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Review Article

Dietary patterns, inflammation, and dyslipidemia: A review of evidence and implications

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Abstract

Dyslipidemia refers to elevated levels of LDL (bad) cholesterol and triglycerides, accompanied by low HDL-cholesterol in the body which is one of the most significant modifiable contraindications for cardiovascular illness, and the leading cause of death worldwide. Inflammation plays a key role in the pathophysiological process of cardiovascular disease among dyslipidemic people. Numerous systematic reviews have revealed a link among biological markers of inflammation, including C-reactive protein (CRP), interleukin (IL)-1β, IL-6, tumor necrosis factor-alpha (TNF-α), IL-4, and IL-10 and cardiometabolic disease as well as proinflammatory diet impact on inflammation. Several studies have found that preventing and managing dyslipidemia may considerably decrease fatality and morbidness caused by atherosclerosis and cardiac ischemia. The three major strategies to cure dyslipidemia are to improve one's lifestyle, eat a healthier diet, and use medications of these three, a healthy dietary habit is the most important lifestyle component that influences an inflammatory response. The inclusion of various foodstuffs plus healthy eating habits may exert a positive impact on wellness due to the anti-inflammatory qualities they possess. The purpose of the present review article is to assess the overall results obtained from current articles whose research has explored the relationship between dyslipidemia with Dietary Inflammatory Index (DII) as well as its correlation with an elevated risk of cardiovascular disease. The dietary inflammatory index score is significant in the relationship between inflammation and cardiovascular disease among Dyslipidemia patients and is thoroughly evaluated, particularly in the context of healthy dietary habits. In conclusion, our analysis suggests that the Dietary Inflammatory Index (DII) tool appears as an effective scoring algorithm for assessing the proinflammatory effect of an eating regimen and helps to understand complex relationships among dietary habits, inflammation, and risk of cardiovascular disease among dyslipidemia individuals.

Keywords cardiovascular disease, dietary inflammatory index, inflammation

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Introduction

Dyslipidemia is a significant risk factor for CHD (coronary heart disease) and stroke [1]. CHD is one of several leading causes of fatalities among the Indian population [2]. High cholesterol is the major cause of atherosclerosis, which is the primary health risk associated with CHD [3]. Dyslipidemia is described by the altered composition of the fats in the blood [4], which includes elevated levels of total cholesterol, triglycerides, low-density lipoprotein (LDL) cholesterol, as well as lower levels of high-density lipoprotein (HDL) cholesterol, and is widely recognized as being a risk factor for

cardiovascular disease [5]. As stated by the World Health Assembly (WHO), Cardiovascular illness (CVD) is the most prevalent cause of death internationally [6]. Elevated levels of total cholesterol and increased incidence of hypertension along with poor eating habits and being sedentary increase the likelihood of all causes underlying CVD [7].

One of the main causes and effects of atherosclerosis is inflammation. There is significant variability activates biological effectors and tissue context within the larger category of inflammation. Since LDL-Cholesterol containing ApoB is the causative factor for atherosclerotic inflammation and cardiovascular disease development, so lipid-related inflammation is important [8]. And an unhealthy diet contributes significantly to several NCDs. In 2017, an improper diet accounted for 11 million deaths and 255 DALYs. A poor diet contributes significantly to the pathophysiology of obesity, diabetes, and cardiovascular disease, owing in part to low-grade chronic inflammation [9]. While preventing ASCVD is crucial, dyslipidemia is linked to many clinical problems that necessitate preventative treatments. The link between acute pancreatitis development and elevated levels of triglycerides (TG >10 mmol/L or >885 mg/dL) is most clinically significant. Pancreatic autodigestion and inflammation may result from aberrant lipolysis caused by mislocalized exocrine pancreatic lipase, albeit the underlying pathophysiology is unknown [10].

