

Review Article

Type 2 diabetes mellitus with dyslipidemia: risk factors, prevalence, pathophysiology, and nutritional management-a narrative review

Chandana M. S.¹, Savitha Vijaykumar², Pooja Anudhar G.^{1*}

¹Department of Nutrition and Dietetics, School of Life Sciences, JSS Academy of Higher Education and Research, Mysore, Karnataka, India

²Department of Medicine, JSS Medical College, JSS Academy of Higher Education and Research Mysore, Karnataka, India

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*Correspondence:

Dr. Pooja Anudhar G.,

E-mail: poojaanudharg@jssuni.edu.in

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ABSTRACT

Diabetic dyslipidemia is the most prevalent medical condition, which is associated with an increase in the risk of cardiovascular disease. In diabetic patients, Insulin resistance alters metabolic functions. It triggers the abnormalities of the lipid profile by elevating free fatty acid which is characterized by an increase in total cholesterol (TC), triglycerides, low level of high-density lipoprotein, and low level of low-density lipoprotein. Globally, 80-90% of diabetes patients have dyslipidemia mainly due to several factors like hyperglycemia, lifestyle, genetics, age, and history of other comorbidities. Management of the condition by incorporating a healthier lifestyle and dietary pattern also includes a calorie-deficit diet, low glycemic foods, fiber-rich foods, and unsaturated fatty acids foods. These nutrient-dense functional components reduce insulin resistance followed by free fatty acid flux. These changes can optimize the blood glucose and lipid profile levels in individuals. Hence, main purpose of review is to understand pathophysiology of diabetic dyslipidemia, risk factors, dietary modifications, and particular nutrient-dense functional foods to reduce the conditions. This study also includes some of the clinical studies with functional foods in managing dyslipidemia in diabetes. In conclusion says that incorporating nutrient-dense components along with healthy lifestyle modifications can reduce the risk of dyslipidemia and avoid the risk of cardiovascular diseases in diabetic individuals.

Keywords: Dyslipidemia, Insulin resistance, Metabolic function, Hyperglycemia, Nutrients

INTRODUCTION

Diabetes mellitus (DM) is an endocrine disease caused due to deficiency or insufficient secretion of insulin by the pancreas. It mainly results in an increase or decrease in glucose concentration in the body. DM is characterized as insulin-dependent DM (IDDM, type 1 DM) and non-insulin-dependent DM (NIDDM, type 2 DM). Type 1 DM mostly affects youngsters with a lack of insulin secretion and has an autoimmune basis. Typically, the most occurrence form is type 2 DM (T2DM), due to the beta cell dysfunction resulting in insulin deficiency and insulin resistance. This mechanism disturbs the

metabolism of protein, carbohydrates, and lipids.¹ This metabolic dysfunction affects various organs such as the eyes, kidneys, nerves, heart, and blood vessels which also leads to microvascular diseases like neuropathy and nephropathy.²⁻⁴ The majority of T2DM is associated with cardiovascular diseases, which are affected, mainly by insulin resistance, abdominal obesity, non-alcoholic fatty liver disease, hypertension, low-grade inflammation, and increased oxidative stress. These factors also promote atherogenic activity and cause dyslipidemia. Considering as dyslipidemia by abnormal levels of lipid profiles, mainly TC and high concentration of plasma triglyceride (TG). Other changes include a reduction of high-density

lipoprotein cholesterol (HDL-C) and an increase in low-density lipoprotein cholesterol (LDL-C).⁵ As insulin secretion and insulin action together cause dyslipidemia, patients with diabetes should be given priority for the treatment. Diabetic dyslipidemia includes not only the range of abnormal lipoprotein range but also its characteristics and abnormal metabolic rate which altogether contribute to the development of atherosclerotic complications. Glycated hemoglobin (HbA1c) has a positive correlation with TC, low LDL-C, and high HDL-C. Patients having >7.0% of HbA1c value have a higher value of TC and low level of HDL-C as compared to the patients with HbA1c $\leq$ 7.0%. HbA1c is the indicator of long-term blood glucose level which imitates the 2-3 months of glucose level. In addition, it can be used as a potential dual marker of glycemic control and dyslipidemia in T2DM as it is strongly associated with lipid profile abnormalities.⁶

