QUALITY IN SPORT

eISSN 2450-3118 · Open Access · Peer-reviewed

apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: KOWALCZYK, Olga, KOZŁOWSKI, Sebastian, BULICZ, Anna, ZDUNEK, Marta, STODULSKI, Maciej, KASPRZYCKA, Izabela, FIKS, Justyna, KUKLA, Monika, DZIARNOWSKA, Joanna and KMIECIK, Izabela. The Impact of Physical Activity on Lipid Profile and Dyslipidemia: What Type of Exercise Is Most Effective in Preventing Cardiovascular Diseases? Quality in Sport. 2026;57:72387. https://doi.org/10.12775/QS.2026.57.72387

ARTICLE TIMELINE

Received: 22.05.2026. Revised: 25.05.2026. Accepted: 31.05.2026. Published: 10.06.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines: Medical Sciences; Health Sciences.

Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Nauki medyczne; Nauki o zdrowiu. © The Authors 2026.

OPEN ACCESS · CC BY-NC-SA 4.0 This article is published with open access under the License Open Journal Systems of Nicolaus Copernicus University in Toruń, Poland, and is distributed under the terms of the Creative Commons Attribution Non-commercial Share Alike License (http://creativecommons.org/licenses/by-nc-sa/4.0/), which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the work is properly cited. The authors declare no conflict of interest regarding the publication of this paper.

The Impact of Physical Activity on Lipid Profile and Dyslipidemia: What Type of Exercise Is Most Effective in Preventing Cardiovascular Diseases?

Olga Kowalczyk, https://orcid.org/0009-0003-6908-7618

olga.kowalczyk1@stud.umed.lodz.pl

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Sebastian Kozłowski, https://orcid.org/0009-0000-1892-1869

sebastian.jan.kozlowski@gmail.com

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Anna Bulicz, https://orcid.org/0009-0008-7336-7096

anna.bulicz@stud.umed.lodz.pl

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Marta Zdunek, https://orcid.org/0009-0006-4920-4460

marta.zdunek18@gmail.com

University Clinical Hospital No. 2, Medical University of Lodz,

Zeromskiego 113, 90-549 Lodz, Poland

Maciej Stodulski, https://orcid.org/0009-0001-1614-3511

maciej.stodulski1999@gmail.com

Municipal Hospital of St. John Paul II,

Jana Amosa Komenskiego 35, 82-300 Elblag, Poland

Izabela Kasprzycka, https://orcid.org/0009-0009-9416-4156

i.k.kasprzycka@gmail.com

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Justyna Fiks, https://orcid.org/0009-0003-3833-7194

justynafiks08@gmail.com

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Monika Kukla, https://orcid.org/0009-0006-1136-4194

monikakukla10@gmail.com

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Joanna Dziarnowska, https://orcid.org/0009-0001-7615-8408

joannadziarno@gmail.com

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Izabela Kmiecik, https://orcid.org/0009-0005-9905-3704

izabela.kmiecik@stud.umed.lodz.pl

Medical University of Lodz,

Kosciuszki 4, 90-419 Lodz, Poland

Corresponding Author

Monika Kukla, monikakukla10@gmail.com

Table of Contents

1. **Abstract**
2. **Introduction**
3. **Materials and Methods**
4. **Results**

    4.1. Pathophysiology of Dyslipidemia and Atherosclerosis

    4.2. Mechanisms of Physical Activity in Modulating Lipid Profiles and Atherogenesis

4.2.1. LDL-C

4.2.2. HDL-C

4.2.3. Triglycerides

4.3. Exercise Modalities and Specific Lipid Modulation

4.3.1. Aerobic Exercise

4.3.2. Resistance Training

4.3.3. High-Intensity Interval Training (HIIT)

4.3.4. Combined Training

5. **Discussion**
6. **Conclusions**

References

1. Abstract

Background. Cardiovascular diseases (CVDs) remain the leading cause of mortality worldwide. Dyslipidemia significantly contributes to atherosclerosis, endothelial dysfunction, and chronic vascular inflammation, increasing the risk of ischemic cerebral and cardiac events. Regular physical activity is a key component of cardiovascular prevention due to its beneficial effects on lipid metabolism and systemic inflammation.

