

Review Article

The Link Between Culture, Cuisine, And Cancer (A Nigerian Perspective)

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Abstract:

Background: Cancer is a public health challenge in Nigeria, with cases rising in recent times. Research shows that dietary patterns play a substantial yet underestimated role in cancer incidence and death. Some Nigerian diets, which feature high consumption of red and processed meats, deep-fried foods, and the use of plastic bags when preparing, have been identified as culprits in the increased exposure to dietary carcinogens and elevation of cancer risk. Foods such as fruits, vegetables, and whole grains possess cancer-protective properties; however, their intake remains inadequate in combating diet-related cancers.

Aim: This review examines the relationship between diet and cancer in Nigeria, focusing on the current cancer burden of Nigeria, carcinogenic risks linked to food preparation, and preventive nutritional strategies.

Methods: Information was sourced from PubMed, Scopus, Google Scholar, and ResearchGate, alongside reports from global health agencies. Search terms included "diet," "cancer," "Nigeria," "food," "carcinogens," and "prevention." Only articles written in English language and that met predefined inclusion criteria were included.

Results: Evidence suggests that the consumption of carcinogens formed through traditional cooking methods increases the risk of cancer. Notable concerns include barbecue-style cooking, repeated use of cooking oils, and heavy metal contamination in local alcoholic beverages. Protective effects were observed in diets rich in fruits and vegetables.

Conclusion: Dietary practices in Nigeria significantly shape cancer risk. Targeted interventions promoting nutrition education, protective food intake, and safer cooking methods are essential to reduce carcinogen exposure and lower the national cancer burden.

Keywords: Diet; Cancer; Nigeria; Dietary Carcinogens; Toxins

Introduction

With a steady rise in both incidence and mortality rates, cancer has emerged as a major public health challenge in Nigeria (1). Among the various risk factors implicated in cancer development, diet stands out as a significant yet often overlooked contributor (2). In Nigeria, food is not merely a source of sustenance—it serves as a profound expression of culture, identity, and tradition. For generations, food has played a central role in fostering community bonds and reinforcing cultural values (3). However, the ingredients and preparation methods of many beloved Nigerian dishes may carry hidden health risks that silently contribute to cancer development.

While many foods are naturally nutritious, their preparation methods often turn them into sources of carcinogenic exposure. For example, the open-flame grilling of meat (suya - charred spiced meat), which involves direct contact with fire, can produce polycyclic aromatic hydrocarbons (PAHs). Similarly, deep-frying snacks like akara and puff-puff can lead to higher levels of acrylamide, a recognized carcinogen. These harmful compounds are by-products of high-temperature cooking methods that are widely used but rarely

scrutinized (4, 5). Smoked fish, (6) mouldy groundnuts and maize (contaminated with aflatoxins), (7) deep-fried street foods (e.g., akara, puff-puff), (8) and locally brewed alcohol (ogogoro) (9, 10), have also been linked with increased cancer risks due to contamination with carcinogenic compounds like PAHs, acrylamides, methanol, and aflatoxins. In addition, the repeated use of cooking oil for frying and high intake of salt-cured foods like stockfish and dried meats may further increase the risk of cancer, especially in a population already challenged by limited screening and dietary awareness. Diets high in fruits, vegetables, and essential micronutrients, such as vitamin C and folate, are associated with a lower risk of cancer. (9).

This article explores the connection between diet, cultural food practices, and cancer risk in Nigeria, emphasizing how evolving cooking traditions and modern dietary transitions may silently accelerate the nation's cancer burden. By critically examining the links between diet and carcinogenesis, we hope to illuminate a path toward culturally grounded, diet-based cancer prevention strategies.

Methodology

A comprehensive literature search was conducted using various databases, including PubMed, ResearchGate, Google Scholar, and a direct Google search, supplemented by grey literature from the World Health Organization (WHO) and the International Agency for Research on Cancer (IARC) reports. Keywords include diet, nutrition, cancer, Nigeria, dietary carcinogens, red and processed meat, polycyclic aromatic hydrocarbons (PAHs), heterocyclic amines (HCAs), toxins, and metals. The search strategy used combinations of keywords and Boolean operators, including: (diet OR nutrition) AND cancer AND Nigeria; (PAH OR HCAs) AND cancer AND Nigeria; food carcinogens OR cooking methods AND cancer. Only articles written in the English language were considered. EndNote 2025 was used to manage the references received from the search throughout the entire writing and review process. Two researchers independently screened the search results based on the title and abstract, and the other authors helped confirm eligibility based on the inclusion and exclusion criteria.

The authors read through the following sections: title, abstract, material and methodology, results, and discussion, to look for information of interest.

Inclusion criteria were:

1. Articles published in English from 2000 to 2025;
2. Studies focusing on diet-related carcinogens, nutritional epidemiology, or public health interventions relevant to Nigeria or Sub-Saharan Africa;
3. Reports providing quantitative or qualitative data on dietary patterns, cooking practices, or associated cancer risks.

