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Review on the chemical changes in the vegetable oils release harmful carcinogens upon deep fry or reused

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Abstract
The objective of the present review is on Chemical Changes In the vegetable oils release harmful carcinogens upon deep fry or reused. Depending on what it is you're cooking, refined oils can be reheated the following number of times: For breaded or battered foods – three to four times. For clean frying, such as making chips or crisps – eight times, although you could reuse it far more than that if you replenish it with fresh oil after the eighth time. Vegetable oils, as used in foods, are comprised of complex mixtures of triacylglycerols (TAGs; usually > 95%) with some minor amounts of diacylglycerols (usually < 5%). Other minor components are tocopherols/tocotrienols (up to 900 mg kg^{-1}) and phytosterol esters/phytosterols (up to 1%). Reusing cooking oil in food preparation, especially during deep-frying, is a common practice to save costs. Repeated heating of the oil accelerates oxidative degradation of lipids, forming hazardous reactive oxygen species and depleting the natural antioxidant contents of the cooking oil. Repeatedly heated cooking oils can generate varieties of compounds, including polycyclic aromatic hydrocarbons, some of which have been reported as carcinogenic. Heating for prolonged periods as well as exposure to air/oxidation are known to decrease the nutritive value of oils. Thermal decomposition of flavanols and poly phenols has been reported after heating of oil at 220 °C for 100 min and on storage at 25 °C for 2 weeks.

Keywords: Vegetable oils, reused**,** heating process, harmful carcinogens**,** policy loopholes

1. Introduction
Oil's chemical constituents dictate its fragrance, taste, and therapeutic properties, and a single oil can have many different uses, thanks to its complex chemical profile. Understanding the chemistry can therefore help you to get the most out of your essential oils and give you a better understanding of which oils to use and any safety concerns. A typical essential oil contains several hundred individual chemicals, with the great majority at levels of less than 1%. The chemical compounds in an essential oil typically have hydrogen, carbon and oxygen as their building blocks and can be divided into two main categories:- Hydrocarbons and Oxygenated compounds. Hydrocarbons contain only carbon and hydrogen. This group is made up almost exclusively of terpenes - monoterpenes and sesquiterpenes. From hydrocarbons a plant can make the second main group – oxygenated compounds. A plant makes oxygenated compounds from hydrocarbons - mainly esters, aldehydes, ketones, alcohols, phenols, and oxides. Some oils such as Pine are dominated by hydrocarbons whereas others such as Clove comprise mainly of oxygenated compounds. It is the oxygenate constituents that typically determine the oil's aroma and taste. They also give them some solubility in water and considerable solubility in alcohol. Sometimes essential oils obtained from botanically identical plants can have a significantly different set of chemical components and therapeutic actions. This can be due to different growing conditions, location, climate etc. [1].

Consumption of repeatedly heated cooking oil has been a regular practice without knowing the harmful effects of use. The present study is based on the hypothesis that, heating of edible oils to their boiling points results in the formation of free radicals that cause oxidative stress and induce damage at the cellular and molecular levels [2]. Polycyclic aromatic hydrocarbons (PAHs) are a large class of organic compounds that are produced through the incomplete combustion or pyrolysis of organic matter and are persistent, bio-accumulative, carcinogenic and mutagenic [3].

Food business operators (FBOs) are supposed to discard vegetable oils after four times of frying or when its total polar compound (TPC) levels reach 25, say the Food Safety and Standards Authority of India (FSSAI) rules. TPCs are associated with diseases such as atherosclerosis, hypertension, liver diseases, and Alzheimer's, FSSAI says. The oils tend to be used repeatedly to reduce expenses. The heating of oils to their boiling points repeatedly results in the formation of free reactive oxygen (free radicals) which is responsible for oxidative stress causing elevated levels of glucose, creatinine, and cholesterol in the body, studies say. Repeated frying also alters Poly Unsaturated Fatty Acids (PUFA) molecules present in the oil resulting in the formation of oxidized monomers, dimmers, and polymers which further break down into toxic Malondialdehyde which is linked to coronary heart disease and cancers. The present review is focused on the Chemical Changes in the vegetable oils release harmful carcinogens upon deep fry or reused.

