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Review article

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Dietary acrylamide-linked burden of cancers in four sub-sahara African countries: A review and data synthesis

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ABSTRACT

Acrylamide (AA) is a food processing byproduct that forms at high temperatures and is classified as a probable human carcinogen. Previous studies have linked AA to kidney, uterus, and ovary cancer burdens, but its study in African countries remains underexplored. This study systematically used six recent articles on dietary AA concentration data from scholarly databases using specific search terms. We also collected health metrics secondary data from the Institute of Health Metrics and Evaluation and other sources for the period 2015–2019. We used a Monte-Carlo simulation to integrate the dietary AA exposure, risks, and health metrics to estimate the cancer burdens. The results showed that the modal healthy life years lost ranged from 0.00488 (Ghana) to 0.218 (Ethiopia) per 100,000 population. The median statistic indicated 1.2 and 26.10 healthy life years lost for Ghana and Ethiopia, respectively, due to the three cancer types. The four-country study areas' total disability-adjusted life years (DALYs) were 63.7 healthy life-year losses. Despite the limitations of the non-standardized age-related food consumption data and the few inclusive articles, the probabilistic approach may account for the uncertainties and provide valid conclusions.

1. Introduction

Acrylamide (AA) is a vinyl-substituted primary amide, classified as group 2 A "probable human carcinogen" by the International Agency for Research on Cancer (IARC) [1]. Studies have shown that AA forms through the Maillard reaction when food groups such as carbohydrates, fats, or proteins are thermally processed by baking, roasting, frying, or grilling, especially at high temperatures [2,3]. Acrylamide (AA) can form from various food components through different mechanisms. One of the main mechanisms is the Maillard reaction, which occurs in starchy foods when reducing sugars and the amino acid asparagine react at high temperatures. This reaction involves a series of steps: first, the reducing sugars and the amino group form N-glycoside, also known as a Schiff's base; second, the N-glycoside rearranges to an Amadori compound; third, the Amadori compound decarboxylates and degrades into acrylamide and other products [4–6]. Some of the intermediate products of the Maillard reaction, such as 3-amino propionamide, can also be converted to acrylamide in aqueous conditions. Therefore, it is unsurprising that dietary AA has been detected in pasteurized fruit juices [7]. The Maillard reaction is driven by heat, so it is ubiquitous in many foods that undergo roasting, baking, frying, or toasting, such as

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Fig. 1. Map of the West and East African Regions highlighting Ghana, Nigeria, Ethiopia, and Kenya.

cocoa beans, coffee, cereal products, potato crisps, baked goods, and baby foods [8–15]. Since the bulk of the human diet is obtained from such food categories, it raises a concern that everybody may be at risk of dietary AA exposure [16,17]. Exposure to acrylamide (AA) has various effects on human health, especially when it is biotransformed during its metabolism after ingestion. Some studies have reported that AA binds to hemoglobin and forms adducts in the blood [18,19] and long-term exposure can also result in nerve damage [20,21]. Moreover, there is evidence of a link between AA exposure and reproductive problems, especially in animal models where sperm motility has been impaired [22]. The toxicity of AA is enhanced when it is biotransformed by cytochrome P450 2E1 (CYP2E1) to the genotoxic epoxide glycidamide [23]. This epoxide is highly reactive and attacks DNA to form an adduct at the nitrogen 7 of guanine (N⁷-GA-Gua), which indicates a potential carcinogenic pathway [24]. The dose-response relationships of cancers associated with dietary AA are uncertain [25]. However, while some authors have reported weak or no statistical significance between dietary AA exposure and cancer, others have reported a significant increase in endometrial, kidney, and ovarian cancers [26,27].

Traditionally, risk assessors often use either the margin of exposure (MOE) or excess lifetime cancer risks (ELTCR) to report carcinogenic risks. While MOE values $<10^4$ indicate high public health concerns [28], ELTCR values $>10^{-4}$ indicate significant risks [29]. Unfortunately, this method of reporting risks has little impact on accounting for the overall disease burden. However, the burden of disease approach can convert risks into morbidity and mortality. Thus, it can provide determinant information that indicates the population's health loss as a function of time [30]. Expressed as the Disability-adjusted life years (DALYs), it presents as the sum of the number of years lived with disability (YLDs) and the number of years of life lost (YLLs) due to premature mortality (Equation (1)). The YLDs are estimated by multiplying the number of deaths and life expectancy at the age of death [31]. The DWs express the proportional reduction of quality of life subject to sequelae scaled from 0 to 1, where 0 equals perfect health and 1 equals death [32].

YLL + YLD = DALYs.

(1)

Although methodological approaches limit the comparability of resulting DALY estimates across studies, the DALY identifies essential health gaps and highlights risk factors that policymakers may overlook. The Institute of Health Metrics and Evaluation (IHME) performs frequent updates of the Global Burden of Disease (GBD) study, which aims to determine the incidence, prevalence, YLL, YLD, and DALYs of several diseases and risk factors [33]. Some studies have suggested that dietary AA intake may be linked to certain types of cancer; thus, this information has influenced public health policies in some regions where AA exposure is high [34–36]. However, the situation in African countries is poorly reported and lacks data for similar policies on dietary AA and cancer risk. This knowledge gap has led to inconsistent public education and a possible public health crisis due to dietary AA-related cancer burden. This study supports the Sustainable Development Goal 3 (SDG3), which aims for good health and well-being for all people of all ages, thus creating the need for this work. Therefore, we estimated the Disability adjusted life years (DALYs) rates due to dietary AA exposure in four sub-Saharan African countries: Ghana, Nigeria, Ethiopia, and Kenya.

2. Methods

2.1. Exposure to acrylamide

The concentration of dietary acrylamide: We reviewed literature using the PRISMA and Cochrane guidelines to obtain the distribution of dietary AA across the selected sub-Sahara African region [37,38] where the study occurred. Details of the process are captured in a file (Supplementary 1) with the corresponding MS Excel file Supplementary 1 .xlsx) available in the link provided.

Food consumption data: We collected food consumption data of the four countries under study (Fig. 1) from two database sources relating to the food categories identified and corresponding to the AA-contaminated foods obtained from the literature search. These

food categories were sourced from the Food and Agricultural Organization (FAO)/World Health Organization (WHO) Global Individual Food consumption data Tool (GIFT) database (www.fao.org/gift-individual-food-consumption/data/en) and the WHO/(Global Evaluation and Monitoring Systems (GEMS) Food Cluster Diets database (www.who.int/data/gho/samples/food-cluster-diets).

Though the health metrics data was collected from 2015 to 2019, the food consumption data was collected from 2012 to 2019. The decision was based on the assumption that food eating habits have a significant environmental and social impact based on solid cultural dynamics and sustainable eating habits [39]. Thus, we assumed that the food consumption pattern of the consumers would stay the same within the study period over a short time. For the two East African countries, the food categories identified from the foods reporting AA concentrations included beverages (cocoa and coffee), cereals and their products, roots, tubers, plantains and their products, and fats and oils. The food categories represented in the foods from the two West African countries included sweets and sugars, fats and oils, pulses, seeds and nuts, cereals and their products, roots and tubers, and plantains and their products. Thus, the masses consumed daily for the relevant food categories were collected from WHO/GIFT and WHO/GEMS foods.

Body weight: We used the WHO standard default average body weight (60 kg) [40] to determine the exposure. With the various elements of exposure (concentration of dietary AA, mass of food ingested and body weight) in place, we determined the probabilistic distributions of the dietary AA.

2.2. Kidney, ovary and uterus cancer data

This study investigated the association between dietary AA exposure and the risk of kidney, ovarian, and uterine cancers. We were motivated by meta-analyses that reported a modest increase in the risk of renal cancer [41] and a recent study that suggested a positive linear relationship between dietary AA exposure and the risk of ovarian and uterine cancers [25]. We synthesized the evidence on the association between dietary AA exposure and the risk and burden of these cancers based on the prevalence, mortality, YLL, and YLD of these cancers in Ethiopia, Kenya, Ghana, and Nigeria from 2015 to 2019 from the Global Burden of Disease (GBD) Compare database [33]. All data were collected by age categories (5–19, 20–54, and 55–89 years). The kidney cancer data were collected for both males and females.

2.3. Probabilistic approach for data analysis

Based on the recommendation of The National Research Council Science and Judgement, we used the Monte Carlo simulations model to enumerate the associated uncertainties while harmonizing the estimates using iterations (10⁵) in simulation [42]. In doing so, the uncertainties and the variabilities were quantified as a minimum, maximum, mode, mean, median and various percentiles [43]. In this study, the Palisade @Risk software tools were used to fit the distributions of all the variable input datasets collected within the 5-year study period (2015–2019), which were documented and available through the link provided (Supplementary 2 .xlsx). Among the expressed outcomes, we collected the mode (most frequently occurring event) and the 50th percentile (median) and studied them. These central tendencies were selected to avoid biases often arising from outliers [44].

2.4. Determination of dietary acrylamide exposure and cancer risk assessment

We estimated chronic exposure to AA in food as the product of the concentration of the hazard and the mass of food ingested per body weight per day [45,46]. Thus, the distribution of the estimated exposure (E_{EST}) of dietary AA among the population in the study area was determined using Equation (2):

$$E_{EST} = \frac{AA_{SRL} \times M_F}{B_W},\tag{2}$$

where the AA_{SRL} and M_F are, respectively, the distributions of the concentration of dietary AA (mg/kg) and the mass of foods consumed (kg/day) reported in the study, and body weight (BW) is 60 kg, as recommended by the WHO [40]. Subsequently, we determined the distributions of the risk of years of life lost (R_{LL}) and the risk of years of life with a disability (R_{LD}) of each specific cancer endpoint using Equations (3a) and (3b).

$$R_{LL} = CSF_{LL} \times E_{EST}, \tag{3a}$$

$$R_{LD} = CSF_{LD} \times E_{EST,}$$
(3b)

where *CSF* is the cancer slope factor for a specific cancer endpoint expressing the lifetime risk of cancer per unit exposure. The US EPA indicates an AA cancer slope factor of 0.51 per mg/kg-day [47]. However, for each of the specific cancers studied, their scaled cancer slope factors (CSF_{sp}) were determined using their mortality (Equation (4a)) and prevalence (Equation (4b)). Since the CSF is not the same for each cancer endpoint, they were derived as illustrated in other studies [24,35] and exemplified in Equations (4a) and (4b):

$$CSF_{sp} = CSF \frac{Mort_{sp}}{Mort_{tot}} = CSF_{LL},$$
(4a)

$$CSF_{sp} = CSF \frac{Prev_{sp}}{Prev_{tot}} = CSF_{LD_{s}}$$
(4b)



Fig. 2. (a) Schematic pathways showing dietary AA-induced cancer risks expressed as YLL rates. (b) Schematic pathways showing dietary AA-induced cancer risks expressed as YDL rates.

