# A Review on Health Risks from Processing Contaminants in Edible Oils and Foodstuffs

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## Abstract

**Abstract** – Processing contaminants are chemical substances produced during processing of edible oils and foodstuffs. They also might result from environmental contamination and may pose a risk to animal and human health. Edible oils and foodstuffs processing methods include fermentation, smoking, drying, refining and high-temperature cooking that have been the sources of 3-monochloropropanediol (3-MCPD) and mutagens. Exposure to these processing contaminants could be intentional in terms of food additives and adulterants or unintentional in the case of contaminants such as mycotoxins, pesticides, and metals. The mechanism of action of these chemicals is mainly through DNA damage, which causes mutagenic effects such as chromosomal aberrations, point mutations, frameshift mutation, and cellular transformations, thereby altering the genetic information. In the long run, these changes get translated into various pathologies such as tumor and cancer of various vital organs, thereby posing great threat to the health of consumers. 3-MCPD, mutagens and processing contaminants emerging during thermal processing of edible oils and foodstuffs will be discussed briefly in the current review to provide an overview of current knowledge regarding the critical health risks associated with processing contaminants of edible oils and foodstuffs.

**Keywords**: Edible Oils; Foodstuffs; Health Risks; Processing Contaminants

  

# Introduction

## 1.1 Official Definition of Contaminant

A "contaminant" is defined in legislation (Regulation (EEC) No 315/93) as any substance not intentionally added to food which is present in such food as a result of the production (including operations carried out in crop husbandry, animal husbandry and veterinary medicine), manufacture, processing, preparation, treatment, packing, packaging, transport or holding of such food or as a result of environmental contamination [1].

## 1.2 Health Risks from 3-MCPD Processing Contaminant in Refined Edible Oils

Refining of edible oils at a high temperature generates byproducts such as 3-monochloropropanediol (3-MCPD) which is the most toxic food contaminant formed during thermal processing of refined edible oils [2, 3]. 3-MCPD exists either in free form, or in bound form called 3-MCPD esters in different foodstuffs that has passed through cooking processes using cooking oil. In order to produce safe edible oils, usage of new materials and methods in refining process are explored, and the effects of byproducts generated during this process on human body are closely monitored [4]. Deodorization is a key step during oil refining for overall oil quality, especially the deodorization temperature is the direct and critical parameter [5]. The UK Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment [6] has considered the carcinogenicity of 3-MCPD and its fatty acid esters. European Commission [7], the regulatory arm of the European Union, namely the European Commission (EC) Scientific Committee on Food (SCF), adopted the tolerable daily intake (TDI) of 2 µg/kg body weight (bw) for 3-MCPD. Furthermore, The Joint WHO/FAO Expert Committee on Food Additives (JECFA) also recommended a provisional maximum TDI of 2 µg/kg bw for 3-MCPD [8, 9]. In March 2016, the European Food Safety Authority (EFSA) issued an extensive report warning about the possible health consequences of contaminants created during the processing of edible oils [10, 11]. EFSA specifically identified 3-MCPD, which was classified as a possible human carcinogen by International Agency for Research on Cancer (IARC) and documented in IARC Monographs [12]. Finally, there is a growing concern associated with release of bound 3-MCPD into its free form that results in bioavailability and absorption of 3-MCPD into human body fluids and tissues, e.g. human breast milk, which requires additional studies

  

[13-15]. Kidney is one of the primary target organs for 3-MCPD [16] with induced renal injury [17], toxicity in kidney, lung, testis, and heart [18-20], immunosuppressive activity [21], toxic effects on male reproduction [22] and is regarded as a rat carcinogen inducing testicular lesions, Leydig-cell and mammary gland tumors in males and kidney tumors in both genders [23, 24]. Combination of histopathological examination, clinical chemistry and metabolomics analyses resulted in systematic comprehensive assessment of the long-term toxicity of 3-MCPD, where renal tubular hyaline cast accumulation with epithelium cell degeneration and potential kidney toxicity were major histopathological changes with thin appearance and subdued behavior accompanied by decreases in bw. Microscopy revealed tubular basophilia in kidneys, exfoliated degenerative germ cells in the lumen of the seminiferous tubule of testes, vacuolation in the brain, axonal degeneration of sciatic nerve and cardiomyopathy [25, 26].

