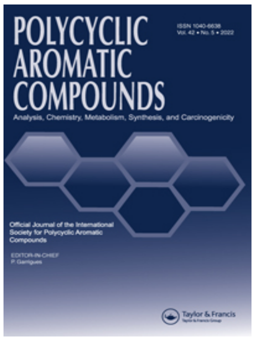

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Dietary Risk Assessment Due to the Consumption of Polycyclic Aromatic Hydrocarbon in Two Commonly Consumed Street Vended Foods

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ABSTRACT

Polycyclic aromatic hydrocarbons (PAH) are present and pervasive in the environment. Handling of the foods by the vendors is therefore likely to introduce PAHs into the meals. Quantification of PAH was made using HPLC-FID and consumption data by face–face interviews with 188 regular consumers for the vendors. Data obtained were iterated 10,000 times using palisade @risk software and results presented at 95th percentile level of consumption showing the worst case scenario. Dietary risk assessment of 12-priority PAHs was carried out on the components of two staple foods in Ghana. Naphthalene was detected in all food samples at concentrations ranging from 1.7 to 6.5 mg kg^{-1} at 95th percentile consumption level. Benzo(a)pyrene and dibenz-a,h-anthracene were detected in fried chicken samples with the stochastic concentrations of 1.2E–02 mg kg^{-1} and 3.2E–03 mg kg^{-1}, respectively, at the 95th percentile level of consumption. Incremental life cancer risk values fell within the deminis value of $\leq 10^{-6}$ to 10^{-4} except at the 95th percentile level of consumption of chicken, hot pepper sauce, and soup components of the meals which were higher (10^{-3}). The 95th percentile level of consumption of fried rice meal as well as the 50th and 95th percentile level of consumption of the fufu meal had a hazard index above 1, indicating consumers may be suffering the non-carcinogenic effects due to PAH in the respective meals. The study, therefore, showed that high consumption levels of the street vended foods are likely to cause dietary risk to the health of its consumers.

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Exposure; food consumption; food contamination; hazard index; polycyclic aromatic hydrocarbon; risk assessment

Introduction

There is a general perception that street-vended foods in the developing countries are unsafe for consumption, considering the conditions in which they are prepared, sold, or consumed.[1] Street-vended foods have unique flavors and taste, depicting the cultural and social heritage of societies

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where they are found,[2] thus boosting the tourism industry by attracting tourists.[3] Street-vended foods are highly patronized where a consumer may depend on totally for daily meals. Unhygienic conditions at the locations of vending and poor knowledge on food hygiene among vendors can result in the exposure of the food to contamination and thereby rendering them unsafe for consumption.[4] Epidemiological data, mostly from microbiological standing, points to the incidence of food-borne diseases resulting from the consumption of popular street-vended foods.[2,5] The cooking procedures used by the vendors in meal preparations may increase the content of some process contaminants, such as polycyclic aromatic hydrocarbons (PAHs).[6] Chemical contaminants usually have a long-term intoxication effects due to chronic exposures unlike the microbiological intoxication with short-term effects realized at a maximum of 1 week. The effects of chemical contaminants on human health depends mainly on the length and route of exposure, the concentration one is exposed to, as well as the relative toxicity of the contaminants.

PAHs are ubiquitous environmental contaminants which are produced mainly as a result of oil spillage into open fires and/or incomplete combustion of organic materials including wood, fossil fuels and petroleum products.[7] Many of the PAH congeners are mutagenic[8] while some have also proven to be carcinogenic.[9,10] These hazardous effects of the PAH raise concerns about their presence in the environment and in foods.[11,12] PAHs are lipophilic, hence they can be adsorbed on atmospheric particles such as dust and be transported over distances[13] and can also accumulate in lipid tissues of plants and animals used as food.[14] However, food processing procedures such as smoking, drying, and cooking,[6] are commonly thought to be the major sources of PAH contamination in food.[10] There are several mechanisms for the formation of PAH such as heated fat that undergoes pyrolysis when it drips into open heat during grilling of meat, for example, the grilling of khebabs and fish. Also, the pyrolysis of meat which occurs due to the high temperatures used during roasting.[9] Overall, consumption of food containing PAH is the major route of human exposure to PAH.[9,15,16] Inhalation and dermal exposures are other routes by which PAH can enter into the human body.[17–20] The health effects of PAH could be classified as short- and long-term, carcinogenic and non-carcinogenic. Some long-term non-carcinogenic effects of PAH are hemolytic anemia, fatigue, restlessness, cataracts, kidney and liver damage (e.g., jaundice), breathing problems, asthma-like symptoms, and lung function abnormalities.[21] Some short-term non-carcinogenic effects of PAH may include eye irritation, nausea, vomiting, diarrhea, and confusion.[21]

Generally, streets are characterized by high levels of PAH in the air as a result of the smoke from the exhaust fumes of vehicles.[13] Street food vending activity is very common at transport terminals and major streets in the major cities of Ghana.[22] Again, preparation of street-vended foods are characterized by such processes as grilling, smoking, over-cooking of some starch, and protein-rich foods at high temperatures.[6] These cooking processes are likely to introduce PAH into the street-vended cooked foods. Frequent patronage or consumption of such contaminated foods is most likely to expose consumers to PAH. However, information on the concentrations of PAH in street-vended foods as well as the possible risk they pose to the consuming population is scanty. In this work, we determined the concentrations of 12 USEPA priority PAH in two commonly consumed street-vended foods (fufu and fried rice) in Kumasi, Ghana. The risks they pose to consumers were evaluated at the 50th and 95th levels of consumption. The findings of the study will enable both consumers and policy makers to estimate the level of hazards of PAH in consuming street-vended foods and its associated risks at the various levels of consumption. Such information will be useful for the revision of existing policies on vendor-cooking procedures that have the potential of introducing PAH into the foods.

