



## Implementation of a Low-Fat Dietary Regimen in Nursing Practice

Maytha Ali Aldosari <sup>(1)</sup>, Noor Ahmed Yahya Almudyir <sup>(2)</sup>, Tahani Mohammed Basher <sup>(3)</sup>, Laila Mohammed Hawi Alnashri <sup>(4)</sup>, Salihah Ibrahim Ahmed Mawkili <sup>(4)</sup>, Norah Hmoud Alahmari <sup>(5)</sup>, Maryam Naser Abdullah Albdullah <sup>(6)</sup>, Essam Essa Mansour Alajam <sup>(7)</sup>, Fadiyah Naja Almutir <sup>(8)</sup>, Ahood Obeed Alanazi <sup>(8)</sup>, Ameinah Mousem Almutairi <sup>(8)</sup>, Ahlam Muteb Almutairi <sup>(9)</sup>

(1) Al-Dar Al-Bayda Primary Health Care Center – Ministry of Health, Saudi Arabia,

(2) Specialized Dental Center – Ministry of Health, Saudi Arabia,

(3) Specialized Dental Center – Jazan – Ministry of Health, Saudi Arabia,

(4) Erada Mental Health Hospital – Jazan Health Cluster – Ministry of Health, Saudi Arabia,

(5) Ministry of Health Headquarters, Saudi Arabia,

(6) Mahasin Primary Health Care Center – Al-Ahsa – Ministry of Health, Saudi Arabia,

(7) Vector Control Center – Al-Eidabi – Ministry of Health, Saudi Arabia,

(8) Al Nahda Western Health Center – Second Health Cluster – Ministry of Health, Saudi Arabia,

(9) Erada Mental Health Complex – Ministry of Health, Saudi Arabia

### Abstract

**Background:** Cardiovascular disease, cancer, and obesity remain leading global health concerns, with dietary fat intake identified as a major modifiable risk factor. Traditional low-fat diets have been widely recommended; however, emerging evidence questions their effectiveness and highlights unintended consequences of fat reduction strategies.

**Aim:** This study aims to evaluate the clinical significance, controversies, and implications of implementing low-fat dietary regimens in nursing practice.

**Methods:** A comprehensive review of epidemiological, clinical, and guideline-based evidence was conducted to assess the impact of low-fat diets on cardiovascular health, cancer risk, and obesity, alongside practical considerations for nursing and interprofessional interventions.

**Results:** Findings reveal that indiscriminate fat reduction, particularly when replaced by refined carbohydrates, may worsen lipid profiles and metabolic outcomes. Evidence supports prioritizing fat quality—favoring unsaturated fats—over total fat reduction. Interprofessional strategies, including patient-centered counseling and lifestyle interventions, are essential for effective implementation.

**Conclusion:** Low-fat diets alone are insufficient for optimal health outcomes. Nursing practice should emphasize balanced dietary patterns, fat quality, and individualized counseling within a collaborative care model.

**Keywords:** Low-fat diet, cardiovascular disease, cancer, obesity, nursing practice, dietary counseling, interprofessional care..

### Introduction

There is broad agreement across clinical and public health disciplines that reducing dietary fat intake plays a central role in lowering the risk of cardiovascular disease and related mortality. Cardiovascular disorders remain a leading cause of death worldwide, and dietary patterns have been consistently identified as modifiable risk factors. A low-fat diet is commonly defined as a dietary pattern in which 30 percent or less of total daily caloric intake is derived from fat. This approach has been promoted as a preventive and therapeutic strategy, particularly in populations with high rates of ischemic heart disease and dyslipidemia. Population-based and correlational studies have demonstrated clear associations between national dietary fat consumption patterns and cardiovascular mortality rates, reinforcing the role of

diet in shaping long-term cardiovascular outcomes.<sup>[1]</sup> From a practical nutritional standpoint, low-fat foods are often identified based on their caloric contribution from fat. A commonly used guideline is that a food providing 100 calories with three grams of fat or less can be classified as low fat. This definition assists clinicians, nurses, and patients in making informed food choices without requiring complex nutritional calculations. Foods that naturally fall within this category include a wide range of plant-based and lean animal products. Vegetables, fruits, and whole grain cereals form the foundation of low-fat dietary patterns and contribute essential vitamins, minerals, and dietary fiber. Lean protein sources such as egg whites, skinless poultry, legumes including beans and lentils, peas, seafood, and low-fat dairy products provide adequate protein while limiting excess fat intake.

These foods collectively support cardiovascular health while meeting nutritional requirements.

