Arsenic contamination in processed chicken and turkey products, available in Trinidad and Tobago supermarkets and the associated health risks to children[a]

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ABSTRACT

Arsenic is an established human carcinogen but can also lead to non-carcinogenic health effects such as diabetes and hypertension. This study analysed processed chicken and turkey products, both locally produced and imported, for total arsenic (tAs) and inorganic arsenic (iAs) concentrations using a combination of Soxhlet Extraction and Hydride Generation Atomic Absorption Spectroscopy (HG-AAS). Samples were purchased from supermarkets throughout the country and represented all the brands commercially available to the population at the time. The results revealed that four out of six locally produced samples (A, B, C, and E) exceeded the US FDA maximum limit of 500 µg/kg for tAs, with concentrations ranging from 269 to 1475 µg/kg. Additionally, for all six local products the estimated daily intake (EDI) for iAs exceeded the USEPA limit of 0.3 µg/kg BW/day across all three child age groups i.e. toddlers 1 - 3 years, pre-teens < 13 years, and teens 13 - 17 years, with values ranging from 0.60 to 10.30 µg/kg BW/day. The Hazard Quotient (HQ) for the six locally processed products was greater than 1, indicating potential health risks with HQ values ranging from 2.1 to 31.8. The Incremental Lifetime Cancer Risk (ILCR) assessment suggested low increased cancer risk, across all three age groups in Trinidad and Tobago based on consumption patterns and average body weight. Among imported samples, only one out of 11 products studied exceeded the US FDA regulatory limit for tAs, with a concentration of 534 µg/kg, and an estimated daily intake (EDI) for iAs of 1.3 to 6.2 µg/kg BW/day across all three age groups.

Introduction

Arsenic is a naturally occurring metalloid distributed in soil, water, and air. It exists in both inorganic and organic forms, with inorganic arsenic bonded to elements like sulfur, oxygen, and chlorine, while organic arsenic is bonded to hydrogen and carbon (Mandal and Suzuki 2002). Organic arsenic (oAs) is generally considered non-toxic, whereas inorganic arsenic (iAs), such as arsenite (Arsenic 3+ (As(III))) and arsenate (Arsenic 5+ (As(V))), are toxic and pose significant health risks (Sloth et al. 2003). Organic arsenic compounds, although less toxic than their inorganic counterparts, must still be metabolised into As(V) (Singh et al. 2011). Inorganic arsenic, classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC), includes As(III) and As(V), both of which are linked to serious health effects (International Agency for Research on Cancer 2018). Of the two, As(III) is generally more toxic, as it binds to sulfur-containing proteins, disrupting enzyme function and causing greater cellular damage (Valko et al. 2005).

The carcinogenicity of arsenic is mainly associated with As (III), which is more potent due to its ability to inhibit enzymes such as pyruvate dehydrogenase. As(V) must first be reduced to As(III) to enter cells and exhibit toxicity (Valko et al. 2005). Studies have confirmed that As(III) has a lower lethal dose (LD50) compared to As(V), making it more toxic to humans and animals (Hughes et al. 2011).

Chronic exposure to inorganic arsenic is associated with a variety of non-carcinogenic effects, such as diabetes and hypertension

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(Hong et al. 2014). Arsenic exposure has been linked to the development of Type 2 diabetes, possibly through mechanisms like oxidative stress and inflammation (Wang et al. 2007). In Trinidad and Tobago, increasing rates of childhood obesity and Type 2 diabetes could be linked to arsenic exposure through dietary sources (Batson et al. 2013; Ghouralal 2019b).

Arsenic-based compounds, such as Roxarsone, have been used in poultry feed to prevent parasitic diseases and promote growth. Once ingested, Roxarsone can degrade into toxic inorganic arsenic (Arsenic 3+ (As(III)) and Arsenic 5+ (As(V))) under anaerobic conditions, leading to arsenic accumulation in chicken tissues and litter (Nachman et al. 2013; Stolz et al. 2006). Studies suggest that these inorganic forms can be re-ingested by chickens, posing a risk to human consumers (Garbarino et al. 2003).

Arsenic can undergo various chemical transformations in the environment, such as oxidation, reduction, and methylation, which affect its mobility and toxicity (Sharma and Sohn 2009). In anaerobic environments, organic arsenic like Roxarsone can be converted to its more toxic inorganic forms, potentially contaminating groundwater and crops (Rehman et al. 2021).

Arsenic contamination comes from both natural and anthropogenic sources. Natural emissions include volcanic activity and weathering of minerals, while human activities like coal combustion and pesticide use significantly contribute to atmospheric and soil arsenic levels (Pacyna and Pacyna 2001). Global anthropogenic sources contribute over 23,600 t of arsenic annually (Arya and Basu 2016).

In soil, arsenic can persist for long periods due to its resistance to degradation. It can accumulate in plants, including crops, and enter the food chain, posing health risks to humans and animals (Rehman et al. 2021). Soil arsenic concentrations are influenced by local geology and industrial activity, with contaminated sites often showing significantly higher levels. Soil moisture also plays a crucial role, influencing both the redox conditions and the potential for arsenic leaching. Arsenic contamination in soil can impact poultry in several ways, depending on the arsenic levels, its forms, and how it interacts with the environment (Chen et al. 2020). Human exposure to arsenic from soil, water, and air is a growing concern, with contaminated water and food being the primary sources of dietary intake. Arsenic's behaviour in soil is highly dependent on the soil oxygen levels and moisture content (Chen et al. 2020). Anaerobic conditions generally increase arsenic mobility due to the formation of soluble arsenite, while aerobic conditions stabilise arsenic in less mobile forms like arsenate (Xu et al. 2016). The movement of arsenic through soil, water, and air is complex, involving various chemical transformations. In soil and water, arsenic can be oxidised, reduced, methylated, or volatilised, depending on environmental conditions. These processes influence its bioavailability and toxicity (Chen et al. 2020).

In water, arsenic primarily exists as arsenite (As (III)) or arsenate (As(V)), depending on the oxidation state. Natural sources of arsenic in water include the dissolution of arsenic-bearing minerals, while anthropogenic activities like mining and agricultural runoff contribute to elevated arsenic levels in groundwater and surface water (Flora 2015). Arsenic-contaminated water, particularly groundwater, poses significant health risks, as it is a primary source of drinking water for millions globally.

In the air, arsenic is emitted primarily through industrial processes such as metal smelting and burning fossil fuels. Atmospheric arsenic is transported over large distances, eventually settling onto land and water, where it can further contaminate the environment (Pacyna and Pacyna 2001). Airborne arsenic is typically present as particulate matter and is inhaled by humans, contributing to respiratory diseases and long-term health effects.