DIABETES PREVALENCE

In 2021 international diabetes federation (IDF), assessed that approximately 537 million (9.8%) people are existing with diabetes across the world. This number will surge up to 643 million (10.8%) by 2030 followed by 783 million (11.2%) by 2045. IDF also proclaimed that the South East Asia region will be increased with diabetes population from 10% in 2021 to 11.3% in 2045. Across the world, China has the highest diabetes population which will increase from 140 million 10.6% to 170 million, 10.5% by 2045. India has the second highest diabetes population, 74 million or 9.6% of adults were living with diabetes in 2021 which is projected to be 125 million which is 10.8% in 2045. According to a study published in the Lancet diabetes and endocrinology, union territories and the states in India with the highest prevalence of diabetes are Goa (26.4%), Puducherry (26.3%), Kerala (25.5%) and Karnataka in 9th place.⁷

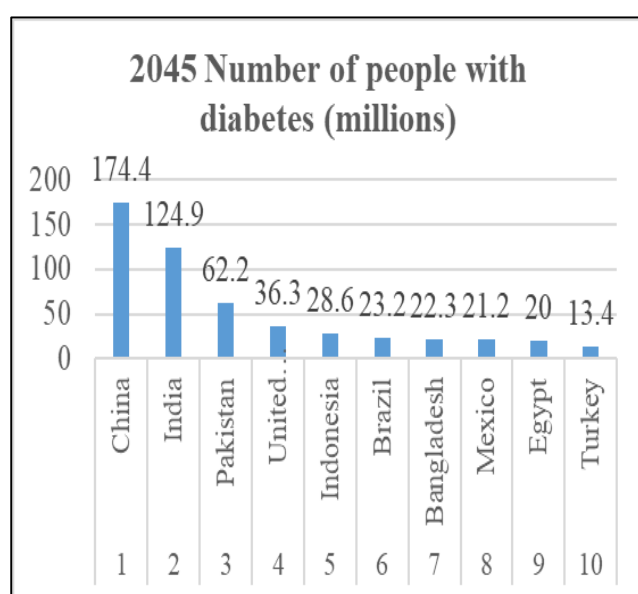


Figure 1: Diabetes prevalence.

In a study conducted by the Madras diabetes research foundation in collaboration with the Indian council of medical research (ICMR), approximately 10.6% of the population has diabetes in Karnataka.⁸ In a cross-sectional study, 2,806 individuals were screened in Karnataka and found that 30.18% were diagnosed with diabetes.⁹ Several research studies suggested that the diabetes prevalence rate is typically higher in urban areas (10.8%) than rural areas (7.2%).¹⁰ It is also more common in high-income countries (10.4%) than in low-income countries (4.0%).¹¹

PATHOPHYSIOLOGY OF T2DM

T2DM develops mainly due to beta cell dysfunction impaired insulin secretion or increased insulin resistance, these remain the primary pathophysiological characteristics of condition. Mainly glucose homeostasis process is controlled by hormones called glucagon and insulin secreted by the pancreas and nervous system activities and glucose uptake by the liver, muscle, and fat tissues. Neurons presented in the brain stem and hypothalamus recognize the hyper/hypoglycemia level and pass the signals to pancreatic cells through the nerve impulses to reduce the effects. This overall process directly affects the physiological and behavioral response of the human body system.

Beta cell dysfunction means beta cell death or apoptosis. Pancreatic beta cells the responsible for the secretion of insulin, but it doesn't recognise the high glucose content in the blood and fail to secrete sufficient insulin and increase the blood glucose level. This might be because of more complex network interactions between factors like environment and other metabolic pathways. Metabolic abnormalities like blood glucose level, high body mass index, and abnormal level of lipid profile affect the beta cells in the pancreas, with impaired secretion of insulin and other chronic inflammations. Inflammations like endoplasmic reticulum (ER) stress, metabolic stress, inflammatory stress, and amyloid stress reduce the efficiency of islets in the pancreas and affect insulin production. Less secretion of insulin causes, excess of free fatty acid, and high glucose levels in the bloodstream triggers the beta cell dysfunction by the activation of apoptotic unfolded protein responses (UPR) pathway by inducing ER stress. Lipotoxicity, glucotoxicity, and a combination of glucolipotoxicity in the bloodstream increases the metabolism and oxidative stress and leads to excessive UPR activation. Depositing a high amount of fat disrupts the ER calcium (Ca^{2+}) balance, leading to protein misfolding and the production of reactive oxygen species (ROS), which exacerbate inflammation. This stress triggers the UPR, impairing proinsulin processing and promoting the accumulation of misfolded proinsulin. Ultimately, this leads to beta cell apoptosis and results in a lack of insulin secretion.

