EuroSciCon

World Congress on
Nutrition and Dietetics

June 18-19, 2018
Paris, France

J Clin Nutr Diet 2018 Volume: 4
DOI: 10.4172/2472-1921-C1-003

POLYUNSATURATED FATTY ACID OXIDATION IS PRODUCING CONCERNS GLOBALLY

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Eicosapentaenoic acid (EPA) and Docosahexaenoic acid (DHA) both are the ω-3 polyunsaturated fatty acids having preventive role in the outbreak of chronic disorders. Presently its preventive role is questioned by masses due to its oxidation during storage, processing, cooking and even after ingestion. The resultant oxidative compounds not only affect the quality of the product but also its nutritional value. Chronic disease including cardiovascular diseases, cancer, obesity and diabetes are increasing alarmingly round the globe. These chronic disorders are the major causes of increasing public health burden globally. As per FAO and WHO recommendations, the adults need to take 0.25-2g EPA+DHA per day. American Heart Association recommends daily intake of 0.5-1 g EPA+DHA per day per adult. Oxidation of polyunsaturated fatty acid is either free medical mediated or mediated by ultraviolet/singlet oxygen. Enzymes involved in its oxidation are cyclooxygenases, lipoxygenases and cytochromes P450. Non enzymatic oxidation is mediated by free radical producing hydroperoxide and further broken down into ketones and melanoaldehydes. Fish and plant oils are major source of polyunsaturated fatty acids. Oxidation of polyunsaturated fatty acid produces off flavor due to aldehyde and ketone formation during storage, processing and even after ingestion. Temperature and light is major contributor of poly unsaturated fatty acid oxidation. Lipid oxidation decreases nutritional value of the final produce by the destruction of essential fatty acids and fat soluble vitamins A, D, E, and K as well as decrease caloric value. Oxidation of polyunsaturated fats results in 4-Hydroxy-2-nonenal, a product and it has been found in many diseases including atherosclerosis, neurodegenerative diseases and cancer. Oxidized PUFAs (rich in HNE and HHE) induced oxidative stress and inflammation in mice and in human intestinal Caco-2/TC7 cells. D-series resolvin and protectin/neuroprotectin inhibit neutrophil infiltration into injured kidneys, block toll-like receptor-mediated inflammatory activation of macrophages and mitigate renal dysfunctions.

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