Over the past couple of centuries, the volume of investigation addressing the relationship between inflammation and disease has increased dramatically. The focus on how diet affects inflammation has increased in tandem with this rise, with the body of research on the subject doubling roughly for every 4 years once and the DII's purpose was to provide a scientific method for assessing the relationship between diet and medical consequences, which include blood levels of inflammatory cytokines or long-term disease [11]. To evaluate the possibility of inflammation in the body caused by diet, the tool Dietary Inflammatory Index (DII) was created and DII is the nutrient-based tool. It was predicted that "more nutritious" dietary habits would be connected to anti-inflammatory DII scores. Chronic, systemic inflammation is significantly moderated by diet [12], for example, the Mediterranean diet, a well-known healthy eating pattern, has anti-inflammatory effects [13], and the Western food patterns with a high fat content and energy dense foods can greatly heighten inflammation in the body [14]. Thus, having an integrated approach, such as the DII, is vital for assessing the inflammatory effect of a diet. Although the prevalence of dyslipidemia varies by region, it is believed that over 50% of adults worldwide suffer from the condition. There are reports on the increased incidence of elevated levels of bad cholesterol and reduced levels of good cholesterol, hypertriglyceridemia, and hypercholesterolemia [15].

Prevalence

The data highlights significant trends and patterns from 2017 to 2023, offering insight into altered levels of lipids in the blood is one of the major risk factors for atherosclerosis. A study of population-based data across India reveals rising average TC (total cholesterol) levels. According to the latest research, 25-30% of urban and 15-20% of rural persons have high cholesterol. The incidence is lower than in nations with high incomes. The most typical dyslipidemias in India include borderline excessive levels of LDL, inadequate HDL, and elevated Triglycerides. According to study findings, total cholesterol, LDL cholesterol, and triglycerides have been reported to be high in the urban population in recent years, and the prevalence of CHD events is strongly correlated with elevated levels of Apo-B, total cholesterol (TC), and LDL cholesterol. In contrast, Apo-A and HDL cholesterol levels are typically protective, reducing the risk of CHD events. Despite its significance, there is a paucity of research on lipid problems in children, and hypercholesterolemia remains poorly understood, undertreated, and inadequately controlled in India [16], there is another study published in 2023, which involved a randomly selected cohort of 2976 participants from diverse geographical locations, including rural, suburban, and urban areas in Tamil Nadu, India, with a distribution of 29.1% from rural areas (865), 34.6% from suburban areas (1030), and 36.3% from urban areas (1081). Lipid profiles varied significantly across urban, suburban, and rural areas, with suburban women showing high rates of poor HDL cholesterol (approximately 80%) and urban areas exhibiting higher prevalence rates of hypercholesterolemia, hypertriglyceridemia, and elevated LDL-C compared to suburban

and rural populations. The same study revealed a significant gender-age interaction in the development of dyslipidemia, where - Males were more likely to develop dyslipidemia than females before the age of 40 However, after the age of 40, the risk of dyslipidemia shifted, and females were more likely to develop dyslipidemia than males. Apart from decreased HDL levels other factors such as sex, age, the habit of smoking and drinking, uncontrolled body weight, DM, and HTN will also have a strong association with altered lipid profile [17].

Causes of dyslipidemia

The primary secondary causes of dyslipidemia have been given in Table 1 **[18].**

Table 1. Most common cause of dyslipidemia

Primary causes		Secondary causes	
⇧ Bad cholesterol	⇩ Good cholesterol	⇧ Bad cholesterol	⇩ Good cholesterol
Deficient LDLreceptor	Deficiency of Apo A-1	Obesity	Metabolic syndrome
Familial homozygous	Mutation of Apo A-1	High consumption of fat	Diabetes mellitus
hyperlipidemia	Deficiency of LCAT (partial or complete)	Hypothyroidism	Overweight
	Tangler's disease	Diabetes mellitus	Lack of Physical activity
	Familial hypoalphalipoproteinemia	Nephrotic syndrome	Smoking/Tabaco
		Medication	Patients with Beta blocker therapy
		Obstructive Hepatobiliary disease	Low fat consumption or more dependent on polyunsaturated fatty acids and steroids

