Ann Appl Sport Sci, 12(02): e1300, 2024.
http://www.aassjournal.com; e-ISSN: 2322–4479; p-ISSN: 2476–4981. 10.61186/aassjournal.1300

REVIEW ARTICLE

Impact of Exercise on High-Density Lipoprotein Cholesterol in Adults with Overweight and Obesity: A Narrative Review

[1,2]Sameer Badri Al-mhanna, [3]César Leão, [2]Wan Syaheedah Wan Ghazali*, [2]Mahaneem Mohamed, [4]Alexios Batrakoulis, [5]Hafeez Abiola Afolabi, [6]Bishir Daku Abubakar, [7]Monira I. Aldhahi, [8]Mehmet Gülü, [9]Fatma Hilal Yagin, [10]Pantelis T. Nikolaidis

[1]Center for Global Health Research, Saveetha Medical College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, India. [2]Department of Physiology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia. [3]Research Center in Sports Performance, Recreation, Innovation and Technology (SPRINT), Melgaço, Portugal. [4]Department of Physical Education and Sport Science, School of Physical Education, Sport Science and Dietetics, University of Thessaly, Trikala, Greece. [5]Department of General Surgery, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia. [6]Department of Human Physiology, Federal University Dutse, Jigawa State, Dutse, Nigeria. [7]Department of Rehabilitation, College of Health and Rehabilitation Sciences, Princess Nourah Bint Abdulrahman University, Riyadh, Saudi Arabia. [8]Department of Sports Management, Faculty of Sport Sciences, Kirikkale University, Kirikkale, Turkey. [9]Department of Biostatistics and Medical Informatics, Faculty of Medicine, Inonu University, Inonu, Turkey. [10]School of Health and Caring Sciences, University of West Attica, Athens, Greece.

Submitted November 01, 2023; Accepted in final form January 09, 2024.

ABSTRACT

Background. Sedentary lifestyles and insufficient physical activity contribute significantly to the rising global epidemic of obesity, fostering an environment where excess calories are stored as adipose tissue. Lack of regular physical activity and diminished cardiorespiratory fitness are key factors in the emergence of obesity-related illnesses, leading to cardiovascular and metabolic complications. However, the extent to which low levels of physical activity and obesity directly cause low HDL-C levels is uncertain, and the specific impact of obesity on reducing HDL-C as well as the associated risks are not well clarified. The influence of different types of exercise on HDL function is intricate and depends on various factors, including exercise intensity and individual characteristics. **Objectives.** This study aims to investigate the effect of exercise on HDL-C levels in overweight and obese adults, as well as review the mechanisms and pathways by which various exercise types influence HDL-C metabolism in this population. **Methods.** A literature search was conducted using the PubMed, Scopus, and Google Scholar databases. Studies were included if they were published in English. **Results.** A sequential moderate-to-high-intensity exercise regimen resulted in sustained or gradual improvements in HDL-C levels. For increased HDL-C levels, frequent moderate-to-high-intensity, long-duration exercise at an aerobic threshold in conjunction with body mass reduction and dietary modification is observed from the reviewed studies. **Conclusion.** Regular exercise can improve HDL-C levels in overweight and obese individuals. A combination of moderate-to-high-intensity, long-duration exercise at an aerobic threshold with body mass reduction and dietary modification is most effective. Overweight and obese individuals should engage in regular exercise to improve their HDL-C levels and overall health.

KEYWORDS: ***Body Weight, Physical Activity, Aerobic Exercise, Lipid Metabolism.***

*. Corresponding Author:
Wan Syaheedah Wan Ghazali, Ph.D.
E-mail: syaheeda@usm.my

INTRODUCTION

Numerous prospective epidemiological studies have indicated a consistent inverse correlation between concentrations of high-density lipoprotein cholesterol (HDL-C) in the serum and the risk of coronary heart disease (CHD) (1). However, recent evidence from genetic and clinical research has begun to challenge the longstanding belief that elevated HDL-C levels are invariably advantageous, while lower levels are consistently harmful (2). HDL-C has traditionally been recognized as "good cholesterol," playing a positive role in overall well-being and, notably, cardiovascular health (3). In obesity, the role of HDL-C is vital, since individuals with impaired lipid metabolism are very likely to develop obesity-related illness resulting in metabolic dysregulation (4, 5). In summary, abnormal HDL-C levels are associated with upregulation of glucose homeostasis and resting blood pressure promoting metabolic syndrome and cardiovascular disease among people with excessive weight (6).

A growing body of literature highlights the negative impact of obesity in altering the blood lipids (7), where certain metabolic disorders, such as raised blood pressure and decreased HDL-C (8) are the key risk factors for CVD, even though the claimed independent correlation among plasma triglycerides (TG) and CVD is more contentious (9). Notwithstanding, many studies correlate other risk factors with altered TG levels (e.g., lower HDL-C) (10). HDL-C is a proxy of CVD (11) where the risk of CVD correlates with high LDL and low HDL-C (i.e. <40 mg/dl and <50 mg/dl for men and women, respectively) levels (12). Apart from obesity, chronic inflammation and a high risk of sepsis have also been linked with lower HDL-C concentrations (13). On the other hand, physical inactivity and decreased cardiorespiratory fitness are major risks of developing obesity and CVD (14). Although the management of obesity is achievable, it demands self-perseverance and regimen compliance (15). Over the past decade, most research on obesity has highlighted the effect of several treatments, including diet, physical activity or exercise, behavioral adjustment, and pharmacological approaches in obesity management. Of most, exercise strategies have provided successful weight management and obesity prevention among the different therapeutic methods (16) and have been documented as popular trends for practitioners within the global exercise community (17). The extent of the impact of physical training on obesity should be understood due to its ample evidence of reversing the progression of certain types of CVD (16). Partaking in regular physical activity is believed to partially decrease the risk of CVD, improving blood lipid profile (18), and should be encouraged as a strategy to optimize lipid homeostasis and reduce the incidence of CVD among individuals with obesity and/or hyperlipidemia (19).

The large majority of researchers examining normolipidemic groups reported decreases in TG and rises in HDL-C following exercise. Physical exercise is one of the lifestyle factors traditionally recommended for individuals with high BMI to improve lipid metabolism, such as increasing HDL-C (20, 21). However, people with excess weight tend to present low compliance rates to various exercise interventions due to inherent obesity-related psychophysiological complications (22, 23). Strong evidence indicates dose-response associations between HDL-C and duration of exercise, with regular or even single exercise sessions positively impacting the body's cholesterol metabolism (24); however, further research is needed in this direction (25). In general, various types of exercise demonstrate beneficial alterations in the lipid profile among people with excessive weight (26-30). Research on lipids and lipoproteins response to exercise in hypercholesterolemic males showed a decrease in TC, TG, and an increase in HDL-C level (31). In this review, we aimed to analyze the mechanism responsible for changes in HDL-C metabolism and the magnitude of those changes in response to various types of exercise in overweight and obese individuals, aiming to describe the potential pathways of exercise influencing HDL-C values.

STRUCTURE OF HDL-C

Cholesterol is a crucial part of cellular membranes and precursors of steroid hormones and other essential biomolecules. Human cholesterol is derived exogenously from ingesting animal products and endogenously from the liver and other tissues. Together with TG, apoproteins, and phospholipids, these cholesterols are synthesized into complex lipoprotein molecules. The lipoproteins, along with enzymes such as lipoprotein lipase (LPL) and lecithin-cholesterol

acyltransferase, comprise a complex mechanism that delivers fatty acid and cholesterol to peripheral tissues while also returning cholesterol to the liver for conversion to bile acids and subsequent excretion (32). There are five main classes of these lipoproteins based on their lipid content, as their lower lipid content increases the particulate density (33). They include the large TG, which are considered rich particles exerted by the intestine after meals; VLDL (very low-density lipoprotein) secreted by the liver and is the most responsible for TG transport; IDL (intermediate-density lipoprotein) that is a transient lipolytic product of VLDL and a source of LDL; the LDL which is the primary particle transporting cholesterol in the plasma; and HDL that has an approximately equal amount of lipid and protein with a molecular size of about 200-400 KDa and densities 1.21 to 1.6 g per cubic centimeter (34).