### 1.3 Determination of 3-MCPD in some Edible Oils using GC-MS/MS

Almoselhy et al., [27] recently carried out an investigation of 3-MCPD in some edible oils using optimized recent updated and validated enhanced swift analytical indirect method for determining 3-MCPD in consumed edible oils (palm, palm olein, extra virgin olive, corn, sunflower, soybean, olive pomace) and blend of 5% sunflower oil with extra virgin olive oil, using selective and sensitive gas chromatography tandem triple quadrupole mass spectrometry (GC-MS/MS) employing deuterated 3-MCPD (3-MCPD-d5) as internal standard (IS) during the entire analytical procedure to obtain precise and accurate results. The occurrence and variation of 3-MCPD contents among the studied oils were found in different levels ranged from 93.1 µg/kg to 5634.1 µg/kg oil samples, with maximum value assigned for palm oil (5634.1 µg/kg) followed by palm olein (5576.8 µg/kg), corn oil (2447 µg/kg), sunflower oil (1817.3 µg/kg), soybean oil (1486.1 µg/kg), olive pomace oil (572.5 µg/kg), blend of 5% sunflower oil with extra virgin olive oil (210 µg/kg) and extra virgin olive oil (93.1 µg/kg). Palm, palm olein, corn, sunflower and soybean oils were found out of the limits recommended by the Commission Regulation (EU) 2020/1322, whereas, extra virgin olive oil, olive pomace oil and blend of 5% sunflower oil with extra virgin olive oil were in compliance and within the limits recommended by EU. Moreover, 3-MCPD content could be used as a good tool for authenticity and quality of genuine extra virgin olive oil [27].

  

The mechanism for the formation of 3-MCPD esters and palm oil related compounds is assumed that glycerol, monoacylglycerols (MAGs), diacylglycerols (DAGs) and phospholipids are precursors on the way to the esters. The formation of free 3-MCPD strongly depends on temperature and the content of lipids, glycerol, salt and water. Additionally to triacylglycerols (TAGs), fats and oils contain varying amounts of free fatty acids, MAGs and DAGs depending on the history of the raw material before processing. While oils like rapeseed, sunflower, olive or soybean oil contain between 1 and 3% DAGs, in palm oil amounts between 6 and 10% can be found resulting from the activity of lipases after maturation before inactivation. Crude coconut, palm and palm kernel oils are distinguished by high amounts of free fatty acids up to 7%, while the other oils only contain between 1 and 2% [28]. Studying of factors impacting the formation of 3-MCPD during palm oil production, a root-cause analysis was performed in order to map the parameters potentially responsible for the occurrence of MCPD diesters in refined palm oil and related fractions [29].

Chlorine-containing compounds exist in the form of either inorganic or organic. Inorganic chlorine salts of calcium chloride ($CaCl_2$), magnesium chloride ($MgCl_2$), iron (III) chloride ($FeCl_2$) and iron (III) chloride ($FeCl_3$) originate from fertilizers and irrigation process. $FeCl_2$ and $FeCl_3$ were reported to have higher contents compared to other sources of inorganic chlorine. The occurrence of organochlorines in crude palm oil (CPO) prior to processing indicates that the chlorinated compounds might be present in the oil palm fruitlets even before harvesting. The correlations between total chlorine content in vegetable oil and 3-MCPD level have also been established by many researchers. It was suggested that organochlorines might indirectly act as a chlorine donor during oil deodorization process, which takes place at a temperature above 180 $^{o}$C. During the process, the sum of organochlorines depleted as the sum of 3-MCPD diesters increased and HCl was formed. Therefore, HCl is suspected to be one of the reactive compounds contributing to the formation of 3-MCPD. It has also been identified that the 3-MCPD ester level increases with the addition of ionic bound chlorine, for instance tetrabutylammonium chloride (TBAC) during the modeling of deodorization for vegetable oil. A similar finding, involving a laboratory scale of CPO physical refining highlighted the potential of natural organochlorine as a chlorine precursor due to its oil solubility, which inhibits its removal during rinsing step with water. Conventionally, among fertilizers used in the oil palm plantation are ammonium

