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Acrylamide Exposure and Risks in Most Frequently Consumed Foods in a Total Diet Study

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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 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.

Keywords: *acrylamide, lifetime cancer risk, margin of safety, hazard quotient*

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1. Introduction

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 [1] 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 [2]. 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 [3,4]. In order to quantify risks associated with acrylamide in foods, total diet studies should be the way forward [5,6], 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 [7,8]. 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 [9]. 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 [10]. 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 [11]. 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 [12].

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, warrants 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 [9,10]. 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 [9]. This is also partly due to the fact that, the same raw food product after processing can have variable acrylamide content [13], which makes the levels of acrylamide in foods vary significantly.

According to the World Health Organization (WHO), more than 8.8 million died from cancer globally in the year 2015 [14]. 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 [15] 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 [16], making cancer the fourth cause of death in the country. It has been documented [17] that,

20 – 50% of these 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 [1]. The fact that, it is not a food contaminant, but rather, forms during food processing, makes its presence in foods unavoidable [2], 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 [18]. There is therefore the need to determine if the concentrations of acrylamide in our frequently eaten foods are enough to pose a cancer risk or any related toxicities.

The study sought to determine the carcinogenic and neurotoxic risks associated with the consumption of the frequently consumed foods. Specifically, the project determined the food consumption data within the Kumasi metropolis, from which the dietary acrylamide exposures and risks across the day and also among sections of the consumers were evaluated.

2. Materials and Methods

2.1 Materials

2.1.1. 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.

2.1.2. Standards and Reagents

Hexane and acetonitrile were obtained from Prolabo VWR International (Paris-France) and Merck (Darmstadt, Germany) respectively. Salts ($MgSO_4$ 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).

2.2. Methods

2.2.1. 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 [19]. 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 [20]. 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 [21,22].

2.2.2. 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.

2.2.3. 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 study 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.

2.2.4. Sampling and Sample Preparation 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.

2.2.5. 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 [23]. 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.

2.2.6. HPLC Analysis

A Cecil-Adept binary pump HPLC with a Dynamic Absorbance detector was used for the HPLC analysis [24]. 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 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.

2.2.7. 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 [25]. 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, with an r^2 of 0.998.

2.2.8. 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 1 [11], based on Monte Carlo simulation (Palisade @Risk) software [26], 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 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 [27].

$$CDI = \frac{C_L \times C_R \times EF \times ED}{B_W \times AT}. \quad (1)$$

To characterize the tumorigenic and neurogenic effects resulting from the dietary exposure to acrylamide, the margin of exposure (MOE) was estimated using Equation 2 [28]. The $BMDL_{10}$ (represented the bench mark dose lower limit) values used for tumorigenesis and

neurogenicity were 0.17 and 0.43 mg/kg bw/day respectively, as proposed by regulation [9].

$$MOE = \frac{BMDL_{10}}{CDI}. \quad (2)$$

The non-cancer risk for systemic toxicity study, known as the hazard quotient (HQ) was estimated using Equation 3 [8]. 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 [29].

$$HQ = \frac{CDI}{R_iD}. \quad (3)$$

The lifetime cancer risk (R) resulting from acrylamide exposure was estimated using Equation 4 [10]. The PF used was 0.5 $(mg/kg\ day)^{-1}$, as recommended by regulation [10].

$$R = CDI \times PF. \quad (4)$$

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.

3. Results and Discussion

3.1. 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 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 [30, 31]. 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 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 were detected in porridge and bread, and the lowest, was in 'Fufu' and meat soup. Low levels were also detected in the 'Kenkey' and fish. The porridge and bread food samples contributed the greatest acrylamide content probably because of the presence of bread, since bread has been reported [32] to have high acrylamide concentrations (7×10^{-5} - 43×10^{-5} mg/g). There has also been a report [33] 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 [34]. 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 [35]. 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 [32,34].

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 [13]. 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 [9].

3.2. Acrylamide Exposure

The food consumption data profiling of consumers in the communities are also presented in Table 2 through to Table 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 2 through to Table 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 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 acrylamide exposure 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 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.

Table 2. Statistical distributions of acrylamide and elements of exposures in male respondents across the day

			Central tendency metrics				Percentiles		
	Variable	Statistical distribution	Min	Max	Mean	Mode	5th	50th	95th
BREAKFAST	Hazard (×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, 1197.9)	104	1094	531	304	192	469	982
	EF (days)	Geomet (0.0050205)	0	1196	198	364	0	208	364
	ED (years)	Geomet (0.071189)	1	65	13	10	1	8	44
	BW (kg)	Negbin (29, 0.30064)	20	100	67	64	39	68	94
LUNCH	Hazard (×10^{-3} mg/g)	Triang (0, 0, 14.946)	0	13.23	4.67	0	0	3.63	11.02
	Mass of food (g)	InvGauss (431.16, 1833.17, -18.438)	99	1031	412	199	192	347	782
	EF (days)	IntUniform (0, 364)	0	364	156	0	0	156	364
	ED (years)	Negbin (2, 0.11402)	1	73	15	10	2	10	44
SUPPER	Hazard (×10^{-3} mg/g)	Triang (0, 0, 15.867)	0	14.39	4.88	0	0	3.63	13.23
	Mass of food (g)	Triang (118.02, 231.11, 985.63)	132	884	462	208	192	391	795
	EF (days)	IntUniform (0, 364)	0	364	153	0	0	156	364
	ED (years)	Negbin (2, 0.11124)	1	73	15	5	2	10	44