2. Heating Process of Vegetable Oil

Lipid peroxidation is thought to be an important factor in the pathophysiology of a number of diseases and in the process of ageing, but its measurement *in vivo* has been difficult. The aim of this thesis was to evaluate methods for measurement of lipid peroxidation *in vivo* that are suitable for clinical investigations, and to apply these methods in animal and human studies investigating basal conditions and situations associated with increased lipid peroxidation.

Cooking oil is exposed to an extremely high temperature in the presence of air and moisture during the frying process, under such conditions, a complex series of chemical reactions takes place, resulting in loss of both quality and nutritional values of the cooking oil. Repeatedly heating the cooking oils initiates a series of chemical reactions, modifying the fat constituents of cooking oil through oxidation, hydrolysis, polymerization, and isomerization, eventually resulting in lipid peroxidation [4]. Lipid peroxidation generates a wide spectrum of volatile or non-volatile components, including free fatty acids, alcohols, aldehydes, ketones, hydrocarbons, trans-isomers, cyclic and epoxy compounds [5-6]. As a result, when the same cooking oil is reused excessively, the chemical reactions enhance foaming, darkening of oil color, increased viscosity, and off-flavor. Hence, repeated heating of the oil can lead to degradation of the cooking oil, both chemically and physically.

Although the chemical reactions provoked by thermal treatment are complex, they interact with and affect each other. Exposure to oxygen at high temperatures leads to oxidation of triacylglycerides, which generates hydroperoxides. Hydroperoxides are unstable intermediates and rapidly break down into reactive free radicals to initiate autoxidation, generally through a three-phase process (initiation, propagation and termination). Autoxidation is therefore suggested to be a principal mechanism of lipid peroxidation. The extreme heat during frying is the main initiator for autoxidation, in addition to other factors such as photonic agents, ionizing radiation, free radicals and chemical impacts. The initiation phase involves homolytic cleavage of hydrogen bonds, particularly those in the a-position relative to the double bond of the fatty acid chain, to form alkyl radicals ($L_1\cdot$; Reaction 1).

$$[L_1\cdot H \rightarrow L_1\cdot\cdot + H\cdot] \quad (1)$$

$L_1\cdot\cdot$ radicals are highly unstable intermediates. They stabilize themselves by reacting with oxygen to generate peroxyradicals ($L_1\cdot OO\cdot$; Reaction 2).

$$[L_1\cdot + {}^3O_2 \rightarrow L_1OO\cdot] \quad (2)$$

The resulting peroxyradical then abstracts a hydrogen from other unsaturated fatty acid (L_2H) to form a hydroperoxide (L_1OOH) and another alkyl radical ($L_2\cdot$; reaction 3), thus replenishing the reaction (1). This phase is called propagation. It propagates sustainably at a high rate.

$$[L_1OO\cdot + L_2H \rightarrow L_1OOH + L_2\cdot] \quad (3)$$

The propagation phases continue until a maximum concentration of hydroperoxide is reached, at which time point the collision between the individual moieties becomes more frequent. This stage marks the onset of the termination phase, in which the double bond adjacent to the hydroperoxyl group is broken down to yield hydrocarbons, aldehydes, alcohols and ketones (reaction 4).

$$[L_2OO\cdot + L_3OO\cdot \ {}_1 \text{ Non-radical compounds}] \quad (4)$$

Hydrolysis, another key pathway of lipid peroxidation, is initiated by water vapor found in food and the atmosphere. Activated water molecules break down the esterified bonds of triacylglycerides to generate glycerol, free fatty acids, monoacylglycerides and diacylglycerides. The breakdown products in turn accelerate the hydrolysis rate. At the same time, high temperatures induce polymerization of the hydrolysis products to form high-molecular weight cyclic fatty acid monomers, dimers or oligomers, which subsequently speeds up the hydrolytic reaction.

Superoxide radicals ($O_2^{\bullet-}$), hydrogen peroxide (H_2O_2), hydroxyl radicals (•OH), and singlet oxygen ($^1O_2$) are commonly defined reactive oxygen species (ROS); they are generated as metabolic by-products by biological systems. Processes, like protein phosphorylation, activation of several transcriptional factors, apoptosis, immunity, and differentiation, are all dependent on a proper ROS production and presence inside cells that need to be kept at a low level [7]. When ROS production increases, they start showing harmful effects on important cellular structures like proteins, lipids, and nucleic acids [8]. A large body of evidences shows that oxidative stress can be responsible, with different degrees of importance, in the onset and/or progression of several diseases (i.e., cancer, diabetes, metabolic disorders, atherosclerosis, and cardiovascular diseases) [9].