  

chloride, $NH_4Cl$ and potassium chloride, KCl. In addition, the herbicides used in the plantation, namely diuron,2,4-D amine, dicamba and fluroxypyr also contained chlorine compound. As most of the herbicides used in the plantation are water soluble, the oil palm fruitlet is likely to be exposed to the chlorine compound through nutrient uptake by the palm trees and through leaching process, where the chlorine compound dissolved in groundwater which will then be absorbed by the palm trees during cultivation. The irrigation water used in the oil palm plantations is also a possible source of chlorine precursor, in addition to the treated wastewater from the treatment facilities that used $FeCl_3$ as flocculants. Moreover, the bruising of fresh fruit bunches (FFBs) was identified to correspond with the increase in FFA content in the palm oil during harvesting and transportation to the mills. The FFA formation is equivalent to the formation of diacylglycerols (DAGs) and monocylglycerols (MAGs), which influence the formation of 3-MCPD [30, 31].

### 1.4 Health Risks from Mutagens Processing Contaminants in Refined Edible Oils

An array of evidences has proved that some of the dietary components or the compounds produced during processing of food have been the sources of mutagens. Exposure to these mutagens could be intentional in terms of food additives and adulterants or unintentional in the case of contaminants such as mycotoxins, pesticides, and metals. The mechanism of action of these chemicals is mainly through DNA damage, which causes mutagenic effects such as chromosomal aberrations, point mutations, frameshift mutation, and cellular transformations, thereby altering the genetic information. In the long run, these changes get translated into various pathologies such as tumor and cancer of various vital organs, thereby posing great threat to the health of consumers [32].

Edible oils, the source of essential fatty acids procured from plant, animal, or synthetic fat, is used in frying, baking, and other types of cooking/food preparations. These are also used in various food preparations and flavorings, which do not involve any heat source, such as salad dressings and bread dips. From extraction, refinement and processing during various food preparations induce various alterations in the composition and physicochemical properties of the cooking medium. Therefore, choice of oil should be judiciously chosen for the purpose of

fulfilling its specific property. Edible oils and fats is one commodity that is prone to various modes of adulterations. The well-known oil adulterants are cheaper oils compromising the oil

  

quality along with other nonedible oils or compounds such as argemone oil (AO), tricresyl phosphate (TCP), and butter yellow. Studies suggest that consumption of mustard oil adulterated with AO leads to a clinical condition known as epidemic dropsy. Sanguinarine, a benzophenanthridine alkaloid of AO, is an electrophilic molecule that intercalates in the GC-rich regions of DNA, thereby causing genotoxicity as evidenced in comet assay, chromosomal aberration, and micronuclei test. AO has been shown to be carcinogenic in nature and it is feared that it is one of the etiological agent for gall bladder cancer (GBC) in the population of Indo-Gangetic Basin, where consumption of mustard oil is maximum. Butter yellow, a fat-soluble azo nonpermitted dye, is often mixed with colorless cheap oil along with the mustard pungency factor, allyl isothiocyanate, to look and appear like mustard oil. Butter yellow interacts with macromolecules including DNA and proteins potentiating genotoxic and mutagenic responses [32].

It has been found as a contributory factor to cancer of respiratory tract, liver, and skin, due to which this color has been banned for food usage since 1950s. Recently, this dye has also been shown to produce GBC in experimental animals. TCP is an odorless, colorless, industrial, and organophosphorus chemical. It is used in lacquers and varnishes as a plasticizer and. TCP has been shown to cause paralysis of hands and feet in animals and humans. However, its mode of action is not yet fully understood. It appears that ortho isomer of TCP is more toxic than its meta or para isomers. Outbreaks of TCP poisoning have occurred in past in the United States, Morocco, India, Durban, and Sri Lanka. In July 1988, an outbreak through the consumption of adulterated rapeseed oil took place at Behala area in Southwest outskirts of Kolkata, where about 600 victims of polyneuritis were reported to the hospital, of which 343 were admitted. An 18-month follow-up study of these patients revealed that only 37.9% patients recovered from TCP-induced paralysis, whereas the majority (62.1%) still had neurological deficits. It was observed that alcoholics showed less response to recovery because of the formation of a metabolite saligenin cyclic-o-tolyl phosphate, which is five times more toxic than the parent compound. Although saligenin cyclic-o-tolyl phosphate is an alkylating agent, no studies have been conducted on mutagenicity and genotoxicity of TCP. Another incident of mutagens in edible oil was recorded in the year 1981 in Spain, where consumption of

contaminated colza oil led to the development of multisystem disorder, in which over 20,000 were affected and over 300 people lost their lives. This was later called as toxic oil syndrome,

  

in which prevalence of mutation was reported in the genes encoding for N-acetyl transferase 2. Figure (1) shows mutagens produced in oils with respective effect on target organs [32].

