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Review Article

Know Your Cooking Oil

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Abstract

Most of the cooking oils are vegetable oil. They are very rich in Polyunsaturated Fatty Acids (PUFA), omega-6 (n-3) and omega-3 (n-3) in the ratio of 16-20:1 against the recommended requirement of 1-4:1 and they lack their natural anti-oxidants. Therefore they are very prone to oxidation even at room temperature. Heating and reheating during cooking process enhances the oxidation, hydrolysis and polymerization of lipids and produce varieties of free radicals which creates oxidative stress in the body after consumption. N-6 fatty acids derivatives are pro-inflammatory while n-3 derivatives are anti-inflammatory. Since n-6: n-3 ratio is higher in the oils, there exists a dominance of inflammatory mechanisms over anti-inflammatory mechanisms that results into inflammation. Oxidative stress and inflammation together contribute to the pathogenesis of many diseases like Rheumatoid Arthritis (RA), cancer, Coronary Artery Disease (CAD), diabetes, Non Alcoholic Fatty Liver Disease (NAFLD) etc. Therefore, it has been suggested that fast food and repeated heating of cooking oil at home, must be avoided and n-3 fatty acids must be supplemented in the diet to fight the inflammatory effects of n-6PUFA.

Introduction

The cooking oils from plant are triglycerides, usually liquid at room temperature, extracted from plant using hexane and subjected to heat and chemical treatments for refining. Most of the oils are usually very rich in PUFAs; n-6 and n-3 in the ratio of 16-20:1 against the recommended requirement of 1-4:1 [1,2], results into inflammation [3,4]. Because of possession of double bonds, PUFAs are also susceptible to oxidation, which is enhanced during cooking process by heat. Consumption of such oil along with food generates oxidative stress in the body. Thus the two problems of oil consumption; inflammation and oxidative stress conclude into various physiological disorders.

Therefore, the objective of this mini review is to discuss inflammation and oxidative stress in terms of oil consumption and health consequences for awareness of common people.

Inflammation, Oxidative stress and cooking oil

Inflammation is the normal physiological phenomena against infection and injury to get rid of invaders and damaged tissue with the aid of leukocytes. Leukocytes involved in the process get activated by certain factors and synthesize cytokines; tumor necrosis factor-(TNF- α); interleukins (IL) like IL-1, IL-6 and IL-8; 2 series prostaglandins (PGE₂), leukotriene (LTB₄) and other mediators [5]. Anti-inflammatory cytokines such as IL-10 and receptor antagonist IL-1 [6] oppose inflammation. PGE₂, PGI₂ and LTB₄ are generated by n-6 PUFA [6] and can be further increased by consuming Arachidonic Acid (ARA) or Linoleic Acid (LA) in the diet [7]. Anti-inflammatory, leukotriene LTB₅ and resolves; RVE1 and RVD are generated by n-3 PUFA [8]. Metabolism products of these fatty acids are important mediators of many physiological processes. Since their metabolism use the same rate limiting enzymes, they compete for their metabolizing enzymes. Because of high concentration of n-6 in the diet, enzymes involved show a preference for n-6 than n-3 and thus there persist a dominance of inflammatory reactions over anti-inflammatory producing inflammation [9].

Oxidative stress arises as a result of an imbalance between reactive oxygen species and antioxidant defenses. Reactive oxygen species reacts with macromolecules and culminates into per-oxidative chain reaction leading to structural damage and disease. Short term oxidative stress is employed by the body to get rid of damaged tissue by trauma, heat, excessive exercise and to kill pathogens via inflammation. Free radicals are formed continuously in mitochondria during respiratory chain reactions, archidonate pathways, and cytochrome P-450 system and also derived from various external sources like exposure to X-rays, cigarette smoke, air pollutants in the air, pesticides, antibiotics and analgesics [10]. Antioxidant system reduces surplus free radicals and keeps the body at normal pace. Thus production of free radicals and their reduction by anti-oxidants is natural phenomena in the biological system. Sometimes, oxidative stress surpasses this balance, as in the scenario stated above, leading to damage of lipid, protein and ultimately the DNA molecule [11]. Cooking oils rich in PUFA contributes enormously to oxidative stress in various ways. PUFAs, especially ARA and LA, are primary targets for free radical and singlet oxygen oxidations that results into oxidative stress [12,13]. As stated above, cooking oil rich in n-6, contributes to inflammation which is always accompanied by oxidative stress [14]. During frying process, oil undergoes oxidation, hydrolysis and polymerization producing variety of free radicals and destruction of

anti-oxidants present in it. Consumption of such oil loaded with free radicals generates oxidative stress [15-17] and concludes into various physiological disorders.

