ZARZYCKI, Adam, NOWAK, Dominika, PRÓCHNICKA, Magdalena, SIWEK, Michał, HAMOUTA, Jakub, DŁUGOSZ, Patrycja, KONAT, Julia, DOROSZUK, Wiktor, NOSKOWICZ, Jan and URBAN, Jan. The Impact of Endurance Running Training on the Lipid Profile in Professional and Amateur Athletes: A Comprehensive Review. Quality in Sport. 2025;42:60824. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.42.60824
https://apcz.umk.pl/QS/article/view/60824

The journal has been awarded 20 points in the parametric evaluation by the Ministry of Higher Education and Science of Poland. This is according to the Annex to the announcement of the Minister of Higher Education and Science dated 05.01.2024, No. 32553. The journal has a Unique Identifier: 201398. Scientific disciplines assigned: Economics and Finance (Field of Social Sciences); Management and Quality Sciences (Field of Social 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: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).
The authors declare that there is no conflict of interest regarding the publication of this paper.
Received: 08.05.2025. Revised: 17.05.2025. Accepted: 14.06.2025. Published: 16.06.2025.

The Impact of Endurance Running Training on the Lipid Profile in Professional and Amateur Athletes: A Comprehensive Review

Adam Zarzycki
adam.zarzycki0709@gmail.com
ORCID:0009-0004-9589-1842
University Clinical Hospital No. 4 in Lublin
ul. Doktora Kazimierza Jaczewskiego 8, 20-090 Lublin

Dominika Nowak
mika1200@onet.pl
ORCID:0000-0002-0195-1102
University Clinical Hospital No. 4 in Lublin
ul. Doktora Kazimierza Jaczewskiego 8, 20-090 Lublin

Magdalena Próchnicka
maadiii1210@gmail.com
ORCID:0000-0002-0641-2079
University Clinical Hospital No. 4 in Lublin
ul. Doktora Kazimierza Jaczewskiego 8, 20-090 Lublin

Michał Siwek
michal.siwek21371488@gmail.com
ORCID:0000-0002-0930-9333
University Clinical Hospital No. 4 in Lublin
ul. Doktora Kazimierza Jaczewskiego 8, 20-090 Lublin

Jakub Hamouta
kubahamouta@gmail.com
ORCID:0009-0003-6368-223X
1st Military Clinical Hospital with Outpatient Clinic SPZOZ in Lublin
Raclawickie Avenue 23, 20-049 Lublin

Patrycja Długosz
patrycjadlugosz1999@gmail.com
ORCID:0000-0003-4517-4832
University Clinical Hospital No. 4 in Lublin
ul. Doktora Kazimierza Jaczewskiego 8, 20-090 Lublin

Julia Konat
juliakonat11@gmail.com
ORCID:0009-0008-6607-6725
Provincial Specialist Hospital in Lublin
Krasnickie Avenue 100, 20-718 Lublin

Wiktor Doroszuk
wiktor.doroszuk@gmail.com
ORCID:0009-0004-2446-726X
1st Military Clinical Hospital with Outpatient Clinic SPZOZ in Lublin
Raclawickie Avenue 23, 20-049 Lublin

Jan Noskowicz
jasieknoskowicz@gmail.com
ORCID:0009-0009-6578-0398
Independent Public Healthcare Institution of the Ministry of Internal Affairs and Administration named after Sergeant Grzegorz Załoga in Katowice
ul. Wita stwosza 39/41, 40-042 katowice

Jan Urban
jan.urban019@gmail.com
ORCID:0009-0004-8583-6970
Independent Public Healthcare Institution of the Ministry of Internal Affairs and Administration named after Sergeant Grzegorz Załoga in Katowice
ul. Wita stwosza 39/41, 40-042 katowice

Abstract

Endurance running is one of the most popular forms of physical activity, influencing many aspects of health, including lipid metabolism. Regular endurance exercise positively affects the lipid profile, which plays a key role in preventing cardiovascular diseases (CVD). Dyslipidemia, characterized by abnormal levels of cholesterol and triglycerides, is a significant risk factor for the development of these diseases.

The purpose of this study is to analyze the impact of endurance running on the lipid profile of amateur and professional athletes, considering the differences in metabolic adaptations resulting from training intensity and lifestyle.

A review of the available literature was conducted by searching official databases such as Pubmed and Google Scholar using the following keywords: dyslipidemia, endurance running, cardiovascular, lipoprotein lipase.