HDL is the smallest lipoprotein particle 5-17 nm, the densest with the largest percentage of protein to lipids. It contains mainly cholesterol I esters, and TG with its surface containing mostly free cholesterol, phospholipids, and A, C, E apo-proteins (34). Furthermore, HDL particles are sub-classified based on size and density in a high salt solution by measuring their ultra-centrifugal flotation rate and mass concentration of the lipoprotein particles (35). Together HDLs were further subclassified into “2a, 2b, 3a, 3b, and 3c” HDLs, using gel electrophoresis and apolipoprotein AI composite (35, 36) (Figure 1). HDL is thought to have a role in the reverse transfer of cholesterol from the peripheral tissue to the liver (37). It originates from the liver and intestine that secrete discoid nascent HDL particles containing mostly apoprotein A-1 and phospholipids that grow into mature spherical HDL particles by incorporating free cholesterol from the peripheral tissues cell membrane and other lipoproteins. These cholesterol sequestering processes and lecithin- cholesterol acyltransferase enzymatic cholesterol conversion to cholesterol esters give rise to newly synthesized large spherical HDLs (with 4 or 3 apoprotein A-1) (37). The mechanism of transporting cholesterol esters to the liver is complex, involving unique membrane-bound receptors for the transfer to a lipoprotein containing both apoprotein B (38). HDL is the most important lipoprotein in terms of quantity, which has essential survival functions and can also influence different parts of the human body (39). On the other hand, HDL particles (HDLs) are essential to the 'reverse transfer of cholesterol,' a mechanism that transfers excess cholesterol contained in peripheral cells, such as foam cells, to the liver for the excretion into bile; such scavenged cholesterols are termed HDL-Cs. Reverse cholesterol transport (RCT) is the pathway by which excess cholesterol from peripheral tissues is transported for redistributed to the other tissues or the liver for onward gallbladder excretion; however, HDL-C is the primary lipoprotein implicated in this process (40).

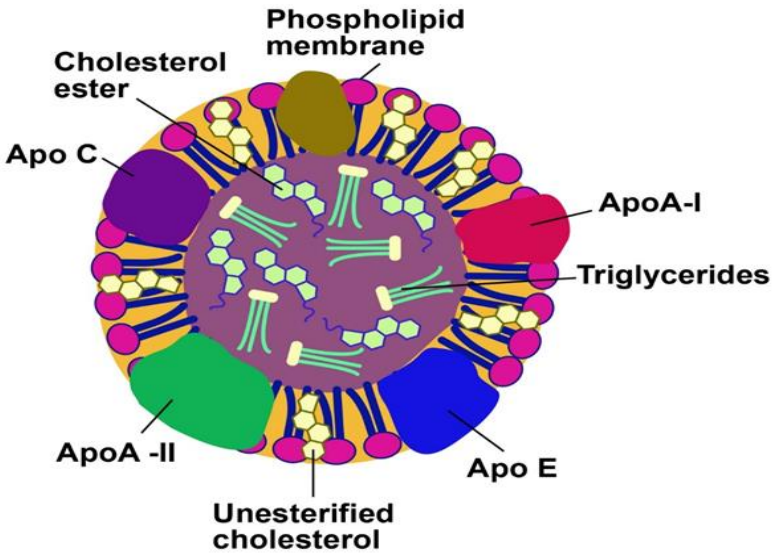

Figure 1. HDL-C structure.

HDL-C AS A RISK FACTOR

The biological function of HDL is to transport excess cellular cholesterol, and there is a possible causal relationship between cellular cholesterol content and immune function (41). It has been suggested that different components of HDL molecules may be implicated in the development of autoimmune disease from multiple mechanisms by which HDL modulates the immune response (42) where the possibility of infectious disease is associated with HDL (43) and contributes to the profound risk of autoimmune disease (42). Among the numerous lipid fractions, HDL-C holds a distinct place, also dubbed as "good cholesterol". A major risk factor for CVD in obese persons is reduced HDL-C concentration, along with increased LDL-C concentrations, which also induces HDL-C dysfunction (44). Obesity raises CVD risk by increasing TG and LDL-C and reducing HDL-C values. In that sense, HDL-C may be a good index to classify people at risk of dyslipidemia, HBP, and metabolic syndrome (MTS) (45).

Several reports have shown low serum HDL-C concentrations, defined as <1 mmol/L (40 mg/dL) in both sexes or <1 mmol/L in males and <1.3 mmol/L (50 mg/dL) in females (46), are an independent predictor for CHD in obese people and diabetic patients (47). For instance, the Emerging Risk Factors Collaboration in a record of 302,430 people without prior vascular illness revealed low HDL-C levels to correlate with an elevated risk of CVD, irrespective of other identified risk factors (48). Studies indicated that both in obese and non-obese, decreased HDL levels are linked with an elevated risk of developing CHD (49) and CVD (50). To be specific Ashen and Blumenthal (2005) (51) stated a cut-off point of an elevated risk of CHD in individuals with low HDL-C levels to be about less than 40 mg per deciliter [1.03 mmol per liter] in men and less than 50 mg per deciliter [1.30 mmol per liter] in women. It was also observed an increased risk of post-angioplasty re-stenosis and cardiovascular death especially when diabetes was present. Cardiovascular dysfunction and poor muscle strength are also linked with total cholesterol (TC), LDL-C, and HDL-C (52). An inverse relationship between HDL-C concentrations and CVD has been identified for many decades (53). Therefore improving HDL-C level has the potential to minimize the risk of CVD, CHD morbidity, and mortality (54) and in obese, CHD risk in women is reduced by 3% and 2% in men for each 1 mg/dL increase in HDL-C (2). In addition, HDLs have some extra anti-atherogenic effects apart from cholesterol homeostasis presence. It has been identified as a contributing factor in decreasing inflammation, oxidation (55), vascular thrombosis (56), enhancing endothelial activity, facilitating endothelial repair (57), and improving insulin sensitivity (58).

AUGMENTING EFFECT OF EXERCISE ON HDL-C

The impact of various modalities of exercise on HDL function is complex and depends on many variables, including the intensity and duration of exercise. For that reason, the effects of regular exercise on these measures remain unclear (59). The molecular composition of HDL particles, especially the components of proteins and lipids, is thought to mediate the biological functions of HDL (60, 61). Kannan et al., (2014) (62) indicated that moderate to intense exercise improved LDL and HDL levels. Studies have shown that concomitant improvements in HDL-C and/or apoA-I levels have been the best predictors of exercise-induced improvement in cholesterol efflux capacity (63, 64). The improvement in cholesterol efflux potential was linked positively with improvements in the overall concentration of HDL particles and the mean size of HDL (59). In a study by Wood et al. (1985), it was shown that one year of mild EX among 84 overweight and obese men induced a reduction in the mean body fat that significantly mediated an increase in plasma levels of HDL-C compared to a hypocaloric diet (65). Moderate exercise and weight reduction are reported to mediate differently and independently increased HDL-C and showed summative effect when combined (66). The evidence that body fat reductions are easier to obtain and maintain when exercising as part of a weight-loss regimen in obese people confirms the significance of exercise in raising HDL cholesterol levels when enrolled in a restricted diet for weight loss (67). Dose-response associations between the amount of exercise and improvements in blood lipids among obese people show that exercise may change blood lipids positively at low levels of training. Still, the results will not be measurable until certain exercise thresholds are reached (around 1200-2200 kcal/wk energy expenditure) (68). It was

reported that moderate-intensity exercise at 60% of heart rate reserve for a distance of 2 miles, three days a week, induces adequate changes in the HDL profile (69). Another study reported substantial HDL-C differences among male exercisers and those in the low physical activity category (~6 mg/dl) due to exercise energy expenditures of 1500 to 3000 kcal/wk. The findings also showed increasing exercise intensity could result in additional improvement of the lipid fraction since the difference in HDL-C improved by 3.5 mg/dl in those reporting exercise of energy expenditures exceeding 3000 kcal/wk (70).

Based on the rate of stated changes in HDL-C and TG levels following the exercise program, HDL-C lipid variables are more reactive to regular EX than TC and LDL-C, with significantly increased HDL-C and reduced TG values following exercise (71). Sarzynski, et al., (2018) (59) stated that six months of exercise shows little effect on cholesterol efflux capability and exchange of HDL-apoA-I (apolipoprotein A-I), with only the greatest intensity of exercise increasing radio-labeled efflux capacity, and thus frequent sustained intense exercise enhances some but not all HDL functions. Another study found significant rises in HDL-C and reductions in TC among serum lipids in abdominally obese middle-aged women following a combined 60-minute workout routine four days/week for 12 weeks (72). Similarly, large reductions in TC and substantial rises in HDL-C have been recorded in obese people after a 40-minute of aerobic exercise (4 days/week for 12 weeks) (73). Studies have also shown that the Combination Workout Regimen has effectively minimized LDL-C, TC, and TG values for 12 weeks in women with abdominal obesity and has substantially improved HDL-C levels (74). Several authors uphold that exercise can boost cardiopulmonary functions and help prevent and alleviate CVD and hyperlipidemia (75). Furthermore, the effects of 24 weeks of endurance exercise induced favorable variations in plasma lipoprotein and lipid profiles, particularly HDL particle size independent of diet and baseline or alteration in body fat (76). On the other hand, a study by Nassef et al., (2019) (77) conducted among 7797 persons, of which 3,559 males and 4,238 females aged 30-70 years, showed subjects' participation in aerobic exercise and daily badminton correlated with greater HDL-C values. In other trials, moderate routine exercise 2 days a week did not substantially improve body composition over 12 months in obese adults, and regular exercise reduced HDL-C in overweight and obese persons after 6 and 12 months of exercise instruction (78). Similarly to other research, serum cholesterol, HDL, and LDL levels in 45 poorly trained men were not significantly impacted by a 9-week ergometer training, concluding that anaerobic threshold training has no negative effect on blood lipoprotein profiles with aerobic proven beneficial (79). Swain and Franklin (2006) (80) indicated that the change in lipid profile related to exercise relies on each workout's intensity, frequency, and duration. Moreover, contradictory findings of the impact of exercise on blood lipid profiles are limited to people with typical lipid normal values (81). For instance, a study by Gordon et al., (1998) (82) conducted acute EX among 12 women aged 18–35 years by running for 24–48 km a week over six months (35.7-9.1 km/wk) showed an immediate rise in HDL-C post-exercise when uncorrected for plasma volume shifts. However, after adjusting for changes in plasma volume, they detected a more significant HDL-C response. The HDL-C level was reported to be most increased after the 48-hour HDL-C measurement. These results highlight the necessity to adjust for plasma volume when measuring absolute HDL-C concentration changes following exercise (83) (Table 1).