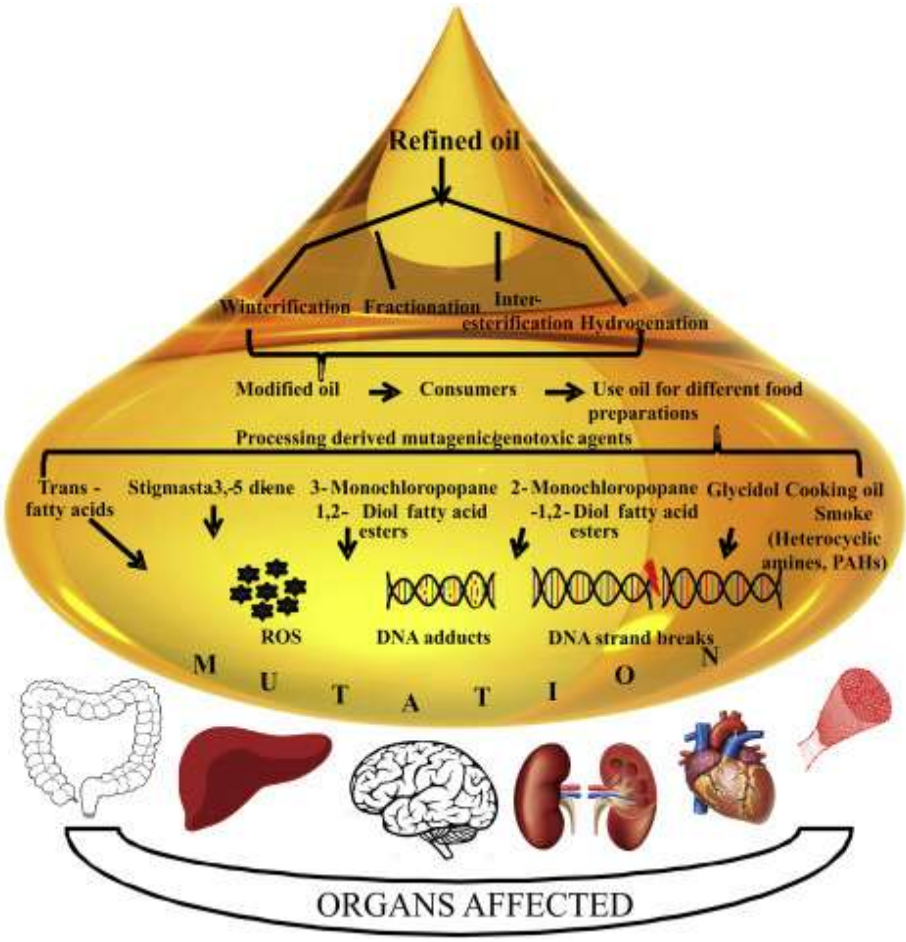


**Figure (1) Mutagens produced in oils with respective effect on target organs**

It has been observed that the generation of undesired process gives rise to contaminants possessing mutagenic and genotoxic potential such as trans-fatty acids, stigmasta-3,5-diene, and 3-monochloropropane-1,2-diol (3-MCPD) fatty acid esters. These are generated as a result of vegetable oils subjected to high temperature. These chemicals tend to generate oxidative stress in the system, thus forming DNA adducts, leading to chromosomal aberrations, and if these manifestations prevail for a longer period in the system, it may get converted into mutagenic outcome. Cooking oil fumes (COF) has caused passive smoking in the nonsmoking individuals, increasing the risk of lung cancer. Several epidemiological studies from China and Taiwan showed that subjects exposed to COF had major bulky DNA

  

adducts (benzo[a]pyrene 7,8-diol 9,10-epoxide-N-2-deoxyguanosine (BPDE-N-2-dG) adduct), causing p53 hot spot mutations, leading to lung tumors. In another study, on exposure to COF, there was induction of 8-hydroxydeoxyguanosine in calf thymus as a result of oxidative DNA damage, which may also be linked to the etiology of lung cancer [32].