*Mort*_{sp} and *Prev*_{sp} are the mortality and prevalence of a specific cancer endpoint, and *Mort*_{tot} and *Prev*_{tot} are the total mortality and prevalence during the five-year study period (2015–2019).

2.5. Burden of disease

This current study was designed to use three variable elements: concentration of dietary AA, mass of food ingested per day, and body weight. These elements were used to compute dietary AA exposure (Equation (2)), which were then converted to cancer risks of YLL (R_{LL}) and that of YLD (R_{LD}) (Equations (3a) and (3b)). Next, to obtain the average years of life lost (YLL) and years lived with disability (YLD) due to kidney, ovary, and uterus cancer cases during the five years, we used Equations (5a) and (5b), respectively. Equation (5a) calculates the YLL per fatal case (YLLpp) by incorporating each specific cancer's total YLL and mortality rate. Equation (5b) calculates the YLD per case (YLDpp) by incorporating the total YLD and the prevalence of each specific cancer. Thus, the dietary AA attributable to the YLL and YLD of each specific cancer and its corresponding rate (per 100,000 persons) were computed. In (Fig. 2), we illustrate the nexus connecting dietary AA exposures and the health metrics to yield the rates resulting from YLL and YLD, the components of DALY rates. The details are provided in the Supplementary Table 4s.

$$YLL_{pp} = \frac{YLL_{sp}}{Mort_{sp}},$$
(5a)

$$YLD_{pp} = \frac{YLD_{sp}}{Prev_{sp}},$$
(5b)

Subsequently, the five-year range of the specific cancer prevalence (5-YRC_{sp} same as F-YRC_{sp}), YLL (*F*-YRC_{sp} _{LL}) and YLD (*F*-YRC_{sp} _{LD}) attributable to dietary AA exposure were determined according to Equations (6a) and (6b): where N_{pop} and LE_{pop} are the



Fig. 3. Mass of food consumed from the two Eastern African countries (expressed in Log10 of mass of food (kg/day)).



Fig. 4. Mass of food consumed from the two Western African countries (expressed in Log₁₀ of mass of food (kg/day)).

specific national populations [48] and their age-related life expectancies [49], respectively.

$$F-YRC_{sp\ LL} = \frac{N_{pop}}{LE_{pop}} \times R_{LL},$$
(6a)
$$F-YRC_{sp\ LD} = \frac{N_{pop}}{LE_{pop}} \times R_{LD},$$
(6b)

Thus, the dietary AA-induced YLLs and YLDs distributions per specific cancer were subsequently derived according to Equations



Fig. 5. Acrylamide concentrations ingested from food categories derived from the two East African countries.



Fig. 6. Acrylamide concentrations ingested from food categories derived from the two West African countries.

Table 1	
Statistical outcomes of acrylamide ingestion and estimated exposures in Eastern and Western African countries.	

	Ethiopia-Kenya		Ghana-Nigeria			
	Acrylamide (mg/kg)	Exposure mg/kg (bw)-day	Acrylamide (mg/kg)	Exposure mg/kg (bw)-day		
Mode	7.81	4.07×10^{-6}	$5.54 imes10^{-2}$	$1.03 imes 10^{-5}$		
Median	4.93	1.64×10^{-3}	2.56×10^{-1}	3.19×10^{-4}		

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Table 2a

Modal and median risk of Years of Life Lost and Years of Life lived in disability of kidney, ovarian and uterine cancers of age groups in the two East African countries.

		Risk of Year	rs of Life Lost		ability				
		Kidney Cancer		Ovarian Cancer	Uterine Cancer	Kidney Cancer		Ovarian Cancer	Uterine Cancer
		Ethiopia							
Age Group	Gender Statistics	Male	Female	Female	Female	Male	Female	Female	Female
5–19	Mode	$1.78 imes$ 10^{-7}	1.91 imes 10 ⁻⁷	$5.08 imes 10^{-7}$	0.00	1.99×10^{-7}	$rac{2.82 imes}{10^{-7}} imes$	1.01×10^{-6}	0.00
	Median	$1.86 imes 10^{-4}$	2.00 imes 10 ⁻⁴	$\begin{array}{c} 5.26 \times \\ 10^{-4} \end{array}$	0.00	$2.05 imes$ 10^{-4}	2.95 imes 10 ⁻⁴	$\textbf{1.04}\times \textbf{10}^{-3}$	0.00
20–54	Mode	$\frac{1.37}{10^{-6}}\times$	9.31×10^{-7}	$9.28\times \\10^{-6}$	1.63×10^{-6}	$7.17 imes 10^{-7}$	$6.77 imes 10^{-7}$	$\textbf{6.46}\times \textbf{10}^{-6}$	$\textbf{2.11}\times \textbf{10}^{-6}$
	Median	$\begin{array}{c} 1.42 \times \\ 10^{-3} \end{array}$	$9.69 imes$ 10^{-4}	$9.63 imes 10^{-3}$	$\textbf{1.68}\times \textbf{10}^{-3}$	$\begin{array}{c} \textbf{7.42}\times\\ \textbf{10}^{-4} \end{array}$	$7.00 imes 10^{-4}$	$\textbf{6.69}\times \textbf{10}^{-3}$	$\textbf{2.19}\times \textbf{10}^{-3}$
55–89	Mode	$5.72 imes$ 10^{-6}	$3.77 imes 10^{-6}$	$1.49 imes$ 10^{-5}	$\textbf{6.79}\times 10^{-6}$	$9.42 imes$ 10^{-7}	$7.38 imes 10^{-7}$	$\textbf{3.23}\times 10^{-6}$	$\textbf{2.76}\times \textbf{10}^{-6}$
	Median	$5.93 imes$ 10^{-3}	$3.90 imes 10^{-3}$	$1.55 imes 10^{-2}$	$\textbf{7.04}\times 10^{-3}$	$9.73 imes$ 10^{-4}	$7.64 imes 10^{-4}$	$\textbf{3.33}\times \textbf{10}^{-3}$	$\textbf{2.88}\times \textbf{10}^{-3}$
Kenva									
5–19	Mode	$\begin{array}{c} 1.39 \times \\ 10^{-7} \end{array}$	1.68×10^{-7}	$4.73 imes 10^{-7}$	0.00	$2.90 imes 10^{-7}$	$4.03 imes$ 10^{-7}	1.57×10^{-6}	0.00
	Median	1.87 imes 10 ⁻⁴	$rac{2.23}{10^{-4}} imes$	$6.30 imes$ 10^{-4}	0.00	$2.15 imes$ 10^{-4}	2.99 imes 10 ⁻⁴	1.16×10^{-3}	0.00
20–54	Mode	$1.38 imes 10^{-6}$	$rac{1.13 imes}{10^{-6}} imes$	$1.25 imes$ 10 $^{-5}$	$\textbf{2.17}\times \textbf{10}^{-6}$	$rac{1.61}{10^{-6}} imes$	$rac{1.64}{10^{-6}} imes$	1.62×10^{-5}	$\textbf{6.07}\times 10^{-6}$
	Median	1.84×10^{-3}	$1.49 imes 10^{-3}$	1.68×10^{-2}	$\textbf{2.88}\times \textbf{10}^{-3}$	1.19×10^{-3}	$1.23 imes 10^{-3}$	1.21×10^{-2}	$\textbf{4.47}\times 10^{-3}$
55–89	Mode	$3.15 imes 10^{-6}$	$3.55 imes 10^{-6}$	$1.92 imes 10^{-5}$	$\textbf{7.45}\times10^{-6}$	$1.16 imes$ 10^{-6}	$1.30 imes 10^{-6}$	$\textbf{7.36}\times 10^{-6}$	$\textbf{6.23}\times10^{-6}$
	Median	$4.17 imes 10^{-3}$	4.75×10^{-3}	$2.55 imes 10^{-2}$	$\textbf{9.95}\times 10^{-3}$	$8.58 imes 10^{-4}$	9.59×10^{-4}	5.50×10^{-3}	$\textbf{4.62}\times 10^{-3}$

(7a) and (7b). Consequently, the specific DALY rates were determined per 100,000 population as the algebraic sums of individual YLL_{sp} and YLD_{sp} (Equation (8)).

$\text{YLL}_{\text{sp}} = \text{F-YRC}_{\text{sp LL}} \times \text{YLL}_{\text{pp}},$	(7a)
$YLD_{sp} = F-YRC_{sp \ LD} \times YDL_{pp,}$	(7b)
$YLL_{sp} + YLD_{sp} = DALY_{sp.}$	(8)

3. Results

3.1. Acrylamide exposure and excess lifetime cancer risks assessment

The masses of foods consumed from the four main categories of heat-processed foods reported across the two Eastern African countries are presented in the logarithmic scale (Fig. 3). The average mass of foods consumed per person daily ranged from a minimum of 2 g/day ($log_{10} = 0.30$), identified as beverages, cocoa and coffee, to a maximum of 398 g/day ($log_{10} = 2.52$), which presented as cereal products. The rest of the categories of foods consumed ranged between these thresholds. In all, 75 separate food samples were collected from the published papers, constituting four categories of foods sampled from the research area (Fig. 3). In Ethiopia, beverages such as *keribo*; fermented, medium, light, deep, malted and unmalted drinks were included [50].

At the same time, French fries, unbranded potato crisps and branded potato crisps were also collected from Kenya. The food samples were carefully selected for the study period (2015–2019) in the two East African countries. Conversely, the five leading food categories representing frequently consumed foods in the two West African countries (Fig. 4) were collected from the FAO/WHO-GEMS database and a comprehensive survey of foods published in Nigeria [51]. Unlike the East African countries, the minimum mass of food consumed ranged from 18 g/day ($log_{10} = 1.34$), presenting as fats and oil-based foods, to a maximum of 631 g/day ($log_{10} = 2.92$), representing cereal-based products. In all, 89 separate food samples were collected from published papers, constituting five categories of foods collected (Fig. 4).

In Nigeria, published articles showing the quantities of popularly fried foods such as yam, sweet potato fries, French fries, deepfried ripe plantain, fried *akara*, baked *akara*, roasted plantain, and baked meat pie were included in the study [52,53]. Cereal-based foods such as *banku* with fish or meat, *kenkey* and fish, and porridge with bread were included in the articles collected in

Table 2b

Modal and median risk of Years of Life Lost and Years of Life lived in disability of kidney, ovarian and uterine cancers of age groups in the two West African countries.