Screening of dyslipidemia

Dyslipidemia screening has long been recommended as a component of community health approaches used to treat various other illnesses, such as diabetes. The major objectives of this strategy are to find as many impacted instances as possible, which will lessen the long-term effects of cholesterol (if early management is taken), and to prevent or postpone problems (such as cardiovascular events). Sadly, overall dyslipidemia screening rates are low (2.5% - 3.2%), even though 4.8% to 12.3% of the tested population had a diagnosis of dyslipidemia [19]. The standard lipid profile is the most commonly recommended screening procedure according to guidelines. It comprises total cholesterol, good cholesterol (HDL), bad cholesterol (LDL), and triglycerides [20]. Some guidelines, in addition to the usual lipid profile, mention the measurement of ApoB-100, which considers all lipoproteins that are categorized as atherogenic [10].

Diagnosis of dyslipidemia

- Dyslipidemia is typically asymptomatic and is discovered by chance or screening. However, in extreme situations, the patient may exhibit one of the symptoms of the consequences (either peripheral artery disease or coronary disease), including dizziness, palpitations, fainting, swelling of the lower limb, or enlargement of veins (such as those in the stomach or neck).
- The fasting lipid count, which is the standard biochemical test for evaluating hypercholesteremia, requires the individuals to fast for a minimum of 12 hours before to giving blood. The LDL-C concentration can be computed using the Fried Ewald equation, and the fasting lipid test measures the following key components of cholesterol including TC, TG, and HDL-C levels.
- When the TG levels are less than 400 mg/dL (4.5 mmol/L), this formula is employed. Still, the physician must request an LDL-C direct assay if the result is higher. Some clinicians prefer a non-fasting lipid profile as an essential test, which can provide an early assessment of lipid levels. Assuming the results show elevated value (triglycerides greater than four hundred milligrams per decilitre), a follow-up fasting lipid

profile is recommended later. For each of the four lipids, the precise values to consider are high, ideal, or low (in the case of HDL).
- To determine the best course of action for patients with borderline readings, the doctor must be aware of any co-risk factors. A lipoprotein study ought to be performed if the patient has low HDL and borderline high cholesterol levels. If the patient has two risk factors or high cholesterol, the analysis is also recommended. These risk factors include advanced age (over 45 for men and over 55 for women), early menopause (without estrogen replacement therapy), diabetes, smoking, hypertension, cerebral or peripheral blood vessel disease, and genetic history of CVD [19].

Dyslipidemia increases Inflammation which leads to cardiovascular disease

The pathophysiology of dyslipidemia is shown in Figure 1.
- Dyslipidemia contributes to atherosclerosis, which is the initial step in the development of cardiovascular disease. Dyslipidemia-related cholesterol buildup is oxidized, which quickens the explanation of the roles played by vascular selectin and inter-cell adhesion molecule-1 in adherence of monocyte, this leads to a sequential response of monocyte influx and subsequent release of cytokine, fuelling inflammation.
- Monocytes differentiate into macrophages and synthesize MCP-1 (Monocyte Chemoattractant protein-1), which enhances more monocytes, amplifying the inflammatory response. Furthermore, monocytes release pro-inflammatory cytokines, such as interleukin (IL)-6, which in turn produce oxidizing chemicals and accelerate lipid oxidation. After engulfing oxidized lipids, macrophages transform into foam cells, which accumulate and persist in the vascular walls, contributing to the development of atherosclerotic plaques and cardiovascular disease in this way.
-The extracellular matrix of atherosclerotic lesions is thought to be formed by the transport and retention of plasma low-density lipoprotein (LDL) through the endothelial cell layer the area below the endothelium.
- Once inside the artery wall, LDL undergoes nonenzymatic glycation and oxidation, which chemically alters it. The slightly oxidized LDL (soxLDL) acts as a chemotactic agent, attracting monocytes into the arterial walls.
- Afterward, monocytes convert into macrophages, leading to increased oxidation of LDL cholesterol. The repeated destruction and healing of the atherosclerotic plaque eventually results in the formation of a fibrous cap, which protects a complex mixture of calcium deposits, lipid accumulation, collagen fibers, and inflammatory cells, including T-lymphocytes.
- Preservation of the fibrous cap is crucial to maintain plaque stability and prevent its rupture, which can lead to catastrophic consequences, such as coronary thrombosis.
- Due to a strong relationship between cellular oxidative stress and increased formation of reactive oxygen species, ROS generation is a primary fundamental pathogenic process of atherosclerosis.
- To the stimulation of the vascular inflammatory response and endothelial dysfunction. Accelerated formation of ROS is closely tied to common cardiovascular risk factors, including smoking, diabetes, hypertension, and hypercholesterolemia, which collectively enhance ROS production and accelerate atherosclerotic progression.
- Atherosclerosis, an inflammatory condition affecting medium and large arteries, is significantly influenced by cytokines since cytokines are present throughout all stages of the disease. Pro-atherogenic cytokines, including interleukin (IL)-1, IL-6, and tumor necrosis factor-alpha (TNF-α), play a crucial role in the pathophysiology of atherosclerosis. These cytokines are produced by various cell types, including Natural killer cells, Vascular smooth muscle cells, Lymphocytes, and Macrophages. Triggering a cascade of events that includes cytokine production, adhesion molecule upregulation, and vascular cell activation, ultimately contributing to plaque development and progression [22].