ROS are mainly produced by mitochondria, during both physiological and pathological conditions, that is, $O_2^{\bullet-}$ can be formed by cellular respiration, by lipoxygenases (LOX) and cyclooxygenases (COX) during the arachidonic acid metabolism, and by endothelial and inflammatory cells [6]. Despite the fact that these organelles have an intrinsic ROS scavenging capacity [27], it is worth to note that this is not enough to address the cellular need to clear the amount of ROS produced by mitochondria [11].

Cells deploy an antioxidant defensive system based mainly on enzymatic components, such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), to protect themselves from ROS-induced cellular damage [12].

3. Non-enzymatic lipid peroxidation

Reusing cooking oil in food preparation, especially during deep-frying, is a common practice to save costs. Repeated heating of the oil accelerates oxidative degradation of Lipids, forming hazardous reactive oxygen species and depleting the natural antioxidant contents of the cooking oil. Lipid peroxidation – a free radical fingerprinting method. Lipid peroxidation is probably the most extensively investigated free radical- induced process [13]. One of the earliest descriptions of the different stages of lipid peroxidation was given in the late 1820s by de Saussure, who used a simple mercury manometer to study the uptake of oxygen by a layer of walnut oil on water (reviewed in [14]. Polyunsaturated fatty acids (PUFAs) are particularly susceptible to peroxidation and once the process is initiated, it proceeds as a free radical-mediated chain reaction involving initiation, propagation and termination [15]. The lipid peroxidation chain reaction Initiation of lipid peroxidation is caused by attack of any species that has sufficient reactivity to abstract a hydrogen atom from a methylene group upon a PUFA [16-19]. (Fig. 1). Since a hydrogen atom in principle is a free radical with a single unpaired electron, its removal leaves behind an unpaired electron on the carbon atom to which it was originally attached. The carbon-centred radical isstabilised by a molecular rearrangement to form a conjugated diene, followed by reaction with oxygen to give a peroxyl radical. Peroxyl radicals are capable of abstracting a hydrogen atom from another adjacent fatty acid side-chain to form a lipid hydroperoxide, but can also combine with each other or attack membrane proteins. When the peroxyl radical abstracts a hydrogen atom from a fatty acid, the new carbon-centred radical can react with oxygen to form another peroxyl radical, and so the propagation of the chain reaction of lipid peroxidation can continue. Hence, a single substrate radical may result in conversion of multiple fatty acid side chains into lipid hydroperoxides. The length of the propagation chain before termination depends on several factors e.g. the oxygen concentration and the amount of chain-breaking antioxidants present. Hydroperoxides are fairly stable molecules, but their decomposition can be stimulated by high temperatures or by exposure to transition metal ions (iron andcopper ions). Decomposition of hydroperoxides generates a complex mixture of secondary lipid peroxidation products such as hydrocarbon gases (e.g. ethane and penthane) and aldehydes (e.g. malondialdehyde (MDA) and 4-hydroxynonenal [20].