Organic arsenicals, such as those used in agriculture, can degrade into more toxic inorganic forms, increasing environmental

risks (Sánchez-Rodas et al. 2006). In particular, Roxarsone, used in poultry feed, can transform into arsenite (As(III)) and arsenate (As(V)) under anaerobic conditions, posing a risk when introduced into the environment through poultry litter (Stolz et al. 2006).

International regulatory agencies have established various guidelines for arsenic exposure. The US FDA set a maximum limit of 500 µg/kg for total arsenic (tAs) in chicken tissue (Wallinga 2006), while the European Union and other regions have banned the use of arsenic-based feed additives (Baynes et al. 2016). However, these standards often differ across regions and may not adequately consider modern consumption patterns and arsenic toxicity (WHO 2011). In 1991, the United States Environmental Protection Agency (USEPA) established a reference dose (RfD) of 0.3 µg/iAs kg/BW per day (US Environmental Protection Agency 1991). The RfD is also called the toxicity value for non-cancerous effects from ingestion exposure. This regulation has been adopted worldwide and is frequently used today, as it is unlikely to cause the manifestation of adverse health effects. This study examined the effects of arsenic in processed food products consumed by children aged 1 – 17 in Trinidad and Tobago, the three age groups studied were toddlers 1 - 3 years, pre-teens < 13 years, and teens 13 - 17 years.

Methodology

Sampling plan

Seventeen processed chicken and turkey products comprising hotdogs, bologna, and salami were sampled across major grocery stores in Trinidad and Tobago during the period November 2023 to January 2024. These samples were available in all of the major grocery stores visited during this period. These samples represent all the hotdog, bologna and salami brands commercially available to the population at the time. They included six locally produced commercial brands and 11 imported commercial brands. Batch numbers were recorded to ensure that the products represented batch consistency and homogeneity. As these processed products are manufactured on the same production line, these items were sampled three times from the same grocery store by purchasing three separate batches of each product, and the average concentrations of the three batches/samples were reported.

Sample preparation

Samples were stored in a freezer at - 20 °C before analysis. They were then thawed to room temperature, then homogenised using a food processor.

Analysis of total arsenic

Approximately 0.5 g of each homogenized sample was weighed directly into boiling tubes. Each boiling tube was first placed in a beaker on the analytical balance, the balance was zeroed, and samples were added until approximately 0.5 g was achieved. Once weighed, the samples were pre-digested with 10 mL of ACS-grade concentrated nitric acid at room temperature for 24 h. Samples were further digested at 130 °C for 90 m on a heating block. The samples were allowed to cool to room temperature and filtered through a Whatman no. 541 filter paper into 50 mL volumetric flasks and diluted to 50 mL with deionised water (Mohammed et al. 2017); 5 mL of the diluted digests were extracted into a 25 mL volumetric flask and treated with 5 mL of 5 M HCl and 5 mL of 1% KI, then diluted to 25 mL; samples were allowed to stand at room temperature for 50 min. This process reduced As (V) to As (III), which formed the stable hydride needed for analysis. In hydride generation, sodium borohydride ($NaBH_4$) and hydrochloric acid (HCl) are usually employed to convert As species in aqueous solutions into volatile hydrides (Mohammed et al. 2017).

Analysis of inorganic arsenic

Following the procedure of Yuan et al. (2005), and as optimised previously, 100 mL of methanol and 100 mL of water were added to a 250 mL round bottom flask with 5 - 8 boiling chips; 10 g of the homogenised sample was added to a cellulose thimble and extracted with the methanol/water solvent for 6 hours using soxhlet extraction The solvent was removed from the extract with the use of a rotary evaporator, leaving only the inorganic extract and minimal solvent. The inorganic extract was dried in a desiccator overnight and solubilised with 50 mL of 1 M nitric acid at room temperature. This extract was then quantitatively transferred to a 50 mL volumetric flask and the volume made up to 50 mL.

To 5 mL of the diluted extract, 0.25 g KI, 0.5 g ascorbic acid, and 2.5 mL 1.2 M HCl were added and left standing for 50 m to facilitate complete reduction of As (V) to As (III). The volume was then made up to 25 mL with deionised water (Dos Santos et al. 2017; Mindak et al. 2014). Thus, the final concentrations of KI and ascorbic acid were 1% (m/v) and 0.2 % (m/v), respectively. This process was necessary as only As (III) forms the stable hydride needed for HG-AAS (hydride generation atomic adsorption spectrophotometry) analysis. Samples were allowed to stand at room temperature for 50 m; this reduced As (V) to As (III), which formed the stable hydride needed for analysis. In hydride generation, sodium borohydride ($NaBH_4$) and hydrochloric acid (HCl) are usually employed to convert As species in aqueous solutions into volatile hydrides (Mohammed et al. 2017).

Instrumentation

Analysis of total arsenic was performed using a Varian atomic absorption spectrophotometer (SpectrAA880), equipped with the VGA77 hydride generation accessory and the quartz flow cell. Arsenic high-intensity UltraAA coded single element hollow cathode lamps, with Agilent UltraAA boosted lamp supply were also used (Mohammed et al. 2017).

Quality assurance

All glassware were washed with a non-ionic detergent, rinsed with distilled water, and placed in an acid bath for 24 h or until ready for use. The glassware was then removed from the acid bath, rinsed twice with deionised water, and allowed to air dry overnight. A series of three sample blanks were analysed with each batch of samples, blanks were matrix-matched.

A DORM-5 fish protein-certified reference material (CRM), which has a certified total arsenic value of 13300 ± 700µg/kg, was used to validate the results. A 0.5 g sample of the certified reference material was analysed in triplicate using the identical method for the samples. A recovery of 98.99% for arsenic was obtained, and this was considered acceptable.

A calibration curve was generated from serial dilutions of the stock arsenic atomic absorption standard at a concentration of 1000 µg/kg. Working standard concentrations were 10, 30, and 50 µg/kg and were matrix-matched to ensure consistency.

The method detection limit (MDL) was calculated using the formula MDL = (3 σ/m) x 10, where σ is the standard deviation of a known low concentration, i.e., 0.05 µg/kg solution where (n = 10) and m is the slope of the calibration curve (Miller and Miller 2018).

Health risk assessment

Health risk assessment is a process in which information is analysed to determine if a hazard might cause adverse effects to humans following exposure under defined conditions to a risk source (Page 2019). Risk assessments are characterised into two categories, non-cancerous and cancer-related. The most common measures are the Hazard

Quotient (HQ) which is used to assess the non-cancerous effects and the Incremental Lifetime Cancer Risk (ILCR) for cancer effects (Saipan and Ruangwises 2009).