Reduced metabolic response of insulin-responsive cells or desensitization to insulin are symptoms of insulin resistance. This indicates that when insulin fails to be

absorbed effectively by muscle, liver, and adipose tissue, blood glucose levels spike. The condition known as relative insulin resistance occurs when the body continues to produce insulin, but the insulin is not sufficient to meet bodily needs.¹² Moreover, β -cells may eventually stop secreting enough insulin, which would further impair glucose homeostasis. Insulin function may be impaired by hormonal imbalances, such as increased glucagon, cortisol, and adrenaline output. In insulin-resistant individuals, muscle cells struggle to absorb glucose, the liver continues producing glucose even when it is not needed, and fat cells fail to store triglycerides efficiently, leading to fat accumulation and increased free fatty acids in circulation. Normally, insulin regulates blood sugar in coordination with growth hormones after eating, while glucagon and other hormones maintain glucose levels during fasting. However, excessive hormone secretion can worsen insulin resistance. As IR progresses, persistent hyperglycemia places increased strain on β -cells, leading to their dysfunction and eventual failure.¹³

DYSLIPIDEMIA IN T2DM: GLOBAL PREVALENCE

Among T2DM patients, lipid profiles are widely varied due to many factors like genetic factors, associated diseases, lifestyle practices, pharmacological, and other factors that can affect the lipid values and vary the lipid profile. Dyslipidemia is more common among T2DM patients, there is a link between CVD and glucose, cholesterol, and triglyceride levels among them. There is a higher impact of lipid level abnormalities in diabetes patients than in the normal population. Dyslipidemia is characterized by an increase in TC, LDL C, TG, and high levels of HDL-C. The various studies highlighted that most T2DM patients are associated with dyslipidemia as there is a significant impact on HDL cholesterol levels. Due to the lack of insulin secretion, TG levels will be increased which promotes process of conversion of HDL into LDL and accelerates the excretion of HDL.^{14,15} LDL is a major risk factor for atherosclerotic CVD, including coronary artery disease. CVD is major cause of morbidity and mortality in both men and women with T2DM.¹⁶

Globally prevalence of dyslipidemia increasing $\geq 80\%$ to 90% in different countries. Studies in Jordan, Somali, South Africa, Northwest China, Thailand, South Korea, Nepal, Malawi, Saudi Arabia showed high dyslipidemia prevalence among T2DM patients.^{14,17-21} In India, more than 90% of patients with diabetes are estimated to have dyslipidemia associated with atherosclerosis. Dyslipidemia is mainly divided into two types: isolated dyslipidemia and combined dyslipidemia. A combined type of dyslipidemia is most common where LDL and TG are increased and HDL is decreased. Combined dyslipidemia was seen in 41.1% of males and 33.9% of females whereas isolated dyslipidemia was found in 26.0% of males and 29.5% of females. Presents of combined dyslipidemia among T2DM individuals lead to increases in morbidity and mortality because of

cardiovascular, cerebrovascular, and peripheral arterial diseases. The study, which was conducted in Western Cape, South Africa showed that 78% of male and 62% of female T2DM patients had low HDL this indicates that the most affected lipid profile component was HDL.^{22,14}