		Risk of Yea	rs of Life Lost						
		Kidney Cancer		Ovarian Cancer	Uterine Cancer	Kidney Cancer		Ovarian Cancer	Uterine Cancer
		Ghana							
Age Group	Gender Statistics	Male	Female	Female	Female	Male	Female	Female	Female
5–19	Mode	4.68×10^{-7}	3.45×10^{-7}	3.87 imes 10 ⁻⁷	0.00	$4.43 imes$ 10^{-5}	$2.14 imes$ 10^{-6}	2.23×10^{-6}	0.00
	Median	8.89×10^{-5}	$6.58 imes 10^{-5}$	$\begin{array}{c} 7.37 \times \\ 10^{-5} \end{array}$	0.00	$1.11 imes 10^{-3}$	$\frac{1.22}{10^{-4}}\times$	1.80×10^{-4}	0.00
20–54	Mode	1.19×10^{-6}	$\frac{8.72}{10^{-7}}\times$	$7.25 imes$ 10^{-6}	1.36×10^{-6}	6.46×10^{-5}	$\frac{2.09}{10^{-6}}\times$	1.53×10^{-5}	$\textbf{4.44}\times 10^{-5}$
	Median	$2.29 imes$ 10^{-4}	$1.65 imes 10^{-4}$	$1.37 imes 10^{-3}$	2.64×10^{-4}	1.65×10^{-3}	$rac{1.72 imes}{10^{-4}} imes$	1.26×10^{-3}	1.39×10^{-3}
55–89	Mode	2.88 imes 10 ⁻⁶	$1.43 imes$ 10^{-6}	1.15×10^{-5}	$\textbf{9.08}\times10^{-6}$	$4.54 imes 10^{-5}$	9.47×10^{-7}	$\textbf{7.09}\times 10^{-6}$	1.15×10^{-4}
	Median	$\begin{array}{c} 5.54 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} 2.74 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} \textbf{2.20}\times\\ \textbf{10}^{-3} \end{array}$	1.72×10^{-3}	$\begin{array}{c} 1.15 \times \\ 10^{-3} \end{array}$	7.78×10^{-5}	5.80×10^{-4}	3.63×10^{-3}
5–19	Mode	Nigeria 2.18 \times 10 ⁻⁷	2.51×10^{-7}	$3.25 imes 10^{-7}$	0.00	1.01×10^{-6}	1.96 imes 10 ⁻⁶	$\textbf{2.84}\times \textbf{10}^{-6}$	0.00
	Median	$4.43 imes$ 10^{-5}	$5.13 imes$ 10^{-5}	6.45×10^{-5}	0.00	$5.68 imes 10^{-5}$	$8.57 imes 10^{-5}$	1.61×10^{-4}	0.00
20–54	Mode	$1.28 imes$ 10^{-6}	$8.64 imes$ 10^{-7}	$6.50 imes 10^{-6}$	9.03×10^{-7}	$3.37 imes 10^{-6}$	$3.95 imes 10^{-6}$	1.97×10^{-5}	$\textbf{5.49}\times \textbf{10}^{-6}$
	Median	$2.63 imes 10^{-4}$	$rac{1.76 imes}{10^{-4}} imes$	$rac{1.33 imes}{10^{-3}} imes$	1.84×10^{-4}	$rac{1.90}{10^{-4}} imes$	$rac{1.73 imes}{10^{-4}} imes$	1.11×10^{-3}	3.21×10^{-4}
55–89	Mode	3.01×10^{-6}	$rac{1.81}{10^{-6}} imes$	1.12 imes 10 ⁻⁵	$\textbf{4.58}\times10^{-6}$	2.44×10^{-6}	$1.29 imes 10^{-6}$	1.09×10^{-5}	$\textbf{6.94}\times10^{-6}$
	Median	$\begin{array}{c} \textbf{6.14}\times\\ \textbf{10}^{-4} \end{array}$	$\begin{array}{c} 3.72 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} 2.33 \times \\ 10^{-3} \end{array}$	9.32×10^{-4}	$\begin{array}{c} 1.38 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} 1.05 \times \\ 10^{-4} \end{array}$	$\textbf{6.22}\times 10^{-4}$	$\textbf{5.64}\times \textbf{10}^{-4}$

Ghana. Other tuber and root-based foods such as Fufu with fish soup, *gari* and beans, and *ampesi* with stew; all forms of rice products and fish, oats with bread, Hausa *Koko* with *koose*, or Buffloaf; and beverages such as tea and cocoa drinks were included [54]. Details of the dietary AA concentrations are shown in Figs. 5 and 6. The concentrations ranged from a minimum of 0.13 mg/kg dietary AA (fats and oils) to 9.73 mg/kg (roots, tubers, plantains and their products) for published works in East Africa.

On the other hand, for the West African study area, the concentrations of dietary AA ranged from a minimum of 0.025 mg/kg to a high of 14.39 mg/kg (cereals and their products). In contrast, the other food categories presented dietary AA concentrations within the range. Subsequently, the exposure estimated in the two study areas is shown in Table 1. We observed that the most frequently occurring dietary AA concentration in the Ethiopia-Kenya study area (7.81 mg/kg) was significantly higher than that of the Ghana-Nigeria study area (5.54×10^{-2} mg/kg). However, the exposure in the Ghana-Nigeria study area was higher (1.03×10^{-5} mg/kg (bw)-day) relative to the Ethiopia-Kenya study area (4.07×10^{-6} mg/kg (bw)-day).

3.2. Risk of years of life lost

Kidney cancer: Generally, the risk of AA-induced YLL of kidney cancers in the four countries (Tables 2a and 2b) presented similar insignificant ($<10^{-6}$) trends across the youthful age groups to significant for the aged years ($>10^{-6}$). In all these cases, the median statistic was higher than the most frequently occurring risk, which was low ($\sim 10^{-6}$). The highest median risk was recorded for the male-aged group in Ethiopia (5.93×10^{-3}), followed by the aged female group in Kenya (4.75×10^{-3}). However, Ghana's aged female group recorded the lowest median risk (2.74×10^{-4}).

Ovarian and Uterine cancers: For the risk of years of life lost to ovarian and uterine cancer diseases, a similar pattern emerged across the study areas where the modal risk was insignificant among the youth, increasing to significant risks among the aged groups. The highest median risk for ovarian and uterine cancer diseases was recorded in Kenya, whereas the lowest ovarian and uterine cancer risks were respectively recorded in Ghana (2.20×10^{-3}) and Nigeria (9.32×10^{-4}) .

3.3. Risk of years of living with a disability

Kidney Cancer: There seemed to be a higher modal risk of YLD of kidney cancer disease (Tables 2a and 2b) in both male and female populations $(10^{-6}-10^{-5})$ in the Ghana study area. Relative to the Ethiopia and Kenya study areas, there were low modal risks across the age groups ranging between $10^{-7}-10^{-6}$. Similarly, the median risk of years of life with disability of kidney cancer disease across the age

Table 3a

The dietary AA-induced five-year cancer prevalence rates YLD, YLL and DALY of cancers in kidney, ovaries, and uteri among age-related populations in Ghana.

Age group		5-YRC		YLL		YLD		DALY		% Cancer	
(years)		Mode	Median	Mode	Median	Mode	Median	Mode	Median	Mode	Median
	Gender	Kidney C	ancer								
5–19	Male	23.95	642	$\begin{array}{c} 1.65 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} 3.99 \times \\ 10^{-2} \end{array}$	$\begin{array}{c} 1.34 \times \\ 10^{-5} \end{array}$	$\begin{array}{c} \textbf{5.40}\times\\ \textbf{10}^{-4}\end{array}$	$rac{1.78 imes}{10^{-4}} imes$	$\begin{array}{c} 4.04 \times \\ 10^{-2} \end{array}$	$\frac{1.02}{10^{-1}}\times$	2.74
	Female	0.35	66	$\begin{array}{c} 1.10 \times \\ 10^{-4} \end{array}$	$2.70 imes 10^{-2}$	$2.76 imes 10^{-7}$	5.19×10^{-5}	$\frac{1.10}{10^{-4}}\times$	$rac{2.71 imes}{10^{-2}}$	$1.79 imes$ 10^{-3}	0.34
20–54	Male	23.37	949	$\begin{array}{c} 2.51 \times \\ 10^{-4} \end{array}$	6.11×10^{-2}	$2.78 imes 10^{-5}$	9.05×10^{-4}	2.79×10^{-4}	$6.20 imes 10^{-2}$	4.75×10^{-2}	1.93
	Female	0.49	93	$1.58 imes 10^{-4}$	3.93×10^{-2}	$\begin{array}{c} \textbf{4.42}\times\\ \textbf{10}^{-7}\end{array}$	8.43×10^{-5}	$\frac{1.58}{10^{-4}}\times$	$3.94 imes$ 10^{-2}	$2.11 imes 10^{-4}$	0.04
55–89	Male	16.45	664	$\begin{array}{c} 3.01 \times \\ 10^{-4} \end{array}$	7.33×10^{-2}	$2.63 imes$ 10^{-5}	$1.06 imes 10^{-3}$	$\begin{array}{c} 3.27 \times \\ 10^{-4} \end{array}$	$7.44 imes 10^{-2}$	$1.55 imes 10^{-2}$	0.63
	Female	0.22	42	$\begin{array}{c} 1.34 \times \\ 10^{-4} \end{array}$	$3.33 imes$ 10^{-2}	$3.09 imes$ 10^{-7}	5.89×10^{-5}	$1.34 imes$ 10^{-4}	$3.34 imes$ 10^{-2}	$1.55 imes 10^{-4}$	0.03
	Female	Ovarian	Cancer								
5–19		0.54	98	$\begin{array}{c} 1.17 \times \\ 10^{-4} \end{array}$	2.95×10^{-2}	$6.99 imes 10^{-7}$	$7.16 imes 10^{-5}$	$\frac{1.18}{10^{-4}}\times$	$3.00 imes$ 10^{-2}	$2.58 imes 10^{-3}$	0.49
20–54		3.71	683	$1.32 imes$ 10^{-3}	$3.34 imes 10^{-1}$	$4.19 imes 10^{-6}$	$7.64 imes 10^{-4}$	$\frac{1.32}{10^{-3}}\times$	$3.35 imes 10^{-1}$	$1.61 imes 10^{-3}$	0.30
55–89		1.73	314	$\begin{array}{c} 1.10 \times \\ 10^{-3} \end{array}$	$rac{2.78 imes}{10^{-1}} imes$	$3.19 imes 10^{-6}$	$\begin{array}{c} 5.81 \times \\ \mathbf{10^{-4}} \end{array}$	$\begin{array}{c} 1.10 \times \\ 10^{-3} \end{array}$	$\begin{array}{c} \textbf{2.79}\times\\\textbf{10}^{-1}\end{array}$	$\begin{array}{c} 1.21 \times \\ 10^{-3} \end{array}$	0.22
	Female	Uterine	Cancer								
5–19		0	0	0	0	0	0	0	0	0	0
20–54		10.81	742	$\begin{array}{c} 2.34 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} 5.92\times\\ 10^{-2} \end{array}$	$\begin{array}{c} 8.94 \times \\ 10^{-6} \end{array}$	$6.12 imes 10^{-4}$	$2.43 imes 10^{-4}$	$\begin{array}{c} 5.98 \times \\ 10^{-2} \end{array}$	4.68×10^{-2}	0.32
55–89		28.30	1932	$\begin{array}{c} \textbf{8.81}\times\\\textbf{10}^{-4}\end{array}$	$rac{2.22 imes}{10^{-1}} imes$	$2.76 imes 10^{-5}$	$\frac{1.89}{10^{-3}}\times$	9.09×10^{-4}	$\begin{array}{c} \textbf{2.24}\times\\ \textbf{10}^{-1} \end{array}$	$\frac{1.98}{10^{-2}}\times$	1.36
TOTAL		109.92	6225					$4.88 imes 10^{-3}$	1.20	$9.23 imes$ 10^{-1}	8.4