Exclusion criteria were:

Studies unrelated to human cancer; 2. Articles lacking verifiable references or published in non-peer-reviewed outlets (except for authoritative agency reports). Given the heterogeneity of study designs and outcomes, a “narrative synthesis” approach was adopted, integrating biochemical, epidemiological, and cultural perspectives without quantitative meta-analysis.

Cancer Burden in Nigeria

As of July 9, 2025, data from the World Health Organization (WHO) indicated that Nigeria had a cancer burden of 269,109 prevalent cases (over a 5-year period), 127,763 new cases, and 79,542 deaths in 2022. Prostate, colorectal, and liver cancers were the top three

leading cancers among males, while breast, cervix uteri, and colorectal cancers were the most common among females (11). This represents a marked increase compared to the 102,079 cases documented in 2012 (12), indicating a rising cancer burden in the country. When

compared to other diseases, cancer, alongside other non-communicable diseases (NCDs), was responsible for up to 27% of deaths in Nigeria in 2019, and caused 565 deaths per 100,000 in males and 546 deaths in females in 2021. This shows that cancer is not an isolated problem but part of a larger NCD crisis in Nigeria. This burden is made worse by Nigeria's fragile healthcare infrastructure and a critical shortage of skilled health workers. With a density of only 1.83 skilled health workers per 1,000 people, Nigeria falls

significantly short of the WHO's recommended minimum of 4.45 per 1,000. This deficit is further strained by the increasing emigration of medical professionals and a projected population surge to about 263 million by 2030, suggesting that the country's cancer control efforts will face mounting challenges unless urgent investments in healthcare human resources are made (13). Table 1 shows the cancer burden in Nigeria based on World Health Organization data for 2022.

Table 1. Cancer Burden in Nigeria – Statistics at a Glance (2022)

Metric	Males	Females	Both Sexes
Population	109,872,878	106,874,055	216,746,933
Number of New Cancer Cases	48,096	79,667	127,763
Age-Standardized Incidence Rate	93.9	133.3	113.6
Cumulative Risk of Developing Cancer (% < 75 yrs)	10.4	13.8	12.1
Top 3 Leading Cancers (by new cases)	Prostate, Colorectum, Liver	Breast, Cervix uteri, Colorectum	Breast, Prostate, Cervix uteri
Number of Cancer Deaths	32,905	46,637	79,542
Age-Standardized Mortality Rate	68.4	81.4	74.6
Cumulative Risk of Dying from Cancer (% < 75 yrs)	7.4	8.7	8.0
Top 3 Leading Cancers (by deaths)	Prostate, Colorectum, Liver	Breast, Cervix uteri, Colorectum	Breast, Prostate, Cervix uteri
5-Year Prevalent Cases	94,487	174,622	269,109

Data from the World Health Organization (2022) shows the cancer burden of Nigeria over 5 years, with breast and prostate cancer leading the mortality for women and men, respectively (11)

Cancer-Linked Substances Found in Traditional Nigerian Dishes

Heterocyclic Amines (HCAs): Heterocyclic amines are a class of carcinogenic and mutagenic compounds formed during the cooking of muscle meats such as beef, pork, poultry, and fish at high temperatures. These compounds are produced through the reaction of amino acids and creatine or creatinine when meat is grilled, fried, or otherwise cooked using methods involving intense heat. HCAs are generated in significantly higher quantities when meats are overcooked, blackened, or charred, as is commonly observed in local delicacies such as suya. To date, more than 25 HCAs have been identified, with seventeen specifically formed from the cooking of muscle meats. Among these, the International Agency for Research on

Cancer (IARC) has classified four HCAs—IQ, MeIQ, MeIQx, and PhIP—as probable or possible human carcinogens. The carcinogenic potential of HCAs is significantly enhanced after metabolic activation by cytochrome P450 1A2-mediated oxidation, followed by acetylation or sulfation, which transforms these compounds into reactive mutagens capable of forming DNA adducts and inducing genetic mutations that initiate carcinogenesis (14). HCAs have been implicated in various cancer types, such as colon, breast, and prostate cancer (15). Given the popularity of grilled meat in Nigeria, understanding the role of HCAs in diet-related cancer (16, 17). The structural formula of HCAs is given in Figure 1.

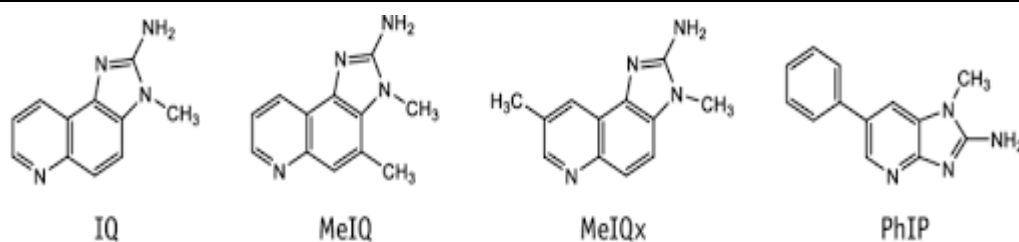


Fig. 1. Structural formula of four heterocyclic Amines (HCAs). “Source: (18)”