Hazard Quotient (HQ)

The Hazard Quotient (HQ) is the most appropriate method for assessing the non-carcinogenic effect of arsenic-contaminated chicken on the population based on the frequency of use in literature. It is used by the USEPA to assess the health risks of air toxins and carcinogens, if the value of HQ is less than or equal to 1.0, the risk is considered negligible to low, and no unacceptable effects will occur in the exposed population. An HQ greater than 1.0, is considered indicative of an unacceptable hazard for various non-cancerous effects such as hypertension, diabetes, and infertility (Singh and Ghosh 2012).

$$HQ = \frac{C_{ias} * IR}{BW * RfD}$$

where:

C_{iAs} = concentration of inorganic arsenic in the sample (mg/kg)

IR = daily ingestion rate of sample specific to Trinidad and Tobago (0.15kg/day) (Evans and Linden 2015;US Environment Protection Agency 1989)

BW: body weight of individual

RfD: $3x10^{-4}$ mg/kg for arsenic (US Environment Protection Agency 1989)

USEPA daily estimated intake / daily ingestion rate

In Trinidad and Tobago, current consumption data exists for meats such as chicken, pork, beef, and goat; however, no data is available for processed products like salami, bologna, and hotdogs. To estimate the processed poultry intake for children aged 1-17, an assumption was made based on typical consumption patterns. For instance, a child might consume two 57 g hot dogs and one 28 g slice of bologna three times a week, either for breakfast or for lunch. This results in a daily intake of 142 g, a weekly intake of 426 g, and an annual intake of 22.15 kg, based on consumption three times a week. Over the course of 156 days in a year, this equates to a daily ingestion rate of approximately 0.14 kg/day.

The inorganic arsenic (iAs) intake was calculated as follows:

iAs = amount of chicken ingested (kg/day) * iAs in chicken (µg/kg)/body weight (kg)

Body weight plays a critical role in these calculations, with the average body weight for children in different age groups being 13 kg for toddlers, 34.75 kg for pre-teens, and 63 kg for teens (Grummer-Strawn et al. 2009). The reference dose (RfD) for inorganic arsenic, as specified by the USEPA, is $3x10^{-4}$ mg/kg of body weight per day (US Environment Protection Agency 1989).

Incremental Lifetime Cancer Risk (ILCR)

Incremental Lifetime Cancer Risk (ILCR) is defined by the USEPA as the incremental probability that an individual will develop cancer over their lifetime due to exposure to a carcinogen (Wiltse and Dellarco 1996). ILCR is expressed as a unit less probability, representing the risk of developing cancer in a population, and is influenced by exposure frequency and contact duration with a chemical agent (US Environment Protection Agency 2012).

ILCR values between 1×10^{-6} and 1×10^{-4} suggest that cancer rates are acceptable, meaning 1 additional cancer case in a population of 10,000 to 1,000,000 people (World Health Organization 2001). A ILCR value less than 1×10^{-6} is considered negligible, while a value exceeding 1×10^{-4} indicates a significant risk (US Environment Protection Agency 2012). ILCR values are typically

presented with one significant digit for clarity, where, for example, an ILCR of 1.3×10^{-6} is rounded to 1×10^{-6}, representing an acceptable risk, whereas an ILCR of 1.5×10^{-3} is rounded to 2×10^{-3}, indicating unacceptable risk (World Health Organization 2010). Table 1 outlines how different ILCR values relate to cancer risk levels.

Table 1: Quantifying and interpreting Incremental Lifetime Cancer Risk (ILCR) levels

ILCR value	Risk expression	Number at risk	Risk level
$1x10^{-1}$	$1.0E^{-1}$	One in ten	Very high increased risk
$1x10^{-2}$	$1.0E^{-2}$	One in a hundred	High increased risk
$1x10^{-3}$	$1.0E^{-3}$	One in a thousand	Moderate increased risk
$1x10^{-4}$	$1.0E^{-4}$	One in ten thousand	Low increased risk
$1x10^{-5}$	$1.0E^{-5}$	One in a hundred thousand	Very low increased risk
$1x10^{-6}$	$1.0E^{-6}$	One in a million	Extremely low increased risk
$1x10^{-9}$	$1.0E^{-9}$	One in a billion	No risk

ILCR calculation for arsenic

The ILCR for a specific heavy metal, such as inorganic arsenic (iAs), measures the incremental increase in cancer cases in an exposed population compared to an unexposed one (US Environmental Protection Agency 2011). ILCR is calculated by multiplying the lifetime average daily dose (LADD) of iAs (mg/kg/day) by its corresponding cancer slope factor (CSF) (mg/kg BW/day) (Liu et al. 2015). The CSF estimates the probability of developing cancer from lifetime exposure to 1.0 mg/kg/day of a carcinogen (typically calculated over 70 years) (US Environmental Protection Agency 2011).

$$LADD = (CiAs \times IR \times ED \times EF) / (BW * 365 \text{ days/year} * LE)$$

Where:

CiAs = concentration of inorganic arsenic in poultry (mg/kg)
IR = daily ingestion rate (0.15 kg/day)
ED = exposure duration (years) (World Health Organization 2019).
EF = exposure frequency (144 days/year)
BW = body weight (kg), depending on age group (Grummer-Strawn et al. 2009):
- Toddler 1 – 3 years (13 kg)
- Pre-teen < 13 years (34.75 kg)
- Teen 13 – 17 years (63 kg)

LE = life expectancy (73.67 years for adults in Trinidad and Tobago) (World Health Organization 2019).

The ILCR is then calculated by:

$$ILCR = LADD \times CSF_{iAs}$$

Results

Tables 2, 3, and 4 show that four out of six locally produced products (A, B, C, and E) of Trinidad and Tobago (TT) origin analysed exceeded the US FDA's regulatory limit for total arsenic of 500 µg/kg (542 µg/kg - 1475 µg/kg) and all six locally produced products (A - F) exceeded the USEPA's regulatory limit (EDI) of 0.3 µg/kg for inorganic arsenic (0.6 - 10.3 µg/kg/day/BW). These values far exceed the regulatory limits and pose significant health risks to consumers.

All locally produced products had an HQ that greatly exceeded 1, ranging from 10.4 – 34.3 for toddlers (1 - 3 year-olds, 13kg), 3.9 - 12.8 for pre-teens (< 13 year-olds, 34.75kg), and 2.1 – 7.1 for teens (13 - 17 year-olds, 63kg). These results strongly suggest that consuming these products will place children in all age groups at severe risk of developing non-cancerous effects such as diabetes, hypertension, neurological impairment, and infertility, with the youngest age group (1 - 3 years) being more profoundly affected. The ILCR for locally produced processed products were found to range from $2.7*10^{-4}$ - $8.9*10^{-4}$ (toddler), $1.0*10^{-4}$- $3.3 * 10^{-4}$ (pre-teen), and $1.3*10^{-4}$ - $4.3*10^{-4}$ (teen).

