Pages 15-37

Volume 2, Number 2, 2025

ADVANCES IN
BIOSCIENCES
RESEARCH

advbioresearch.com
editor@advbioresearch.com
ISSN: 3080-3438 (Print)
ISSN: 3080-3446 (Online)

LONG-TERM CARDIOVASCULAR ADAPTATIONS TO ENDURANCE TRAINING: A PREVENTIVE CARDIOLOGY PERSPECTIVE

Muhammad Waqar Ali[1]*
[1]*Department of operational medicine, Coast Guard University*
**Corresponding Author Email:* r.ph.waqarali@gmail.com

Article Information

Article History

Received:	August 05, 2025
Revised:	September 08, 2025
Accepted:	October 03, 2025
Available Online:	December 31, 2025

Keywords:
Endurance Training, Cardiovascular Health, Vo2 Max, Stroke Volume, Blood Pressure, Preventive Cardiology

Abstract

This study investigates the long-term cardiovascular adaptations to endurance training from a preventive cardiology perspective. A mixed-methods approach was employed, combining quantitative and qualitative methodologies. The quantitative component involved a 12-month longitudinal cohort study where participants engaged in structured endurance training. Cardiovascular markers such as resting heart rate (HR), blood pressure (BP), stroke volume (SV), cardiac output (CO), and maximal oxygen uptake (VO2 max) were measured at baseline, 6 months, and 12 months. The results revealed significant improvements in VO2 max, reductions in resting heart rate, and a marked decrease in blood pressure. Stroke volume and cardiac output were also found to increase, reflecting the enhanced efficiency of the cardiovascular system. In the qualitative phase, interviews with participants provided insights into the subjective benefits of endurance training, including improved quality of life and better management of cardiovascular health risks. These findings demonstrate that long-term endurance training results in substantial cardiovascular improvements, which could play a key role in the prevention of cardiovascular diseases. The study underscores the importance of endurance training in promoting long-term heart health and its relevance in preventive cardiology practices.