Although fats are often targeted for reduction, they remain an essential component of human nutrition. Dietary fats serve critical physiological functions, including energy provision, absorption of fat-soluble vitamins, cell membrane integrity, and hormone synthesis. The emphasis of low-fat dietary recommendations is therefore moderation and quality rather than complete elimination. Dietary fats are broadly categorized into four main types based on their chemical structure and physiological effects: saturated fats, trans fats, monounsaturated fats, and polyunsaturated fats. These categories differ in their degree of saturation and physical characteristics. Saturated and trans fats are typically solid at room temperature, whereas monounsaturated and polyunsaturated fats are generally liquid. Despite these differences, all fats provide nine calories per gram, making them the most energy-dense macronutrient compared with carbohydrates and proteins. The health impact of dietary fat is determined largely by fat type rather than total fat alone. Saturated and trans fats have been shown to raise serum low-density lipoprotein cholesterol levels, which are strongly associated with atherosclerotic cardiovascular disease. Common sources of saturated fat include fatty meats, full-fat dairy products, butter, and certain processed foods. Trans fats, often found in partially hydrogenated oils and commercially baked products, are particularly harmful due to their dual effect of raising LDL cholesterol while lowering high-density lipoprotein cholesterol. In contrast, monounsaturated and polyunsaturated fatty acids exert more favorable lipid effects. These fats reduce LDL cholesterol levels and are associated with improved endothelial function and reduced inflammatory markers. Sources of monounsaturated fats include olive oil and certain nuts, while polyunsaturated fats are found in fish, seeds, and plant oils.

Clinical dietary guidelines have translated this evidence into specific quantitative recommendations. The National Cholesterol Education Program guidelines for adults, as outlined in the Adult Treatment Panel III report, emphasize limiting saturated fat intake to less than seven percent of total daily calories and restricting dietary cholesterol to under 200 milligrams per day. These guidelines also recognize the importance of fat quality by recommending that polyunsaturated fats constitute up to ten percent of total caloric intake and monounsaturated fats up to twenty percent.[2] Such recommendations reflect a balanced approach that limits harmful fats while permitting beneficial ones within controlled limits. Extensive clinical and epidemiological research supports the role of dietary fat modification in regulating serum cholesterol levels. Elevated serum cholesterol, particularly LDL

cholesterol, is a key contributor to the development of atherosclerosis. Atherosclerotic plaque formation leads to narrowing and stiffening of arteries, increasing the risk of myocardial infarction, stroke, and peripheral vascular disease. Evidence indicates that reducing total fat intake, especially saturated and trans fats, leads to measurable reductions in serum cholesterol concentrations. These changes translate into meaningful reductions in cardiovascular risk over time. For this reason, low-fat dietary patterns have been widely endorsed in clinical practice. Healthcare providers frequently recommend low-fat diets as part of comprehensive cardiovascular risk reduction strategies that also include physical activity, smoking cessation, and weight management. In nursing practice, dietary counseling on low-fat nutrition plays an important role in patient education, chronic disease management, and preventive care. Nurses often serve as the primary point of contact for reinforcing dietary guidance, monitoring adherence, and addressing barriers to lifestyle change. In summary, the rationale for promoting low-fat diets is grounded in strong scientific evidence linking dietary fat intake to serum cholesterol levels and cardiovascular outcomes. By emphasizing reduced intake of saturated and trans fats, encouraging consumption of nutrient-dense low-fat foods, and aligning practice with established clinical guidelines, low-fat diets remain a cornerstone of cardiovascular disease prevention and management. The consistent advocacy of low-fat dietary patterns reflects their proven role in reducing cardiovascular morbidity and mortality across diverse populations.

#### **Issues of Concern**

Despite longstanding endorsement of low-fat diets for cardiovascular risk reduction, significant concerns and controversies have emerged regarding their overall health impact and practical implementation. One of the most prominent issues relates to how the low-fat concept has been translated into food production and consumer behavior. In many cases, the reduction of fat content in processed foods has been accompanied by the addition of large quantities of refined carbohydrates and added sugars to preserve palatability and texture. This substitution undermines the intended health benefits of fat reduction and introduces new metabolic risks. Diets high in refined carbohydrates have been associated with increased insulin resistance, elevated triglyceride levels, and a higher incidence of metabolic disorders. As a result, products marketed as low fat may paradoxically contribute to adverse cardiometabolic outcomes rather than reduce them. Epidemiological and clinical studies have increasingly demonstrated that dietary patterns rich in carbohydrates and low in unsaturated fats can negatively influence lipoprotein profiles. High carbohydrate intake, particularly from refined sources, has been linked to reduced high-density lipoprotein cholesterol levels and increased triglyceride concentrations. These changes represent

an unfavorable lipid profile that is associated with increased cardiovascular risk. Evidence suggests that replacing dietary fat indiscriminately with carbohydrates does not confer cardiovascular protection and may, in some cases, exacerbate atherogenic processes.[3] This has challenged the traditional assumption that lowering total fat intake alone is sufficient to improve cardiovascular health.

Further concern has arisen regarding the functional quality of high-density lipoprotein cholesterol. Beyond its concentration in serum, HDL exerts cardioprotective effects through reverse cholesterol transport, anti-inflammatory activity, and antioxidant properties. A proposed mechanism suggests that refined carbohydrate intake may impair these protective functions by altering HDL metabolism and composition. Such alterations could diminish HDL efficiency in cholesterol efflux and vascular protection, thereby weakening its role in cardiovascular risk reduction.[4] This perspective has shifted scientific focus away from isolated lipid values toward the functional integrity of lipoproteins. Another critical issue is the lack of clarity regarding the type and quality of carbohydrates used to replace dietary fat. While early low-fat dietary recommendations emphasized reducing fat intake, they often failed to specify whether replacement carbohydrates should be whole grains, fruits, and vegetables or refined sugars and starches. This ambiguity has contributed to widespread dietary patterns characterized by high intake of refined carbohydrates with low nutritional value. Research has highlighted that the metabolic effects of carbohydrate replacement vary substantially depending on carbohydrate quality, yet this distinction has historically received insufficient emphasis in dietary guidance.[5] The absence of clear guidance has limited the effectiveness of low-fat dietary strategies in real-world settings.