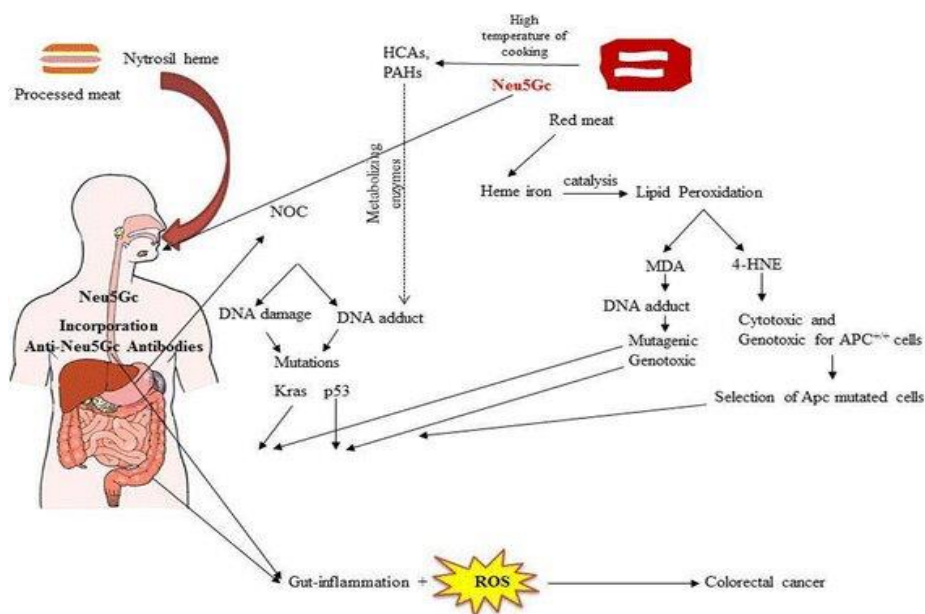


Fig. 2. Mechanisms underlying the carcinogenesis of PAHs and HCAs in colorectal cancer. PAHs and HCAs are produced in significant amounts when red meat is cooked at high temperatures. PAHs and HCAs, when present in the human body, are metabolized by phase I enzymes such as cytochrome P450 monooxygenases (CYP) and phase II enzymes and eventually bind to DNA, forming a DNA adduct. The formation of these DNA adducts results in DNA mutations, alteration of gene expression profiles, and tumorigenesis. Furthermore, heme produced from red meat by lipid peroxidation and endogenous formation of N-nitroso compounds (NOCs) can provoke several DNA mutations responsible for colorectal cancer. Lastly, the association of products of lipid peroxidation (induced by heme), reactive oxygen species (ROS) present in red and processed meat, and N-glycolylneuraminic acid (Neu5GS) would result in inflammation, which can lead to the development of colorectal cancer. “Source: (19, 20)”

Suya (Red meat): The preparation method of suya, which is a traditional Nigerian grilled meat delicacy, has been linked to the formation of carcinogenic compounds, particularly heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs) (21). Studies have shown that grilling meat over open charcoal flames, as commonly practiced in suya preparation, facilitates the production of HCAs such as PhIP, MeIQ, and MeIQx, which form when creatine and amino acids in meat react at high temperatures (22). These HCAs, along with PAHs formed when fat drips onto hot coals and produces smoke, are classified as probable human carcinogens by the International

Agency for Research on Cancer (IARC) (23, 24). Aside from the presence of HCAs and PAHs in suya, which pose health risks, a study by Oyewole et al. (2024) identified dangerously high levels of arsenic, chromium, and cadmium in suya samples sold across Ibadan, Nigeria. These heavy metals exceeded safety thresholds and were associated with incremental lifetime cancer risk (ILCR) values above acceptable limits (25).

Polycyclic Aromatic Hydrocarbons (PAHs):

Polycyclic aromatic hydrocarbons (PAHs) are compounds composed of multiple fused aromatic rings, which are primarily formed through the incomplete combustion of organic materials, such as wood, fossil fuels, and biomass. They represent one of the most concerning classes of environmental carcinogens linked to diet-related cancer risk. Although PAHs are naturally occurring, they can also be formed during food processing methods such as grilling, roasting, drying, and smoking. From a toxicological standpoint, PAHs are not inherently carcinogenic in their native state. However, upon entering the body, these compounds undergo metabolic activation, particularly in the liver, where cytochrome P450 enzymes transform them into reactive epoxide intermediates. These electrophilic metabolites, such as PAH-derived diol epoxides, can form covalent adducts with nucleophilic sites on DNA. This process results in

mutations that can initiate the development of cancer. PAHs have been implicated in various cancer types, such as lung, skin, and bladder cancer (26). In Nigeria, dietary exposure to PAHs is of concern due to cultural cooking practices. The preparation of popular foods such as suya (spiced, flame-grilled meat), smoked fish, and roasted plantains typically involves direct flame or smoke contact, which are conditions that favor PAH formation (27-30). Four representative examples of PAHs are shown below in Figure 3.