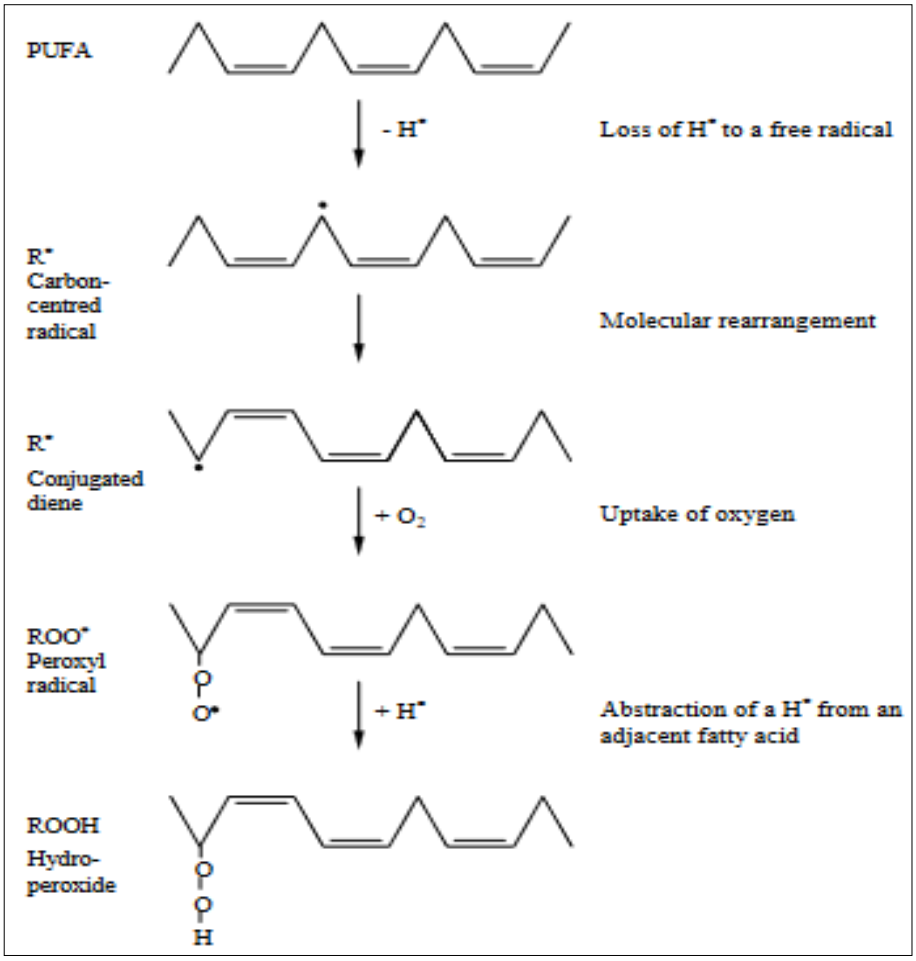

Fig 1: Mechanism of non-enzymatic lipid peroxidation

4. Types of Chemical Reactions

When vegetable oil is heated for a long time in the open air at high temperature, various chemical reactions (hydrolysis, oxidation and polymerization) occurs. As a result of reactions, cooking oil produce volatile components which result into degradation the oil.

5. Hydrolysis

Hydrolysis of vegetable oils and fats is an endothermic reaction (Fig 2). The extent of hydrolysis increases with an increase in temperature. Additionally, the miscibility of water in lipid increases at high temperatures and pressures, thereby enhancing the rate of the hydrolysis reaction [21].

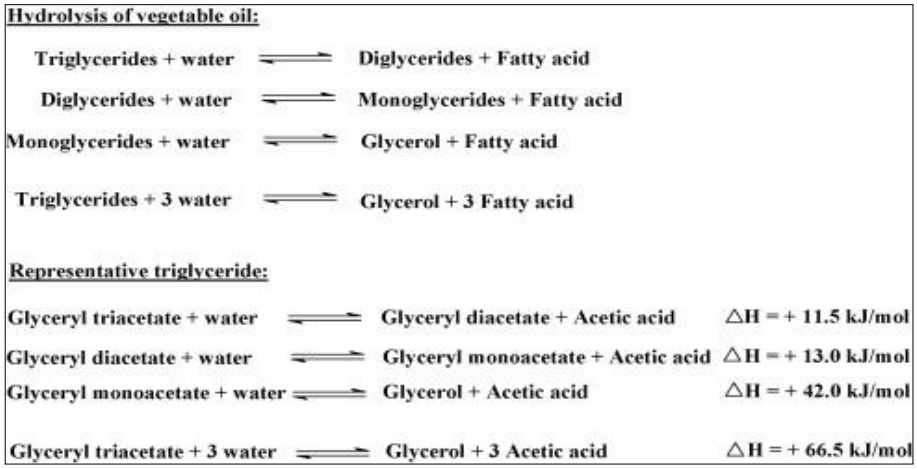

Fig 2: Hydrolysis of vegetable oil

6. Oxidation

When cooking oil is exposed to heat, light and oxygen, it can oxidize. Oxidized oils can produce harmful compounds and toxic by-products and make your food taste bad (Fig 3). The more an oil can resist reacting with oxygen and breaking down, the better for cooking. Oxidation is an undesirable series of chemical reactions in oil that degrades its quality and value. Some oils naturally oxidize faster than others due to their composition of fatty acids and antioxidant. When cooking oil is exposed to heat, light and oxygen, it can oxidize. Oxidized oils can produce harmful compounds and toxic by-products and make your food taste bad. The more an oil can resist reacting with oxygen and breaking down, the better for cooking. This quality is measured as *oxidative stability* and is considered by many oil experts as the best predictor of how an oil performs during cooking. Oxidative stability is measured via induction time. This is the point when an oil breaks down and potentially produces harmful compounds. A greater induction time indicates an oil is more resistant to oxidation, a shorter induction time means the oil will oxidize easily [22].