These accumulating concerns have prompted exploration of alternative dietary approaches that prioritize fat quality rather than fat quantity. Diets emphasizing unsaturated fats, such as Mediterranean-style dietary patterns, have gained support due to their favorable effects on lipid profiles, glycemic control, and inflammatory markers.[6] Such approaches challenge the low-fat paradigm by demonstrating that higher fat intake, when derived from healthy fat sources, can coexist with reduced cardiovascular risk. This shift reflects a broader reevaluation of macronutrient balance and dietary patterns. Clinical studies have also raised concerns about specific lipid alterations associated with low-fat, high-carbohydrate diets. Evidence indicates that such diets may lower HDL cholesterol concentrations and raise triglyceride levels, particularly in individuals with insulin resistance or metabolic syndrome. Postprandial lipemia, characterized by prolonged elevation of triglyceride-rich lipoproteins after meals, has also been observed following high-carbohydrate

intake.[7][8] These postprandial changes are increasingly recognized as independent cardiovascular risk factors, further complicating the presumed benefits of low-fat dietary patterns.

The evolving evidence base has influenced major clinical guideline recommendations. The 2013 American Heart Association and American College of Cardiology Guideline on Lifestyle Management to Reduce Cardiovascular Risk did not provide specific recommendations for dietary cholesterol intake. The guideline acknowledged insufficient evidence to conclusively demonstrate that lowering dietary cholesterol independently reduces low-density lipoprotein cholesterol levels.[9] This marked a significant departure from earlier guidance that strongly emphasized cholesterol restriction and reflected growing uncertainty regarding its direct impact on cardiovascular outcomes. This shift was reinforced by the 2015 Dietary Guidelines Advisory Committee, which chose not to maintain the long-standing recommendation to limit dietary cholesterol to less than 300 milligrams per day. Instead, the committee emphasized overall dietary patterns rather than isolated macronutrient targets. The focus moved toward encouraging consumption of nutrient-dense foods, balanced eating patterns, and healthy fat sources rather than strict numerical limits on fat or cholesterol intake.[10] This change signaled recognition that cardiovascular risk is influenced by complex dietary interactions rather than single nutrients in isolation. Collectively, these issues highlight the limitations of a simplistic low-fat dietary model. The evidence suggests that reducing total fat intake without regard to fat type or carbohydrate quality may lead to unintended metabolic consequences. The historical focus on fat reduction has, in some cases, diverted attention from the harmful effects of refined carbohydrates and the benefits of unsaturated fats. As scientific understanding of nutrition advances, dietary recommendations increasingly reflect a more nuanced approach that prioritizes food quality, metabolic impact, and overall dietary patterns. In clinical and nursing practice, these controversies underscore the importance of individualized dietary counseling and evidence-based guidance. Rather than promoting low-fat diets in isolation, healthcare professionals are encouraged to consider the broader nutritional context, patient metabolic profile, and long-term cardiovascular risk. Awareness of these concerns allows clinicians to guide patients toward balanced dietary patterns that support cardiovascular health without introducing new risks.

### Clinical Significance

#### Association with Cardiovascular Disease

A substantial body of scientific evidence supports a direct association between dietary fat intake and the development of cardiovascular disease. Cardiovascular disease remains a leading cause of morbidity and mortality worldwide, and dietary composition has been consistently identified as a

major modifiable risk factor. Early observational and experimental studies demonstrated that populations consuming diets high in total fat and saturated fatty acids experience higher rates of coronary artery disease, whereas populations with lower intake exhibit reduced cardiovascular risk.[11] This relationship has driven decades of research aimed at clarifying how different types of dietary fat influence lipid metabolism and atherosclerotic processes. Dietary cholesterol has also received considerable attention due to its influence on blood cholesterol concentrations and subsequent cardiovascular risk. The link between dietary intake and circulating cholesterol levels provided a biological rationale for dietary modification as a preventive strategy.[12] Among lipid markers, low-density lipoprotein particles have emerged as the most reliable predictor of cardiovascular events. Elevated LDL particle concentration promotes cholesterol deposition within the arterial wall, leading to plaque formation and progression of atherosclerosis.[13][14] Consequently, dietary interventions that reduce LDL levels are considered central to cardiovascular risk reduction. Research has demonstrated that the effect of dietary fat on serum cholesterol is not uniform and depends largely on fatty acid composition. Saturated fatty acids have consistently been shown to increase total and LDL cholesterol levels, thereby elevating cardiovascular risk. In contrast, polyunsaturated fatty acids reduce serum cholesterol concentrations by enhancing LDL clearance and improving lipid metabolism. Monounsaturated fatty acids have generally been shown to exert a neutral or modestly beneficial effect on cholesterol levels when they replace saturated fats in the diet.[15] These differential effects have reshaped nutritional recommendations to emphasize fat quality rather than total fat alone.