Table 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 (×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	33
	BW (kg)	Negbin (24, 0.27554)	20	120	63	N/A	38	63	91
LUNCH	Hazard (×10^{-3} mg/g)	Expon (4.0003, 0.0291992)	0	13.23	4.00	0	0	3.63	11.02
	Mass of food (g)	InvGauss (325.05, 819.62, 4.1002)	66	997	329	132	104	276	736
	EF (days)	IntUniform (0, 364)	0	364	135	0	0	156	364
	ED (years)	Geomet (0.083102)	1	50	11	1	1	8	33
SUPPER	Hazard (×10^{-3} mg/g)	Triang (0, 0, 15.400)	0	14.39	4.59	0	0	3.63	13.23
	Mass of food (g)	Triang (57.949, 166.22, 971.84)	77	939	376	166	128	347	982
	EF (days)	Geomet (0.0073839)	0	364	134	0	0	156	364
	ED (years)	Geomet (0.078902)	1	50	11	5	1	8	33

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 2). This observation seems to be in line with the report previously published by the Norwegian Food Control Authority [36], 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).

The exposure of acrylamide by the three age groups (Table 4, Table 5 and Table 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.

3.3. 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 2, and was consumed during breakfast. The 5th percentile 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 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 [37]. 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 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 50th percentile group was 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 [38]. 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 [38,39,40,41]. In contrast however, in countries such as Germany, Portugal, Finland, Sweden, Hungary and Russia, lunch is the main meal of the day [42,43,44,45,46]. 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, Table 5 and Table 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 [47].

Table 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 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 (×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 (×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 (×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 6. Statistical distributions of acrylamide and elements of exposures of adults (40 years and above) across the day

			Central tendency metrics				Percentiles		
	Variable	Statistical Distribution	Min	Max	Mean	Mode	5th	50th	95th
BREAKFAST	Hazard ($\times10^{-3}$ mg/g)	Uniform (-0.21478, 14.605)	0	14.39	5.65	0	0	6.17	14.39
	Mass of food (g)	ExtValue (376.34, 165.53)	138	1094	470	138	138	437	875
	EF (days)	Geomet (0.0051422)	0	1196	193	N/A	0	156	364
	ED (years)	Geomet (0.042929)	1	65	22	N/A	2	25	50
	BW (kg)	Negbin (57, 0.43769	48	120	73	N/A	52	72	96
LUNCH	Hazard ($\times10^{-3}$ mg/g)	Uniform (-0.20354, 13.434	0	13.23	0.52	0	0	3.63	13.23
	Mass of food (g)	InvGauss (414.21, 1279.35, -29.035)	66	1031	385	28	138	317	782
	EF (days)	Geomet (0.0076798)	0	364	129	0	0	156	364
	ED (years)	Geomet (0.043781)	1	73	21	N/A	2	20	50
SUPPER	Hazard ($\times10^{-3}$ mg/g)	Uniform (-0.22484, 14.615)	0	14.39	6.75	0	0	8.76	13.23
	Mass of food (g)	Triang (62.175, 347.23, 906.19)	104	830	444	347	154	407	782
	EF (days)	IntUniform (0, 364)	0	364	163	156	0	156	364
	ED (years)	Geomet (0.040767)	1	73	23	30	1	22	50

3.4. 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 7, Table 9 and Table 10. The mean and 95th percentile 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 7 and Table 9). This chronic intake seems to be slightly higher compared to the estimated intakes from many other countries, as reported in Table 9. 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 [48]. For instance, a decrease in the concentration of acrylamide in foods has been reported [48], following an increase and decrease of the sulphur and nitrogen levels in the soil respectively.

Table 9 shows a very interesting observation. It appears the adults age group (40 years and above) recorded the greatest chronic exposures across the study. 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 [55] 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 [32], 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 [56,57] lower chronic exposures of children and teenagers.

Table 7. Chronic dietary exposures of male and female respondents for the risk of tumorigenesis and neurotoxicity

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

Table 8. Dietary acrylamide exposures of respondents from different countries

Country	Acrylamide exposure (mg/kg bw/day)		References
	Mean	95th Percentile	
Italy	4.52×10^{-4}	1.54×10^{-3}	[37]
Canada	5.80×10^{-4}	2.19×10^{-3}	[49]
France	4.30×10^{-4}	1.02×10^{-3}	[50]
Belgium	3.50×10^{-4}	1.12×10^{-3}	[51]
China	2.86×10^{-4}	4.90×10^{-4}	[52]
United States of America	4.30×10^{-4}	1.30×10^{-3}	[53]
The Netherlands	4.80×10^{-4}	6.0×10^{-4}	[5]
Sweden	3.10×10^{-2}	6.20×10^{-2}	[54]

Table 9. Respondent age groups and their chronic dietary exposures for the risk of tumorigenesis and neurotoxicity

	Exposures during breakfast (mg/kg-day)			Exposures during lunch (mg/kg-day)			Exposures 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
Tumorigenesis									
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}
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}
Neurotoxicity									
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}
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}

Exposures to acrylamide in foods depend on variable factors including; the population, age of consumers and their eating preferences [58]. Thus, it is these factors that form the basis of variations in the exposures to acrylamide.

3.5. Risk Characterization

3.5.1. 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 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.