Aim. The study aimed to evaluate the impact of physical activity on lipid profile parameters and cardiovascular risk, with particular emphasis on the effects of aerobic, resistance, high-intensity interval training (HIIT), and combined exercise models on LDL-C, HDL-C, and triglyceride levels.

Materials and Methods. A narrative literature review was performed using PubMed and Google Scholar databases. The search included keywords such as “dyslipidemia,” “lipid profile,” “physical activity,” and “cardiovascular risk.” Original studies, randomized controlled

trials, systematic reviews, meta-analyses, and current clinical guidelines on cardiovascular prevention were analyzed.

Results. Regular physical activity improves lipid profile by lowering LDL-C and triglyceride levels while increasing HDL-C concentration. Aerobic exercise is the most extensively studied modality in cardiovascular prevention and demonstrates significant cardioprotective effects. Resistance training improves insulin sensitivity and body composition, indirectly supporting lipid metabolism. HIIT induces rapid metabolic adaptations and may be an effective alternative for selected patients. Combined training integrates the benefits of aerobic and resistance exercise, providing comprehensive cardiovascular and metabolic effects.

Conclusions. Regular physical activity, regardless of exercise type, plays a fundamental role in improving lipid profile and reducing cardiovascular risk. The greatest benefits are associated with combined training programs and regular moderate-intensity aerobic exercise.

Keywords: dyslipidemia; physical activity; atherosclerosis; lipid profile; LDL-C; HDL-C; triglycerides; cardiovascular diseases; aerobic exercise; resistance training; HIIT; combined training; cardiovascular prevention; vascular inflammation.

2. Introduction

Cardiovascular events persistently represent the primary cause of mortality both in Poland and globally. More than four out of five deaths attributable to cardiovascular diseases (CVDs) result from myocardial infarctions and ischemic strokes (1), which most frequently arise from advanced atherogenesis. Advanced atherosclerosis ultimately leads to critical arterial stenosis, culminating in the ischemia of vital organs, predominantly the heart and the brain.

In many instances, an acute myocardial infarction serves as the initial manifestation of coronary artery disease, thereby limiting the opportunities for timely therapeutic intervention and underscoring the critical importance of primary prevention. Because dyslipidemia plays a foundational role in the initiation and progression of atherosclerosis, managing the lipid profile—specifically targeting the reduction of low-density lipoprotein cholesterol (LDL-C)—constitutes a primary therapeutic objective.

An elevated cardiovascular risk profile combined with lipid derangements invariably necessitates therapeutic lifestyle changes. The World Health Organization (WHO) provides specific guidelines regarding the duration and intensity of physical activity required to mitigate cardiovascular mortality. According to these recommendations, adults should engage in at least 150–300 minutes of moderate-intensity aerobic physical activity, or at least 75–150 minutes of vigorous-intensity aerobic physical activity per week (2). Regular exercise has been demonstrated to significantly improve lipid parameters, leading to reductions in LDL-C and circulating triglycerides, alongside concomitant increases in high-density lipoprotein cholesterol (HDL-C) (3).

Given the profound relationship between physical exertion and lipid metabolism, it is clinically essential to delineate which specific exercise modalities demonstrate the highest efficacy in optimizing the lipid profile, preventing dyslipidemia, and mitigating overall cardiovascular disease risk.

3. Materials and Methods

To evaluate the impact of various physical activity modalities on the lipid profile and global cardiovascular risk, a narrative review of the scientific literature indexed in the PubMed and Google Scholar biomedical databases was performed.

The search strategy relied on specific keyword sequences and their Boolean combinations. The following terms were utilized: "dyslipidemia", "lipid profile", "lipoproteins", "atherosclerosis", "physical activity", "exercise", "aerobic exercise", "resistance training", "high-intensity interval training" (HIIT), "combined training", "cardiovascular risk", and "vascular inflammation".