The main causes of dyslipidemia are dietary, systemic, and genetic disorders. Dietary regimens that reduce inflammation, such as the Nordic, Mediterranean, and Dash diets, are thought to offer preventative benefits. Enhancing insulin resistance, changing the gut flora, lowering inflammatory responses in the mucosa and across the body, and influencing epigenetic connections like DNA methylation and acetylation are a few possible methods.

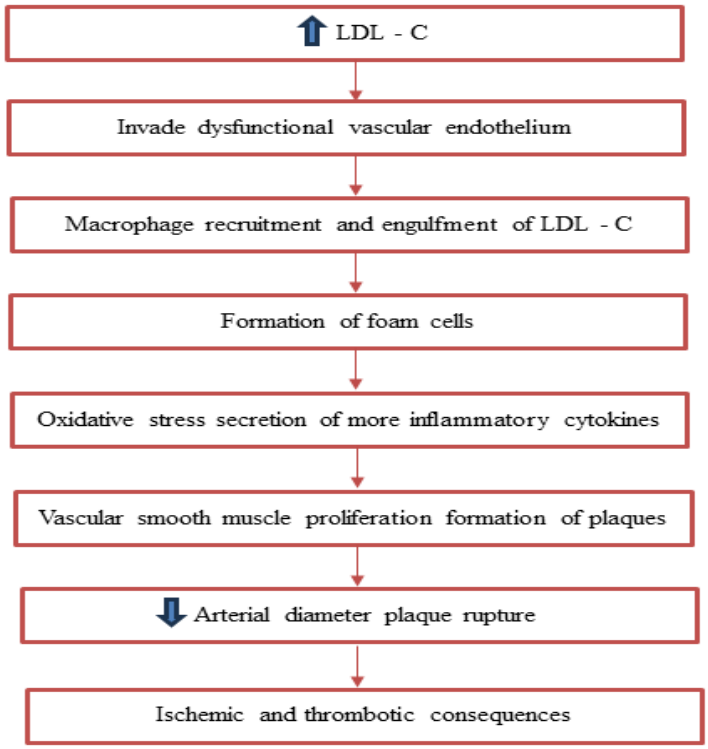

Figure 1. Pathophysiology of Dyslipidemia
(Adopted from Su et al., [21])

Another significant risk factor for dyslipidemia, which is known to be a chronic low-grade inflammatory condition, is obesity. A diet heavy in fat and sugar raises toxic substances and chronic inflammation, both of which accelerate the development of obesity [23]. Which increases the risk of cardiovascular disease. Cardiovascular diseases (CVDs) refer to a broad spectrum of disorders affecting the heart and circulatory system. Which is one of the major causes of death across the globe [24]. They include endocarditis, hypertensive disease, [25] atherosclerosis, arrhythmias, cardiomyopathy, aortic aneurysm, congenital heart diseases, hypertension, plaque, myocardial infarction, stroke, blockage of an artery in the lungs (pulmonary embolism), and reduced blood flow to the heart muscle (CHD) are among disorders of heart and blood vessels collectively referred to as cardiovascular diseases [26].