5-YRC: Five year-range cancers for specific cancers; YLL: Years of life lost for specific cancers; YLD: Years of life lived with a disability for specific cancers; DALYs: Disability adjusted life years for specific cancers.

groups ranged between 10^{-5} - 10^{-4} , with no clear patterns across these age groups relative to the Ghana study area where the risk was higher (10^{-4} - 10^{-3}).

Ovarian and Uterine cancers: The modal risk of YLD rates for ovarian and uterine cancer diseases across all four study areas ranged between 10^{-6} and 10^{-5} for all the age groups without showing any clear patterns. Generally, the median risk increased across the age groups for all the study areas, presenting the highest risk of ovarian (5.5×10^{-3}) and uterine (4.62×10^{-3}) cancer diseases in Kenya. The least risk was recorded in the Nigeria study area for ovarian (6.22×10^{-4}) and uterine (5.64×10^{-4}) cancer diseases.

3.4. Cancer burdens

Table 3a-d present the median and the mode of 5-YRCs, YLL rates, YLD rates, DALY rates, and the dietary AA-induced percentage cancer in Ghana, Nigeria, Ethiopia, and Kenya within the study period. These statistics were chosen to avoid biases that may arise from outliers. The metrics were obtained for the specific cancer endpoints for the three cancers studied per 100,000 population.

3.4.1. Kidney cancer

3.4.1.1. Disability adjusted life years (DALYs) rates. The dietary AA-induced cancer burdens obtained across the four countries were such that the male population generally presented higher DALY rates than the female population. The median DALY rates from Ethiopia (Table 3c) were the highest (0.29–2.47), whereas, in Ghana (Table 3a), the lowest DALY rates (0.0271–0.0744) were recorded. There was a consistent increase in DALY rates from the younger male population to the older male population. Similarly, the median DALY rates of the female population followed a similar trend. It is observed that the YLL rates component of the DALY rates for kidney cancer in the four countries were between 10^{-5} (Kenya) and 10^{-1} (Ethiopia) due to dietary AA exposure.

3.4.1.2. Acrylamide-induced (AA-induced) five-year range cancer prevalence rates (5-YRC) in kidney. The median dietary AA-induced cancer prevalence rates over the five years of study were highest in Ethiopia (Table 3c) among all the four-country study areas. It consistently increased from the young male population (396) to the adult male population (1,882). The dietary AA-induced kidney cancer cases in the female population also increased across the same trend, presenting the highest median 5-YRC prevalence (1,371). The median 5-YRC prevalence in Kenya (Table 3d) was high among the middle-aged for both the male (1,091) and female (1,053)

Table 3b

The dietary AA-induced five-year cancer prevalence rates YLD, YLL and DALY of cancers in kidney, ovaries, and uteri among age-related populations in Nigeria.

		5-YRC	5-YRC			YLD		DALY		% Cancer	
		Mode	Median	Mode	Median	Mode	Median	Mode	Median	Mode	Median
Age group (years)	Gender	Kidney (Cancer								
5–19	Male	1.14	210	$\begin{array}{c} 9.05 \times \\ 10^{-4} \end{array}$	0.129	$9.63 imes 10^{-7}$	$\frac{1.76}{10^{-4}}\times$	$9.06\times \\ 10^{-4}$	$\begin{array}{c} 1.29 \times \\ 10^{-1} \end{array}$	$rac{1.56}{10^{-3}} imes$	0.29
	Female	2.83	300	$5.40 imes 10^{-4}$	0.139	$2.21 imes 10^{-6}$	$2.34 imes 10^{-4}$	$\begin{array}{c} 5.42 \times \\ 10^{-4} \end{array}$	$1.39 imes 10^{-1}$	$3.75 imes 10^{-3}$	0.40
20–54	Male	3.80	703	2.97×10^{-3}	0.422	$3.67 imes 10^{-6}$	$6.76 imes 10^{-4}$	$2.97 imes$ 10^{-3}	$4.23 imes 10^{-1}$	$3.08 imes$ 10^{-3}	0.57
	Female	5.71	605	1.06×10^{-3}	0.273	4.93 imes 10 ⁻⁶	5.22 imes 10 ⁻⁴	$1.06 imes$ 10^{-3}	$2.74 imes 10^{-1}$	$5.84 imes 10^{-4}$	0.06
55–89	Male	2.76	508	$1.97 imes$ 10^{-3}	0.497	$4.09 imes$ 10^{-6}	$7.52 imes 10^{-4}$	$rac{1.97 imes}{10^{-3}} imes$	$4.98 imes 10^{-1}$	4.14 imes 10 ⁻⁴	0.08
	Female	3.45	366	$1.14 imes 10^{-3}$	0.293	$4.79 imes 10^{-6}$	$5.13 imes 10^{-4}$	$1.14 imes$ 10^{-3}	$2.94 imes$ 10^{-1}	$5.39 imes$ 10^{-4}	0.06
	Female	Ovarian	Cancer								
5–19		5.27	563	$6.79 imes$ 10^{-4}	0.17	$\begin{array}{l} 3.88 \times \\ 10^{-6} \end{array}$	4.10×10^{-4}	$6.83 imes 10^{-4}$	$\frac{1.70}{10^{-1}}\times$	$7.00 imes$ 10^{-3}	0.75
20–54		36.60	3901	$7.88 imes 10^{-3}$	2.02	$4.34 imes 10^{-5}$	4.59×10^{-3}	$7.92\times \\10^{-3}$	2.02	3.74 imes 10 ⁻³	0.40
55-89		20.46	2187	7.52×10^{-3}	1.94	3.65×10^{-5}	3.90×10^{-3}	$7.56 imes$ 10^{-3}	1.94	3.20×10^{-3}	0.34
	Female	Uterine	Cancer								
5–19		0	0	0	0	0	0	0	0	0	0
20–54		10.30	1122	$1.05 imes 10^{-3}$	0.27	$\begin{array}{c} 8.23 \times \\ 10^{-6} \end{array}$	$8.73 imes 10^{-4}$	$1.06 imes 10^{-3}$	$\begin{array}{c} \textbf{2.71}\times\\\textbf{10}^{-1}\end{array}$	$1.05 imes$ 10^{-3}	0.12
55-89		18.53	1970	2.79×10^{-3}	0.72	$1.78 imes$ 10^{-5}	1.90×10^{-3}	$rac{2.81}{10^{-3}} imes$	7.22	2.90×10^{-3}	0.31
TOTAL		110.85	12,435					2.86×10^{-2}	13.40	2.78 imes 10 ⁻²	3.38

5-YRC: Five year-range cancers for specific cancers; YLL: Years of life lost for specific cancers; YLD: Years of life lived with a disability for specific cancers; DALYs: Disability adjusted life years for specific cancers.

populations. While the median 5-YRC prevalence in Ghana (Table 3a) was the lowest, it also indicated that the female population consistently presented lower values (42.06–92.94) relative to the male population (642.28–948.63). Characteristically, the female population presented the least 5-YRC prevalence (42) among the four countries. Similar to Kenya, the middle-aged population in Nigeria (Table 3b) presented a high 5-YRC prevalence for the male (701) and female (605) populations. Among the three age categories, the adult female population in Ghana (Table 3a) presented the lowest percentage of dietary AA-induced kidney cancer cases (0.03 %). The highest cases in the young male population (2.74 %) were also recorded in Ghana. The percentage of kidney cancer across the three age groups in Nigeria was consistently low (0.06–0.57 %) compared to the three other countries in the study area.

3.4.2. Ovarian cancer

3.4.2.1. Disability adjusted life years (DALYs) rates. The median dietary AA-induced DALY rates recorded across the four-country study areas for ovarian cancer closely followed that of kidney cancer. The highest cases were presented in Ethiopia (0.755–8.42), followed by Kenya (0.401–6.39). However, the most minor cases were recorded in Ghana (0.0030–0.0335). Although the median DALY rates recorded in Ethiopia and Kenya were high, the most frequently occurring (mode) DALY rates presented were low, ranging from 1.18×10^{-4} (Ghana) to 7.92×10^{-3} (Ethiopia). The ovarian cancer cases increased consistently from the younger to the older age group. However, the middle-aged group in Ghana, rather than the older group, presented the highest median DALY rates (0.335). Similar to the dietary AA-induced kidney cancer trends, YLL rates rather than the YLD rates contributed significantly to the ovarian cancer disease DALY rates across the study area.

3.4.2.2. Acrylamide-induced (AA-induced) five-year range cancer (5-YRC) prevalence rates in ovaries. Across the study area, the middle-aged group presented the highest median 5-YRC prevalence rates apart from Ethiopia, which recorded the highest (1,863) among the younger age group. It ranged from 683 (Ghana) to 10,343 (Kenya). However, the cases were low for the most frequently occurring (mode) dietary AA-induced 5-YRC prevalence across the study area. Ethiopia recorded the lowest (5.49×10^{-3}) for the older age group, whereas Nigeria presented the highest (36.6). The percentage of dietary AA attributable to ovarian cancer across the study area consistently decreased from the younger to the older age group. However, in the Ethiopia and Kenya study areas, the median dietary AA-induced ovarian cancer cases were higher (2.58–7.50 %) than that of the Ghana-Nigeria areas (0.22–0.75 %). The most frequently occurring (mode) 5-YRC prevalence of dietary AA-induced ovarian cancer cases remained under ~ 10^{-3} .

