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Why Fried Food is Unhealthy: Heat Induced Food Toxicants and Associated Health Risks

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ABSTRACT

Frying is a widespread process used in food preparation and manufacturing as reflected in a large spectrum of products. Fried foods such as potato chips, French fries, expanded snacks, roasted nuts, extruded noodles, doughnuts and fried fish and chicken have gained worldwide popularity due to their unique organoleptic characteristics like distinctive flavour, aroma, appearance, and crunchy texture. During frying, as several physical and chemical changes occur in foods that impart desirable characteristics, heat induced food toxicants such as acrylamide, hydroxymethylfurfural, heterocyclic amine, nitrosamines and polyaromatic hydrocarbons could also be produced. However, these toxicants and the oil used for frying can cause non-communicable diseases such as coronary heart diseases, diabetes, cancer, overweight obesity and hypertension. Diet and nutrition play a key role in the promotion and maintenance of good health. Therefore, use of antioxidants, frying time and temperature control, recipe optimization, use of saturated oil, and vacuum frying are some of the techniques used to reduce the formation of heat generated food toxicants. In addition, increasing consumer awareness of the relationship between nutrition and health and fried food consumption is also crucial.

Keywords: Frying, Fried food, Thermal degradation, Heat-induced food toxicants, Health effects.
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INTRODUCTION

Frying is one of the most popular thermal processing methods for food preparation and manufacturing worldwide. It is a cheap and fast process of simultaneous heat and mass transfer that imparts unique sensory characteristics to food (Ngadi and Xue, 2016) and changes the nutritional characteristics as a result of complex interactions between food and oil (Ziaiifar et al., 2008). In addition, food frying has benefits in reducing risk of microbial spoilage and extends product shelf-life by thermal destruction of microorganisms, enzymes, and reduction of water activity on the surface of the food (Fellows, 2009). Frying of food is widely used in the food industry in food manufacturing, at home and street with a significant impact on the final quality of foods. Deep frying, shallow or pan frying, stir-frying and sautéing are all standard frying techniques. Fried foods such as potato chips, French fries, expanded snacks, roasted nuts, extruded noodles, doughnuts and fried fish and chicken have gained worldwide popularity. Heat generated food toxicants such as acrylamide (Figure 1), hydroxymethylfurfural (HMF), heterocyclic amine (2-2-amino-1-methyl-6-phenylimidazo-pyridine), nitrosamines and polyaromatic hydrocarbons such as benzoapyrene and chloropropanols (for example, 3-monochloropropane-1-diol, 3-MCPD) are produced during food frying (Stadler, 2012). Such compounds cause different types of DNA damage like nucleotide alterations and gross chromosomal aberrations. Most genotoxic compounds begin their action at the DNA level by forming carcinogen–DNA adducts, which result from the covalent binding of a carcinogen or part of a carcinogen to a nucleotide (Jägerstad and Skog, 2005). The major pathway for acrylamide formation in foods is Maillard reaction with free asparagine as main precursor (Mottram et al., 2002; Stadler et al., 2002; Zyzak et al., 2003; Stadler et al., 2004). Furan also forms as an intermediate in the Maillard Reaction (Ames,1992) and from direct dehydration of sugars under acidic conditions (caramelisation) during thermal treatments applied to foods (Kroh, 1994). 3-MCPD is formed from glycerol

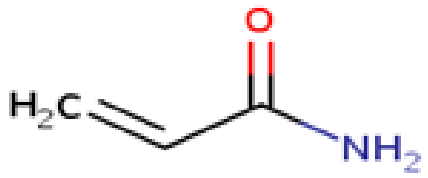

Figure 1. The structure of Acrylamide (Friedman, 2003).

or acylglycerols and chloride ions in heat-processed foodstuffs that contain fat with low water activity (Calta et al., 2004). Diet and nutrition play a key role in the promotion and maintenance of good health, as they are important modifiable risk factors for chronic diseases (Mozaffarian et al., 2015). World Health Organization recommends limiting the consumption of saturated and trans-fats (hydrogenated fats), sugars and salt in the diet, which are often found in snacks, processed foods and drinks (Nishida et al., 2004). Evidence suggests that regular and excessive consumption of energy-dense foods high in fat, particularly saturated fat, and in refined carbohydrates can lead to weight gain, obesity and pose an increased risk for non-communicable diseases (NCDs) (Mozaffarian et al., 2015). NCDs are the leading causes of death globally, killing more people each year than all other causes combined (Nishida et al., 2004). Of 56.4 million global deaths in 2015, 39.5 million, or 70%, were due to non-communicable diseases.