Dietary approach helps to reduce inflammation and improves lipid profile and cardiovascular health

Cardio-metabolic risk variables are significantly influenced by diet and exercise. Our diet and physical activity are regulated and modified easily by measured parameters such as weight of the body, blood pressure, visceral fat, blood lipid and glucose level, and more. As well as Inflammation, stress caused by oxidation, endothelial health and the microbiome of the gut are examples of complex interactions, most likely through hormonal and other influences [23]. Cardiovascular disease (CVD)-related morbidity and mortality can be reduced by controlling and preventing modifiable risk factors of CVD which include diabetes, obesity, high blood pressure, elevated cholesterol level, current smoking along with diet [27]. Increasing physical activity is unlikely to be effective on its own, but it might offer a slight supplement to the effects of dietary restriction. It should also be promoted to eat less fat, especially saturated fat. The

American Diabetes Association (ADA) suggests limiting cholesterol, saturated fat, and trans-fat in one's diet. Unrefined carbohydrates and omega-3, linoleic, or oleic fish oils can partially replace saturated fat in individuals who do not have a significant rise in serum triglycerides but are not obese [28]. Intake of fat is the main dietary factor that determines blood lipoprotein levels. In contrast, selecting good fats

(Omega-6, PUFA) such as those found in vegetable oils, nuts, and oil seeds lowers elevated cholesterol in the bloodstream. While limiting or avoiding saturated and trans-fats which elevates cholesterol levels. In controlled clinical trials, diets rich in these polyunsaturated fatty acids aid in lessening the incidence of CHD and they also decrease the ratio of total to HDL cholesterol [29].

Dietary inflammatory index (DII)

DII is an instrument that helps to determine an individual's diet as an anti-inflammatory or proinflammatory diet, to establish the person's level of inflammation induced by their eating behaviors, and serves to shed light on the connection between inflammation caused by diet and different metabolic illnesses [30]. DII was a population-centered dietary score created by Shivappa et al., [31], after reviewing the literature and doing a detailed analysis of 1943 papers released between 1950 and the end of 2010. To better understand the relationship between individual diet and inflammation as well as the health consequences of inflammation, the literature search was limited to papers that investigated the association of one or more of 45 dietary and nutritional factors with six previously-established biomarkers that indicate inflammation. TNF-α, CRP, IL1β, IL4, IL6, and IL10 are markers that help in comprehending the link underlying diet and inflammation, as well as the health effects of inflammation. The year 2009 witnessed the release of the DII's initial iteration. The publications linked not less than 1 out of six biomarkers of inflammation (TNF-α, IL1β, IL4, IL6, and IL10, and C – reactive protein) to any part of the diet. In contrast to the list of biomarkers for inflammation [11].