The analysis included original research articles (including randomized controlled trials [RCTs]), prospective cohort studies, meta-analyses, systematic reviews, and current clinical guidelines or position statements issued by international scientific societies, with particular emphasis on the European Society of Cardiology (ESC) and the American Heart Association (AHA). The review focused on studies evaluating the physiological and pathophysiological mechanisms linking physical exercise to lipid metabolism, modifications in specific lipoprotein fractions (LDL-C, HDL-C, TG), and hard clinical endpoints (e.g., myocardial infarction, stroke, or cardiovascular mortality).

Regarding population criteria, the review considered studies involving sedentary adults, individuals with overweight or obesity, metabolic syndrome, or diagnosed dyslipidemia. Editorial articles, conference abstracts, single case reports, and studies with imprecise or unquantifiable training protocols were excluded from the analysis. Data extracting the type, intensity, duration, and frequency of the exercise interventions underwent a qualitative synthesis, enabling a rigorous comparison of the clinical efficacy of aerobic, anaerobic, interval, and combined exercise models in modulating the lipid profile and inhibiting atherogenesis.

4. Results

4.1. Pathophysiology of Dyslipidemia and Atherosclerosis

Dyslipidemia represents a cornerstone in the pathogenesis of atherosclerosis and subsequent cardiovascular diseases. It is characterized by abnormal plasma concentrations of lipids and lipoproteins, typically manifesting as elevated total cholesterol (TC), high LDL-C, elevated triglycerides (TG), reduced HDL-C, and aberrant levels of lipoprotein(a). Low-density lipoproteins (LDL), whose primary structural protein is apolipoprotein B (apoB), play a mandatory role in the development of the atheromatous plaque (4).

The atherogenic process initiates with vascular endothelial dysfunction, which promotes increased endothelial permeability and the subsequent retention of apoB-containing lipoproteins within the subendothelial space. These trapped lipoproteins undergo subsequent chemical modifications, such as oxidation or glycation, which stimulate endothelial cell activation and initiate a localized inflammatory response (5). This is followed by the recruitment of immune cells, predominantly circulating monocytes, which differentiate into tissue macrophages. These macrophages internalize the modified lipoproteins via scavenger receptors, transforming into cholesterol-laden foam cells. Concurrently, pro-inflammatory signaling pathways are upregulated, amplifying the influx of additional immunocytes and further accelerating lipoprotein modification, thereby driving the progression of atherosclerotic lesions (6).

Triglycerides and their carrier lipoproteins—namely chylomicrons, very-low-density lipoproteins (VLDL), and their respective remnants, including intermediate-density lipoproteins (IDL)—also contribute significantly to atherogenesis. These remnant particles can be directly taken up by macrophages to participate in foam cell formation, actively accelerating plaque expansion (7).

Conversely, high-density lipoproteins (HDL) represent another critical lipoprotein fraction; diminished HDL-C concentrations are strongly correlated with an increased incidence of cardiovascular events and accelerated atherosclerosis (8). HDLs exert a potent atheroprotective effect primarily via their role in reverse cholesterol transport (RCT). This mechanism mediates the clearance of excess cholesterol from peripheral cells and its subsequent transport back to the liver for excretion, thereby restricting foam cell formation and decelerating plaque progression.

In summary, dyslipidemia promotes atherogenesis through complex, interconnected mechanisms encompassing lipid metabolic derangements and chronic vascular inflammation. Deciphering these pathways is fundamental to developing robust primary and secondary prevention strategies for cardiovascular disease.

4.2. Mechanisms of Physical Activity in Modulating Lipid Profiles and Atherogenesis

Physical activity directly mitigates vascular inflammation, the primary driving force behind atherogenesis. In physically active individuals, a threefold increase in the phosphorylation efficiency of endothelial nitric oxide synthase (eNOS) is observed. This upregulates the synthesis and bioavailability of nitric oxide (NO), inducing potent vasodilation. Additionally, regular exercise downregulates the systemic production and expression of pro-inflammatory cytokines.