The four main NCDs are cardiovascular diseases, cancers, diabetes and chronic lung diseases (WHO, 1998). The burden of these diseases is rising disproportionately among lower and middle-income countries and populations. In addition, of the 57 million deaths that occurred globally in 2008, 36 million almost two-thirds were due to NCDs, comprising mainly cardiovascular diseases, cancers, diabetes and chronic lung diseases (Alwan et al., 2010). The association between dietary fats and chronic diseases has been extensively studied with evidence indicating that dietary fats play an important role in the development of coronary heart diseases, obesity, stroke, cancer and diabetes. Improving population diets play an important role in preventing chronic NCDs. According to estimates by (WHO, 2009), without preventative measures, the number of deaths by NCDs will increase by 17% on a global scale over the next ten years. Therefore, it is necessary to understand the fat absorption, mechanism of formation and mitigation of heat generated foods toxicants during food frying and the associated health risks of fried food consumption on long-term. Thus, this paper reviews the existing evidence on the generation of heat-induced food toxicants, mitigation mechanisms and the relation of fried food consumption with the major non-communicable diseases. In this review article, all relevant evidence from different sources such as Scopus, ProQuest, SpringerLink, Taylor and Francis, ACS, Wiley Online, and ScienceDirect were systematically reviewed based on some important questions and using key words related to the title and the collected informations were compiled for interpretation.

FRYING GENERATED FOOD TOXICANTS

During frying, a variety of reactions cause a spectrum of physical and chemical changes. In the presence of oxygen, food moisture and high temperature, the oil undergo different deleterious reactions particularly hydrolysis caused by water, oxidation, and thermal alteration caused by oxygen and heat. These are tremendously complex reactions that cause the formation of numerous polymerization products, of which over 400 have been identified (Paul and Mittal, 1997).

Acrylamide

Acrylamide is a white odorless crystalline solid, soluble in water, ethanol, ether, and chloroform. Formerly, acrylamide was known only as a constituent of cigarette smoke and products of plastics and water treatment chemicals. In early 2002, acrylamide was detected in a range of foods heated during production or preparation (Tareke et al., 2002; Rosén and Hellenäs, 2002). It is one of the chemicals known as Maillard reaction products, which formed when foods are heated at high temperatures (Mottram et al., 2002; Stadler et al., 2002; Becalski et al., 2003). Particularly high concentrations were found in products of plant origin heated to high temperatures, such as potato chips, French fries, pan-fried potato products, or crisp bread, whereas the contents in foods rich in protein were low (Tareke et al.,

2002). The primary pathway for the formation of acrylamide in foods is via Strecker degradation of asparagine with dicarbonyls by the reaction between an amino acid called asparagines and reducing sugars such as glucose and fructose (Mottram et al., 2002). Strecker degradation is a chemical reaction which converts α-amino acid into an aldehyde containing the side chain, by way of an imine intermediate. This reaction generally occurs at higher temperatures (Biedermann et al., 2002) and in low moisture conditions and it is part of the Maillard reaction that provides color, flavor, and aroma in cooked foods. Alternatively, it can be produced from precursors such as 3-aminopropionamide, acrylic acid, and acrolein, and reactions between other amino acids such as alanine, arginine, cysteine and other sugars like galactose, lactose, and sucrose. In addition, it has been reported that acrylamide could be directly generated from N-glycosides formed from sugars and amino acids during an early stage of the Maillard reaction (Stadler et al., 2002) (Stadler et al., 2002). The water content is one of the most important factors in the formation of acrylamide, besides the reaction temperature and time (Slayne and Lineback, 2005; Gertz and Klostermann, 2002).

The minimum of acrylamide formation was observed at the water content between 25 and 40%; outside of this range, the acrylamide concentration was higher. Fructose was more effective for the acrylamide formation in comparison with glucose (Yaylayan et al., 2003). In general, carbohydrate degradation products are necessary to form acrylamide, activating asparagine by forming a Schiff base, which subsequently decarboxylates upon heating. The decarboxylated Schiff base can form acrylamide directly or degrade into 3-aminopropionamide (3-APA), which in turn yields acrylamide by the elimination of ammonia.