The mechanism responsible for the acute rise in HDL-C following exercise is likely associated with the catabolism of lipoproteins rich in triglycerides through LPL (83). Increased activity of LPL following exercise results in hydrolysis of chylomicron and VLDL, thereby decreasing plasma triglyceride levels (71), and subsequently, surface remnants are converted into nascent HDL-C; nevertheless, following exercise, the levels of HDL-C can increase. In comparison, drops in plasma TG levels following acute activity tend to correlate with the amount of intensity and duration of exercise (84). Another study showed that prolonged strenuous exercise could induce a reduction in TG levels within 24 hours (85), while shorter workout durations will lead to only moderate decreases in plasma triglycerides in obese women (86) or no improvement at all (87). In brief, there are conflicting findings regarding the changes in lipid profile in response to exercise interventions of

different intensities and durations. In that matter, we observe studies conducted in overweight and obese individuals showing different results in HDL-C levels namely, an increase (63, 64), a decrease (88), and some other studies reporting no changes in the same variable (89). Such inconsistencies in the effect of exercise on the HDL-C levels can be explained by the discrepancies observed in the experimental design. For instance, not all the experiments were controlled for collateral changes in diet, alcohol consumption, and smoking, which could significantly modify lipoprotein profiles. Furthermore, the timing of plasma collection and processing may have a profound influence on levels of HDL-C (49).

DYSFUNCTIONAL HDL-C AND DEPLETION MECHANISM IN OVERWEIGHT/OBESE INDIVIDUALS

The inverse association between HDL-C and obesity is well-reported (90, 91), but the degree to which obesity leads to low HDL-C is unknown. Moreover, obesity's relative contribution to reducing HDL-C and the estimated risks of reduced HDL-C due to obesity is unclear. Bora et al., (2017) (92) indicated that a significant percentage of diminished HDL-C values were observed in obese subjects and the risk to develop a decreased HDL-C is four (individuals with visceral obesity) to five times greater than non-obese counterparts. Therefore, obesity has a link to a significant percentage of the disease burden of low HDL-C dyslipidemia. The mechanism that can cause low HDL and apoA-I might involve the inflammation process because inflammation affects the expression of hepatic genes and contributes to changes in HDL's protein structure. The levels of apoAI and cholesterol ester transport protein on HDL are reduced throughout the inflammation, or in laboratory models in which bacterial endotoxin lipopolysaccharide is used (Figure 2). In contrast, the quantities of serum amyloid A and secretory phospholipase A2 acute-phase reactant proteins on HDL are significantly higher, these improvements are correlated with reduced HDL cholesterol acceptor activity (93). Some studies postulate antioxidant activity of HDL-C does not adequately exist. For instance, Sorrentino et al., (2010) (94) observed that patients with T2DM had low endothelial antioxidant function when HDL-C was isolated from their blood plasma; besides, reduced potential for cellular cholesterol efflux was illustrated as an additional key factor in obese patients causing cardiovascular cerebral problems. The HDL-C obesity pathway demonstrated strongly elevated free fatty acids and very high LDL; in exchange, this induces excessive RCT stimulation, further accelerating the catabolism of HDL-C (95). Compared to healthy adults, cellular cholesterol efflux ability reflected by HDL-C levels is, therefore, less informative on the state of atherosclerosis (96). Also worth mentioning are cholesterol ester transport protein LCAT, liver lipid proteinase, and protein phospholipid, which are also altered in obese patients (49). Bertière et al., (1988) (97) compared the HDL levels in obese subjects aged 68, and in the control group, the cholesterol and protein ratio of HDL in the obese subjects was 50% lower. Other studies carried out between overweight, obese women and older individuals of both sexes and varying adiposity found intra-abdominal fat as an independent indicator of low levels of HDL2-cholesterol than mid-thigh fat (98), BMI, or total percentage of body fat (99). Bora et al., (2017) (92) showed that 79.8% of the decline in HDL-C was observed in people with overweight or obesity, whereas 72.8% of the decline in HDL-C was due to increased waist circumference among centrally obese individuals. In the overall population, the burden of reduced HDL-C due to obesity was also moderately high: 57.1% of low HDL-C was due to obesity, while 36% of low HDL-C was due to central obesity. Furthermore, a lack of changing TG levels and elevations in HDL concentration were found in severely obese men and women following weight reduction by an exercise intervention. Decreased HDL levels in obesity have been attributed to both an enhancement in the uptake of HDL by adipocytes and an increase in the catabolism of apolipoprotein A-I on HDL particles. Additionally, there is a reduction in the conversion of the pre-beta1 subfraction, the initial acceptor of cholesterol from peripheral cells to pre-beta-2 particles. Conversely, as a means of reversing the reduction in HDL levels in obesity, sustained weight loss is an effective method. Alternative mechanisms may play a role in decreasing HDL-2 in obese individuals who increase cholesteryl ester transfer protein mass and high activity levels in the obese and can potentially reduce HDL levels (49).

Table 1. The Effect of Exercise on HDL-C in Overweight and Obese Individuals

Study	Participants	Intervention	Result	Reference
Effect of exercise intervention	10 obese men / 59±8 years	Cycling Walking /jogging/, 3/w/=40 min for 6month	Not significant Pre VS post 31±6 VS 32±6	(100)
Effect of exercise intervention	n=22 obese women that completed endurance training; n=17 obese women that completed combined training	Endurance (60–80% HRmax) and combined (20 min of strength exercises, 50–60% 1 RM and 25 min of endurance training, 60–80% HRmax) training groups underwent a 3-mo physical training program	In both groups, but especially in women undergoing combined training, a decrement in visceral adiposity index was observed together with a decrement in plasma atherogenic index, TC, and LDL-C. Conversely, an HDL-C increment was observed	(101)
Effect of exercise intervention	Basketball (n=10), swimming (n=9), long distance (LD) running (n=23) and wrestling (n=35); also a group of non-athletes as controls (n=19).	Participants trained at least 2 h/day for more than 3 years	Immediately after a maximal effort, all groups (controls included) showed significant HDL-C increases from rest values, while LD running showed the highest values. HDL2 levels increased in LD running, basketball and wrestling, while HDL3 ones in all groups and controls ($p<0.001$)	(102)
Effect of exercise intervention	Male Army recruits (n=115, age 22±0.3 years)	Completed a 12-WK moderate-intensity exercise program. A subset of 51 subsequently completed a 15-WKk high-intensity exercise program.	Moderate-intensity exercise increased HDL-C and apolipoprotein A-I levels (6.6%, 11.6% respectively), and decreased low-density lipoprotein cholesterol and apolipoprotein B levels (7.2%, 4.9% respectively) (all $P<0.01$). HDL-C and apolipoprotein A-I levels further increased by 8.2% ($P<0.001$) and 6.3% ($P<0.05$) after high-intensity exercise.	(103)
Effects of exercise intervention	Thirteen obese females. 38±5.37 years	For =14 months 4-5 times /WK 90 mis sessions included cycling, swimming, Walking jogging, and aerobic dance	Significant increase pre-VS post 0.60±0.08 VS 0.85±0.24	(104)
Effects of exercise intervention	Thirty-one obese females. Aged 35.4±5.1 years	Exercised for 90 minutes 55% of VO2max .4-5 times /WK for 6 months	1.17±0.24 VS 1.15±0.21	(105)
Effects of resistance training	Sixteen obese females, and the age from 30-40 years	Resistance training; 12wk; for 3 times /WK Performing 3 sets, of 6 to 8 repetitions	No significant change in HDL-C level after exercise intervention	(106)
Effect of exercise intervention	Eleven obese Females: aged 37. 1±I .2 years	Aerobic exercise for 5 WK 5 times /WK	Significant different pre-VS post 1.54±0.10 VS 1.64±0.08	(107)
Effect of exercise tolerance	Seven obese females. Aged 21.0±0.8 years	Aerobic exercise for 6WK included Jogging, rowing cycling and walking. three times /WK for 50 minutes/ session	No significant change HDL-C	(108)
Effect of exercise intervention	8 Obese adolescents. Aged 14.9±3.6 years DR, HA	Ten months of supervised exercise intervention	Significant increase in HDL level after ten-month intervention pre-VS post 42.13±4.04 VS 48.50±4.63	(109)
Effect of exercise intervention	Twenty tow Obese men. Aged range 46–76 years means 62.8 years	Daily treadmill walks at 70–85% of HR max for 45–60 min. for Three WK	A decrease in HDL-C pre VS post 43.7 (10.4) 39.4 (10.1)	(110)
Effect of combined exercise	Ten obese women. Aged 57.20±2.57 years	Aerobic and resistance exercise for 12 WK. for the aerobic Running 1-6 WK HRR 40-55% 7-12 WK HRR 56-75% for 3 times weekly for 40 mins.	Significant increase in HDL-C level pre VS post 50.97±3.86 VS 55.74±2.33	(111)