Table 2: Total and inorganic arsenic concentrations in processed chicken and turkey products and the risk impacted for toddlers (1 - 3 years, 13 kg)

Product	**Origin**	**Inorganic As µg/kg**	**EDI**	**Total As µg/kg**	**HQ**	**ILCR**	**Meaning of ILCR**
A	TT	826	9.5	1463	31.8	0.00082	Low increased risk
B	TT	893	10.3	1475	34.3	0.00089	Low increased risk
C	TT	620	7.2	926	23.8	0.00062	Low increased risk
D	TT	269	3.1	542	10.4	0.00027	Low increased risk
E	TT	525	6.1	813	20.2	0.00052	Low increased risk
F	TT	335	3.9	683	12.9	0.00033	Low increased risk
G	Venezuela	534	6.2	825	20.5	0.00053	Low increased risk
H	US	0	0.0	0	0.0	0.00000	No risk
I	US	0	0.0	0	0.0	0.00000	No risk
J	US	0	0.0	0	0.0	0.00000	No risk
K	US	0	0.0	0	0.0	0.00000	No risk
L	US	0	0.0	6.6	0.0	0.00000	No risk
M	US	0	0.0	6.5	0.0	0.00000	No risk
N	US	0	0.0	6.7	0.0	0.00000	No risk
O	US	0	0.0	6.5	0.0	0.00000	No risk
P	US	0	0.0	6.5	0.0	0.00000	No risk
Q	US	0	0.0	0	0.0	0.00000	No risk

As: arsenic, EDI: estimated daily intake, HQ: Hazard Quotient, ILCR: Incremental Lifetime Cancer Risk, TT: Trinidad and Tobago, US: United States. Method detection limits: tAs = 11.58 µg/kg; MDL iAs = 8.37 µg/kg ± SD within 5% of the values reported.

Table 3: Total and inorganic arsenic concentrations in processed chicken and turkey products and the risk impacted for pre-teens (< 13 years, 34.75 kg)

Product	**Origin**	**Inorganic As µg/kg**	**EDI**	**Total As µg/kg**	**HQ**	**ILCR**	**Meaning of ILCR**
A	TT	826	3.6	1463	11.9	0.00031	Low increased risk
B	TT	893	3.9	1475	12.8	0.00033	Low increased risk
C	TT	620	2.7	926	8.9	0.00023	Low increased risk
D	TT	269	1.2	542	3.9	0.00010	Low increased risk
E	TT	525	2.3	813	7.6	0.00020	Low increased risk
F	TT	335	1.5	683	4.8	0.00012	Low increased risk
G	Venezuela	534	2.3	825	7.7	0.00020	Low increased risk
H	US	0	0.0	0	0.0	0.00000	No risk
I	US	0	0.0	0	0.0	0.00000	No risk
J	US	0	0.0	0	0.0	0.00000	No risk
K	US	0	0.0	0	0.0	0.00000	No risk
L	US	0	0.0	6.6	0.0	0.00000	No risk
M	US	0	0.0	6.5	0.0	0.00000	No risk
N	US	0	0.0	6.7	0.0	0.00000	No risk
O	US	0	0.0	6.5	0.0	0.00000	No risk
P	US	0	0.0	6.5	0.0	0.00000	No risk
Q	US	0	0.0	0	0.0	0.00000	No risk

As: arsenic, EDI: estimated daily intake, HQ: Hazard Quotient, ILCR: Incremental Lifetime Cancer Risk, TT: Trinidad and Tobago, US: United States. Method detection limits: tAs = 11.58 µg/kg; MDL iAs = 8.37 µg/kg ± SD within 5% of the values reported.

Table 4: Total and inorganic arsenic concentrations in processed chicken and turkey products and the risk impacted for teens (13 - 17 years, 63 kg)

Product	Origin	Inorganic As µg/kg	EDI	Total As µg/kg	HQ	ILCR	Meaning of ILCR
A	TT	826	2.0	1463	6.6	0.00040	Low increased risk
B	TT	893	2.1	1475	7.1	0.00043	Low increased risk
C	TT	620	1.5	926	4.9	0.00030	Low increased risk
D	TT	269	0.6	542	2.1	0.00013	Low increased risk
E	TT	525	1.3	813	4.2	0.00025	Low increased risk
F	TT	335	0.8	683	2.7	0.00016	Low increased risk
G	Venezuela	534	1.3	825	4.2	0.00026	Low increased risk
H	US	0	0.0	0	0.0	0.00000	No risk
I	US	0	0.0	0	0.0	0.00000	No risk
J	US	0	0.0	0	0.0	0.00000	No risk
K	US	0	0.0	0	0.0	0.00000	No risk
L	US	0	0.0	6.6	0.0	0.00000	No risk
M	US	0	0.0	6.5	0.0	0.00000	No risk
N	US	0	0.0	6.7	0.0	0.00000	No risk
O	US	0	0.0	6.5	0.0	0.00000	No risk
P	US	0	0.0	6.5	0.0	0.00000	No risk
Q	US	0	0.0	0	0.0	0.00000	No risk

As: arsenic, EDI: estimated daily intake, HQ: Hazard Quotient, ILCR: Incremental Lifetime Cancer Risk, TT: Trinidad and Tobago, US: United States. Method detection limits: tAs = 11.58 µg/kg; MDL iAs = 8.37 µg/kg ± SD within 5% of the values reported.

All imported samples analysed fell well under the US FDA's regulatory limit for total arsenic i.e., 500 µg/kg, and USEPA's regulatory limit of 0.3 µg/kg for inorganic arsenic, except for product G of Venezuelan origin. Venezuela, like Trinidad and Tobago, does not have regulations governing arsenical use in the poultry industry, because of this, the product is not for sale in the US but is exported to other countries that do not have governing regulations for arsenical use (Khan et al. 2020).

All imported products were found to have an HQ of zero, except for product G which had HQs of 20.5 (toddler), 7.7 (pre-teen), and 4.2 (teen). Product G had ILCRs of $5.3*10^{-4}$ (toddler), $2.0*10^{-4}$ (pre-teen), and $2.6*10^{-4}$ (teen). This indicates a low increased risk of developing cancer, hypertension, and diabetes in the children who consume these products three times a week. Product G contained more arsenic than several of the Trinidad and Tobago products. If product G, is considered to be

typical of other products originating from Latin America, then similar health issues as a result of arsenic exposure will be experienced in Latin America.