In the past years many methods have been developed to quantitatively analyse the acrylamide content in food. The majority are classical methods based on LC-MS/MS or GC technique. However, because of the complexity of food matrices, these methods do not suffice for the analysis of acrylamide in heat-treated foods at trace levels. Particularly, they lack selectivity and the additional degree of analyte certainty required to confirm the presence of a small molecule, such as acrylamide in a complex food matrix.

Gas chromatography (GC) - mass spectrometry (MS) and HPLC analysis are both acknowledged as the major useful and authoritative methods for the acrylamide determination. A simple and rapid method was developed and validated for the determination of acrylamide in potato and cereal-based foods by using a single quadrupole LC-MS interfaced
with positive atmospheric pressure chemical ionization (APCI+) (Kim et al., 2010). In addition, a reverse phase LC-MS based on stable isotope dilution assay was used for acrylamide analysis.

Furthermore, a new Volta metric biosensor was developed to detect acrylamide in food sample.

Mitigation Mechanisms of Acylamide Formation

In 2011, the food and drink Europe (FDE) identified the main measures that may lead to a reduction of acrylamide in French fries, breakfast cereals, biscuits and bakery wares. The mitigation strategies include agronomical techniques (control of reducing sugars in potato, control of tuber storage temperature, use of sprout suppressants to prevent sweetening during storage, maintaining sulphur levels for cereal cultivation), recipes formulations techniques (selection of potato varieties and cereal varieties low in acrylamide precursors, addition of proteins, glycine, cysteine and other amino acids, organic acids and acidulants, calcium ions, cyclodextrin, natural antioxidants or antioxidant extracts etc., replacement of reducing sugars with sucrose and of ammonium bicarbonate with sodium bicarbonate) and during preparation and processing (use of asparaginase enzyme, optimization of time-temperature of frying or baking, changing in the type of oven, prolonged fermentation) (Palermo et al., 2016; Torres and Parreño, 2009). Moreover, Anese et al. (2009) proposed the removal of acrylamide after formation by means of vacuum but its impact on manufacturing practices and food quality has not yet been clearly established.

FURAN AND 2-METHYLFURAN

Furan (C_4H_4O) is volatile with the boiling point of 31^0C and colorless liquid and is classified as a possible human carcinogen by the International Agency for Research on Cancer. Similar to acrylamide, furan in food could potentially become a serious problem due to its widespread presence in many types of products. It has been reported to occur in a number of foods that undergo heat treatment, such as canned and jarred foods (US FDA, 2004). There are multiple pathways underlying furan formation, such as thermal degradation or rearrangement of carbohydrates alone or in the presence of amino acids, thermal degradation of certain amino acids, oxidation of ascorbic acid under high temperatures, and oxidation of polyunsaturated fatty acids and carotenoids. According to Maga and Katz (1979), the primary source of furans in food is thermal degradation of carbohydrates such as glucose, lactose, and fructose. Becalski and Seaman (2005) also reported the formation of furan from ascorbic acid and its derivatives. In addition, they demonstrated that furan can be formed from the oxidation of polyunsaturated fatty acids at elevated temperatures and that the addition of commercially available antioxidants (such as tocopherol acetate) reduced the formation of furan by up to 70%. More recently, Perez Locas and Yaylayan (2004) studied the formation of furan from model systems using pyrolysis-GC-MS analysis and $^{13}C_3$-labelled sugars, amino acids and ascorbic acid. They observed that certain amino acids, such as serine

and cysteine, can degrade to form furan, but that other amino acids, such as aspartic acid, threonine and -alanine, require the presence of sugar to form furan. 2-Methylfuran and 3-methylfuran, have been found concurrently with furan, and apparently, are also formed during thermal processing and are likely to undergo a similar metabolic fate to furan. 3-methylfuran is the analogue of 2-methylfuran. Like furan and 2-methylfuran, 3-methylfuran is also aromatic in nature. It can be supposed that its chemical properties are similar, but information is scarce.