Materials and methods

This study was conducted in Kumasi, the second largest city in Ghana which has 10 metropolitan areas. Three of the metropolitan areas namely, Asawase, Oforikrom, and Subin, were purposefully selected due to high concentration of street food vending activity for the study. A total of 18 vendors each of fried rice and fufu (pounded cassava and plantain) participated in the study. Informed or oral consent was sought from the vendors who had been in operation for at least 3 years. Vendors who agreed and consented to the study were recruited into the study.

All food sampling and analysis were done according to IARC guidelines for risk assessment (Internal Report 14/001). Sampling of food was conducted between the period from May to August, 2014. Food samples were bought from the vendors and separated into the various components of each meal at point of purchase by the vendors. The typical components of fried rice meal are rice, fried chicken, hot pepper sauce (shito), cut vegetables as salad, macaroni, ketchup, and mayonnaise. Typical components of a fufu meal are fufu (pounded cooked cassava and plantain) and soup. In total, 120 components of the fried rice meal and 36 components of the fufu meal samples were collected from 18 fried rice and 18 fufu vendors. Individual weights of the food samples were recorded and the food samples were stored in a $-20\,^{\circ}C$ deep freezer (HTF-519GB, Lagos, Nigeria) prior to the analysis. Raw (uncooked) or unprocessed food samples were collected from the vendors and used as control samples. Site-specific exposure parameters[23,24] such as ingestion rate (IR), exposure frequency (EF) and duration (ED) were obtained using face-to-face interviews with 188 regular consumers of both meal types (fufu and fried rice) from the selected vendors. The severest possible outcome (risk) (worst case scenario) that can reasonably be projected to occur in the consumption of the street vended foods was desired hence the use of regular consumers.

All analytical works were done on the food samples as they were obtained. PAH was first extracted from the samples using the method described by AOAC.[25] In brief, 10 mL of acetonitrile (HPLC grade) was added to 5 g portion of each of the food component samples in a 50 mL centrifuge tube and vortexed for 1 min. A 6 g portion of anhydrous magnesium sulfate ($MgSO_4$) and 1.5 g of anhydrous sodium acetate (NaOAc) were added and vortexed for 1 min after which the tube and its contents were centrifuged at 4000 rpm for 5 min. A 6.0 mL aliquot of the upper acetonitrile layer was transferred into an AOAC QuEChERS dispersive solid phase extraction tubes (15 mL tube) which contains 400 mg of primary secondary amines, 40 mg of C_{18} EC and 1200 mg of anhydrous $MgSO_4$. The mixture was vortexed for 1 min and then centrifuged at 4000 rpm for 5 min. A 4 mL aliquot of the extract was filtered through a 0.45 µm polyvinylidene fluoride syringe filter and then 0.1 mL of the extract was injected into the HPLC system.

The standard mix of PAH, in methylene chloride: methanol (1:1) was purchased from Sigma Aldrich (catalogue number, 861291). The analysis was based on Shimadzu Application Note (LC-022) Demuro protocol with some modifications: A Cecil-Adept binary pump HPLC coupled with Shimadzu 10AxL fluorescence detector (Excitation: 254 nm, Emission: 390) with Phenomenex HyperClone BDS C18 Column (150×4.60 mm, 5 µm). Mobile phase composition was Pump A (Acetonitrile) and Pump B (Deionized Water) at 0.8 mL/min. Gradient elution was used with the following combination, $0-5$ min $=$ 60% A, 40% B; $5-15$ min $=$ 90% A, 10% B; $15-28$ min $=$ 100% A, 0% B; $28-30$ min $=$ 60% A, 40% B. The PAH in samples were identified using the retention times against the standards and quantified using the calibration curve developed (Figure 1). The concentration of the PAH in the standard mix were as follows: 2.5 µg kg^{-1} for Naph, 1MN, 2MN, and Ace; 5 µg kg^{-1} for Ant, Pyr, Flu, BbF, and BkF; 10 µg kg^{-1} for Flu and BaP then 20 µg kg^{-1} for DahA.

Legend

1: Naphthalene; 2: 1-methylnaphthalene; 3: 2-methynaphthalene; 4: Acenaphthene; 5: Fluorene; 6: Anthracene; 7: Fluoranthene; 8: Pyrene; 9: benzo-b-fluoranthene; 10: benzo-k-fluoranthene; 11: benzo-a-pyrene; 12: dibenz-a,h-anthracene