Discussion

The cancer risk is accepted in ranges $1*10^{-6}$ to $1*10^{-4}$ which indicates a negligible carcinogenic health risk. Most of the local products did not meet the criteria, and the ranges were found to be $2.7*10^{-4}$ - $8.9*10^{-4}$ for toddlers (1 - 3 years old), $1.0*10^{-4}$ - $3.3*10^{-4}$ for pre-teens (< 13 years) and $1.3*10^{-4}$ - $4.3*10^{-4}$ for teens (13 - 17 years). D was the only product that met the criteria but only for the pre-teen group, with an ILCR of $1.0*10^{-4}$.

Products manufactured in the United States, are subjected to USFDA and USEPA regulations, and since arsenic-based additives are banned for commercial use within the United States, the zero or very low levels were expected, and adhered to regulatory limits.

The American Institute for Cancer Research AICR/WCRF defines processed meat as meat preserved by smoking, curing or salting, or the addition of chemical preservatives. Meat is typically listed as the first ingredient, followed by water, on the label of processed meat products (Bender 2015).

A study conducted by Prayson et al. 2008) analysed eight varied products of hot dogs to determine what each hot dog consisted of, while "meat" i.e., skeletal muscle was listed as the first ingredient in all eight products, the actual skeletal muscle in each product ranged from 2.9 – 21.2% by volume, while water accounted for 44 – 69% of the weight of each hot dog. Though it is predominantly water and a small amount of meat, it was shown to collectively be a variety of tissues; including bone, collagen, blood vessels, plant material, peripheral nerve, fat, cartilage, and skin. In countries outside of North America, it is popular for hotdogs, lunch meat, and other processed meats, to contain organ material, such as kidney, liver, and heart (Ahmad and Badpa 2014).

Two decades of data show that processed meat such as hotdogs, bologna, sausages, and salami is an immensely popular breakfast food worldwide and these are particularly popular among age groups 2 – 19 years. Parents often feed their children hot and cold processed meats, because they are inexpensive and easy to prepare, and are a common school lunch. (Saksena et al. 2018; Wang et al. 2021).

The World Health Organization's Agency for Research on Cancer (WHO/IARC), classifies processed meat such as hot dogs, sausages, bacon, and ham, as group 1 carcinogens (Harvard T.H. Chan School of Public Health 2015). This puts processed foods in the same category as arsenic, alcohol, and tobacco. The WHO states that consuming 50 g of processed meat a day, which is equivalent to one standard-sized hot dog, can increase the risk of developing bowel cancer by 18% (Harvard T.H. Chan School of Public Health 2015).

Toxicity is complex and can vary depending on characteristics such as age, weight, sex, and frequency of exposure, in the case of this research frequency of exposure was assumed to be 3 days a week i.e 156 days a year. The average child weighs less than the average adult, and this puts them at higher risk of arsenic poisoning, according to the USEPA daily reference dose. The average 3 year-old weighs 13 kg (Buchan et al. 2007) because of this, the daily intake of arsenic would increase by 22% for the same serving when compared to an adult. In 1999, in Trinidad and Tobago it was found that 2.4 % of children 5 - 18 were diagnosed with obesity, in 2009 this rose to 13% and in 2017 to 55 %, this is a risk factor for developing diabetes, and it is projected that juvenile diabetes will be on the rise because of this (Ghouralal 2019a). In 2009 among school children in Trinidad and Tobago 10.4/100,000 were prevalent with Type 2 diabetes, and 7.5/100,000 with impaired glucose intolerance (Batson et al. 2013).

Latin America and the Caribbean countries have the fastest-growing prevalence of Type 2 diabetes in the world (Gallardo-

Rincón et al. 2021). In the Caribbean, there is a prevalence of 15/100,000 adolescents with Type 2 diabetes, whereas in the USA there is a prevalence of 13.8/100,000 adolescents with Type 2 diabetes (Perng et al. 2023), in the United Kingdom the prevalence is 1.35/100,000 (Candler et al. 2018). This shows childhood Type 2 diabetes in the Caribbean is higher than in the USA and UK.

The fertility rate to replace a population is 2.1, each mother should have 2.1 babies (Gietel-Basten and Scherbov 2020). In Trinidad and Tobago, currently, the fertility rate is 1.67, the population is not being renewed; the rate of births is now at about 17,000 yearly and is steadily declining (Hamilton-Davis 2022). The average fertility in the wider Caribbean is 1.85, and globally it stands at 2.4 (Central Intelligence Agency 2020). Many factors contribute to the declining population of the Caribbean including lower than average fertility rates, migration, psychological stress, maternal age, and mortality rates (O'Gam and Robey 2014). The effects of arsenic cannot be ruled out (Lei et al. 2015) as the Caribbean has no regulations concerning arsenic use, and it has one of the highest global meat consumptions (HeligiLibrary 2018).

In the United States, there are 17.4 cancer diagnoses per 100,000 children ages younger than 15 years (Barr et al. 2016). Cancer is the second most common cause of childhood deaths in Latin America and the Caribbean; over 29,000 children and adolescents (0 - 19 years) are diagnosed with cancer annually with 10,000 of them succumbing to the disease (Da Silva 2023).

Although several factors can contribute to childhood cancers including genetic pre-disposition, genetic-environmental interaction, exposures to certain viruses, pesticides, and tobacco smoking (Bunin 2004) the impact of long-term arsenic exposure throughout the child's life and well into adulthood cannot be ruled out (Palma-Lara et al. 2020).

In 2018, it was revealed that Trinidad and Tobago ranks second for the highest consumption of chicken in the Caribbean on a per capita basis and fourth in the world, among 180 countries assessed (HeligiLibrary 2018). This can very well be linked to the non-communicable diseases and cancer rates. Arsenic is a real contributor to population incidents of diabetes, hypertension, poor fertility, and various cancers (Jomova et al. 2011; Lei et al. 2015). Nutrition-related chronic non-communicable diseases (NCDs) account for 60% of all deaths with cancers being ranked second globally (Razzaghi et al. 2019). The prevalence of diabetes in Trinidad and Tobago is 13%, while 26% of the population is affected by hypertension. Numerous animal and human studies have shown that arsenic exposures induce infertility by altering the levels of follicle-stimulating hormone and promoting oxidative stress (Zargari et al. 2022).

The impact of NCDs may result in increased poverty and lower quality of life. NCDs can have negative impacts on the economic growth of developing Caribbean economies when finances have to be diverted to treat these diseases. They also causes fatigue and are tremendous burdens on public health care systems.