		For resistance exercise In WK 1–6, 60% of 1RM was used for 8 to 10 RP. In WK 7–12, 70% 1RM was used for 10–12 RP. Each session lasted for 30 MN 3 times a WK.		
Effect of aerobic exercise	21 obese females. Aged 45.6±1.1 years	Aerobic exercise for 12WK (5 times weekly 45 MN sessions/ WK)	Significant increase in HDL-C level pre VS post 1.29±0.06 VS 1.34±0.07	(112)
Effect of Endurance Exercise Training	Fifteen moderately obese men. Aged 59±6 years	Endurance exercise intervention for 9 months. exercised included cycling, treadmills, and ski machines 3 times weekly. The duration and intensity of exercise 45 to 60 MN	Significant increase in HDL-C level pre VS post 0.85±0.16 VS 0.91±0.18	(113)
Effect of resistance exercise o	Twenty-six elderly obese females. Aged 70.92±6.60 years	Resistance exercise for 12 WK. using elastic bands, leg raise, shoulder bridge For 40- MN.	Not significant in HDL-C Pre VS post 50.08±8.55 VS 52.23±12.25	(114)
Effect of wand stretching exercise program	124 overweight and obese males and females. Aged 62+4 50/10	Wand stretching exercise program for 15 The exercise is performed in the standing position while holding a 770 g wand. Stretching of upper and lower body or 40 min per day, 5 days per week	Significant increase in HDL-C level pre VS post Ha 55.3±2.57 VS 59.6±2.68	(115)
Effect of low-intensity exercise program	Twenty-two obese males and females. Aged 43.4±2.3 years	low-intensity exercise for 4 MO. 2 weekly sessions of 25 MN	Significant increase in HDL-C level pre VS post 40.9 (1.6) VS 46.2 (1.9)	(116)

RP=repetitions, WK=week, MO=month, MN=minute.

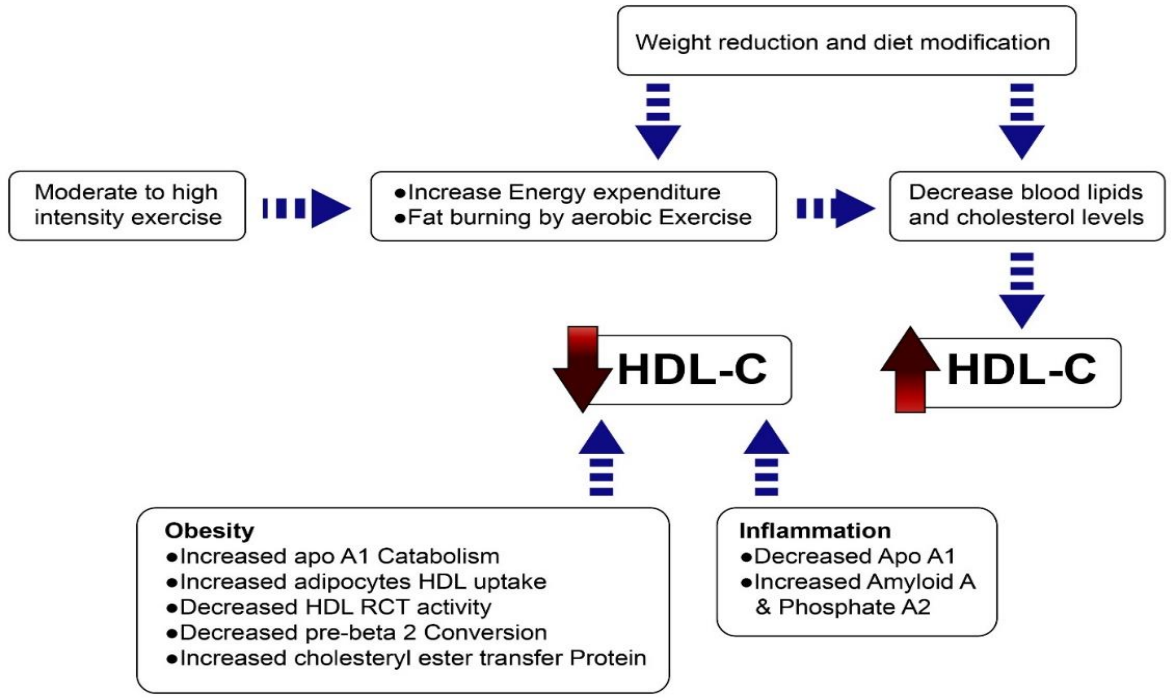

Figure 2. The influence of exercise on HDL-C in obesity and overweight.

ROLE OF EXERCISE INTENSITY, DURATION, AND FREQUENCY ON HDL-C CONCENTRATION

Daily activity can be an exciting technique to modulate lipid profile and work as a prevention feature, preventing disease risk due to exercise's anti-inflammatory and anti-atherogenic features, or it can be used as a therapeutic intervention. In addition, the ability to monitor multiple training variables, such as mode, intensity, duration, and recovery interval, can be used to optimize the benefits of cardiovascular health promotion exercise and to increase HDL-C (117). There is also evidence that changes in HDL-C levels can be met with more efficacy when following principles of training, like individualization, specificity, progression, and loading. A study performed by Leclerc et al., (1985) (118) suggested a minimum level of habitual exercise duration with an energy expenditure of 5 to 6 METs or more as the criterion for beneficial improvements in HDL-C. Nieman et al., (2002) (119) investigated the effect of aerobic exercise for 12 weeks in 21 obese females aged 45.6±1.1 (5 days/wk, 45-min sessions) and found that a significant increase in HDL-C level pre vs. post 1.29±0.06 vs. 1.34±0.07. Mogharnasi et al., (2014) (120) revealed that engaging in both endurance and resistance exercises three days/week over an eight-week period resulted in a significant elevation of HDL-C levels. A study by Sigal et al., (2007) (121) found that a 22-week combined aerobic and resistance training program significantly increased HDL-C levels in obese patients. Azarbayjani et al., (2014) (122) examined the effect of combined aerobic and resistance training for 16 weeks among obese women, the results showed significant reductions in TC, LDL-C, and TG, while increasing HDL-C levels.

In contrast, the impact of 12 weeks of resistance exercise in 26 elderly obese females aged 70.92±6.60 using elastic bands for 40 min did not change HDL-C levels pre vs. post (50.1±8.6 vs. 52.2±12.3) (123). A recent systematic review and meta-analysis comprising 13 studies with a collective enrolment of 2,454 patients showed that the combination of aerobic exercise and diet did not lead to an elevation in HDL-C levels among obese type 2 diabetes patients (124).

It was reported that plasma HDL-C levels did not improve with endurance running in stable, middle-aged, sedentary men unless they ran at least 10 min/wk for nine months at 70%–85% of the maximum heart rate (125).

On the other hand, the exercise intensity threshold has also been implicated in influencing HDL-C levels. Volunteers who exercised at 75% of the maximum heart rate for 12 weeks had increased HDL-C levels, but no changes were reported in those who exercised at 65% of the maximum heart rate (126). A more remarkable rise in HDL-C values is reported in longer duration low-intensity exercises rather than short duration high intensity was observed when considering the relationship between exercise intensity and duration (127). In addition, the running distance was more strongly related to HDL-C than running velocity, Williams (1998) (128) indicating that exercise duration is a more significant contributor to HDL-C levels than training intensity, recorded that weekly running distance was positively related to HDL-C levels in women. Regardless of confounding variables such as food, alcohol consumption, and body weight, running time is often linked to HDL-C levels. In short, as correlated with body weight, physical exercise often tends to have an independent effect on health-related performance, indicating that sufficient fitness levels will mitigate the detrimental impact of body weight on health outcomes. Thus, to promote the adoption and maintenance of an appropriate amount of physical exercise to regulate body weight and increase the HDL-c level in obese individuals, it is essential to target exercise intervention strategies.

CONCLUSION

Physical inactivity and decreased cardiorespiratory fitness led to the possibility of developing obesity and CVD. Obesity has been related to low levels of HDL-C, which is an independent risk factor for potential CVD. Lifestyle improvements, including regular exercise, should provide a systematic approach to reaching optimum HDL-C levels. Exercise protocols characterized by moderate to high intensity appear promising for sustaining or gradually elevating HDL-C levels. In particular, such protocols with a high-volume approach in conjunction with weight loss and favorable dietary modifications seem to be the optimal strategy for improving HDL-C. Nevertheless, further studies with more participants and other

forms of exercise, in type and duration, are needed to better establish the impact of these interventions. In conclusion, regular exercise is a beneficial tool for people with excessive weight seeking to improve their blood lipid profile, enhance the immune system, reduce body mass, and lower cardiovascular disease risk factors. Thus, such favorable alterations can play a vital role in reducing obesity-related illness.