PATHOPHYSIOLOGY OF DIABETES WITH DYSLIPIDEMIA

Dyslipidemia in T2DM is very common, and its independent association with an increased risk of morbidity and mortality from CVD (cardiovascular diseases). Lipids are transported in the form of lipoprotein in the bloodstream and lipid abnormalities are not just quantitative which means measuring the particular lipoproteins (LDL and HDL) but also qualitative which measures the particle factors like density and size of the particles. The main quantitative lipid abnormalities of diabetic dyslipidemia are increased TG and reduced HDL-C which are categorized based on their density and are determined by their increased, chylomicrons (CM), VLDL, and IDL. Small, dense LDL particles are considered the most significant contributors to the development of CVD, and CM, VLDL, and IDL tend to be elevated in T2DM individuals. Insulin is an important hormone in lipid metabolism which is disrupted by increased insulin production and insulin action which may explain the connection between metabolic diseases like diabetes. Individuals who are insulin-resistant but do not have T2DM also exhibit the same lipid abnormalities. It is evident from the indication that the primary underlying abnormality causing dyslipidemia is insulin resistance. Hypertriglyceridemia, prolonged postprandial hyperlipidemia, increased levels of remnant particles (Remnants are cholesterol-rich particles that are small enough to penetrate the vessel wall, and thus, they are considered to be atherogenic lipoproteins), and decreased HDL-C levels due to an accelerated rate of HDL catabolism are quantitative abnormalities and increased VLDL particle size is the most common qualitative abnormality, which is potentially atherogenic.²³

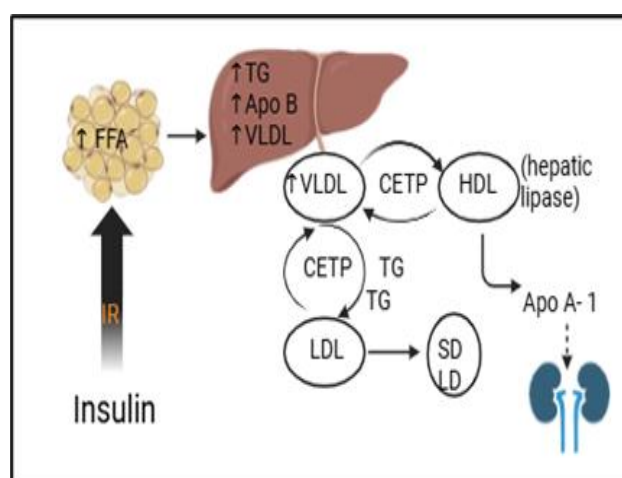


Figure 2: Pathophysiology of dyslipidaemia in insulin resistance.^{5,25}

Usually, insulin withholds the breakdown of triglycerides into free fatty acids (FFA) and glycerol from the adipose tissues by inhibiting the function of an enzyme called hormone-sensitive lipase. However, in T2DM patients, though insulin secretion increases, it reduces the ability to interact with the fat cells and causes excessive lipolysis. This leads to elevated FFA in the bloodstream and increased FFA influx into the liver. These FFA are also called non-esterified fatty acids (NEFA). Elevated levels of NEFA in the bloodstream increase hepatic triglyceride production and it is associated with increased secretion of apolipoprotein B (apoB) especially in insulin-resistant states. VLDL is an important lipoprotein which has responsible for transporting the triglyceride in the bloodstream than any other lipoproteins. It is synthesized by the liver; its production is encouraged by increased delivery of free fatty acids. When insulin levels are frequently raised, it makes the liver less responsive to the inhibitory effects of insulin on VLDL secretion hence when insulin levels are high, the secretion of VLDL becomes high. A high level of VLDL in blood plasma makes the protein called cholesteryl ester transfer protein (CETP) to exchange the triglycerides in VLDL to HDL and cholesterol from HDL to VLDL. This results in major lipid changes which are highly atherogenic, VLDL is rich in cholesterol, and low levels of HDL lose the ability to protect the heart conditions. Triglyceride-rich HDL particle breaks down itself and detaches the protein apolipoprotein A-I (apo A-I). Protein helps HDL to eradicate the excess cholesterol from the cells and transmit it to liver for excretion. However, dissociation of protein causes HDL to lose the cholesterol as well as reduce and weaken the particles. Insulin resistance leads to cholesterol-depleted and therefore smaller and denser LDL particles. The underlying etiology of small, dense LDL particles in insulin resistance is, again, hypertriglyceridemia, and the mechanism is similar to that which causes low HDL levels.²⁴