4.2.1. LDL-C

Physically active cohorts exhibit reductions in circulating LDL-C, the primary substrate for atherosclerotic plaque architecture. One underlying molecular mechanism is the exercise-induced downregulation of proprotein convertase subtilisin/kexin type 9 (PCSK9), an enzyme responsible for the endosomal degradation of hepatic LDL receptors. Mitigating PCSK9 activity preserves a higher density of LDL receptors (LDL-R) on the surface of hepatocytes, enhancing the clearance of circulating LDL-C from the bloodstream and lowering its serum concentration (9). Furthermore, exercise-induced weight loss and reduction in adiposity synergistically optimize the lipid profile. It is estimated that a 10 kg reduction in body mass correlates with a total cholesterol reduction of approximately 0.23 mmol/L in certain populations (10).

4.2.2. HDL-C

Depressed HDL levels compromise systemic anti-inflammatory capacity and impair reverse cholesterol transport (11, 12). Conversely, an elevation in HDL-C is one of the most consistent lipid alterations induced by chronic physical exercise. This mechanism is primarily driven by an upregulation of lipoprotein lipase (LPL) activity within skeletal muscle and adipose tissues. LPL catalyzes the hydrolysis of triglycerides within chylomicrons and VLDLs, prompting the net transfer of surface lipids and apolipoproteins onto HDL particles, thereby increasing their maturation, size, and circulating concentration (13).

Additionally, physical activity modulates the activity of cholesteryl ester transfer protein (CETP). Regular exercise training has been shown to downregulate CETP activity, which inhibits the transfer of cholesteryl esters from HDL to VLDL and LDL, thus preserving higher plasma concentrations of cardioprotective HDL. Crucially, exercise augments reverse cholesterol transport (RCT) by upregulating the expression of ATP-binding cassette transporter A1 (ABCA1) in macrophages, facilitating the initial efflux of cholesterol from peripheral tissues to nascent HDL particles prior to hepatic clearance (14).

4.2.3. Triglycerides

The reduction of serum triglycerides (TG) via physical activity occurs through both acute post-exercise responses—often observable after a single exercise bout—and chronic long-term adaptations. The primary mechanism is the aforementioned upregulation of LPL activity, which accelerates the catabolism of triglyceride-rich lipoproteins (VLDLs and chylomicrons) (15).

Physical exertion markedly increases the metabolic demands of skeletal muscle, stimulating fatty acid beta-oxidation. Long-term training enhances systemic insulin sensitivity, which effectively suppresses lipolysis within adipose tissue and diminishes the flux of free fatty acids (FFAs) to the liver. Consequently, hepatic synthesis and secretion of VLDL particles are curtailed, directly reducing serum TG levels (16). Notably, the magnitude of triglyceride reduction correlates closely with total energy expenditure; a greater movement-induced caloric deficit yields a more pronounced decline in circulating TG.

4.3. Exercise Modalities and Specific Lipid Modulation

Selecting an appropriate exercise model is essential for individualizing non-pharmacological cardiovascular therapy. Distinct exercise modalities utilize different predominant metabolic pathways, directly influencing their efficacy in modifying specific lipoprotein fractions.

4.3.1. Aerobic Exercise

Aerobic exercise, characterized by sustained physical effort involving large muscle groups at a constant intensity (e.g., brisk walking, running, swimming, cycling), represents the most comprehensively validated modality in cardiovascular prevention. Meta-analyses of clinical trials demonstrate that total training volume (total energy expenditure) is the primary parameter governing lipid profile improvements. Regular aerobic training typically yields a significant increase in HDL-C concentrations (averaging 3–10%) and a concurrent reduction in TG levels (ranging from 5–20%) (17). Regarding LDL-C, aerobic exercise shifts the particle distribution toward larger, less atherogenic, buoyant LDL molecules (*large buoyant LDL*). This significantly diminishes their propensity to accumulate in the subendothelial space, even when absolute quantitative changes in total LDL-C concentrations are modest (18).

4.3.2. Resistance Training

Historically, resistance (strength) training was less frequently prescribed for dyslipidemia management; however, contemporary evidence underscores its distinct metabolic advantages. This modality enhances vascular compliance and myocardial performance through structural muscular and hemodynamic adaptations (19). The primary driving mechanism is the accretion of lean body mass (LBM), which elevates the basal metabolic rate (BMR) and augments resting fatty acid oxidation. Furthermore, resistance training stimulates the translocation of glucose transporter type 4 (GLUT-4) receptors within skeletal muscle, enhancing insulin sensitivity and reducing the substrates required for hepatic VLDL assembly (20).