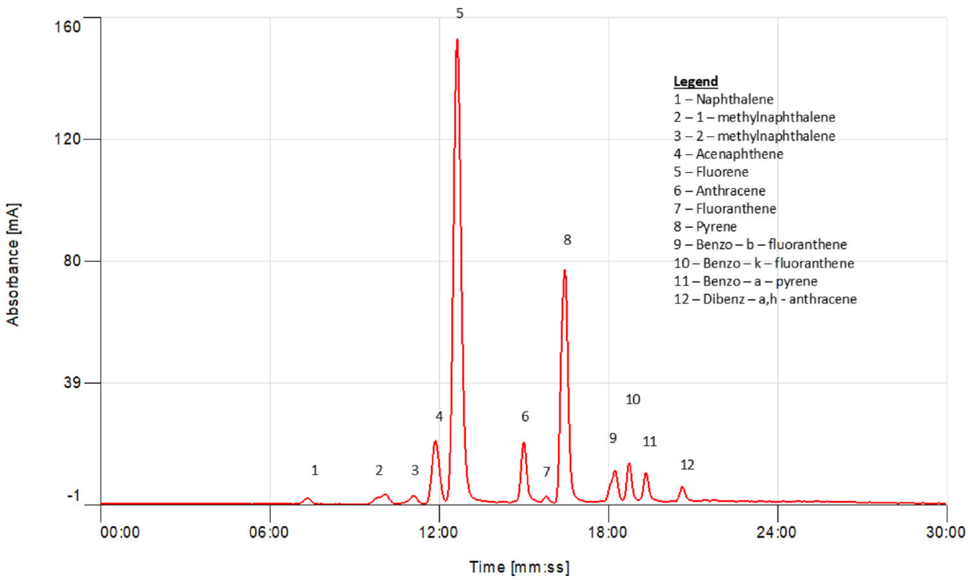

Figure 1. Chromatogram of the standard mix used to identify the peaks of the PAH in the sample.

Quantitation was performed using a 5-point internal standard calibration with $r^2 \geq 0.98$. Limits of detection (LOD) were calculated based on 3SD/*s* (SD is the standard deviation of the response of five replicate measurements and *s* is the slope of the calibration graph). LOD of PAH were in the range 0.05–6.46 ng/g. Recovery assessment was done by spiking matrix with naphthalene (Naph): 100 ng/g; fluorine (Fle): 40 ng/g; pyrene (Pyr): 20 ng/g; benzo [a] pyrene (BaP): 20 ng/g, prior to extraction with standard mix. The average recovery was 114% ± 1.42%. The final PAH concentrations were not corrected from the recoveries of the internal standards. Blanks were run periodically and contained no detectable amounts of target analyte. No significant difference ($p > 0.05$) was observed in PAH concentration of duplicate samples.

Responses from the consumer interviews were transcribed into an excel worksheet as consumption data, and analyzed using the @Risk version 6 software with Palisade Inc, USA. The PAH concentrations and consumption data were initially fitted into a distribution curve and then iterated 10,000 times in the first order Monte Carlo to calculate the stochastic concentrations of the hazards, IR, exposure frequency, and duration (input factors). Results were presented in the 5th, 50th, and 95th percentile level of consumption.[26] These levels give a complete picture of the distributions of the hazards at the levels of consumption. The 50th percentile for a normal distribution indicates, the median and mean.

The dietary risks due to consumption of PAH was categorized into cancer risks and non-cancer risks according USEPA.[10] Dietary risk assessment was conducted using the hazard index (HI) (Equations (1) and (2)).[24] The HI calculations were used for non-cancer PAH and incremental lifetime cancer risk (ILCR) calculations for carcinogenic PAH (Equations (3) and (4)).[24] An hazard quotient (HQ) >1 means that there is a chance of adverse effects of the hazards, with an increasing probability as the value increases.[27]

$$HQ = \frac{CDI}{RfD} \quad (1)$$

where the oral reference doses (R_fD) for naphthalene (Naph), I-methylnapthalene (1MN), 2-methylnaphtahlene (2MN), acenaphthene (Ace), fluorene (Fle), antracene (Ant), fluoranthene (Flu), and pyrene (Pyr) are given as 2.0×10^{-2}, 7.0×10^{-2}, 4.0×10^{-3}, 6.0×10^{-2}, 4.0×10^{-2}, 3.0×10^{-2}, 4.0×10^{-2}, and 1.0×10^{-3} mg kg^{-1} day^{-1}, respectively.[28]

It has been reported that exposure to two or more pollutants may result in additive and/or interactive effects.[27] Therefore, the HI of the non-cancer causing PAH for individual food components was treated as the arithmetical sum of the HQ of the individual PAH (Equation (3)).[24]

$$\mathrm{HI} = \mathrm{HQ}_{(n=1)} + \mathrm{HQ}_{(n=2)} + \mathrm{HQ}_{(n)} \tag{2}$$

where n refers to the HQ of the individual PAH.

Chronic daily intake (CDI) for each PAH was calculated using Equation (3).

$$\mathrm{CDI} = \frac{\mathrm{Cs} \times \mathrm{IR} \times \mathrm{EF} \times \mathrm{ED}}{\mathrm{BW} \times \mathrm{AT}} \tag{3}$$

where the CDI represents the amount of chemical (hazard) intake per kilogram body weight per day (μg kg^{-1} day^{-1}). The input factors for the calculation of the CDI are as follows: Cs represents the average concentration of a particular PAH (μg kg^{-1}); IR represents the ingestion rate (kg day^{-1}) for a given meal (given in the stochastic determination from the consumption studies); EF represents the exposure frequency (days/year) of consumption, ED represents the exposure duration (year), the total number of years the consumers have been exposed to the hazard, BW represents the body weight of the consumer (a 70 kg body weight was assumed for the adult consumers), and AT represents the pathway-specific period of exposure, for non-carcinogenic effects (ED × 365 days/year) and 70-year lifetime for carcinogenic effects (i.e., 70 year × 365 days/year), averaging time.[24]

For the carcinogenic PAH, the ILCR, in humans was determined by multiplying the CDI of the PAH with the respective potency factor (PF) according to the USEPA guidelines[24] using the Equation (4) which is given as follows:

$$\text{Incremental Lifetime Cancer Risk (ILCR)} = \mathrm{CDI} \times \mathrm{PF} \tag{4}$$

The PF for the carcinogenic PAH, benzo-b-fluoranthene (BbF), benzo-k-fluoranthene (BkF), benzo-a-pyrene (BaP), and dibenz-a,h-anthracene (DahA) are 7.3E–01, 7.3E–02, 7.3E + 00 and 7.3E + 00, respectively. Data were presented at the 95th percentile level of consumption as the worst case scenario was desired.