Table 3c

The dietary AA-induced five-year cancer prevalence rates YLD, YLL and DALY of cancers in kidneys, ovaries, and uteri among age-related populations in Ethiopia.

Age group		5-YRC	5-YRC			YLD		DALY	DALY %		% Cancer		
(years)		Mode	Median	Mode	Median	Mode	Median	Mode	Median	Mode	Median		
	Gender	Kidney Car	icer										
5–19	Male	0.35	396	$\begin{array}{c} 2.32 \times \\ 10^{-4} \end{array}$	0.29	$3.00 imes 10^{-7}$	$\begin{array}{c} 3.36 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} 2.32 \times \\ 10^{-4} \end{array}$	$\begin{array}{c} \textbf{2.90}\times\\ \textbf{10}^{-1}\end{array}$	$7.95\times \\ 10^{-4}$	0.89		
	Female	0.45	531	$2.35 imes 10^{-4}$	0.29	$3.58 imes$ 10^{-7}	$4.21 imes 10^{-4}$	$\frac{2.35}{10^{-4}}\times$	$\begin{array}{c} \textbf{2.90}\times\\ \textbf{10}^{-1} \end{array}$	$8.36 imes 10^{-4}$	0.99		
20–54	Male	1.27	1440	9.50×10^{-4}	1.19	1.33×10^{-6}	1.51×10^{-3}	$9.51 imes$ 10^{-4}	$1.19 imes 10^{-1}$	1.16×10^{-3}	1.32		
	Female	1.06	1262	$6.45 imes$ 10^{-4}	0.79	3.53×10^{-5}	4.42×10^{-2}	$6.80 imes$ 10^{-4}	$\frac{8.34}{10^{-1}}\times$	$2.28 imes$ 10^{-4}	0.27		
55–89	Male	1.66	1882	$198 imes 10^{-3}$	2.47	$\begin{array}{c} \textbf{2.87}\times\\ \textbf{10}^{-6} \end{array}$	3.23 imes 10 ⁻³	$1.98 imes 10^{-1}$	2.47	$\frac{8.42}{10^{-4}}\times$	0.95		
	Female	1.16	1371	$1.25 imes$ 10^{-3}	1.52	1.93×10^{-6}	2.28×10^{-3}	$1.25 imes 10^{-3}$	1.52	4.98 imes 10 ⁻⁴	0.59		
	Female	Ovarian Ca	ancer										
5–19		1.60	1863	$5.93 imes$ 10^{-4}	0.72	$\begin{array}{c} 2.89 \times \\ 10^{-5} \end{array}$	3.52×10^{-2}	$6.22 imes$ 10^{-4}	$7.55 imes 10^{-1}$	$2.98 imes$ 10^{-3}	3.48		
20–54		$6.53 imes 10^{-3}$	8	$6.53 imes 10^{-3}$	7.97	$3.63 imes 10^{-4}$	$4.45 imes 10^{-1}$	$6.89 imes$ 10^{-3}	8.42	2.21 imes 10 ⁻³	2.58		
55-89		$\begin{array}{c} \textbf{5.49}\times\\\textbf{10}^{-3}\end{array}$	7	$\begin{array}{c} \textbf{5.49}\times\\ \textbf{10}^{-3} \end{array}$	6.68	$\begin{array}{c} 3.00 \times \\ 10^{-4} \end{array}$	$3.73 imes 10^{-1}$	$\begin{array}{c} 5.79\times\\\mathbf{10^{-3}}\end{array}$	7.05	$2.20 imes 10^{-3}$	2.58		
	Female	Uterine Ca	ncer										
5-19		0	0	0	0	0	0	0	0	0	0		
20–54		3.29	3960	$\begin{array}{c} 1.14 \times \\ 10^{-3} \end{array}$	1.34	$\begin{array}{c} 6.42 \times \\ 10^{-5} \end{array}$	$\begin{array}{c} \textbf{7.82}\times\\ \textbf{10}^{-2} \end{array}$	$\frac{1.20}{10^{-3}}\times$	1.42	$7.04 imes 10^{-4}$	0.86		
55-89		4.43	5196	$\begin{array}{c} 2.36 \times \\ 10^{-3} \end{array}$	2.77	$\begin{array}{c} 1.22 \times \\ 10^{-4} \end{array}$	$rac{1.51}{10^{-1}} imes$	$\begin{array}{c} \textbf{2.48}\times\\\textbf{10}^{-3}\end{array}$	2.92	$\begin{array}{c} 1.90 \times \\ 10^{-3} \end{array}$	2.23		
TOTAL		15.27	17,916					$egin{array}{c} 2.18 imes 10^{-1} \end{array}$	26.10	$4.18 imes 10^{-1}$	16.74		

5-YRC: Five year-range cancers for specific cancers; YLL: Years of life lost for specific cancers; YLD: Years of life lived with a disability for specific cancers; DALYs: Disability adjusted life years for specific cancers.

3.4.3. Uterine cancer

3.4.3.1. Disability adjusted life years (DALYs) rates. Across the study area, the young age group presented no uterine cancer DALY rates. However, the median dietary AA-induced uterine cancer disease DALY rates were the lowest in Ghana (5.98×10^{-2}) but highest in Nigeria (7.22). The DALY rates, which increased across the age groups, were more influenced by their YLL rates component than the YLD rates. Characteristically, the most frequently occurring (mode) dietary AA-induced uterine cancer DALY rates were very low (10^{-4} - 10^{-3}).

3.4.3.2. Acrylamide-induced (AA-induced) five-year range cancer (5-YRC) prevalence rates in the uterus. The prevalence rates were highest in Ethiopia (3960–5196) and Kenya (3846–3961) regions but comparatively lower in Ghana (742–1932) and Nigeria regions (1122–1970). However, the most frequently occurring (mode) 5-YRC were significantly low $(10^{-4}-10^{-3})$ across the study area. The older age groups across the region characteristically presented higher 5-YRCs. The four study areas also showed no records of the percentage of cancer for the younger age groups. However, the middle age group in Nigeria presented the lowest median dietary AA-induced percentage uterine cancer prevalence (0.12%), while the highest was recorded in Kenya (1.70%). For the middle and older age groups, the median dietary AA-induced uterine percentage cancer cases increased consistently across all four study areas. The highest (4.68×10⁻²%) most frequently occurring (mode) dietary AA-induced percentage uterine cancer disease was recorded in Ghana, while the middle age group in Ethiopia recorded the least (7.04×10⁻⁴%).

4. Discussion

In this study, we estimated DALY rates (algebraic sum of YLLs and YLDs) for dietary AA-induced kidney, ovary, and uterine cancer diseases in four study areas: Ghana, Nigeria, Ethiopia, and Kenya. We estimated the most frequently occurring (mode) DALY rates as ranging from a low (0.00488) in Ghana to the highest (0.218) in Ethiopia per 100,000 population. We observed that the modal DALY rates were lower across the study area than the median DALY rates that presented higher values. Generally, the two West African countries presented lower DALY rates than East African countries. For example, we estimated a median of 1.2 healthy life years lost per 100,000 population of the three cancer diseases in Ghana.

Meanwhile, 26.10 healthy life years lost per 100,000 population were recorded in Ethiopia. Relative to a similar study in Denmark [35], 1.8 healthy life years per 100,000 population were lost based on dietary AA-induced cancers. While the low DALY rates recorded

Table 3d

The dietary AA-induced five-year cancer prevalence rates YLD, YLL and DALY of cancers in kidneys, ovaries, and uteri among age-related populations in Kenya.

Age group		5-YRC		YLL		YLD		DALY		% Cancer	
(years)		Mode	Median	Mode	Median	Mode	Median	Mode	Median	Mode	Median
	Gender	Kidney	Cancer								
5–19	Male	0.23	196	$3.65 imes 10^{-5}$	0.06	2.04×10^{-7}	1.76×10^{-4}	$\begin{array}{c} 3.67 \times \\ 10^{-5} \end{array}$	$6.02 imes 10^{-2}$	$\begin{array}{c} 1.46 \times \\ 10^{-3} \end{array}$	1.27
	Female	0.31	255	$9.05 imes$ 10 $^{-5}$	0.15	$\begin{array}{c} 1.92 \times \\ 10^{-5} \end{array}$	$\frac{1.89}{10^{-2}}\times$	$1.10 imes$ 10 $^{-4}$	$\frac{1.69}{10^{-1}}\times$	$\begin{array}{c} \textbf{2.20}\times\\ \textbf{10}^{-3} \end{array}$	1.82
20–54	Male	1.25	1091	$\begin{array}{c} 2.01 \times \\ 10^{-4} \end{array}$	0.31	1.29×10^{-6}	$1.11 imes$ 10^{-3}	$2.02 imes$ 10^{-4}	$3.11 imes 10^{-1}$	$\begin{array}{c} 1.92 \times \\ 10^{-3} \end{array}$	1.68
	Female	1.24	1053	$3.45 imes$ 10 $^{-4}$	0.57	$\begin{array}{l} \textbf{7.99}\times\\ \textbf{10}^{-5}\end{array}$	$7.27 imes$ 10^{-2}	4.25 imes 10 ⁻⁴	$6.43 imes 10^{-1}$	$5.49 imes 10^{-4}$	0.47
55–89	Male	0.90	783	$2.44 imes$ 10^{-4}	0.38	1.47×10^{-6}	$\frac{1.27}{10^{-3}}\times$	$2.45 imes$ 10^{-4}	$\begin{array}{c} \textbf{3.81}\times\\\textbf{10}^{-1}\end{array}$	$6.92 imes$ 10^{-4}	0.60
	Female	0.98	826	$\begin{array}{c} 5.33 \times \\ 10^{-4} \end{array}$	0.88	$\begin{array}{c} 1.42 \times \\ 10^{-4} \end{array}$	$rac{1.28}{10^{-1}} imes$	$6.75 imes 10^{-4}$	1.01	$7.77 imes 10^{-4}$	0.65
	Female	Ovaria	1 Cancer								
5–19		0	0	$\begin{array}{c} \textbf{2.41}\times\\ \textbf{10}^{-4} \end{array}$	0.40	$\frac{8.89}{10^{-7}}\times$	$7.41 imes 10^{-4}$	$2.42 imes$ 10^{-4}	$\begin{array}{c} \textbf{4.01}\times\\\textbf{10}^{-1}\end{array}$	8.48×10^{-3}	7.10
20–54		12.30	10,343	$\begin{array}{l} 3.83 \times \\ 10^{-3} \end{array}$	6.38	$1.44 imes 10^{-5}$	$\frac{1.21}{10^{-2}}\times$	$3.84 imes$ 10^{-3}	6.39	$5.43 imes 10^{-3}$	4.57
55–89		5.62	4717	$\begin{array}{c} 3.20 \times \\ 10^{-3} \end{array}$	5.31	$\begin{array}{c} 1.04 \times \\ 10^{-5} \end{array}$	$\frac{8.63}{10^{-3}}\times$	$\begin{array}{c} 3.21 \times \\ 10^{-3} \end{array}$	5.32	$4.45 imes 10^{-3}$	3.73
	Female	Uterine	Cancer								
5–19		0	0	0	0	0	0	0	0	0	0
20–54		4.59	3846	$6.45 imes 10^{-4}$	1.07	3.81×10^{-6}	$3.14 imes$ 10^{-3}	$6.49 imes 10^{-4}$	1.07	$\begin{array}{c} \textbf{2.03}\times\\ \textbf{10}^{-3} \end{array}$	1.70
55–89		4.73	3961	$\begin{array}{c} 1.13 \times \\ 10^{-3} \end{array}$	1.88	$\begin{array}{c} \textbf{5.08}\times\\\textbf{10}^{-6}\end{array}$	$\begin{array}{c} \textbf{4.24}\times\\\textbf{10}^{-3}\end{array}$	$1.14 imes$ 10^{-3}	1.88	$3.74 imes$ 10^{-3}	3.13
TOTAL		32.15	27,071					$rac{1.08 imes}{10^{-2}} imes$	17.60	$egin{array}{c} 2.95 imes 10^{-2} \end{array}$	26.72