Calculation of DII

The DII is intended to be utilized with nutritional information gathered through various techniques of assessment. An "opulent" approach involves collecting information on all 45 food parameters, such as food records or interviews lasting a whole day (24-hour diet recall and FFQ). For comparative reasons, they present seven scenarios with Dietary inflammatory index scores computed using an array of probable consumption levels for each of the 45 food characteristics that pertaining the overall DII. Based on their ability to cause inflammation, these values span the 45 food factors that make up the DII ranging between the highest value to the 90th, 75th, median, 25th, and 10th lowest value. For instance, they added up the highest proinflammatory value of each food characteristic to determine the maximum positive inflammatory DII. Food intake measurements are utilized for estimating the DII; these data are subsequently combined with a globally represented regional dataset, which yields a precise estimation of the mean and standard deviation (SD) of every component. These are then used as multipliers to express an individual's exposure in terms of a Z-score relative to the "standard global mean." To do the aforementioned, deduct the reported amount from the "standard mean" and divide the final value by the standard deviation of that parameter. This value's "rights skewing" effect is reduced by converting it to a percentile score. To create a symmetrical distribution, every percentile score is doubled after which 1 is deducted. This transformation centers the values around 0 (null), ranging from -1 (maximally antiinflammatory) to +1 (maximally proinflammatory). To determine a "food parameter-specific DII score," multiply the centered percentile value for each food parameter by the associated "overall food parameter-specific inflammatory effect score." Eventually, the overall DII score" for a particular person is calculated by adding up individual "food parameter-specific DII scores [31]. The highest possible proinflammatory DII score of a diet is +7.98, while the highest possible antiinflammatory DII score is -8.87. Forty-five foods, substances, or nutrients are included in the DII score because they may cause inflammation in the diet. A pro-inflammatory diet has a high DII score, while one that is anti-inflammatory has a low score [32].

Evidence from earlier studies

Numerous studies have demonstrated that substantial elevation in systemic inflammatory biomarkers, such as TNF-α, CRP, IL1β, IL4, IL6, and IL10 are linked to greater DII scores, this indicates the food pattern's inflammatory potential. Previous studies have connected a higher DII score with an increased risk of metabolic disorders, cancer, CVD, and fatality [31]. The findings indicate that sufficient incorporation of

beneficial dietary habits, like the Mediterranean diet (plant-based and healthy fat), like fruits and veggies, or macronutrients/micronutrients, like fiber or vitamins C and E, are linked to lower degree of positive inflammatory biomarkers, primarily TNF-α, IL6, and CRP. Conversely, adhering to a modern dietary approach, along with consumption of food groups such as added sugars, macronutrients, and unhealthy fatty foods (saturated & trans-fat), is linked to an upsurge of the above-mentioned Inflammatory biomarkers [33]. Limited investigations have demonstrated a greater correlation between DII and high cholesterol (Dyslipidemia). Chen et al.'s study included a survival analysis in the dyslipidemia group to assess whether DII is linked to a higher probability of mortality from all causes or CVD-related deaths in those who had the condition [30]. Based on earlier research, they included covariables linked to dyslipidemia and DII. Gender, ethnicity, aging, schooling, body mass index, C-reactive protein (CRP), booze consumption, smoking history, elevated blood pressure, insulin resistance, coronary heart disease, and cerebrovascular accident were among the factors. The subjects in their study with dyslipidemia exhibited higher DII, BMI, and blood pressure. Additionally, those with dyslipidemia were older, with a higher CRP level as well and they were more likely to have comorbidities such as hypertension, diabetes mellitus, coronary artery disease, coronary heart disease, cerebrovascular illness, and cancer. Hence they proved Dyslipidemia and DII were positively and substantially correlated and the Low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, Total cholesterol, and Triglycerides are quietly notable. The DII Pro-inflammatory scores were greater in persons with dyslipidemia than with normal lipid profile [30].

Individuals with dyslipidemia stated that their Body mass index (BMI), Waist-hip ratio (WHR), Visceral adiposity index (VAI), Systolic blood pressure (SBP), and Diastolic blood pressure (DBP) were higher. Additionally, people with dyslipidemia had a greater likelihood of having higher CRI and CRI-II. This was demonstrated by another study and strongly associated with DII [34]. This investigation came from the Ravansar Non-Communicable Diseases (RaNCD) study, in which 51.9% of whom were men. Among those with dyslipidemia, the higher adherence to the DII score resulted in a substantial increase in the mean body weight and body mass index [35]. Also, another study revealed the correlation between DII and T2DM, obesity, along with heart and blood vessel diseases (CVD). It also proved the relationship between T2DM, obesity, and DII with cardiovascular disease and its risk factors, which include insulin resistance, insulin secretion, hypertension, and dyslipidemia [9]. The possibility of passing away was strongly as well as directly correlated with a more positive DII score in patients with hyperlipidemia was recently proven [36] and Lower HDL levels were associated with greater DII score [37]. The study's findings indicated a small connection between the DII value and HDL as well as TG elements of metabolic syndrome, indicating the role diet plays in controlling inflammation. Consequently, it is critical to shift a person's dietary pattern from one that is proinflammatory (higher DII scores) to one that is Mediterranean (lower DII scores), which has a lower propensity for inflammation and is high in antioxidants. An anti-inflammatory diet reduces the negative consequences of persistent inflammation and the cardiovascular risks that go along with it [38].