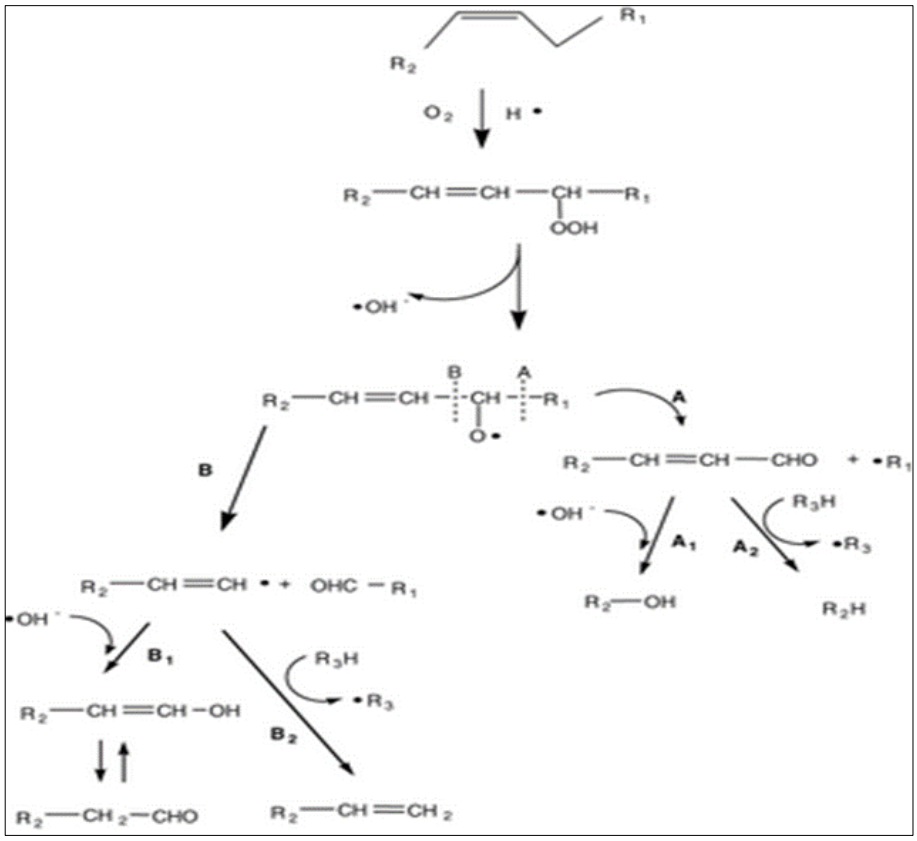

Fig 3: Mechanisms and Factors for Edible Oil Oxidation

7. Influences Oxidative Stability of an Oil

There are three major factors that determine an Oil's stability.

A). Number of Antioxidants

As the name suggests, antioxidants protect against oxidation, which is the undesirable process that occurs

quicker when an oil is exposed to high heat for extended periods. Therefore, antioxidants play a key role in an oil's oxidative stability and health benefits. It's important to consider which oils contain and retain their naturally high antioxidant levels.

B). Type and Ratio of Fats

A fat is more resistant to heat if it contains fewer double bonds between its molecules. Double bonds oxidize and break down easier when exposed to heat.

- **Saturated fats (SFA):** These do not contain any double bonds and are solid at room temperature.
- **Monounsaturated fats (MUFA):** These contain only one double bond but are also stable at high temperatures.
- **Polyunsaturated fats (PUFA):** These have two or more double bonds, which makes them prone to oxidation [23].