Obesity is an important factor contributing to inflammation and oxidative stress and n-6 PUFAs have been reported to induce obesity [18]. 2-arachidonoylglycerol (2-AG) is produced after the hydrolysis of ARA, is a predominant ligand of cannabinoid receptors of brain that stimulates food intake and lipogenesis crowning to obesity [19].

2-AG levels were found to be high in mice fed on safflower oil; high in LA but deficient in α linolenic acid (LNA) and reduced in rats supplemented with DHA rich fish oil [20]. SREBP-1c is a transcription factor required for insulin mediated synthesis of triglycerides and fatty acids through the activation of fatty acid synthase [21]. On the other hand, transcription factor PPAR α exerts hypolipidemic effects through activation of genes encoding for lipid oxidizing enzymes in skeletal muscles, cardiac muscles and liver. N-3 PUFA derivatives, EPA (Eicosapentaenoic acid), and DHA activate PPAR α and suppresses the expression of SREBP-1c and thus inhibits the gene expression for lipid synthesis [4]. Diet rich in n-6 fails to activate PPAR α and inhibit SREBP-1c. This situation favors fatty acids and triglycerides synthesis over fatty acid oxidation and development of obesity and oxidative stress because of triglycerides induced oxidative stress by inhibiting enzymatic anti-oxidants [4].

Health Consequences

It is not possible to include all the disorders related to inflammation and oxidative stress in this short review but some are listed here;

Rheumatoid Arthritis (RA) - RA is caused by the inflammation of joint and surrounding tissue. Most frequent eicosanoids, PGI₂ [22], PGE₂ and LTB₄ [6] are found in the synovial fluid of patients with RA.

NAFLD is characterized by accumulation of fat in the liver and impaired bioavailability of liver n-6 and n-3 PUFAs. PUFAs are more susceptible to oxidation and diet rich in high n-6 PUFA and low n-3 PUFA lead to depletion of n-3 PUFA and oxidation of n-6 PUFA leading to the production of pro-inflammatory eicosanoid derivatives that lead to the development of NAFLD [1,23].

CAD is caused by atherosclerosis, a chronic low-grade inflammatory disease of the vessel wall. Usually endothelium releases and maintain a balance between pro and anti-inflammatory molecules but during atherosclerosis, production of pro-inflammatory cytokines; IL-1, 2, 6 and TNF- α , surpass the production of anti-inflammatory molecules leading to the progression of atherosclerosis [24].

Blood Pressure-Oxidative stress generated by oxidation of PUFA leads to increased vascular activity and reduction of vascular regulatory nitric oxide that causes the pathogenesis of blood pressure also [25,26].

Diabetes- Obesity induces oxidative stress and Inflammation which culminates into LTB₄ induced insulin resistance followed by type 2 diabetes. High levels of inflammatory mediators TNF- α and IL-6 also indicates involvement of inflammation in the pathogenesis of type 2 diabetes [27].

Cancer- Inflammation and oxidative stress are one of the contributing factors of carcinogenesis [28,29]. N-6 PUFA derivative,

PGE₂ has been reported to increase the methylation and hence the suppression of tumor suppressor genes by increasing the expression of DNA methyl transferases during colorectal cancer and tumor growth in ApcMin/+ mice [30,31]. PGE₂ has also been linked with breast cancer through its capacity to increase mRNA expression of aromatase enzyme which converts androgens to estrogen, resulting in estrogen biosynthesis, a key driver of estrogen-receptor positive breast cancer. EPA, LAA derived or from the diet, reduces the level of PGE₂ and LTB₄ and thus renders protection by synthesizing anti-inflammatory eicosanoids, PGE₃ and LTB₅ [32,33].

Conclusion

It has been suggested from the review above that fast food and repeated heating of cooking oil at home, must be avoided and n-3 fatty acids must be supplemented in the diet to compensate the inflammatory effects of n-6PUFA.

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