The results indicate that regular endurance running enhances LPL activity, fatty acid oxidation, and cholesterol transport. Both moderate and high-intensity training lower LDL-C and triglycerides, while raising HDL-C. Lipid responses vary between amateur and professional athletes due to differences in training and lifestyle. Overall, endurance running with a healthy lifestyle effectively improves lipid profiles and reduces cardiovascular risk.

Keywords: dyslipidemia; endurance running; cardiovascular; lipoprotein lipase

1. Introduction

Cardiovascular diseases (CVD) remain the foremost cause of death globally, accounting for nearly 18 million deaths annually [1]. Among the many risk factors contributing to CVD, dyslipidemia holds a pivotal role in the initiation and progression of atherosclerosis [2]. Dyslipidemia is characterized by abnormal concentrations of plasma lipids, including elevated total cholesterol (TC), LDL-C, and triglycerides (TG), along with decreased HDL-C. This lipid imbalance promotes endothelial dysfunction, plaque formation, and ultimately leads to ischemic events such as myocardial infarction and stroke [3].

Pharmacological therapies targeting lipid abnormalities, such as statins, have proven effective; however, lifestyle modifications including diet and physical activity remain foundational in prevention and management strategies [4]. Endurance running is a form of aerobic exercise involving sustained, rhythmic physical activity that enhances cardiovascular fitness and metabolic health. It has been extensively studied for its favorable impact on lipid metabolism and overall cardiovascular risk reduction [5].

Despite widespread acknowledgment of the benefits of endurance running, variability exists in individual lipid responses, influenced by training volume, intensity, genetic predisposition, and baseline health status. Additionally, differences in adaptations between amateur and professional runners may further complicate generalizations. This review synthesizes existing research, emphasizing mechanisms of lipid modulation via endurance running, and contrasts effects observed in different athlete populations [6].

2. Epidemiology of Dyslipidemia and Cardiovascular Risk

Dyslipidemia is a global health problem with prevalence estimates exceeding 40% in adult populations of developed countries. Elevated LDL-C and TG levels are strongly correlated with increased cardiovascular risk, while HDL-C is inversely related to disease incidence. According to the American College of Cardiology/American Heart Association (ACC/AHA) guidelines, optimal lipid targets include LDL-C below 100 mg/dL and HDL-C above 40 mg/dL for men and 50 mg/dL for women to minimize atherosclerotic risk [2,3,7].

Sedentary lifestyle significantly contributes to lipid abnormalities. Data indicate that physical inactivity is responsible for an estimated 10% of coronary heart disease cases worldwide. Conversely, regular physical activity improves lipid metabolism and reduces cardiovascular morbidity and mortality [4].

3. Physiological Adaptations to Endurance Running

Endurance running induces a multitude of physiological adaptations that collectively contribute to improved lipid profiles and cardiovascular health. Cardiovascular adaptations include increased maximal oxygen uptake (VO2max), stroke volume, and capillary density in skeletal muscle, enhancing oxygen delivery and utilization. These changes optimize substrate metabolism, favoring increased fatty acid oxidation during prolonged exercise [8,9]. On a molecular level, endurance training upregulates enzymes involved in lipid metabolism. Lipoprotein lipase (LPL), critical for hydrolyzing circulating TG into free fatty acids (FFA), is increased in skeletal muscles of trained individuals, facilitating enhanced TG clearance [10].

The activity of hepatic LDL receptors is also upregulated, promoting clearance of LDL-C from circulation [11]. Moreover, endurance running positively affects HDL-C by enhancing production of apolipoprotein A-I, a major structural protein of HDL particles, thereby promoting reverse cholesterol transport (RCT) — the process by which cholesterol is removed from peripheral tissues to the liver for excretion [12].

4. Mechanisms of Lipid Profile Modulation through Endurance Running

4.1 Lipoprotein Lipase (LPL) Activation

Lipoprotein Lipase hydrolyzes TG-rich lipoproteins such as very low-density lipoproteins (VLDL) and chylomicrons, releasing FFA for muscle uptake and oxidation. Endurance training increases LPL gene expression and enzymatic activity, primarily in skeletal muscles, which accelerates TG clearance and reduces plasma TG concentrations. Exercise intensity and duration modulate the extent of LPL activation, with moderate to high intensity exercise producing greater effects [10,13].

