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ACRYLAMIDE EXPOSURE AND RISKS IN MOST FREQUENTLY CONSUMED FOODS IN A TOTAL DIET STUDY

A THESIS SUBMITTED TO THE DEPARTMENT OF FOOD SCIENCE AND TECHNOLOGY IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MPHIL FOOD SCIENCE AND TECHNOLOGY

BY

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DECLARATION

I hereby declare that this submission is my own work toward the award of the MPhil degree and that, to the best of my knowledge, it contains no material previously published by another person or material which has been accepted for the award of any other degree or diploma at Kwame Nkrumah University of Science and Technology, Kumasi or any other educational institution, except where due acknowledgement has been made in the thesis.

ABSTRACT

The neurotoxic and carcinogenic nature of acrylamide, coupled with the recent emphasis of the ―probable carcinogenic‖ status of acrylamide is a cause for concern requiring further studies. The objective of this study was to determine the carcinogenic and neurotoxic risks associated with the consumption of frequently consumed foods in a Total Diet Study (TDS). From a selection of 80 frequently consumed foods, the acrylamide concentrations in the foods were purified by the QuEChERS method of extraction and purification, and the concentrations of acrylamide were determined using the HPLC. Acrylamide was detected in 82% of all the foods analyzed, and the

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levels ranged from $1.33\times10^{-3} \pm 1.89$ to $14.39\times10^{-3} \pm 6.33$ mg/g. The probabilistic approach was used to model the chronic exposures using the Monte Carlo simulation of the Palisade @Risk software. The mean, 50th and 95th percentile values for acrylamide exposures were in the range of 1.56×10^{-3} to 1.88×10^{-2}, 3.21×10^{-4} to 5.85×10^{-3} and 6.16×10^{-3} to 8.32×10^{-2} mg/kg bw/day respectively. The mean and 95th percentile values for the margins of exposure (MOE) for the risk of tumorigenesis and neurotoxicity were below the thresholds, hence posing significant public health concern. Generally, the lifetime cancer risks of male consumers were higher compared to that of the female consumers. The median and 95th percentile consumers presented unacceptable risk, since their lifetime cancer risks were greater than the *de minimus* (10^{-6}). The elements that imparted the most on the overall lifetime cancer risk of the consumers were the exposure duration and the concentration of acrylamide in the foods. To lower these lifetime cancer risks, mitigation studies can thus, be mounted in order to help lower the concentrations of acrylamide in the foods.

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DEDICATION

To the Almighty God, by whose grace I have come this far.

CHAPTER ONE

INTRODUCTION

1.1 Background

The toxicity of acrylamide is well known and recent announcement from International Agency for Research on Cancer (IARC), placing acrylamide as a Group 2A carcinogen and making it a probable human cancer (IARC, 2017) has sparked another round of public discourse on the carcinogenicity of this compound.

Acrylamide is produced in many foods especially when it is baked, toasted or fried. Studies have also shown that, the sources of acrylamide remain ever present in the way we process our foods (Xu *et al.*, 2014). While substantial quantities are generated from Maillard reactions, others are produced from asparagine as the principal precursor. Other studies also show that, fats and oils deliver acrylamide usually at high temperatures via acrolein (Lingnert, 2002; Zyzak *et al.*, 2003).

In order to quantify risks associated with acrylamide in foods, total diet studies should be the way forward (Konings *et al.*, 2003; Shim *et al.*, 2014), since chronic daily intake is related to the risks associated with total food consumption. It is difficult to control the presence of acrylamide in foods, meaning consumers shall always be exposed to this hazard. It is therefore important to consider all aspects of risk indices in order to make judgement on the risks posed by the presence of this hazard. The hazard quotient (HQ) for instance, which is used to quantify the risk associated with acrylamide exposure, is defined as the ratio of the chronic human exposures to the reference dose of acrylamide (USEPA, 2001; USEPA, 2005). The margin of exposure (MOE), which is the ratio of the bench mark dose lower limit ($BMDL_{10}$) to the estimated exposure of

a hazard can also be used to evaluate the risk (EFSA, 2015). Another method is the lifetime risk, which uses the integrated product of the potency factor (PF) and human exposures of hazards (usually determined as the chronic daily intake -CDI). The potency factor (also known as the slope factor), is usually derived from institutional compendium, and is defined as the risk produced by a lifetime average dose of 1 mg/kg-day (USEPA, 2010).

In order to make a better judgement of the exposure of acrylamide in consumers, detailed food consumption data, particularly related to the most frequently consumed food must be analyzed. The United States Environmental Protection Agency (USEPA) integrates all the elements for the determination of CDI, as presented in Equation 1. Essentially, the CDI is the product of the average daily intake and the consumption level related to the exposure frequency and duration per the averaging time (USEPA, 2002). Exposure to acrylamide may be determined using a national food consumption data. However, the reliability of such data could be flawed when sub populations are the target of the study.

The exposure processes themselves are subject to biases and errors when questionnaire administrators are not properly trained (Douglass and Tennant, 1997). In order to make risks quantification consequential, the quantified values must be compared to thresholds. Institutions such as the European Food Safety Authority (EFSA) and USEPA recommend that values of HQ greater than 1 represent risks, and thus, warrant public health concern. Similarly, MOE values less than 10,000 and 125 warrants public health concern for tumorigenic and neurotoxic studies respectively. Lifetime risks studies with values greater than the recommended *de minimis* (10^{-6}) is also regarded

to imply a probable risk of developing cancer as far as acrylamide exposure is concerned (EFSA, 2015; USEPA, 2010).

Collective information from these risk indices should empower risk managers and communicators to review the status of acrylamide and recommend possible avenues to control the probable carcinogenic properties of acrylamide. The European food safety authority (EFSA) have still not set any maximum level for acrylamide in foods, because of their perception that, any minute level of exposure to a carcinogenic and genotoxic substance like acrylamide, will cause damage to the DNA, leading to cancer (EFSA, 2015). This is also partly due to the fact that, the same raw food product after processing can have variable acrylamide content (Esposito *et al.*, 2017), which makes the levels of acrylamide in foods vary significantly.

1.2 Problem statement and justification

According to the World Health Organization (WHO), more than 8.8 million died from cancer globally in the year 2015 (WHO, 2015). About 70% of these deaths were recorded from low and middle income countries including Ghana. This total number of cancer cases is expected to increase (WHO/IARC, 2008) to 15 million by 2020. In Ghana for instance, 16,600 cases of cancer are reported yearly, with about 12,700 deaths reported in 2008 (IARC, 2008), making cancer the fourth cause of death in the country. It has been documented (Doll, 1998) that, 20 – 50% of cancer cases were diet-related, hence particular attention must be given to our dietary intakes.

There is an overwhelming evidence that, acrylamide is a hazard, with the probability of causing cancer in humans (IARC, 2017). The fact that, it is not a food contaminant, but rather, forms during food processing, makes its presence in foods unavoidable (Xu

et al., 2014), thus, exposing consumers to the hazard. Since the alert of the presence of acrylamide in foods in 2002, many advanced countries have worked extensively to determine the total dietary intake of acrylamide in their foods. However, the same cannot be said about developing countries such as Ghana, where the food consumption data is even unavailable. It is also unreliable to continue making extrapolations from the risk assessments of acrylamide from these developed countries, since there are variations in the global dietary patterns, and also the foods that contribute acrylamide intake differ from country to country (Mucci and Wilson, 2008). There is therefore the need to determine if the concentrations of acrylamide in frequently eaten Ghanaian foods are enough to pose a cancer risk or any related

toxicities.

1.3 Objectives

The study sought to determine the carcinogenic and neurotoxic risks associated with the consumption of the frequently consumed foods.

1.3.1 Specific objectives

Specifically, the project sought to determine the food consumption data within the Kumasi metropolis, from which the

- dietary acrylamide exposures and
- the three risk indices (HQ, MOE, lifetime risk) across the day and also among sections of the consumers were evaluated.

CHAPTER TWO

LITERATURE REVIEW

2.1 Chemistry and properties of acrylamide

Acrylamide (2-propeamide, acrylic amide, ethylene carboxamide) is a white, odorless, crystalline solid, with a molecular formula and mass of C_3H_5NO and 71.08 respectively. It has a boiling point of 136 °C, a melting point of 84.5 °C, and a vapor pressure of 0.007 mmHg at 25 °C (Arribas-Lorenzo and Morales, 2012). Acrylamide has been used for ages as a chemical intermediate in polyacrylamides production, mainly because it is highly reactive (Borda and Alexe, 2011), and soluble in a number of solvents, including; ethanol, chloroform, ether and water. It also decomposes in the presence of acids, bases, oxidizing agents and iron salts
(Friedman, 2003).

The polyacrylamides produced from acrylamides are used in the textile, paper and cosmetic industry, and also as a flocculants during water treatment (Claeys *et al.*, 2010). Apart from their industrial applications, polyacrylamide gels are also applied during protein separation by electrophoresis in many research laboratories. There have also been reports of their presence in the smoke of cigarettes (IARC, 1994; JECFA, 2005). The presence of acrylamide in food is however, of serious concern because it is a hazard, and thus, ―has the potential to cause adverse health effects upon exposure‖ (Strachan, 1984). It has been reported that, every hazard poses risk, which is explained as ―the estimate of the probability of the occurrence of the adverse health effects and its severity‖ (CCFH, 1998).

2.2 Toxicity of acrylamide

Many consumers are health conscious and are keen to follow the public discourse on carcinogenicity. The seriousness of the threat, engage the attention of research scientists on efforts to mitigate the presence of these hazards in foods (FAO/WHO,

2002). Recently, the International Agency for Research on Cancer (IARC, 2017) reviewed the profile of acrylamide, and classified it as Group 2A carcinogens—probable human carcinogen‖. This means, there is enough evidence that shows that, acrylamide causes cancer in laboratory animals, but with little and ongoing studies of its carcinogenicity in humans. The review by IARC and the European Union was based on evidence that strongly suggested that, acrylamide goes through phase I metabolism to produce a genotoxic hazard (glycinamide), which occur in both test animals and human beings (IARC, 1994; EC, 2002). The European Chemicals

Agency (ECHA) also added acrylamide to the European Union‘s list of substances of —very high concern‖, due to the many deleterious effects it causes (ECHA, 2009).

2.3 Acrylamide in foods

Currently, most of the foods consumed by humans are heat processed, using such methods as; baking, toasting and frying. Acrylamide has been reported to be present in many foods such as breakfast cereals (Tareke *et al.,* 2002), as well as in heat processed meat and fish (Krishnakumar and Visvanathan, 2014). Potato chips (Gaikwad *et al.,* 2016), biscuits (Eerola *et al.,* 2007) and baby foods (EFSA, 2015) have all been reported to contain acrylamide. Studies have shown that, some acrylamide and its related products may be produced from variables such as proteins undergoing heat treatment (Yaylayan and Stadler, 2005). The study explain that the amino acid, asparagine, is the principal precursor of acrylamide formation and that, it reacts with the carbonyl group of reducing sugars through Maillard reaction, to initiate the formation of acrylamide (Mottram *et al.*, 2002; Stadler *et al.*, 2002). Maillard reaction is known to occur at high temperatures, accompanied by very low moisture

conditions (Tareke *et al.*, 2002; Yaylayan *et al.*, 2003; Semla *et al.*, 2017). It is during these high temperature processing that high quantities of acrylamides are formed (Hoenicke and Gatermann, 2005; Lantz *et al.*, 2006). Heat treated processes such as frying, baking, toasting and roasting, are known to generate high levels of acrylamide contrary to boiling of foods, where low or non-detectable levels of acrylamide are reported (Friedman, 2003). However, this information has been challenged by other studies that reported of acrylamide in boiled starch (Ezeji *et al.*, 2003).

Other variables that affect acrylamide formation in foods have been reported to include; pH (Jung *et al.*, 2003), duration for heating (Taubert *et al.*, 2004) and the presence of components that bind water (Rydberg *et al.*, 2003). These variables together with the quantity of asparagine and reducing sugars (fructose and glucose), determine the total amount of acrylamide that are produced (Lukac *et al.*, 2007). Acrylamide formation through Maillard reaction is regarded as the major pathway in foods (Zyzak *et al.*, 2003). Acrolein, a primary precursor of acrylamide reacts with residual amines to form acrylamide. The acrolein itself, is believed to form by dehydration from glycerol, when the long chain fatty acids are decomposed from it (Antal *et al.*, 1985; Lingnert *et al.*, 2002).

Some studies suggest that, the acrylamide content of foods has links to the incidences of acrylamide related diseases (Hogervorst *et al.*, 2007; Hogervorst *et al.*, 2008). Though, Wilson *et al.* (2010) countered that there was no strong correlation between the presence of acrylamide and disease endpoints, gradual support appears to be rising (Hirvonen *et al.*, 2010).

In order to arrive at a reasonable exposure of acrylamide in foods, it is better if food consumption data based on total diets of consumers were evaluated, using the appropriate exposure assessment methods. In this way, a reliable risk estimate of the genotoxicity or systemic toxicity of the consuming public could be evaluated. The quantitative risk assessment process is a strong tool of choice for estimating probabilistic risk because, the method quantifies the totality of risks including their uncertainties (Kettler *et al*., 2015).

There are three approaches to determine food systems risks. One approach, the probability-based food systems risks, can be used to quantify a lifetime risk. This method uses the integrated product of the potency factor (PF) and human exposures of hazards (usually determined as the chronic daily intake -CDI). Regulatory institutions, such as US Environmental Protection Agency (USEPA, 2010), define potency factor as the _risk produced by a lifetime average dose of 1 mg/kg-day‘. Another method, which is the hazard quotient (HQ) approach, is the _ratio of the human exposures to the reference dose‘ (R_fD) (USEPA, 2001; USEPA, 2005). The hazard quotient and the life time risk methods are applied when quantifying systemic risks (non-cancer) and genotoxic risk (cancer) respectively. There is also the margin of exposure (MOE) approach, which is a method used to assess whether exposures to hazards, present a public health concern or not. The margin of safety is defined by the European Food Safety Authority (EFSA, 2015) as the _ratio of the bench mark dose lower limit ($BMDL_{10}$) to the estimated exposure of a hazard‘.

2.4 Acrylamide risk analysis process

Acrylamide is hazardous and one major route of formation is through the Maillard reaction which gives the golden brown color, flavor or aroma of many baked, fried or toasted foods (Xu *et al.*, 2014). The carcinogenicity resulting from the genotoxicity of acrylamide, continue to place this toxicant in the priority list of scientists. Many studies have suggested that there may be an epidemiological threat in terms of association of cancer and the diet consumed (Erdreich and Friedman, 2004;

Hogervorst *et al.*, 2007; Konings *et al.*, 2010; Bongers *et al.*, 2012; Chen *et al.*, 2012; Lujan-Barroso *et al.*, 2014; Hogervorst *et al.*, 2014). However, no clear association and mechanisms have been outlined. The need arises to continue to monitor the risk analysis process of acrylamide, involving; its hazard identification, characterization, exposure assessment and risk quantification.