In a report on the risk of acrylamide in Romanian food [59], 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 [54,59] 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 [29]. 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). 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.

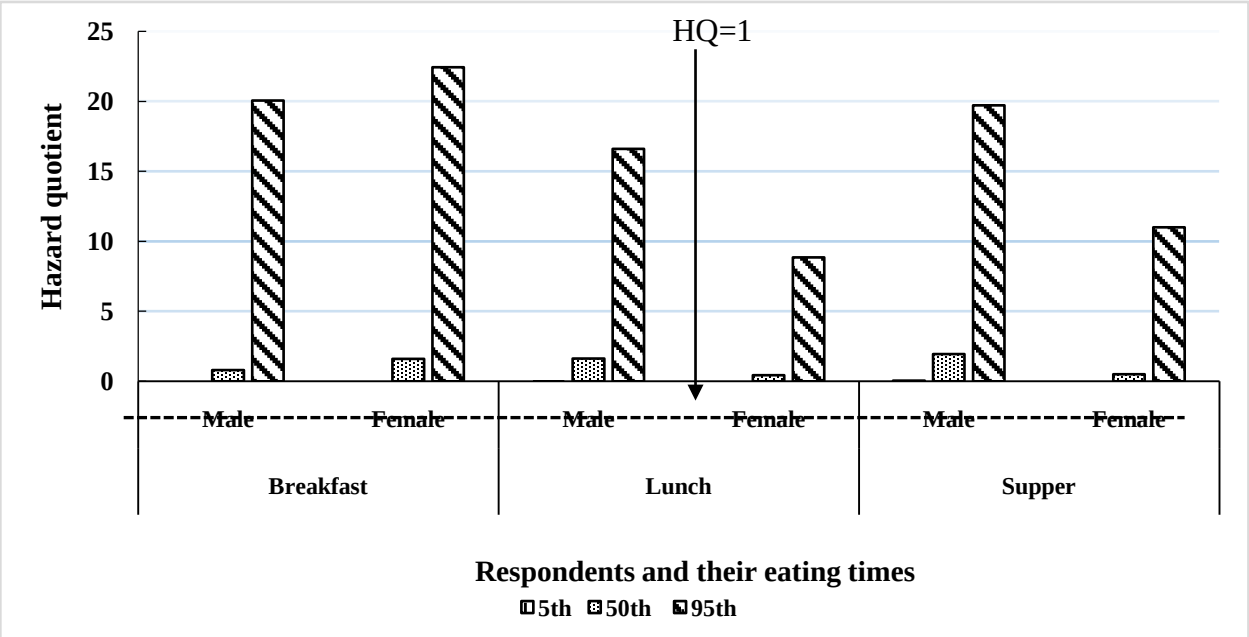

Figure 1. Estimated hazard quotients of male and female respondents from acrylamide ingestion across the day

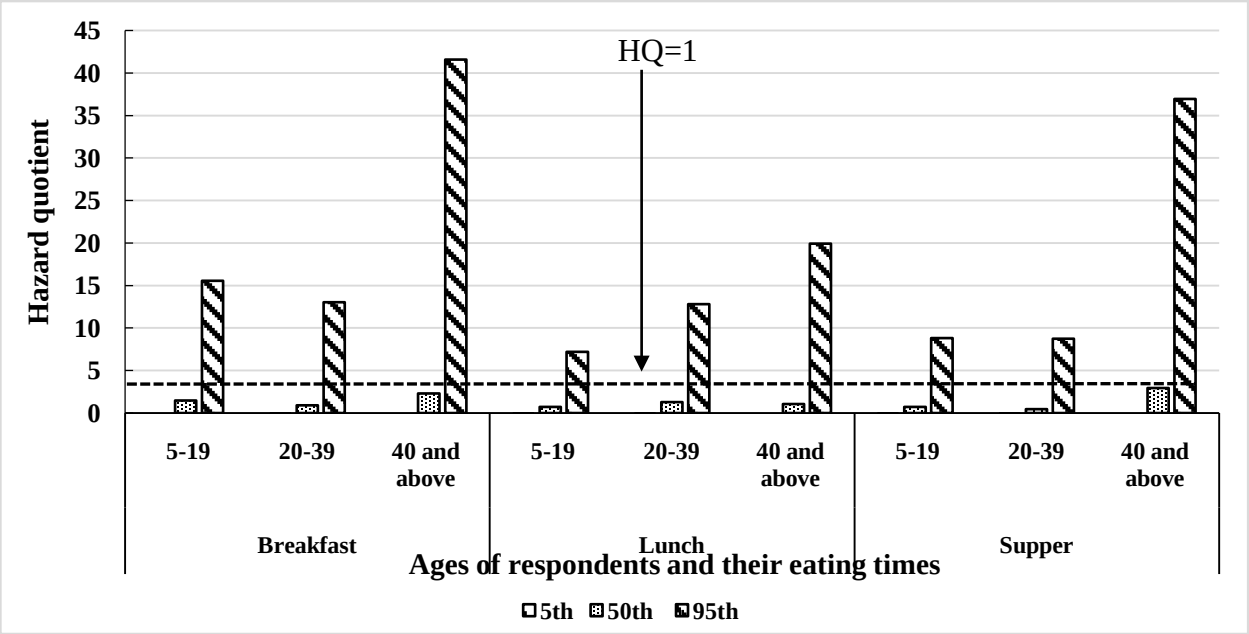

Figure 2. Estimated hazard quotients of age groups of respondents from acrylamide ingestion across the day