Results and discussion

The LOQs for selected PAH compounds, were under 2 μg/kg and the linearity of the calibration curves of each PAH compound had a high regression levels in the food samples, with coefficient (R^2 = 0.99). Therefore, these results showed sufficient sensitivity for the detection of PAH.[29] The stochastic concentrations of the non-carcinogenic PAH in the components of the meals types used in the study are as shown in Table 1. The non-carcinogenic PAH detected were Naph, 1MN, 2MN, Ace, Flu, Ant, Fle, and Pyr. Naphthalene was the predominant PAH detected in all the food samples. The stochastic concentration was as high as 6500 μg kg^{-1} (Table 1) in rice and hot pepper sauce samples at 95th percentile level of consumption in the components. The rice was cooked/boiled with stored water and the ingredients for the hot pepper sauce was stored in the storage rooms prior to use. The ingredients were again mixed with some stored water before preparing the hot pepper sauce. The recorded levels of Naph could be due to the use of Naph balls (moth balls), which vendors put in stored water (as water purification agent) and also placed at vantage places in storage rooms as insect repellent.[30] This stored water is used for the all cooking and vending processes. In fufu preparation, for example, excess raw cassava is stored in water for use the next day, therefore the water gets imbibed into the raw cassava which could lead to the high levels of Naph in the meal (1.7 E + 03 μg kg^{-1} at 95th percentile level of consumption). The same stored water is used to prepare the soup (2.8 E + 05 μg kg − 1 at 95th percentile level of consumption, Table 1). The soup contains about 92% water. There is, therefore, the need to find alternative means of storing water without the

Table 1. Stochastic concentrations, chronic daily intakes and hazard indices values of the non-carcinogenic PAH.

		Concentration (μg kg^{-1})		CDI (mg kg^{-1} day^{-1})	RfD (mg kg^{-1} day^{-1})	HI
Food sample	PAH	50th percentile	95th percentile	95th percentile		95th percentile
Rice	Naphthalene	1.7E + 03	6.5E + 03	2.0E–02	1.4E–02	1.94E + 00
	1-methylnapthalene	1.8E–01	8.4E–01	7.0E–02	9.0E–05	
	2- methylnapthalene	3.9E + 00	1.8E + 01	4.0E–03	1.9E–03	
	Acenaphthene	2.7E + 01	1.2E + 02	6.0E–02	1.3E–02	
	Fluorene	2.0E + 01	9.5E + 01	4.0E–02	9.5E–03	
	Anthracene	8.8E–01	4.1E + 00	3.0E–01	4.3E–04	
	Fluoranthene	2.7E + 01	1.3E + 02	4.0E–02	1.3E–02	
	Pyrene	5.8E–01	2.7E–00	3.0E–02	2.7E–04	
Chicken	Naphthalene	1.8E + 01	2.3E + 03	2.0E–02	2.3E–02	2.77E + 00
	1-methylnapthalene	4.2E + 01	2.0E + 02	7.0E–02	4.3E–03	
	2- methylnapthalene	5.0E + 01	2.3E + 02	4.0E–03	5.0E–03	
	Acenaphthene	9.4E + 00	4.4E + 01	6.0E–02	9.2E–04	
	Fluorene	1.1E + 01	5.2E + 01	4.0E–02	1.1E–03	
	Anthracene	7.7E + 00	3.6E + 01	4.0E–02	7.5E–04	
	Fluoranthene	8.6E–03	4.0E–02	3.0E–02	8.3E–07	
Hot pepper sauce	Naphthalene	1.7E + 03	6.5E + 03	2.0E–02	4.4E–02	4.71E + 00
	1-methylnapthalene	6.6E + 01	3.1E + 02	7.0E–02	2.0E–03	
	2- methylnapthalene	3.7E + 02	1.7E + 03	4.0E–03	1.1E–02	
	Acenaphthene	4.6E + 01	2.1E + 02	6.0E–02	1.4E–03	
	Fluorene	3.6E + 01	1.7E + 02	4.0E–02	1.1E–03	
	Anthracene	5.2E + 00	2.4E + 01	3.0E–01	1.6E–04	
	Fluoranthene	9.9E + 01	4.6E + 02	4.0E–02	2.9E–03	
	Pyrene	6.3E–01	2.9E + 00	3.0E–02	1.9E–05	**1.12E + 01**
Fufu	Naphthalene	1.3E + 01	1.7E + 03	2.0E–02	1.5E–01	4.78E + 01
	1-methylnapthalene	2.1E + 01	9.5E + 01	7.0E–02	2.1E–02	
	2- methylnapthalene	1.1E + 02	5.3E + 02	4.0E–03	1.2E–01	
	Acenaphthene	5.0E + 00	2.3E + 01	6.0E–02	5.2E–03	
	Fluorene	3.5E–01	1.6E + 00	4.0E–02	3.6E–04	
Soup	Naphthalene	2.2E + 01	2.8E + 03	2.0E–02	2.3E–01	6.46E + 01
	1-methylnapthalene	6.4E + 01	2.9E + 02	7.0E–02	5.9E–02	
	2- methylnapthalene	1.7E + 02	8.0E + 02	4.0E–03	1.6E–01	
	Acenaphthene	4.2E + 01	8.5E + 01	6.0E–02	2.6E–02	
	Fluorene	2.8E–01	1.3E + 00	4.0E–02	2.6E–04	
	Anthracene	6.3E–01	2.9E + 01	3.0E–01	5.8E–04	
	Fluoranthene	4.1E + 01	1.9E + 02	4.0E–02	3.6E–02	
	Pyrene	4.3E–01	2.0E + 00	3.0E–02	3.7E–04	**1.30E + 02**

Figure in **bold** indicate the total Hazard Indices of the respective meals.
CDI: chronic daily intake; RfD: oral reference dose; HI: hazard index; PAH: polycyclic aromatic hydrocarbon.

use of moth balls. Good house-keeping practices could prevent the use of Naph as insect repellent and hence reduce the levels of Naph present in the food samples.