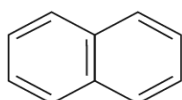
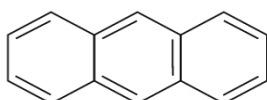
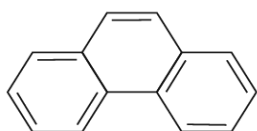
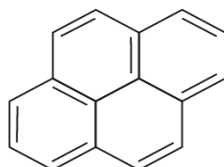
Naphthalene**Anthracene****Phenanthrene****Pyrene**

Fig. 3. Chemical structure of four polycyclic aromatic hydrocarbons (PAHs). Source: (31)."

Kilishi: Kilishi, a popular sun-dried and spiced meat delicacy in Nigeria, was found to contain concerning levels of PAHs when processed using traditional smoking techniques. A study by Fakolade et al. (2017) revealed that commercially prepared Kilishi samples contained PAH concentrations as high as 8.80 µg/kg, exceeding the European Commission's safety threshold of 5.0 µg/kg for benzo[a]pyrene, a marker for total PAH contamination. Similarly high levels (6.40 µg/kg) were also detected in laboratory-smoked samples. These elevated values are largely attributed to direct exposure to smoke from coal combustion at high temperatures (220–250 °C) and the use of oily ingredients like groundnut paste, which increase PAH accumulation through pyrolysis. Since PAHs are mutagenic and carcinogenic compounds, their presence in traditionally smoked Kilishi raises significant public health concerns. Routine consumption of such contaminated products could contribute to long-term cancer risk, particularly cancers of the gastrointestinal tract (32).

Smoked fish or meat (e.g., barbecue): Barbecue cooking has been strongly linked to the formation of PAHs, compounds known for their carcinogenic potential (33, 34). A study by Sumer and Oz (2023) investigated the effects of direct and indirect barbecue

cooking on beef. The results revealed that while cooking method had no significant effect on total PAH4 or PAH8 levels, direct barbecuing and higher cooking degrees (well-done) led to increased levels of benzo[a]pyrene (BaP) and dibenzo[a,h]anthracene (DahA), both highly toxic PAHs. Cooking loss and lipid oxidation also increased significantly with more intense cooking, indicating both nutritional degradation and the formation of higher toxic compounds (35). An Earlier study by Rose et al. (2015) studied PAH formation during various home-cooking methods, including barbecuing with and without wood chips. The study showed that grilling, frying, toasting, and roasting produced minimal PAHs, but barbecuing—especially with charcoal and wood chips—led to significant PAH accumulation in meats like beef and sausages. This study also revealed that BaP levels in heat-treated meat products frequently exceeded the European Commission's safety threshold of 5.0 µg/kg (36). These studies' findings indicate that barbecue techniques, particularly direct flame cooking with charcoal, elevate PAH exposure and may increase cancer risk.

Phthalates: Phthalates are chemicals that are commonly used in plastic production for various items (37). They are considered possible human carcinogens based on evidence from several studies (38). Phthalates such as di(2-ethylhexyl) phthalate (DEHP) and benzyl butyl phthalate (BBP) have shown hepatocarcinogenic effects in rodents, primarily through activation of PPARα, a pathway linked to liver tumor formation (39). DEHP, in particular, has been associated with liver, testicular, prostate, and breast cancers in both in vivo and in vitro models. Lifelong exposure to DEHP in male rats led to increased liver and testicular tumor development, while its metabolite MEHP has been shown to promote prostate and breast cancer cell proliferation. Prenatal and lactational exposure to DEHP also increased susceptibility to prostate tumors in male offspring. In breast cancer models, DEHP stimulated tumor growth without inducing apoptosis, and co-exposure with bisphenol A further enhanced mammary tumor risk and reduced latency (40). Phthalates are also endocrine disruptors, capable of mimicking hormones and interfering with reproductive health and development. Some studies suggest phthalates promote breast tumor growth via estrogen receptor (ER) signaling, while others report estrogen-independent and antiestrogenic mechanisms (14). Figure 4 and Figure 5 illustrate the chemical structures of commonly used phthalates.

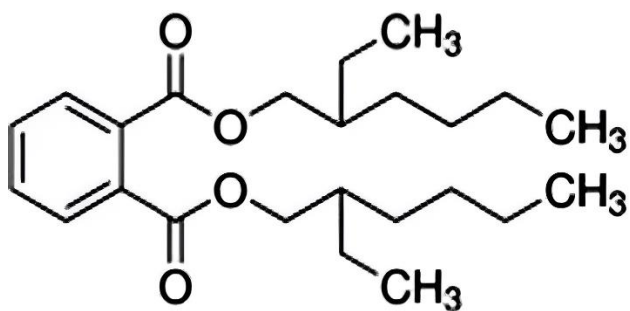


Fig. 4. Structure of di-(2-ethylhexyl) phthalate (DEHP) used in the production of building products (wallpaper, wire and cable insulation), car products, clothing (footwear, raincoats), food packaging, etc. "Source: (41)."

