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Review Article

Correlation between fast food consumption and lipid profiles

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ABSTRACT

Abnormalities in lipid profile, known as dyslipidemia, refer to elevated levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), in addition to low levels of high-density lipoprotein cholesterol (HDL-C) in the blood. Dyslipidemia is widely spread among the population, and this rise in dyslipidemia is attributed to changes in dietary habits and shifts towards fast food predominated diet. The widespread consumption of fast food is linked to adverse health impacts, including rise in obesity, diabetes, and cardiovascular diseases. Fast food is characterized by large portion size and unusually high energy density. It contains higher levels of sugars, fats, and salt, in addition to suboptimal levels of dietary fibers and micronutrients. Moreover, due to the heavy processing and packaging of fast food, it usually contains harmful compounds and by-products that further exacerbate the undesirable health impacts of fast-food diet. This narrative review aims to outline current knowledge regarding the association between the rise in fast food consumption and lipid profile abnormalities, including how the poor nutritional composition of fast food contributes to fluctuations in TC, TG, LDL-C and HDL-C levels in the blood and development of dyslipidemia.

Keywords: Fast-food, Dyslipidemia, Lipid profile, Total cholesterol, Low-density lipoprotein cholesterol, Triglycerides, High-density lipoprotein cholesterol

INTRODUCTION

Consumption of fast food has risen globally, and this rise is linked to various problems including negative health outcomes such as incline in obesity, diabetes, and cardiovascular diseases, prevalence of malnutrition and vitamin deficiency, unsustainable agricultural practices, and environmental pollution.[1-4] Studies report a rising preference towards Western fast food, such as fried chicken, French fries, pizza, and burgers, over local food varieties.

Key factors that contribute to this increase in fast food consumption are convenience, taste, affordable cost, and accessibility, along with social influence from peers and family. Other factors include socioeconomic status, age, gender, education, employment, and health consciousness.[5]

Lipid profile abnormalities are frequently observed among fast food consumers. These abnormalities are known as dyslipidemia, and they include elevated levels of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), and triglycerides (TG), in addition to low levels of high-density lipoprotein cholesterol (HDL-C) in the blood. Dyslipidemia is linked to several clinical conditions such as type 2 diabetes and cardiovascular diseases.[6,7] Dyslipidemia is considered a common disease, with global prevalence of 38.4% for low HDL-C, 28.8% for hypertriglyceridemia, 24.1% for hypercholesterolemia, and 18.93% for high LDL-C.

Differences in dyslipidemia levels are also observed according to gender, country, year, and socioeconomic status. The rise in dyslipidemia prevalence is attributed to changes in dietary habits and shifts towards fast food predominated diet.[8]

Fast food has several characteristics that contribute to its undesirable effects on health. Fast food is characterized by a large serving size and a very high energy density. Moreover, it usually contains higher levels of sugars and processed carbohydrates, saturated and trans-fatty acids, cholesterol, and salt, in addition to diminished levels of micronutrients and dietary fibers.[9] Furthermore, due to the heavy packaging and processing of fast food, it often contains harmful chemicals that have adverse health effects.[10,11] In this narrative review, we aim to outline current evidence regarding the association between the rise in fast food consumption and dyslipidemia, including how the poor nutritional composition of fast food contributes to fluctuations in TC, TG, LDL-C and HDL-C levels in the blood.

METHODS

This narrative review is based on an extensive literature search conducted on 10 November 2025 in PubMed, Cochrane, and Web of Science databases using medical subject headings (MeSH) and relevant keywords. The search aimed to identify studies examining current knowledge regarding the correlation between fast food consumption and abnormalities in lipid profiles. The review focused on articles that address poor nutritional composition and harmful chemicals in fast food and their association with fluctuations in TC, TG, LDL-C, and HDL-C blood levels. No restrictions were applied regarding publication date, language, or type of publication, to ensure a broad and comprehensive investigation of the available literature.

DISCUSSION

High energy density and low nutritional value of fast food

A typical fast-food meal often contains twice the energy density of a recommended healthy diet, leading to passive over-consumption.[12,13] This leads to a substantial increase in caloric intake that exceeds daily energy requirements, consequently resulting in weight gain and obesity.[14]

Several studies reported the association between carbohydrate intake and lipid levels in the blood. Various indicators are used to quantify the carbohydrate content in food, such as glycemic index (GI) and glycemic load (GL). GI is a measurement of the blood glucose raising potential of carbohydrate content of food in comparison to glucose as a reference. It has been shown that foods with similar carbohydrate content do not have the same effect on blood glucose levels due to variation in GI of these foods. While GL comprises both the amount of carbohydrate in the food and the GI of the food, i.e. how each gram of carbohydrate raises blood glucose levels.[15] High dietary GL was reported to be associated with low levels of HDL-C in children and adolescents.[16,17] Moreover, high dietary GI and GL were both associated with increased levels of TC and TG.[18]