APPLICABLE REMARKS

- Exercise protocols characterized by moderate to high intensity appear promising for sustaining or gradually elevating HDL-C levels.
- High-volume training protocols in conjunction with weight loss and favorable dietary modifications seem to be the optimal strategy for improving HDL-C.
- Further research with larger sample sizes investigating other types of exercise characterized by varied training parameters is needed to better establish the impact of exercise on HDL-C.

ACKNOWLEDGMENTS

School of Medical Sciences of the Universiti Sains Malaysia, grant number 304.PPSP.6315639.

AUTHORS' CONTRIBUTIONS

Study concept and design: Sameer Badri Al-mhanna, Alexios Batrakoulis, Hafeez Abiola Afolabi. Acquisition of data: Sameer Badri Al-mhanna. Analysis and interpretation of data: Bishir Daku Abubakar, Monira I. Aldhahi. Drafting the manuscript: Sameer Badri Al-mhanna, Bishir Daku Abubakar. Critical revision of the manuscript for important intellectual content: Wan Syaheedah Wan Ghazali, Mahaneem Mohamed. Administrative, technical, and material support: Mehmet Gülü, Pantelis T. Nikolaidis, César Leão. Study supervision: Wan Syaheedah Wan Ghazali, Mahaneem Mohamed, Fatma Hilal Yagin, Bishir Daku Abubakar.

CONFLICT OF INTEREST

No potential conflict of interest was reported by the authors.

REFERENCES

1. Afolabi, H.A., et al., The relationship between obesity and other medical comorbidities. Obesity Medicine, 2020. 17: p. 100164. [doi:10.1016/j.obmed.2019.100164]
2. Kosmas, C.E., et al., High-density lipoprotein (HDL) functionality and its relevance to atherosclerotic cardiovascular disease. Drugs Context, 2018. 7: p. 212525. [doi:10.7573/dic.212525] [PMid:29623098]
3. Jomard, A. and E. Osto, High Density Lipoproteins: Metabolism, Function, and Therapeutic Potential. Front Cardiovasc Med, 2020. 7: p. 39. [doi:10.3389/fcvm.2020.00039] [PMid:32296714]
4. Endo, Y., M. Fujita, and K. Ikewaki, HDL Functions-Current Status and Future Perspectives. Biomolecules, 2023. 13(1). [doi:10.3390/biom13010105] [PMid:36671490]
5. Afolabi, H.A., et al., Obesity: A Prerequisite for Major Chronic Illnesses, in Obesity-Recent Insights and Therapeutic Options. 2023, IntechOpen.
6. Stadler, J.T. and G. Marsche, Obesity-Related Changes in High-Density Lipoprotein Metabolism and Function. Int J Mol Sci, 2020. 21(23). [doi:10.3390/ijms21238985] [PMid:33256096]
7. Klop, B., J.W.F. Elte, and M. Castro Cabezas, Dyslipidemia in obesity: mechanisms and potential targets. Nutrients, 2013. 5(4): p. 1218-1240. [doi:10.3390/nu5041218] [PMid:23584084]
8. Stadler, J.T. and G. Marsche, Obesity-related changes in high-density lipoprotein metabolism and function. International journal of molecular sciences, 2020. 21(23): p. 8985. [doi:10.3390/ijms21238985] [PMid:33256096]
9. Shahid, S.U. and S. Sarwar, The abnormal lipid profile in obesity and coronary heart disease (CHD) in Pakistani subjects. Lipids in health and disease, 2020. 19(1): p. 1-7. [doi:10.1186/s12944-020-01248-0] [PMid:32290855]
10. Tambalis, K., et al., Responses of blood lipids to aerobic, resistance, and combined aerobic with resistance exercise training: a systematic review of current evidence. Angiology, 2009. 60(5): p. 614-632. [doi:10.1177/0003319708324927] [PMid:18974201]
11. Yusuf, S., et al., Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. The lancet, 2004. 364(9438): p. 937-952. [doi:10.1016/S0140-6736(04)17018-9] [PMid:15364185]

12. März, W., et al., HDL cholesterol: reappraisal of its clinical relevance. Clinical Research in Cardiology, 2017. 106: p. 663-675. [doi:10.1007/s00392-017-1106-1] [PMid:28342064]
13. Parhofer, K.G., Interaction between glucose and lipid metabolism: more than diabetic dyslipidemia. Diabetes & metabolism journal, 2015. 39(5): p. 353-362. [doi:10.4093/dmj.2015.39.5.353] [PMid:26566492]
14. Heymsfield, S.B. and T.A. Wadden, Mechanisms, pathophysiology, and management of obesity. New England Journal of Medicine, 2017. 376(3): p. 254-266. [doi:10.1056/NEJMra1514009] [PMid:28099824]
15. Hills, A.P., L.B. Andersen, and N.M. Byrne, Physical activity and obesity in children. British journal of sports medicine, 2011. 45(11): p. 866-870. [doi:10.1136/bjsports-2011-090199] [PMid:21836171]
16. Balady, G.J., et al., Core components of cardiac rehabilitation/secondary prevention programs: 2007 update: A scientific statement from the american heart association exercise, cardiac rehabilitation, and prevention committee, the council on clinical cardiology; the councils on cardiovascular nursing, epidemiology and prevention, and nutrition, physical activity, and metabolism; and the american association of cardiovascular and pulmonary rehabilitation. 2007. 115(20): p. 2675-2682. [doi:10.1161/CIRCULATIONAHA.106.180945] [PMid:17513578]
17. Kercher, V.M., et al., 2023 Fitness Trends from Around the Globe. ACSMs Health Fit J, 2023. 27(1): p. 19-30. [doi:10.1249/FIT.0000000000000836]
18. Durstine, J.L., et al., Blood lipid and lipoprotein adaptations to exercise: a quantitative analysis. Sports medicine, 2001. 31: p. 1033-1062. [doi:10.2165/00007256-200131150-00002] [PMid:11735685]
19. Superko, H., The most common cause of coronary heart disease can be successfully treated by the least expensive therapy-exercise. ACSM Certified. News, 1998. 8: p. 1-5.
20. Kelley, G.A. and K.S. Kelley, Effects of diet, aerobic exercise, or both on non-HDL-C in adults: a meta-analysis of randomized controlled trials. Cholesterol, 2012. 2012. [doi:10.1155/2012/840935] [PMid:23198142]
21. Lemos, G.d.O., R.S. Torrinhas, and D.L. Waitzberg, Nutrients, physical activity, and mitochondrial dysfunction in the setting of metabolic syndrome. Nutrients, 2023. 15(5): p. 1217. [doi:10.3390/nu15051217] [PMid:36904216]
22. Gilyana, M., A. Batrakoulis, and V. Zisi, Physical Activity, Body Image, and Emotional Intelligence Differences in Adults with Overweight and Obesity. Diseases, 2023. 11(2). [doi:10.3390/diseases11020071] [PMid:37218884]
23. Batrakoulis, A. and I.G. Fatouros, Psychological Adaptations to High-Intensity Interval Training in Overweight and Obese Adults: A Topical Review. Sports (Basel), 2022. 10(5). [doi:10.3390/sports10050064] [PMid:35622474]
24. Drygas, W., et al., Long-term effects of different physical activity levels on coronary heart disease risk factors in middle-aged men. International journal of sports medicine, 2000. 21(04): p. 235-241. [doi:10.1055/s-2000-309] [PMid:10853693]
25. Batrakoulis, A., et al., Dose-response effects of high-intensity interval neuromuscular exercise training on weight loss, performance, health and quality of life in inactive obese adults: Study rationale, design and methods of the DoIT trial. Contemp Clin Trials Commun, 2019. 15: p. 100386. [doi:10.1016/j.conctc.2019.100386] [PMid:31193901]
26. Batrakoulis, A., Psychophysiological Adaptations to Yoga Practice in Overweight and Obese Individuals: A Topical Review. Diseases, 2022. 10(4). [doi:10.3390/diseases10040107] [PMid:36412601]
27. Batrakoulis, A., Psychophysiological Adaptations to Pilates Training in Overweight and Obese Individuals: A Topical Review. DISEASES, 2022. 10(4). [doi:10.3390/diseases10040071] [PMid:36278570]
28. Batrakoulis, A., Role of Mind-Body Fitness in Obesity. Diseases, 2022. 11(1). [doi:10.3390/diseases11010001] [PMid:36648866]
29. Batrakoulis, A., A.Z. Jamurtas, and I.G. Fatouros, High-Intensity Interval Training in Metabolic Diseases: Physiological Adaptations. ACSM's Health & Fitness Journal, 2021. 25(5): p. 54-59. [doi:10.1249/FIT.0000000000000703]