NCDs place a significant economic burden on the nation of Trinidad and Tobago. The estimated annual economic cost incurred from treating hypertensive patients is TT$3.2 billion and from diabetes is TT$3.5 billion (Doon et al. 2023).The annual cost incurred for cancer treatment in Trinidad and Tobago, is TT$13,287,000 across different types of cancers (PAHO 2013). TT$6.80 is approximately US$1.00.

Regulations can correct this situation, and possibly reduce the burden on the health care system. If regulations are enforced, this means Trinidad and Tobago will cease importing poultry products from countries with no regulations, the poultry reared locally will have low levels of arsenic since veterinarians and poultry feed companies will not be allowed to use it as a supplement, this will translate to lower levels in processed products.

Conclusion

The results indicate that four out of six locally produced samples exceeded the US FDA maximum limit of 500 µg/kg for total arsenic (tAs), with concentrations ranging from 269 to 1475 µg/kg. For all six local products the estimated daily intake (EDI) of inorganic arsenic (iAs) surpassed the USEPA safety threshold of 0.3 µg/kg BW/day across all child age groups—toddlers (1 - 3 years), pre-teens (< 13 years), and teens (13 - 17 years)—with values between 0.64 and 10.30 µg/kg BW/day. The Hazard Quotient (HQ) for all six locally processed products was greater than 1, suggesting potential health risks, with HQ values ranging from 2.1 to 31.8. The Incremental Lifetime Cancer Risk (ILCR) analysis indicated a low increased risk of cancer across all child age groups in Trinidad and Tobago based on consumption patterns and average body weight.

For imported samples, only the Venezuelan product, G exceeded the US FDA limit for tAs, with a concentration of 534 µg/kg; for this product the EDI for iAs exceeded the safe threshold across all age groups, with HQ values also above 1; The ILCR was calculated to be 6.2 µg/kg, implying a low increased cancer risk, where approximately 1 in 10,000 children in each age group could be at risk of developing cancer. The remaining ten imported products, all from the US, were within safe limits according to all risk assessment models and regulatory standards.

The regulators, producers, and the population of Trinidad and Tobago need to be educated on the long-term effects and dangers of arsenic in meats and poultry, in order to drive the establishment of appropriate regulations to protect the consumer from arsenic exposure.

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References

Ahmad, S., and A.G. Badpa. 2014. "Meat Products and Byproducts for Value Addition. In: *Food Processing: Strategies for Quality Assessment*, edited by A. Malik, Z. Erginkaya, S. Ahmad and H. Erten, 125–153. Springer, New York.

Arya, A., and S.K. Basu. 2016. *Anthropogenic Pollution: Causes and Concern.* The Readers Paradise.

Barr, R.D., L.A.G. Ries, D.R. Lewis, L.C. Harlan, T.H.M. Keegan, B.H. Pollock, and W.A. Bleyer. 2016. "Incidence and Incidence Trends of the Most Frequent Ancers in Adolescent and Young Adult Americans, Including "Nonmalignant/ Noninvasive" Tumors." *Cancer* **122 (7)**: 1000–8. https://doi.org/10.1002/cncr. 29867.

Batson, Y.A., S. Teelucksingh, R. Maharaj, V. Singh, S. Balkaran, and B. Cockburn. 2013. "Screening for Diabetes in Schoolchildren in Trinidad, West Indies." *Paediatrics and International Child Health* **33 (1)**: 37–41. https://doi.org/10.1179/2046905512y.0000 000032.

Baynes, R.E., K. Dedonder, L. Kissell, D. Mzyk, T. Marmulak, G. Smith, L. Tell, R. Gehring, J. Davis, and J.E. Riviere. 2016. "Health Concerns and Management of Select Veterinary Drug Residues." *Food and Chemical Toxicology* **88**: 112–122.

Bender, A. 2015. "Bacon, Hot Dogs and Lunch Meat – Is It Processed Meat?". American Institute for Cancer Research (AICR), Last Modified October 26, 2015, accessed June 30. https://www.aicr.org/resources/blog/bacon-hot-dogs-and-lunch-meat-is-it-processed-meat/.

Buchan, I.E., P.E. Bundred, D.J. Kitchiner, and T.J. Cole. 2007. "Body Mass Index has Risen More Steeply in Tall Than in Short 3-year Olds: Serial Cross-Sectional Surveys 1988–2003." *International Journal of Obesity* **31 (1)**: 23–29.

Bunin, G.R. 2004. "Nongenetic Causes of Childhood Cancers: Evidence from International Variation, Time Trends, and Risk Factor Studies." *Toxicology and Applied Pharmacology* **199 (2)**: 91–103.

Candler, T.P., O. Mahmoud, R.M. Lynn, A.A. Majbar, T.G. Barrett, and J.P.H. Shield. 2018. "Continuing Rise of Type 2 Diabetes Incidence in Children and Young People in the UK." *Diabetic Medicine* **35 (6)**: 737–744. https://doi.org/10.1111/dme.13609.

Central Intelligence Agency. 2020. *The Cia World Factbook 2020–2021*. New York: Simon and Schuster.

Chen, X., X.-C. Zeng, Y.K. Kawa, W. Wu, X. Zhu, Z. Ullah, and Y. Wang. 2020. "Microbial Reactions and Environmental Factors Affecting the Dissolution and Release of Arsenic in the severely Contaminated Soils Under Anaerobic or Aerobic Conditions." *Ecotoxicology and Environmental Safety* **189**: 109946.

Da Silva, J.B., Jr. 2023. "A Call to Action: Strengthening Services to Improve Childhood Cancer Survival in Latin America and the Caribbean." *Revista Panamericana de Salud Pública* **47**: e157. https://doi.org/10.26633/rpsp.2023.157.

Doon, R., T. Malcolm, Y. Lewis, L. Holder, L. Gulston, A. Hamid, A. Best, R. Mitchell, and P. Lalla. 2023. "Improving Cardiovascular Health with the Patient-Centered, Integrated Primary Care Hearts Model in Trinidad and Tobago." *Revista Panamericana de Salud Pública* **46**: e169.

Dos Santos, G.M., D. Pozebon, C. Cerveira, and D.P. de Moraes. 2017. "Inorganic Arsenic Speciation in Rice Products Using Selective Hydride Generation and Atomic Absorption Spectrometry (Aas)." *Microchemical Journal* **133**: 265–271.

Evans, T., and J. Linden. 2015. Global Poultry Trends 2014: "Growth in Chicken Consumption in Americas Slows." Accessed August 2 2020. https://www.thepoultrysite.com/articles/global-poultry-trends-2014-growth-in-chicken-consumption-in-americas-slows

Flora, S.J.S. 2015. "Arsenic: Chemistry, Occurrence, and Exposure." In *Handbook of Arsenic Toxicology*, 1–49. Cambridge, Massachusetts, USA: Academic Press.