FACTORS AFFECTING DYSLIPIDEMIA IN DIABETES

Age and gender

All age groups are diagnosed with diabetes but it has become more prevalent in the elderly. Even dyslipidemia diabetes occurs at the age of 50 and above. This is due to many reasons such as chronic inflammation, oxidative stress, DNA damage, tissue dysfunction, mainly deficiency of insulin secretion, and growing insulin resistance followed by increased levels of TG and LDL. Early detection and diagnosis in old age often leads to a better outcome, with lower HbA1c and reducing the insulin. It also reduces the morbidity and mortality of individuals. Growing research highlights that gender differences, matter when it comes to many diseases' epidemiology, pathophysiology, management, and outcomes. In many studies, it was found that high prevalence of dyslipidemia among women with diabetes. Due to the increased level of adipose tissue in abdomen, it

releases a high amount of FFA. The heightened flux of FFAs to the liver contributes to dyslipidemia by promoting the production of VLDL and TG.²⁵

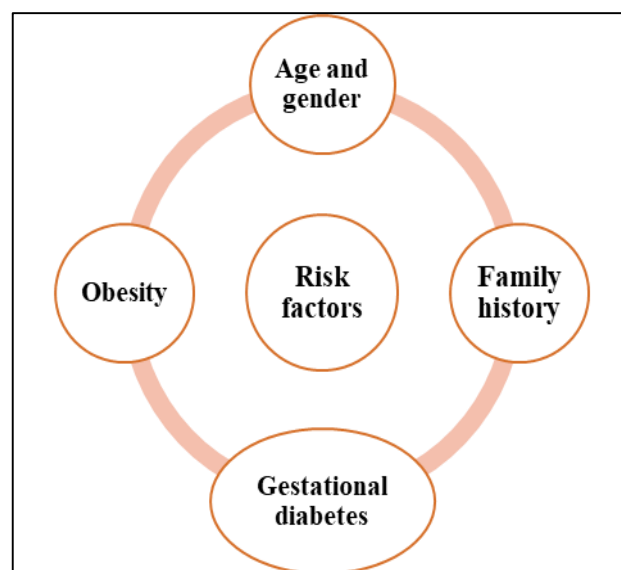


Figure 3: Risk factors of diabetes.

Family history

A family history is an independent risk factor for impaired fasting glucose (IFG) even when obesity is absent. It is common for seemingly healthy, non-obese youngsters with positive FHD to also have hyperinsulinemia and dyslipidemia. There is a correlation between FHD and hyperglycemia, low HDL-C plays a significant role in the development of abnormal glucose and lipid metabolism.²⁶

Gestational diabetes mellitus

Gestational diabetes mellitus (GDM) is a carbohydrate-intolerant phase, which may occur in very small populations (2-3%). During pregnancy, the body's requirement for insulin is more as tissues become less responsive to it, but most women manage to maintain normal blood sugar levels. The prevalence of GDM and T2DM is correlated with maternal age, BMI, race/ethnicity, and family history. The major metabolic disorders are more likely to occur in women with GDM, compared to women with normoglycaemic pregnancy, the history of GDM appears to have a nearly 10-fold increased risk of having dyslipidemia. It also found that HDL-C is significantly lower in women who develop GDM. Given the severity of this risk, it is crucial to take action to delay the emergence of T2DM, especially in the first few years postpartum.²⁷

Body composition

The key indicators to assess obesity and waist-to-hip ratio include body mass index (BMI) and waist-to-hip

circumference. They also measure central or abdominal obesity. The accumulation and distribution of fat in the abdomen region contribute to metabolic abnormalities and an increased risk of cardiovascular diseases.

