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# The Efficacy of Non-Pharmacological Interventions in Reducing Elevated Serum LDL Cholesterol Levels: A Review Paper

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**Abstract**

The review article explores the latest non-pharmacological approaches towards lowering serum levels of a main atherogenic lipoprotein, i.e. low-density lipoprotein (LDL). Elevated low-density lipoprotein cholesterol (LDL-C) levels are associated with a higher risk of cardiovascular disease (CVD)(1). According to World Health Organization (WHO) cardiovascular diseases (CVDs) are the leading cause of mortality worldwide(2). Although drug therapy of elevated LDL-C with statins remains effective, lifestyle modifications should be implemented as a first step in the treatment and also together with prescribed medication for the improved therapeutic outcome. Additionally, the side effects and cost of statins cause more than 30% of patients to discontinue their prescriptions(3), making lifestyle modifications both necessary and increasingly sought after. The article discusses lifestyle

changes, particularly dietary adjustments and increased physical activity, and their potential beneficial effects on reducing elevated LDL-C levels. The **conclusion** emphasizes the critical role of non-pharmacological interventions in managing excess LDL-C in all patients.

Keywords

elevated LDL management, lifestyle medicine, non-pharmacological interventions, LDL, cholesterol, diet, exercise, atherosclerosis

## Introduction

Cardiovascular diseases (CVDs) rank as the primary cause of death in Poland, Europe and in the world. CVDs are the first most common cause of death globally according to World Health Organisation (WHO)(2). In 2019, approximately 17.9 million people died from cardiovascular diseases (CVDs), accounting for 32% of all global deaths, 85% of which were caused by heart attacks and strokes. According to Eurostat(4), which is the statistical office of the European Union, in 2022, circulatory diseases were responsible for nearly one-third (32.7%) of all deaths in the EU. According to Statistics Poland (Główny Urząd Statystyczny - GUS) in 2023, the main causes of death in Poland were circulatory diseases, cancers, and respiratory diseases. Circulatory diseases were the primary cause of death, and they accounted for 36% of all deaths(5). One could state that two conditions are responsible for cardiovascular deaths – myocardial infarction (MI) and stroke and their complications(5). Both conditions are directly linked to hypertension and dyslipidemia, the former affecting around 30% of the Polish population and the latter more than 60%(6).

The article “Association of low-density lipoprotein cholesterol levels with the risk of mortality and cardiovascular events: A meta-analysis of cohort studies with 1,232,694 participants”(1) concludes that LDL-C level of more than 130mg/dL is associated with higher

risk of cardiovascular events such as myocardial infarction (MI) and stroke(7). Therefore, adults should strive to maintain an LDL-C level below 130 mg/dL to help prevent cardiovascular disease (CVD), the leading cause of death in Poland, Europe, and worldwide.

In order to manage elevated LDL cholesterol levels, it is important to reduce the intake of saturated fatty acids and trans fats, while increasing dietary fiber consumption(8). Additionally, incorporating changes in physical activity should be considered.

## Materials and Methods

This review article presents a comprehensive and systematic analysis of the current literature on non-pharmacological approaches to managing elevated LDL-C levels. The primary **aim** was to synthesize and critically assess existing evidence, identify gaps in the research, and propose directions for future investigation. A thorough literature search was conducted across multiple electronic databases, including PubMed, Google Scholar, and Scopus. The search strategy focused on clinical interventions, randomized controlled trials, meta-analyses, and review articles published within the past 20 years. Relevant **keywords** used in the search included “elevated LDL management,” “lifestyle medicine,” “non-pharmacological interventions,” “low-density lipoproteins,” “cholesterol”, “diet”, “exercise”, and “atherosclerosis,” among others.

## Discussion

### 1. Dietary Fat

#### Saturated Fatty Acids (SFA)

Saturated fatty acids (SFAs) contribute to elevated levels of low-density lipoprotein cholesterol (LDL-C) and exhibit prothrombotic properties. It is estimated that a 1% increase in dietary energy derived from SFAs raises LDL-C concentrations by approximately 0.02–

0.04 mmol/L(6). Since SFAs can be endogenously synthesized by the human body, their intake through the diet is unnecessary. Polish dietary guidelines recommend maintaining SFA intake "as low as possible while preserving adequate nutritional quality." Similarly, the European Society of Cardiology advises limiting SFA consumption to below 10% of total energy intake in individuals without cardiovascular risk factors, and to below 7% in those with increased risk(6).

Sadly, SFAs are present in nearly all food products. Major dietary sources include animal-derived fats such as butter and lard, high-fat dairy products (e.g., cream, cheese, full-fat cottage cheese), fatty cuts of meat (e.g., beef and pork), as well as coconut and palm oil.