Gallardo-Rincón, H., A. Cantoral, A. Arrieta, C. Espinal, M.H. Magnus, C. Palacios, and R. Tapia-Conyer. 2021. "Review: Type 2 Diabetes in Latin America and the Caribbean: Regional and Country Comparison on Prevalence, Trends, Costs and Expanded Prevention." *Primary Care Diabetes* **15 (2)**: 352–359.

Garbarino, J.R., A.J. Bednar, D. Rutherford, R. Beyer, and R. Wershaw. 2003. "Environmental Fate of Roxarsone in Poultry Litter. I. Degradation of Roxarsone During Composting." *Environmental Science and Technology* **37 (8)**: 1509–1514.

Ghouralal, D. 2019a. "Diabetes Affecting Children Before Adults." https://tt.loopnews.com/content/diabetes-affecting-children-adults.

Ghouralal, D. 2019b. "Rambharat: Avoid Buying 'Nasty, Old' Imported Chicken." https://tt.loopnews.com/content/rambharat-avoid-buying-nasty-old-imported-chicken.

Gietel-Basten, S., and S. Scherbov. 2020. "Exploring the 'True Value'of Replacement Rate Fertility." *Population Research and Policy Review* **39 (4)**: 763–772.

Grummer-Strawn, L., N.F. Krebs, and C.M. Reinold. 2009. "Use of World Health Organization and CDC Growth Charts for Children Aged 0-59 Months in the United States." *Morbidity and Mortality Weekly Report* **59 (36)**: 1–15.

Hamilton-Davis, R. 2022. "4 Girls Born First on Xmas Day: Fertility Rate Declining, Says Deyalsingh." *Trinidad and Tobago Newsday* Monday December 26 2022.

Harvard T.H. Chan School of Public Health. 2015. "WHO Report Says Eating Processed Meat is Carcinogenic: Understanding the Findings." The Nutrition Source. Accessed June 30 2024. https://www.hsph.harvard.edu/nutritionsource/2015/11/03/report-says-eating-processed-meat-is-carcinogenic-understanding-the-findings/.

HeligiLibrary. 2018. "Poultry Meat Consumption Per Capita." Prague, Czech Republic: Heligi Analytics. https://www.helgilibrary.com/indicators/poultry-meat-consumption-per-capita/

Hong, Y.-S., K.-H. Song, and J.-Y. Chung. 2014. "Health Effects of Chronic Arsenic Exposure." *Journal of Preventive Medicine and Public Health* **47 (5)**: 245–252.

Hughes, M.F., B.D. Beck, Y. Chen, A.S. Lewis, and D.J. Thomas. 2011. "Arsenic Exposure and Toxicology: A Historical Perspective." *Toxicological Sciences* **123 (2)**: 305–332.

International Agency for Research on Cancer. 2018. *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, 14 ed. Lyon Cedex, France: International Agency for Research on Cancer. https://publications.iarc.fr/Book-And-Report-Series/Iarc-Monographs-On-The-Identification-Of-Carcinogenic-Hazards-To-Humans/Red-Meat-And-Processed-Meat-2018

Jomova, K., Z. Jenisova, M. Feszterova, S. Baros, J. Liska, D. Hudecova, C. Rhodes, and M. Valko. 2011. "Arsenic: Toxicity, Oxidative Stress and Human Disease." *Journal of Applied Toxicology* **31 (2)**: 95–107.

Khan, K.M., R. Chakraborty, J. Bundschuh, P. Bhattacharya, and F. Parvez. 2020. "Health Effects of Arsenic Exposure in Latin America: An Overview of the Past Eight Years of Research." *Science of the Total Environment* **710**:136071.

Lei, H.-L., H.-J. Wei, H.-Y. Ho, K.-W. Liao, and L.-C. Chien. 2015. "Relationship Between Risk Factors for Infertility in Women and Lead, Cadmium, and Arsenic Blood Levels: A Cross-Sectional Study from Taiwan." *BMC Public Health* **15**:1220.

Liu, G.-R., X. Peng, R.-K. Wang, Y.-Z. Tian, G.-L. Shi, J.-H. Wu, P. Zhang, L.-D. Zhou, and Y.-C. Feng. 2015. "A New Receptor Model-Incremental Lifetime Cancer Risk Method to Quantify the Carcinogenic Risks Associated with Sources of Particle-Bound Polycyclic Aromatic Hydrocarbons from Chengdu in China." *Journal of Hazardous Materials* **283**:462–468.

Mandal, B.K., and K.T. Suzuki. 2002. "Arsenic Round the World: A Review." *Talanta* **58 (1)**: 201–235.

Miller, J.N., and J.C. Miller. 2018. *Statistics and Chemometrics for Analytical Chemistry*. Harlow, Essex, England: Pearson.

Mindak, W.R., J. Cheng, S.C. Hight, and S.G. Capar. 2014. *Elemental Analysis Manual (EAM) for Food and Related Products*. US Food and Drug Administration.

Mohammed, E., T. Mohammed, and A. Mohammed. 2017. "Optimization of An Acid Digestion Procedure for the Determination of Hg, As, Sb, Pb and Cd in Fish Muscle Tissue." *MethodsX* **4**:513–523.

Nachman, K.E., P.A. Baron, G. Raber, K.A. Francesconi, A. Navas-Acien, and D.C. Love. 2013. "Roxarsone, Inorganic Arsenic, and Other Arsenic Species in Chicken: A US-Based Market Basket Sample." *Environmental Health Perspectives* **121 (7)**: 818–824.

O'Gam, C., and B. Robey. 2014. "Fertility Trends and Factors Affecting Fertility." In *Women in the Third World: An Encyclopedia of Contemporary Issue.* edited by N.P. Stromquist. New York: Routeledge.

Pacyna, J.M., and E.G. Pacyna. 2001. "An Assessment of Global and Regional Emissions of Trace Metals to the Atmosphere From Anthropogenic Sources Worldwide." *Environmental Reviews* **9 (4)**: 269–298.

Page, N.P. 2019. "Human Health Risk Assessment." In *Basic Environmental Toxicology*, edited by L.G. Cockerham and B.S. Shane, 561–580. Boca Raton, Florida: CRC Press.

PAHO. 2013. "The Trinidad and Tobago Experience." Pan American Health Organization.

Palma-Lara, I., M. Martínez-Castillo, J. Quintana-Pérez, M. Arellano-Mendoza, F. Tamay-Cach, O. Valenzuela-Limón, E.