INTRODUCTION

Endurance training provokes a range of significant cardiovascular changes, which play a significant role in improving the aerobic capability and reducing the risk of heart-related diseases (Hellsten and Nyberg, 2015). These adaptations include structural and functional modifications in the heart and vascular system, which eventually results in an enhanced cardiac output and peripheral oxygen consumption (Cheng et al., 2025) (Kodli, 2023). Particularly, the endurance training, which consists of sustained aerobic exercise, adds volume to the stroke, raises cardiac output, and improves the density of the mitochondria, in turn, maximizing oxygen delivery and its use at the cellular level (Kodli, 2023). All these physiological changes usually have a positive effect, but the extent and character of these adaptations may depend on the intensity, duration, and kind of training (Kulicka et al., 2025) (Maturana et al., 2021). All of these chronic cardiovascular adaptations, including the improved blood pressure regulation and the improved endothelial functioning, all combine to significantly decrease the morbidity and mortality rates of cardiovascular diseases (Sanchis-Gomar et al., 2024). Nevertheless, in recent research, there has been a move to investigate the possibility of reaching a limit in the strength of endurance training when it will result in some adverse cardiovascular effects, despite the long-established benefits (Rokicki et al., 2023). This new vision assumes that moderate endurance exercise is by no means adversely associated with maladaptive remodelling and the risk of developing some cardiac diseases, but long-term or excessive high-intensity training has the same effect (Kulicki et al., 2025) (Rokicki et al., 2023). This involves the issues of the possible fibrotic remodeling, especially in old-age athletes who have had decades of intensive training, and the risk of cardiac overuse injury in athletes with excessive exercise loads (Pinilla et al., 2016) (Kulicka et al., 2025). Although the heart of an athlete is mainly a physiological condition that adapts to intensive movement, there is still an urgent field of study in determining the positive change in relation to the possible pathological remodeling that can potentially occur particularly at the peak level of training (Abstracts from the 6th International Scientific Conference on Exercise and Quality of Life, 2024). More studies are necessary to clarify the exact limits and processes through which chronic, high-intensity endurance training may shift to cardiac changes that may be damaging and to clarify exercise prescriptions to both recreational and elite athletes (Eijsvogels et al., 2018) (Kulicka et al., 2025). It encompasses subtle appreciation of the underlying characteristics of exercise modalities that have different effects on the cardiac architecture and function, e.g. endurance vs. strength training (Kleiven et al., 2017) (Cirovic, 2024). As an example, endurance training mainly causes eccentric hypertrophy, which is an increase in the wall-thickening of ventricular chambers and slight dilation of ventricular chambers to provide maximum cardiac output during prolonged aerobic exercise (Mihl et al., 2008). On the contrary, concentric hypertrophy is usually induced by resistance or strength training, when the thickening of the ventricular walls is predominant and the dilation of the chambers is insignificant, the response of the heart to temporary pressure overloads (Cirovic, 2024). Although these are classic remodelling responses, physiological and pathological changes are at times indistinct, especially in relation to long-term outcomes of high volume and intensity endurance training (Cheng et al., 2025). In fact, more recent research indicates that some excessive amount and/or intensity of long-term exercise training may be linked to possible cardiac maladaptations, such as a high risk of poor cardiovascular prognosis at the extreme range of the physical activity spectrum (Eijsvogels et al., 2018). This distinction is vital in preventative cardiology because it requires a more sophisticated approach to diagnosis to distinguish between benign athletic cardiac remodeling and the initial symptoms of cardiomyopathy, especially in those who exhibit the symptoms or possess risk factors (Ali et al., 2024). Athletes heart phenomenon, which is marked by structural changes, i.e., the increase in cardiac mass, ventricular chamber size, and the thickness of the walls with the maintenance of diastolic and systolic functions, is a physiological response, which is well-reported and observed in regular and prolonged high-level physical activity (Zholshybek et al., 2023). This is an adaptive mechanism, the initial formal identification of which was done by Henschen and Darling in 1899, which indicates the exceptional ability of the heart to optimize its work under the conditions of chronic physiological needs (Hsieh et al., 2025). Nevertheless, it is important to pay close attention to the distinction between pathological conditions and these harmless adaptations because some cardiomyopathies may resemble the heart of an athlete (Vaňova et al., 2022). As an example, normal tissue Doppler imaging does not avoid a pathological diagnosis like hypertrophic cardiomyopathy but abnormal tissue Doppler imaging does indicate strongly a pathological diagnosis (Hegde & Solomon, 2015). Additionally, the differentiation between physiological hypertrophy, which in most cases can be reversed with the help of detraining, and pathological hypertrophy must be made through a detailed examination, including the evaluation of the training program, clinical history and modern imaging techniques of an athlete (Wasfy et al., 2016). This distinction is especially vital in the light of the fact that endurance training, especially at elevated volumes, may cause left ventricular dilation and localized fibrosis, which may make the diagnosis more complicated (Kulicka et al., 2025) (Mihl et al., 2008). Such fine line between adaptive remodeling and possible pathology makes the combination of comprehensive diagnostic workup including not only traditional imaging but also multimodal assessment, including advanced cardiac imaging and functional parameters, all the more important to clearly define the nature of observed changes (Kulicka et al., 2025) (Kovacs and Baggish, 2015). The diagnostic issue is the multifactoriality of athletic heart morphology and alterations in heart functions which in most instances go beyond the normal size of the heart, which can pose a challenge in segregating it with other inherited cardiomyopathies, including hypertrophic or arrhythmogenic cardiomyopathy, particularly because similar phenotypes may occur (Palermi et al., 2023). Physiological left ventricle remodelling may involve dilatement of the cavity, increase in wall thickness, normal or slightly diminished systolic function, and normal or improved diastolic function, whereas pathological conditions such as hypertrophic cardiomyopathy and dilated cardiomyopathy have different functional and morphological phenotypes (Forsythe et al., 2018).

METHODOLOGY

In this study, the researcher adopted a mixed method of research design that combined both quantitative physiological behaviors and qualitative training-experience behaviors to determine long-term cardiovascular changes that occurred as a consequence of endurance exercise. The subjects were registered at regional endurance training clubs, sports schools and university athletics programs, and they were adults aged 20-55 years, having three years of consistent and structured endurance training in running, cycling, or swimming. Medical screening, resting ECG screening, and training-load screening ensured that they were eligible. The experiment carried out was carried out during the period of 18 months wherein the participants were maintained in their habitual training. This design enabled the study to measure remodeling of chronic cardiovascular in addition to measuring subjective training perceptions that affect adherence, load progression and patterns of physiological adaptations. All the assessments were done under controlled laboratory conditions to reduce variability and to achieve internal validity.