C). Polymerization of used oils

Polymerization occurs during frying, producing a wide variety of chemical reactions that result in the formation of compounds with high molecular weight and polarity (Fig 5). Polymers can form from free radicals or triglycerides by the Diels–Alder reaction. Viscous gel-like species form in vegetable oils during frying. After fatty acid formation, thermal oxidation of unsaturated chains causes polymerization. Heat treatment of oleic acid was employed to produce and identify the initial polymerization products. Spectroscopic studies revealed that heating oleic acid (210 °C, open to air) causes the formation of oligomers cross-linked by ester groups. The absence of esters, before thermal treatment, facilitated observation of cross-links (ester groups) that propagate to produce heavier insoluble products [24-25].

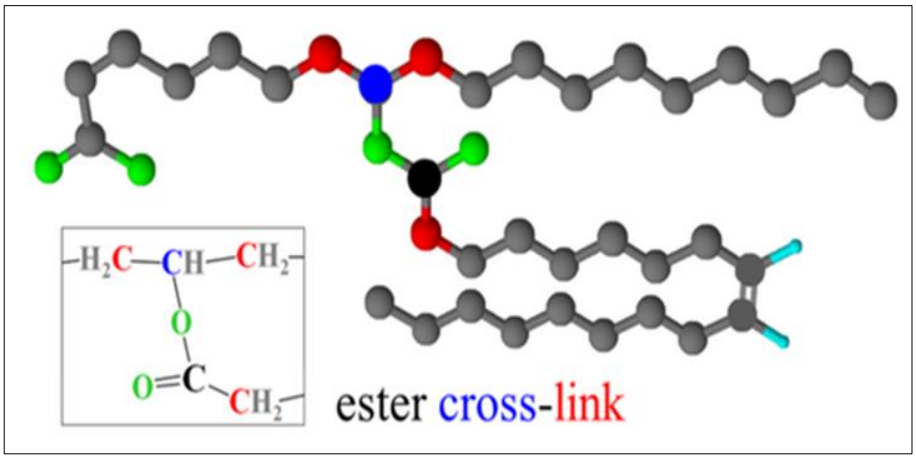

Fig 5: Viscous gel-like species form in vegetable oils during frying

Effect of polymerization on quality of oil decreases the unsaturated fatty acids of oil as well as increases foaming, color, viscosity, density, specific heat, as well as contents of free fatty acids, polar materials, and polymeric compounds [26].

8. Conclusions

1. Long-term intake of diet comprising reheated vegetable oil leads to endothelial dysfunction. Repeatedly heated dietary vegetable oil promotes oxidative stress, resulting in NO inactivation and reduced bioavailability. Moreover, antioxidant effect of fresh vegetable oil against free radicals may be reduced gradually as the oil is repeatedly heated. Production of free radicals and reduction of antioxidant and vitamin levels eventually lead to oxidative stress. Oxidative stress and endothelial dysfunction play pivotal roles in the pathogenesis of cardiovascular diseases, which may be controlled by diet modification. Ingestion of repeatedly heated vegetable oil should be restricted due to the detrimental consequences on health
2. Oxidative stress and free radicals are generally known to be detrimental to human health. A large amount of studies demonstrates that in fact free radicals contribute to initiation and progression of several pathologies, ranging from CVD to cancer. We can reach to the conclusion that oxidative stress, as phenomenon, although being one of the major harms to individuals' wellness and health, it can also be exploited as a treatment tool when and if we will be able to operate a fine tuning of this process inside human organism.
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4. Policy Loopholes, the FSSAI rules apply to FBOs that consume 50 liters or more vegetable oil per day for frying. Hotels often record less than 50 liters of being consumed to bypass the rules. Consumers are unaware of health risks and authorities are ignorant. Hence, hotels are using the same oil 10-14 times. Public health sector is suffering due to a lack of political will."

9. References

1. Tisserand R, Balacs T. Essential Oil Safety, a Guide For Health Care Professionals. New York: Churchill Livingstone; c1995.
2. Rekhadevi PV, Rajagopal S. Evaluation of the deleterious health effects of consumption of repeatedly heated vegetable oil. Toxicology Reports. 2016;3:636-643.
3. Alexander C. Pollution history of the Savannah River estuary and comparisons with Baltic Sea pollution history, Limnologica; c1999.