The relationship between high GI/GL food and dyslipidemia is attributed to the impaired physiological responses after meals. After a high GI/GL meal intake, the early postprandial period, which is within the first 2 hours, is characterized by hyperglycemia and hyperinsulinemia. This is followed by drop in blood glucose levels to hypoglycemic range. Meanwhile in the late postprandial period, which is 4 to 6 hours after the meal, gluconeogenic and glycogenolytic pathways are activated by counterregulatory hormones (cortisol, glucagon, and growth hormone) in order to restore euglycemia, which leads to an increase in free fatty acid levels in the blood, contributing to lipid profile abnormalities as a response to the high GL diet.[19,20] Moreover, high GL food consumption causes decreased lipoprotein lipase activity, specifically in the late postprandial period, which leads to decreased clearance of triglycerides, consequently resulting in increased LDL levels, decreased HDL levels, and increased LDL-to-HDL ratios. These events eventually lead to hypercholesterolemia and dyslipidemia.[21]

Fast food is characterized by high fat composition. Saturated fats have been known to lead to elevated levels of LDL-C, and they have been linked to cardiovascular risks. This effect depends on the length of the fatty acid chain. Saturated fatty acids (SFAs) of shorter length have been shown to have a greater influence over raising LDL-C levels, such that lauric acid (12-carbon) raised LDL-C the most, followed by myristic (14-carbon) and palmitic (16-carbon) acids.[22] Fast food also contains trans-fatty acids (TFAs), which are unsaturated fatty acids that can be either naturally occurring or artificially produced, and they contain at least one double bond in the trans configuration. Artificial TFAs are widely used in food manufacturing despite their adverse health effects. A study revealed that higher TFA intake was associated with increasing likelihood of dyslipidemia manifested as hypercholesterolemia and increase in LDL-C levels among adults. Moreover, the association was stronger when higher TFA intake was combined with higher saturated fat intake.[23]

Fast food consumption is associated with lower intake of fruits, vegetables, whole grains, and fiber.[24] Dietary fiber is an essential component for controlling elevated cholesterol levels in the blood. A meta-analysis study investigated the beneficial effects of dietary fiber intake on lipid profiles. Results revealed that higher intake of dietary fiber was associated with significant decrease in TC and LDL-C levels, in addition to reducing inflammation and blood pressure.[25] The main mechanism of cholesterol reduction in blood by fiber comprises binding to bile acids, which are made from cholesterol, and decreasing their

reabsorption in the small intestine and promoting their excretion, consequently resulting in a decrease in circulating cholesterol levels.[26] Another mechanism involves reduced glycemic response, which leads to lower insulin production, and therefore, reduced cholesterol biosynthesis in the liver. Moreover, fermentation of dietary fiber in the intestines by gut microbiota results in release of short chain fatty acids, which play a role in inhibiting hepatic cholesterol biosynthesis.[27]

Presence of harmful chemicals in fast food

Fast food is heavily packaged, and this leads to leakage of harmful chemicals such as phthalates and bisphenol A (BPA). Fast food consumption has been linked to phthalates and BPA exposures.[10] Phthalates are widely used as plasticizers in food packaging materials. They have endocrine disrupting properties, and they are linked to numerous adverse effects, such as neurotoxicity, infertility, growth reduction, allergy, obesity, and insulin resistance. BPA is used in the lining material of canned foods and beverages as well as thermal receipts. It also has endocrine disrupting properties, and it is associated with several health risks, such as obesity, type 2 diabetes, impaired metabolism, cardiovascular diseases, and cancer.[28] The association between dyslipidemia and phthalates and BPA exposure has been reported. Studies revealed that accumulation of phthalates can lead to elevated TG and reduced HDL-C levels in children and adolescents, while long-term exposure to BPA was associated with increase in LDL-C and TG levels and decrease in HDL-C levels in middle-aged and elderly people.[29-30]

Furthermore, heat processing of certain fast foods such as French fries, chicken nuggets, and burger leads to generation of harmful products, including acrylamide, advanced glycation end products, furan and its derivatives, and heterocyclic amines.[11] Acrylamide is a well-known food contaminant that is generated from the Maillard reaction between amino acids, especially asparagine, and reducing sugars such as glucose. This reaction is common in heat processing of food, including baking and frying processes. Acrylamide gets metabolized into glycidamide. These two compounds, acrylamide and glycidamide, are classified as carcinogens and neurotoxins and have been linked to various poor health outcomes.[31-33] Acrylamide exposure has been reported to increase the risk of cardiovascular diseases in adults, by mediating oxidative stress and inflammation. Moreover, it was associated with elevated LDL-C and TC levels.[34] Glycidamide has been reported to be more relevant to dyslipidemia. Long-term glycidamide exposure was found to be associated with elevated TC levels but not associated with other serum lipids in adolescents.[35] Another study revealed association between glycidamide exposure and increase in TC, LDL-C, and TG levels, in addition to decrease in HDL-C levels in adults.[36]