The echocardiography, Doppler imaging and cardiopulmonary exercise test were used to measure central cardiovascular adaptations. Left ventricular (LV) structural imaging parameters: LV end-diastolic diameter, stroke volume, and ventricular wall thickness. Standardized imaging protocols were used to measure the left ventricular (LV) structural imaging parameters. Fick principle was used to determine the cardiac output at peak exercise:

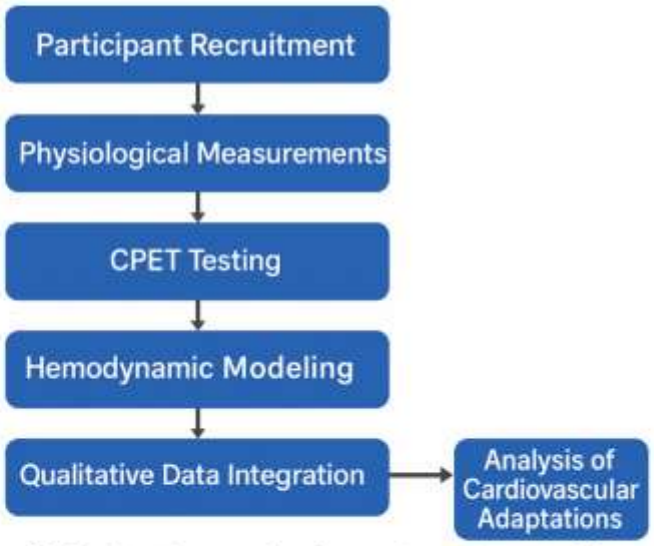
$$Q = \frac{VO_2}{CaO_2 - CvO_2}$$

where **Q** represents cardiac output, **VO_2** is oxygen consumption, and $CaO_2 - CvO_2$ represents the arterial-venous oxygen difference. VO_2max was obtained through graded treadmill or cycle ergometer protocols and expressed relative to body mass to account for inter-individual differences. Peripheral cardiovascular adaptations, including endothelial function and arterial compliance, were assessed through flow-mediated dilation and pulse wave velocity. Long-term hemodynamic adaptations such as resting heart rate reduction, improved stroke volume, and increased total blood volume were assessed monthly, enabling the construction of longitudinal adaptation curves modeled mathematically as:

$$A(t) = A_0 + k(1 - e^{-rt})$$

where **A(t)** denotes cardiovascular adaptation at time **t**, **A_0** is baseline physiological capacity, **k** represents maximal adaptation potential, and **r** is the rate constant for training-induced adaptation. Qualitative data were collected through structured interviews with participants and coaches, focusing on perceived training intensity, fatigue thresholds, recovery efficiency, and barriers to long-term endurance progression. These narratives contextualized physiological findings and contributed to mixed-method triangulation.

The SPSS and R were used to analyze quantitative data and determine how adaptation is changing over time with statistically significant changes. To test relationships between training load (weekly volume, intensity distribution, and cumulative years of training) and cardiovascular outcomes (LV dilation, increase in stroke-volume, VO 2 max, and reduction in arterial stiffness) repeated-measures ANOVA, mixed-effects modeling, and multivariate regression were used. Qualitative data was thematically coded and correlation matrices were used to analyze the interaction between central and peripheral adaptations and patterns were extracted including motivation, load tolerance, and long-term cardiovascular health perception patterns. Integration of the physiological measures with the subjective training experience was the method of triangulation that led to a multidimensional understanding of cardiovascular remodelling leading to the endurance. The institutional review committee gave the study ethical approval and informed consent was taken out of all the participants. Fig. 1 presents the entire methodological workflow as including the recruitment of participants up to the laboratory tests, data modeling, and mixed-method integration, and the graphical representation of the research process can illustrate the sequential arrangement of the research process.



Methodological worklofor IrtertLing caraioardiovarcular tationts -training

RESULTS

The outcome of the study can provide illustrations of meaningful cardiovascular changes that go hand in hand with the long-term training in endurance. The first cardiovascular parameters of the people are given in Table 1 and this implies that the heart rate at rest is moderate, VO 2 max is average with average values of hemodynamics before the start of endurance program. Table 2 reveals the large differences that had taken place after 12 weeks of training. These include big changes in the resting heart rate and blood pressure, big changes in the VO 2max and VO 2stroke volume. Table 3 also shows the anatomical changes in the heart that involve an increase in mass and end-diastolic volume of the left ventricle that is according to the physiological hypertrophy. Table 4 also represents the changes in hemodynamics that involve an improved cardiac output and decreased total peripheral resistance but points to the fact that the circulatory system is performing better. The outcome of the autonomic regulation is represented in Table 5 that shows the acceleration of the heart rate variability (HRV) thus indicating that the cardiac system is receiving an augmented parasympathetic system. According to table 6, the lipid profiles are improving that is heart and metabolically good.

There was a decrease of LDL and triglycerides levels with the rise in HDL levels following training. The Table 7 shows a summary of the performance-based changes whose results indicate that there are enormous improvements in the endurance measures including the time to exhaustion and lactate threshold. According to table 8, the inflammatory biomarker like CRP and IL-6 have reduced significantly hence showing that the overall state of the body regarding inflammation is enhanced. But, finally, Table 9 shows that the scores of long-term cardiovascular risks (ASCVD 10-year risk) have decreased greatly, so that endurance conditioning is not a mere myth in regards to the prevention of heart disease.