Dietary management

Principle of diet

For patients with high blood cholesterol levels, dietary management remains the primary line of treatment. A recommended dietary approach includes Low calories, low in saturated fat and trans-fat with 7% of total calories, and a diet high in PUFA and MUFA will be beneficial along with low carbohydrates, moderate protein, and high fiber diet with the increased number of antioxidants is recommended.

- To control lipid diseases, patients should have a balanced diet that includes limited additional sugars (< 10% kcal/d), cholesterol (< 200 mg/d), and SF (< 7% kcal/d). They should also abstain from alcohol. A variety of diets can meet the general principles that should include healthy eating behavior. For example, Low salt diet (DASH Diet) and Low-fat diet [39].
- Diet Plans follow lower levels of fat and carbs influence cholesterol readings. According to a meta-analysis, eating less fat had the greatest positive impact on blood levels of total cholesterol and low-density

lipoprotein cholesterol, while eating less carbohydrates had the best effects on blood levels of TG and HDL-C [40].

Protein- Dietary patterns must emphasize the intake of high-quality protein sources, including fish, shellfish, nuts, seeds, legumes, and lean animal proteins, while also emphasizing non-tropical plant oils over saturated fats.

Carbohydrate- Given that both high and low-carbohydrate diets are linked to increased mortality, the ingestion of carbohydrates (CHO) ought to vary from 45 to 55 percent of total energy intake [41].

Fat- The eating habits should restrict calories from saturated fats to < 7% and cholesterol should be < 200 mg/d of total calories [42], avoid trans-fat from highly processed food.

MUFA and PUFA- are advised to consume together makeup not higher than 10% and 15% of total calorie intake, respectively. Monounsaturated fats are those abundant within canola, almond, and groundnut oil, and seem to possess fewer negative impacts on HDL-C than polyunsaturated fatty acids. Fish, which is high in omega-3 fatty acids, can help decrease the level of triglycerides and have minimal effect on LDL cholesterol in individuals with normal triglyceride levels.

Fiber- Dietary recommendations for fiber intake range from 25 to 40 grams per day, with at least 7 to 13 grams of soluble fiber having a safe and beneficial impact on cholesterol regulation [43].

Alcohol- limiting or abstaining from drinking. The effects of alcohol on lipoprotein metabolism vary, partially according to dosage. People who consume one or two drinks a day have reduced serum triglycerides, but larger intakes negatively impact older men's LDL cholesterol levels and increase triglyceride levels.

Probiotics- Adding probiotics to the diet in the form of fermented milk products or capsules is beneficial in lowering the amount of TC level by 8.5 mg/dl and LDL-C levels by 6.5 mg/dl [44].

Conclusion

The present review demonstrates that chronic inflammation is a major cause of CVD-related deaths among individuals with dyslipidemia. Additionally, diet can significantly influence inflammation, either positively or negatively. The Dietary Inflammatory Index appears to be a valuable tool in assessing the inflammatory potential of a diet. Studies have shown that dyslipidemia sufferers with higher DII scores tend to have elevated inflammatory levels and an increased risk of mortality from cardiovascular disease and related causes, although further research is necessary to confirm these findings. The DII could also help identify the relationship between inflammation, diet, and CVD risk in individuals with dyslipidemia. Therefore, it is suggested that dyslipidemic patients should shift from a pro-inflammatory diet to a healthy, anti-inflammatory eating pattern, which may reduce inflammation and lower the risk of future cardiovascular disease.

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