**Monounsaturated Fatty Acids (MUFA)**

Monounsaturated Fatty Acids (MUFA) are often referred to as "healthy" or "good" fats due to their ability to maintain healthy high-density lipoprotein (HDL) levels while not contributing to elevations in low-density lipoprotein cholesterol (LDL-C). Although the human body can synthesize MUFAs, they are especially important in the context of recommendations to limit saturated fatty acid (SFA) intake. Consequently, MUFAs should constitute a substantial proportion of total daily fat intake.

Common sources of MUFAs include olive oil, other vegetable oils, nuts, and seeds. It is important to acknowledge, however, that MUFAs may also contain varying amounts of SFAs. Therefore, even though MUFAs are considered “healthy” fats, they should still be consumed in moderation to avoid the potential risk of elevating LDL cholesterol levels. A balanced diet that keeps fat intake below 35% of total daily energy is recommended for optimal health(6).

**Trans Fatty Acids (TFA)**

Even though trans fatty acids (TFAs) are technically a type of monounsaturated fatty acid (MUFA), it is strongly recommended that TFA intake should be kept as low as possible(6), or ideally eliminated entirely(9). TFAs are primarily formed during the industrial hydrogenation of vegetable fats, such as in the production of margarine. These fats have been shown to have

detrimental effects on cardiovascular health by lowering high-density lipoprotein (HDL) levels and raising low-density lipoprotein (LDL) levels(6), making them even more harmful to cardiovascular health than SFAs.

The main sources of TFAs include hydrogenated vegetable oils, which are found in processed foods like margarine, cakes, cookies, crackers, and potato chips(9).

**Polyunsaturated Fatty Acids (PUFAs)**

Polyunsaturated fatty acids (PUFAs) are divided into two main families: omega-3 (n-3) and omega-6 (n-6). Some PUFAs are classified as essential fatty acids (EFAs) because the human body cannot synthesize them, although they can be enzymatically modified(6).

**Omega-3 Fatty Acids**

Alpha-linolenic acid (ALA) is the essential omega-3 fatty acid that must be obtained through the diet, with a recommended intake of 0.5% of total energy(6). ALA serves as a precursor to the longer-chain omega-3s, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

ALA can be found in plant-based sources such as flaxseeds, chia seeds, hemp seeds, walnuts, and oils like flaxseed, canola, and soybean oil.

Although EPA and DHA are also considered essential fatty acids, they can be synthesized from ALA in the body. However, the conversion efficiency is relatively low, with only about 5-21% of ALA being converted to EPA and 2-9% to DHA. Consequently, the recommended intake for healthy adults is a combined 250 mg of EPA and DHA daily(6).

EPA and DHA are primarily found in fatty marine fish, fish oil, and marine microalgae oil.

In general, omega-3 fatty acids do not affect LDL cholesterol levels. However, high doses of omega-3 fatty acids have been shown to raise LDL cholesterol levels in individuals with hypertriglyceridemia(10). As a result, the National Cholesterol Education Program (NCEP) guidelines do not recommend the use of fish oils for lowering cholesterol, though they do encourage the consumption of fish as part of both Step I and Step II diets.

### Omega-6 Fatty Acids

Linoleic acid (LA) is an omega-6 fatty acid classified as an essential fatty acid (EFA). It helps reduce LDL cholesterol and triglyceride levels while increasing HDL cholesterol. The recommended intake for LA is 4% of total energy(6).

LA is primarily found in oilseed plants and their oils, including canola, sunflower, corn, and soybean oils.

Arachidonic acid (ARA), also part of the omega-6 family, can be synthesized from LA, therefore there is no established dietary requirement for it(6).

ARA is mainly found in animal-based products, particularly egg yolks.

**Dietary cholesterol**

Dietary cholesterol increases LDL-C levels, with an estimated rise of 0.05–0.5 mmol/L for every 100 mg consumed(6). While its impact on LDL-C is less pronounced than that of saturated fatty acids (SFAs), it is important to note that about three-quarters of the cholesterol in the human body is produced endogenously(6), making dietary cholesterol intake unnecessary.

The main sources of dietary cholesterol include high-fat animal products, such as butter, lard, eggs, and organ meats.

In contrast, epidemiological studies and meta-analyses discussed in the article “Is There a Correlation between Dietary and Blood Cholesterol?”(11) indicate that dietary cholesterol does not have a direct impact on blood cholesterol levels. However, it is important to note that the majority of the existing research on dietary cholesterol uses eggs as its primary food source. This highlights the need for further research that would explore other dietary sources of cholesterol and their impact on plasma LDL-C.

**2. Exercise**

The article “Effects of Diet and Exercise in Men and Postmenopausal Women with Low HDL Cholesterol and High LDL Cholesterol”(7) examined the impact of exercise alone, diet alone, and their combination on reducing serum LDL-C levels. Plasma LDL cholesterol levels of participants were greater than 125 mg per deciliter but below 210 mg per deciliter for women and below 190 mg per deciliter for men.