2.4.1 Hazard identification

Since the Swedish team of the University of Stockholm in April, 2002 broke the news on the presence of acrylamide in foods (SNFA, 2002; Tareke *et al.*, 2002), many studies have focused on acrylamide. The UK Food Standards Agency (FSA) for instance, immediately studied and published data on acrylamide in foods, which were in agreement with that of the Swedish National Food Administration (FSA, 2002). It is believed that, public outcry demanded confirmation because, many foods consumed by the general public are usually treated to temperatures above 120 ˚C (Törnqvist, 2005). While Törnqvist (2005) reported of such temperatures as prerequisite for acrylamide formation, other studies have however, reported of lower temperatures for acrylamide formation in specific products (Biedermann-Brem *et al.*,

2003; Amrein *et al.*, 2004; Granvogl *et al.*, 2004).

The formation of acrylamide in foods is summarized under three main stages (Lingnert *et al.*, 2002), in which first stage involves the condensation of the carbonyls of reducing sugars with the free amino acid (asparagine). The series of reactions eventually lead to the formation of an Amadori compound (Stadler *et al.*, 2004; Granvogl and Schieberle, 2006). The second stage (Strecker degradation stage), shows the degradation of the Amadori products into a number of flavor chemicals and other degraded products. Among such degraded products include; carbon dioxide, ammonia and aldehydes, specifically acetaldehyde, formaldehyde and propenaldehyde (acrolein) (Arvanitoyannis and Dionisopoulou, 2014). The third stage involves the formation of acrylamide from the reaction between propenaldehyde and ammonia.

Acrolein (2-propenal, propenaldehyde) can also form from the transformation of fats and oils, where at high temperatures above the smoke point of oils, these oils are hydrolyzed into glycerol and fatty acids (Umano and Shibamoto, 1987). The glycerol is further degraded to acrolein by the removal of water through an acid catalyzed mechanism (Lingnert *et al.*, 2002). The acrolein or its derivative, acrylic acid, can further react with ammonia (liberated from amino acids) to form acrylamide (Becalski *et al.*, 2003; Keramat *et al.*, 2011). The acrolein pathway is known to be the minor pathway for acrylamide formation in foods (Ehling *et al.*, 2005; Zhang *et al.*, 2005).

2.4.1.1 Absorption and distribution

Studies have shown that, acrylamide is soluble in water, ethanol and acetone (Zhang and Zhang, 2007), thus, it is fairly distributed (Sirot *et al.*, 2012) and excreted with

virtually no bioaccumulation in the human body. Their rapid absorption and elimination following exposure, means acrylamide has a very short half-life of 3.1 – 3.5 h (Fennell *et al.,* 2006) as it goes through biotransformation. However, it is by this rapid absorption and elimination properties of acrylamide, that ensures it is present in fetuses and breastmilk of pregnant animals (JECFA, 2011). Acrylamide is eliminated from the human system via the urine in the form of mercapturic acid conjugates (Friedman, 2003).

2.4.1.2 Biotransformation

The metabolism of acrylamide have been studied extensively and is believed to involve cytochrome P_{450} (CYP 2E1) and glutathione-S-transferase (GST) (Kadry *et al.,* 1999; Barber *et al.*, 2001; Xu *et al.*, 2014). The conjugation of acrylamide with glutathione (GSH), catalyzed by the GST serve as the major pathway for the metabolism and excretion of acrylamide as urinary metabolites. On the other hand, acrylamide can undergo epoxidation to form an epoxide; 2,3-epoxypropionamide (glycidamide), which is the main metabolite formed under the mediation of CYP 2E1 through the phase I (Sumner *et al.*, 1999; Virk-Baker *et al.*, 2014). It is this metabolic activation pathway, which serves as the major route underlying the toxicity of acrylamide, since the oxidized metabolite is even more toxic (Xu *et al.*, 2014).

Studies have shown that, acrylamide usually goes through the epoxidation pathway in situations where GSH levels in the body is low. This condition could result from the consumption of diets containing low levels of sulphur amino acids (methionine and cysteine), essential for the synthesis of GSH (Khanna *et al.*, 1988; Khanna *et al.*, 1992). Liver damage could also result in low GSH levels because, the human liver is expected

to maintain a GSH concentration of (3 to 5) $\times 10^{-3}$ mmol/g of liver wet weight (Mulders *et al.,* 1992).

Two major adverse health effects; carcinogenicity and systemic toxicity, have been put forward by the UK FSA (FSA, 2002) and the Scientific Committee on Food (SCF 2002), stemming from the genotoxicity of the glycidamide and acrylamide (Ghanayem *et al.*, 2005). Firstly, with the neurotoxic and genotoxic features of acrylamide, its exposure to humans and animals have led to gene mutation and DNA damage, leading to carcinogenicity (Mojska *et al.,* 2010). A number of animal models have established acrylamide and its metabolite, glycidamide, as a multi-organ carcinogen (Virk-Baker *et al.,* 2014). Studies have also shown the potential toxicities and their end response in humans including such devastating illness such as cancer of the brain (Hogervorst *et al.*, 2009a), lung (Hogervorst *et al.*, 2009b) and gastrointestinal tract (Mucci *et al.,* 2006). Olesen *et al.* (2008) and Hogervorst *et al.* (2008) have argued in favor of a positive correlation between the dietary intake of acrylamide and the risk of breast, kidney cancer and endocrine tumors. This is evident with the appearance of tumors on certain organs (thyroid, uterus, adrenal and mammary gland) of laboratory animals exposed to this toxicant (Mojska *et al.*, 2012).

Studies have shown that, the nervous system is the primary site for acrylamide toxicity (LoPachin, 2005), and that, the neurotoxicity or damage to the nervous systems resulting from acrylamide exposure is cumulative. This means, short term or sub chronic exposure results mostly in numbness of limbs, tingling and ataxia, which can lead to the impairment of copulatory functions. Chronic exposure on the other hand results in more adverse effects such as cerebellar dysfunction and neuropathy (He *et*

al., 1989; Pennisi *et al.*, 2013). These effects leading to neuropathy stems from the ability of acrylamide to target especially the sulphur amino acids of proteins in the neurons (Friedman, 2003), as well as suppress the incorporation of amino acids into proteins in the nervous system.

The bifunctional structure of acrylamide (alkene and amide) could be another possible reasoning behind its neurotoxicity. Friedman (2003) has explained that, as the alkene part of acrylamide undergo hydrophobic interactions, the amide part also interact through hydrogen bonding with cell components respectively. It is these properties that speed up their diffusion and penetration into the terminal sites of the nerves and interrupts the normal functioning of the nervous system and cell membranes. Friedman (2003) further explained that, the hydrophilic nature of acrylamide affords it the ability to diffuse passively throughout the entire body. This makes every tissue in the human system a target for acrylamide carcinogenesis (Ghanayem *et al.*, 2005). There have been reports of a direct relationship between neurotoxicity and reproductive toxicity resulting from acrylamide exposure (Hagmar *et al.*, 2001). Here, the toxicity of the reproductive system, like decreased and abnormal sperm count in males, and reduced litter size of pregnant females of rats have also been reported after acrylamide exposure (Shipp *et al.*, 2006; Lineback and

Jones, 2011).

The second effect is that of systemic toxicity. Here, there is a suggestion of erectile dysfunction resulting from damaged nerves in the sexual organs (Erkekoğlu and Baydar 2010). There is still the suggestion that, the locomotion instruments of the sperm cells are also damaged as a result of the sulfhydryl groups of kinesin motor

proteins not able to function well when the levels of GSH is low (JECFA, 2002). Though the exact mechanisms are not fully understood, it appears when the level of glutathione is depleted, this prompts proteins/amino acids/enzymes involved in nerve transmissions to step in and react with glycidamide through their sulfhydryl cysteine, thereby rendering these kinesin proteins ineffective (Erkekoğlu and Baydar 2014).

There have been reports on the close association between acrylamide and the red blood cells, where acrylamide and its metabolite, glycidamide binds to hemoglobin's amino acids in the red blood cells (Schettgen *et al.*, 2003; Fennell *et al.*, 2004). The acrylamide and glycidamide exposure of the US population was studied by Vesper *et al.* (2010) by measuring their levels of hemoglobin adducts of acrylamide (HbAA) and glycidamide (HbGA). Lingnert *et al.* (2002) have reported that, hemoglobin acrylamide adduct acts as a biomarker, as it is one way used for measuring humans' dietary exposure to acrylamide.

Other effects of acrylamide and glycidamide in the human system include oxidative stress (Yousef and El-Demerdash, 2006; Jiang *et al.*, 2007), resulting from glutathione depletion and aneuploidy, where acrylamide bind to the proteins involved in cell division (Sickles *et al.*, 2007; Hogervorst *et al.*, 2010). The most frustrating thing about these health effects is that, it affects the unborn child. Many experimental studies have revealed that, the placenta acts as the medium for the exposure of the fetuses (Annola *et al.*, 2008). For instance, Schettgen *et al.* (2004) reported of HbAA adducts inside the blood of the umbilical cord, when they performed a pilot study on the exposure of neonates to acrylamide though the placenta of 11 pregnant women. In a report by Sörgel *et al.* (2002), 10 - 50% of dietary acrylamide was transferred through the blood

via the placenta to the fetus of pregnant women, with the breast milk of these women containing up to 18.8×10^{-3} mg/L of acrylamide. They also reported of the ability of acrylamide to damage the brain, because of its potential to cross the blood-brain barriers. A research by Srivastava *et al.* (1986) also revealed acrylamide‘s potential to reduce the levels of the GSH in the brain and to suppress the action of the brain‘s GST. In this way, epoxidation of acrylamide to glycidamide occurs due to the low levels of both the GST and GSH.

2.4.2 Exposure assessment

The exposure of acrylamide must be quantified in order to proceed to reliably estimate the risk. Reports show that, the major route for humans‘ exposure to acrylamide is through food (Boon *et al.*, 2005). The total dietary exposure to acrylamide depends on the acrylamide content in the food, the quantity consumed and the frequency of consumption of the food (Virk-Baker *et al.*, 2014). The Food and Agriculture Organization/World Health Organization (FAO/WHO, 2002) have recommended that, the mean and 95th percentile dietary exposure to acrylamide be set in the range of 0.2×10^{-3} to 1.0×10^{-3} mg/kg bw/day and 0.6×10^{-3} to 1.8×10^{-3} mg/kg bw/day respectively (JEFCA, 2006; Zhou *et al.*, 2013).

Basically, exposure assessment has been defined as ―the concentration of the hazard per body weight of consumers‖ (USEPA, 2002). Indeed, regulatory institutions such as the Codex Alimentarius Commission (CAC) define exposure assessment as ―food consumption data and the concentration of the food toxicant in food‖ (Codex, 1989).

The dietary exposure has been simplified in the general expression in Equation 2.1.

$$\text{Dietary exposure} = \frac{\text{Hazard concentration} \quad \text{Food consum}}{\text{Body weight}} \times \text{ed} \quad \text{.....2.1}$$

The evaluation of exposure assessment has been achieved through a number of approaches. In order to arrive at a reliable food consumption data, the detailed food consumption patterns and concentrations of specific foods containing acrylamide must be determined (Dybing *et al.*, 2005; Kearney, 2010). Different studies have used different methods for this purpose, including the stepwise approach of the WHO tiered systems (Fransen *et al.*, 2010). Generally, the more meticulous the procedure for estimating the concentration of the hazard and the food consumption data, the higher the cost. There are inherent unavoidable challenges when any of the survey methods are used. Such difficulties border on the validity of the information collected from consumers, rather than on the analysis of chemical concentration of the hazards.

Total diets study remain one good approach through which the total exposure of acrylamide can be quantified, and from which a reliable exposure of acrylamide and risk of consumers can be estimated. For exposure assessment, the food frequency questionnaire (FFQ) is the method of choice for reasons such as; measuring habitual long-term dietary exposure and cost (Konings *et al.*, 2003; Shim *et al.*, 2014) .

A couple of studies have shown similar trends for determining the total diets. Many of these studies show a reliable national food consumption data, that present types and quantities of food groups consumed as breakfast, lunch and supper for an individual in those study areas (Claeys *et al.*, 2010; Bongers *et al.*, 2012; Sirot *et al.*, 2012). For instance, in their studies to estimate the exposures of dietary acrylamide in UK and Ireland, Mills *et al.* (2008) estimated exposures of the hazard using the UK Food

Standards Agency and the Food Safety Authority of Ireland national database. Acrylamide exposure was then estimated depending on their consumption patterns and food groups determined in the study area. For such studies, low limit of detection (LOD) are used in quantifying acrylamide levels in these food groups, simply because they expect low levels of acrylamide in foods. On the other hand, there is paucity of data for study areas where there are no reliable national food consumption data. For such areas, it should be possible to determine the localized total diet, using the appropriate survey tool to study the total mix of foods consumed throughout the day.

Exposure assessments are best described as probabilistic estimates, due to inherent problems such as the validity of the data collected from individuals. Acrylamide dietary exposure have been estimated using statistical modelling methods such as Monte Carlo simulation (Mills *et al.*, 2008; Cummins *et al.*, 2009). From such probabilistic methods, variable percentiles such as 95^{th}, 97.5^{th}, 99^{th} and sometimes 99.9^{th} are used to describe high exposure of hazards and risks. On the other hand, low exposures of hazards are described with 5^{th} percentiles whereas average exposures are described with the 50^{th} percentiles or the median (Konings *et al.*, 2003; Hirvonen *et al.*, 2010). Many authors have reviewed methods of exposure assessment of foods and concluded that, the use of food frequency questionnaire (FFQ) is the best method because it offers the habitual dietary habits of consumers (Shim *et al.*, 2014). However, there have been reports of uncertainties of the food consumption data surveys (Douglass and Tennant, 1997). Such issues, resulting from tiredness or fatigue, include biases either from the side of the questionnaire administrators or from the consumers. Thus, it is no surprise that uncertainties in acrylamide risks are present (Amrein *et al.*, 2003; Halford *et al.*, 2012). Other sources of uncertainties of acrylamide content in

foods is believed to be as a result of the genetic makeup of the food material as well as the various processing protocols (Bhaskar *et al.,* 2010; Ye *et al.,* 2010). Uncertainties in acrylamide risk analysis have also been traced to analytical methods and statistical evaluations (Becalski *et al.*, 2003; Hedegaard *et al.*, 2007). In all these cases, the EFSA Scientific Committee has given guidelines as to how to use iterations to better quantify these uncertainties (EFSA SC, 2018).

2.4.3 Hazard and risk characterization

Hazard characterization generally involves the dose-response study of a hazard and the profile characterizes the adverse health effects (Xu *et al.*, 2014). Risk characterization on the other hand seeks to compare the toxicity levels of the hazard with exposure doses to ascertain if risk would be implicated. Generally, the assessment of human risk acrylamide exposure is usually made when the results from animal studies are extrapolated to human epidemiological studies (Hogervorst *et al.*, 2010), on the basis of the chronic carcinogenicity assay of these animals. Different studies use different approaches to study human risk and health concern associated with acrylamide exposure.

The margin of exposure (MOE) approach, for instance, seem to be at center of the characterization of the exposure or level of safety of acrylamide in foods (Sirot *et al.*, 2012), without necessarily quantifying the lifetime risk based on the slope factor (EFSA, 2015). The ―margin‖ stands for the _safety buffer‘ between the dose of the disease endpoints or adverse effects from the test animals, and human exposure. The preference for this approach stems from EFSA‘s decision not to set a reference dose (R_fD) for acrylamide in foods (WHO, 1996; WHO, 1998), probably because it may not be possible to control acrylamide in food due to the ubiquitous nature of the hazard.