Further differentiation within saturated fatty acids has revealed variable biological effects. Studies indicate that myristic and palmitic acids exert strong cholesterol-raising effects, whereas stearic acid appears to have little or no impact on serum cholesterol levels.[16] This distinction underscores the complexity of dietary fat metabolism and highlights the limitations of treating all saturated fats as a single category. Trans fatty acids have been shown to behave similarly to saturated fats, with the added disadvantage of lowering high-density lipoprotein cholesterol while raising LDL levels.[17] This dual effect significantly increases atherogenic risk and has led to strong recommendations for minimizing trans fat intake. Collectively, these findings support dietary patterns characterized by low intake of saturated and trans fatty acids and higher intake of polyunsaturated fatty acids.[15] Such patterns favorably modify lipid profiles and reduce cardiovascular risk. Longitudinal cohort studies have provided strong evidence for this approach. The Nurses' Health Study demonstrated that women who consumed diets low in saturated and trans

fats and relatively high in unhydrogenated monounsaturated and polyunsaturated fats experienced the lowest incidence of cardiovascular events.[18] This large-scale observational study reinforced the importance of fat substitution rather than simple fat reduction.

Interventional studies have further clarified the clinical impact of modifying dietary fat intake. In men, reducing total fat intake from approximately 36 percent of total energy to 27 percent, along with a reduction in saturated fat from 12 percent to 8 percent of energy, resulted in significant declines in total and LDL cholesterol levels.[19][20] These findings illustrate that even moderate changes in dietary fat composition can yield meaningful improvements in lipid profiles, thereby lowering cardiovascular risk. Despite these advances, population-level dietary patterns remain a concern. Over recent decades, the percentage of dietary fat intake has declined modestly in some populations. However, this reduction has been accompanied by a paradoxical increase in absolute fat consumption, reflecting an overall rise in total energy intake. This trend suggests that portion size expansion and increased caloric consumption have offset improvements in dietary composition. As a result, many individuals continue to consume excessive amounts of total fat and saturated fatty acids.

	<b>High Fat</b>
Cereal Foods	Croissants Pastries Doughnuts Cakes Biscuits Fried Bread Puddings
Fruit, Vegetables & Nuts	Chips Fried or Roast Potatoes Fried, creamed, buttered or cheezed vegetables Crisps and potato snacks Coconut Brazil nuts Roasted peanuts Cooking oil
Fish	Fish roe Caviar
Meat	Visible fat on meat Crackling Sausages Pâtés Duck, goose Meat pies and pasties Processed meats like salami Lamb mince Beef mince

**Fig. 1:** Low Fat Diet.

In the United States, a significant proportion of the population still exceeds recommended limits for saturated fat intake, placing them at ongoing risk for cardiovascular disease. This persistent gap between dietary guidelines and actual consumption highlights the challenge of translating scientific evidence into sustained behavioral change. From a clinical perspective, it underscores the need for continued emphasis on dietary education, counseling, and public health strategies that address both fat quality and total energy intake. In summary, the association between dietary fat and cardiovascular disease is well established and supported by epidemiological, clinical, and mechanistic evidence. Saturated and trans fatty acids increase LDL cholesterol and cardiovascular risk, whereas polyunsaturated and monounsaturated fats exert protective or neutral effects. Effective cardiovascular prevention requires not only reducing harmful fats but also promoting healthier fat substitutions within balanced dietary

patterns. Continued attention to these principles remains essential for reducing the global burden of cardiovascular disease.

#### Association with Cancer

The relationship between dietary fat intake and cancer risk has been the subject of extensive research, with multiple epidemiologic studies demonstrating consistent associations across a range of malignancies. Evidence suggests that dietary fat may influence the development and progression of cancers such as breast, prostate, colorectal, and lung cancer, although the strength of these associations varies depending on cancer type, fat source, and overall dietary pattern.[21] Breast cancer has been the malignancy most extensively studied in relation to dietary fat intake. Epidemiological data indicate that high consumption of total fat, particularly from animal sources, may be associated with increased breast cancer risk.[22] Several biological mechanisms have been proposed to explain this link. One mechanism involves the conversion of essential fatty acids into short-lived, hormone-like lipid mediators that can influence cell proliferation and differentiation. Dietary fat may also increase oxidative stress through the generation of reactive oxygen species, which have the potential to induce DNA damage, genomic instability, and alterations in gene expression that promote tumorigenesis.[23] Additionally, fat intake may modulate the hypothalamic-pituitary axis, resulting in hormonal changes that affect estrogen production and receptor activity. Such alterations can influence breast tissue proliferation and the risk of malignant transformation. Dietary fat may also affect enzyme systems involved in estrogen metabolism, further modulating exposure of breast tissue to proliferative signals. Other mechanisms include changes in cell membrane structure and function, signaling pathways, and immune surveillance, all of which can contribute to carcinogenic processes.[24]

The type of dietary fat appears to be a significant determinant of cancer risk. Polyunsaturated fatty acids, particularly omega-3 fatty acids found in fish and certain plant oils, have demonstrated protective effects against cancer development. Omega-3 fatty acids may reduce inflammation, improve immune function, and modulate cellular proliferation pathways, collectively decreasing the likelihood of malignant transformation. Conversely, high intake of saturated fats and certain animal fats has been associated with increased cancer risk, with some studies reporting the strongest positive correlation between animal fat consumption and breast cancer incidence. These observations highlight the importance of fat quality rather than total fat alone in influencing cancer outcomes. Prostate cancer has also been linked to dietary fat intake, with proposed mechanisms involving alterations in sex hormone levels. High-fat diets may increase circulating androgens, which can promote prostate cell proliferation and enhance tumor progression.