Pyrene, was the least detected PAH in fried rice, hot pepper sauce, and soup samples. The average concentrations of Pyr detected were 2.7, 2.9, and 2.0 μg kg^{-1}, respectively, in fried rice, hot pepper sauce and soup samples at the 95th percentile level of consumption. In a related study conducted in Turkey, Pyr was detected in barbecued beef meat and pan fried beef meat with concentrations of 1.72 and 1.62 μg kg^{-1}, respectively.[29] Pyrene is of concern because it is believed to act as a precursor to BaP,[31] the most potent carcinogenic PAH.[32] However, no safe limits have been established for dietary concentration of Pyr.

Benzo-a-pyrene was detected in fried chicken, hot pepper sauce, and soup at concentrations of 12, 20, and 6.3 μg kg^{-1}, respectively, at the 95th percentile level of consumption (Table 2). The results showed that at the 95th percentile level of consumption, the consumers of the fried rice meal were exposed to DahA at concentrations of 3.2 μg kg^{-1}. The presence of BaP and DahA in these meals is not desired because of their carcinogenic properties.[33] These possible human carcinogens do not have safe limits of exposure, so all exposure routes should be reduced to the barest minimum.[34] Nine of the vendors were located at the major transport terminal and six were

Table 2. Stochastic concentrations and incremental life cancer risks values of carcinogenic PAH.

		Concentrations (μg kg^{-1})		ILCR	
Food component	Polycyclic aromatic hydrocarbon	50th percentile	95th percentile	50th percentile	95th percentile
Rice	Benzo-b-fluoranthene	7.4E–01	3.4E + 00	1.6E–05	2.5E–04
	Benzo-k-flouranthene	6.4E + 00	3.0E + 01	1.3E–05	2.2E–04
Chicken	Benzo-b-fluoranthene	8.4E–03	3.9E–02	3.4E–08	6.1E–07
	Benzo-k-flouranthene	1.5E–02	6.9E–02	6.1E–09	1.1E–07
	Benzo-a pyrene	2.6E + 00	1.2E + 01	1.1E–04	1.8E–03
	Dibenz-a,h-anthracene	7.0E–01	3.2E + 00	2.9E–05	5.0E–04
Hot pepper sauce	Benzo-b-fluoranthene	2.3E + 01	1.1E + 02	3.0E–05	5.0E–04
	Benzo-k-flouranthene	4.3E + 00	2.0E + 01	5.3E–07	9.1E–06
	Benzo-a pyrene	5.8E + 00	2.7E + 01	7.3E–05	1.3E–03
Soup	Benzo-a pyrene	1.4E + 00	6.3E + 00	3.7E–04	9.0E–03

ILCR: incremental life cancer risk.

located beside busy roads. At these locations, exhaust fumes from vehicles can easily contaminate the exposed fried chicken while cooling after frying. Some vendors actually grill chicken on naked fire which ultimately leads to the charring of certain parts of the chicken as it was grilled. The charring processes could induce the formation of PAH in the meat.[35]

No PAH was detected in some of the components of fried rice, namely, vegetables, ketchup mayonnaise, and macaroni for all the samples as well as in the control. The salads which consisted of chopped cabbage, onions, lettuce leaves, and grated carrots and sometimes sliced cucumbers were stored in airtight containers by the vendors. Possibly, preparation procedures such as peeling of the carrots and onions, the removal of the outer layer of the cabbage before shredding, could have helped reduce the PAH on the vegetables.[15] Interestingly in a study by Ofosu et al.,[36] B(b)F concentrations of 4.0, 2.0, and 2.0 μg kg^{-1} was detected in some vegetables samples collected from street food vending sites in namely Asafo, KNUST, and Adum, respectively, in Kumasi, Ghana. The rest of the investigated PAH were below the detected limits. It is documented that careful washing and cleaning may remove up to 50% of the total PAH on the average.[14,37] The observed results may mean that the vendors washed the vegetables very well before vending. Mayonnaise and ketchup are industrial products that are obtained from the market and used as toppings on the salads. They are poured into refill bottles after mixing it up with some water to increase the volume and flow. At the vending site, these are not left exposed but are dispensed in bottles hence environmental contact with these products is minimal.[4]

The boiled macaroni, a component of the fried rice meal did not contain PAH. No studies have been published on PAH levels in cooked macaroni. Fluorene, Pyr, and BaP were expected in the cooked macaroni because these PAH are known to be associated with domestic combustion of wood charcoal,[38] which was the primary fuel used for cooking by the vendors. The presence of PAH in the rice and chicken were probably as a result of vendor-cooking practices because none of them was detected in the uncooked rice and water.