The article discusses the NCEP Step 2 diet, developed by the National Cholesterol Education Program (NCEP). This diet recommends limiting total fat intake to less than 30%, saturated fat to under 7%, and cholesterol to less than 200 mg per day. Saturated fats were intended to be replaced with complex carbohydrates, low-fat dairy products, and lean meats.

The exercise regimen involved aerobic activity three times per week, with each session lasting one hour. Participants were required to complete at least 16 km of fast-paced walking or jogging weekly.

Unfortunately, the NCEP Step 2 diet alone did not lower LDL cholesterol levels in men or women with high-risk lipoprotein profiles. Similarly, the group that exercised but did not modify their diet, failed to lower their LDL-C levels. “The diet-plus-exercise group” was found to reduce LDL cholesterol levels by 15-20%(7). A decrease of 14.5+/-22.2 mg per deciliter was observed among women and a decrease of 20.0+/-17.3 mg per deciliter was noted among men. This emphasizes the essential role of physical activity in managing high LDL cholesterol, but only when paired with nutritional modifications.

The article “Effectiveness of Low to Moderate Physical Exercise Training on the Level of Low-Density Lipoproteins: A Systematic Review“(12) investigated the impact of exercise alone on reducing serum LDL-C levels. It pooled nearly 800 participants from 11 studies. Most participants were of Caucasian ethnicity aged 18-75 with lower physical activity profiles, their LDL levels were mostly normal or high borderline. In most cases the intervention

involved 30-45 min of supervised aerobic exercise with warm-up and cool-down phases 3-5 times a week for a period ranging from 8-24 weeks. The exercise was the equivalent of nearly 20 km of walking or jogging per week. Unfortunately, in most cases study showed no significant reduction in the levels of LDL, which means that exercise alone cannot result in therapeutic outcomes in lowering of LDL-C levels. However, a positive effect on LDL subfractions was observed. And again, in one study that the review included, significant changes in LDL-C levels were observed in the exercise group that applied dietary modifications compared to exercise alone.

**3. Weight reduction**

Weight reduction in overweight individuals is associated with lower LDL-C levels. Even a modest weight loss of 5–10% significantly reduces LDL-C levels(13). A meta-analysis of 70 studies(14) confirmed this link, demonstrating that weight loss leads to substantial reductions in LDL cholesterol (LDL-C). The relationship between weight loss and decreased LDL-C was statistically significant ($P \leq 0.001$), with a correlation coefficient ($r = 0.29$), indicating a moderate association.

However, dyslipidemia increases the risk of cardiovascular disease (CVD) even in non-obese individuals(15). Since weight gain is associated with dyslipidemia (one of its key manifestations being elevated LDL-C), it is crucial to prevent weight gain, even in those with a healthy Body Mass Index (BMI). Moreover, rather than relying solely on BMI, the Fat-to-Muscle Ratio (FMR) may serve as a more effective measure for predicting the risk of failing to achieve optimal LDL-C targets(16). FMR offers a more accurate reflection of body composition by comparing fat mass to muscle mass. The higher FMR, the greater proportion of fat relative to muscle, the higher the risk of elevated LDL-C levels.

As of 2019, 57% of the Polish population was overweight or obese(17), and approximately 60% suffered from dyslipidemia(6). Weight reduction could help prevent elevated LDL-C

levels, thereby reducing the risk of cardiovascular events such as myocardial infarction and stroke.

**4. Dairy**

The fat found in milk and dairy products contains approximately 70% saturated fatty acids (SFAs) and 2.5% trans fats(6), both of which are known to raise LDL cholesterol (LDL-C) levels and are generally recommended to be limited or even eliminated entirely. However, evidence suggests that dairy consumption does not have to impact LDL-C levels significantly(18). The article "Influence of Habitual Dairy Food Intake on LDL Cholesterol in a Population-Based Cohort"(18) examined the impact of milk and ricotta cheese consumption on LDL-C levels. Individuals who consumed higher amounts of milk tended to have lower serum LDL-C levels, whereas greater ricotta cheese intake was associated with higher LDL-C concentrations. These findings highlight the importance of further research to better understand the effects of various dairy foods on LDL-C levels.

Another study discussed in *Lifestyle Medicine*(6) demonstrated the varying impacts of different dairy products on LDL-C levels. Participants were divided into two groups: one consumed milk, while the other consumed butter, both with equivalent amounts of saturated fatty acids (SFAs). Within the milk group, individuals were further divided based on whether they consumed skimmed or full-fat milk. The full-fat milk group experienced an LDL-C increase that was 0.32 mmol/L higher than the skimmed milk group, indicating that SFAs from full-fat dairy can raise LDL cholesterol(6). In contrast, the same amount of SFAs from butter led to a significantly greater rise in LDL-C compared to cheese, with a difference of 0.22 mmol/L per 40 g of milk fat.