Mitigation Strategies to Reduce Furan Concentration in Foods

No available mitigation strategies specifically addressed to reduce furan content in foods because of the nature of its precursors and formation pathways. HMF forms through Maillard reaction and caramelisation, which mostly contribute to desired colour, taste and aroma of heated foodstuffs. Unfortunately, HMF formation follows the same pathways leading to brown and flavour compounds. For instance, a high correlation between HMF content and browning development has been repeatedly reported (Capuano et al., 2009; Capuano and Fogliano, 2011) so that modelling the time-temperature profile by reducing heating times and/or temperatures is likely to reduce HMF concentrations in the same time resulting in a reduction of browning development which can potentially compromise the quality and acceptability of final products. The same happens when mitigation strategies based on changes in recipes are applied, for example by replacing reducing sugars with non-reducing sugars or polyalcohols. Currently, no official standard methods are available for the analysis of furan and methylfurans in foods. Three analytical approaches are used most often for the determination of furan and methylfuran in foods. All of them are based on a mass-spectrometric (MS) detection and quantification using stable isotope dilution assays with *d*4-furan and *d*3-2-methylfuran. Separation is accomplished by capillary gas chromatography.

HETEROCYCLIC AMINES AND POLYCYCLIC AROMATIC HYDROCARBONS

Heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs) are chemicals formed when meat, including beef, pork, fish, muscle or poultry is cooked or fried using high-temperature methods, such as pan frying or grilling directly over an open flame (Cross and Sinha, 2004). Polycyclic aromatic hydrocarbons (PAHs) are organic compounds containing only carbon and hydrogen that are composed of multiple aromatic rings.
Heterocyclic amines (HCAs) are chemical compounds containing at least one heterocyclic ring, which has atoms of at least two different elements, as well as at least one amine group. HCAs are formed when amino acids, sugars, and creatine (a substance found in muscle) react at high temperatures and long cooking times. Amino-imidazo-azaarenes (AIA) and amino carbolines are the two common toxic compounds formed during frying of meat. PAHs are produced through incomplete combustion of organic matter. They are formed when fat and juices from meat grilled directly over an open fire drip onto the fire, causing flames. PAHs can also be formed during other food preparation processes, such as smoking of meats (Cross and Sinha, 2004). Cooking methods that expose meat to smoke or charring contribute to PAH formation (Jägerstad and Skog, 2005). The major contributors to PAH intake in the average diet are oils and fats, cereals, and vegetables (Moret and Conte, 2000). Purcaro et al., (2006) has set a maximum level of 2 ppb for benzo[a]pyrene (BaP) in oils and fats intended for direct consumption or for use as an ingredient in foods. The amount and variety of AIAs and carbolines formed in fried meat products primarily depend on processing conditions, of which temperature, time, and method of frying are the most important (Chiu et al., 2016; Chen et al., 2016). A large variety of AIAs and carbolines often occur in cooked meat products under drastic conditions such as frying 200 or 300^0C for 10 min. Liquid chromatography-mass spectrometry (LC-MS) is a technique used to quantify the number of HCAs and PAHs in foods.

Ways to Reduce HCA And PAH Formation

Even though no specific guidelines for HCA/PAH consumption exist, concerned individuals can reduce their exposure by using several cooking or frying methods like avoiding direct exposure of meat to an open flame or a hot metal surface and avoiding prolonged cooking times (especially at high temperatures) can help reduce HCA and PAH formation. Using a microwave oven to cook meat prior to exposure to high temperatures can also substantially reduce HCA formation by reducing the time that meat must be in contact with high heat to finish cooking. Furthermore, continuously turning the meat over on a high heat source can substantially reduce HCA formation compared with just leaving the meat on the heat source without flipping it often and removing charred portions of meat and refraining from using gravy made from meat drippings can also reduce HCA and PAH exposure (Knize and Felton, 2005).

HEALTH RISKS ASSOCIATED WITH CONSUMPTION OF FRIED FOODS

Many of the Maillard reaction produce compounds that contribute to the flavors and aromas of food during food frying or cooking. However, compounds having adverse physiological effects or potential health risks are also formed. Nowadays, the consumption of deep-fried food has gained popularity which may cause increased risk of non-communicable diseases. To reduce the expenses,

the oil tends to be used repeatedly for frying. When heated repeatedly, changes in physical appearance of the oil will occur such as increased viscosity and darkening in color, which may alter the fatty acid composition of the oil. With each reuse, oil becomes more degraded, and more gets absorbed into food, which can contribute to weight gain, higher cholesterol, and higher blood pressure all risk factors for type 2 diabetes and heart disease (Cahill et al., 2014).