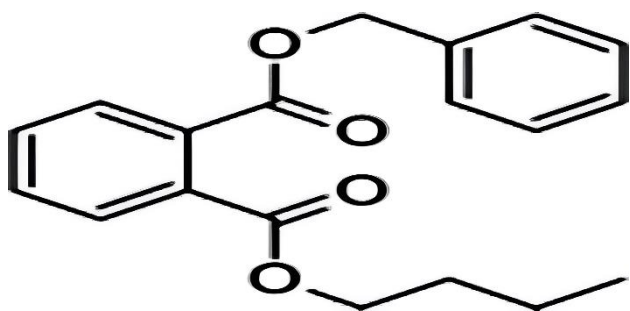


Fig. 5. Structure of benzyl butyl phthalate (BBP), used in the traffic cones, food conveyor belts, and artificial leather. "Source: (41)."

Moi Moi: Moi Moi is a steamed bean paste dish typically prepared from blended cowpeas mixed with seasonings and various other ingredients (42). Moi Moi, as a dish, doesn't pose any danger to health, but the method of cooking is where the problem lies. A study by Iguh et al. (2023) found that cooking Moi Moi in polyethylene film (nylon) led to the migration of di-butyl, di-2-ethylhexyl, and benzylbutyl phthalates into the food. These phthalates were not detected in the raw paste but appeared after cooking and increased further after three days of frozen storage and reheating. This migration occurs because phthalates are not chemically bonded to the plastic, allowing them to leach under

high heat. Although detected levels were below safety limits, their presence raises concern due to the carcinogenic and endocrine-disrupting potential of phthalates (43).

Acrylamides: Acrylamide is a chemical monomer commonly used in producing plastics, dyes, adhesives, and food packaging. However, since 2002, it has gained attention as a food contaminant formed unintentionally during high-temperature cooking methods such as frying, baking, and roasting. Carbohydrate-rich foods like fried potatoes, coffee, biscuits, and bread are notable sources. Acrylamide forms through the Maillard reaction—a chemical interaction between asparagine (an amino acid) and reducing sugars, especially under conditions of high heat and low moisture (44, 45). This compound poses significant health risks. The International Agency for Research on Cancer (IARC) classifies acrylamide as a probable human carcinogen. Mechanistic studies show that it is genotoxic, forming glycidamide, a metabolite that binds to DNA and proteins, promoting mutations. Beyond its cancer risk, acrylamide has been associated with neurotoxicity and reproductive toxicity in animal models (46). In Nigeria, the rise in consumption of fried and baked street foods—such as puff-puff, buns, and yam fries—may increase acrylamide exposure, especially in urban areas. The structural formula of acrylamide is presented in Figure 6, while its metabolic pathway is shown in Figure 7.

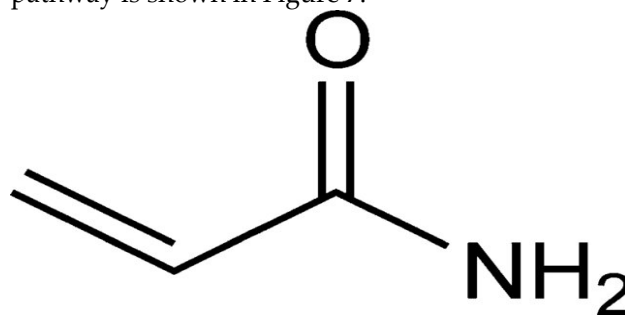


Fig. 6. Structural formula of acrylamide. "Source: (47)."

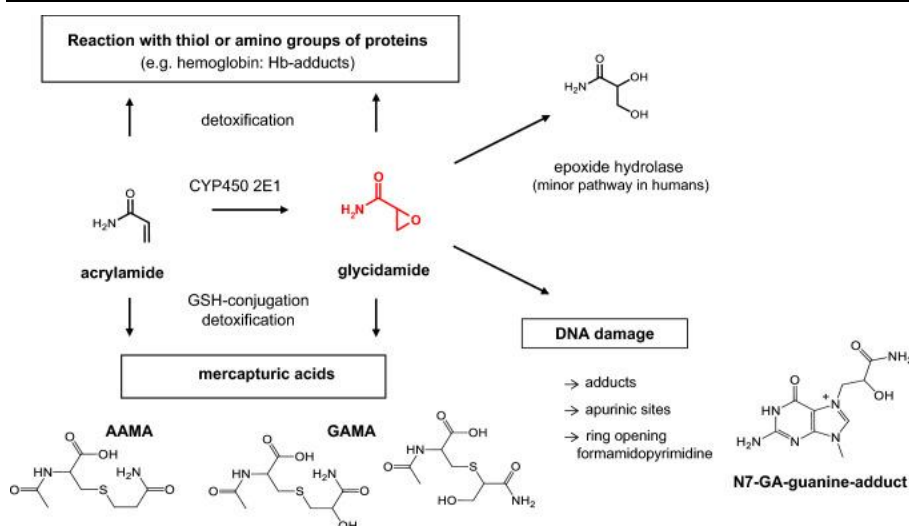


Fig. 7. Metabolic pathway of acrylamide in carcinogenesis.

