Distance from residence to the smokehouse				
			.0	close	far	18.0	Total
Cough	no	Count	$1_{a, b}$	128_{b}	36_{a}	$0_{a, b}$	165
		Expected Count	.6	137.0	26.8	.6	165.0
	yes	Count	$0_{a, b}$	117_{b}	12_{a}	$1_{a, b}$	130
		Expected Count	.4	108.0	21.2	.4	130.0
Total		Count	1	245	48	1	295
		Expected Count	1.0	245.0	48.0	1.0	295.0

Each subscript letter denotes a subset of distance from residence to the smokehouse categories whose column proportions do not differ significantly from each other at the .05 level.

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	10.489[a]	3	.015	.004
Likelihood Ratio	11.662	3	.009	.004
Fisher's Exact Test	10.541			.004
N of Valid Cases	295			

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .44.

Phlegm/wheezing * Distance from residence to the smokehouse

			Distance from residence to the smokehouse				
			.0	close	far	18.0	Total
Phlegm/ wheezing	no	Count	$1_{a, b}$	171_{b}	41_{a}	$1_{a, b}$	214
		Expected Count	.7	177.7	34.8	.7	214.0
	yes	Count	$0_{a, b}$	74_{b}	7_{a}	$0_{a, b}$	81
		Expected Count	.3	67.3	13.2	.3	81.0
Total		Count	1	245	48	1	295
		Expected Count	1.0	245.0	48.0	1.0	295.0

Each subscript letter denotes a subset of distance from residence to the smokehouse categories whose column proportions do not differ significantly from each other at the .05 level.

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	5.679[a]	3	.128	.106
Likelihood Ratio	6.731	3	.081	.060
Fisher's Exact Test	5.895			.070
N of Valid Cases	295			

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .27.

Breathlessness * Distance from residence to the smokehouse

			Distance from residence to the smokehouse				
			.0	close	far	18.0	Total
Breathlessness	no	Count	$1_{a, b}$	195_{b}	45_{a}	$1_{a, b}$	242
		Expected Count	.8	201.0	39.4	.8	242.0
	yes	Count	$0_{a, b}$	50_{b}	3_{a}	$0_{a, b}$	53
		Expected Count	.2	44.0	8.6	.2	53.0
Total		Count	1	245	48	1	295
		Expected Count	1.0	245.0	48.0	1.0	295.0

Each subscript letter denotes a subset of distance from residence to the smokehouse categories whose column proportions do not differ significantly from each other at the .05 level.

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	5.900[a]	3	.117	.110
Likelihood Ratio	7.430	3	.059	.043
Fisher's Exact Test	6.815			.075
N of Valid Cases	295			

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .18.

Respiratory (chest cold, chest illness) * distance from residence to the smokehouse

			Distance from residence to the smokehouse				
			.0	close	far	18.0	Total
Respiratory (chest cold, chest illness)	no	Count	$1_{a, b}$	117_{b}	35_{a}	$1_{a, b}$	154
		Expected Count	.5	127.9	25.1	.5	154.0
	yes	Count	$0_{a, b}$	128_{b}	13_{a}	$0_{a, b}$	141
		Expected Count	.5	117.1	22.9	.5	141.0
Total		Count	1	245	48	1	295
		Expected Count	1.0	245.0	48.0	1.0	295.0

Each subscript letter denotes a subset of distance from residence to the smokehouse categories whose column proportions do not differ significantly from each other at the .05 level.

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	12.028[a]	3	.007	.002
Likelihood Ratio	13.163	3	.004	.001
Fisher's Exact Test	12.045			.001
N of Valid Cases	295			

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .48.

Asthma * Distance from residence to the smokehouse

			Distance from residence to the smokehouse				
			.0	close	far	18.0	Total
Asthma	no	Count	$1_{a, b}$	210_{b}	47_{a}	$1_{a, b}$	259
		Expected Count	.9	215.1	42.1	.9	259.0
	yes	Count	$0_{a, b}$	35_{b}	1_{a}	$0_{a, b}$	36
		Expected Count	.1	29.9	5.9	.1	36.0
Total		Count	1	245	48	1	295
		Expected Count	1.0	245.0	48.0	1.0	295.0

Each subscript letter denotes a subset of distance from residence to the smokehouse categories whose column proportions do not differ significantly from each other at the .05 level.

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	5.858[a]	3	.119	.250
Likelihood Ratio	8.187	3	.042	.042
Fisher's Exact Test	7.615			.057
N of Valid Cases	295			

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .12.

Pneumonia * Distance from residence to the smokehouse

			Distance from residence to the smokehouse				
			.0	close	far	18.0	Total
Pneumonia	no	Count	$1_{a, b}$	213_{b}	47_{a}	$1_{a, b}$	262
		Expected Count	.9	217.6	42.6	.9	262.0
	yes	Count	$0_{a, b}$	32_{b}	1_{a}	$0_{a, b}$	33
		Expected Count	.1	27.4	5.4	.1	33.0
Total		Count	1	245	48	1	295
		Expected Count	1.0	245.0	48.0	1.0	295.0

Each subscript letter denotes a subset of distance from residence to the smokehouse categories whose column proportions do not differ significantly from each other at the .05 level.

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)
Pearson Chi-Square	5.122[a]	3	.163	.244
Likelihood Ratio	7.113	3	.068	.052
Fisher's Exact Test	6.768			.072
N of Valid Cases	295			

a. 4 cells (50.0%) have expected count less than 5. The minimum expected count is .11.