Epidemiologic studies suggest that men consuming diets high in saturated fats, particularly from animal sources, may be at higher risk for aggressive forms of prostate cancer.[25] Colorectal cancer provides another example of the complex relationship between dietary fat and malignancy. Mortality and incidence data have correlated high consumption of animal fats with increased colorectal cancer risk. Mechanistic explanations include increased bile acid secretion, which may promote colonocyte proliferation and generate carcinogenic secondary metabolites, as well as changes in intestinal microbial metabolism that influence epithelial health and inflammatory status.[26] Conversely, populations consuming diets rich in olive oil or marine-derived oils have shown significantly lower incidence rates of colorectal cancer. These observations underscore the importance of dietary fat quality, with unsaturated fats from plant and marine sources providing protective effects while high intake of saturated and processed animal fats increases risk.[27]

Collectively, the evidence indicates that dietary fat plays a multifaceted role in cancer development, mediated by hormonal, metabolic, and cellular mechanisms. Fat type, source, and overall dietary patterns are critical in determining whether dietary fat contributes to increased cancer risk or provides protective effects. For clinical and nursing practice, these findings highlight the importance of dietary counseling focused not solely on reducing total fat intake but on optimizing fat quality by encouraging polyunsaturated and monounsaturated fat consumption and limiting saturated and trans fats. Such guidance can contribute to comprehensive cancer prevention strategies that integrate nutritional modification with other lifestyle and risk-reduction interventions. The interplay between dietary fat and cancer underscores the need for continued research to elucidate precise mechanisms and quantify risk across diverse populations. Nevertheless, existing evidence strongly supports a dietary approach that emphasizes unsaturated fats, minimizes animal and processed fats, and incorporates nutrient-dense foods as part of a preventive strategy against multiple forms of cancer.

#### **Association with Obesity**

Obesity is recognized as a chronic, multifactorial disease associated with numerous comorbidities, including type 2 diabetes mellitus, dyslipidemia, hypertension, non-alcoholic fatty liver disease, and obstructive sleep apnea, among others.[28][29][30] Its etiology is complex, encompassing both intrinsic and extrinsic factors, but environmental influences, particularly dietary habits, are considered among the most significant contributors. Within this context, dietary fat intake has been identified as a key factor influencing energy balance and body weight regulation. The development of obesity is fundamentally driven by prolonged energy imbalance, in which caloric intake exceeds

energy expenditure. Modern sedentary lifestyles, combined with the availability of high-calorie foods, have amplified this imbalance, contributing to the rapid rise in obesity prevalence globally. In the United States, using body mass index (BMI) as the standard measure, more than one-third of adults are classified as overweight or obese.[31] This reflects not only individual dietary and activity behaviors but also broader societal trends, including increased consumption of energy-dense foods, larger portion sizes, and limited physical activity. The global prevalence of obesity has similarly increased at an alarming rate, highlighting the role of modifiable lifestyle factors such as diet composition. Dietary fat, in particular, is highly energy-dense, providing nine calories per gram, compared with four calories per gram for carbohydrates or protein. This characteristic renders high-fat diets especially potent in promoting positive energy balance and excess weight gain when not counterbalanced by energy expenditure. Epidemiological research has explored the relationship between fat intake and obesity through multiple study designs, including ecological, cross-sectional, and prospective cohort studies.[32] Cross-sectional analyses generally reveal that individuals with higher BMI consume a greater proportion of their daily energy from fat compared to individuals with normal BMI.[33] These findings support the notion that high-fat diets contribute to increased caloric intake and, by extension, weight gain. However, the relationship is complex, as fat intake alone may not fully explain variations in body weight. Metabolic differences, physical activity levels, and dietary patterns collectively influence energy balance and weight outcomes.

Beyond its caloric density, dietary fat has been hypothesized to contribute to obesity through metabolic mechanisms. Some studies suggest that individuals with obesity may exhibit reduced fat oxidation, either systemically or at the cellular level, leading to less efficient utilization of dietary fat for energy.[34] This impaired capacity to metabolize fat could predispose individuals to greater fat storage, further exacerbating weight gain over time. Fat oxidation capacity varies among individuals due to genetic, hormonal, and physiological differences, which may explain why some individuals gain weight more readily than others under similar dietary conditions. Despite these associations, recent population-level data indicate that reducing absolute fat intake or decreasing the percentage of dietary energy derived from fat has not consistently translated into meaningful reductions in body weight across populations.[35] This finding suggests that obesity prevention and treatment strategies cannot rely solely on lowering fat intake; rather, they must consider total energy consumption, macronutrient quality, physical activity, and broader lifestyle factors. For instance, replacing dietary fat with refined carbohydrates may

not reduce overall energy intake and can even exacerbate metabolic dysregulation by increasing postprandial glucose and insulin levels, thereby contributing to adiposity. From a clinical and nursing perspective, these findings underscore the importance of comprehensive dietary counseling and individualized interventions for obesity management. Emphasis should be placed not only on limiting excess energy intake from fat but also on promoting balanced diets rich in nutrient-dense foods, including fruits, vegetables, whole grains, and lean protein sources. Behavioral strategies, education on portion control, and integration of regular physical activity are essential components of effective obesity prevention and treatment programs. Addressing obesity requires a multifaceted approach that recognizes the complexity of dietary influences, metabolic differences, and lifestyle behaviors. In summary, dietary fat intake is closely associated with obesity due to its energy density and potential metabolic effects. While high-fat diets contribute to increased caloric intake, they do not act in isolation; obesity development is influenced by a combination of dietary patterns, energy expenditure, and individual metabolic capacity. Effective interventions must therefore address the broader context of energy balance, emphasizing quality, quantity, and behavioral strategies to mitigate the rising prevalence of obesity and its related comorbidities [35].