5-YRC: Five year-range cancers for specific cancers; YLL: Years of life lost for specific cancers; YLD: Years of life lived with a disability for specific cancers; DALYs: Disability adjusted life years for specific cancers.

However, the most frequently occurring (mode) 5-YRC were significantly low $(10^{-4} \cdot 10^{-3})$ across the study area. The older age groups across the region characteristically presented higher 5-YRCs. The four study areas also showed no records of the percentage of cancer for the younger age groups. However, the middle age group in Nigeria presented the lowest median dietary AA-induced percentage uterine cancer prevalence (0.12 %), while the highest was recorded in Kenya (1.70 %). For the middle and older age groups, the median dietary AA-induced uterine percentage cancer cases increased consistently across all four study areas. The highest (4.68×10^{-2} %) most frequently occurring (mode) dietary AA-induced percentage uterine cancer disease was recorded in Ghana, while the middle age group in Ethiopia recorded the least (7.04×10^{-4} %).

in Denmark may be attributed to more robust healthcare infrastructure, the low to moderate DALY rate outcomes observed in this current study may be attributed to other factors. Studies have shown that ethnicity and the interactions between socio-environmental, behavioral and biological factors contribute to cancer development [55–57]. Thus, variations in these factors may have contributed to the low metric rates observed in some zones in the current study area. However, a study in China [24] presented about 26,688 healthy life years lost attributed to dietary AA-induced cancers. Relatively, such DALY rates are enormous compared to the low median values (1.2-26.10) obtained in this current study. The median dietary AA exposures varied from a low of 3.19×10^{-4} in the Ghana-Nigeria study area to a high of 1.64×10^{-3} mg/kg (bw)-day in the Ethiopia-Kenya study areas. Consequentially, we estimated the median 5-YRC prevalence of dietary AA-induced cancer diseases between 2015 and 2019 from 6225 (Ghana) to 27,071 (Kenya) per 100,000 populations. Thus, the dietary AA-induced risks were higher for the older than the younger age group. The general trend was that age advancement could be one of the most critical risk factors for dietary AA-induced cancer diseases, as may be for many other cancer types [58]. These median morbidity and mortality rates of dietary AA-induced cancer diseases in the four individual countries led to a total loss of healthy lives of about 63.7 DALY rates per 100,000 population. It is evident from the results that these DALY rates could be avoided if dietary AA-related food safety policies were in place. In the case of uterine cancer, no data was reported on the mortality and prevalence for the younger age group, according to the IHME database [33]. It does not mean this group is immune from dietary AA-induced cancer burdens; instead, it suggests the general trend concerning the age-related prevalence of cancer diseases [58]. Undoubtedly, the 5-YRC prevalence, which significantly impacts the DALY rates, might have arisen from different cultural food processing methods specific to the ethnicities in the study areas [56]. They could also arise from different dietary habits, leading to differences in dietary AA concentrations, resulting in exposure differences [26]. Many studies have shown that these exposure elements directly contribute to dietary AA exposure variations in the population [17]. However, the elements of exposure collected for this study from literature are undoubtedly laden with uncertainties relating to the food consumption practices within the population, body weight and dietary AA concentrations. The dietary AA concentrations have been shown to vary widely depending on the service provider, the time for cooking, and the method and temperature used during cooking [59]. The final regional DALY rates show

Table 4s

Details and illustrations of the nexus connecting AA exposures and the health metrics to yield YLL and YLD, the components of DALYs.



different patterns of how dietary AA exposure affects consumers' health. Thus, the differences in the dietary AA-linked burden of cancers observed could be attributed to socio-environmental, behavioral and biological factors among closely related ethnic groups [56] and their eating patterns.

4.1. Study limitations

There are some limitations associated with this study, and this is why the most frequently occurring (mode) and median cases were

used in this report. For this reason, probabilistic models with their iterations were employed to provide a more realistic estimate of the dietary AA-induced health outcomes. Thus, care must be exercised in the interpretations of the outcome of this study. Another challenge was the relatively low number (six) of articles from the restrictive inclusion years of study (2015–2019), from which dietary AA concentrations were collected. This limitation might impact our results. However, the probabilistic approach adopted may leverage the results of the two uncertainty outcomes (modal and median values) presented in this report. Age-related standardized national and international data on food consumption in the African region were unavailable. Thus, the mass of food consumed was the average daily consumption by the whole population. Also, the non-availability of age and gender-related body weight distribution presented another challenge. Therefore, we used fixed body weight, a default value proposed in such situations by EFSA [47]. However, reasonable conclusions have been adduced once decisions or outcomes are interpreted using the associated uncertainties in a probabilistic approach [60].

5. Conclusion

Among other uncertainties, the study estimated the total median cancer burdens in the four study areas to be 63.7 DALY rates, ranging between 1.2 and 26.1. For individual countries, they presented as Ethiopia (26.1) > Kenya (17.6) > Nigeria (13.4) > Ghana (1.2) per 100,000 population. These health metrics indicators represent the median dietary AA doses in foods consumed, expressed as the associated exposures and risks relating to human cancer burdens in the three organs (kidney, ovary, and uterus). The model developed in this study can produce results based on the quality of the elements of exposure supplied as input variables. The output of this study establishes the processes that connect the exposures and risks of dietary AA-contaminated foods in a model designed to estimate the related cancer burdens transmitted through foods.

Data availability statement

Data associated with this study can be assessed from the Mendeley data repository via: https://data.mendeley.com/datasets/tkkbgm4b83/1.

Consent to publish

The authors agree to publish the manuscript.

Ethics approval and consent to participate

Not applicable.

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Availability of data and materials

All datasets supporting the conclusion made in the study will be deposited in Mendeley repository.

CRediT authorship contribution statement

Naa K.-A Quartey: Writing - original draft, Data curation. Juanita A. Haagsma: Writing - review & editing, Resources. Lea S. Jakobsen: Writing - review & editing, Resources. Isaac W. Ofosu: Writing - review & editing, Writing - original draft, Supervision, Resources, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2023.e23075.

Abbreviations

AA	Acrylamide
E _{EST}	Estimated exposure
AA _{SRL}	Distribution of the concentrations of acrylamide searched through literature
$\mathbf{B}_{\mathbf{W}}$	Body weight of population
M _F	Mass of food consumed
CSF	Cancer slope factor
CSF _{sp}	Cancer slope factor scaled for a specific cancer endpoint
Mort _{sp}	Mortality of a specific cancer endpoint
Mort _{tot}	Total mortality of cancers
CSFLL	Cancer slope factor scaled for a specific cancer endpoint for risk of Years of life lost
CSFLD	Cancer slope factor scaled for a specific cancer endpoint for risk of Years of life lived with a disability
Prev _{sp}	Prevalence for a specific cancer end point
Prevtot	Total prevalence of cancers
R _{LL}	Risk of years of life lost
R _{LD}	Risk of years of life with a disability
5-YRC	Five year-range cancers
5-YRC _{sp}	Five year-range cancers for specific end point
5-YRC _{sp}	LL Five year-range cancers for specific end point for life lost
5-YRC _{sp}	LD Five year-range cancers for specific end point for life lived with a disability
Npop	National population
LEpop	Life expectancy of the population
DW	disability weight
YLL	Years of life lost
YLL _{sp}	Years of life lost for a specific cancer endpoint
YLLpp	Years of life lost per person
YLD	Years of life lived with a disability
YLD _{sp}	Years of life lived with a disability for a specific cancer endpoint
YLD _{pp}	Years of life lived with a disability per person
DALYs	Disability adjusted life years
FAO	Food and Agriculture Organization
EFSA	European Food Safety Authority
IARC	International Agency for Research on Cancer
IHME	Institute for Health Metrics and Evaluation
WHO	World Health Organization
US NTP	United States National Toxicology Programme
ELTCR	Excess Lifetime Cancer Risk
MOE	Margin of Exposure
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
GIFT	Global Individual Food consumption data Tool
GEMS	Global Environment Monitoring System
NOS	Newcastle-Ottawa Scale
GBD	Global Burden of Disease