Once ingested and absorbed from the gastrointestinal tract, acrylamide is converted by cytochrome P450 2E1 (CYP450 2E1) into the genotoxic epoxide glycidamide. Glycidamide, in turn, forms DNA adducts, primarily at N7 of guanine (N7-GA-Gua). This interaction of glycidamide with DNA is what is considered to represent the key event resulting in genotoxicity and carcinogenicity of acrylamide. "Source: (48)."

Puff puff and Akara: Puff-puff and akara are two widely consumed West African fried foods, but research has shown that they contain varying levels of acrylamide, which associates them with potential cancer risks. A study by Akinosun et al. (2025) (49) found that frying puff-puff and akara instead of baking resulted in a 4.40% and 8.09% increase in acrylamide, respectively. In this study, temperature and cooking time were 2 critical factors that influenced acrylamide formation. This study also indicated that an increase in cooking temperature from 150 °C to 180 °C resulted in a 157.63% rise in puff-puff and a 227.78% increase in akara acrylamide levels. Further heating to 210 °C produced an additional 21.51% (puff-puff) and 11.2% (akara) rise. These findings emphasized that higher temperatures and longer durations significantly elevate acrylamide content (49). To minimize health risks, the authors recommend processing puff-puff and akara at temperatures below 150 °C and for less than 5 minutes. When this is not feasible, cooking should not exceed 15 minutes to limit acrylamide accumulation.

Trans fat: Trans fatty acids (TFAs), also known as trans fats, are a type of unsaturated fat with at least one non-conjugated double bond in the trans configuration, as defined by Regulation (EU) No 1169/2011 (50). While small amounts occur naturally in meat and dairy products from ruminants, the primary dietary source is industrially produced partially hydrogenated oils, which are used in processed and deep-fried foods. (51) TFAs have been implicated in the development of several malignancies, notably breast and colorectal cancers. Their consumption disrupts

lipid metabolism, promotes systemic inflammation, and increases oxidative stress, conditions conducive to initiating carcinogenesis (52, 53). Emerging research suggests that trans fats may influence hormonal pathways, potentially contributing to hormone-related cancers. Beyond cancer, TFAs are associated with other chronic conditions, including cardiovascular disease, diabetes, and pregnancy-related complications, which may indirectly affect cancer susceptibility (52). Figure 8 below shows the mechanism of action of trans fat in carcinogenesis.

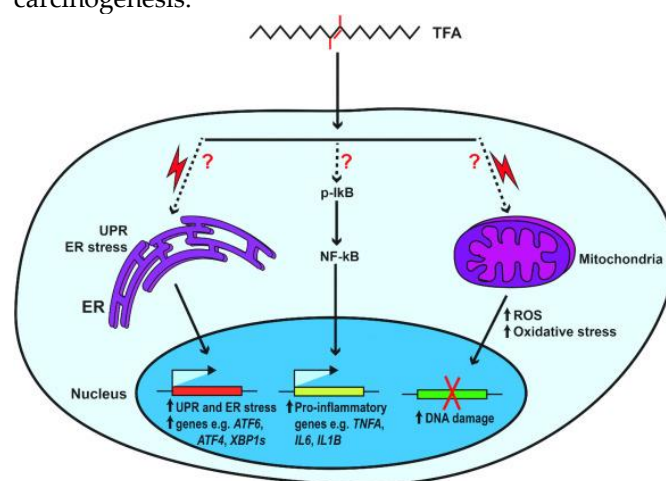


Fig. 8. The mechanism of action of trans fat in carcinogenesis. Trans fat may stimulate inflammation through the induction of NF-κB signaling and endoplasmic reticulum stress. Trans fat can also induce reactive oxygen species (ROS) production, which leads to DNA damage. Inflammation and DNA damage both contribute to cancer development. "Source: (54)."

Fried plantain: Fried plantain, a popular deep-fried dish across West Africa, particularly in Ghana and Nigeria, has become a dietary staple. However, recent evidence has raised concerns about their safety due to the presence of trans fatty acids (TFA). A study by Addo et al. (2023) found significant levels of TFAs in plantain chips sold in Accra, Ghana, especially trans isomers of linoleic acid. These harmful fats were linked

to poor frying practices, with 97% of vendors reusing oil multiple times (55).

Heavy Metals: Heavy metals such as chromium (Cr), nickel (Ni), manganese (Mn), and iron (Fe) are environmental contaminants known for their toxicity and carcinogenic potential. Due to their non-biodegradable nature, they accumulate in tissues and are linked to oxidative stress, organ dysfunction, and cancer (56, 57). A study by Ogidi et al. (2024) analyzed

seven locally produced Nigerian beverages and found that Cr, Ni, Mn, and Fe levels in many samples exceeded WHO limits. While non-carcinogenic risk values were below hazard thresholds, carcinogenic risk values for several metals surpassed the acceptable 1×10^{-4} limit, suggesting long-term health hazards (10). Figure 9 illustrates the mechanisms through which heavy metals contribute to carcinogenesis.