4.2 Elevation of High-Density Lipoprotein Cholesterol (HDL-C)

Exercise-induced increases in HDL-C are well documented. Enhanced HDL production results from increased apolipoprotein A-I synthesis and improved efficiency of RCT. HDL particles also possess antioxidant and anti-inflammatory properties, contributing to atheroprotection. Regular endurance running can increase HDL-C by 5-10%, with effects being dose-dependent [5,12,14].

4.3 Reduction of Low-Density Lipoprotein Cholesterol (LDL-C)

LDL-C reduction following endurance exercise is mediated by increased LDL receptor expression in the liver, facilitating enhanced clearance from plasma. Exercise also reduces hepatic synthesis of VLDL, precursor to LDL, thereby lowering LDL particle concentrations.

However, the magnitude of LDL-C reduction varies and is generally more modest compared to HDL-C increases [11,13].

4.4 Triglyceride (TG) Reduction

Endurance exercise promotes utilization of TG as an energy substrate during prolonged activity, decreasing circulating TG levels. Increased LPL activity and improved mitochondrial function enhance TG clearance. Studies have demonstrated reductions in fasting TG levels by 15-20% following structured aerobic training programs [13,14].

4.5 Anti-inflammatory and Antioxidant Effects

Chronic inflammation contributes to dyslipidemia and atherogenesis. Endurance running decreases pro-inflammatory cytokines (e.g., TNF-α, IL-6) and increases anti-inflammatory markers such as adiponectin. Reduced systemic inflammation improves endothelial function and lipid metabolism. Additionally, regular training enhances antioxidant defenses, mitigating oxidative modification of lipoproteins [15].

5. Comparative Effects in Amateur vs. Professional Runners

The lipid profile response to endurance running differs significantly between amateur and professional athletes, primarily due to variations in training volume, intensity, duration, and physiological adaptations. Understanding these differences is essential to tailor training recommendations and optimize cardiovascular health outcomes in different populations.

5.1 Training Volume and Intensity

Professional runners typically engage in high-volume training programs, often exceeding 100 kilometers per week, incorporating a mix of long runs, tempo workouts, interval training, and recovery runs. This substantial training load induces pronounced cardiovascular and metabolic adaptations, including enhanced lipid metabolism. In contrast, amateur runners generally train between 20 to 50 kilometers per week, with less structured intensity and variability [6,16]. Studies indicate that increased training volume correlates positively with higher HDL-C levels and lower LDL-C and triglyceride concentrations [17]. It has been demonstrated that elite runners had HDL-C levels approximately 15% higher than sedentary controls, while recreational runners demonstrated intermediate increases. This gradient suggests a dose-response relationship between training load and lipid improvements [18].

5.2 Physiological Differences

The enhanced lipid profile observed in professional athletes is partly due to superior mitochondrial density, greater oxidative enzyme activity, and increased capillarization within skeletal muscle, all of which improve fatty acid oxidation. Furthermore, professional runners exhibit elevated lipoprotein lipase (LPL) activity, facilitating more efficient triglyceride clearance from circulation [9,13]. However, excessive training intensity and volume without adequate recovery in professionals can lead to transient elevations in oxidative stress and inflammatory markers, which may temporarily blunt beneficial lipid effects. This highlights the importance of periodization and recovery strategies even among elite athletes [19].

5.3 Impact of Age and Gender

Age and gender modulate lipid responses to endurance running. Women typically have higher baseline HDL-C levels and may experience smaller relative increases with training compared to men [12]. Aging is associated with reduced maximal oxygen uptake (VO2max) and altered lipid metabolism, potentially attenuating training-induced lipid improvements Nevertheless, sustained endurance training mitigates age-related declines and preserves favorable lipid profiles in master athletes [17].

5.4 Psychological and Lifestyle Factors

Professional athletes often maintain strict dietary regimens, optimized sleep patterns, and comprehensive recovery protocols that synergistically enhance lipid metabolism. Conversely, amateurs may have less disciplined lifestyles, with dietary indiscretions and inconsistent sleep potentially limiting training benefits on lipid profiles [6,20]. These lifestyle factors must be considered when interpreting lipid data across different runner populations.

6. Training Protocols and Their Impact on Lipid Profiles

Exercise prescriptions designed to optimize lipid profile improvements through endurance running encompass a range of training modalities, intensities, and durations. The selection and structuring of these training protocols are critical to maximize cardiovascular benefits while minimizing risks of overtraining and injury.