The WHO and European Union have however proceeded to set a legal acrylamide maximum level in drinking water as 0.5×10^{-3} mg/L (WHO, 2008) and 1×10^{-3} mg/L (EC, 1998) respectively. EFSA (2015) does not agree with this maximum level, because they argue that, any minute quantities of exposure to a carcinogenic substance such as acrylamide, could lead to genotoxicity and subsequently to carcinogenesis.

They have, however, recommended and predetermined an estimated dose, known as the Benchmark Dose (BMD). EFSA (2015) argued that, it would be better to accept a pre-determined *de minimus* risk in human exposure compared to a subjective reference dose. The Benchmark Dose Lower Confidence Limit ($BMDL_{10}$), is ―the minimum dose of a hazard that produces a clear, low level disease end point, usually in the range of a 1-10% of consumers‖. Subsequently, EFSA scientists have set a $BMDL_{10}$ of 0.17 and 0.43 mg/kg bw/day for tumorigenesis and neurotoxicity in humans respectively. Thus, researchers can now point out the ―level of health concern‖, referred to as the margin of exposure, by comparing test animals $BMDL_{10}$ to human dietary exposure to acrylamide (JECFA, 2005).

The Scientific Committee of the European Food Safety Authority (EFSA, 2015) considers an MOE value of 10,000 or higher for tumorigenesis to be of low concern for the health. Similarly, neurotoxicity value is set at 125 or higher. Thus, the higher the MOE, the lower the risk of exposure (Claeys *et al.*, 2010). In place of the $BMDL_{10}$, the no-observed-adverse-effect-level (NOAEL) has also been used in evaluating the MOE (JECFA, 2011).

2.5 Acrylamide risk management

Risk management is a platform for dissemination and subsequent control of risk. It thrives when research scientists provide strategies to mitigate hazards. Acrylamide exposures in foods have been characterized from which guidelines have been developed. Reports have been made of such mitigation measures by the Confederation of the European Food and Drink Industry (CIAA) (Borda and Alexe, 2011). These guidelines include agronomical, biotechnological, use of additives and conventional process parameters to control acrylamide in foods. Agronomical control measures focus on the reduction of precursors of acrylamide such as asparagine and reducing sugars in the raw food. It is believed that soil fertility profile resulting from fertilizer application can impart immensely on these precursors in the raw food product (Adebo *et al.*, 2017). Studies show that, increasing the sulphur levels, while decreasing the nitrogen levels in the soil can help to greatly reduce the levels of asparagine in the food product (Halford *et al.*, 2012; Stojanovska and Tomovska, 2015).

The biotechnological factors on the other hand, focus on application of enzymes such as asparaginase to pre-treat and ferment foods to remove specifically, asparagine (Anese *et al.*, 2009). It has also been reported that, fermenting yeasts is capable of devouring some 60 to 90% of free asparagine in cereal products (Claus *et al.*, 2008). It is also reported that, fermentation time of about 1 h reduced the acrylamide levels significantly in dough during bread making (Fredriksson *et al.*, 2004). Again, lactic acid fermentation and blanching of potatoes before deep frying, has been reported to produce between 79 – 94% reduction of acrylamide levels (Baardseth *et al.*, 2006; Anese *et al.*, 2009). Addition of asparaginase to some biscuits reduced the acrylamide levels from 0.4 mg/kg to 0.17 mg/kg (Borda and Alexe, 2011).

The conventional process parameters affecting acrylamide formation in foods include; heating temperature, relative humidity, as well as the duration of the heating process. There have been reports of low acrylamide levels in bakery products that were produced when the relative humidity was high (Ahrné *et al.*, 2007; De Vleeschouwer *et al.*, 2007). In another study, the acrylamide concentration was increased from 0.265 to 2.13 mg/kg, following an increase in temperature from 150 to 190 °C (Jackson and Al-Taher, 2005).

2.6 Acrylamide risk communication

The final stage of the risk analysis process is risk communication, which have been defined by the United States Environmental Protection Agency (USEPA) as a ―process of informing consumers about the potential hazards that may adversely affect them‖ (USEPA, 2002). The joint Food and Agriculture Organization of the United Nations/World Health Organization (FAO/WHO) on the other hand also define risk communication as ―the exchange of information and opinions concerning risk and risk-related factors among risk assessors, risk managers, consumers and other interested parties‖ (FAO/WHO, 2003). Codex Alimentarius also describe risk communication, to be ―the interactive exchange of information and opinions throughout the risk analysis process‖ (FAO/WHO, 2013). In fact, the risk analysis process relate to hazards and risks, risk-related factors and risk perceptions. It also include the dissemination of these risk indices among risk assessors, risk managers, consumers, industry, the academic community and other interested parties. Above all, the explanation of the risk assessment findings and the basis of risk management decisions are also made.

After the report of the presence of acrylamide in foods, the Swedish National Food Administration (SNFA) on 24th April, 2002 made a press release headlined ―Acrylamide is formed during the preparation of food and occurs in many foodstuffs‖. These scientists at the SNFA had found very high levels of acrylamide in foods, which was up to about 500 times the levels found in drinking water (Löfstedt, 2003). They also made some statements concerning the carcinogenicity of the hazard, its methods of analysis and the possible ways of its mitigation (Troxell and Posnick, 2003). The joint Food and Agriculture Organization of the United Nations/World Health Organization (FAO/WHO) in response, also made a press statement in June 2002, declaring their support of all the findings by the SNFA. Following these press statements and declarations, the sale of fried foods, particularly chips fell by about 30 – 50% (Löfstedt, 2003). The communication of risk undeniably is one important aspect of the risk analysis process, which if not communicated well, can impart negatively on consumers.

2.7 Pressing matters

Even with the many health implications associated with acrylamide exposure, many countries have not yet introduced a routine practice to monitor their presence in foods. In as much as many studies have been done by researchers in developed and some developing countries on acrylamide formation, their exposure routes and health implications, (Olesen *et al.*, 2008; Hogervorst *et al.*, 2009a; Hirvonen *et al.*, 2010; Keramat *et al.*, 2011; Chen *et al.*, 2012; Pennisi *et al.*, 2013; Virk-Baker *et al.*, 2014; Stojanovska and Tomovska, 2015; Claeys *et al.*, 2016; Altissimi *et al.*, 2017), same cannot be said of some African countries particularly Ghana. There is paucity of data on the exposure levels of the Ghanaian population to acrylamide through our locally prepared foods, even though there is a possibility that there could be high levels of

them in the frequently consumed foods. Also, there is no reliable national food consumption data. This study thus seeks to obtain baseline information on the eating habits of consumers and their potential risks associated with acrylamide exposure.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Materials

3.1.1 Sampling of Foods

The food samples selected for the analysis were based on the total dietary intakes of individuals in the Kumasi metropolis. In total, about 80 food samples were collected, in the proportion of the heavily eaten to the least eaten foods. There were rice (28), ―Fufu‖ (9), ―Banku‖ (11), ―Kenkey‖ (6), ―Porridge‖ (12), Tea (3), ―Oats‖ (2), ―Ampesie‖ (1) and their accompaniments. These samples were collected based on the time of day the food was consumed. Thus, breakfast foods were sampled in the morning between the hours of 07.00 GMT and 10.00 GMT, lunch foods between the

hours of 13.00 GMT and 15.00 GMT and the supper foods were sampled in the evenings, between the hours of 16.00 GMT and 19.00 GMT.

3.1.2 Standards and reagents

Hexane and acetonitrile were obtained from Prolabo VWR International (ParisFrance) and Merck (Darmstadt, Germany) respectively. Salts (MgSO4 and NaCl) were obtained from Sigma Aldrich (Germany). The acrylamide standard was also obtained from Acros Organics (New Jersey, USA). Analytical starch was purchased from the Ayensu Starch Company, (Central Region, Ghana).

3.2 Methods

3.2.1 Study area

The study area was Kumasi, one of the largest metropolitan areas in Ghana, with a population of 2,035,064 people, according to the 2010 population census (GSS, 2010). It has been credited as the second biggest city of Ghana. Many people from different regions of Ghana access the city for a number of business activities daily, mainly because it serves as the primary center for the trading of several commodities (Adarkwa, 2011). The city is endowed with a number of large markets; Kejetia, Bantama and Tafo. The Kejetia market is believed to be the biggest open space market in West Africa (KMA, 2010; Amoah and Jørgensen, 2014).

3.2.2 Survey

The stratified random sampling procedure was applied for the selection of the specific study areas in the metropolis. The locations were (Kwadaso, Tafo/Pankrono, Tek, Ayigya, Kotei, Buokrom, Suame, Bantama, Asokwa and Kejetia). The three

experienced assistants who were recruited to assist in the data collection, were further trained on the questionnaire administration process. As part of the training, they were enlightened on the details of the questionnaire and how to discharge the questions to the respondents. The English language and the Twi local dialect was used as the medium of instruction for the questionnaire administration. The collection of the data was randomized, and in total, about 300 respondents who were willing, were interviewed from all the ten locations in the study area, on their total dietary intakes.

3.2.3 Outline of questionnaire and localized food consumption data

The questionnaire was a structured one, containing relevant information about the dietary intake of consumers. This included questions on the quantity of food consumed at a sitting, the number of times that particular food was consumed in a week (in order to determine the exposure frequency), and the number of years that food has been consumed (exposure duration). Other information present on the questionnaire were on the biodata of consumers; their weight, age, religion, gender, work and educational background. Prior to the beginning of the actual survey, a baseline study was performed on the validity of the questionnaire, using 50 respondents in the study area. The feedback received from this test was used to modify the questionnaire. Respondents were asked questions based on foods they consumed for breakfast, lunch and supper, and their responses recorded accordingly. The biodata of the respondents, together with their consumption patterns were used as the localized food consumption data in the study area. The responses were processed in a Microsoft excel spreadsheet by grouping the similar foods together and sorted to rank the foods in the required category (breakfast, lunch and supper). The top five food groups which were consumed most in each category were selected to represent the most frequently eaten foods.

3.2.4 Sampling, Sample Preparation and Storage of Food Groups

The frequently eaten foods were randomly sampled in another round of survey according to the time of the day they were eaten. Sampled foods were quantitatively homogenized with specified amount of water in a Crompton blender (cq Sierra 500, India) and packaged into Ziploc bags and stored at -2°C until further analysis.

3.2.5 Extraction and Clean-up

In this study, a slight modification of food sample mass of 2 g was used instead of the 5 g recommended (UCT, 2012). Respective masses of $MgSO_4$ (4000 mg) and NaCl (1000 mg), together with the food sample masses were weighed and transferred into 50 ml centrifuge tubes. Five (5) ml of hexane was added and vortexed (Wilten and Co. B.V., Holland) for 1 min to help separate the hydrophilic and hydrophobic components of the food. Acetonitrile (10 ml) and distilled water (10 ml) were added and further vortexed for 1 min and later centrifuged (LHW 24958, Wageningen) at 3000 rpm for 5 min. The resulting aqueous acetonitrile phase (1 ml) was subsequently treated with 1500 and 500 mg of $MgSO_4$ and NaCl respectively, vortexed and agitated at 4000 rpm for 5 min. Finally, 2 ml of the supernatant was siphoned for HPLC analysis.

3.2.6 HPLC analysis

A Cecil-Adept binary pump HPLC with a Dynamic Absorbance detector was used for the HPLC analysis (Gökmen *et al.,* 2005). The column used was an Agilent eclipse plus C18 column (4.6 mm × 150 mm, 3.5 µm), and the column oven was set at 25°C. The mobile phase was made up of acetonitrile and water (20:80 v/v), and was adjusted to pH 3.5 with orthophosphoric acid. The flow rate of the mobile phase was set at 1 ml/min, and it was detected at 225 nm. For both the samples and standards, a volume of 60 µl was injected into the HPLC for the analysis, using the auto sampler.

Acrylamide present were detected and quantified by matching their peaks with the standard retention time and subsequently, the area under the peaks were automatically integrated by the Cecil-Adept PowerStream (CE 4300, UK) and expressed as the concentrations of acrylamide in the food samples.

3.2.7 Quality control

The recovery of the method was determined by spiking 2 g of analytical starch with different concentrations (20, 50 and 100 µg) of acrylamide standard. The extraction and purification procedure used for this recovery test followed the same procedure as that used for the various food samples. The mean recovery was at 97%, which shows that the accuracy of the method used was sufficient (Chen *et al.*, 2012). The analytical method used had a limit of detection (LOD) and limit of quantification (LOQ) of 0.03 µg/g and 0.1 µg/g respectively. The calibration curve for this method was linear (Appendix A), with an r^2 of 0.998.

3.2.8 Data analysis

The data obtained from the survey were captured into Microsoft Excel and grouped according to the gender and the different age groups; children and teenagers (5-19), young adults (20-39) and adults (above 40 years). The dietary exposure, CDI was then estimated using Equation 3.1 (USEPA, 2002), based on Monte Carlo simulation (Palisade @Risk) software (Claeys *et al.*, 2016), as a Microsoft Excel add-in. The concentrations of acrylamide in the foods, as obtained from the HPLC analysis, the body weights of the respondents, and the averaging time were expressed as C_L, B_W and AT respectively. The contact rate (C_R), is the total mass of food consumed per day. The exposure frequency (EF) and exposure duration (ED) respectively, represent the number of times the food is consumed in a week, and the number of years that

particular food has been consumed. All these variables (Equation 3.1), except for the AT, were fitted to their respective distributions. The values generated, were then used to estimate the CDI. For the AT, 30 and 70 years were used in estimating for the CDI leading to non-cancer (neurotoxicity) and cancer risk

(tumorigenesis) respectively (Gerba, 1999).

$$CDI = \frac{C_L \times C_R \times EF \times ED}{AT} \times B_W \quad \ldots\ldots\ldots\ldots 3.1$$

To characterize the tumorigenic and neurogenic effects resulting from the dietary exposure to acrylamide, the margin of exposure (MOE) was estimated using Equation 3.2 (JEFCA, 2005). The $BMDL_{10}$ (represented the bench mark dose lower limit) values used for tumorigenesis and neurotoxicity were 0.17 and 0.43 mg/kg bw/day respectively, as proposed by regulation (EFSA, 2015).

$$MOE = \frac{BMDL_{10}}{CDI} \quad \ldots\ldots\ldots\ldots 3.2$$

The non-cancer risk for systemic toxicity study, known as the hazard quotient (HQ) was estimated using Equation 3.3 (USEPA, 2005). The reference dose (R_fD) used was 2.0×10^{-3} mg/kg-day, adopted from the regional screening level (RSL) generic table released by the USEPA in November, 2017 (USEPA, 2017).

$$HQ = \frac{CDI}{\quad}.$$

$$R\ D_i \quad \ldots\ldots\ldots\ldots\ 3.3$$

The lifetime cancer risk (R) resulting from acrylamide exposure was estimated using Equation 4 (USEPA, 2010). The PF used was 0.5 $(mg/kg\ day)^{-1}$, as recommended by regulation (USEPA, 2010).

$$R = CDI \times PF \quad \ldots\ldots\ldots\ldots\ 3.4$$

The lifetime cancer risk values generated were iterated 100,000 times using the Palisade @Risk software, to give the lifetime cancer risk curves. For the regression studies, the regression option was selected, and this displayed the regression coefficients of all the individual variables that were integrated to give the lifetime risk cancer values.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.1 Acrylamide levels in foods

Acrylamide was detected in 82% of all the foods analyzed, with a mean concentration ranging from 1.33×10^{-3} - 14.39×10^{-3} mg/g as shown in Table 4.1. A similar wide variation was also reported in Austrian and Polish foods, where acrylamide levels of 0.03×10^{-3} - 1.50×10^{-3} mg/g and 0.01×10^{-3} - 3.645 mg/g were presented respectively (Murkovic, 2004; Mojska *et al.*, 2010). However, the acrylamide concentrations obtained in this study seem to be higher. It is very likely that the variation could be resulting from the foodstuffs peculiar to the different geographical regions.