4. Sodergren E. Lipid peroxidation *in vivo*. Evaluation and application of methods for measurement. Acta Universitatis Upsaliensis. Comprehensive Summaries of Uppsala Dissertations from the Faculty of Medicine. 2000;949:78. Uppsala. ISBN 91-554-4791-0.
5. Choe E, Min DB. Chemistry of deep-fat frying oils. J Food Sci. 2007;72:R77-86.
6. Kaviyani M, Ghazi NM, Shariati MA, Atarod S. The study of frying oils properties. Int. J Adv Eng Res Tech. 2014;2:90-96.
7. Zhang Q, Saleh AS, Chen J, Shen Q. Chemical alterations taken place during deep-fat frying based on certain reaction products: A review. Chem Phys Lipids. 2012;165:662-681.
8. Sato H, Shibata H, Shimizu T, Shibata S, Toriumi H, Ebine T. Differential cellular localization of antioxidant enzymes in the trigeminal ganglion, Neuroscience. 2013;248:345-358.
9. Navarro-Yepes J, Zavala-Flores L, Anandhan A, Wang F, Skotak M, Chandra N. Antioxidant gene therapy against neuronal cell death, Pharmacology & Therapeutics. 2014;142:206-230.
10. Edori OS, Odoemelam SA. Concentration, source apportionment and ring size analysis of polycyclic Aromatic Hydrocarbons (PAHs) in water from Kolo Creek, Niger Delta, Nigeria. Int. J Adv. Chem. Res. 2022;4(1):36-41.
DOI: 10.33545/26646781.2022.v4.i1a.45
11. Wu QJ, Kosten TR, Zhang XY. Free radicals, antioxidant defense system, and schizophrenia, Progress in Neuro-Psychopharmacology & Biological Psychiatry. 2013;46:200-206.
12. Taniyama Y, Griendling KK. Reactive oxygen species in the vasculature, Hypertension. 2003;42:1075-1081.
13. Al-Gubory KH, Garrel C, Faure P, Sugino N. Roles of antioxidant enzymes in corpus luteum rescue from reactive oxygen species-induced oxidative stress, Reproductive Biomedicine Online. 2012;25:551-560.
14. Hansen JM, Go YM, Jones DP. Nuclear and mitochondrial compartmentation of oxidative stress and redox signalling, Annual Review of Pharmacology and Toxicology. 2006;46:215-234.
15. Glasauer A, Chandel NS. Targeting antioxidants for cancer therapy, Biochemical Pharmacology. 2014;92:90-101.
16. Deponte M. Glutathione catalysis and the reaction mechanism of glutathione-dependent enzymes, Biochimica et Biophysica Acta; c2013. p. 3217-3266, 1830.
17. Halliwell B, Gutteridge JMC. Free radicals in biology and medicine. 3rd ed. Oxford: Oxford University Press; c1999.
18. Gutteridge JM, Halliwell B. The measurement and mechanism of lipid peroxidation in biological systems. Trends Biochem Sci. 1990;15:129-135.
19. Halliwell B, Gutteridge JM. Role of free radicals and catalytic metal ions in human disease: an overview. Methods Enzymol. 1990;186:1-85.
20. Halliwell B, Chirico S. Lipid peroxidation: its mechanism, measurement, and significance. Am J Clin Nutr. 1993;57:715S-724S.
21. Gutteridge JM. Lipid peroxidation and antioxidants as biomarkers of tissue damage. Clin Chem. 1995;41:1819-1828.
22. Moore K, Roberts LJ II. Measurement of lipid peroxidation. Free Radic Res. 1998;28:659-671.
23. De Zwart LL, Meerman JH, Commandeur JN, Vermeulen NP. Biomarkers of free radical damage applications in experimental animals and in humans. Free Radic Biol Med. 1999;26:202-226.
24. Martinez-Yusta A, Goicoechea E, Guillen MD. A review of thermo-oxidative degradation of food lipids studied by ^{1}H NMR spectroscopy: Influence of degradative conditions and food lipid nature. Comprehensive Reviews in Food Science and Food Safety. 2014;13:838-859.
25. Evuen UF, Apiamu A, Ugbeni OC. Toxicological potentials of repeated frying on antioxidant status of vegetable oils. Int J Eng Res Tech; c2013.
26. Gordon MH, Kourisma L. The effects of antioxidants on changes in oils during heating and deep frying. J Sci. Food Agric. 1995;68:347-353.
27. Rajendran P, Nandakumar N, Rengarajan T, Palaniswami R, Gnanadhas EN, Lakshminarasaiah U. Antioxidants and human diseases, Clinica Chimica Acta. 2014;436:332-347.