Numerous prospective and cross-sectional studies have confirmed that there is an association between T2DM, dyslipidemia, and obesity. The majority of T2DM individuals are either overweight or obese. Since obesity, dyslipidemia, and T2DM are linked to insulin resistance and insulin deficiency, they impair the breakdown of TG-rich lipoproteins hinder their clearance from the blood circulation system, and lead to elevated TG levels. Increased TG levels can activate cholesteryl ester transfer protein (CETP), developing TG-rich HDL particles, which are more prone to hydrolysis by plasma lipases. This process ultimately reduces HDL-C levels. In obese individuals with high insulin resistance, TG metabolism releases free fatty acids (FFAs) into the bloodstream. FFAs originate primarily from dietary intake and the breakdown of adipose tissue through lipolysis contributes to the accumulation of fat in the abdominal region. This abdominal fat exacerbates insulin resistance, creating a vicious cycle that increases the risk of dyslipidemia in diabetic individuals.²⁸

Depression

Depression is a mental illness and a major public health challenge worldwide. Despite extensive research to understand its underlying mechanisms, the exact cause remains unclear. Recent studies suggested that the occurrence of depression is closely related to metabolic syndrome especially dyslipidemia, diabetes, and insulin resistance as they share similar biological pathways. Also, they have a bidirectional relationship among them. Insulin resistance is considered a key link between depression and diabetes. In 40-60% of individuals with major depressive disorder, the hypothalamic-pituitary-adrenal (HPA) axis becomes overactive, which leads to excess cortisol production. Elevated cortisol disrupts blood sugar regulation, increases insulin levels, and promotes insulin resistance, which can contribute to both diabetes and dyslipidemia.²⁹

Obstructive sleep apnea

Obstructive sleep apnea (OSA), is a condition that disrupts breathing during sleep. It is the most common type of Sleep-disordered breathing (SDB) and it is linked to adverse cardiometabolic effects such as hypertension and T2DM, ultimately raising the risk of cardiovascular disease. Many studies have shown that OSA is connected to the development of insulin resistance, glucose intolerance and thus leading to T2DM, conversely, studies investigating the incidence of OSA in individuals with T2DM have found remarkably high rates of OSA but compared to non-OSA population, OSA patients are more susceptible to develop T2DM. The common risk factor for both T2DM and OSA is obesity and all three

conditions collectively contribute to the development of cardiovascular diseases.³⁰

NUTRITION STRATEGIES FOR MANAGING BLOOD SUGAR AND LIPID DISORDERS

The current generation is undergoing fast changes in nutrition habits and choosing dietary habits towards a Westernized diet. It has very low fiber content and over caloric diet; a sedentary lifestyle has significantly contributed to T2DM and dyslipidemia. Diabetic dyslipidemia treatment can be done in two ways of therapies, non-pharmacological and pharmacological. Pharmacological therapy includes statins, cholesterol absorption inhibitors, niacin, fibrates, bile acid sequestrants (BAS), and PCSK9 inhibitors to reduce the lipid profile and glycemic level. Whereas non-pharmacological includes weight loss, physical activity, sleep patterns, and mainly medical nutrition therapy.³¹

Lifestyle changes, dietary practices, and effective nutrition management are very crucial to reducing the risk of metabolic disorders like diabetes and dyslipidemia. Different kinds of approaches especially diets with low carb, low fat, low glycemic, and high fiber significantly improve glycemic control, lipid metabolism, and insulin sensitivity. To achieve optimal lipid levels in diabetics, efforts should focus on reaching near-normal blood sugar levels followed by lipid profiles through lifestyle changes. Dietary regimens with differing macronutrient compositions can be utilized safely and efficaciously in the short term (1-2 years) to help individuals with diabetes. It is crucial to develop a personalized meal plan that includes nutrient-dense foods like fruits, vegetables, legumes, dairy, lean protein sources (including plant-based sources in addition to lean meats, fish, and poultry), nuts, seeds, and whole grains, as well as guidance on reaching the appropriate energy deficit.^{32,33} Optimal nutrition, along with appropriate aerobic and resistance exercise, is fundamental in managing dyslipidemia in diabetes. Reducing total body and visceral fat through weight loss and improved body composition can significantly impact serum lipid levels, often achieving results comparable to many medications.³⁴