### **Nursing, Allied Health, and Interprofessional Team Interventions**

Effective dietary management requires a coordinated, interprofessional approach to optimize patient outcomes and promote long-term health. Healthcare practitioners—including nurses, dietitians, physician assistants, primary care providers, and allied health personnel—must collaborate to ensure that patients receive consistent, evidence-based guidance on nutrition and lifestyle modifications. Current recommendations emphasize dietary patterns rather than isolated macronutrient targets, reflecting the understanding that overall diet quality, rather than simply reducing fat intake, is most influential in preventing and managing metabolic and cardiovascular conditions. This approach aligns with the Therapeutic Lifestyle Changes (TLC) program, which is the lifestyle component of the third report of the National Cholesterol Education Program Adult Treatment Panel (ATP III) guidelines. The TLC framework is endorsed by multiple organizations, including the American Heart Association, the American Diabetes Association, and The Obesity Society, as a recommended strategy to improve metabolic health and reduce cardiovascular risk. The first step in implementing an interprofessional dietary strategy is education and skill development among healthcare team members. Nursing staff, allied health professionals, and clinicians must understand the principles of TLC, including the rationale behind recommended dietary patterns, the physiological

benefits of nutrient-dense foods, and the evidence linking these patterns to improved cardiovascular and metabolic outcomes. Educational tools, workshops, and simulation exercises can facilitate this understanding and enable team members to confidently counsel patients. A shared knowledge base ensures that all team members communicate consistent dietary advice, reducing confusion for patients and increasing adherence to recommended practices [35].

Patient-centered counseling is a critical component of this approach. The interprofessional team should guide patients to adopt a dietary pattern that prioritizes vegetables, fruits, whole grains, legumes, low-fat dairy, poultry, fish, nontropical oils, and minimal consumption of red meats, sugar-sweetened beverages, and sweets. These recommendations align with the TLC and Dietary Approaches to Stop Hypertension (DASH) frameworks, both of which have been shown to improve lipid profiles, reduce blood pressure, and mitigate the risk of metabolic syndrome. Team members must also address behavioral components of lifestyle change, including strategies for meal planning, grocery shopping, portion control, and cooking methods that reduce fat and sodium content while maintaining palatability. Physical activity is an integral complement to dietary intervention. Healthcare providers should encourage patients to engage in regular aerobic activity to support reductions in low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and overall cardiovascular risk. Nurses and physical therapists can provide guidance on safe and effective exercise routines tailored to individual patient capabilities and health status. Coordinated counseling that integrates diet and exercise reinforces lifestyle changes and promotes sustainable behavior modification. An interprofessional approach also requires careful monitoring and follow-up. Nurses and allied health professionals play a key role in assessing patient adherence, monitoring clinical markers such as lipid levels, blood pressure, and body weight, and identifying barriers to implementation. Physicians and nurse practitioners oversee clinical decision-making and adjust interventions based on laboratory results and patient progress. Dietitians provide individualized nutritional counseling, ensuring that dietary recommendations are culturally appropriate, feasible, and aligned with patient preferences [33][34][35].

Shared decision-making between the healthcare team and the patient is essential for successful dietary modification. Patients must understand the health implications of high-fat diets and the benefits of adopting healthier eating patterns. Collaborative goal-setting, realistic planning, and ongoing support increase patient engagement and adherence. By integrating dietary counseling with lifestyle interventions and leveraging the skills of the entire interprofessional team, healthcare providers can

effectively address obesity, hyperlipidemia, hypertension, diabetes, and cardiovascular risk, fostering long-term health outcomes. In conclusion, interprofessional collaboration is fundamental to implementing evidence-based dietary interventions. Through education, shared knowledge, patient-centered counseling, and ongoing monitoring, nurses, allied health professionals, and clinicians can collectively guide patients toward healthier dietary patterns and improved metabolic and cardiovascular outcomes. This comprehensive, team-based approach ensures that dietary modifications are not only theoretically beneficial but also practically achievable for patients [35].

### Conclusion:

The traditional emphasis on reducing total fat intake as a universal strategy for disease prevention is increasingly recognized as overly simplistic. Evidence demonstrates that the health impact of dietary fat depends largely on its type and quality rather than quantity alone. Saturated and trans fats elevate cardiovascular risk, while unsaturated fats confer protective benefits. Furthermore, replacing fat with refined carbohydrates can lead to adverse metabolic effects, undermining intended health gains. For nursing and clinical practice, these findings underscore the need for individualized, evidence-based dietary counseling that prioritizes nutrient-dense foods and healthy fat sources. Interprofessional collaboration is critical to ensure consistent messaging, patient engagement, and long-term adherence. By shifting focus from restrictive low-fat paradigms to holistic dietary patterns, healthcare providers can more effectively reduce the burden of cardiovascular disease, cancer, and obesity, promoting sustainable health outcomes across diverse populations.