6.1 Aerobic Continuous Training

Continuous aerobic training involves sustained running at a moderate intensity, typically between 60-75% of maximal heart rate (HRmax), for durations ranging from 30 to 60 minutes per session. This modality promotes improvements in VO2max, mitochondrial density, and enzymatic activity associated with lipid metabolism. Several studies have demonstrated that moderate-intensity continuous training effectively increases HDL-C by approximately 5-10% and reduces triglycerides by 10-20%. These changes are dose-dependent; training volumes exceeding 150 minutes per week elicit more pronounced effects [4,9,12,14].

6.2 High-Intensity Interval Training (HIIT)

HIIT alternates brief bouts of high-intensity running (85-95% HRmax) with recovery periods. HIIT has gained popularity due to its time efficiency and potent cardiovascular adaptations [21]. Recent meta-analyses report that HIIT can produce equal or greater improvements in lipid profiles compared to continuous training [22]. Mechanistically, HIIT enhances lipoprotein lipase activity and mitochondrial biogenesis more robustly than moderate continuous exercise. Studies show HDL-C increases of 7-12% and reductions in LDL-C and triglycerides by up to 15% following HIIT programs lasting 8-12 weeks [23,24].

6.3 Combined Training Protocols

Programs combining continuous moderate-intensity runs with HIIT sessions offer synergistic benefits. For example, a weekly regimen including three continuous runs and two HIIT workouts improves lipid metabolism more than either modality alone. This approach balances cardiovascular endurance development with metabolic stress, optimizing enzyme activation related to lipid clearance [25].

6.4 Training Volume and Frequency

The American Heart Association and American College of Sports Medicine recommend 150-300 minutes per week of moderate-intensity aerobic activity or 75-150 minutes of vigorous activity to reduce cardiovascular risk. For lipid improvements, training at the higher end of this range yields superior results. A minimum frequency of 3-5 sessions per week is optimal to sustain enzymatic adaptations such as increased lipoprotein lipase activity and improved HDL synthesis. Periodization involving incremental increases in weekly mileage with rest days to prevent overtraining is essential [4,13].

6.5 Nutritional Considerations

Training effects on lipid profiles are modulated by dietary intake. Adequate carbohydrate availability supports high-quality training sessions, while dietary fats influence circulating lipid concentrations. Incorporation of omega-3 fatty acids, fiber-rich foods, and reduced saturated fat intake synergize with training to improve lipid profiles [20,26].

7. Clinical Implications and Recommendations for Endurance Runners

The lipid profile improvements induced by endurance running carry significant clinical relevance for cardiovascular disease (CVD) prevention and management. This section outlines practical recommendations and considerations for athletes and clinicians.

7.1 Cardiovascular Risk Reduction

Elevated HDL-C and reduced LDL-C and triglycerides are well-established protective factors against atherosclerosis and CVD [27]. Endurance running's capacity to favorably modify these lipid parameters translates into reduced incidence of coronary artery disease, stroke, and related morbidity [14]. Meta-analyses confirm that regular moderate to vigorous endurance exercise reduces all-cause mortality by approximately 30% and CVD mortality by 35% [28]. For runners, this underscores the dual role of exercise for performance enhancement and long-term health.

7.2 Personalized Training Guidelines

Given individual variability in lipid responses, training programs should be personalized. Factors such as baseline fitness, age, gender, and genetic predispositions influence lipid metabolism adaptations [29]. Clinicians should encourage incremental increases in running volume and intensity while monitoring lipid profiles periodically to assess efficacy. Integration of nutrition counseling and behavioral support optimizes outcomes [4].

7.3 Potential Risks and Overtraining

While endurance running is largely beneficial, excessive training without adequate recovery can induce transient dyslipidemia, increased oxidative stress, and immune suppression. Monitoring for symptoms of overtraining syndrome and adjusting load accordingly is essential [19].

7.4 Special Populations

Certain populations, including older adults, individuals with metabolic syndrome, and those with familial hypercholesterolemia, require tailored exercise prescriptions. Combining endurance running with dietary and pharmacologic interventions enhances lipid management in these groups [30].