Table 4.1: Concentrations of acrylamide in some selected foods

Food samples	**Acrylamide level ($\times10^{-3}$ mg/g)**	
	Mean ±Standard deviation	**Min - Max**
_Banku‘ and fish	8.76 ±9.39	0.00 – 27.82
_Banku‘ and meat	1.99 ± 2.39	0.30 – 3.68
_Fufu‘ and fish	13.23 ± 5.04	8.80 – 20.27
_Gari‘ and beans	5.85 ± 2.22	1.18 – 8.88
_Kenkey‘ and fish	4.64 ± 4.94	0.00 – 10.58
_Fufu‘ and meat	1.33 ± 1.89	1.00 – 2.67
Porridge and bread	14.39 ± 6.33	9.28 – 27.18
Rice and fish	10.20 ± 11.48	0.00 – 34.60
Porridge and buff loaf	9.80 ± 0.00	9.80 – 9.80
Oats and bread	7.81 ± 11.04	0.00 – 15.62
Rice and meat	3.63 ± 3.96	0.00 – 12.12
Tea and bread	6.17 ± 5.31	2.72 – 12.28
_Ampesie‘ and _Kontomire‘	11.02 ± 0.00	11.02 – 11.02

Porridge and _koose‘	1.44 ± 0.04	1.40 – 1.50

The highest mean acrylamide concentrations in this study (14.39 ± 6.33) were detected in porridge and bread, and the lowest (1.33 ± 1.89), was in _Fufu‘ and meat soup. Low levels were also detected in the _Kenkey‘ and fish (4.64 ± 4.94). The porridge and bread food samples contributed the greatest acrylamide content probably because of the presence of bread, since bread has been reported (Friedman, 2003) to have high acrylamide concentrations (7×10^{-5} - 43×10^{-5} mg/g). There has also been a report (Atwa *et al.,* 2010) of high acrylamide concentrations in roasted bakery products (96.8×10^{-5} mg/g), therefore, such observation is not surprising.

The low concentrations recorded for the _Kenkey‘ with fish, is not surprising because _Kenkey‘ is a boiled food product, and thus, it is expected to have non-detectable to very low levels of acrylamide (Arvanitoyannis and Dionisopoulou, 2014). Again, it is a fermented food product, and there is a report of the reduction of acrylamide content in foods that have undergone yeast fermentation (Huang *et al.,* 2008). The report show that, increasing the fermentation time from 0 to 240 min significantly reduced the asparagine and acrylamide content from 15.45 to 7.48 mg/100 g and 3.43×10^{-4} to 1.25×10^{-4} mg/g respectively. Other studies have shown that, fishes have very low levels of glucose/fructose, thus, low levels of precursors for acrylamide formation. The lowest acrylamide concentration was recorded for the _Fufu‘ and meat soup dish, and the reason could probably be that the _Fufu‘ is a boiled food product, which is known to have negligible acrylamide content (Friedman, 2003;

Arvanitoyannis and Dionisopoulou, 2014).

Up to date, the maximum level for acrylamide in foods has not been established, mainly because, the same raw food product after processing can have variable acrylamide content (Esposito *et al.*, 2017). European Food Safety Authority has however, set some indicative values to be used during the detection of acrylamide in foods. Although, these are non-legal thresholds, they serve as guidelines, above which corrective actions are needed (EFSA, 2015).

4.2 Acrylamide exposure

The food consumption data profiling of consumers in the communities are also presented in Table 4.2 through to Table 4.6. Variables of the values that were integrated to give the various parameters; the hazard, and mass of food, exposure frequency (EF), exposure duration (ED) and body weight (B_W) all presented different statistical distributions as shown in Table 4.2 through to Table 4.6. The mean acrylamide consumption for male consumers ranged from 4.67×10^{-3} to 4.88×10^{-3} mg/g across the day (Table 4.2). This trend was further enforced with a higher 50^{th} percentile exposures of 3.63×10^{-3} mg/g across the day compared to the negligible 5^{th} percentile exposures. Another observation was that, the 95^{th} percentile or the top 5% acrylamide exposure of consumers during breakfast (14.39×10^{-3} mg/g) was the highest among the 95^{th} percentile group.

Comparably, the mean acrylamide exposure for the female consumers during breakfast (Table 4.3) seems to be higher (6.26×10^{-3} mg/g) compared to male exposures (4.75×10^{-3} mg/g). This could probably stem from the females‘ high consumption of the foods which contained high levels of acrylamide (porridge and

bread: 14.39×10^{-3} mg/g; rice and fish: 10.20×10^{-3} mg/g) during breakfast. It could also be because, the breakfast foods ingested by the male consumers were mostly _Banku‘, _Fufu‘ and _Kenkey‘, which contained lower concentrations of acrylamide.

The mean acrylamide exposure during lunch (4.00×10^{-3} mg/g) and supper (4.59×10^{3} mg/g) for the female consumers were lower compared to that of the male consumers, which were 4.67×10^{-3} and 4.88×10^{-3} mg/g respectively (Table 4.2). This observation seems to be in line with the report previously published by the Norwegian Food Control Authority (NFCA, 2002), where the estimated acrylamide exposures from food was higher for male consumers (2.70×10^{-2} mg) relative to female consumers (2.00×10^{-2} mg). The Norwegian Food Control Authority again stated in the report that, male exposures to acrylamide in coffee beverages were higher (1.39×10^{-2} mg), compared to females (1.16×10^{-2} mg).

Table 4.2: Statistical distributions of acrylamide and elements of exposures in male respondents across the day

	Variable	Statistical distribution	Central tendency metrics				Percentiles		
			Min	Max	Mean	Mode	5th	50th	95th
BREAKFAST	Hazard ($\times 10^{-3}$ mg/g)	Expon (4.7541, -0.032456)	0	14.39	4.75	0	0	3.63	14.39
	Mass of food (g)	Triang (76.073, 304.18,104 1197.9)			531	304	192	469	982
	EF (days)	Geomet (0.0050205)	0	1094	198	364	0	208	364 44
	ED (years)	Geomet (0.071189)	1		13	10	1	8	94
	BW (kg)	Negbin (29, 0.30064)	20	1196 65	67	64	39	68	11.02
				100					782
LUNCH	Hazard ($\times 10^{-3}$ mg/g)	Triang (0, 0, 14.946)	0	13.23	4.67	0	0	3.63	364
	Mass of food (g)	InvGauss (431.16, 1833.17, 18.438)	99	1031	412	199	192	347	44 13.23
	EF (days)	IntUniform (0, 364)	0	364	156	0	0	156	
	ED (years)	Negbin (2, 0.11402)	1	73	15	10	2	10	795
									364
SUPPER	Hazard ($\times 10^{-3}$ mg/g)	Triang (0, 0, 15.867)	0	14.39	4.88	0	0	3.63	44
	Mass of food (g)	Triang (118.02, 231.11,132 985.63)			462	208	192	391	
	EF (days)	IntUniform (0, 364)	0	364	153	0	0	156	
	ED (years)	Negbin (2, 0.11124)	1	73	15	5	2	10	

Table 4.3: Statistical distributions of acrylamide and elements of exposures in female respondents across the day

			Central tendency metrics				Percentiles		
	Variable	**Statistical Distribution**	**Min**	**Max**	**Mean**	**Mode**	**5th**	**50th**	**95th**
BREAKFAST	Hazard ($\times 10^{-3}$ mg/g)	Uniform (-0.11155, 14.502)	0	14.39	6.26	0	0	6.17	14.39
	Mass of food (g)	Triang (89.305, 196.60, 1165.1)	104	1094	481	166	166	437	884
	EF (days)	IntUniform (0, 364)	0	364	152	N/A	0	156	364
	ED (years)	Geomet (0.084864)	1	44	10	10	1	9 63	91 33
	BW (kg)	Negbin (24, 0.27554)	20	120	63	N/A	38	3.63 276	11.02
LUNCH	Hazard ($\times 10^{-3}$ mg/g)	Expon (4.0003, 0.0291992)	0	13.23	4.00	0	0	3.63 156	13.23 736
	Mass of food (g)	InvGauss (325.05, 819.62, 4.1002)	66	997	329	132	104	8	33 364
	EF (days)	IntUniform (0, 364)	0	364	135	0	0		
	ED (years)	Geomet (0.083102)	1	50	11	1	1	347	
				14.39					

SUPPER	Hazard ($\times 10^{-3}$ mg/g)	Triang (0, 0, 15.400)	0		4.59	0	0	1568	9823
		Triang (57.949, 166.22, 971.84)	77	939	376	166	128		3643
	Mass of food (g)	Geomet (0.0073839)	0	364	134	0	0		
	EF (days)	Geomet (0.078902)	1	50	11	5	1		
	ED (years)								

The exposure of acrylamide by the three age groups (Table 4.4, Table 4.5 and Table 4.6); children and teenagers (5-19), young adults (20-39) and adults (40 and above) also followed a similar trend as that observed for the males and females consumers. That is, for all the three age groups, the 5th percentile consumers recorded negligible exposures to acrylamide.

4.3 Mass of food consumed

There was a general trend in all the food groupings, showing heavy food consumption in the morning, least consumption in the afternoon and topping up lightly in the evening. The mean mass of food consumed by the male consumers was highest (531 g), as shown in Table 4.2, and was consumed during breakfast. The 5th percentile or the bottom 5% consumers showed the lowest mass of food consumed (192 g) across the day. Fifty percent of the male consumers ingested mass of food ranging between 347 g during lunch, to 469 g during breakfast. The 95th percentile or the top five percent male consumers also showed a similar trend; highest mass of food during breakfast (982 g), lowest during lunch (782 g) and topping up during supper (795 g). On the other hand, the mean mass of food consumed by the female consumers were generally lower (329-481 g), relative to the mean mass consumed by male consumers (412-531 g). The trend of consumptions observed in this study is similar to what has been reported in a study in Italy (Altissimi *et al.*, 2017). In their study, the mean mass of food consumption of the targeted population showed male consumers ingesting more grams of foods relative to female consumers in most of the foods studied.

The female food consumption pattern (Table 4.3) also followed a similar trend of highest consumption during breakfast (481 g), least during lunch (329 g) and topping

up during supper (376 g). The food consumption pattern of the 50% of the consumers were again, in the decreasing order of breakfast, supper and lunch. The masses of food consumed during lunch were the lowest, probably because of the short break time of between 10 and 30 min (WTD, 2015). These short break periods may be a convention adopted from the Switzerland‘s National and EU legislations, which prescribe such breaks; between 10 and 30 min, after six consecutive hours of work. It is within this short time frame that workers are expected to arrange for something to eat and visit the restrooms as well. This might be leading to low patronage of lunch in these countries (WTD, 2015; Sebastian *et al.*, 2015; Deliens *et al.*, 2014; Apple,
2009). In contrast these however, in countries such as Germany, Portugal, Finland, Sweden, Hungary and Russia lunch is the main meal of the day (Albyn, 1993; Fogelholm, 2001; Schulte-Peevers and Gray, 2007; Albala, 2011; Long, 2015). Thus, it is usually a full hot meal, and consisting of more than one course. For such countries the masses of food consumed during lunch may be higher and
subsequently, the risk may be greater.

The food consumption patterns and acrylamide exposures for the three different age groups; children and teenagers (5-19), young adults (20-39) and adults (40 and above) are presented in Table 4.4, Table 4.5 and Table 4.6 respectively. The food consumption pattern of all the three age groups followed the trend of heaviest breakfast, moderate supper and lightest lunch. The young adults‘ age group recorded the highest mass of food consumed for breakfast (537 g), lunch (404 g) and supper
(438 g), with the children and teenagers group recording the lowest. These observations were similar to the results from the National Nutrition Survey (NNS) in Australia, which reported that, young adults of the age group 25-29 years had the

highest mean daily intake of food and beverages, and children between the ages 2-11 years had the lowest mean daily intake (McLennan and Podger, 1999).

Table 4.4: Statistical distributions of acrylamide and elements of exposures of children and teenagers (5-19 years) across the day

			Central tendency metrics				Percentiles		
	Variable	**Statistical Distribution**	**Min**	**Max**	**Mean**	**Mode**	**5th**	**50th**	**95th**
BREAKFAST	Hazard (×10^{-3} mg/g)	Uniform (-0.22484, 14.615)	0	14.39	6.30	0	0	3.63	14.39
	Mass of food (g)	Weibull (1.8467, 458.05, 71.151)	104	1094	478	437	138	437	875
	EF (days)	IntUniform (0, 364)	0	364	179	364	0	156	364
	ED (years)	Negbin (2, 0.23278)	1	18	6	1	1	5	15
	BW (kg)	Negbin (16, 0.23088)	20	85	53	N/A	24	54	79
LUNCH	Hazard (×10^{-3} mg/g)	Triang (0, 0, 12.121)	0	10.2	3.67	0	0	3.63	10.2
	Mass of food (g)	Expon (184.16, 95.731)	99	997	283	99	99	207	664
	EF (days)	IntUniform (0, 364)	0	364	143	0	0	104	364
	ED (years)	IntUniform (1, 18)	1	18	7	9	1	8	16
SUPPER	Hazard (×10^{-3} mg/g)	Expon (4.1960, 0.0626264)	0	14.39	4.20	0	0	3.63	11.02
	Mass of food (g)	Triang (54.997, 166.22, 937.90)	77	830	368	166	128	308	782
	EF (days)	IntUniform (0, 364)	0	364	161	156	0	156	364
	ED (years)	Negbin (4, 0.33727)	1	19	7	5	1	6	17

Table 4.5: Statistical distributions of acrylamide and elements of exposures of young adults (20-39 years) across the day

			Central tendency metrics				Percentiles		
	Variable	**Statistical Distribution**	**Min**	**Max**	**Mean**	**Mode**	**5th**	**50th**	**95th**
BREAKFAST	Hazard ($\times 10^{-3}$ mg/g)	Expon (4.9983, 0.0357020)	0	14.39	4.99	0	0	4.64	14.39
	Mass of food (g)	Triang (91.571, 208.34, 234.8)	104	1094	537	332	196	491	1060
	EF (days)	IntUniform (0, 364)	0	364	166	0	0	156	364
	ED (years)	Negbin (2, 0.17532)	1	33	9	10	1	8	26
	BW (kg)	Negbin (60, 0.47097)	26	100	67	N/A	50	67	94
LUNCH	Hazard ($\times 10^{-3}$ mg/g)	Triang (0, 0, 14.482)	0	13.23	4.31	0	0	3.63	11.02
	Mass of food (g)	Triang (115.43, 208.34, 965.81)	132	939	404	256	181	345	782
	EF (days)	IntUniform (0, 364)	0	364	155	0	0	156	364
	ED (years)	Negbin (2, 0.14091)	1	38	12	10	1	10	33
SUPPER	Hazard ($\times 10^{-3}$ mg/g)	Expon (4.2480, 0.0294999)	0	14.39	4.24	0	0	3.63	13.23
	Mass of food (g)	Triang (110.58, 208.34, 999.66)	132	939	438	332	173	347	782
	EF (days)	Geomet (0.0074689)	0	364	132	0	0	156	364
	ED (years)	Negbin (2, 0.14582)	1	35	11	N/A	1	9	30