The stochastic CDI due to PAH in the two meal types are shown in Table 1. The levels of exposure due to PAH at the 50th percentile level of consumption were lower than the respective oral R_fD (Table 1). This indicates that the amount of the PAH in the foods being consumed at the 50th percentile level of consumption were within safe levels.[27] At the 95th percentile level of consumption for fufu and soup, the CDI for Naph and 2MN were higher than the R_fD. This indicates that consumers at the 95^{th} percentile level of consumption stand a chance of suffering the adverse health effects of Naph and 2MN.[24]

Health risk assessment

The HI of the fried rice meal components was <1 at the 50th percentile levels of exposure. However, at the 95th percentile level of exposure, all the meal types in the study had an HI of

Table 3. Sensitivity analysis on the impact of input factors on the hazard index.

Fufu meal				Rice meal			
IF	PAH	CC	RC	IF	PAH	CC	RC
ED	–	0.69		ED	–	0.72	
EF	–	0.41		EF	–	0.47	
Soup	2MN	0.25		Shito	2MN	0.18	
Fufu	2MN	021		Shito	Naph	0.13	
Soup	Naph	012	0.52	Chicken	Naph	0.13	1.00
Fufu	Naph	0.11	0.14	Rice	Naph	0.09	0.04
IR (Soup)	–	0.07		Chicken	2MN	0.04	
IR (Fufu)	–	0.05		Rice	2MN	0.04	

IF: input factors; CC: correlation coefficient; RC: regression coefficient; ED: exposure duration; EF: exposure frequency; IR: ingestion rate; 2MN: 2-methylnaphthalene; Naph: naphthalene.

>1, with the HI of hot pepper sauce being the highest (Table 1). This implies that even though small quantities of hot pepper sauce are added to the fried rice meal, the heavy consumers (at 95th percentile level of consumption) stand the risk of suffering the non-carcinogenic effects. However, HI >1 was observed for the 50th percentile level consumers of the fufu meal. This indicates that about 50% of the consumers of fufu used in the study may be experiencing the non-carcinogenic effects due to the presence of the quantified PAH in the meals.

The mean incremental life cancer risks (ILCR) values for the clearly labeled carcinogenic PAH by USEPA, are as displayed in Table 2. At the 5th percentile level of consumption, the ILCR value was $\leq 10^{-6}$ and was between $\leq 10^{-6}$ and 10^{-4} for the 50th percentile level of consumption. Benzo-a-pyrene in all the components of the fried rice meals at the 95th percentile level of consumption was higher than 10^{-4}. These values represent the risk of getting cancer due to consumption of BaP in the components in the fried rice meals. High total exposure risks was also recorded for BaP(eq) contamination of vegetables since the median and modal ILTCR fell within both unacceptable risk ($\geq 10^{-6} \leq 10^{-4}$).[36] The carcinogenic risk from eating the chicken component of the fried rice meal is 2 out of 1000 people in the study population. The cancer risk for hot pepper sauce was approximately 1 out of 1000 people in the study population (Table 2). These values are unacceptable as the maximum point of acceptance is 1 out of 1,000,000.[39]

The ILCR due to BaP in the soup samples was higher than 10^{-3}, translating to 9 people out of 1000 in the study population. According to Whipple,[39] the de-minimis for cancer risk is 10^{-6}, meaning, 1 out of a million people in any risk calculation was acceptable. The safe limit for PAH cancer risk according to Bortey-Sam et al.,[20] ranged from $\leq 10^{-6}$ to 10^{-4}. Therefore, consumers at the 95th percentile level of consumption of soup and chicken were at risk of cancer at the given concentrations of BaP consumed.

Sensitivity analysis

A sensitivity analysis was performed on the overall HI to determine which input factors have the greatest effect on the risk estimates. The results of the sensitivity analysis are as shown in Table 3 as the spearman rank order correlation coefficients. These coefficients show the extent to which the two variables are linearly correlated. Exposure duration and exposure frequency were the most influential input variables on the HI for the fufu, with correlation coefficients of 0.69 and 0.41, respectively (Table 3). If the relationship is perfectly linear, then the correlation coefficient is +1 if there is a positive correlation and −1 if the line has a negative slope. There will be no linear relationship between the variables if the correlation coefficient is zero.[40]

In Table 3, the concentration of Naph in soup had the best linear relationship comparing with the concentration of Naph in the fufu sample on the HI. This indicates that the Naph in soup must be focused on for intervention and all possible exposure routes of Naph in the consumption of the soup reduced. A probable source of Naph and its derivatives in the fufu meal is the use of the stored water for the cooking process since Naph balls were observed in the stored water.[30]

The sensitivity analysis (Table 3) for the HI of the fried rice implicated the following input factors in descending order of correlation with the HI. The input factors are exposure duration, exposure frequency, concentrations of Naph, 1MN, and 2MN in hot pepper sauce, chicken and rice. Consumer based interventions needs to be focused on exposure duration (0.72) and frequency (0.47), since these factors impacted the HI most. A reduction in the duration and frequency of exposure of the hazard in the food can be focused on for interventions. However, since it is the presence of the hazard in the food and not the food itself, the actual intervention must be that for the hazard which is the presence of Naph in the rice, chicken, and hot pepper sauce. The possible source of this hazard may be as a result of the vendor and raw material supplier practice.

Table 3 shows the regression coefficients of the input factors on the HI of the fried rice meal. The presence of Naph in chicken and rice were the main input factors with strong regression effects on the HI. This meant that reducing the levels of Naph in this meal will greatly affect the HI, by reducing it, thereby improving the safety of the meal.