30. Batrakoulis, A., et al., Comparative Efficacy of 5 Exercise Types on Cardiometabolic Health in Overweight and Obese Adults: A Systematic Review and Network Meta-Analysis of 81 Randomized Controlled Trials. Circ Cardiovasc Qual Outcomes, 2022: p. 101161CIRCOUTCOMES121008243. [doi:10.1161/CIRCOUTCOMES.121.008243] [PMid:35477256]
31. Grandjean, P.W., S.F. Crouse, and J.J. Rohack, Influence of cholesterol status on blood lipid and lipoprotein enzyme responses to aerobic exercise. Journal of Applied Physiology, 2000. 89(2): p. 472-480. [doi:10.1152/jappl.2000.89.2.472] [PMid:10926628]
32. Lund-Katz, S. and M.C. Phillips, High density lipoprotein structure-function and role in reverse cholesterol transport. Subcell Biochem, 2010. 51: p. 183-227. [doi:10.1007/978-90-481-8622-8_7] [PMid:20213545]
33. Kasia, B.E., V.Y. Nyondia, and P.O. Oseajeh, Evaluation of lipid profile pattern among apparently healthy students of Niger Delta University. Annals of Tropical Pathology, 2020. 11(2): p. 146.
34. Kunitake, S.T., K. La Sala, and J.P. Kane, Apolipoprotein AI-containing lipoproteins with pre-beta electrophoretic mobility. Journal of lipid research, 1985. 26(5): p. 549-555. [doi:10.1016/S0022-2275(20)34341-8] [PMid:3926924]
35. Rosenson, R.S., et al., HDL measures, particle heterogeneity, proposed nomenclature, and relation to atherosclerotic cardiovascular events. Clinical chemistry, 2011. 57(3): p. 392-410. [doi:10.1373/clinchem.2010.155333] [PMid:21266551]
36. Arungovind, G., A. Kamalanathan, and K. Venkataraman, Atherogenic Dyslipoproteinemia in Type 2 Diabetes Mellitus. Mechanisms of Vascular Defects in Diabetes Mellitus, 2017: p. 451-467. [doi:10.1007/978-3-319-60324-7_20]
37. Trajkovska, K.T. and S. Topuzovska, High-density lipoprotein metabolism and reverse cholesterol transport: strategies for raising HDL cholesterol. Anatolian journal of cardiology, 2017. 18(2): p. 149. [doi:10.14744/AnatolJCardiol.2017.7608] [PMid:28766509]
38. Khovidhunkit, W., et al., Effects of infection and inflammation on lipid and lipoprotein metabolism: mechanisms and consequences to the host. The Journal of Lipid Research, 2004. 45(7): p. 1169-1196. [doi:10.1194/jlr.R300019-JLR200] [PMid:15102878]
39. Madsen, C.M. and B.G. Nordestgaard, Is it time for new thinking about high-density lipoprotein? 2018, Am Heart Assoc. p. 484-486. [doi:10.1161/ATVBAHA.118.310727] [PMid:29467220]
40. Chapman, M.J., et al., Cholesteryl ester transfer protein: at the heart of the action of lipid-modulating therapy with statins, fibrates, niacin, and cholesteryl ester transfer protein inhibitors. European heart journal, 2010. 31(2): p. 149-164. [doi:10.1093/eurheartj/ehp399] [PMid:19825813]
41. Ito, A., et al., Cholesterol accumulation in CD11c+ immune cells is a causal and targetable factor in autoimmune disease. Immunity, 2016. 45(6): p. 1311-1326. [doi:10.1016/j.immuni.2016.11.008] [PMid:28002731]
42. Catapano, A.L., et al., HDL in innate and adaptive immunity. Cardiovascular research, 2014. 103(3): p. 372-383. [doi:10.1093/cvr/cvu150] [PMid:24935428]
43. Madsen, C.M., et al., U-shaped relationship of HDL and risk of infectious disease: two prospective population-based cohort studies. European heart journal, 2018. 39(14): p. 1181-1190. [doi:10.1093/eurheartj/ehx665] [PMid:29228167]
44. Rader, D.J. and H.H. Hobbs, Disorders of lipoprotein metabolism. Harrisons principles of internal medicine, 2005. 16(2): p. 2286.
45. Quijada, Z., et al., The triglyceride/HDL-cholesterol ratio as a marker of cardiovascular risk in obese children; association with traditional and emergent risk factors. Pediatric diabetes, 2008. 9(5): p. 464-471. [doi:10.1111/j.1399-5448.2008.00406.x] [PMid:18507788]
46. Chapman, M.J., et al., Triglyceride-rich lipoproteins and high-density lipoprotein cholesterol in patients at high risk of cardiovascular disease: evidence and guidance for management. Eur Heart J, 2011. 32(11): p. 1345-61. [doi:10.1093/eurheartj/ehr112] [PMid:21531743]
47. Kontush, A. and M.J. Chapman, Functionally defective high-density lipoprotein: a new therapeutic target at the crossroads of dyslipidemia, inflammation, and atherosclerosis. Pharmacol Rev, 2006. 58(3): p. 342-74. [doi:10.1124/pr.58.3.1] [PMid:16968945]
48. Di Angelantonio, E., et al., Major lipids, apolipoproteins, and risk of vascular disease. Jama, 2009. 302(18): p. 1993-2000. [doi:10.1001/jama.2009.1619] [PMid:19903920]

49. Rashid, S. and J. Genest, Effect of obesity on high-density lipoprotein metabolism. Obesity (Silver Spring), 2007. 15(12): p. 2875-88. [doi:10.1038/oby.2007.342] [PMid:18198293]
50. Ahmed, H.M., et al., Primary Low Level of High-Density Lipoprotein Cholesterol and Risks of Coronary Heart Disease, Cardiovascular Disease, and Death: Results From the Multi-Ethnic Study of Atherosclerosis. Am J Epidemiol, 2016. 183(10): p. 875-83. [doi:10.1093/aje/kwv305] [PMid:27189327]
51. Ashen, M.D. and R.S. Blumenthal, Clinical practice. Low HDL cholesterol levels. N Engl J Med, 2005. 353(12): p. 1252-60. [doi:10.1056/NEJMcp044370] [PMid:16177251]
52. Golbidi, S. and I. Laher, Exercise induced adipokine changes and the metabolic syndrome. J Diabetes Res, 2014. 2014: p. 726861. [doi:10.1155/2014/726861] [PMid:24563869]
53. Mazidi, M., D.P. Mikhailidis, and M. Banach, Associations between risk of overall mortality, cause-specific mortality and level of inflammatory factors with extremely low and high high-density lipoprotein cholesterol levels among American adults. Int J Cardiol, 2019. 276: p. 242-247. [doi:10.1016/j.ijcard.2018.11.095] [PMid:30473336]
54. Chapman, M.J., et al., Raising high-density lipoprotein cholesterol with reduction of cardiovascular risk: the role of nicotinic acid--a position paper developed by the European Consensus Panel on HDL-C. Curr Med Res Opin, 2004. 20(8): p. 1253-68. [doi:10.1185/030079904125004402] [PMid:15324528]
55. Barter, P.J., et al., Antiinflammatory properties of HDL. Circ Res, 2004. 95(8): p. 764-72. [doi:10.1161/01.RES.0000146094.59640.13] [PMid:15486323]
56. Mineo, C., et al., Endothelial and antithrombotic actions of HDL. Circ Res, 2006. 98(11): p. 1352-64. [doi:10.1161/01.RES.0000225982.01988.93] [PMid:16763172]
57. Tso, C., et al., High-density lipoproteins enhance progenitor-mediated endothelium repair in mice. Arterioscler Thromb Vasc Biol, 2006. 26(5): p. 1144-9. [doi:10.1161/01.ATV.0000216600.37436.cf] [PMid:16528007]
58. Fryirs, M.A., et al., Effects of high-density lipoproteins on pancreatic beta-cell insulin secretion. Arterioscler Thromb Vasc Biol, 2010. 30(8): p. 1642-8. [doi:10.1161/ATVBAHA.110.207373] [PMid:20466975]
59. Sarzynski, M.A., et al., Effects of Increasing Exercise Intensity and Dose on Multiple Measures of HDL (High-Density Lipoprotein) Function. Arterioscler Thromb Vasc Biol, 2018. 38(4): p. 943-952. [doi:10.1161/ATVBAHA.117.310307] [PMid:29437573]
60. Shah, A.S., et al., Proteomic diversity of high density lipoproteins: our emerging understanding of its importance in lipid transport and beyond. J Lipid Res, 2013. 54(10): p. 2575-85. [doi:10.1194/jlr.R035725] [PMid:23434634]
61. Kontush, A., M. Lhomme, and M.J. Chapman, Unraveling the complexities of the HDL lipidome. J Lipid Res, 2013. 54(11): p. 2950-63. [doi:10.1194/jlr.R036095] [PMid:23543772]
62. Kannan, U., et al., Effect of exercise intensity on lipid profile in sedentary obese adults. J Clin Diagn Res, 2014. 8(7): p. Bc08-10. [doi:10.7860/JCDR/2014/8519.4611] [PMid:25177556]
63. Boyer, M., et al., Impact of a one-year lifestyle modification program on cholesterol efflux capacities in men with abdominal obesity and dyslipidemia. Am J Physiol Endocrinol Metab, 2018. 315(4): p. E460-e468. [doi:10.1152/ajpendo.00127.2018] [PMid:29870675]
64. Furuyama, F., et al., Effects of Cardiac Rehabilitation on High-Density Lipoprotein-mediated Cholesterol Efflux Capacity and Paraoxonase-1 Activity in Patients with Acute Coronary Syndrome. J Atheroscler Thromb, 2018. 25(2): p. 153-169. [doi:10.5551/jat.41095] [PMid:28855433]
65. Wood, P.D., et al., The effects on plasma lipoproteins of a prudent weight-reducing diet, with or without exercise, in overweight men and women. N Engl J Med, 1991. 325(7): p. 461-6. [doi:10.1056/NEJM199108153250703] [PMid:1852180]
66. Sopko, G., et al., The effects of exercise and weight loss on plasma lipids in young obese men. Metabolism, 1985. 34(3): p. 227-36. [doi:10.1016/0026-0495(85)90005-8] [PMid:3974451]
67. Martin, J.E. and P.M. Dubbert, Exercise applications and promotion in behavioral medicine: current status and future directions. J Consult Clin Psychol, 1982. 50(6): p. 1004-17. [doi:10.1037/0022-006X.50.6.1004] [PMid:7174968]
68. Durstine, J.L., et al., Blood lipid and lipoprotein adaptations to exercise: a quantitative analysis. Sports Med, 2001. 31(15): p. 1033-62. [doi:10.2165/00007256-200131150-00002] [PMid:11735685]