3.5.2. 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 3, Figure 4, Figure 5 and Figure 6). This range is not surprising, because of reported MOE values, ranging from 129 to 1.43×10^7 [13]. Another study however, reported of less varied MOE values, ranging between 110.5 and 951.3 [37]. For the risk of tumorigenesis, all the female 5th percentile consumers recorded MOE values which were above the threshold (10,000) mark, thus, showing safe levels across the day (Figure 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 5th 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 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 3), thus, implying risk, since the MOE reported (89 – 425) is below the 10,000 threshold mark recommended by the European Food Safety Authority [9]. Studies on the risk assessment of the dietary exposure to acrylamide in the Norwegian population also revealed an MOE of 189 [60]. However, one study [37] 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 3), which is much higher than the range 50 and 283, reported by the European Food Safety Authority [9], hence implying higher risk of tumorigenesis. In other studies, the 95th percentile exposures of 106 and 110.46 have been reported [37,60] 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 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 [9] 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 5). The mean exposure MOE of the consumers ranged from 40.57 to 106.17 (Figure 5), which is much lower than the values reported by the European Food Safety Authority [9] to be in the range of 226 to 1075. This value is also below the safety limit or threshold of 125 set by regulation [9], and thus, poses serious health concern. The 95th percentile consumers recorded MOEs ranging between 9.59 and 24.29 (Figure 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 [9]. 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 6), hence posing public health concern. These values were again, lower than the values reported by the European Food Safety Authority [9].

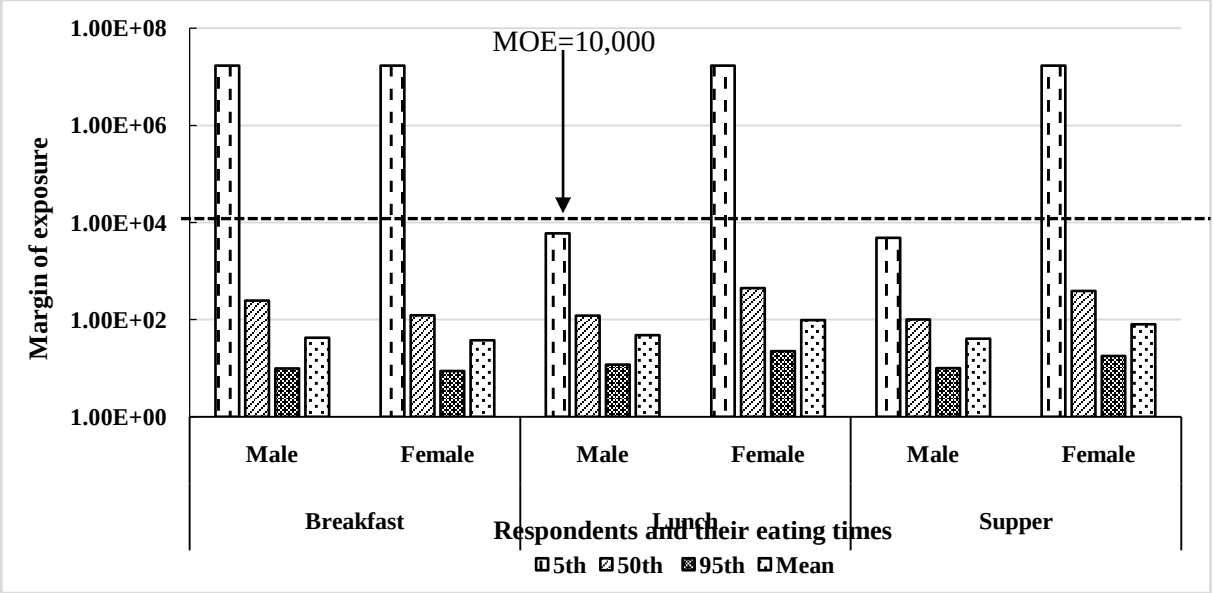

Figure 3. Estimated MOE for the gender of respondents for the risk of tumorigenesis resulting from acrylamide exposures across the day