4.3.3. High-Intensity Interval Training (HIIT)

High-Intensity Interval Training (HIIT) has emerged as an efficient therapeutic alternative, particularly for time-constrained populations. It involves brief bursts of near-maximal exertion (85–95% of maximal heart rate, HRmax) interspersed with periods of active recovery.

Comparative trials suggest that HIIT may induce more rapid and pronounced reductions in triglyceride concentrations than traditional moderate-intensity continuous training (MICT), a phenomenon attributed to the robust acute surge of lipolytic hormones, specifically catecholamines and growth hormone (21). HIIT also exerts a powerful beneficial effect on arterial stiffness and cardiorespiratory fitness, rendering it a highly effective tool for lowering global cardiovascular risk in patients free of cardiac contraindications.

4.3.4. Combined Training

The latest clinical guidelines from the European Society of Cardiology (ESC) and the American Heart Association (AHA) recommend combined training as the optimal therapeutic model. The synergy between aerobic and resistance exercise allows patients to simultaneously maximize cardiorespiratory fitness and optimize body composition. This dual approach demonstrates the highest efficacy in reducing visceral adipose tissue, which is metabolically active and directly implicated in the pathogenesis of dyslipidemia and vascular inflammation (22).

5. Discussion

Physical activity represents a foundational recommendation for patients at elevated cardiovascular risk. Exercise-induced reductions in body weight and adiposity consistently optimize lipid metabolism, ultimately preventing or retarding atherogenesis (22, 23). Among the analyzed exercise modalities, aerobic training appears to be the most preferred for primary and secondary CVD prevention. Its superiority in modulating the lipid profile stems primarily from its reliance on oxidative metabolism and fatty acid oxidation. It effectively improves plasma lipid concentrations and favorably modifies LDL particle size in overweight and obese cohorts.

Conversely, resistance training exerts distinct metabolic actions by enhancing insulin sensitivity, expanding lean body mass, and escalating resting energy expenditure (24).

High-Intensity Interval Training (HIIT) has also been scrutinized for its lipid-lowering potential. Notably, one meta-analysis reported that HIIT outperformed MICT in lowering LDL-C and total cholesterol (TC) within a cohort of young overweight or obese adults, though it demonstrated no significant impact on HDL-C or TG levels (25). However, the demanding nature of HIIT may limit its clinical utility and adherence in patients with extensive comorbidities or low exercise tolerance.

An expanding body of evidence highlights the clinical utility of combined training, which merges aerobic and resistance exercise. Consequently, the highest efficacy in optimizing the global cardiovascular risk profile is currently attributed to either isolated aerobic training or a combined aerobic-resistance framework (26, 27).

When interpreting these data, several study limitations must be considered. Many clinical interventions differed substantially regarding duration, intensity, and overall training volume, and several trials relied on small sample sizes. Furthermore, the explicit effects of physical activity are frequently confounded by concurrent dietary habits, pharmacological therapies, age, sex, and baseline fitness levels. These confounding variables preclude the formulation of a single, universal exercise prescription applicable to all clinical demographics.

Table 1: Comparative Analysis of Exercise Modalities on the Lipid Profile and Cardiovascular Risk

Exercise Modality	Primary Mechanism	Impact on Lipid Profile	Clinical Advantages
Aerobic Exercise	Up-regulated fatty acid oxidation and favorable modification of LDL particle buoyancy.	Reduction in small dense LDL; lower TG; elevation of HDL-C.	Most thoroughly documented and validated modality.
Resistance Training	Accretion of lean body mass; enhancement of peripheral insulin sensitivity.	Enhanced resting lipid oxidation; reduction of substrate availability for hepatic VLDL synthesis.	Direct metabolic benefits; favorable restructuring of body composition.

High Intensity Interval Training (HIIT)	Acute, robust surge of lipolytic hormones (catecholamines, growth hormone).	Reductions in LDL-C and TC; no significant impact on HDL-C and TG levels.	Rapid metabolic adaptations; highly time-efficient.
Combined Training	Synergistic integration of metabolic, muscular, and hemodynamic adaptations.	Most profound impact on the global lipid profile and visceral fat reduction.	Optimal model per ESC/AHA guidelines; comprehensive CV risk reduction.