Table 4.6: Statistical distributions of acrylamide and elements of exposures of adults (40 years and above) across the day

	Variable	Statistical Distribution	Min	Max	Central tendency metrics	Percentiles		95th
							50th	
BREAKFAST	Hazard (×10^{-3} mg/g)	Uniform (-0.21478, 14.605)	0	14.39	5.65	0	6.17	14.39
		ExtValue (376.34, 165.53)	138	1094	470	138	437	875
	Mass of food (g)	Geomet (0.0051422)	0	1196	193	N/A	156	364
	EF (days)	Geomet (0.042929)	1	65	22	N/A	25	50
	ED (years)	Negbin (57, 0.43769	48	120	73	N/A	72	96
	BW (kg)						3.63	13.23
LUNCH	Hazard (×10^{-3} mg/g)	Uniform (-0.20354, 13.434)	0	13.23	0.52	0	317	782
		InvGauss (414.21, 1279.35, 29.035)	66	1031	385	28	156	364
	Mass of food (g)	Geomet (0.0076798)	0	364	129	0	20	50
	EF (days)	Geomet (0.043781)	1	73	21	N/A	8.76	13.23
	ED (years)			14.39	6.75	2	407	782
						0	156	364
						0	22	50

SUPPER	Hazard ($\times 10^{-3}$ mg/g)	Uniform (-0.22484, 14.615)	0	830	444	347	154
	Mass of food (g)	Triang (62.175, 347.23, 906.19)	104	364	163	156	0
				73	23	30	1
	EF (days)	IntUniform (0, 364)	0				
	ED (years)	Geomet (0.040767)	1				

4.4 Chronic exposures

The chronic daily intakes (CDI) with respect to both tumorigenesis and neurotoxicity for consumers generally ranged from 1×10^{-8} to 8.32×10^{-2} mg/kg bw/day across the day, as shown in Table 4.7 and Table 4.9. The mean and 95th percentile (top 5%) consumers' chronic dietary exposures were in the range of 1.56×10^{-3} - 1.88×10^{-2} mg/kg bw/day and 6.16×10^{-3} - 8.32×10^{-2} mg/kg bw/day respectively (Table 4.7 and Table 4.9). This chronic intake seems to be slightly higher compared to the estimated intakes from many other countries, as reported in Table 4.8. This could probably be due to the nature of the foods and their resulting higher acrylamide levels determined in this study. Again, the food culture in Ghana is totally different from that of these other countries, thus, the variation could also result from many factors including; processing conditions, substrate composition (asparagine and fructose/glucose), and the sulphur and nitrogen levels of our soils (Adebo *et al.*, 2017). For instance, a decrease in the concentration of acrylamide in foods has been reported (Adebo *et al.*, 2017), following an increase and decrease of the sulphur and nitrogen levels in the soil respectively.

The adults age group (40 years and above) recorded the greatest chronic exposures across the study (Table 4.9). This was followed by the children and teenagers group (5-19 years), and finally, the young adults group (20-39 years). In contrast, a study (Jankowska *et al.*, 2009) has reported of highest dietary exposure by children and teenagers in Kraków, Poland. The study reported that, bread, which is one of the key sources of acrylamide (Friedman, 2003), was consumed daily by this age group, and this could probably be the reason supporting the high exposures. They also reported that, the lower body weight of this age group contributed to their high exposures. In contrast to this, and in line with this present study, other studies have reported (Wyka

et al., 2015; Cummins *et al.,* 2008) lower chronic exposures of children and teenagers. Exposures to acrylamide in foods depend on variable factors including; the population, age of consumers and their eating preferences (Semla *et al.,* 2017). Thus, it is these factors that form the basis of variations in the exposures to acrylamide observed.

Table 4.7: Chronic dietary exposures of male and female respondents for the risk of tumorigenesis and neurotoxicity

	Exposures during breakfast (mg/kg bw/day)		**Exposures during lunch (mg/kg bw/day)**		**Exposures during supper (mg/kg bw/day)**	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**
Tumorigenesis						
Mean	3.99×10^{-3}	4.52×10^{-3}	3.55×10^{-3}	1.74×10^{-3}	4.19×10^{-3}	2.11×10^{-3}
5th	1×10^{-8}	1×10^{-8}	2.83×10^{-5}	1×10^{-8}	3.52×10^{-5}	1×10^{-8}
50th	6.84×10^{-4}	1.38×10^{-3}	1.40×10^{-3}	3.78×10^{-4}	1.67×10^{-3}	4.35×10^{-4}
95th	1.72×10^{-2}	1.92×10^{-2}	1.42×10^{-2}	7.59×10^{-3}	1.69×10^{-2}	9.42×10^{-3}
Neurotoxicity						
Mean	9.32×10^{-3}	1.06×10^{-2}	8.29×10^{-3}	4.05×10^{-3}	9.78×10^{-3}	4.92×10^{-3}
5th	1×10^{-8}	1×10^{-8}	6.60×10^{-5}	1×10^{-8}	8.22×10^{-5}	1×10^{-8}
50th	1.60×10^{-3}	3.21×10^{-4}	3.26×10^{-3}	8.82×10^{-4}	3.89×10^{-4}	1.01×10^{-3}
95th	4.01×10^{-2}	4.49×10^{-2}	3.32×10^{-2}	1.77×10^{-2}	3.94×10^{-2}	2.20×10^{-2}

Table 4.8: Dietary acrylamide exposures of respondents from different countries

Country	**Acrylamide exposure (mg/kg bw/day)**		
	Mean	**95th Percentile**	**References**
Italy	4.52×10^{-4}	1.54×10^{-3}	Altissimi *et al.,* 2017
Canada	5.80×10^{-4}	2.19×10^{-3}	Normandin *et al.,* 2013
France	4.30×10^{-4}	1.02×10^{-3}	Sirot *et al.,* 2012
Belgium	3.50×10^{-4}	1.12×10^{-3}	Claeys *et al.,* 2010

China	2.86×10^{-4}	4.90×10^{-4}	Chen *et al.*, 2008
United States of America	4.30×10^{-4}	1.30×10^{-3}	Dybing *et al.*, 2005
The Netherlands	4.80×10^{-4}	6.0×10^{-4}	Konings *et al.*, 2003
Sweden	3.10×10^{-2}	6.20×10^{-2}	Svensson *et al.*, 2003

Table 4.9: Respondent age groups and their chronic dietary exposures for the risk of tumorigenesis and neurotoxicity

	Exposures during breakfast (mg/kg bw/day)			Exposures during lunch (mg/kg bw/day)			Exposures during supper (mg/kg bw/day)		
	Children and teenagers (5-19 years)	Young adults 20-39 years	Adults 40 years and above	Children and teenagers (5-19 years)	Young adults 20-39 years	Adults 40 years and above	Children and teenagers (5-19 years)	Young adults 20-39 years	Adults 40 years and above
Tumorigenesis									
Mean	3.31×10^{-3}	2.61×10^{-3}	8.08×10^{-3}	1.56×10^{-3}	2.75×10^{-3}	3.98×10^{-3}	1.83×10^{-3}	1.74×10^{-3}	7.47×10^{-3}
5th	1×10^{-8}	3.57×10^{-6}	1×10^{-8}	1.81×10^{-5}	1.82×10^{-5}	1×10^{-8}	6.26×10^{-6}	2.52×10^{-6}	1×10^{-8}
50th	1.28×10^{-3}	7.92×10^{-4}	1.98×10^{-3}	6.20×10^{-4}	1.09×10^{-3}	9.09×10^{-4}	6.05×10^{-4}	4.09×10^{-4}	2.51×10^{-3}
95th	1.33×10^{-2}	1.12×10^{-2}	3.56×10^{-2}	6.16×10^{-3}	1.1×10^{-2}	1.71×10^{-2}	7.57×10^{-3}	7.49×10^{-3}	3.17×10^{-2}
Neurotoxicity									
Mean	7.72×10^{-3}	6.08×10^{-3}	1.88×10^{-2}	3.65×10^{-3}	6.42×10^{-3}	9.29×10^{-3}	4.28×10^{-3}	4.07×10^{-3}	1.74×10^{-2}
5th	1×10^{-8}	8.32×10^{-6}	1×10^{-8}	4.22×10^{-5}	4.55×10^{-5}	1×10^{-8}	1.46×10^{-5}	5.87×10^{-6}	1×10^{-8}
50th	2.98×10^{-3}	1.85×10^{-3}	4.63×10^{-3}	1.45×10^{-3}	2.55×10^{-3}	2.12×10^{-3}	1.41×10^{-3}	9.55×10^{-4}	5.85×10^{-3}
95th	3.11×10^{-2}	2.61×10^{-2}	8.32×10^{-2}	1.44×10^{-2}	2.56×10^{-2}	3.99×10^{-2}	1.77×10^{-2}	1.75×10^{-2}	7.39×10^{-2}

4.5 Risk characterization

4.5.1 Hazard quotient

The 5th percentile group of the male and female consumers did not show any risks resulting from acrylamide exposure because, they recorded HQ values less than 1 across the day (Figure 4.1). The median (50th percentile group) consumers did not present any trend across the day in both the male and female consumers, mainly because in some cases they presented HQ of less than 1, and in other cases they presented HQ greater than 1. That is, they showed confounding safe and unsafe levels across the day. However, the highest exposures (95th percentile groups of both males and females) showed unsafe levels across the day, since all their HQ values were greater than 1. This implies that, consumers who are highly exposed to acrylamide (top 5%) through their dietary intakes are at a higher risk compared to the bottom 5% consumers.

In a report on the risk of acrylamide in Romanian food (Oroian *et al.,* 2015), the HQ values for both the male (0.55) and female (0.70) consumers were less than 1, signifying safe levels of chronic-toxic exposures. In determining the HQ of the exposures, a reference dose (R_fD) of 0.5×10^{-3} mg/kg bw/day was used (Svensson *et al.,* 2003; Oroian *et al.,* 2015) whereas the R_fD applied in this present study was 2.0×10^{-3} mg/kg-day as documented in the Regional Screening Level (RSL) generic table, released by the USEPA in November, 2017 (USEPA, 2017). This could probably be the reason behind the differences in the HQ values reported above. Also, the different chronic exposures could be another factor contributing to these differences.

The HQ values for the respondents belonging to the three age groups showed a similar trend as was recorded for the gender. Here, the 5th percentile group once again recorded HQ values less than 1 across the day (Figure 2). That is, the levels of acrylamide they were exposed to through eating did not imply any significant health risk. On the other hand, the 95th percentile presented HQ values above 1 for all the age groups across the day, thus, showing unsafe levels of acrylamide exposure. This implies that, the highest (top 5%) food consumers stand a higher risk of acrylamide toxicity than the low (bottom 5%) food consumers.

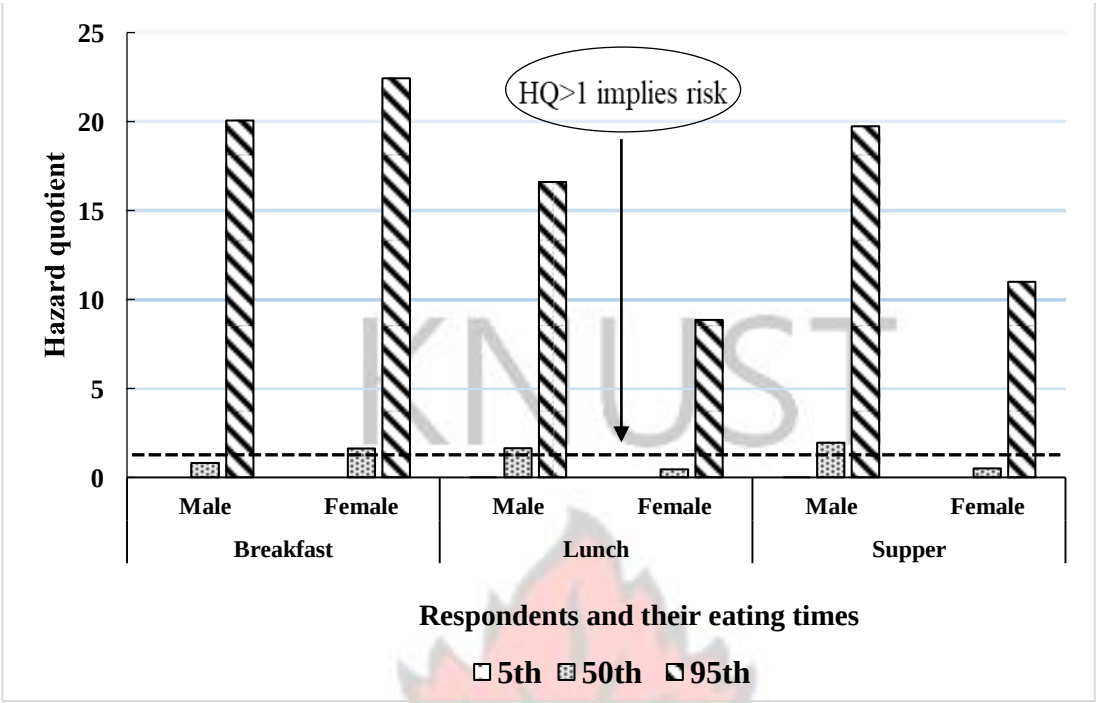

Figure 4.1: Estimated hazard quotients of male and female respondents from acrylamide ingestion across the day

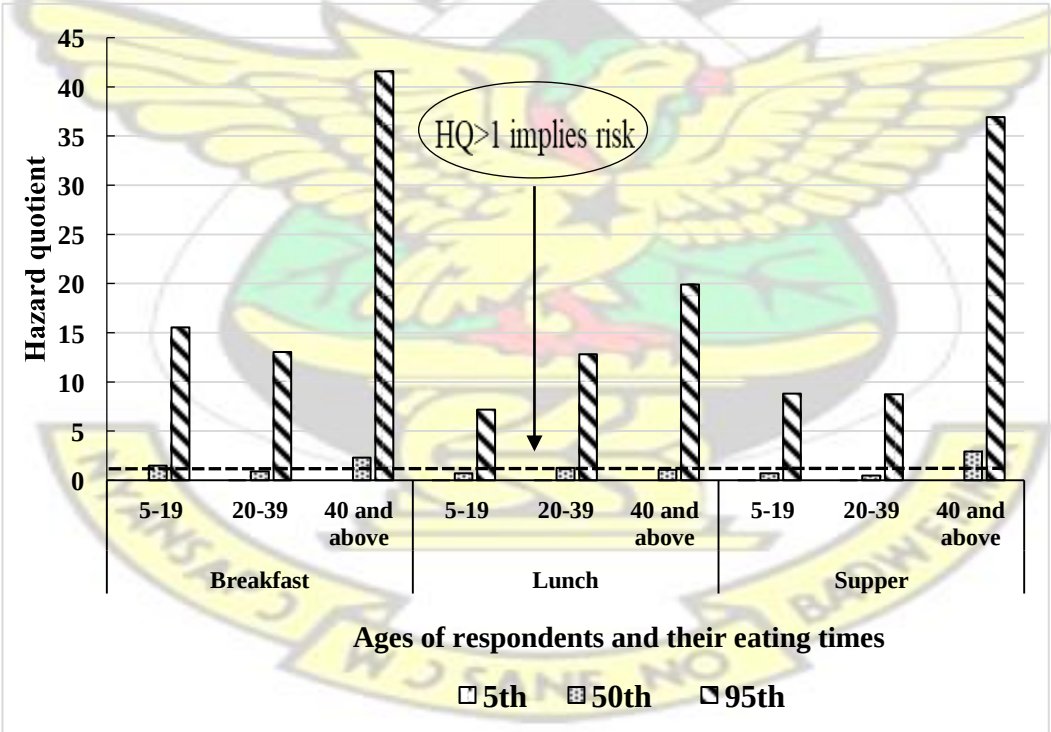

Figure 4.2: Estimated hazard quotients of age groups of respondents from acrylamide ingestion across the day

4.5.2 Margin of Exposure (MOE)

In this study, the MOE values for the risk of tumorigenesis and neurotoxicity ranged between 4.8 and 4.3×10^7 (Figure 4.3, Figure 4.4, Figure 4.5 and Figure 4.6). This range is not surprising, because of reported MOE values, ranging from 129 to 1.43×10^7 (Esposito *et al.,* 2017). Another study however, reported of less varied MOE values, ranging between 110.5 and 951.3 (Altissimi *et al.,* 2017).