### 1.5 Health Risks from Processing Contaminants in Foodstuffs

Consumer exposure to furan and methylfurans in food could lead to possible long-term liver damage. The most exposed group of people are infants, mainly through consumption of ready-to-eat jarred or canned foods. Exposure in other population groups is mainly from consumption of grain-based foods and coffee, depending on age and consumer habits. Furan and the related compounds 2- and 3-methylfurans are chemical contaminants that naturally form during heated food processing, including cooking. These substances have always been present in cooked or heated foods. The European Commission asked EFSA for a scientific evaluation on the risk to human health of the presence of furan and methylfurans (2-methylfuran, 3-methylfuran and 2,5-dimethylfuran) in food. They are formed in foods during thermal processing and can co-occur. Furans are produced from several precursors such as ascorbic acid, amino acids, carbohydrates, unsaturated fatty acids and carotenoids, and are found in a variety of foods including coffee and canned and jarred foods. Regarding furan occurrence, 17,056 analytical results were used in the evaluation. No occurrence data were received on methylfurans. The highest exposures to furan were estimated for infants, mainly from ready-to-eat meals. Grains and grain-based products contribute most for toddlers, other children and adolescents. In adults, elderly and very elderly, coffee is the main contributor to dietary exposure. Furan is absorbed from the gastrointestinal tract and is found in highest amounts in the liver. It has a short half-life and is metabolised by cytochrome P450 2E1 (CYP2E1) to the reactive metabolite, cis-but-2-ene-1,4-dialdehyde (BDA). BDA can bind covalently to amino acids, proteins and DNA. Furan is hepatotoxic in rats and mice with cholangiofibrosis in rats and hepatocellular adenomas/carcinomas in mice being the most prominent effects. There is limited evidence of chromosomal damage in vivo and a lack of understanding of the underlying mechanism. Clear evidence for indirect mechanisms involved in carcinogenesis include oxidative stress, gene expression alterations, epigenetic changes, inflammation and increased cell proliferation. The CONTAM Panel used a margin of exposure (MOE) approach for the risk characterization using as a reference point a

  

benchmark dose lower confidence limit for a benchmark response of 10% of 0.064 mg/kg body weight (bw) per day for the incidence of cholangiofibrosis in the rat. The calculated MOEs indicate a health concern. This conclusion was supported by the calculated MOEs for the neoplastic effects [33].

Thermal process contaminants, which exert carcinogenic or mutagenic effects on human health, are generated in foods during common thermal processing such as frying, baking, and roasting. Heterocyclic aromatic amines (HAAs), acrylamide, 5-hydroxymethyl-2-furfural (HMF), α-dicarbonyl compounds (α-DCs), advanced glycation end products (AGEs), chloropropanols (3-MCPD) and related esters, and lipid oxidation products are the most important examples for thermal process contaminants in thermally processed foods. The presence of varying amounts of thermal process contaminants in widely consumed foods daily is considered as a major concern by public authorities worldwide. Therefore, efforts to reduce the amount of these substances in heat-treated foods have gained importance. Most thermal process contaminants have reactive moieties in their structures that may react with other food constituents during digestion and may decrease by reacting with amino and sulfhydryl compounds under intestinal conditions or may increase by forming from their precursor intermediates under gastric conditions. During digestion, certain process contaminants are formed from their precursors. On the other hand, some are eliminated as a result of reactions with amino or sulfhydryl compounds released in the digestive tract. Reviewed data indicate that the levels of process contaminants in foods do not reflect the exact amount that human is exposed to. This suggests that potential elimination and formation reactions of process contaminants during digestion should be taken into consideration to accurately estimate their bioavailabilities [34].

Acrylamide is a process contaminant already regulated in Europe whereas the toxicological effects of certain heat-induced furan derivatives such as hydroxymethylfurfural (HMF) and furfural are still under revision. Acrylamide, HMF and furfural content were evaluated in 113 coated fried foods provided by food services (restaurants and school canteens). Sampling included battered and breaded foods intended for frying and were grouped according to meat-products (ham & cheese, chicken nuggets), fish-products (fish sticks, fish fillets), seafood (squid rings), vegetables (onion rings), and doughs (croquettes). Acrylamide ranged between 23.8 and 130.4 µg/kg, HMF between 0.11 and 16.07 mg/kg, and furfural between 0.01 and

  