8. Future Directions and Research Gaps

Despite extensive research on endurance running and lipid metabolism, several key gaps remain that warrant further investigation to optimize training protocols and clinical applications. Emerging molecular techniques, such as transcriptomics and metabolomics, offer promising avenues to uncover novel pathways through which endurance running modulates lipid metabolism, while insights into genetic variability could help tailor personalized exercise prescriptions. However, most existing studies are limited by their cross-sectional or short-term design; longitudinal research tracking lipid profiles over years of training is needed to clarify the sustainability of benefits and long-term health outcomes [17,21]. Additionally, current evidence lacks diversity, with underrepresented populations—including ethnic minorities, women, older adults, and individuals with comorbidities—highlighting the need for more inclusive studies to improve the generalizability of exercise recommendations. Future research should also explore the interplay between endurance running, dietary interventions, and lipid-lowering medications to identify synergistic effects and refine treatment paradigms. Finally, technological advancements, such as wearable devices and digital health platforms, could revolutionize real-time monitoring of training load, metabolic markers, and recovery, though rigorous validation is required to integrate these tools into individualized programming. Addressing these gaps will advance both scientific understanding and practical applications in the field [20,26,29].

Conclusions

Endurance running has a beneficial effect on the lipid profile in both amateur and professional athletes. Typical changes include an increase in HDL cholesterol and a decrease in LDL cholesterol and triglycerides. These effects result from increased lipoprotein lipase activity, improved fatty acid oxidation, and enhanced reverse cholesterol transport. The effectiveness of these mechanisms depends on training volume, intensity, and duration. Due to higher training loads, professional athletes achieve more pronounced metabolic adaptations; however, excessive training without adequate recovery may temporarily reduce these benefits. Both moderate-intensity continuous training and high-intensity interval training (HIIT) improve lipid parameters, and combining them may produce synergistic effects. Training programs should be tailored to individual characteristics such as age, sex, fitness level, and genetic predisposition.

Clinical implications of this review suggest that endurance running should be an integral component of dyslipidemia prevention and cardiovascular disease management. Given the considerable interindividual variability in training responses, a personalized approach considering risk factors and lifestyle is essential. Further research is needed, particularly long-term cohort studies and investigations in underrepresented populations such as older adults, women, and individuals with metabolic syndrome or chronic diseases. Future efforts should also focus on integrating training, nutritional, and pharmacological data to develop comprehensive strategies for optimizing lipid profiles and preventing cardiovascular diseases.

Disclosures

Authors do not report any disclosures

Author's contribution

Conceptualization: Adam Zarzycki,Dominika Nowak, Magdalena Próchnicka, Michał Siwek,

Jan Noskowicz, Jan Urban, Jakub Hamouta, Julia Konat, Wiktor Doroszuk, Patrycja Długosz
Formal analysis: Adam Zarzycki, Dominika Nowak, Michał Siwek, Jan Noskowicz, Jan Urban, Jakub Hamouta, Julia Konat, Wiktor Doroszuk, Patrycja Długosz
Methodology: Adam Zarzycki, Dominika Nowak, Magdalena Próchnicka, Jan Noskowicz, Jan Urban, Wiktor Doroszuk, Michał Siwek, Jakub Hamouta, Patrycja Długosz, Julia Konat
Investigation: Adam Zarzycki, Dominika Nowak, Magdalena Próchnicka, Michał Siwek, Jan Noskowicz, Resources: Dominika Nowak, Michał Siwek, Wiktor Doroszuk, Patrycja Długosz, Julia Konat
Writing- rough preparation: Adam Zarzycki Dominika Nowak, Jakub Hamouta, Patrycja Długosz, Julia Konat, Wiktor Doroszuk, Jan Urban, Jan Noskowicz, Magdalena Próchnicka,
Writing- review and editing: Adam Zarzycki Dominika Nowak, Magdalena Próchnicka, Michał Siwek, Jan Noskowicz, Jakub Hamouta, Julia Konat
Supervision: Adam Zarzycki
All authors have read and agreed with the published version of the manuscript

Funding statement:
Not applicable.
Institutional Review Board Statement:
Not applicable.
Informed Consent Statement:
Not applicable.
Data Availability:
Not applicable.
Acknowledgments:
Not applicable.
Conflict or interest statement:
Authors declare no conflict of interest.

Declaration of generative AI and AI-assisted technologies in the writing process
During preparing this work the authors have used ChatGPT for the purpose of improving language and readability. After using this tool, the authors have reviewed and edited the

content as needed and accept full responsibility for the substantive content of the publication.

References

[1] WHO. Global Health Estimates 2020: Deaths by Cause, Age, Sex, by Country and Region, 2000-2019. Geneva: World Health Organization; 2021. English.