69. Spate-Douglas, T. and R.E. Keyser, Exercise intensity: its effect on the high-density lipoprotein profile. Arch Phys Med Rehabil, 1999. 80(6): p. 691-5. [doi:10.1016/S0003-9993(99)90174-0] [PMid:10378497]
70. Drygas, W., A. Jegler, and H. Kunski, Study on threshold dose of physical activity in coronary heart disease prevention. Part I. Relationship between leisure time physical activity and coronary risk factors. Int J Sports Med, 1988. 9(4): p. 275-8. [doi:10.1055/s-2007-1025021] [PMid:3263343]
71. Durstine, J.L. and W.L. Haskell, Effects of exercise training on plasma lipids and lipoproteins. Exerc Sport Sci Rev, 1994. 22: p. 477-521. [doi:10.1249/00003677-199401000-00017] [PMid:7925552]
72. Park, S.K., et al., The effect of combined aerobic and resistance exercise training on abdominal fat in obese middle-aged women. J Physiol Anthropol Appl Human Sci, 2003. 22(3): p. 129-35. [doi:10.2114/jpa.22.129] [PMid:12808225]
73. Ho, S.S., et al., The effect of 12 weeks of aerobic, resistance or combination exercise training on cardiovascular risk factors in the overweight and obese in a randomized trial. BMC Public Health, 2012. 12: p. 704. [doi:10.1186/1471-2458-12-704] [PMid:23006411]
74. Park, S.M., Y.S. Kwak, and J.G. Ji, The Effects of Combined Exercise on Health-Related Fitness, Endotoxin, and Immune Function of Postmenopausal Women with Abdominal Obesity. J Immunol Res, 2015. 2015: p. 830567. [doi:10.1155/2015/830567] [PMid:26075288]
75. Bruning, R.S. and M. Sturek, Benefits of exercise training on coronary blood flow in coronary artery disease patients. Prog Cardiovasc Dis, 2015. 57(5): p. 443-53. [doi:10.1016/j.pcad.2014.10.006] [PMid:25446554]
76. Halverstadt, A., et al., Endurance exercise training raises high-density lipoprotein cholesterol and lowers small low-density lipoprotein and very low-density lipoprotein independent of body fat phenotypes in older men and women. Metabolism, 2007. 56(4): p. 444-50. [doi:10.1016/j.metabol.2006.10.019] [PMid:17378998]
77. Nassef, Y., et al., The Impact of Aerobic Exercise and Badminton on HDL Cholesterol Levels in Adult Taiwanese. Nutrients, 2019. 11(3). [doi:10.3390/nu11030515] [PMid:30823398]
78. Gondim, O.S., et al., Benefits of Regular Exercise on Inflammatory and Cardiovascular Risk Markers in Normal Weight, Overweight and Obese Adults. PLoS One, 2015. 10(10): p. e0140596. [doi:10.1371/journal.pone.0140596] [PMid:26474157]
79. Aellen, R., W. Hollmann, and U. Boutellier, Effects of aerobic and anaerobic training on plasma lipoproteins. Int J Sports Med, 1993. 14(7): p. 396-400. [doi:10.1055/s-2007-1021198] [PMid:8244606]
80. Swain, D.P. and B.A. Franklin, Comparison of cardioprotective benefits of vigorous versus moderate intensity aerobic exercise. Am J Cardiol, 2006. 97(1): p. 141-7. [doi:10.1016/j.amjcard.2005.07.130] [PMid:16377300]
81. Mazurek, K., et al., Effects of aerobic interval training versus continuous moderate exercise programme on aerobic and anaerobic capacity, somatic features and blood lipid profile in collegate females. Ann Agric Environ Med, 2014. 21(4): p. 844-9. [doi:10.5604/12321966.1129949] [PMid:25528932]
82. Gordon, P.M., et al., Effects of acute exercise on high density lipoprotein cholesterol and high density lipoprotein subfractions in moderately trained females. Br J Sports Med, 1998. 32(1): p. 63-7. [doi:10.1136/bjsm.32.1.63] [PMid:9562167]
83. Visich, P.S., et al., Effects of exercise with varying energy expenditure on high-density lipoprotein-cholesterol. Eur J Appl Physiol Occup Physiol, 1996. 72(3): p. 242-48. [doi:10.1007/BF00838646] [PMid:8820893]
84. Farber, H.W., et al., The endurance triathlon: metabolic changes after each event and during recovery. Med Sci Sports Exerc, 1991. 23(8): p. 959-65. [doi:10.1249/00005768-199108000-00013] [PMid:1956272]
85. Lamon-Fava, S., et al., Acute changes in lipid, lipoprotein, apolipoprotein, and low-density lipoprotein particle size after an endurance triathlon. Metabolism, 1989. 38(9): p. 921-5. [doi:10.1016/0026-0495(89)90243-6] [PMid:2505019]
86. Lee, R., et al., The effects of acute moderate exercise on serum lipids and lipoproteins in mildly obese women. Int J Sports Med, 1991. 12(6): p. 537-42. [doi:10.1055/s-2007-1024730] [PMid:1797694]