1.04 mg/kg. Presence of these compounds was mostly restricted to the outer part of foods (coating material), since browning reactions are sped up in the crust. Onion rings showed a significantly higher concentration than the other samples being studied. Home-style (freshly made) chicken nuggets exhibited significantly lower acrylamide content than frozen par-fried nuggets from an industrial source. This finding suggests that increasing the complexity of the formulation of the coating through the introduction of ingredients or additives aimed at improving palatability might lead to the development of heat induced process contaminants. As a consequence, the increase of harmful compounds during thermal treatment of coated food should be carefully considered during the industrial formulation of coatings. Although the recent Regulation 2017/2158 for acrylamide does not mention specific values or mitigation strategies for coated foods, its contribution to daily acrylamide exposure within the population must not be dismissed. This was the first systematic prospective evaluation of process contaminants in real operative conditions at food services in Spain. Mean contents were 44 µg/kg (acrylamide), 1.98 mg/kg (HMF) and 0.17 mg/kg (furfural). Contaminants were higher in the crust of the coated fried foods than in the food core. Battered and breaded onion rings exhibited the highest concentrations of acrylamide and the acrylamide level in home-style nuggets was lower than industrially manufactured [35, 36].

Hydroxymethylfurfural (HMF), furaldehyde (FAL) and acrylamide (AA) are produced during the food roasting process. The content of these toxic products in a common ready-to-eat breakfast, known as “Bsissa”, was investigated. This North African traditional food is mainly based on roasted cereals, leguminous and spices. HMF and FAL were detected in most samples, with values not exceeding 24.71 mg/100 g and to 3.91 mg/100 g, respectively. This analysis may help in the mitigation step of furanic compounds by choosing the appropriate Bsissa ingredients. [37]. PAHs or polycyclic aromatic hydrocarbons (benzo[a]anthracene, chrysene, benzo[b]fluoranthene, and benzo[a]pyrene) reported as generated during the manufacture of smoked pork sausage [38]. N-nitrosodimethylamine (NDMA), dimethylamine, carbonyl content and thiobarbituric acid reactive substances (TBARS) generated during in the production and cooking of fermented sausages [39].

TBARS are formed as a by-product of lipid peroxidation. Malondialdehyde (MDA) is one of several end products formed through the decomposition of lipid peroxidation products. The TBARS method is nonspecific for MDA; fatty peroxide-derived decomposition products

  

other than MDA are Thiobarbituric Acid (TBA) positive. In general, MDA/TBA reactivity is a reliable estimator of lipid peroxidation. Increased circulating concentrations of TBARS have been observed in leptin-deficient obese mice, as well as in men and women with obesity and with metabolic syndrome. Besides the reported obesity-associated increase in systemic TBARS, several studies have found increased levels of TBARS in muscle tissues. For example, increased levels of TBARS have been observed in cardiac muscle of obese Zucker Fatty rats and in diet-induced obese Wistar rats, which were returned to normality by treatment with statins. Levels of TBARS are also increased in the soleus muscle of rats fed ad libitum with a high-fat diet for 12 weeks. Importantly, TBARS levels are elevated in skeletal muscle of ob/ob mice, and return to normality after leptin replacement but not after pair feeding, suggesting a direct positive effect of leptin on oxidative stress in the skeletal muscle. Obesity is associated with a chronic low-grade inflammation and increased oxidative stress that leads to increased comorbidity. While these processes have been extensively studied in adipose tissue, information regarding inflammation and oxidative stress in skeletal muscle in the context of obesity is not so profuse. Several cytokines, myokines, and lipid molecules produced by the skeletal muscle have been claimed to play a role in the control of inflammation both in the skeletal muscle and at the systemic level. On the other hand, several biomarkers of oxidative stress and antioxidant enzymes are contributing to the unbalanced redox state of the skeletal muscle in obesity [40].