[2] Grundy SM, Stone NJ, Bailey AL, Beam C, et al. Guideline on the Management of Blood Cholesterol. Journal of the American College of Cardiology. 2018;73(24):285-350. https://doi.org/10.1016/j.jacc.2018.11.003. English.

[3] Stone NJ, Robinson JG, Lichtenstein AH, et al. Guideline on the Treatment of Blood Cholesterol to Reduce Atherosclerotic Cardiovascular Risk in Adults. Circulation. 2013;129:1-45. https://doi.org/10.1161/01.cir.0000437738.63853.7a. English.

[4] Thompson PD, Buchner D, Piña IL, et al. Exercise and Physical Activity in the Prevention and Treatment of Atherosclerotic Cardiovascular Disease. Circulation. 2003;107(24):3109-3116. https://doi.org/10.1161/01.CIR.0000075572.40158.77. English.

[5] Kraus WE, Houmard JA, Duscha BD, Knetzger KJ, Wharton MB, McCartney JS, Bales CW, Henes S, Samsa GP, Otvos JD, Kulkarni KR, Slentz CA. Effects of the Amount and Intensity of Exercise on Plasma Lipoproteins. New England Journal of Medicine. 2002;347(19):1483-1492. https://doi.org/10.1056/NEJMoa020194. English.

[6] Mann S, Beedie C, Jimenez A. Differential Effects of Aerobic Exercise, Resistance Training and Combined Exercise Modalities on Cholesterol and the Lipid Profile. Sports Medicine. 2014;44(2):211-221. https://doi.org/10.1007/s40279-013-0110-5. English.

[7] Mora S, Cook N, Buring JE, Ridker PM, Lee I. Physical Activity and Reduced Risk of Cardiovascular Events: Potential Mediating Mechanisms. Circulation. 2007;116(19):2110-2118. https://doi.org/10.1161/CIRCULATIONAHA.107.729939. English.

[8] Leon AS, Sanchez OA. Response of Blood Lipids to Exercise Training Alone or Combined with Dietary Intervention. Medicine & Science in Sports & Exercise. 2001;33(6):502-515. https://doi.org/10.1097/00005768-200106001-00022. English.

[9] Holloszy JO, Coyle EF. Adaptations of Skeletal Muscle to Endurance Exercise and Their Metabolic Consequences. Journal of Applied Physiology. 1984;56(4):831-838. https://doi.org/10.1152/jappl.1984.56.4.831. English.

[10] Seip RL, Mair K, Cole TG, Semenkovich CF. Induction of Human Skeletal Muscle

Lipoprotein Lipase Gene Expression by Short-Term Exercise. Circulation Research. 1993;72(6):1225-1230. English.

[11] Halverstadt A, Phares DA, Wilund KR, Goldberg AP, Hagberg JM. Endurance Exercise Training Raises High-Density Lipoprotein Cholesterol and Lowers Small Low-Density Lipoprotein and Very Low-Density Lipoprotein Independent of Body Fat Phenotype. Metabolism. 2007;56(4):444-450. https://doi.org/10.1016/j.metabol.2006.10.019. English.

[12] Kokkinos PF, Fernhall B. Physical Activity and High Density Lipoprotein Cholesterol Levels: What is the Relationship? Sports Medicine. 1999;28(5):307-314. https://doi.org/10.2165/00007256-199928050-00002. English.

[13] Durstine JL, Grandjean PW, Cox CA, Thompson PD. Lipid and Lipoprotein Adaptations to Exercise: A Quantitative Analysis. Sports Medicine. 2001;31(15):1033-1062. https://doi.org/10.2165/00007256-200131150-00002. English.

[14] Kodama S, Tanaka S, Saito K, Shu M, Sone Y, Onitake F, Suzuki E, Shimano H, Yamamoto S, Kondo K, Ohashi Y, Yamada N, Sone H. Effect of Aerobic Exercise Training on Serum Levels of High-Density Lipoprotein Cholesterol: A Meta-Analysis. Archives of Internal Medicine. 2007;167(10):999-1008. https://doi.org/10.1001/archinte.167.10.999. English.

[15] Tambalis K, Panagiotakos DB, Kavouras SA, Sidossis LS. Responses of Blood Lipids to Aerobic, Resistance, and Combined Exercise Training: A Systematic Review of Current Evidence. Angiology. 2009;60(5):614-632. https://doi.org/10.1177/0003319708324927. English.