For the risk of tumorigenesis, all the female 5^{th} percentile consumers recorded MOE values which were above the threshold (10,000) mark, thus, showing safe levels across the day (Figure 4.3). The higher the MOE value, the less likely it is for concentrations of the hazard (acrylamide) to reach toxicity levels. On the other hand, the 5^{th} percentile male consumers presented MOE values below the threshold during lunch (6.01×10^3) and supper (4.82×10^3), thus, their exposures pose greater public health concern. The study shows that, the male consumers generally presented higher risk of tumorigenesis relative to the female consumers across the day, which could probably be because of the high chronic exposures of the male consumers as reported earlier (Table 4.7).

The mean exposure estimates of MOE of risk of tumorigenesis of both male and female consumers ranged between 37.61 and 97.70 (Figure 4.3), thus, implying cancer risk, since the MOE reported (89 – 425) is below the 10,000 threshold mark recommended by the European Food Safety Authority (EFSA, 2015). Studies on the risk assessment of the dietary exposure to acrylamide in the Norwegian population also revealed an MOE of 189 (Brantsæter *et al.,* 2015). However, one study

(Altissimi *et al.*, 2017) reported of a higher value (376.11), showing that variability of MOE exist because they can be estimated from the exposures of different food samples. For the 95th percentile group of consumers, the MOE values recorded in this study was low and ranged between
8.84 and 22.40 (Figure 4.3), which is much higher than the range 50 and 283, reported by the European Food Safety Authority (EFSA, 2015), hence implying higher risk of tumorigenesis for this group of consumers.

In other studies, the 95th percentile exposures of 106 and 110.46 have been reported (Altissimi *et al.*, 2017; Brantsæter *et al.*, 2015) and these values are much higher than what was obtained in this present study. The disparity in the MOE values could be arising from the different foods analyzed and also different chronic exposure estimates of the consumers. Figure 4.4 presented the observations of the MOE values for the risk of tumorigenesis of the respondents belonging to the three age groups, showed values ranging from 4.77 to 1×10^{7}. The mean exposure estimates of the adult age group (40 years and above) generally recorded the lowest MOE, between 21.04 and 42.71 across the day, thus exhibiting the highest public health concern. These values are much higher compared to the values ranging between 283 and 425, reported by the Scientific Committee of the European Food Safety Authority (EFSA,
2015) for consumers belonging to the adult age group.

Generally, female consumers were at a lower risk of neurotoxicity, because they recorded higher MOEs compared to male consumers (Figure 4.5). The mean exposure MOE of the consumers for the risk of neurotoxicity ranged from 40.57 to
106.17 (Figure 4.5), which is much lower than the values reported by the European

Food Safety Authority (EFSA, 2015) to be in the range of 226 to 1075. This value is also below the safety limit or threshold of 125 set by regulation (EFSA, 2015), and thus, poses serious health concern. The 95th percentile consumers recorded MOEs ranging between 9.59 and 24.29 (Figure 4.5), which is below the safety limit or threshold, hence posing risk of neurotoxicity. These values are again, much lower relative to values ranging between 126 and 717 that was reported by the European Food Safety Authority (EFSA, 2015). The disparity in exposures could result from variable factors such as chronic daily intakes (CDI) of the consumers, and the acrylamide concentrations in the different foods analyzed.

The mean and 95th percentile exposures MOEs (for the risk of neurotoxicity) of consumers of the three age groups were below the threshold of 125 (Figure 4.6), hence posing public health concern. These values were again, lower than the values reported by the European Food Safety Authority (EFSA, 2015). This implies that, the mean and top 5% consumers of the population stand a high risk of neurotoxicity, due to their calculated MOE values.

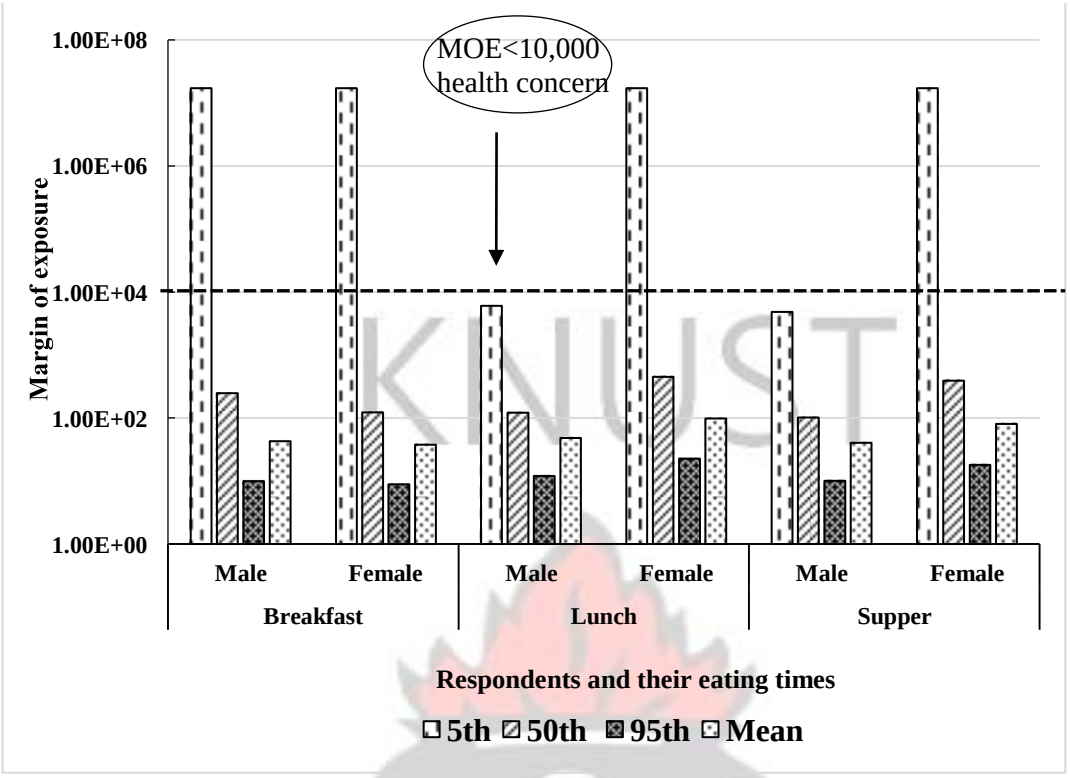

Figure 4.3: Estimated MOE for the gender of respondents for the risk of tumorigenesis resulting from acrylamide exposures across the day

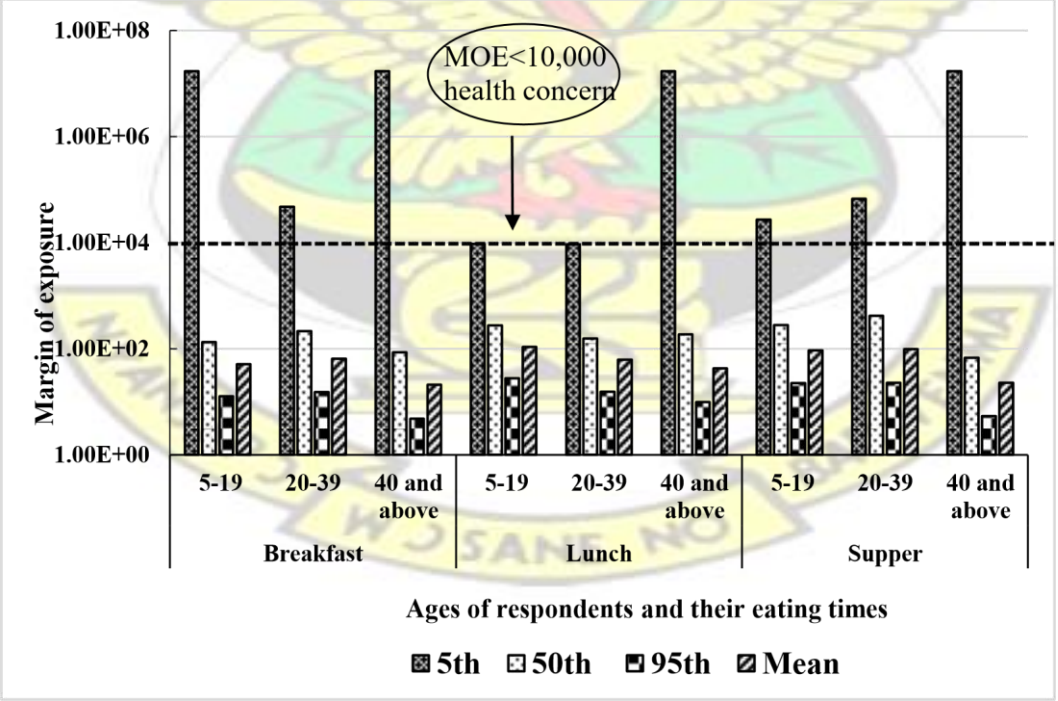

Figure 4.4: Estimated MOE of age groups of respondents for the risk of tumorigenesis resulting from acrylamide ingestion across the day

Figure 4.5: Estimated MOE of respondents for the risk of neurotoxicity resulting from acrylamide ingestion across the day

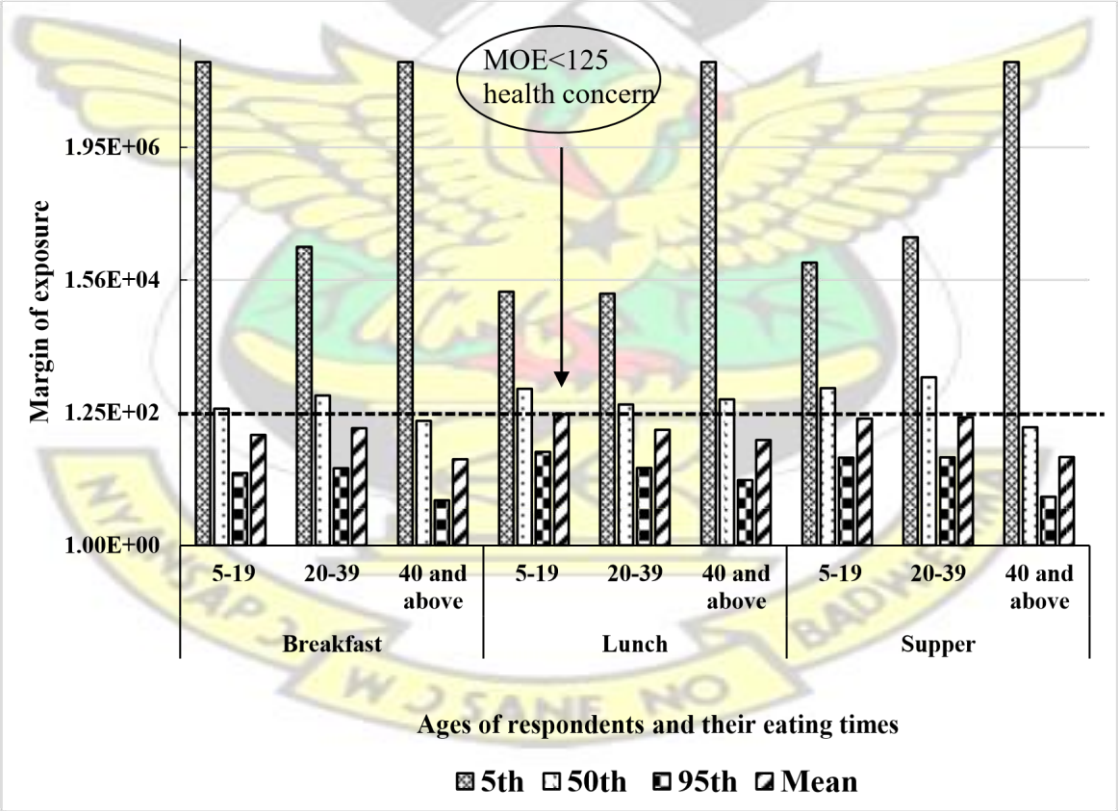

Figure 4.6: Estimated MOE of age groups of respondents for the risk of neurotoxicity resulting from acrylamide ingestion across the day

4.5.3 Lifetime risk

In this study, the lifetime human cancer risk resulting from dietary exposure of acrylamide ranged from 5.0×10^{-9} to 1.76×10^{-2}. That is, values less than 10^{-6} are generally believed to be inconsequential, hence, warranting no public health concern, whiles risk values greater than 10^{-6} pose significant health concern. Male consumers were generally at a higher risk compared to female consumers across the day (Table 4.10). This is in line with a study which reported a higher risk for male consumers (2.1×10^{-3}) relative to female consumers (1.9×10^{-3}) (Dybing and Sanner, 2003). There has also been a reported case of higher cancer risk for male consumers (in the range of 3.8×10^{-6} to 1.9×10^{-5}) relative to female consumers (3.0×10^{-6} to 1.5×10^{-5}) (Chen *et al.,* 2012).