References

- [1] L. Lipworth, Acrylamide: a human cancer risk? Eur. J. Cancer Prev. 22 (2013) 193–194, https://doi.org/10.1097/CEJ.0b013e328358194d.
- [2] M.N. Lund, C.A. Ray, Control of Maillard reactions in foods: strategies and chemical mechanisms, J. Agric. Food Chem. 65 (2017) 4537-4552.
- [3] M. Zeng, L. Manyes, L. Li, Y. Zhuang, X. Zou, M. Chen, B. Cui, Y. Jiao, Y. Cheng, Advanced Glycation End Products: A Comprehensive Review of Their Detection and Occurrence in Food, 2023, https://doi.org/10.3390/foods12112103.
- [4] F. Pedreschi, M.S. Mariotti, K. Granby, Current issues in dietary acrylamide: formation, mitigation and risk assessment, J. Sci. Food Agric. 94 (2014) 9-20.
- [5] R.H. Stadler, V. Gökmen, in: V. Gökmen, B.A.B.T.-A. in F., Second E. Mogol (Eds.), Chapter 1 Acrylamide Formation Mechanisms, Academic Press, 2024, pp. 1–17, https://doi.org/10.1016/B978-0-323-99119-3.00017-5.
- [6] A.A. Maan, M.A. Anjum, M.K.I. Khan, A. Nazir, F. Saeed, M. Afzaal, R.M. Aadil, Acrylamide formation and different mitigation strategies during food processing-a review, Food Rev. Int. 38 (2022) 70–87.
- [7] A. Becalski, B. Brady, S. Feng, B.R. Gauthier, T. Zhao, Formation of acrylamide at temperatures lower than 100 C: the case of prunes and a model study, Food Addit. Contam. 28 (2011) 726–730.
- [8] G. Strocchi, P. Rubiolo, C. Cordero, C. Bicchi, E. Liberto, Acrylamide in coffee: what is known and what still needs to be explored. A review, Food Chem. 393 (2022), 133406, https://doi.org/10.1016/j.foodchem.2022.133406.
- [9] B. Basaran, F. Aydin, Estimating the acrylamide exposure of adult individuals from coffee: Turkey, Food Addit. Contam. 37 (2020) 2051–2060.
- [10] B. Başaran, F. Aydın, Determination of acrylamide levels in infant formulas and baby biscuits sold in Turkey, Lett. Appl. NanoBioScience. 11 (2022) 3155–3165.

- [11] M.A. Schouten, C. Fryganas, S. Tappi, S. Romani, V. Fogliano, Influence of lupin and chickpea flours on acrylamide formation and quality characteristics of biscuits, Food Chem. 402 (2023), 134221, https://doi.org/10.1016/j.foodchem.2022.134221.
- [12] Y. Huang, M. Li, J. Lu, H. Hu, Y. Wang, C. Li, X. Huang, Y. Chen, M. Shen, S. Nie, M. Xie, Inhibitory effect of hydrocolloids and ultrasound treatments on acrylamide and 5-hydroxymethylfurfural formation in French fries, Food Hydrocolloids 133 (2022), 107839, https://doi.org/10.1016/j.foodhyd.2022.107839.
- B. Başaran, H. Turk, The influence of consecutive use of different oil types and frying oil in French fries on the acrylamide level, J. Food Compos. Anal. 104 (2021), 104177, https://doi.org/10.1016/j.ifca.2021.104177.
- [14] B. Basaran, P. Anlar, Z.F. Yılmaz Oral, Z. Polat, G. Kaban, Risk assessment of acrylamide and 5-hydroxymethyl-2-furfural (5-HMF) exposure from bread consumption: Turkey, J. Food Compos. Anal. 107 (2022), 104409, https://doi.org/10.1016/j.jfca.2022.104409.
- [15] M. Mesias, C. Delgado-Andrade, F.J. Morales, An updated view of acrylamide in cereal products, Curr. Opin. Food Sci. 46 (2022), 100847, https://doi.org/ 10.1016/j.cofs.2022.100847.
- [16] H. Pekmezci, B. Basaran, Dietary heat-treatment contaminants exposure and cancer: a case study from Turkey, Foods 12 (2023), https://doi.org/10.3390/ foods12122320.
- [17] T. Filippini, T.I. Halldorsson, C. Capitão, R. Martins, K. Giannakou, J. Hogervorst, M. Vinceti, A. Åkesson, K. Leander, A. Katsonouri, O. Santos, A. Virgolino, F. Laguzzi, Dietary acrylamide exposure and risk of site-specific cancer: a systematic review and dose-response meta-analysis of epidemiological studies, Front. Nutr. 9 (2022), https://doi.org/10.3389/fnut.2022.875607.
- [18] M. Pedersen, E. Vryonidis, A. Joensen, M. Törnqvist, Hemoglobin adducts of acrylamide in human blood what has been done and what is next? Food Chem. Toxicol. 161 (2022), 112799 https://doi.org/10.1016/j.fct.2021.112799.
- [19] S. Mundi, R.E. Aluko, Effects of NaCl and pH on the structural conformations of kidney bean vicilin, Food Chem. 139 (2013) 624–630, https://doi.org/10.1016/ j.foodchem.2012.12.051.
- [20] M. Zhao, B. Zhang, L. Deng, The mechanism of acrylamide-induced neurotoxicity: current status and future perspectives, Front. Nutr. 9 (2022) 488.
- [21] W. Rungratanawanich, Y. Qu, X. Wang, M.M. Essa, B.-J. Song, Advanced glycation end products (AGEs) and other adducts in aging-related diseases and alcoholmediated tissue injury, Exp. Mol. Med. 53 (2021) 168–188, https://doi.org/10.1038/s12276-021-00561-7.
- [22] M. Anvari, A.R. Talebi, E. Mangoli, A. Shahedi, M.R. Ghasemi, M. Pourentezari, Effects of acrylamide in the presence of vitamin E on sperm parameters, chromatin quality, and testosterone levels in mice, Clin. Exp. Reprod. Med. 47 (2020) 101–107, https://doi.org/10.5653/cerm.2019.03230.
- [23] A.M. Khaneghah, Y. Fakhri, A. Nematollahi, F. Seilani, Y. Vasseghian, The concentration of acrylamide in different food products: a global systematic review, meta-analysis, and meta-regression, Food Rev. Int. 38 (2022) 1286–1304.
- [24] L. Yiling, J. Liu, Y. Wang, S. Wei, Cancer risk and disease burden of dietary acrylamide exposure in China, 2016, Ecotoxicol. Environ. Saf. 238 (2022), 113551.
- [25] C. Pelucchi, C. Bosetti, C. Galeone, C. La Vecchia, Dietary acrylamide and cancer risk: an updated meta-analysis, Int. J. Cancer 136 (2015) 2912–2922, https:// doi.org/10.1002/ijc.29339.
- [26] M.K. Virk-Baker, T.R. Nagy, S. Barnes, J. Groopman, Dietary acrylamide and human cancer: a systematic review of literature, Nutr. Cancer 66 (2014) 774–790, https://doi.org/10.1080/01635581.2014.916323.
- [27] C. Fitzmaurice, C. Allen, R.M. Barber, L. Barregard, Z.A. Bhutta, H. Brenner, D.J. Dicker, O. Chimed-Orchir, R. Dandona, L. Dandona, T. Fleming, M. H. Forouzanfar, J. Hancock, R.J. Hay, R. Hunter-Merrill, C. Huynh, H.D. Hosgood, C.O. Johnson, J.B. Jonas, J. Khubchandani, G.A. Kumar, M. Kutz, Q. Lan, H. J. Larson, X. Liang, S.S. Lim, A.D. Lopez, M.F. MacIntyre, L. Marczak, N. Marquez, A.H. Mokdad, C. Pinho, F. Pourmalek, J.A. Salomon, J.R. Sanabria, L. Sandar, B. Sartorius, S.M. Schwartz, K.A. Shackelford, K. Shibuya, J. Stanaway, C. Steiner, J. Sun, K. Takahashi, S.E. Vollset, T. Vos, J.A. Wagner, H. Wang, R. Westerman, H. Zeeb, L. Zoeckler, F. Abd-Allah, M.B. Ahmed, S. Alabed, N.K. Alam, S.F. Aldhahri, G. Alem, M.A. Alemayohu, R. Ali, R. Al-Raddadi, A. Amare, Y. Amoako, A. Artaman, H. Asayesh, N. Atnafu, A. Awasthi, H.B. Saleem, A. Barac, N. Bedi, I. Bensenor, A. Berhane, E. Bernabé, B. Betsu, A. Binagwaho, D. Boneya, I. Campos-Nonato, C. Castañeda-Orjuela, F. Catalá-López, P. Chiang, C. Chibueze, A. Chitheer, J.-Y. Choi, B. Cowie, S. Damtew, J. das Neves, S. Dey, S. Dharmaratne, P. Dhillon, E. Ding, T. Driscoll, D. Ekwueme, A.Y. Endries, M. Farvid, F. Farzadfar, J. Fernandes, F. Fischer, T.T.G./ Hiwot, A. Gebru, S. Gopalani, A. Hailu, M. Horino, N. Horita, A. Husseini, I. Huybrechts, M. Inoue, F. Islami, M. Jakovljevic, S. James, M. Javanbakht, S.H. Jee, A. Kasaeian, M. S. Kedir, Y.S. Khader, Y.-H. Khang, D. Kim, J. Leigh, S. Linn, R. Lunevicius, H.M.A. El Razek, R. Malekzadeh, D.C. Malta, W. Marcenes, D. Markos, Y.A. Melaku, K.G. Meles, W. Mendoza, D.T. Mengiste, T.J. Meretoja, T.R. Miller, K.A. Mohammad, A. Mohammadi, S. Mohammed, M. Moradi-Lakeh, G. Nagel, D. Nand, O. Le Nguyen, S. Nolte, F.A. Ogbo, K.E. Oladimeji, E. Oren, M. Pa, E.-K. Park, D.M. Pereira, D. Plass, M. Qorbani, A. Radfar, A. Rafay, M. Rahman, S.M. Rana, K. Søreide, M. Satpathy, M. Sawhney, S.G. Sepanlou, M.A. Shaikh, J. She, I. Shiue, H.R. Shore, M.G. Shrime, S. So, S. Soneji, V. Stathopoulou, K. Stroumpoulis, M.B. Sufiyan, B.L. Sykes, R. Tabarés-Seisdedos, F. Tadese, B.A. Tedla, G.A. Tessema, J.S. Thakur, B.X. Tran, K.N. Ukwaja, B.S.C. Uzochukwu, V.V. Vlassov, E. Weiderpass, M. Wubshet Terefe, H.G. Yebyo, H.H. Yimam, N. Yonemoto, M.Z. Younis, C. Yu, Z. Zaidi, M.E.S. Zaki, Z.M. Zenebe, C.J.L. Murrav, M. Naghavi, Global, regional, and national cancer incidence, mortality, years of life lost, years lived with disability, and disability-adjusted life-years for 32 cancer groups, 1990 to 2015: a systematic analysis for the global burden of disease study, JAMA Oncol. 3 (2017) 524-548, https://doi.org/10.1001/jamaoncol.2016.5688.
- [28] D. Benford, P.M. Bolger, P. Carthew, M. Coulet, M. DiNovi, J.C. Leblanc, A.G. Renwick, W. Setzer, J. Schlatter, B. Smith, W. Slob, G. Williams, T. Wildemann, Application of the Margin of Exposure (MOE) approach to substances in food that are genotoxic and carcinogenic, Food Chem. Toxicol. 48 (2010) S2–S24, https://doi.org/10.1016/j.fct.2009.11.003.
- [29] M. Alsafran, K. Usman, M. Rizwan, T. Ahmed, H. Al Jabri, The carcinogenic and non-carcinogenic health risks of metal(oid)s bioaccumulation in leafy vegetables: a consumption advisory, Front. Environ. Sci. 9 (2021) 1–11, https://doi.org/10.3389/fenvs.2021.742269.
- [30] T.H. Tulchinsky, E.A. Varavikova, Measuring, Monitoring, and Evaluating the Health of a Population, New Public Heal, 2014, pp. 91–147, https://doi.org/ 10.1016/B978-0-12-415766-8.00003-3.
- [31] B. Devleesschauwer, A.H. Havelaar, C. Maertens de Noordhout, J.A. Haagsma, N. Praet, P. Dorny, L. Duchateau, P.R. Torgerson, H. Van Oyen, N. Speybroeck, DALY calculation in practice: a stepwise approach, Int. J. Public Health. 59 (2014) 571–574.
- [32] B. Devleesschauwer, A.H. Havelaar, C. Maertens de Noordhout, J.A. Haagsma, N. Praet, P. Dorny, L. Duchateau, P.R. Torgerson, H. Van Oyen, N. Speybroeck, Calculating disability-adjusted life years to quantify burden of disease, Int. J. Public Health 59 (2014) 565–569.
- [33] G.B.D. Compare, IHME, University of Washington., Seattle, WA, 2017. https://vizhub.healthdata.org/gbd-compare/.
- [34] Y. Li, J. Liu, Y. Wang, S. Wei, Cancer risk and disease burden of dietary acrylamide exposure in China, 2016, Ecotoxicol. Environ. Saf. 238 (2022), 113551, https://doi.org/10.1016/j.ecoenv.2022.113551.
- [35] L.S. Jakobsen, K. Granby, V.K. Knudsen, M. Nauta, S.M. Pires, M. Poulsen, Burden of disease of dietary exposure to acrylamide in Denmark, Food Chem. Toxicol. an Int. J. Publ. Br. Ind. Biol. Res. Assoc. 90 (2016) 151–159, https://doi.org/10.1016/j.fct.2016.01.021.
- [36] B. Başaran, B. Çuvalcı, G. Kaban, Dietary acrylamide exposure and cancer risk: a systematic approach to human epidemiological studies, Foods 12 (2023) 346, https://doi.org/10.3390/foods12020346.
- [37] J.P.T. Higgins, J. Thomas, J. Chandler, M. Cumpston, T. Li, M.J. Page, V.A. Welch, Cochrane Handbook for Systematic Reviews of Interventions, John Wiley & Sons, 2019.
- [38] L. Shamseer, D. Moher, M. Clarke, D. Ghersi, A. Liberati, M. Petticrew, P. Shekelle, L.A. Stewart, D.G. Altman, A. Booth, A.W. Chan, S. Chang, T. Clifford, K. Dickersin, M. Egger, P.C. Gøtzsche, J.M. Grimshaw, T. Groves, M. Helfand, J. Higgins, T. Lasserson, J. Lau, K. Lohr, J. McGowan, C. Mulrow, M. Norton, M. Page, M. Sampson, H. Schünemann, I. Simera, W. Summerskill, J. Tetzlaff, T.A. Trikalinos, D. Tovey, L. Turner, E. Whitlock, Preferred reporting items for systematic review and meta-analysis protocols (prisma-p) 2015: elaboration and explanation, BMJ 349 (2015) 1–25, https://doi.org/10.1136/bmj.g7647.
- [39] N. Nemeth, I. Rudnak, P. Ymeri, C. Fogarassy, The role of cultural factors in sustainable food consumption—an investigation of the consumption habits among international students in Hungary, Sustainability 11 (2019) 3052.
- [40] Who, Principles and Methods for the Risk Assessment of Chemicals in Food, International Programme on Chemical Safety, vol. 240, environmental health criteria, 2009.
- [41] G. Adani, T. Filippini, L.A. Wise, T.I. Halldorsson, L. Blaha, M. Vinceti, Dietary intake of acrylamide and risk of breast, endometrial, and ovarian cancers: a systematic review and dose–response meta-analysis, Cancer Epidemiol. Biomarkers Prev. 29 (2020) 1095–1106.