6. Conclusions

Based on this comprehensive literature review, the mandatory role of physical activity in the prevention and management of dyslipidemia is confirmed. It serves as the primary non-pharmacological intervention capable of optimizing the lipid profile, inducing weight loss, and fostering favorable cardiovascular adaptations, which collectively translate into reduced cardiovascular risk. Data examining the incidence of atherosclerotic complications in physically active versus sedentary cohorts unequivocally support an active lifestyle. Specifically, regular physical activity correlates with an approximate 27% reduction in stroke risk (28) and a 10–20% reduction in coronary artery disease incidence compared to sedentary individuals (28). Ultimately, a combined exercise model integrating aerobic and resistance training, or sustained, moderate-intensity continuous aerobic exercise, represents the most effective lifestyle strategy for the prevention of cardiovascular diseases.

References

1. World Health Organization, *Cardiovascular diseases*, (2023), https://www.who.int/health-topics/cardiovascular-diseases#tab=tab_1

2. World Health Organization, HEARTS: Technical package for cardiovascular disease management in primary health care: risk-based CVD management, Geneva, World Health Organization, (2020), https://iris.who.int/handle/10665/333221

3. Neil A Smart , David Downes, Tom van der Touw, Swastika Hada, Gudrun Dieberg, Melissa J Pearson, Mitchell Wolden, Nicola King, Stephen P J Goodman, The Effect of Exercise Training on Blood Lipids: A Systematic Review and Meta-analysis, (2025), https://doi.org/10.1007/s40279-024-02115-z

4. Ference BA, Ginsberg HN, Graham I, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease. A consensus statement from the European Atherosclerosis Society Consensus Panel. Eur Heart J. 38(32):2459–2472, (2017), https://doi.org/10.1093/eurheartj/ehx144

5. Borén, J., Packard, C.J., Binder, C.J., *Apolipoprotein B-containing lipoproteins in atherogenesis*, Nature Reviews Cardiology, (2025), https://www.nature.com/articles/s41569-024-01111-0

6. Hansson GK, Hermansson A. The immune system in atherosclerosis. Nat Immunol. 12(3):204–212, (2011), https://doi.org/10.1038/ni.2001

7. Reyes-Soffer, Gissette, Triglyceride-rich lipoproteins and atherosclerotic cardiovascular disease risk: current status and treatments, (2021), https://doi.org/10.1097/med.0000000000000619

8. Rohatgi A. High-density lipoprotein function measurement in human studies. *Circulation.* 130(25):2402–2416, (2015), doi.org/10.1016/j.pcad.2015.05.004

9. Tirandi A, Montecucco F, Liberale L. Physical activity to reduce PCSK9 levels. Front Cardiovasc Med. (2022); https://doi.org/10.3389/fcvm.2022.988698

10. Poobalan A, Aucott L, Smith W, et al. Effects of weight loss in overweight/obese individuals and long-term lipid outcomes – a systematic review. Obesity Reviews. (2004); 5(1): 43–50, https://doi.org/10.1111/j.1467-789x.2004.00127.x

11. S. Nazir, V. Jankowski, G. Bender, S. Zewinger, K.A. Rye, E.P.C.van der Vorst Interaction between high-density lipoproteins and inflammation: Function matters more than concentration! (2020) https://doi.org/10.1016/j.addr.2020.10.006

12. A. Rohatgi, M. Westerterp, A. von Eckardstein, A. Remaley, K.-A. Rye HDL in the 21st century: a multifunctional roadmap for future HDL research, (2021), https://doi.org/10.1161/CIRCULATIONAHA.120.044221

13. Wang, Y., & Xu, D. "Effects of aerobic exercise on lipids and lipoproteins." Lipids in Health and Disease. (2017), https://doi.org/10.1186/s12944-017-0515-5

14. Jonathan J Ruiz-Ramie, Jacob L Barber, Mark A Sarzynski Effects of exercise on HDL functionality, (2019), https://doi.org/10.1097/MOL.0000000000000568