87. Swank, A.M., et al., The effect of acute exercise on high density lipoprotein-cholesterol and the subfractions in females. Atherosclerosis, 1987. 63(2-3): p. 187-92. [doi:10.1016/0021-9150(87)90120-1] [PMid:3827980]
88. Leon, A.S., et al., Effects of a vigorous walking program on body composition, and carbohydrate and lipid metabolism of obese young men. Am J Clin Nutr, 1979. 32(9): p. 1776-87. [doi:10.1093/ajcn/32.9.1776] [PMid:474467]
89. Lewis, S., et al., Effects of physical activity on weight reduction in obese middle-aged women. Am J Clin Nutr, 1976. 29(2): p. 151-6. [doi:10.1093/ajcn/29.2.151] [PMid:1251807]
90. Petrenya, N., et al., Obesity and obesity-associated cardiometabolic risk factors in indigenous Nenets women from the rural Nenets Autonomous Area and Russian women from Arkhangelsk city. Int J Circumpolar Health, 2014. 73: p. 23859. [doi:10.3402/ijch.v73.23859] [PMid:25147770]
91. Bora, K., et al., Variation in Lipid Profile Across Different Patterns of Obesity - Observations from Guwahati, Assam. J Clin Diagn Res, 2015. 9(11): p. Oc17-21. [doi:10.7860/JCDR/2015/15334.6787] [PMid:26672627]
92. Bora, K., et al., Association of Decreased High-Density Lipoprotein Cholesterol (HDL-C) With Obesity and Risk Estimates for Decreased HDL-C Attributable to Obesity: Preliminary Findings From a Hospital-Based Study in a City From Northeast India. J Prim Care Community Health, 2017. 8(1): p. 26-30. [doi:10.1177/2150131916664706] [PMid:27531078]
93. van der Westhuyzen, D.R., F.C. de Beer, and N.R. Webb, HDL cholesterol transport during inflammation. Curr Opin Lipidol, 2007. 18(2): p. 147-51. [doi:10.1097/MOL.0b013e328051b4fe] [PMid:17353662]
94. Sorrentino, S.A., et al., Endothelial-vasoprotective effects of high-density lipoprotein are impaired in patients with type 2 diabetes mellitus but are improved after extended-release niacin therapy. Circulation, 2010. 121(1): p. 110-122. [doi:10.1161/CIRCULATIONAHA.108.836346] [PMid:20026785]
95. Rashid, S., K.D. Uffelman, and G.F. Lewis, The mechanism of HDL lowering in hypertriglyceridemic, insulin-resistant states. J Diabetes Complications, 2002. 16(1): p. 24-8. [doi:10.1016/S1056-8727(01)00191-X] [PMid:11872362]
96. Khera, A.V., et al., Cholesterol efflux capacity, high-density lipoprotein function, and atherosclerosis. N Engl J Med, 2011. 364(2): p. 127-35. [doi:10.1056/NEJMoa1001689] [PMid:21226578]
97. Bertière, M.C., et al., Low high density lipoprotein-2 concentrations in obese male subjects. Atherosclerosis, 1988. 73(1): p. 57-61. [doi:10.1016/0021-9150(88)90163-3] [PMid:3178932]
98. Després, J.P., et al., Adipose tissue distribution and plasma lipoprotein levels in obese women. Importance of intra-abdominal fat. Arteriosclerosis, 1989. 9(2): p. 203-10. [doi:10.1161/01.ATV.9.2.203] [PMid:2923576]
99. Ostlund, R.E., Jr., et al., The ratio of waist-to-hip circumference, plasma insulin level, and glucose intolerance as independent predictors of the HDL2 cholesterol level in older adults. N Engl J Med, 1990. 322(4): p. 229-34. [doi:10.1056/NEJM199001253220404] [PMid:2403660]
100. Coon, P.J., et al., Effects of body composition and exercise capacity on glucose tolerance, insulin, and lipoprotein lipids in healthy older men: a cross-sectional and longitudinal intervention study. 1989. 38(12): p. 1201-1209. [doi:10.1016/0026-0495(89)90160-1] [PMid:2687639]
101. Ratajczak, M., et al., Effects of Endurance and Endurance-Strength Training on Endothelial Function in Women with Obesity: A Randomized Trial. Int J Environ Res Public Health, 2019. 16(21). [doi:10.3390/ijerph16214291] [PMid:31694237]
102. Sgouraki, E., A. Tsopanakis, and C. Tsopanakis, Acute exercise: response of HDL-C, LDL-C lipoproteins and HDL-C subfractions levels in selected sport disciplines. J Sports Med Phys Fitness, 2001. 41(3): p. 386-91.
103. Stanton, K.M., et al., Moderate- and High-Intensity Exercise Improves Lipoprotein Profile and Cholesterol Efflux Capacity in Healthy Young Men. J Am Heart Assoc, 2022. 11(12): p. e023386. [doi:10.1161/JAHA.121.023386] [PMid:35699182]
104. Despres, J.P., et al., Loss of abdominal fat and metabolic response to exercise training in obese women. 1991. 261(2): p. E159-E167. [doi:10.1152/ajpendo.1991.261.2.E159] [PMid:1872379]

105.Lamarche, B., et al., Is body fat loss a determinant factor in the improvement of carbohydrate and lipid metabolism following aerobic exercise training in obese women? 1992. 41(11): p. 1249-1256. [doi:10.1016/0026-0495(92)90017-5] [PMid:1435299]
106.Manning, J.M., et al., Effects of a resistive training program on lipoprotein--lipid levels in obese women. 1991. 23(11): p. 1222-1226. [doi:10.1249/00005768-199111000-00003]
107.Nieman, D.C., et al., Reducing-diet and exercise-training effects on serum lipids and lipoproteins in mildly obese women. 1990. 52(4): p. 640-645. [doi:10.1093/ajcn/52.4.640] [PMid:2403058]
108.Szmedra, L., et al., Exercise tolerance, body composition and blood lipids in obese African-American women following short-term training. 1998. 38(1): p. 59-65.
109.Wesnigk, J., et al., Impact of lifestyle intervention on HDL-induced eNOS activation and cholesterol efflux capacity in obese adolescent. 2016. 2016. [doi:10.1155/2016/2820432] [PMid:27965912]
110.Roberts, C.K., et al., Effect of a short-term diet and exercise intervention on inflammatory/anti-inflammatory properties of HDL in overweight/obese men with cardiovascular risk factors. 2006. [doi:10.1152/japplphysiol.00345.2006] [PMid:16902063]
111.Park, S.-M., Y.-S. Kwak, and J.-G.J.J.o.i.r. Ji, The effects of combined exercise on health-related fitness, endotoxin, and immune function of postmenopausal women with abdominal obesity. 2015. 2015. [doi:10.1155/2015/830567] [PMid:26075288]
112.Nieman, D.C., et al., Reducing diet and/or exercise training decreases the lipid and lipoprotein risk factors of moderately obese women. 2002. 21(4): p. 344-350. [doi:10.1080/07315724.2002.10719233] [PMid:12166532]
113.Nicklas, B.J., et al., Increases in high-density lipoprotein cholesterol with endurance exercise training are blunted in obese compared with lean men. 1997. 46(5): p. 556-561. [doi:10.1016/S0026-0495(97)90194-3] [PMid:9160824]
114.Roh, H.-T., S.-Y. Cho, and W.-Y.J.J.o.c.m. So, A Cross-Sectional Study Evaluating the Effects of Resistance Exercise on Inflammation and Neurotrophic Factors in Elderly Women with Obesity. 2020. 9(3): p. 842. [doi:10.3390/jcm9030842] [PMid:32244926]
115.Puengsuwan, P., et al., Wand stretching exercise decreases abdominal obesity among adults with high body mass index without altering fat oxidation. 2020. 11. [doi:10.3389/fphys.2020.565573] [PMid:33192562]
116.Marcon, E.R., et al., What is the best treatment before bariatric surgery? Exercise, exercise and group therapy, or conventional waiting: a randomized controlled trial. 2017. 27(3): p. 763-773. [doi:10.1007/s11695-016-2365-z] [PMid:27620342]
117.Marques, L.R., et al., Reverse Cholesterol Transport: Molecular Mechanisms and the Non-medical Approach to Enhance HDL Cholesterol. Front Physiol, 2018. 9: p. 526. [doi:10.3389/fphys.2018.00526] [PMid:29867567]
118.Leclerc, S., et al., High density lipoprotein cholesterol, habitual physical activity and physical fitness. Atherosclerosis, 1985. 57(1): p. 43-51. [doi:10.1016/0021-9150(85)90136-4] [PMid:4074463]
119.Nieman, D.C., et al., Reducing diet and/or exercise training decreases the lipid and lipoprotein risk factors of moderately obese women. J Am Coll Nutr, 2002. 21(4): p. 344-50. [doi:10.1080/07315724.2002.10719233] [PMid:12166532]
120.Mogharnasi, M., R. Eslami, and B. Behnam, Effects of endurance and circuit resistance trainings on lipid profile, heart rate, and hematological parameters in obese male students. Annals of Applied Sport Science, 2014. 2(4): p. 11-22. [doi:10.18869/acadpub.aassjournal.2.4.11]
121.Sigal, R.J., et al., Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes: a randomized trial. Annals of internal medicine, 2007. 147(6): p. 357-369. [doi:10.7326/0003-4819-147-6-200709180-00005] [PMid:17876019]
122.Azarbayjani, M.A., et al., Effects of combined aerobic and resistant training on lipid profile and glycemic control in sedentary men. Int Med J, 2014. 21(2): p. 132-136.
123.Roh, H.T., S.Y. Cho, and W.Y. So, A Cross-Sectional Study Evaluating the Effects of Resistance Exercise on Inflammation and Neurotrophic Factors in Elderly Women with Obesity. J Clin Med, 2020. 9(3). [doi:10.3390/jcm9030842] [PMid:32244926]
124.Al-Mhanna, S.B., et al., Effects of combined aerobic exercise and diet on cardiometabolic health in patients with obesity and type 2 diabetes: a systematic review and meta-analysis. BMC Sports Science,

Medicine and Rehabilitation, 2023. 15(1): p. 165. [doi:10.1186/s13102-023-00766-5] [PMid:38049873]
125. Williams, P.T., et al., The effects of running mileage and duration on plasma lipoprotein levels. Jama, 1982. 247(19): p. 2674-9. [doi:10.1001/jama.1982.03320440022026] [PMid:6952022]
126. Stein, R.A., et al., Effects of different exercise training intensities on lipoprotein cholesterol fractions in healthy middle-aged men. Am Heart J, 1990. 119(2 Pt 1): p. 277-83. [doi:10.1016/S0002-8703(05)80017-1] [PMid:2301216]
127. Myhre, K., et al., Relationship of high density lipoprotein cholesterol concentration to the duration and intensity of endurance training. Scand J Clin Lab Invest, 1981. 41(3): p. 303-9. [doi:10.1080/00365518109092049] [PMid:7313515]
128. Williams, P.T., Relationships of heart disease risk factors to exercise quantity and intensity. Arch Intern Med, 1998. 158(3): p. 237-45. [doi:10.1001/archinte.158.3.237] [PMid:9472203]