- [42] A. Maertens, E. Golden, T.H. Luechtefeld, S. Hoffmann, K. Tsaioun, T. Hartung, Probabilistic risk assessment the keystone for the future of Toxicology, ALTEX 39 (2022) 3–29, https://doi.org/10.14573/altex.2201081.
- [43] A. Botchkarev, Assessing Excel VBA Suitability for Monte Carlo Simulation, 2015 arXiv preprint arXiv:1503.08376, https://doi.org/10.48550/arXiv.1503.08376.
- [44] J.W. Osborne, A. Overbay, The power of outliers (and why researchers should always check for them), Practical Assess. Res. Eval. 9 (2004) 6.
- [45] A. Gavrieli, A. Naska, C. Konstantinidi, R. Berry, M. Roe, L. Harvey, P. Finglas, M. Glibetic, M. Gurinovic, A. Trichopoulou, Dietary Monitoring Tools for Risk Assessment, EFSA Support, vol. 11, Publ., 2017, https://doi.org/10.2903/sp.efsa.2014.en-607.
- [46] U.S.E.P.A. USEPA, Characterizing risk and hazard, in: Hum. Heal. Risk Assess. Protoc., U.S. EPA, Office of Solid Waste, 2005, pp. 1–15.
- [47] EFSA, Overview of the procedures currently used at EFSA for the assessment of dietary exposure to different chemical substances, EFSA J. 9 (2011) 2490.
- [48] W. Bank, The World Bank Annual Report 2022, The World Bank, 2022.
- [49] E. Vardell, Global health observatory data repository, Med. Ref. Serv. Q. 39 (2020) 67-74.
- [50] K. Dibaba, L. Tilahun, N. Satheesh, M. Geremu, Acrylamide occurrence in Keribo: Ethiopian traditional fermented beverage, Food Control 86 (2018) 77–82, https://doi.org/10.1016/j.foodcont.2017.11.016.
- [51] R.A. Sanusi, A. Olurin, Portion and serving sizes of commonly consumed foods, in ibadan, southwestern Nigeria, Afr. J. Biomed. Res. 15 (2012) 149–158.
- [52] Z. Ajani, O.T. Fatunsin, A.O. Oyeyiola, K.O. Olayinka, Risk assessment of acrylamide for some commonly eaten fried foods, FUW Trends Sci. Technol. Journal, Www.Ftstjournal.com e-ISSN. 4 (2048) 149–155. www.ftstjournal.com.
- [53] O.O. Elizabeth, S. Anuoluwapo, O.J. John, Effect of deep and infrared rays frying on the acrylamide concentration formation in musa paradisiaca, Am. J. Food Technol. 12 (2017) 385–389, https://doi.org/10.3923/ajft.2017.385.389.
- [54] M. Oppong Siaw, I.W. Ofosu, H.E. Lutterodt, G.M. Ankar-Brewoo, Acrylamide exposure and risks in most frequently consumed foods in a total diet study, Adv. J. Food Sci. Technol. 6 (2018) 123–137, https://doi.org/10.12691/ajfst-6-4-1.
- [55] L.C. Peres, H. Risch, K.L. Terry, P.M. Webb, M.T. Goodman, A.H. Wu, A.J. Alberg, E. V Bandera, J. Barnholtz-Sloan, M.L. Bondy, M.L. Cote, E. Funkhouser, P. G. Moorman, E.S. Peters, A.G. Schwartz, P.D. Terry, A. Manichaikul, S.E. Abbott, F. Camacho, S.J. Jordan, C.M. Nagle, M.A. Rossing, J.A. Doherty, F. Modugno, K. Moysich, R. Ness, A. Berchuck, L. Cook, N. Le, A. Brooks-Wilson, W. Sieh, A. Whittemore, V. McGuire, J. Rothstein, H. Anton-Culver, A. Ziogas, C.L. Pearce, C. Tseng, M. Pike, J.M. Schildkraut, Racial/ethnic differences in the epidemiology of ovarian cancer: a pooled analysis of 12 case-control studies, Int. J. Epidemiol. 47 (2018) 460–472, https://doi.org/10.1093/ije/dyx252.
- [56] V.A. Zavala, P.M. Bracci, J.M. Carethers, L. Carvajal-Carmona, N.B. Coggins, M.R. Cruz-Correa, M. Davis, A.J. de Smith, J. Dutil, J.C. Figueiredo, R. Fox, K. D. Graves, S.L. Gomez, A. Llera, S.L. Neuhausen, L. Newman, T. Nguyen, J.R. Palmer, N.R. Palmer, E.J. Pérez-Stable, S. Piawah, E.J. Rodriquez, M.C. Sanabria-Salas, S.L. Schmit, S.J. Serrano-Gomez, M.C. Stern, J. Weitzel, J.J. Yang, J. Zabaleta, E. Ziv, L. Fejerman, Cancer health disparities in racial/ethnic minorities in the United States, Br. J. Cancer 124 (2021) 315–332, https://doi.org/10.1038/s41416-020-01038-6.
- [57] Y. Wang, Q. Chang, Y. Li, Racial differences in urinary bladder cancer in the United States, Sci. Rep. 8 (2018), 12521, https://doi.org/10.1038/s41598-018-29987-2.
- [58] M.C. White, D.M. Holman, J.E. Boehm, L.A. Peipins, M. Grossman, S.J. Henley, Age and cancer risk: a potentially modifiable relationship, Am. J. Prev. Med. 46 (2014) S7–S15, https://doi.org/10.1016/j.amepre.2013.10.029.
- [59] E. Tareke, P. Rydberg, P. Karlsson, S. Eriksson, M. Törnqvist, Analysis of acrylamide, a carcinogen formed in heated foodstuffs, J. Agric. Food Chem. 50 (2002) 4998–5006.
- [60] K.F. Park, Z. Shapira, in: M. Augier, D.J. Teece (Eds.), Risk and Uncertainty BT the Palgrave Encyclopedia of Strategic Management, Palgrave Macmillan UK, London, 2017, pp. 1–7, https://doi.org/10.1057/978-1-349-94848-2_250-1.