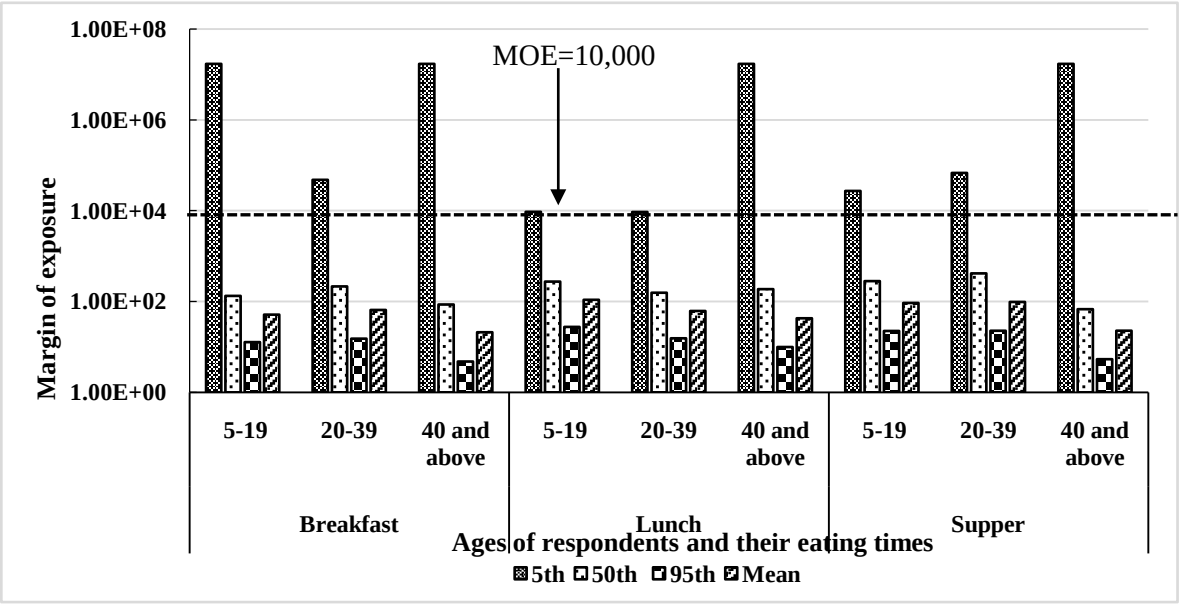

Figure 4. Estimated MOE of age groups of respondents for the risk of tumorigenesis resulting from acrylamide ingestion across the day

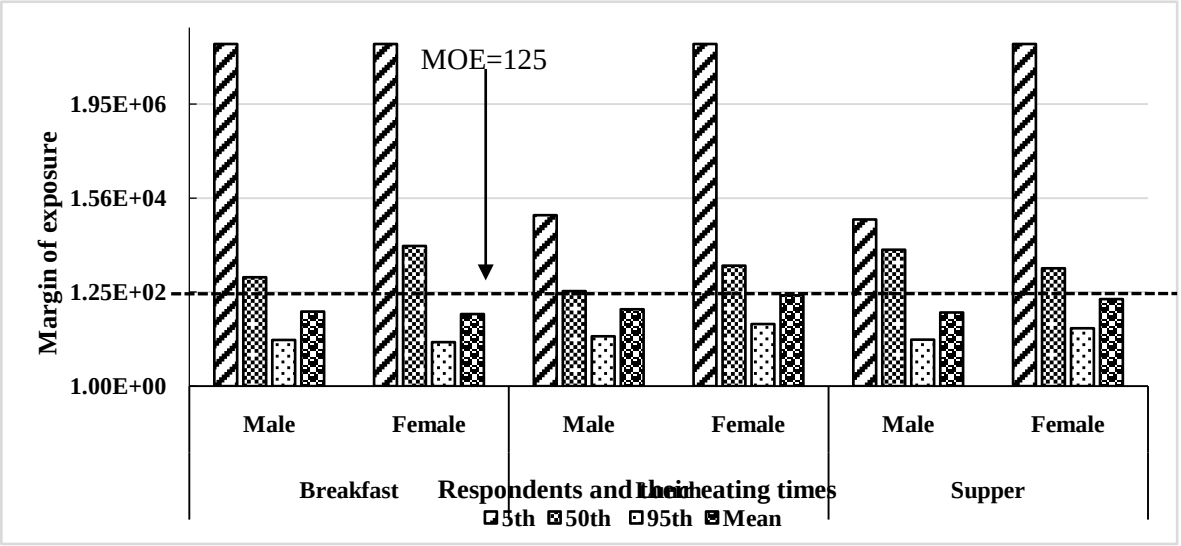

Figure 5. Estimated MOE for the gender of respondents for the risk of neurotoxicity resulting from acrylamide ingestion across the day

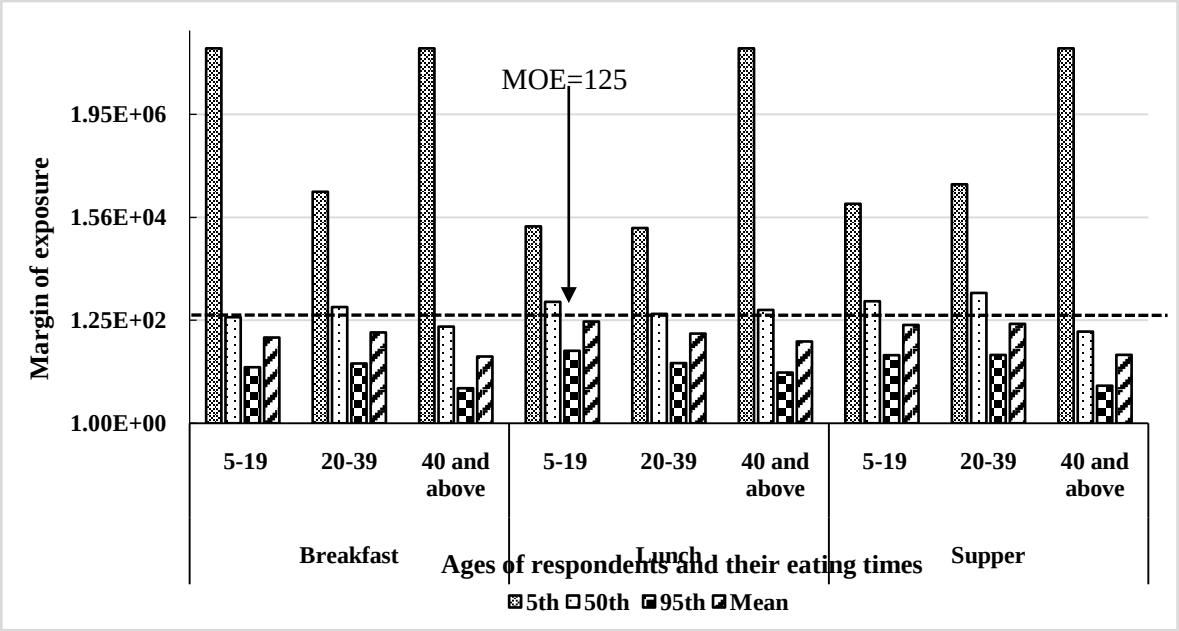

Figure 6. Estimated MOE of age groups of respondents for the risk of neurotoxicity resulting from acrylamide ingestion across the day