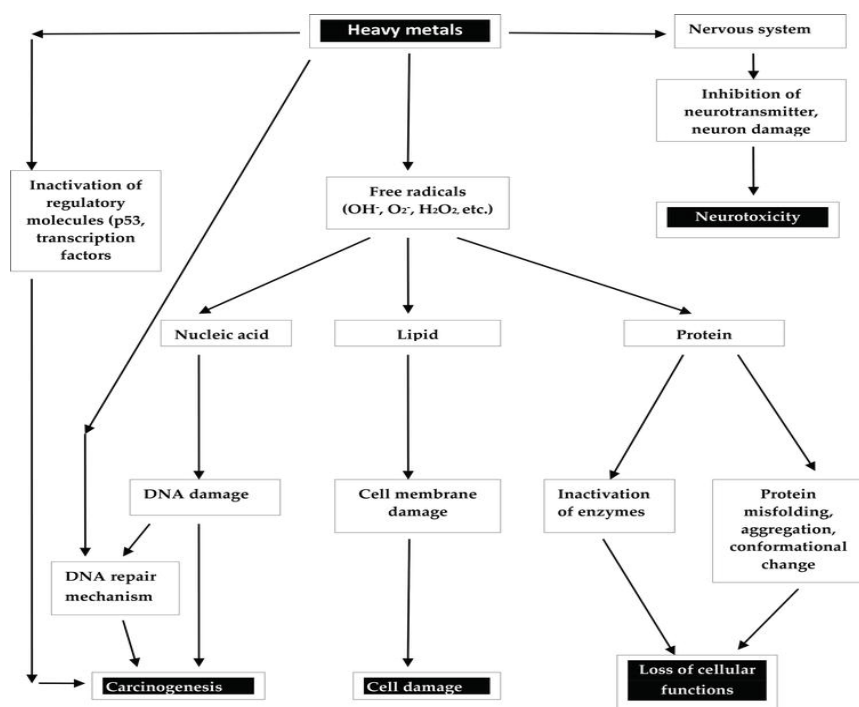


Fig. 9. Mechanism through which heavy metals cause toxicity and contribute to carcinogenesis. "Source: (58)"

Ogogoro: Ogogoro, a distilled spirit derived from fermented *Raphia* palm sap, is consumed widely across southern Nigeria. The study by Ogidi et al. (2024) found that while chromium and nickel levels in ogogoro were within WHO limits, iron levels (1.115 mg/kg) exceeded the safe threshold (0.3 mg/kg). Excess iron has been implicated in the generation of reactive oxygen species (ROS), leading to oxidative damage,

cellular dysfunction, and increased cancer risk, particularly in the liver and pancreas. Although the non-carcinogenic risk was low, the iron-related carcinogenic risk was elevated (10).

Mokite: Mokite, a relatively new marijuana-infused beverage, showed dangerously high levels of iron (1.83 mg/kg) and copper (0.66 mg/kg). The carcinogenic risk values for multiple metals exceeded the acceptable limit. Iron toxicity from Mokite may impair liver function and catalyze ROS production, leading to DNA damage and mutagenesis (10).

Strategies for Cancer Prevention Through Diet

Food regulation: Food regulation by governmental health agencies is important in minimizing cancer risk due to carcinogen exposure. In Nigeria, governmental health regulatory bodies such as the National Agency for Food and Drug Administration and Control (NAFDAC) and the Standards Organization of Nigeria (SON) are tasked with overseeing food safety and creating health policies. Still, the enforcement of these policies remains limited, particularly in informal markets and street food settings where harmful practices like excessive frying, smoking

with firewood, and use of illegal preservatives are common. Not much attention is given to the regulation of known carcinogens such as PAHs, acrylamide, mycotoxins, and heavy metals, all of which are prevalent in local diets through grilled meats, fried snacks, improperly stored grains, and unregulated alcoholic beverages. Strengthening food regulation, educating food handlers on safe preparation techniques, and enlightening the public on healthier food choices should be the priority of these organizations.

Public Health Education: Some diet puts people at risk of cancer; other diets help prevent the risk of cancer (59). The Mediterranean diet is a dietary recommendation that emphasizes eating plant-based foods and healthy fats (60). This diet is characterized by a low saturated fat content, high monounsaturated fat intake (primarily from the high consumption of olive oil), and an abundance of dietary fiber. It includes vegetables, fresh fruit, legumes, non-refined cereals, nuts, poultry, a moderate amount of seafood and dairy products, low intake of red and processed meat, and moderate alcohol consumption. It is rich in antioxidant

compounds and bioactive elements with anti-inflammatory effects, and it has a low glycemic index. Research has shown that adherence to the Mediterranean diet has been associated with decreased risk of several chronic diseases, such as cardiovascular disease, diabetes, neurodegenerative diseases, and cancer (61, 62). As shown in Table 2, the Mediterranean diet emphasizes anticarcinogenic foods such as fruits, vegetables, whole grains, and healthy fats, whose bioactive compounds play protective roles against cancer.