Table 4.10: Estimated lifetime risks of the male and female respondents resulting from acrylamide ingestion across the day

	Risk during breakfast (mg/kg-day)		**Risk during lunch (mg/kg-day)**		**Risk during supper (mg/kg-day)**	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**
5th	5×10^{-9}	5×10^{-9}	1.44×10^{-5}	5×10^{-9}	1.76×10^{-5}	5×10^{-9}
50th	3.45×10^{-4}	6.85×10^{-4}	6.91×10^{-4}	1.91×10^{-4}	8.32×10^{-4}	2.19×10^{-4}
95th	8.61×10^{-3}	9.52×10^{-3}	7.12×10^{-3}	3.82×10^{-3}	8.36×10^{-3}	4.70×10^{-3}

Generally, the 5th percentile female consumers across the day recorded cancer risk levels which were lower (5×10^{-9}) relative to the *de minimus* (10^{-6}), implying negligible cancer risks (Table 4.10). On the other hand, the 50th and 95th percentile consumers of both gender were at risk of developing cancer, since their lifetime human cancer risk levels were higher (1.91×10^{-4} to 9.32×10^{-3}) relative to the *de minimus* (10^{-6}). This implies that, 2 out of 10,000 to 9 out of 1,000 consumers stand the risk of developing cancer. This is similar to a study, where the 50th (0.5×10^{-3}) percentile consumers had

lifetime cancer risk levels greater than the *de minimus* (Dybing and Sanner, 2003).Here, 1 person out of 1000 consumers stand the risk of developing cancer.

Chronic daily intake of hazards is determined based on the elements: concentration of hazard, mass of food, exposure frequency and exposure duration, body weight and averaging time. These elements were integrated to give the lifetime cancer risks across the day. The lowest cancer risk for the 5^{th} and 50^{th} percentile male consumers was recorded during breakfast, followed by lunch and supper (Figure 4.7). On the other hand, the lowest cancer risk for the 5^{th} and 50^{th} percentile female consumers were recorded during lunch, followed by supper and breakfast (Figure 4.8). The lower amount of food mostly consumed by these respondents (Table 4.3) during lunch could probably be the reason for the lower impact of the lunch elements on the cancer risk of the female consumers.

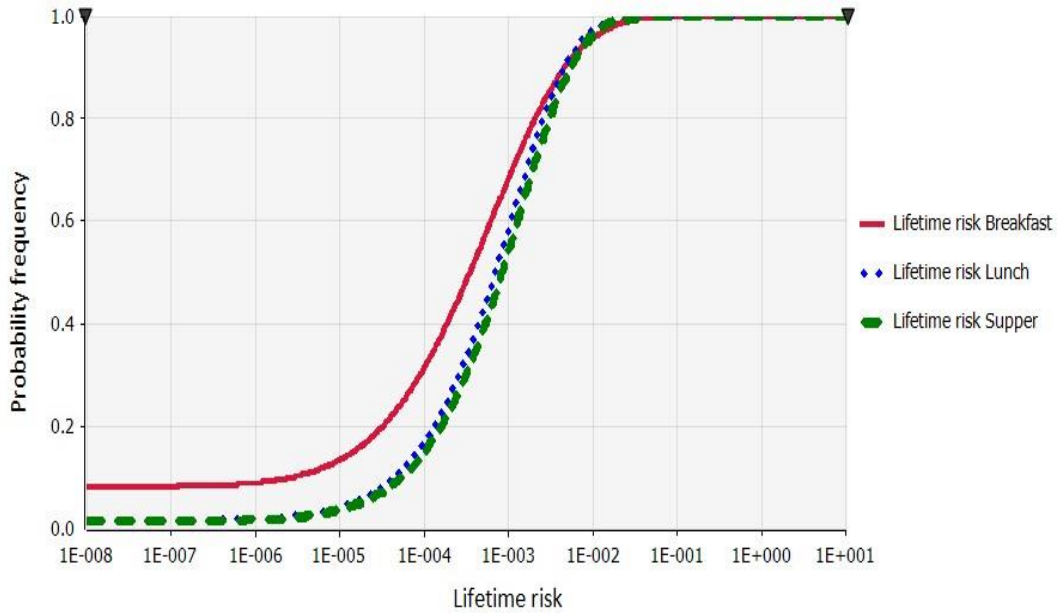

Figure 4.7: Estimated lifetime cancer risks of male respondents resulting from acrylamide ingestion across the day

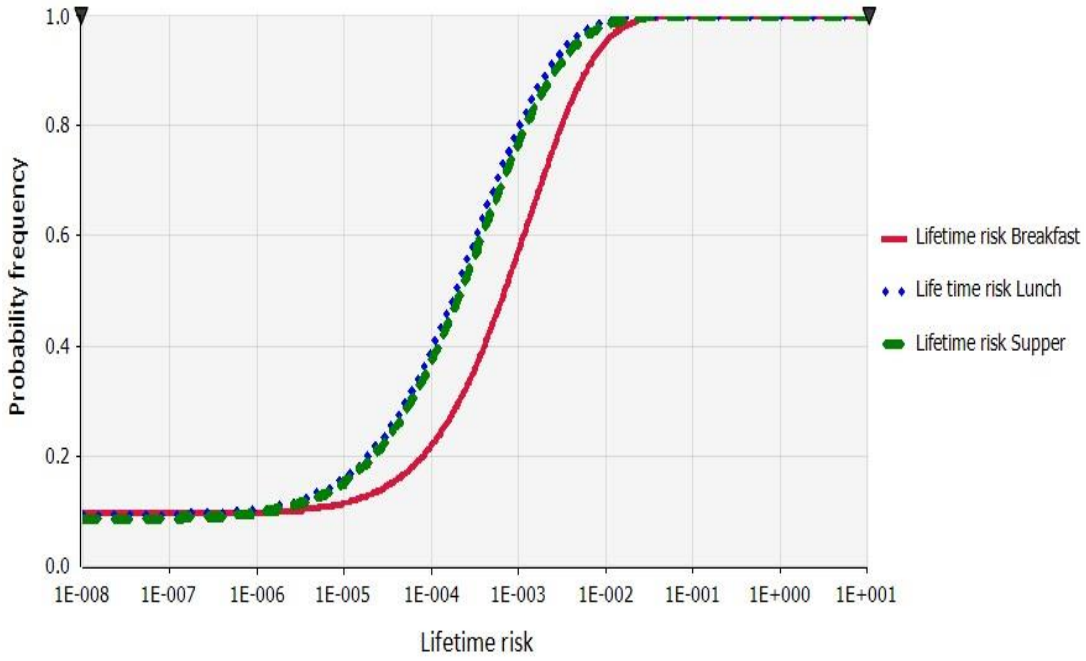

Figure 4.8: Estimated lifetime cancer risks of female respondents resulting from acrylamide ingestion across the day

The cancer risk levels pertaining to acrylamide dietary exposure of the three age group respondents are presented in Table 4.11. The 5th percentile consumers showed a lifetime cancer risk relatively lower (5×10^{-9}) than the *de minimus* (10^{-6}) across the day, thus, exhibiting no public health concern. On the other hand, the 50th and 95th percentile consumers showed high cancer risk, because, their lifetime cancer risk values were relatively greater (2.04×10^{-4} - 1.76×10^{-2}) than the *de minimus* (10^{-6}). In general, the higher the risk value, the greater the risk that will be implicated. Thus, the adults' group (40 years and above) recorded the highest cancer risk value (8.79×10^{-3} - 1.76×10^{-2}), followed by the children and teenagers group (3.11×10^{-3} - 6.64×10^{-3}), and finally the young adults group (3.72×10^{-3} - 5.58×10^{-3}) (Table 4.11).

For the adults' group for instance, between 9 out of 1000 to 2 out of 100 people stand the risk of developing cancer.

Table 4.11: Estimated lifetime risks of the age groups of respondents resulting from acrylamide ingestion across the day

	Risk during breakfast (mg/kg-day)			Risk during lunch (mg/kg-day)			Risk during supper (mg/kg-day)		
	Children and teenagers (5-19 years)	Young adults (20-39 years)	Adults (40 years and above)	Children and teenagers (5-19 years)	Young adults (20-39 years)	Adults (40 years and above)	Children and teenagers (5-19 years)	Young adults (20-39 years)	Adults (40 years and above)
5th	5×10^{-9}	1.75×10^{-6}	5×10^{-9}	8.86×10^{-6}	9.18×10^{-6}	5×10^{-9}	3.31×10^{-6}	1.29×10^{-6}	5×10^{-9}
50th	6.41×10^{-4}	3.97×10^{-4}	9.83×10^{-4}	3.08×10^{-4}	5.46×10^{-4}	4.47×10^{-4}	3.04×10^{-4}	2.04×10^{-4}	1.26×10^{-3}
95th	6.64×10^{-3}	5.58×10^{-3}	1.76×10^{-2}	3.11×10^{-3}	5.55×10^{-3}	8.79×10^{-3}	3.78×10^{-3}	3.72×10^{-3}	1.58×10^{-2}

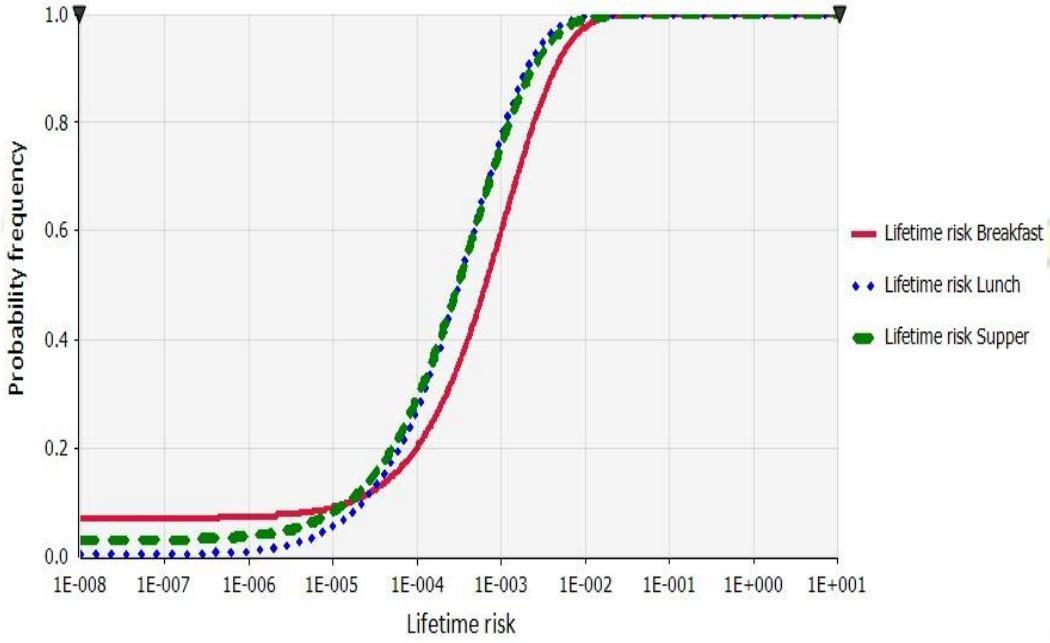

Figure 4.9: Estimated lifetime risks of children and teenagers (5-19 years) resulting from acrylamide ingestion across the day

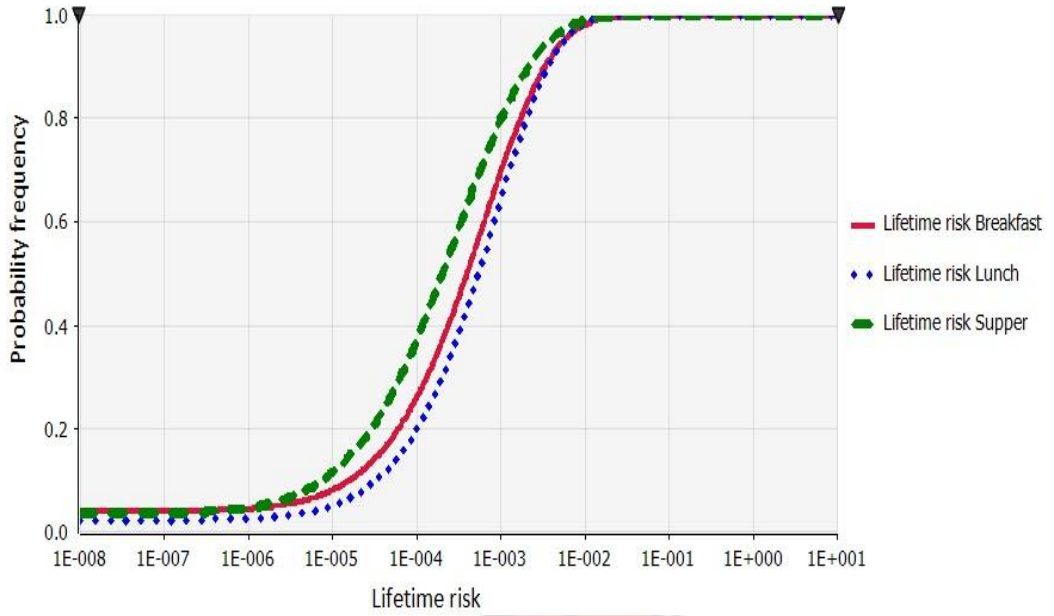

Figure 4.10: Estimated lifetime risks of young adults (20-39 years) resulting from acrylamide ingestion across the day

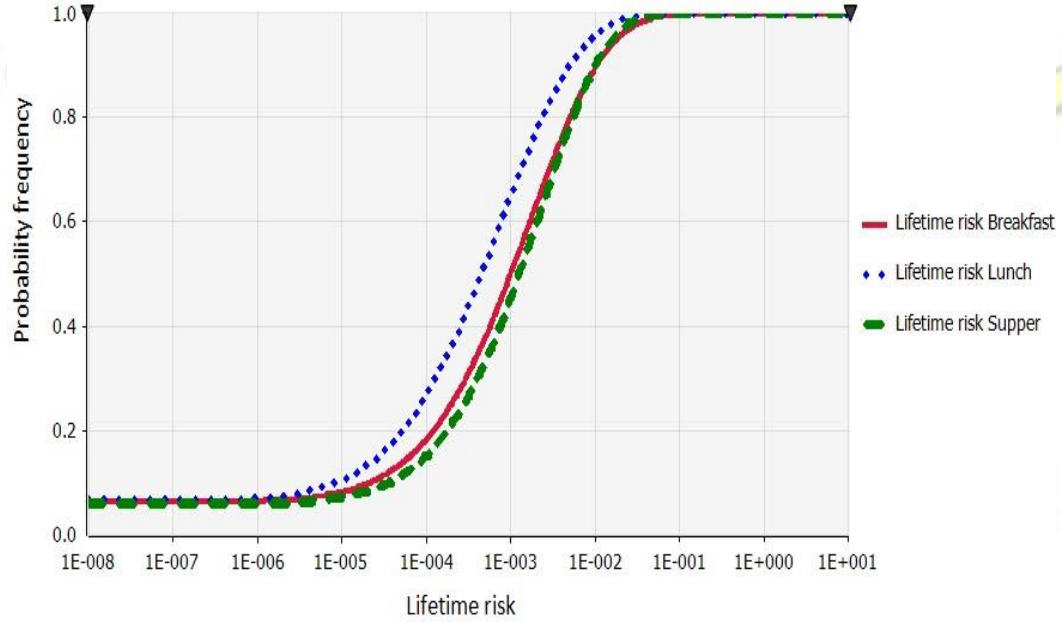

Figure 4.11: Estimated lifetime risks of adults (40 years and above) resulting from acrylamide ingestion across the day

Observations from the regression studies show that, the variable that contributed mostly to the cancer risk was the exposure duration (ED) (Table 4.12). Generally, the ED‘s impact on the cancer risk of the consumers was the greatest (52%), followed by the acrylamide concentration (47%). The impact of the exposure frequency (EF) was

33% while that of the mass of food and body weight were marginal. For the female consumers‘ cancer risks, the impact of the ED was about 52%, while that of the acrylamide concentration and the exposure frequency (EF) were 29% each. With respect to the cancer risk of the male consumers however, the ED‘s impact was 33%, while that of the acrylamide concentration and exposure frequency (EF) contributed 32% each to the risk. The ED was the highest contributor (46%) to the cancer risk of the children and teenagers group. For the young adults‘ group, the acrylamide concentration (47%) was the highest contributor, followed by the ED. The ED was also the highest contributor for the cancer risk of the consumers aged 40 years and above. The high impact of the ED on the cancer risk of the consumers implies that, the longer an individual is exposed to acrylamide through food, the greater that individual‘s risk to developing cancer (Chen *et al.*, 2012).