Human Dietary Exposure Rate to Toxicants in Fried-Foods

Fried foods particularly, potato chips and French fries are among the food items that contain the highest levels of acrylamide, furan, HCAs and PAHs, although concentrations may vary significantly from one item to the other. An individual's exposure to these toxicants reflects the combined intake from diet, smoking, second-hand smoke, drinking water, occupational sources, toiletries and household items. Acrylamide absorption through dermal exposure is much lower because the skin provides a barrier that reduces acrylamide uptake (Fennell et al., 2005).

However, oral exposure is critical in determining the amount of acrylamide and its metabolites that circulate in the body. Becalski et al. (2003) documented concentrations of acrylamide in commercial potato chips and French fries ranging from 530 to 3700 ng/g and 200 to 1900 ng/g, respectively. Dietary acrylamide exposure estimates are mainly available for the general adult population to have been documented to range from 0.3 to 0.8 µg/kg of body weight per day (WHO, 2002). Mean intake of acrylamide in adults averages 0.5 µg/kg body weight per day across populations in several countries.

In Sweden, coffee intake is the major contributor to intake, whereas, in U.S. populations, potato crisps and chips are responsible for the majority of intake (Mucci, 2006).

Dietary acrylamide intake in children, youngsters and adolescents has been suggested to be significantly higher than that of adults (Dybing et al., 2005).

Similarly, WHO (2002) reported that acrylamide intake in children is generally two to threefold higher than that of adults when expressed on a body weight basis. In addition to having a higher average food intake per kg body weight than adults, children and adolescents also consume acrylamide rich-food, such as potato chips and French fries, on a more regular basis than the rest of the population (Dybing et al., 2005). In 2004, the European Food Safety Authority (EFSA) reported the dietary furan exposures of 33.5 µg day^{-1} and 1.1 µg day^{-1} for adults (15 to 75 years old) and children (4 to 6 years old), respectively. Additionally, children were the group which contributed the most to the intake of furan through breakfast cereals. The highest degree of furan inhalation resulted from the frying of chipped potatoes in an open chip pan (between 5 and 35 ng L^{-1}) (Fromberg et al., 2009).

Overweight and Obesity as Resulting from Consuming Fried Foods

Obesity is a chronic disease characterized by the accumulation of excess adipose tissue. Worldwide, 2.8 million people die each year as a result of being overweight and obesity (Resnikoff et al., 2004) and an estimated 35.8 million (2.3%) of global disability-adjusted life year (DALYs) are caused by overweight or obesity (WHO, 2009). In addition, it is estimated that one in 13 annual deaths in the EU is likely to be related to excess weight (Banegas et al., 2003).The risks of coronary heart disease, ischemic stroke and type 2 diabetes mellitus increase steadily with increasing body mass index (BMI), a measure of weight relative to height (WHO, 2002). Raised BMI also increases the risk of cancer of the breast, colon/rectum, endometrium, kidney, esophagus (adenocarcinoma) and pancreas (World Cancer Research Fund/American Institute for Cancer Research., 2007; WHO, 2002). The consumption of high levels of high-energy foods, such as processed foods that are high in fats and sugars, promotes obesity compared to low-energy foods such as fruits and vegetables (WHO, 2003). Fried foods are crunchy, aromatic, highly palatable, and rich in fats. As a consequence, eating fried food in ad libitum conditions may result in higher absolute intake of foods with high energy density and low satiety index. The relatively low satiety index of fats (Blundell, 2002) may be related to their low ability to stimulate insulin and leptin production (Havel et al., 1999). High energy density diet, increased portion size, low physical activity and adoption of a sedentary lifestyle as well as eating disorders are considered as important risk factors for the development of obesity (James, 2008).