### References:

1. Mann J. Complex carbohydrates: replacement energy for fat or useful in their own right? *The American journal of clinical nutrition*. 1987 May;45(5 Suppl):1202-6. doi: 10.1093/ajcn/45.5.1202.
2. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). *JAMA*. 2001 May 16;285(19):2486-97
3. Sacks FM, Lichtenstein AH, Wu JHY, Appel LJ, Creager MA, Kris-Etherton PM, Miller M, Rimm EB, Rudel LL, Robinson JG, Stone NJ, Van Horn LV, American Heart Association. Dietary Fats and Cardiovascular Disease: A Presidential Advisory From the American Heart Association. *Circulation*. 2017 Jul 18;136(3):e1-e23. doi: 10.1161/CIR.0000000000000510.
4. Andraski AB, Singh SA, Lee LH, Higashi H, Smith N, Zhang B, Aikawa M, Sacks FM. Effects of Replacing Dietary Monounsaturated Fat With Carbohydrate on HDL (High-Density Lipoprotein) Protein Metabolism and Proteome Composition in Humans. *Arteriosclerosis, thrombosis, and vascular biology*. 2019 Nov;39(11):2411-2430. doi: 10.1161/ATVBAHA.119.312889.
5. Berglund L, Lefevre M, Ginsberg HN, Kris-Etherton PM, Elmer PJ, Stewart PW, Ershow A, Pearson TA, Dennis BH, Roheim PS, Ramakrishnan R, Reed R, Stewart K, Phillips KM, DELTA Investigators. Comparison of monounsaturated fat with carbohydrates as a replacement for saturated fat in subjects with a high metabolic risk profile: studies in the fasting and postprandial states. *The American journal of clinical nutrition*. 2007 Dec;86(6):1611-20
6. Bolla AM, Caretto A, Laurenzi A, Scavini M, Piemonti L. Low-Carb and Ketogenic Diets in Type 1 and Type 2 Diabetes. *Nutrients*. 2019 Apr 26;11(5):. doi: 10.3390/nu11050962.
7. Ginsberg HN, Kris-Etherton P, Dennis B, Elmer PJ, Ershow A, Lefevre M, Pearson T, Roheim P, Ramakrishnan R, Reed R, Stewart K, Stewart P, Phillips K, Anderson N. Effects of reducing dietary saturated fatty acids on plasma lipids and lipoproteins in healthy subjects: the DELTA Study, protocol 1. *Arteriosclerosis, thrombosis, and vascular biology*. 1998 Mar;18(3):441-9
8. Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins. A meta-analysis of 27 trials. *Arteriosclerosis and thrombosis : a journal of vascular biology*. 1992 Aug;12(8):911-9
9. Eckel RH, Jakicic JM, Ard JD, de Jesus JM, Houston Miller N, Hubbard VS, Lee IM, Lichtenstein AH, Loria CM, Millen BE, Nonas CA, Sacks FM, Smith SC Jr, Svetkey LP, Wadden TA, Yanovski SZ, American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Journal of the American College of Cardiology*. 2014 Jul 1;63(25 Pt B):2960-84. doi: 10.1016/j.jacc.2013.11.003.
10. Jahns L, Davis-Shaw W, Lichtenstein AH, Murphy SP, Conrad Z, Nielsen F. The History and Future of Dietary Guidance in America. *Advances in nutrition* (Bethesda, Md.). 2018 Mar 1;9(2):136-147. doi: 10.1093/advances/nmx025.
11. Nettleton JA, Brouwer IA, Geleijnse JM, Hornstra G. Saturated Fat Consumption and Risk of Coronary Heart Disease and Ischemic Stroke: A Science Update. *Annals of nutrition &*

metabolism. 2017;70(1):26-33. doi: 10.1159/000455681.

12. Tsoupras A, Lordan R, Zabetakis I. Inflammation, not Cholesterol, Is a Cause of Chronic Disease. *Nutrients*. 2018 May 12;10(5):. doi: 10.3390/nu10050604.

13. Upadhyay RK. Emerging risk biomarkers in cardiovascular diseases and disorders. *Journal of lipids*. 2015;2015():971453. doi: 10.1155/2015/971453.

14. Cromwell WC, Otvos JD, Keyes MJ, Pencina MJ, Sullivan L, Vasan RS, Wilson PW, D'Agostino RB. LDL Particle Number and Risk of Future Cardiovascular Disease in the Framingham Offspring Study - Implications for LDL Management. *Journal of clinical lipidology*. 2007 Dec;1(6):583-92. doi: 10.1016/j.jacl.2007.10.001.

15. Siri-Tarino PW, Chiu S, Bergeron N, Krauss RM. Saturated Fats Versus Polyunsaturated Fats Versus Carbohydrates for Cardiovascular Disease Prevention and Treatment. *Annual review of nutrition*. 2015;35():517-43. doi: 10.1146/annurev-nutr-071714-034449.