Table 4.12: Regression coefficients and percentages of the impact of the elements of the CDI on the risk of acrylamide ingestion by respondents

		Hazard (mg/g)	**ED (years)**	**EF (days)**	**Mass of food (g)**	**Bw (kg)**
Male	β	0.32	0.33	0.32	0.15	-0.07
	%	32	33	32	15	7
Female	β	0.29	0.52	0.29	0.25	-0.12
	%	29	52	29	25	12
Age 5-19	β	0.34	0.46	0.33	0.27	-0.17
	%	34	46	33	27	17
Age 20-39	β	0.47	0.37	0.28	0.24	-0.09
	%	47	37	28	24	9
Age 40 and above	β	0.24	0.42	0.4	0.18	-0.08
	%	24	42	40	18	8

CHAPTER FIVE

CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusion

Generally, the chronic dietary exposures to dietary acrylamide of the male consumers were higher than that of the female consumers, thus, placing the males at a higher carcinogenic and neurotoxic risk. The adult group was at the highest risk of developing cancer, followed by children and teenagers' age group, and finally the young adults' age. The mean, median and 95th percentile consumers showing HQ of above 1 and MOEs below the threshold respectively indicate serious health concern. The exposure duration was the element or variable that contributed the most to the cancer risk of the consumers, relative to the mean concentration of acrylamide in the foods.

There was a general trend in all the food groupings, heavy food consumption in the morning, least in the afternoon. The chronic dietary exposures of the male consumers were relatively higher than that of the female consumers, and the adult group was at the highest risk of developing cancer. The highest carcinogenic and neurotoxic risk was recorded during breakfast with 10 out of 1000 people standing the risk of developing cancer.

5.2 Recommendations

In order to impact effectively scientific communication, further studies should be undertaken to specifically map areas in the study area where the chronic dietary exposures and risks were highest, and subsequently, mitigation studies could be seriously mounted in order to control their levels in foods.

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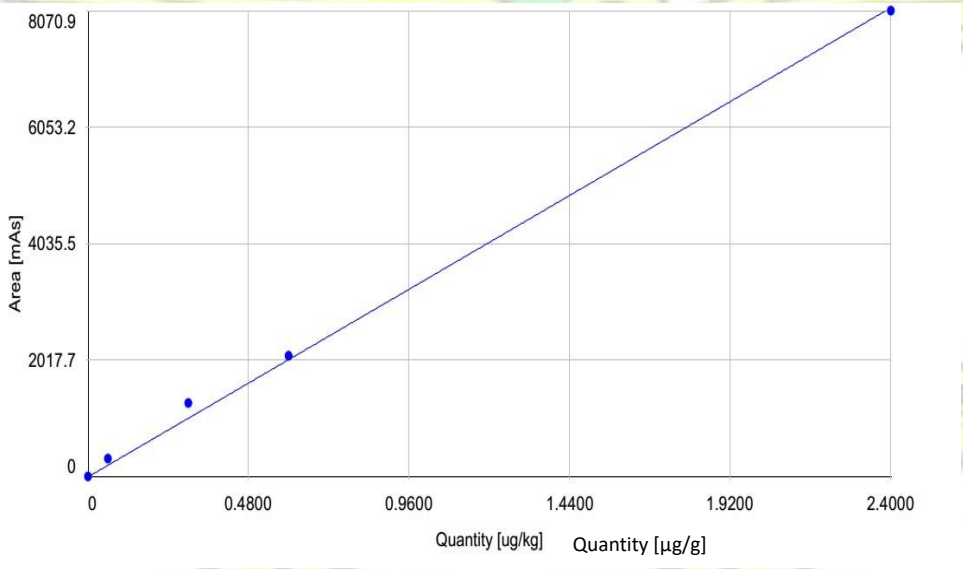
APPENDIX APPENDIX A

Preparation of acrylamide standard solution

About 1.8 mg of acrylamide standard was weighed into a beaker, and dissolved with 100 ml of distilled water. This was considered as the stock solution. From the stock solution, serial dilutions were made to a final concentration of 9 mg/ml by adding 20% acetonitrile solution. Different concentrations of 0.1, 0.5, 1, 2 and 4 mg/ml were prepared into 2 ml glass tubes from this final concentration (9 mg/ml).



A graph showing the calibration curve of acrylamide at pH of 3.5.

APPENDIX B

Estimated hazard quotients of male and female respondents from acrylamide ingestion across the day

	Breakfast		Lunch		Supper	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**
5th	0	0	0.033	0	0.0411	0
50th	0.798	1.61	1.63	0.441	1.94	0.507
95th	20.05	22.43	16.6	8.85	19.72	10.99

Estimated hazard quotients of ages of respondents from acrylamide ingestion across the day

	Breakfast			Lunch			Supper		
	Children and teenagers (5-19 years)	**Young adults 20-39 years**	**Adults 40 years and above**	**Children and teenagers (5-19 years)**	**Young adults 20-39 years**	**Adults 40 years and above**	**Children and teenagers (5-19 years)**	**Young adults 20-39 years**	**Adults 40 years and above**
5th	0	0.00416	0	0.0211	0.0213	0	0.00731	0.00293	0
50th	1.49	0.924	2.31	0.724	1.28	1.06	0.706	0.478	2.93
95th	15.55	13.04	41.58	7.19	12.81	19.93	8.83	8.74	36.95

Estimated MOE for the gender of respondents for the risk of tumorigenesis resulting from acrylamide exposures across the day

	Breakfast		Lunch		Supper	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**
5th	1.70×10^{7}	1.70×10^{7}	6.01×10^{3}	1.70×10^{7}	4.82×10^{3}	1.70×10^{7}
50th	248.538	123.1884	121.4286	449.7354	101.7964	390.8046
95th	9.883721	8.840354	11.94659	22.39789	10.05917	18.04671
Mean	42.60652	37.61062	47.88732	97.70115	40.57279	80.56872

Estimated MOE for the gender of respondents for the risk of neurotoxicity resulting from acrylamide exposures across the day

	Breakfast		Lunch		Supper	
	Male	**Female**	**Male**	**Female**	**Male**	**Female**
5th	4.30×10^{7}	4.30×10^{7}	6.51×10^{3}	4.30×10^{7}	5.23×10^{3}	4.30×10^{7}
50th	268.75	1339.564	131.9018	487.5283	1105.398	425.7426
95th	10.72319	9.585377	12.95181	24.29379	10.90264	19.56324
Mean	46.13734	40.56604	51.86972	106.1728	43.96728	87.39837

Estimated MOE for the ages of respondents for the risk of tumorigenesis resulting from acrylamide exposures across the day

	Breakfast			***Lunch***			***Supper***		
	Children and teenagers (5-19 years)	**Young adults 20-39 years**	**Adults 40 years and above**	**Children and teenagers (5-19 years)**	**Young adults 20-39 years**	**Adults 40 years and above**	**Children and teenagers (5-19 years)**	**Young adults 20-39 years**	**Adults 40 years and above**
5th	1.70×10^{7}	4.77×10^{4}	1.70×10^{7}	9.41×10^{3}	9.33×10^{3}	1.70×10^{7}	2.71×10^{4}	6.76×10^{4}	1.70×10^{7}
50th	132.8125	214.6465	85.85859	274.1935	155.9633	187.0187	280.9917	415.6479	67.72908367
95th	12.75319	15.20572	4.769921	27.58397	15.4827	9.953162	22.45707	22.69693	5.367856015
Mean	51.35952	65.1341	21.0396	108.9744	61.81818	42.71357	92.89617	97.70115	22.75769746

Estimated MOE for the ages of respondents for the risk of neurotoxicity resulting from acrylamide exposures across the day

	Breakfast			**Lunch**			**Supper**		
	Children and teenagers (5-19 years)	**Young adults 20-39 years**	**Adults 40 years and above**	**Children and teenagers (5-19 years)**	**Young adults 20-39 years**	**Adults 40 years and above**	**Children and teenagers (5-19 years)**	**Young adults 20-39 years**	**Adults 40 years and above**
5th	4.30×10^{7}	5.17×10^{4}	4.30×10^{7}	1.02×10^{4}	9.44×10^{3}	4.30×10^{7}	2.94×10^{4}	7.33×10^{4}	4.30×10^{7}
50th	144.2953	232.4324	92.87257	296.5517	168.6275	202.8302	304.9645	450.2618	73.50427
95th	13.82637	16.48773	5.168269	29.90264	16.79032	10.77694	24.33503	24.59954	5.818674
Mean	55.69948	70.72368	22.87234	117.8082	66.97819	46.28633	100.4673	105.6511	24.71264

APPENDIX C

DIETARY QUESTIONNAIRE

QUESTIONS ABOUT WHAT YOU USUALLY EAT OR DRINK

INSTRUCTIONS:

Thus questionnaire is about your eating habits over the past 1 month. Give only 1 answer for each question

PLEASE MARK LIKE THIS:

◯ ◯ ◯ ◯

BIODATA						
GENDER	**RELATION**	**RELIGION**	**EDUCATION**	**WORK**	**AGE (yrs)**	**WGT (kg)**
M ◯	Single ◯	Christian ◯	Informal ◯	Student ◯	0 0	0 0 0
F ◯	Married ◯	Muslim ◯	Basic ◯	Trader ◯	1 1	1 1 1
	Divorced ◯	Traditional ◯	JHS ◯	Civil Servant ◯	2 2	2 2 2
		Others ◯	SHS ◯	Public Servant ◯	3 3	3 3 3
			Tertiary ◯	Others ◯	4 4	4 4 4
					5 5	5 5 5
					6 6	6 6 6
					7 7	7 7 7
					8 8	8 8 8
					9 9	9 9 9

A. BREAKFAST

1. What type of food do you usually eat for breakfast?

a. ◯ I don‘t eat breakfast
b. ◯ Rice and fish
c. ◯ Rice and meat
d. ◯ Rice and chicken

2. How much do you usually eat?

a. ◯ 1 cedi
b. 1.50 cedis ◯
c. 2 cedis ◯
d. 2.50 cedis ◯
e. 3 cedis ◯
f. ◯ 3.50 cedis

e. ◯ Rice and egg g. 4 cedis
f. ◯ Indomie h. 4.50 cedis
g. ◯ Porridge and bread
h. ◯ Porridge and koose
i. ◯ Porridge and buffloaf
j. ◯ Porridge and doughnuts
k. ◯ Porridge and pinkaaso
l. ◯ Fufu and meat
m. ◯ Fufu and fish
n. ◯ Fufu and chicken
o. ◯ Banku and meat
p. ◯ Banku and fish
q. ◯ Oat and bread
r. ◯ Tombrown and bread
s. ◯ Kenkey and fish
t. ◯ etc

4. For how long (in years) have you been eating this food?

a. ◯ 0 ◯ 0
b. ◯ 1 ◯ 1
c. ◯ 2 ◯ 2
d. ◯ 3 ◯ 3
e. ◯ 4 ◯ 4
f. ◯ 5 ◯ 5
g. ◯ 6 ◯ 6
h. ◯ 7 ◯ 7
i. ◯ 8 ◯ 8
j. ◯ 9 ◯ 9

B. <u>LUNCH</u>

1. What type of food do you usually eat for lunch?

a. ◯ I don‘t eat lunch
b. ◯ Rice and fish
c. ◯ Rice and meat
d. ◯ Rice and chicken
e. ◯ Rice and egg
f. ◯ Indomie
g. ◯ Abetie and meat
h. ◯ Abetie and fish
i. ◯ Abetie and chicken
m. ◯ Banku and fish
n. ◯ Banku and meat
o. ◯ Ampesie
p. ◯ Gari and Beans
◯ 5 cedis

2. How many times do you consume this breakfast in a week?

a. ◯ Once
b. ◯ Twice
c. ◯ Thrice
d. ◯ Four times
e. ◯ Everyday

a. How many times do you consume this lunch in a week?

a. ◯ Once
b. ◯ Twice
c. ◯ Thrice
d. ◯ Four times
e. ◯ Everyday

b. For how long (in years) have you been eating this food?

a. ◯ 0 ◯ 0
c. ◯ 1 ◯ 1
d. ◯ 2 ◯ 2
e. ◯ 3 ◯ 3
f. ◯ 4 ◯ 4
g. ◯ 5 ◯ 5
h. ◯ 6 ◯ 6
i. ◯ 7 ◯ 7
j. ◯ 8 ◯ 8
j. ◯ 9 ◯ 9

C. SUPPER

1. What type of food do you usually eat for supper?

q. ◯ Tuo Zaafi and meat
r. ◯ Kenkey and fish
s. ◯ etc

2. How much do you usually eat?

a. ◯. 1 cedi
◯ b. 1.50 cedis
◯ c. 2 cedis
d. ◯ 2.50 cedis
e. ◯. 3 cedis
◯ f. 3.50 cedis
◯ g. 4 cedis
h. ◯ 4.50 cedis
i. ◯ 5 cedis

2. How much do you usually eat?

a. ◯ 1 cedi
b. ◯ 1.50 cedis
c. ◯ 2 cedis
d. ◯ 2.50 cedis
e. ◯ 3 cedis
f. ◯ 3.50 cedis
g. ◯ 4 cedis
h. ◯ 4.50 cedis
i. ◯ 5 cedis

3. How many times do you consume this supper in a week?

a. ◯ Once
b. ◯ Twice
c. ◯ Thrice
d. ◯ Four times
e. ◯ Everyday

a. at supper
b. ◯ Rice and fish
c. ◯ Rice and meat
d. ◯ Rice and chicken
e. ◯ Rice and egg
f. ◯ Indomie
g. ◯ Abetie and meat
h. ◯ Abetie and fish
i. ◯ Abetie and chicken
j. ◯ Fufu and meat
k. ◯ Fufu and fish
l. ◯ Fufu and chicken
m. ◯ Banku and fish
n. ◯ Banku and meat
o. ◯ Ampesie
p. ◯ Tuo Zaafi and meat
q. ◯ Kenkey and fish
r. ◯ etc

4. For how long (in years) have you been eating this food?

a. ◯ 0 ◯ 0
b. ◯ 1 ◯ 1
c. ◯ 2 ◯ 2
d. ◯ 3 ◯ 3
e. ◯ 4 ◯ 4
f. ◯ 5 ◯ 5
g. ◯ 6 ◯ 6
h. ◯ 7 ◯ 7
i. ◯ 8 ◯ 8
j. ◯ 9 ◯ 9