15. Steven Mann, Christopher Beedie, Alfonso Jimenez, Differential Effects of Aerobic Exercise, Resistance Training and Combined Exercise Modalities on Cholesterol and the Lipid Profile: Review, Synthesis and Recommendations, (2013), https://doi.org/10.1007/s40279-013-0110-5

16. Michael Harrison, Niall M Moyna, Theodore W Zderic, Donal J O'Gorman, Noel McCaffrey, Brian P Carson, Marc T Hamilton Lipoprotein particle distribution and skeletal muscle lipoprotein lipase activity after acute exercise, (2012), doi.org/10.1186/1476-511x-11-64

17. Satoru Kodama Shiro Tanaka, Kazumi Saito, Miao Shu, Yasuko Sone, Fumiko Onitake, Emiko Suzuki, Hitoshi Shimano, Shigeru Yamamoto, Kazuo Kondo, Yasuo Ohashi, Nobuhiro Yamada, Hirohito Sone, Effect of aerobic exercise training on serum levels of high-density lipoprotein cholesterol: a meta-analysis, (2007), doi.org/10.1001/archinte.167.10.999

18. Yating Wang, Danyan Xu *Effects of aerobic exercise on lipids and lipoproteins*. Lipids Health Dis.;16(1):132., (2017), https://doi.org/10.1186/s12944-017-0515-5

19. Brown, T.M., Pack, Q.R., Aberegg, E., et al., Core Components of Cardiac Rehabilitation Programs: 2024 Update: A Scientific Statement From the American Heart Association and the American Association of Cardiovascular and Pulmonary Rehabilitation, American Heart Association, (2024)

20. George A. Kelley ⁎, Kristi S. Kelley Impact of progressive resistance training on lipids and lipoproteins in adults: A meta-analysis of randomized controlled trials; J Appl Physiol.;106(4):1164-1170, (2009), https://doi.org/10.1016/j.ypmed.2008.10.010

21. Romeo B Batacan Jr, Mitch J Duncan, Vincent J Dalbo, Patrick S Tucker, Andrew S Fenning Effects of high-intensity interval training on cardiometabolic health: a systematic review and meta-analysis of intervention studies, (2017), https://doi.org/10.1136/bjsports-2015-095841

22. Visseren FLJ, Mach F, Smulders YM, Carballo D, Koskinas KC, Bäck M, et al. 2021 ESC Guidelines on cardiovascular disease prevention in clinical practice: Developed by the Task Force for cardiovascular disease prevention in clinical practice with representatives of the European Society of Cardiology and 12 medical societies with the special contribution of the European Association of Preventive Cardiology (EAPC). Eur Heart J. 2021;42(34):3227–3337. https://doi.org/10.1093/eurheartj/ehab484

23. B K Pedersen, B Saltin Exercise as medicine - evidence for prescribing exercise as therapy in 26 different chronic diseases, (2015), https://doi.org/10.1111/sms.12581Digital Object Identifier (DOI)

24. Stuart M Phillips, Richard A Winett Uncomplicated Resistance Training and Health-Related Outcomes: Evidence for a Public Health Mandate, (2010), https://doi.org/10.1249/jsr.0b013e3181e7da73

25. Christian P McCormick, Garrett Mamikunian, David B Thorp The Effects of HIIT vs. MICT and Sedentary Controls on Blood Lipid Concentrations in Nondiabetic Overweight and Obese Young Adults: A Meta-analysis https://doi.org/10.70252/wgfu4000

26. US Department of Health and Human Services. *Physical Activity Guidelines for Americans*, 2nd Edition, (2018), https://health.gov/our-work/nutrition-physical-activity/physical-activity-guidelines

27. World Health Organization. WHO Guidelines on Physical Activity and Sedentary Behaviour. Geneva: World Health Organization, (2020), https://www.who.int/publications/i/item/9789240015128

28. Leon Smółka, Bartosz Pomianowski, Miłosz Strugała, Krzysztof Osuch, Julia Pakulska, Karolina Blady, Physical activity in the prevention and treatment of atherosclerotic cardiovascular diseases, (2025), https://doi.org/10.5603/aa.104293