It is thought that the fat in milk and certain dairy products may have a less harmful effect on LDL-C levels because it is encapsulated within phospholipid-protein membranes. However, during butter production, these protective membranes are disrupted and removed along with

the buttermilk(6). This may partly explain the differences in cholesterol-raising effects among various dairy products. Nevertheless, the underlying mechanisms remain unclear and should be explored in future research.

Another possible explanation for the differing effects of various dairy products on LDL-C levels lies in the complex composition of dairy fat. It includes a mixture of short-, medium-, and long-chain saturated fatty acids, along with monounsaturated fats, polyunsaturated fats, and compounds such as butyric acid. Some of these components may neutralise the cholesterol-raising effects typically associated with saturated fats(19). Additionally, evidence suggests that not all saturated fatty acids (SFAs) affect LDL-C in the same way(20), which demonstrates that they should not be classified as a single type of fat. However, the 2017 AHA Presidential Advisory on Dietary Fats and CVD concluded that, although individual saturated fatty acids may have slightly different effects, these differences are minor and do not justify changing the current recommendation to limit saturated fat intake. This area remains not fully understood and requires further investigation.

**5. Fiber**

Dietary fiber refers to various plant-based substances that human gastrointestinal enzymes cannot digest. It is classified into two main types: soluble and insoluble fiber(21). Soluble fiber swells in the intestine, forming a gel that prolongs transit time. It also slows fat absorption and increases cholesterol excretion in the stool, helping to lower LDL cholesterol levels(6). These effects are largely attributed to short-chain fatty acids, which are produced during the digestion of soluble fiber. Major dietary sources of soluble fiber include vegetables, fruits, and legumes.

Although soluble fiber has only a modest effect on lowering total and LDL cholesterol levels(21), even small dietary changes can yield meaningful benefits. For instance, consuming just 3 grams of soluble fiber from oat bran daily may reduce total cholesterol by 5–6 mg/dL.

While the impact may appear minor, such improvements are easily achievable and should be embraced rather than overlooked. Current recommendations for adults suggest a daily intake of 20–30 grams of total fiber, with at least 25% derived from soluble fiber(8).

Since fiber is predominantly found in plant-based foods, it should not come as a surprise that plant-based diets are linked to a reduced risk of CVD. Increased fiber intake has been shown to lower LDL cholesterol, which acts as a protective measure against atherosclerosis(25). Furthermore, plant-based diets that are low in processed vegetarian foods typically result in a reduced intake of saturated fats (SFAs) and monounsaturated fats (MUFAs) but an increased consumption of polyunsaturated fats (PUFAs), which has been associated with an improved lipid profile (22).

**Conclusions**

LDL-C, often referred to as "bad" cholesterol, transports cholesterol to various tissues in the body. Elevated serum LDL-C levels increase the risk of cholesterol deposition within tissues(23), which contributes to the development of cardiovascular diseases (CVDs) such as myocardial infarction and stroke. CVDs account for the majority of deaths worldwide(24). Therefore, it is essential to address elevated LDL-C levels early, before they contribute to the onset of disease.

Lifestyle modifications such as adjustments in diet and regular physical activity can significantly lower serum LDL-C levels. Research indicates these interventions can lead to reductions of 15–20% or even more(10), depending on the approach used and the level of compliance.

Addressing elevated LDL-C levels in the future should incorporate a combination of lifestyle modifications. While some patients may require lipid-lowering pharmacological treatment, these medications should be complemented by a healthy diet, regular physical activity, and weight management to maximize therapeutic effectiveness.

## Disclosure

### Author's contribution

Conceptualization: Justyna Janikowka, Monika Wendland; Methodology: Adrianna Domańska, Krzysztof Julian Długosz; Software: Julia Piotrowska, Adrianna Witkowska; Check: Antonina Teresa Witkowska, Monika Wendland; Formal analysis: Barbara Anna Zapalska, Aleksandra Minda; Investigation: Aleksandra Łubińska-Kowalska, Agata Żak-Gontarz, Resources: Justyna Janikowska, Antonina Teresa Witkowska; Data curation: Julia Piotrowska, Barbara Anna Zapalska; Writing - rough preparation: Aleksandra Łubińska-Kowalska and Krzysztof Julian Długosz;; Writing - review and editing: Justyna Janikowska; Visualization: Adrianna Witkowska, Aleksandra Minda, Project administration: Agata Żak-Gontarz, Adrianna Domańska; Receiving funding - no specific funding.

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The authors deny any conflict of interest.

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