Advanced glycation end products (AGEs) are a group of complex and heterogeneous compounds that include brown and fluorescent cross-linking compounds such as pentosidine, non-fluorescent cross-linking compounds such as methylglyoxal-lysine dimers, and non-fluorescent, non-cross-linking compounds such as carboxymethyllysine and pyrraline.[37] They are formed from the nonenzymatic glycation of proteins, lipids, and nucleic acids during the Maillard reaction. As a result, this glycation alters these macromolecules' functions. AGEs contribute to dyslipidemia through mediating inflammation and oxidative stress and glycation of low-density lipoproteins (LDL). LDL glycation is considered a predisposition of atherosclerosis.[38] A study reported the association between AGEs and increased levels of TC and LDL-C in type 2 diabetic patients, with stronger association observed in female subjects compared with males.[39] Another study revealed the atherogenic potential of AGEs in diabetic patients where AGEs exacerbated dyslipidemia in these patients and increased their cardiovascular risk. AGEs mediated the glycation of small, dense LDL particles, converting them to glycated LDLs that are even more atherogenic.[40]

Furan (C4H4O) and its derivatives, 2-methylfuran, 3-methylfuran and 2,5-dimethylfuran, collectively known as methylfurans, are volatile, toxic compounds that cause liver damage and exhibit possible carcinogenic action.[41] These compounds are formed mostly during heat processing of foods. Several mechanisms exist that result in furan generation, including thermal degradation and/or thermal rearrangement of carbohydrates in presence of amino acids, which happens during the Maillard reaction, thermal degradation of certain amino acids, oxidation of ascorbic acid at higher temperatures, and oxidation of polyunsaturated fatty acids and carotenoids.[42]

Effect of dietary furan exposure on lipid profile abnormalities has been investigated in animal models. Findings of one study indicated elevated TC, TG, and LDL-C levels in mice due to high furan intake.[43] Another study revealed an increase in LDL due to dietary furan exposure, in addition to liver and kidney damage in the affected mice.[44]

Heterocyclic amines (HCAs) are compounds that are formed when meat is cooked at high temperatures such as grilling, smoking, roasting, and frying. The precursors for HCAs formation are creatine, creatinine, and other free amino acids found in meat. HCAs are considered mutagens and carcinogens.[45] They have been linked to insulin resistance and type 2 diabetes, however, their effect on lipid profile and their link to cardiovascular diseases is still understudied. A recent study investigated the effect of two common HCAs, MeIQx (2-amino-3,8-dimethylimidazo [4,5-f]quinoxaline) and PhIP (2-amino-1-methyl-6-phenylimidazo [4,5-b]pyridine), on lipid homeostasis in cryopreserved human liver cells. Findings revealed disruption of lipid homeostasis that involved increases in lipid droplets and TG levels in the cells. Moreover, upregulation of key genes involved in lipid synthesis, transport and metabolism was reported. These results

demonstrate the contribution of HCAs in dysregulating lipid production, transport, and storage, leading to fat accumulation in hepatocytes. This area should be further investigated to detect serum lipid abnormalities in response to dietary intake of HCAs.[46]

Future perspectives for public health

The rise in dyslipidemia and its related health risks is tightly linked to fast food consumption. Addressing this global burden requires multi-faceted public health interventions. Dietary education programs, particularly for children and adolescents, are needed to highlight the risks of fast-food consumption and encourage healthier choices. Childhood and adolescence are considered critical periods for developing healthy eating habits with lifelong health outcomes.[47]

Moreover, governments could consider policies such as taxation on unhealthy foods and nutrition labelling of the fast-food products, along with strict restrictions on fast-food marketing directed at children. Furthermore, public and governmental efforts are required to improve the accessibility and affordability of nutritious foods to make healthy eating a viable option for all populations.[48-50]

The food environment heavily impacts healthy food choices. This environment is affected by the availability and cost of healthy food items, in addition to the number of stores in a region. Reduced access to fresh produce and healthy food options greatly affects food choices for low-income populations.[51] Government policies to improve food environment are important for fighting against dyslipidemia and associated health risks and to promote sustainable and healthy nutrition, especially in low and middle-income communities. Such efforts include increasing access to healthy and nutritious food options, applying zoning laws to limit unhealthy foods, and improving healthy food service standards, in addition to increasing the awareness for behavioral changes like reducing food waste and choosing plant-based options more often.[52] Another important aspect is combining dietary changes with promoting physical activity and healthy lifestyle to mitigate the risks of obesity and metabolic disorders.[53]

CONCLUSION

Fast food consumption is associated with poor diet quality resulting in dyslipidemia. Fast food is characterized by high energy intake, high levels of processed carbohydrates and fats, and low fiber intake, in addition to containing harmful chemicals that result from packaging and processing. All these factors contribute to abnormalities in lipid profile, including elevated levels of TC, TG, LDL-C and lower levels of HDL-C. Dyslipidemia is strongly linked to obesity, insulin resistance, and cardiovascular diseases. This raises the need for public health awareness of the detrimental effects of the fast-food diet.

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