García-Montalvo, and A. Hernández-Zavala. 2020. "Arsenic Exposure: A Public Health Problem Leading to Several Cancers." *Regulatory Toxicology and Pharmacology* **110**:104539.

Perng, W., R. Conway, E. Mayer-Davis, and D. Dabelea. 2023. "Youth-Onset Type 2 Diabetes: The Epidemiology of An Awakening Epidemic." *Diabetes Care* **46 (3):** 490-499. https://doi.org/10.2337/dci22-0046.

Prayson, B.E., J.T. McMahon, and R.A. Prayson. 2008. "Applying Morphologic Techniques to Evaluate Hotdogs: What is in the Hotdogs We Eat?" *Annals of Diagnostic Pathology* **12 (2)**: 98–102.

Razzaghi, H., D.N. Martin, S. Quesnel-Crooks, Y. Hong, E. Gregg, G. Andall-Brereton, V. Gawryszweski, and M. Saraiya. 2019. "10-Year Trends in Noncommunicable Disease Mortality in the Caribbean Region." *Revista Panamericana de Salud Pública* **43**:e37. doi:10.26633/RSVP.2019.37

Rehman, M.U., R. Khan, A. Khan, W. Qamar, A. Arafah, A. Ahmad, A. Ahmad, R. Akhter, J. Rinklebe, and P. Ahmad. 2021. "Fate of Arsenic in Living Systems: Implications for Sustainable and Safe Food Chains." *Journal of Hazardous Materials* **417**:126050.

Saipan, P., and S. Ruangwises. 2009. "Health Risk Assessment of Inorganic Arsenic Intake of Ronphibun Residents Via Duplicate Diet Study." *Journal of the Medical Association of Thailand* **92 (6)**: 849–56.

Saksena, M.J., A.M. Okrent, T.D. Anekwe, C. Cho, C. Dicken, A. Effland, H. Elitzak, J. Guthrie, K.S. Hamrick, J. Hyman, Y. Jo, B-H. Lin, L. Mancino, P.W. McLaughlin, I. Rahkorsky, K. Raston, T.A. Smith, H. Stewart, J.E. Todd, and C. Tuttle. 2018. "America's Eating Habits: Food Away From Home." United States Department of Agriculture, Economic Research Service. Economic Information Bulletin No. (ECB-196).

Sánchez-Rodas, D., J. Luis Gómez-Ariza, and V. Oliveira. 2006. "Development of a Rapid Extraction Procedure for Speciation of Arsenic in Chicken Meat." *Analytical and Bioanalytical Chemistry* **385 (7)**: 1172–1177.

Sharma, V.K., and M. Sohn. 2009. "Aquatic Arsenic: Toxicity, Speciation, Transformations, and Remediation." *Environment International* **35 (4)**: 743–759.

Singh, A.P., R.K. Goel, and T. Kaur. 2011. "Mechanisms Pertaining to Arsenic Toxicity." *Toxicology International* **18 (2)**: 87–93.

Singh, S.K., and A.K. Ghosh. 2012. "Health Risk Assessment Due to Groundwater Arsenic Contamination: Children are at High Risk." *Human and Ecological Risk Assessment* **18 (4)**: 751–766.

Stolz, J.F., P. Basu, J.M. Santini, and R.S. Oremland. 2006. "Arsenic and Selenium in Microbial Metabolism." *Annual Revue of Microbiology*. **60 (1)**: 107–130.

US Environmental Protection Agency. 1989. "Risk Assessment Guidance for Superfund (RAGS)." Office of Emergency and Remedial Response, US Environmental Protection Agency.

US Environmental Protection Agency. 1991. "Guidelines for Developmental Toxicity Risk Assessment." Federal Register **56 (234)**: 63798–63826.

US Environmental Protection Agency. 2011. *Exposure Factors Handbook*. 2011 Edition (Final Report). Office of Research and Development, National Center for Environmental Assessment.

US Environmental Protection Agency. 2012. "Human Health Risk Assessment." Accessed 23 February 2022. http://www2.epa.gov/risk/human-health-risk-assessment.

Valko, M., H. Morris, and M. Cronin. 2005. "Metals, Toxicity and Oxidative Stress." *Current Medicinal Chemistry* **12 (10)**: 1161–1208.

Wallinga, D. 2006. "Playing Chicken: Avoiding Arsenic in Your Meat." Institute for Agriculture and Trade Policy, Minneapolis, Minnesota, USA.

Wang, L., E. Martínez Steele, M. Du, J.L. Pomeranz, L.E. O'Connor, K.A. Herrick, H. Luo, X. Zhang, D. Mozaffarian, and F.F. Zhang. 2021. "Trends in Consumption of Ultraprocessed Foods Among US Youths Aged 2-19 Years, 1999-2018." *JAMA* **326 (6)**: 519–530. https://doi.org/10.1001/jama.2021.10238.

Wang, S.-L., F.-H. Chang, S.-H. Liou, H.-J. Wang, W.-F. Li, and D.P. Hsieh. 2007. "Inorganic Arsenic Exposure and its Relation to Metabolic Syndrome in an Industrial Area of Taiwan." *Environment International* **33 (6)**: 805–811.

World Health Organization. 2001. "Principles for the Assessment of Risks to Human Health from Exposure to Chemicals." WHO. Accessed February 19, 2022. https://iris.who.int/handle/10665/42156.

World Health Organization. 2011. Safety Evaluation of Certain Contaminants in Food: Prepared by the Seventy-Second Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). WHO Food Additives Series 63. World Health Organization.

World Health Organization. 2019. "Population, Trinidad and Tobago." Accessed 3rd September 2020. https://data.who.int/countries/780.

Wiltse, J., and V.L. Dellarco. 1996. "US Environmental Protection Agency Guidelines for Carcinogen Risk Assessment: Past and Future." *Mutation Research/Reviews in Genetic Toxicology* **365 (1–3)**: 3–15.

Xu, L., X. Wu, S. Wang, Z. Yuan, F. Xiao, M. Yang, and Y. Jia. 2016. "Speciation Change and Redistribution of Arsenic in Soil Under Anaerobic Microbial Activities." *Journal of Hazardous Materials* **301**:538–546.

Yuan, C.-G., G.-B. Jiang, and B. He. 2005. "Evaluation of the Extraction Methods for Arsenic Speciation in Rice Straw, *Oryza Sativa* L., and Analysis By HPLC-HG-AFS." *Journal of Analytical Atomic Spectrometry* **20 (2)**: 103–110.

Zargari, F., M.S. Rahaman, R. KazemPour, and M. Hajirostamlou. 2022. "Arsenic, Oxidative Stress and Reproductive System." *Journal of Xenobiotics* **12 (3)**: 214–222. https://doi.org/10.3390/jox12030016.