Table 2. Anticarcinogenic foods and their main components

Food/Fruit	Main component	Mechanism of action	Reference
Banana	Flavonoid, phenolics	Inhibits ROS, inhibits cell cycle, and promotes apoptosis.	(63, 64)
Avocado	Triterpenoids, polyphenols, carotenoids, glutathione	Induce cell death, inhibit proliferation, and target the cell cycle	(65-67)
Apple	Triterpenoids, Kaempferol (flavonoid), quercetin	Antioxidant effects induce apoptosis	(68, 69)
Paw paw (papaya)	Acetogenin, alkaloids, terpenoids	Block ATP production, thereby inducing apoptosis and cell cycle arrest	(70, 71)
Pomegranate	Ellagitannins	Inhibit cancer cell proliferation, induce apoptosis, and disrupt signaling pathways.	(72-74)
Grapes	Proanthocyanidins, phenolic antioxidants, kaempferol, and quercetin	Inhibition of cell proliferation, induction of cell cycle arrest in the G1 phase, and apoptosis	(75-79)
Citrus fruits (oranges, lemons, grapefruits)	Naringin (flavonoid)	Inhibiting cancer cell growth, inducing apoptosis (programmed cell death), and modulating various signaling pathways	(80-82)
Berries (blueberries, raspberries, strawberries)	Flavonols, anthocyanins (ACNs), proanthocyanidins (PACs), and phenolic acids	Reduce and repair damage resulting from oxidative stress and inflammation	(83, 84)
Cruciferous vegetables (broccoli, cabbage, kale, Brussels sprouts)	Isothiocyanates, phenolic compounds, flavonoids	Block DNA replication in noncancerous cells, inhibit the binding of carcinogens to DNA, reduce angiogenesis, and increase apoptosis.	(85)
Tomatoes	Lycopene	Antioxidant effect inhibits cancer cell proliferation	(86, 87)
Garlic & Onions	Allicin and sulfur compounds	DNA damage protection, induction of cell death (apoptosis), inhibition of cell proliferation, and blocking of angiogenesis and metastasis	(88-93)
Carrots	B-carotene (carotenoids) and chlorogenic acid (CHA)	Antioxidant effect, inhibiting cell proliferation, inducing apoptosis, and suppressing tumor growth.	(94-96)
Chili pepper	Capsaicinoids (alkaloids)	Initiate apoptosis, cell growth arrest, and suppress angiogenesis and metastasis.	(97, 98)
Turmeric	Curcumin	Suppress cell growth and proliferation, mitigate DNA damage	(99-101)
Whole grains	Dietary fibers	Promote apoptosis, dilute carcinogens, antioxidant effect, and anti-inflammatory effect.	(102, 103)
Legumes (beans, lentils, chickpeas)	Fiber, tannins, flavonoid (quercetin), saponins	Increase growth arrest and apoptosis, antioxidant effect	(104, 105)
Nuts (cashew nuts, walnuts, peanuts)	Alkaloids, polyphenols	Arrest the cell cycle, prevent microtubule formation	(106-108)
Olive oil	Oleuropein (phenols)	Promote apoptosis	(109-112)

Conclusion

The delicate relationship between diet, culture, and cancer in Nigeria highlights an urgent need for multidisciplinary public health intervention. While diets are deeply rooted in the fabric of Nigerian identity, evolving dietary habits—marked by the increasing consumption of fried foods, smoked meats, unregulated alcoholic beverages, and poorly stored grains—pose significant carcinogenic risks. These risks are compounded by socioeconomic challenges, lack of food safety enforcement, and limited public awareness. For the country to overcome the danger posed by diet-related cancers, it must prioritize a comprehensive national strategy that integrates cancer prevention into food systems, health education, and regulatory frameworks. Public enlightenment campaigns should focus on promoting safer cooking methods, recognizing hazardous food additives, and encouraging a shift towards more plant-based, fiber-rich diets. Regulatory bodies must be empowered to monitor and control food contaminants such as PAHs, acrylamide, heavy metals, and mycotoxins, particularly in informal markets and local food production chains.

Recommendation:

The authors recommend establishing a National Dietary and Cancer Prevention Task Force, comprising oncologists, nutritionists, food scientists, policymakers, and community leaders. This body should guide the

formulation of culturally appropriate dietary guidelines, oversee food safety reforms, promote local natural and healthy diets, and champion nutrition education across all levels. Only through such a collaborative and culturally sensitive approach can Nigeria effectively reduce the burden of cancer linked to dietary and lifestyle factors. The Ministry of Health in Nigeria and other associated bodies should encourage, assist, and fund research that produces data on the percentage of cancers in Nigeria attributed to dietary risk, as well as strategies for reducing the cancer burden in Nigeria.

Strengths and Limitations.

This review study addresses an important but underexplored topic using the most recent data and provides practical solutions to combat the problems highlighted in the study. Nonetheless, there are limitations, which include reliance on secondary sources of data; underrepresentation of protective foods in the Nigerian (local) context; the narrative review approach instead of a systematic review pattern; and scarcity of local dietary and cancer data. Despite these challenges, the review offers a valuable foundation for future research and culturally relevant interventions to reduce diet-related cancer risks in Nigeria.

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