LOW-GLYCEMIC DIET'S IMPACT ON DYSLIPIDEMIA IN DIABETES

The dietary glycemic index (GI) was developed to manage blood sugar levels in diabetes individuals by evaluating the quality of food high in carbohydrates based on how they affect blood sugar. Carbohydrates, including sugars and starches, are broken down and digested by human enzymes such as maltase, amylase, and sucrose. In contrast, fiber, a complex carbohydrate, remains undigested and moves to the colon, where it is fermented by gut bacteria. Consuming low-GI foods can help regulate blood glucose levels, prevent insulin spikes, and promote better metabolic health, making them beneficial for diabetes management and overall well-being. Low GI

foods which have GI values of ≤ 55 on the glucose scale, undergo slow digestion, absorption, and metabolism. Most of the low-GI foods are fiber-rich which delays the distension of the gastrointestinal tract, causing increased and prolonged secretion of the gut peptides cholecystokinin, ghrelin, and glucagons, all these factors help in good satiety. High GI foods containing a value of $GI \geq 70$ on the glucose scale where Carbohydrate-rich foods result in rapid digestion and absorption followed by metabolism. High GI foods elicit large insulin response production and inhibit the process of producing new glucose by the liver. Also, it stops releasing the glucose stored in the liver to the bloodstream and suppresses lipolysis. This increases the blood glucose concentration and disrupts lipid metabolism.³⁵

ROLE OF DIETARY FIBER

The edible portion of the plant that is not digested in the small intestine and moves into the large intestine is called Dietary fiber (DF). DF is classified into two types, soluble DF (SDF) and insoluble DF (IDF) based on the solubility in hot water. SDF is soluble in water by forming a viscous gel, and IDF is not soluble in water hence do not form gels. SDF reduces the total calorie intake delays gastric emptying controls the glycemic level, and reduces the blood glucose concentration followed by a reduction in LDL cholesterol level. IDF Increases fecal weight, which improves bowel movement consistency and promotes weight loss. Fibre-rich Diabetes Nutrition (FDN) offers multiple benefits for T2DM patients, including improved glycemic control, reduced glucose spikes, lower hyperinsulinemia, better plasma lipid levels, and weight loss. Insulin is one non-viscous SDF that undergoes intestinal fermentation. It slows down the rate at which the stomach empties, which lowers the amount of glucose absorbed and lowers the rise in blood glucose levels after meals. The antihyperlipidemic advantages of DFs are thought to be caused by five main mechanisms: fermentation, bulking impact, viscosity, binding capacity, and low energy levels. Colon health is supported by SDFs, which are fermented by gut bacteria and play a key role in lipid and glucose metabolism. Among the resulting short-chain fatty acids (SCFA), Butyrate is also one of the main SCFAs is the major end product of indigestible carbohydrates in the gut, has anti-inflammatory effects

and the benefits of insulin sensitivity which are also proven by many animal studies. Propionic acid stands out for its potential to directly influence blood glucose control by suppressing the release of plasma triacylglycerols, a major contributor to insulin resistance. The increased bulk and water content in the intestine dilute nutrients such as sugars and lipids, thereby slowing their movement toward the intestinal walls. Fiber binding also limits dietary cholesterol absorption, leading to a further reduction in circulating lipid levels. DFs are indigestible, contributing little to caloric intake while providing satiety, Reduced energy intake lowers fat synthesis and decreases the accumulation of lipids in the bloodstream. A study found that increasing DFs intake was linked to better blood sugar control and improved heart health. It also showed improvements in insulin sensitivity and reduced inflammation in T2DM patients, even after accounting for other factors. A study on dietary assessment was conducted on 1191 urban-rural populations with diabetes using a validated food frequency questionnaire. There was an association between total DF consumption with TC and LDL-C levels among urban adults with T2DM. People consuming less than the median DF intake (<29 g/day) had a 38% higher risk of hypercholesterolemia and 43% higher risk of high LDL-C compared to those consuming more than the median amount.^{36,37}

OMEGA-3- FATTY ACID

Omega-3 polyunsaturated fatty acids (PUFAs) such as eicosapentaenoic (EPA) and docosahexaenoic (DHA) can help with metabolic complications that include glycaemic control, insulin resistance, obesity, atherosclerosis, and chronic inflammation. Since human body cannot produce these fatty acids, they must be obtained from dietary sources such as walnuts, flaxseeds, fish, canola oil and chia seeds. Omega-3 fatty acids, especially those containing EPA and DHA, reduce TG levels, decrease inflammation and enhance lipid profiles, all of which play a significant role in managing T2DM. Incorporation of TG into VLDL is reduced by EPA and DHA, which decreases hepatic VLDL-TG synthesis and secretion while enhancing clearance of TG from circulating VLDL particles. Additionally, omega-3 fatty acids may suppress hepatic fat production, enhance β -oxidation of fatty acids, and promote breakdown of apoB-100.^{38,39}

Table 1: An overview of dyslipidemia research interventions and findings.