Several studies reported a positive association between fried food intake and being overweight (Srivastava et al., 2009), waist circumference (Krachler et al., 2006) or weight gain among pregnant women (Stuebe et al., 2009). The European Prospective study showed a positive association of fried food consumption with central and general obesity (adjusted odds ratios for general obesity in the highest *versus* the lowest quintile of fried food intake: 1.26 (95% CI: 1.09 to 1.45, *p* for trend <0.001) in men and 1.25 (95% CI: 1.11 to 1.41, *p* for trend <0.001) in women) (Guallar-Castillón et al., 2007). The prevalence of general obesity was 27.6% (out of 12,905) in men and 27.7% (out of 20,637) in women; the prevalence of central obesity was 34.5 and 42.6%, respectively. This study reported that the prevalence of general and central obesity increased with increasing intake of energy from fried food. General obesity is defined as BMI > or = 30 kg/m^2 and central obesity as waist circumstance, WC > or = 88 cm. According to this study, fried meat, fish, potatoes, and eggs were the 4 groups of fried food most frequently consumed by study participants, with >75% of men and women consuming each of those groups of food. The energy intake in men and women, respectively, ranged from to 2.0 and 1.5% for fried eggs to 5.0 and 3.6% for fried meat.

Consumption of fried meat was positively associated with general obesity in men, and the intake of fried fish was associated with general obesity in women. The same pattern was observed for central obesity. In addition, consumption of fried egg was associated with central obesity in men. In gene-diet interaction analysis in three US cohort studies revealed that the association between total fried food consumption and BMI was stronger in participants with a higher genetic risk score than in those with a lower genetic risk score in both the Nurses' Health Study and Health Professionals Follow-up Study (P=0.005 and 0.02, respectively, for interaction) (Qi et al., 2014). Pereira et al., (2004) revealed that greater consumption of fried food away from home was associated with a higher BMI and weight gain in US children and adolescents. In this study, 30.3% (out of 6212 children and adolescents 4 to 19 years old) of study participants ate fast food on any given day, these foods seem to contribute an additional 57 kcal (187 kcal × 30.3%) to the daily diet of the average child in the United States.

Coronary Heart Disease as Related with Consumption of Fried Foods

Laboratory investigations show that fried foods may act through many mechanisms, such that the resulting effect on coronary heart disease is difficult to anticipate. Frying can specifically increase the amount of trans-fatty acids in foods (Litin and Sacks, 1993). Fried foods have been associated with various cardiovascular risk factors in cross-sectional studies. However, only a few studies have evaluated the effect of fried foods on the risk of cardiovascular disease. In a case-control study from India, including 165 patients with coronary heart disease and 199 matched controls, patients with coronary heart disease when compared to controls reported a greater intake of both shallow fried food (24.0 ± 60.4 versus 2.7 ± 17.2 g/day; $p < 0.01$) and deep-fried food (15.2 ± 25.0 versus 1.0 ± 5.1 g/day; $p < 0.01$) (Panwar et al., 2011). Similarly, Djoussé et al. (2015) showed a positive and graded association between fried food consumption and the incidence of heart failure in a prospective cohort study; compared to subjects who reported fried food consumption of <1 per week, the adjusted hazards ratios (95% CI) for heart failure were 1.24 (1.04 to 1.48), 1.28 (1.00 to 1.63) and 2.03 (1.37 to 3.02) for fried food intake of 1 to 3/week, 4 to 6/week and 7+/week, respectively (p for linear trend: 0.0002). In addition, Belin et al. (2011) found that fried fish consumption (>1 serving per week at baseline) was associated with a 48% higher risk of heart failure (HR, 1.48 (95% CI: 1.19 to 1.84). On the contrary, the analysis of the Spanish cohort of the European Prospective Investigation into Cancer and Nutrition found no association between consumption of fried food and the risk of coronary heart disease or cause mortality (Guallar-Castillón et al., 2012). While the existing evidence proposes a higher risk of heart failure in people with frequent fried food consumption, underlying biologic mechanisms remain to be elucidated.