Free radicals and lipid peroxides play an important role in the oxidative stress balance between prooxidant and antioxidant activities. If the balance tilts towards prooxidants, the functional health of an organism is jeopardized. Malonaldehyde (MDA), thiobarbituric acid reactive substances (TBARS), lipid hydroperoxides (LH), and 4-hydroxyalkenals (4-HNE) are examples of lipid peroxidation by-products that have been used as barometers of lipid peroxidation reaction rates. By-products of lipid peroxidation reactions have been investigated in many studies that have focused on exercise using both healthy and diseased models. Increases in MDA, TBARS, LH, or 4-HNE are directly linked to increased lipid peroxidation rates, and indirectly related to electron transport disturbances, uncoupling of oxidative phosphorylation, increased mitochondrial respiration, and/or elevated oxygen uptake (VO2). These lipid peroxidation byproducts are, themselves, toxic. Aldehydes such as MDA may be harmful because of their carcinogenic, mutagenic, and protein cross-linking

  

properties. The basis of the TBARS method is the measurement of MDA, which is one of the secondary products formed during the oxidation of polyunsaturated fatty acids. There are, however, other endogenous sources of MDA in tissues, which come from a variety of reactions such as side products of prostaglandin and thromboxane synthesis. LH are the first separate products of oxidation, and interaction between LH and proteins or any other compounds, can damage membranes and enzymes. 4-HNE is a well characterized oxidation product of polyunsaturated fatty acids and increases in direct proportion to both LDL oxidation and lipid peroxidation reactions. Steady-state MDA, TBARS, LH, and 4-HNE content of a tissue is the net result of the endogenous rate of lipid peroxidation chain reactions, the metabolic removal of lipid peroxidation by-products, and the antioxidant status of the tissue. It is generally agreed that oxygen toxicity is often accompanied by lipid peroxidation by-products. However, it is not clear whether lipid peroxidation byproducts themselves are directly harmful to cells or if lipid peroxidation reaction rates coincidentally increase when oxygen radicals overwhelm antioxidants. An interesting side to the incubus perception of lipid peroxidation is that lipid peroxidation has been shown to be essential for normal cell development. Lipid peroxidation can also be driven by agents other than oxygen or oxygen derived radicals, such as chelated iron, redux metal irons, ascorbate, enzymes such as cyclooxygenase-dependent peroxidation of arachidonate to prostaglandin and thromboxane precursors or the NADPH-cytochrome P450 reductase dependent reactions in microsomes. The rate of lipid peroxidation can also be moderated by the amount and/or activity of certain antioxidants. That lipid peroxidation by-products can increase without evidence of higher oxygen consumption or oxygen derived radical activity, is of interest in studies on lipid peroxidation, disease, and research using different modes of exercise [41].

A surplus of calories in the body is under normal circumstances stored as triglycerides in adipose tissue. However, once the storage capacity is exceeded, as happens in obesity, dyslipidemia appears and lipids accumulate ectopically in different organs, including the kidney, damaging them through toxic processes called lipotoxicity. The increased influx of lipids into the kidneys translates into an increase in their intracellular concentration and promotes insulin resistance, generation of reactive oxygen species, and inflammation. This exposure to oxidative stress also generates endoplasmic reticulum stress, which leads to cell apoptosis and fibrosis through the transcriptional growth factor β pathway, contributing to

  

alterations in the glomerular filtration barrier and renal disease. The molecular structure of lipids makes them susceptible to oxidation. Acrolein, malondialdehyde (MDA), 4-Hydroxynonenal (4-HNE), and thiobarbituric acid reactive substances (TBARS) are produced during lipid peroxidation. All these molecules are water-soluble, low-molecular-weight products, which make them easy to remove in hemodialysis; hence their value as a marker of oxidative stress during hemodialysis is limited. In contrast, lipid hydroperoxide, which is another product of lipid peroxidation, is lipid-soluble and difficult to remove by hemodialysis; thus it is a more reliable biomarker of oxidative stress in end-stage renal disease (ESRD) patients [42].

## 2. Conclusions

Exposure to 3-MCPD and mutagens produced by processing contamination of edible oils and foodstuffs during thermal processing exerts carcinogenic or mutagenic effects on human and animal health. The mechanism of action of these chemicals is mainly through DNA damage, which causes mutagenic effects such as chromosomal aberrations, point mutations, frameshift mutation, and cellular transformations, thereby altering the genetic information. In the long run, these changes get translated into various pathologies such as tumor and cancer of various vital organs, thereby posing great threat to the health of consumers. The current review article discussed briefly the current knowledge regarding the critical health risks associated with 3-MCPD and mutagens produced during processing contamination of edible oils. The data reported here provide new insights in relation to the processing contamination in edible oils to stimulate further studies on the mitigation of these chemical contaminants.

## Conflict of Interest

The author declares no conflict of interest.

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