3.5.3. 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}. Male consumers were generally at a higher risk compared to female consumers across the day (Table 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}) [61]. 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}) [25].

Table 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 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 is similar to a study, where the 50th (0.5×10^{-3}) percentile consumers had lifetime cancer risk levels greater than the *de minimus* [61].

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 5th and 50th percentile male consumers was recorded during breakfast, followed by lunch and supper (Figure 7). On the other hand, the lowest cancer risk for the 5th and 50th percentile female consumers were recorded during lunch, followed by supper and breakfast (Figure 8). The lower amount of food mostly consumed by these respondents (Table 3) during lunch could probably be the reason for the lower impact of the lunch elements on the cancer risk of the female consumers.

The cancer risk levels pertaining to acrylamide dietary exposure of the three age group respondents are presented in Table 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 11).

Observations from the regression studies show that, the variable that contributed mostly to the cancer risk was the exposure duration (ED) (Table 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 [25].

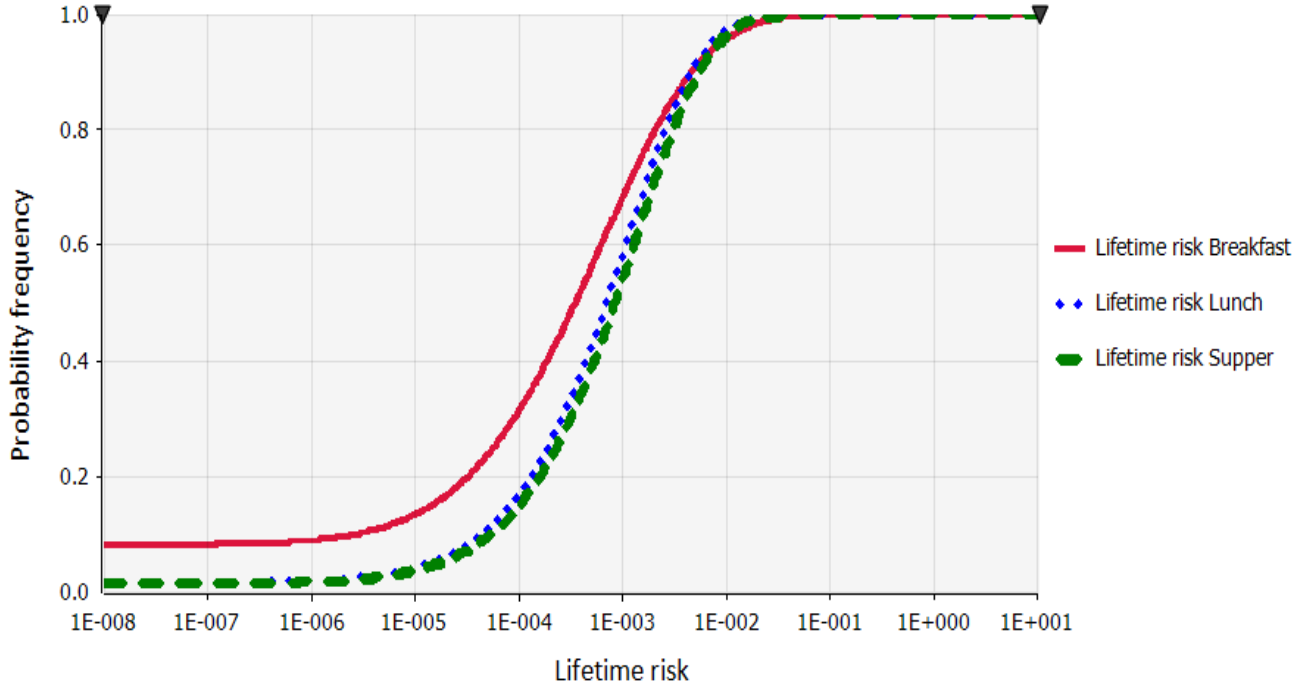

Figure 7. Estimated lifetime cancer risks of male respondents resulting from acrylamide ingestion across the day