16. Tholstrup T, Vessby B, Sandstrom B. Difference in effect of myristic and stearic acid on plasma HDL cholesterol within 24 h in young men. *European journal of clinical nutrition*. 2003 Jun;57(6):735-42.

17. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Saturated fatty acids and risk of coronary heart disease: modulation by replacement nutrients. *Current atherosclerosis reports*. 2010 Nov;12(6):384-90. doi: 10.1007/s11883-010-0131-6.

18. Dhaka V, Gulia N, Ahlawat KS, Khatkar BS. Trans fats-sources, health risks and alternative approach - A review. *Journal of food science and technology*. 2011 Oct;48(5):534-41. doi: 10.1007/s13197-010-0225-8.

19. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Saturated fat, carbohydrate, and cardiovascular disease. *The American journal of clinical nutrition*. 2010 Mar;91(3):502-9. doi: 10.3945/ajcn.2008.26285.

20. Briggs MA, Petersen KS, Kris-Etherton PM. Saturated Fatty Acids and Cardiovascular Disease: Replacements for Saturated Fat to Reduce Cardiovascular Risk. *Healthcare (Basel, Switzerland)*. 2017 Jun 21;5(2):. doi: 10.3390/healthcare5020029.

21. Donaldson MS. Nutrition and cancer: a review of the evidence for an anti-cancer diet. *Nutrition journal*. 2004 Oct 20;3():19.

22. Holmes MD, Willett WC. Does diet affect breast cancer risk? *Breast cancer research : BCR*. 2004;6(4):170-8

23. Ayala A, Muñoz MF, Argüelles S. Lipid peroxidation: production, metabolism, and signaling mechanisms of malondialdehyde and 4-hydroxy-2-nonenal. *Oxidative medicine and cellular longevity*. 2014;2014():360438. doi: 10.1155/2014/360438.

24. Herman JP, McKlveen JM, Ghosal S, Kopp B, Wulsin A, Makinson R, Scheimann J, Myers B. Regulation of the Hypothalamic-Pituitary-Adrenocortical Stress Response. *Comprehensive Physiology*. 2016 Mar 15;6(2):603-21. doi: 10.1002/cphy.c150015.

25. Di Sebastiano KM, Mourtzakis M. The role of dietary fat throughout the prostate cancer trajectory. *Nutrients*. 2014 Dec 22;6(12):6095-109. doi: 10.3390/nu6126095.

26. Ocvirk S, O'Keefe SJ. Influence of Bile Acids on Colorectal Cancer Risk: Potential Mechanisms Mediated by Diet - Gut Microbiota Interactions. *Current nutrition reports*. 2017 Dec;6(4):315-322. doi: 10.1007/s13668-017-0219-5.

27. Liu AG, Ford NA, Hu FB, Zelman KM, Mozaffarian D, Kris-Etherton PM. A healthy approach to dietary fats: understanding the science and taking action to reduce consumer confusion. *Nutrition journal*. 2017 Aug 30;16(1):53. doi: 10.1186/s12937-017-0271-4.

28. Engin A. The Definition and Prevalence of Obesity and Metabolic Syndrome. *Advances in experimental medicine and biology*. 2017;960():1-17. doi: 10.1007/978-3-319-48382-5\_1.

29. Lavie CJ, De Schutter A, Parto P, Jahangir E, Kokkinos P, Ortega FB, Arena R, Milani RV. Obesity and Prevalence of Cardiovascular Diseases and Prognosis-The Obesity Paradox Updated. *Progress in cardiovascular diseases*. 2016 Mar-Apr;58(5):537-47. doi: 10.1016/j.pcad.2016.01.008.

30. Meldrum DR, Morris MA, Gambone JC. Obesity pandemic: causes, consequences, and solutions-but do we have the will? *Fertility and sterility*. 2017 Apr;107(4):833-839. doi: 10.1016/j.fertnstert.2017.02.104.

31. Cetin D, Lessig BA, Nasr E. Comprehensive Evaluation for Obesity: Beyond Body Mass Index. *The Journal of the American Osteopathic Association*. 2016 Jun 1;116(6):376-82. doi: 10.7556/jaoa.2016.078.

32. Satija A, Yu E, Willett WC, Hu FB. Understanding nutritional epidemiology and its role in policy. *Advances in nutrition (Bethesda, Md.)*. 2015 Jan;6(1):5-18. doi: 10.3945/an.114.007492.

33. Galgani J, Ravussin E. Energy metabolism, fuel selection and body weight regulation. *International journal of obesity (2005)*. 2008 Dec;32 Suppl 7(Suppl 7):S109-19. doi: 10.1038/ijo.2008.246.

34. Bergouignan A, Kealey EH, Schmidt SL, Jackman MR, Bessesen DH. Twenty-four hour total and dietary fat oxidation in lean, obese and reduced-obese adults with and without a bout of

---

exercise. *PLoS one.* 2014;9(4):e94181. doi: 10.1371/journal.pone.0094181.

35. Lichtenstein AH, Kennedy E, Barrier P, Danford D, Ernst ND, Grundy SM, Leveille GA, Van Horn L, Williams CL, Booth SL. Dietary fat consumption and health. *Nutrition reviews.* 1998 May;56(5 Pt 2):S3-19; discussion S19-28