Diabetes as Related with Consumption of Fried Foods

Diabetes is the leading cause of renal failure in many populations in both developed and developing countries. Several studies have shown a positive association between a high glycemic diet and the risk of type 2 diabetes (T2D) (Schulze et al., 2004). Consumption of potatoes, red meat and other processed meats have been positively associated with the risk of T2D (Ylönen et al., 2007; Halton et al., 2006; Fung et al., 2004; Pan et al., 2011; Khosravi-Boroujeni et al., 2012). According to Halton et al. (2006), the intakes of potatoes and French fries were positively associated with the incidence of type 2 diabetes in a large prospective cohort of women. The increased risk was more pronounced when potatoes replaced whole-grain products in the diet. This association was independent of known risk factors for type 2 diabetes, including family history, age, BMI, physical activity, smoking status, postmenopausal hormone use, and dietary factors. As expected, the positive association between potato consumption and the risk of type 2 diabetes was seen primarily in obese and sedentary women.

In addition, fried foods from restaurants and fast food consumption were positively associated with T2D (Krishnan et al., 2010; Odegaard et al., 2012; Pereira et al., 2005). Similarly, data from the Nurses' Health Study/Health Professionals Follow-Up Study revealed a strong association between the frequency of fried food consumption and the risk of type 2 diabetes with adjusted RRs (95% CIs) for individuals who consumed fried foods <1, 1 to 3, 4 to 6 or ≥7 times/week of 1.00 (reference), 1.15 (0.97 to 1.35), 1.39 (1.30 to 1.49) and 1.55 (1.32 to 1.83), respectively (Cahill et al., 2014). The frequency of fried food consumption was also associated with the incidence of gestational diabetes (adjusted RR = 2.18 (95% CI: 1.53 to 3.09) comparing fried food intake of 7+ to that of <1 time per week) (Bao et al., 2014). However, a study from Italy demonstrated that in obese (not in lean), insulin-resistant women, consumption of foods fried in extra-virgin olive oil significantly reduced both insulin and C-peptide responses after a meal (Farnetti et al., 2011). Thus, it's possible to conclude as there is strong evidence for a positive association between fried food consumption and the risk of T2D.

Carcinogenic Effects Frying-Induced Food Toxicants

It has been postulated that acrylamide is carcinogenic through a genotoxic pathway (Dybing et al., 2005), after conversion to glycidamide, a DNA-reactive epoxide. According to IARC, (1994) acrylamide concentrations exceeded 1000 μg/k classified as Group 2A probably carcinogenic to humans. Studies have shown that exposure to HCAs and PAHs can cause cancer in animal models (Sugimura et al., 2004). In many experiments,

rodents fed a diet supplemented with HCAs developed tumors of the breast, colon, liver, skin, lung, prostate, and other organs (Kato et al., 1989; Shirai et al., 2002). In laboratory experiments, HCAs and PAHs have been found to be mutagenic that is, they cause changes in DNA that may increase the risk of cancer. HCAs and PAHs become capable of damaging DNA only after they are metabolized by specific enzymes in the body, a process called bioactivation. Studies have found that the activity of these enzymes, which can differ among people, may be relevant to cancer risks associated with exposure to these compounds (Moonen et al., 2005). Population studies have not well established a definitive link between HCA and PAH exposure from fried or cooked foods and cancer in humans.

One difficulty with conducting such studies is that it can be difficult to determine the exact level of HCA and/or PAH exposure a person gets from meats. However, researchers found that high consumption of well-done, fried, or barbecued meats was associated with increased risks of colorectal (Cross et al., 2010), pancreatic (Anderson et al., 2002; Stolzenberg-Solomon et al., 2007), and prostate cancer (Cross et al., 2005; Sinha et al., 2009). Because many of PAHs are carcinogenic in experimental animals, they are widely believed to make a significant contribution to the burden of cancer in humans. Several epidemiological studies conducted in Taiwan and China revealed that Asian women ranked highest in the world for lung cancer, probably because of the exposure to fumes from cooking oil (Wu Williams et al., 1990). Ko et al. (1997) reported that the risk of lung cancer was higher from stir-frying than from deep frying. Some authors suggested that the increased cancer risk observed among people exposed to oil fumes is correlated with the presence of PAHs in the fumes of heated oils (Chen and Chen, 2001; Chiang et al., 1997).