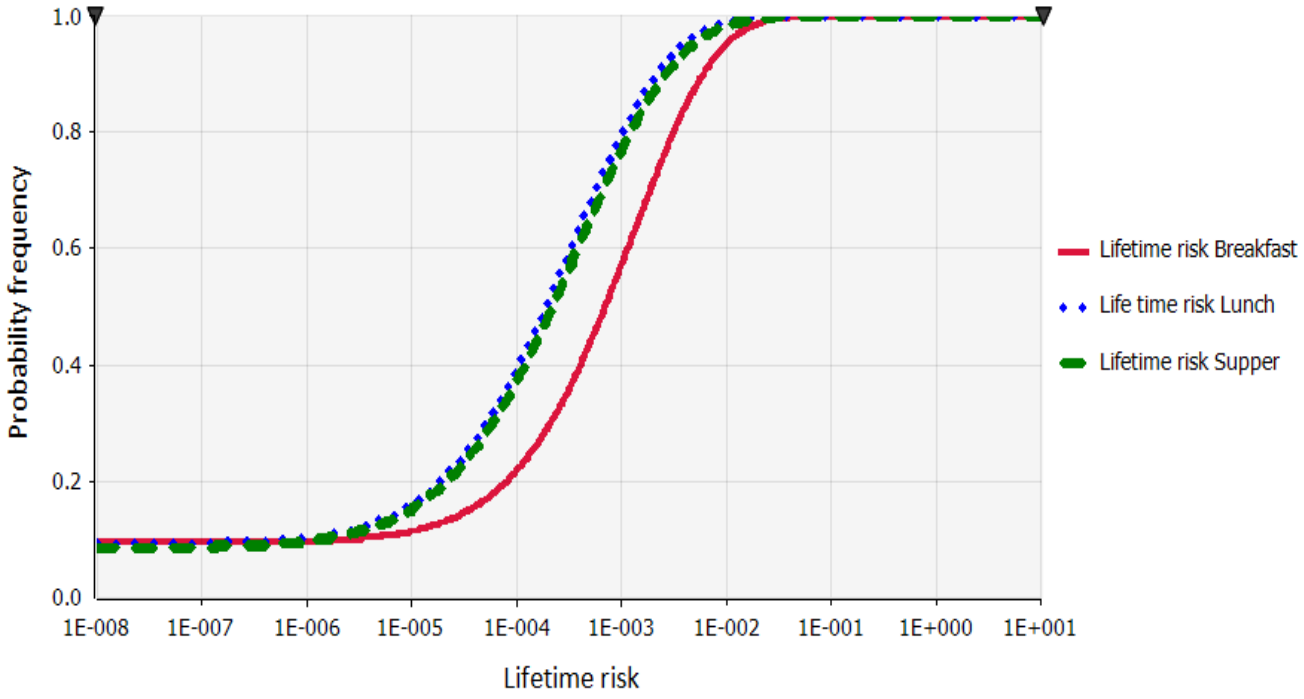

Figure 8. Estimated lifetime cancer risks of female respondents resulting from acrylamide ingestion across the day

Table 11. Estimated lifetime risks of the ages 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)
5^{th}	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}
50^{th}	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}
95^{th}	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}

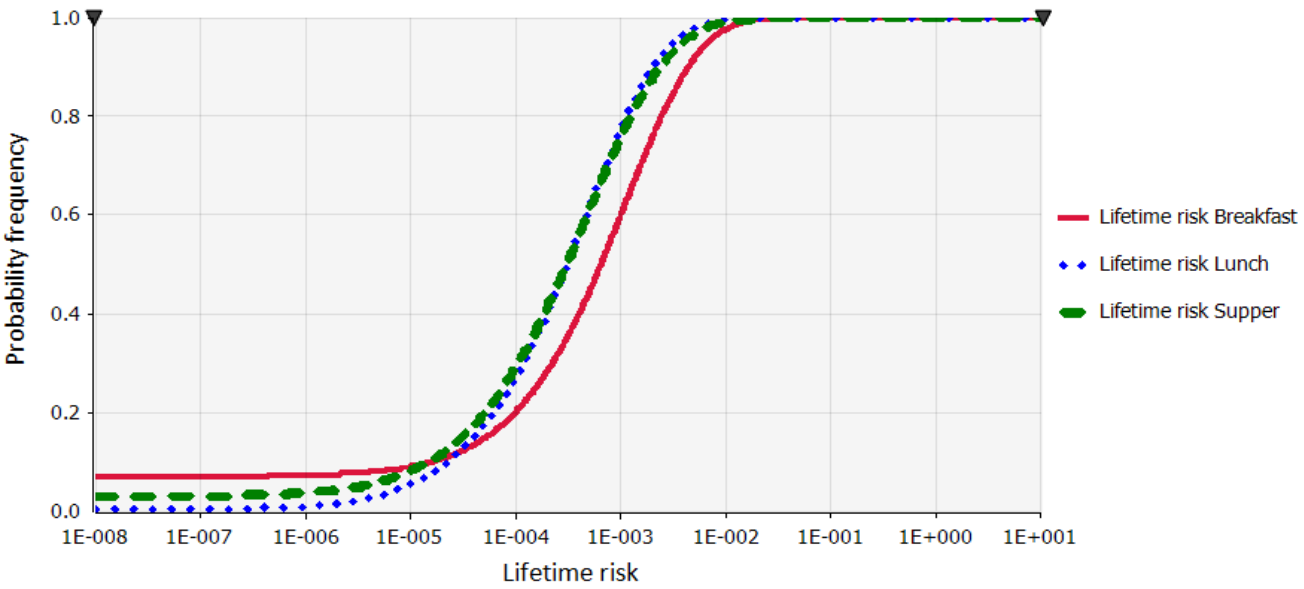

Figure 9. Estimated lifetime risks of children and teenagers (5-19 years) resulting from acrylamide ingestion across the day