Reference and type of diet	Methodology	Conclusion
Low-fat diet⁴⁰	Participants: 221 Duration: 3 weeks Intervention: Group 1 phytosterols-enriched low-fat milk (1.5g phytosterols per day) Group 2 conventional low-fat milk	LDL-cholesterol and TC decreased in group 1 compared.
Low-fat diet⁴¹	Participants: 151 Duration: 6 weeks Intervention: Group 1 Low fat spreads with 2g/ day Plant sterols. Group 2 placebo	Group 1 results in lowering LDL-C and TG.

Continued.

Reference and type of diet	Methodology	Conclusion
Polyphenol diet ⁴²	Participants:150 Duration: 12 weeks Intervention: aqueous extract of <i>Cichorium intybus</i> seeds	There is a significant reduction in hyper glycemia and hyper TG
Polyphenol diet ⁴³	Participants: 18 Duration: 1 week Intervention: Group 1 high fat breakfast for 1 week followed by 20 g cocoa beverage (480 mg flavanols, 201 mg proanthocyanidins, 40 mg epicatechin, and 60 mg polyphenols) group 2 placebo (minimal levels of these beneficial substances)	Decreased VLDL, increased HDL-C.
High protein diet ⁴⁴	Participants: 72 Duration: 6 weeks Intervention: Group 1 Carbohydrate diet Group 2 Carbohydrate reduced high protein diet	There is a greater improvement in TG and HbA1c in group 1 compared to group 2.
Korean diet ⁴⁵	Participants: 24 Duration: 4 weeks for each group Intervention: Group I= Korean diet consists of one serving of grains, fish, poultry, and Kimchi, cooked vegetables of two servings and 1-2 servings of fermented soybeans in every meal. Group II=Control diet includes 1 serving of refined rice, bread or noodles, soup, uncooked vegetables with salad dressing, 1 serving of meat, 0.5 servings of fermented soybeans, and 0.6 servings of Kimchi in each meal.	There is a reduction in TC, non-HDL, and TG concentrations K-diet group compared to the control diet group.
Polyphenol diet ⁴⁶	Participants: 80 Duration: 6 months Intervention: Fruit-rich diet group (FRD)	FRD group had higher levels of TG, TC and LDL-c and a lower level of HDL-c
Low glycemic diet ⁴⁷	Participants: 40 Duration: 2 months Intervention: Group I low GI food, Group II control group received a standard therapy diet.	Both diets reduced TC and TG levels, LGI diet compared to the ST diet was less effective in reducing TG levels.
Low glycemic diet ⁴⁸	Participants: 44 Duration: 6 weeks Intervention: Group 1 control Group 2 performed aerobic resistance exercises Group 3 aerobic-resistance exercises and high-protein, low-GI diet.	LDL C decreased in group 3 as compared to other groups while HDL C remains the same in all the groups.
Low carbohydrate diet ⁴⁹	Participants: 11 Duration: 4 days Intervention: Group 1 low carbohydrate and high-fat diet Group 2 high carbohydrate low fat diet	Group 1 showed a reduced TG level compared to group 2

CONCLUSION

There are limited clinical trials on the effect of nutrition education on diabetes with dyslipidemia. The emphasizing medical nutrition therapy and promoting balanced diet which include low glycemic foods, high-protein, omega-3 fatty acids and DFs, particularly soluble fiber, and improving lifestyle practices. These factors have positive impact on improving the insulin sensitivity and reducing the FFA flux as well as impaired lipid profile. Hence there is a need of incorporating the recommended dietary regime with appropriate approaches in future research.

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Ethical approval: Not required

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