Conclusion

PAHs were detected in all the components of the fried rice except vegetables, ketchup, mayonnaise and macaroni sampled. The CDIs of the fried rice meal at the 95th percentile levels of exposure were lower than the oral R_fD indicating possible consumption at safe levels. The ILCR values fell within the deminis value of $\leq 10^{-6}$ to 10^{-4} except at the 95th percentile level of consumption of chicken, hot pepper sauce and soup components of the meals which were higher $>10^{-3}$. The HI for both meals at 95th percentile level of consumption was >1 indicating unsafe consumption. It is suggested that intervention for reducing HI of the street vended meals was eliminating the use of Naph balls in water.

Recommendation

Policies must be reviewed on the use of moth balls at food preparation areas. Research into the perception of vendors on the use of Naph balls could help understand the messages to be given to the vendors.

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Author's contribution

Gloria M. Ankar-Brewoo – Primary researcher, drafted the manuscript and coordinated in the review and submission of the manuscript. Godfred Darko – Supervised the research and participated in the reviewing of the manuscripts. Robert Clement Abaidoo – Supervised the research and also participated in the review of the manuscript. Anders Dalsgaard – Danish supervisor also on the research and participated in the review of the manuscript. Paa Nii Johnson – a researcher on the project who participated in the review of the manuscript. William Otoo Ellis – supervised the research too and participated in the review of the manuscript. Leon Brimer – Danish supervisor on the research who also participated in the review of the manuscript.

Disclosure statement

No potential conflict of interest was reported by the authors.

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References

1. F. M. Mosupye and A. von Holy, "Microbiological Hazard Identification and Exposure Assessment of Street Food Vending in Johannesburg, South Africa," *International Journal of Food Microbiology* 61, no. 2–3 (2000): 137–45.
2. S. Rane, "Street Vended Food in Developing World: Hazard Analyses," *Indian Journal of Microbiology* 51, no. 1 (2011): 100–6.
3. C. Bellia, M. Pilato, and H. Seraphin, "Street Food and Food Safety: A Driver for Tourism?," *Quality - Access to Success* 17 (2016): 20–7.
4. A. Lucca, and E. A. F. da Silva Torres, "Street-Food: The Hygiene Conditions of Hot-Dogs Sold in São Paulo," *Food Control* 17, no. 4 (2006): 312–6.
5. E. Ekanem, "The Street Food Trade in Africa: Safety and Socio-Environmental Issues," *Food Control* 9, no. 4 (1998): 211–5.
6. I. Proietti, C. Frazzoli, and A. Mantovani, "Identification and Management of Toxicological Hazards of Street Foods in Developing Countries," *Food and Chemical Toxicology* 63, (2014): 143–52.
7. K. Ravindra, R. Sokhi, and R. Vangrieken, "Atmospheric Polycyclic Aromatic Hydrocarbons: Source Attribution, Emission Factors and Regulation," *Atmospheric Environment* 42, no. 13 (2008): 2895–921.
8. M. J. Martena, M. M. P. Grutters, H. N. De Groot, E. J. M. Konings, and I. M. C. M. Rietjens, "Monitoring of Polycyclic Aromatic Hydrocarbons (PAH) in Food Supplements Containing Botanicals and Other Ingredients on the Dutch Market," *Food Additives and Contaminants. Part A, Chemistry, Analysis, Control, Exposure & Risk Assessment* 28, no. 7 (2011): 925–42.
9. Charles A. Menzie, Bonnie B. Potocki, and Joseph Santodonato, "Exposure to Carinogenic PAHs in the Environment," *Environmental Science and Technology* 26, no. 7 (1992): 1278–84.
10. USEPA, "Polycyclic Aromatic Hydrocarbons (PAHs) Factsheet," *JRC Technical Notes* 2008.
11. G. Cornelissen and O. Gustafsson, "Effects of Added PAHs and Precipitated Humic Acid Coatings on Phenanthrene Sorption to Environmental Black Carbon," *Environmental Pollution* 141, no. 3 (2006): 526–31.
12. W. A. Thorsen, W. G. Cope, and D. Shea, "Bioavailability of PAHs: Effects of Soot Carbon and PAH Source," *Environmental Science & Technology* 38, no. 7 (2004): 2029–37.
13. Nesta Bortey-Sam, Yoshinori Ikenaka, Shouta M. M. Nakayama, Osei Akoto, Yared Beyene Yohannes, Elvis Baidoo, Hazuki Mizukawa, and Mayumi Ishizuka, "Occurrence, Distribution, Sources and Toxic Potential of Polycyclic Aromatic Hydrocarbons (PAHs) in Surface Soils from the Kumasi Metropolis, Ghana," *The Science of the Total Environment* 496 (2014): 471–8.
14. European Commission. "*Polycyclic Aromatic Hydrocarbons – Occurrence in Foods, Dietary Exposure and Health Effects*," *Health and Consumer Protection Directorate-General* 29 (2002): A1–A194.
15. M. W. Ashraf and A. Salam, "Polycyclic Aromatic Hydrocarbons (PAHs) in Vegetables and Fruits Produced in Saudi Arabia," *Bulletin of Environmental Contamination and Toxicology* 88, no. 4 (2012): 543–7.
16. N. R. Bishnoi, U. Mehta, and G. G. Pandit, "Quantification of Polycyclic Aromatic Hydrocarbons in Fruits and Vegetables Using High Performance Liquid Chromatography," *Indian Journal of Chemical Technology* 13 (2006): 30–5.
17. C. M. Liao and K. C. Chiang, "Probabilistic Risk Assessment for Personal Exposure to Carcinogenic Polycyclic Aromatic Hydrocarbons in Taiwanese Temples," *Chemosphere* 63, no. 9 (2006): 1610–9.
18. S.-C. Chen and C.-M. Liao, "Health Risk Assessment on Human Exposed to Environmental Polycyclic Aromatic Hydrocarbons Pollution Sources," *The Science of the Total Environment* 366, no. 1 (2006): 112–23.
19. P. Boffetta, N. Jourenkova, and P. Gustavsson, "Cancer Risk from Occupational and Environmental Exposure to Polycyclic Aromatic Hydrocarbons," *Cancer Causes & Control* 8, no. 3 (1997): 444–72.
20. Nesta Bortey-Sam, Yoshinori Ikenaka, Osei Akoto, Shouta M. M. Nakayama, Yared Beyene Yohannes, Elvis Baidoo, Hazuki Mizukawa, and Mayumi Ishizuka, "Levels, Potential Sources and Human Health Risk of Polycyclic Aromatic Hydrocarbons (PAHs) in Particulate Matter (PM(10)) in Kumasi, Ghana," *Environmental Science and Pollution Research International* 22, no. 13 (2015): 9658–67.
21. H. I. Abdel-Shafy and M. S. M. Mansour, "A Review on Polycyclic Aromatic Hydrocarbons: Source, Environmental Impact, Effect on Human Health and Remediation," *Egyptian Journal of Petroleum* 25, no. 1 (2016): 107–23.
22. P. Mensah, D. Yeboah-Manu, K. Owusu-Darko, and A. Ablordey, "Street Foods in Accra, Ghana: How Safe Are They?," *Bulletin of the World Health Organization* 80, no. 7 (2002): 546–54.