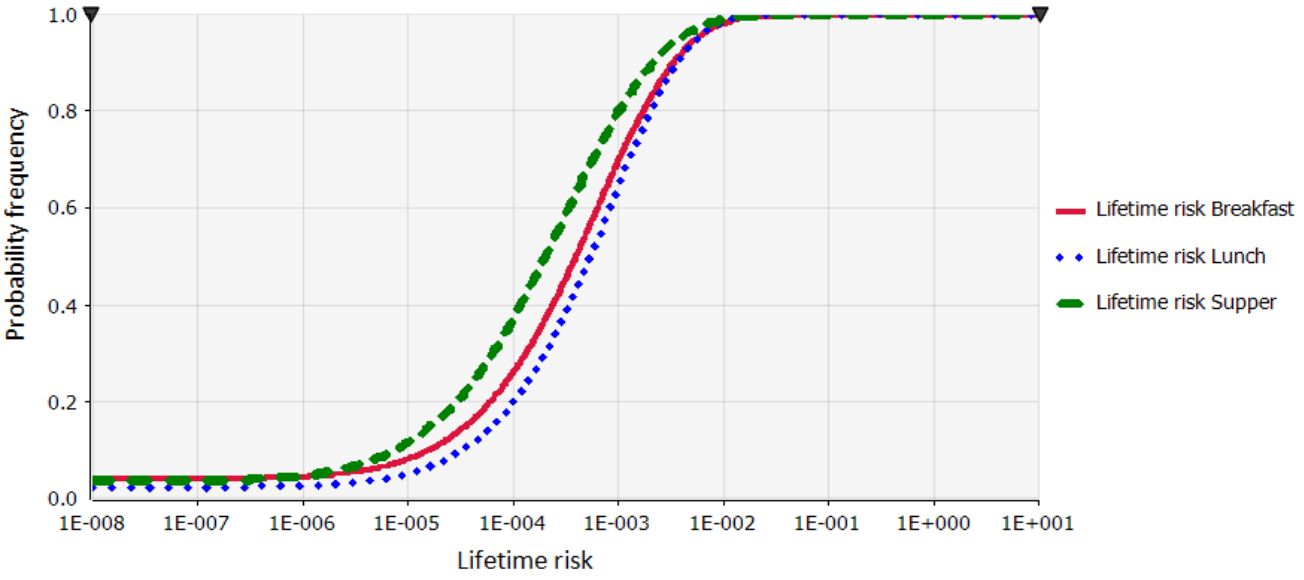

Figure 10. Estimated lifetime risks of young adults (20-39 years) resulting from acrylamide ingestion across the day

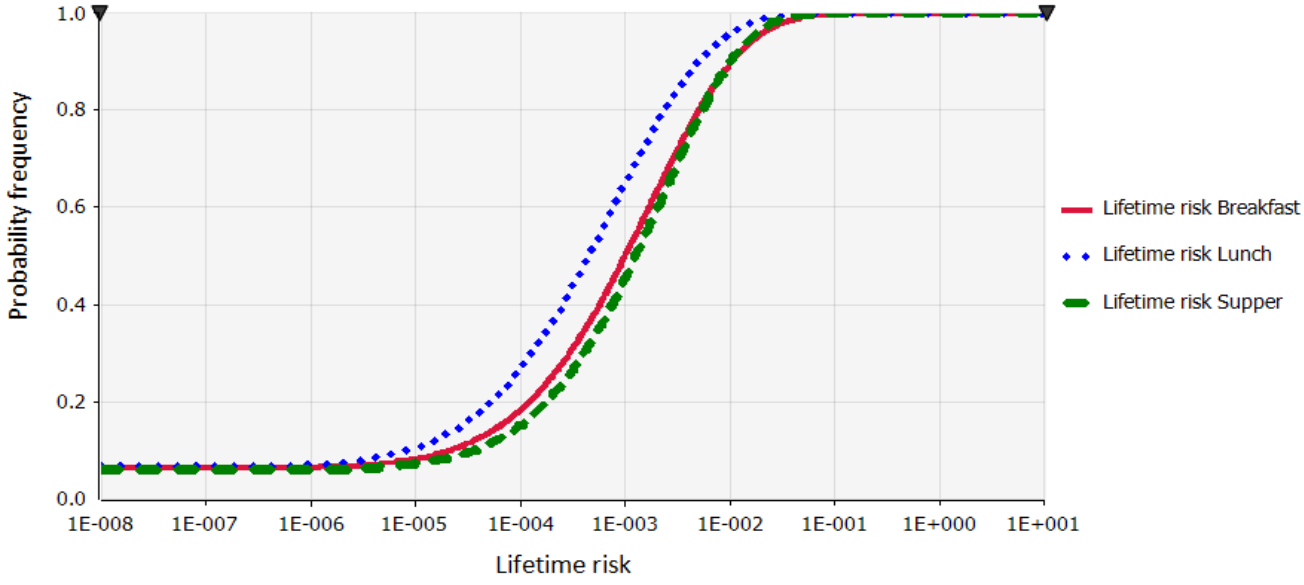

Figure 11. Estimated lifetime risks of adults (40 years and above) resulting from acrylamide ingestion across the day

Table 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)	B_W (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

4. 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 risks. The adult group were 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. Since it would be difficult to control ED, targeting the concentration of acrylamide in the foods, and finding ways to reduce them can be one plausible step, to control their levels in foods. Thus, mitigation studies should be seriously mounted in order to save the lives of the adults and teenagers groups that are at risk.

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Conflict of Interest

None.

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