[16] Basset FA, Howley ET. Limiting Factors for Maximum Oxygen Uptake and Determinants of Endurance Performance. Medicine & Science in Sports & Exercise. 2000;32(1):70-84. https://doi.org/10.1097/00005768-200001000-00012. English.

[17] Laukkanen JA, Lakka TA, Rauramaa R, Kuhanen R. Cardiorespiratory Fitness and Physical Activity as Predictors of Cardiovascular Disease Mortality in Men. JAMA Internal Medicine. 2014;174(8):1335-1340. English.

[18] Lee DC, Xuemei S, Church TS, Lavie CJ, Jackson AS, Blair SN. Changes in Fitness and Fatness on the Development of Cardiovascular Disease Risk Factors. Journal of the American College of Cardiology. 2012;59(7):665-672. https://doi.org/10.1016/j.jacc.2011.11.013. English.

[19] Fisher-Wellman K, Bloomer RJ. Oxidative Stress and Antioxidant Defense Mechanisms

Linked to Exercise. Journal of Cardiopulmonary Rehabilitation and Prevention. 2009;29(4):178-185. English.
[20] Jeukendrup A, Gleeson M. Sport Nutrition: An Introduction to Energy Production and Performance. 3rd ed. Champaign: Human Kinetics; 2018. English.
[21] Gibala MJ, Little JP, MacDonald MJ, Hawley JA. Physiological Adaptations to Low-Volume, High-Intensity Interval Training in Health and Disease. The Journal of Physiology. 2012;590(5):1077-1084. https://doi.org/10.1113/jphysiol.2011.224725. English.
[22] Wewege M, van den Berg R, Ward RE, Keech A. The Effects of High-Intensity Interval Training vs Moderate-Intensity Continuous Training on Body Composition in Overweight and Obese Adults: A Systematic Review and Meta-Analysis. Obesity Reviews. 2017;18(6):635-646. https://doi.org/10.1111/obr.12532. English.
[23] Little JP, Safdar A, Wilkin GP, Tarnopolsky MA, Gibala MJ.A Practical Model of Low-Volume High-Intensity Interval Training Induces Mitochondrial Biogenesis in Human Skeletal Muscle: Potential Mechanisms. The Journal of Physiology. 2011;588(6):1011-1022. English.
[24] Maillard F, Rousset S, Pereira B, Traore A, de Pradel Del Amaze P, Boirie Y, Duclos M, Boisseau N. High-intensity interval training reduces abdominal fat mass in postmenopausal women with type 2 diabetes. Diabetes Metab. 2016 Dec;42(6):433-441. English.
[25] Wilmore JH, Costill DL, Kenney WL. Physiology of Sport and Exercise. 4th ed. Champaign: Human Kinetics; 2001. English.
[26] Mozaffarian D, Micha R, Wallace S. Effects on Coronary Heart Disease of Increasing Polyunsaturated Fat in Place of Saturated Fat: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. PLoS Medicine. 2005;2(6):e123. https://doi.org/10.1371/journal.pmed.0020123. English.
[27] Laufs U, Wassmann S, Czech T, Münzel T, Eisenhauer M, Böhm M, Nickenig G. Physical Inactivity Increases Oxidative Stress, Endothelial Dysfunction, and Atherosclerosis. Arteriosclerosis, Thrombosis, and Vascular Biology. 2005;25(4):809-814. https://doi.org/10.1161/01.ATV.0000158311.24443.af. English.
[28] Nocon M, Hiemann T, Müller-Riemenschneider F, Thalau F, Roll S, Willich SN. Association of Physical Activity with All-Cause and Cardiovascular Mortality: A Systematic Review and Meta-Analysis. European Journal of Cardiovascular Prevention & Rehabilitation. 2008;15(3):239-246. https://doi.org/10.1097/HJR.0b013e3282f55e09. English.

[29] Simoneau JA, Bouchard C. Genetic Determinants of Nonresponse to Training. Canadian Journal of Applied Physiology. 1995;20(3):312-319. https://doi.org/10.1139/h95-024. English.
[30] Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, Macera CA, Heath GW, Thompson PD, Bauman A. Physical Activity and Public Health: Updated Recommendation for Adults. Medicine & Science in Sports & Exercise. 2007;39(8):1423-1434. https://doi.org/10.1249/mss.0b013e3180616b27. English.