23. Yandi Hu, Zhipeng Bai, Liwen Zhang, Xue Wang, Li Zhang, Qingchan Yu, and Tan Zhu, “Health Risk Assessment for Traffic Policemen Exposed to Polycyclic Aromatic Hydrocarbons (PAHs) in Tianjin, China,” *The Science of the Total Environment* 382, no. 2-3 (2007): 240–50.
24. Gerba, C. “Risk Assessment”, in *Environmental Biology*, edited by C. P. Gerba, R. Maier, and I. Pepper. (Elsevier, 2000), 557–71.
25. B. Acosta and I. Cipriani-Avila, “Determination of Polycyclic Aromatic Hydrocarbons (PAHs) in Aqueous Samples by HPLC-FLD with Salting-out Liquid-Liquid Extraction,” *Periodico Tche Quimica* 16 (2019): 95–110.
26. R. R. Johnson and P. J. Kuby, “Elementary statistics,” *Mathematics* (2011): 832.
27. O. Akoto, F. B. Eshun, G. Darko, and E. Adei, “Concentrations and Health Risk Assessments of Heavy Metals in Fish from the Fosu Lagoon,” *International Journal of Environmental Research* 8, (2014): 403–10.
28. USEPA, “Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons,” *Office of Research and Development* 600 (1993).
29. Ö. Kılıç Büyükkurt, E. Aykın Dinçer, İ. Burak Çam, C. Candal, and M. Erbaş, “The Influence of Cooking Methods and Some Marinades on Polycyclic Aromatic Hydrocarbon Formation in Beef Meat,” *Polycyclic Aromatic Compounds* 40, no. 2 (2020): 195–11.
30. S. Soghoian, C. Nyadedzor, B. Ed Nignpense, E. E. K. Clarke, and R. S. Hoffman, “Health Risks of Using Mothballs in Greater Accra, Ghana,” *Tropical Medicine and International Health* 17, no. 1 (2012): 135–8.
31. USEPA, “*Pyrene (CAS Number 129-00-0)*,” *USEPA Archived Document* (2010).
32. I. C. T. Nisbet and P. K. LaGoy, “Toxic Equivalency Factors (TEFs) for Polycyclic Aromatic Hydrocarbons (PAHs),” *Regulatory Toxicology and Pharmacology* 16, no. 3 (1992): 290–300.
33. D. H. Phillips, “Polycyclic Aromatic Hydrocarbons in the Diet,” *Mutation Research* 443, no. 1-2 (1999): 139–47.
34. USEPA, “*Bisphenol an Action Plan (CASRN 80-05-7)*,” (2011).
35. S. Wretling, A. Eriksson, G. A. Eskhult, and B. Larsson, “Polycyclic Aromatic Hydrocarbons (PAHs) in Swedish Smoked Meat and Fish,” *Journal of Food Composition and Analysis* 23, no. 3 (2010): 264–72.
36. I. W. Ofosu, S. Akomea-Frempong, E. De-Graft Johnson Owusu-Ansah, and G. Darko, “Exposure and Risk Assessment of Selected Chemical Hazards in Cabbage and Lettuce,” *Journal of Toxicology and Risk Assessment* 4, no. 1 (2018): 1–10.
37. A. Soceanu, S. Dobrinas, G. Stanciu, and V. Popescu, “Polycyclic Aromatic Hydrocarbons in Vegetables Grown in Urban and Rural Areas,” *Environmental Engineering and Management Journal* 13, no. 9 (2014): 2311–5.
38. O. M. Olabemiwo, “Polycyclic Aromatic Hydrocarbons in Smokes of Two Biomasses Used for Domestic Cooking in Nigeria,” *Chemistry and Materials Research* 6 (2014): 101–6.
39. Chris Whipple, De *Minimis* Risk*: Contemporary Issues in Risk Analysis* (Plenum Press, 1987).
40. A. Hoare, D. G. Regan, and D. P. Wilson, “Sampling and Sensitivity Analyses Tools (SaSAT) for Computational Modelling,” *Theoretical Biology and Medical Modelling* 5 (2008): 4.