Mutagenic Effects Frying-Induced Food Toxicants

High-temperature frying of protein-rich foods generates volatile and non-volatile compounds with mutagenic and carcinogenic properties (Straif et al., 2006). A variety of volatile carcinogens and toxicants have been detected in the fumes from high-temperature frying, including acetaldehyde, acrolein, benzene, 1,3-butadiene, ethylene oxide, heterocyclic amines and polycyclic aromatic hydrocarbons and have been suggested to be responsible for the mutagenic properties of the fumes from cooking oils (Shields et al., 1995). The highest levels of mutagenic activity from fried meat are detected in the pan residue and meat crust, compared with lower levels detected in the cooking fumes (Felton et al., 1980). These mutagenic activity levels in the cooked meat and surrounding air are driven primarily by the cooking temperature rather than cooking duration (Berg et al., 1990).

Genotoxicity Effects of Frying-Induced Food Toxicants

Genotoxicity of furan has been reported in many animal studies. Furan appears to be mutagenic to mouse lymphoma cells, independent of S9 activation. S9 is a crude liver enzyme extract that can, under certain conditions, convert materials without any genotoxic activity to active genotoxic entities.

High doses of furan caused structural chromosome abnormalities but did not affect chromatid exchange in mouse bone marrow cells.

According to a study Mariotti et al. (2013), with a single oral dose of 200 or 100 mg/kg body weight, furan did not cause uncontrolled DNA synthesis in mouse or rat hepatocytes in vivo. Mutagens such as cis-2-butene-1,4-dial are similar to unsaturated compounds that react with DNA. This was directly mutagenic at non-toxic concentrations in a Salmonella enterica Typhimurium strain (TA104) that was sensitive to aldehydes, but not to some other strains (Perez Locas and Yaylayan, 2004). It is possible that furan or cis2-butene-1,4-dial reacts with DNA in target cells and can play a major role in furan-induced tumors. After bioactivation into its metabolites, furan induces loss of ATP, which causes an inevitable uncoupling of hepatic mitochondrial oxidative phosphorylation. This would activate cytotoxic enzymes, including endonucleases that produce DNA double-strand cleavage, leading to cell death (Perez Locas and Yaylayan, 2004).

Hypertension Effects of Frying-Induced Food Toxicants

So far, there is limited and inconsistent epidemiological evidence directly relating fried food consumption and hypertension. A cross-sectional study from Spain reported that consumption of fried foods was associated with a higher prevalence of hypertension (Soriguer et al., 2003). The SUN (Seguimiento Universidad de Navarra) Mediterranean cohort study reported that frequent consumption of fried foods at baseline was associated with a higher risk of hypertension (adjusted hazards ratios = 1.18 (95% CI: 1.03 to 1.36) and 1.21 (95% CI: 1.04 to 1.41) for those consuming fried foods 2 to 4 and >4 times/week, respectively, compared to those consuming fried foods <2 times/week (p for trend = 0.009) (Sayon-Orea et al., 2014). Similarly, Kang and Kim (2016) found that fried food consumption was strongly associated with hypertension among Korean women. However, a significant association was found between the frequency of fried food consumption and hypertension in men. The oxidation process during food frying increases the amount of trans-fatty acids in food and is positively associated with the risk of hypertension (Wang et al., 2010). In addition, this study reported a positive association between dietary intake of trans-fatty acids and the risk of hypertension (adjusted RR in the highest quintile: 1.08; 95% CI: 1.01 to 1.15). However, more evidence is required to further clarify the mechanism and association between fried food consumption and hypertension.

Conclusion and recommendation

Frying is a common and popular process utilized in the food industry and street due to its significant sales and a vast quantity of products. From the consumers' point of view fried food palatability is related to unique sensory characteristics such as flavor, texture and appearance. At the same time, heat generated food toxicants such as acrylamide, hydroxymethylfurfural, heterocyclic amine, nitrosamines and polyaromatic hydrocarbons can be formed. These toxicants and the oil used for frying cause a disease like cancer, diabetes, heart, obesity and DNA complications. To minimize those health risks associated with fried consumption, use of varieties that are low in sugar and an amino acid asparagines, controlling frying temperature (fry foods in the range of 145 to 170ºC), frying in closed system not in open air due to oxidation, frying French fries to a golden yellow rather than a golden brown color, toasting bread to the lightest color, soaking raw potato slices in water for 15 to 30 min before frying or roasting and not storing raw potatoes in the refrigerator are some of the techniques helps to reduce those toxicants during frying process.

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