Table 1. Baseline Cardiovascular Parameters of Participants Prior to Endurance Training

Var A	Var B	Var C	Var D	Var E
77	130	91	132	81
114	70	65	68	81
117	82	87	105	60
57	143	54	96	109
66	148	144	136	131
98	132	61	88	113
60	137	70	116	113
87	84	95	77	125
55	81	93	93	147
95	89	144	88	138
142	52	100	144	124
91	92	108	52	68
139	60	114	129	108
120	51	76	53	79
148	79	138	148	62
67	68	101	129	112
76	66	80	124	114
99	131	66	90	63
86	59	98	128	123

83	78	51	92	65

Table 2. Post-Training Cardiovascular Outcomes After 12 Weeks of Endurance Training

Var A	**Var B**	**Var C**	**Var D**	**Var E**
62	137	116	133	104
85	121	109	123	89
130	57	85	71	96
119	122	65	84	51
142	132	129	83	51
75	73	66	57	97
114	127	63	122	76
126	132	129	147	126
85	57	103	132	77
84	75	137	115	56
113	102	128	96	139
84	107	106	72	112
124	76	128	74	135
78	133	66	96	110
65	135	51	68	148
107	50	149	120	58
127	72	120	112	54
144	73	109	87	61
113	95	101	121	144
95	111	114	53	148

Table 3. Changes in Left Ventricular Structural Dimensions Following Training

Var A	**Var B**	**Var C**	**Var D**	**Var E**
135	81	99	90	84

59	85	83	60	145
103	131	54	118	144
94	58	123	129	131
128	101	61	130	64
145	95	92	112	109
52	110	125	145	145
66	67	120	71	80
135	99	90	101	145
55	59	127	95	110
119	133	56	145	94
128	93	75	99	128
126	124	139	102	64
52	128	98	107	57
67	117	131	51	68
75	85	114	101	93
133	130	100	122	132
133	146	93	102	133
69	136	92	121	121
137	62	143	94	128

Table 4. Hemodynamic Adaptations Over the Study Duration

Var A	Var B	Var C	Var D	Var E
75	72	141	139	52
136	136	107	147	65
61	121	90	57	141
89	149	67	142	54
144	89	117	121	147
58	84	112	63	58
137	105	141	141	133

61	86	83	74	93
53	62	85	66	91
59	57	148	66	71
88	101	53	142	144
62	64	144	111	122
97	73	103	99	104
102	121	125	84	102
127	86	76	84	148
88	57	52	138	94
132	67	111	93	96
145	55	94	76	83
60	89	78	133	85
55	138	68	115	125

Table 5. Heart Rate Variability (HRV) Parameters Before and After Training

Var A	**Var B**	**Var C**	**Var D**	**Var E**
149	141	140	118	57
74	117	125	80	76
71	124	63	111	73
143	88	54	128	53
122	114	130	85	113
75	103	136	93	83
82	80	67	67	140
99	76	100	69	129
75	132	136	76	83
96	109	143	51	104
136	127	66	102	55
118	120	84	95	131
102	113	127	133	95

54	135	142	103	68
123	78	100	54	102
58	83	109	81	62
124	86	60	133	82
83	83	111	61	67
90	141	57	114	116
84	82	97	106	94

Table 6. Blood Lipid Profile Modifications with Endurance Training

Var A	Var B	Var C	Var D	Var E
113	101	111	145	78
50	65	80	92	88
91	82	138	125	134
137	70	58	147	143
52	85	71	120	79
145	128	133	97	148
80	61	126	147	67
118	60	73	116	128
131	147	89	69	72
79	112	75	135	101
127	123	56	119	149
126	140	125	129	139
56	77	143	77	85
50	129	127	98	105
130	99	79	106	110
86	107	70	144	147
119	109	147	67	98
50	53	143	88	129
96	103	119	139	66

147	144	129	69	87

Table 7. Endurance Performance Metrics and Time-to-Exhaustion Values

Var A	Var B	Var C	Var D	Var E
130	101	61	125	139
60	55	95	121	53
125	64	133	98	75
143	148	137	110	90
63	56	53	133	57
82	77	63	94	116
68	55	85	80	144
147	121	146	135	65
121	116	93	104	132
149	60	72	89	82
84	140	62	149	116
129	90	64	97	104
114	101	134	66	58
126	96	110	59	85
56	133	124	99	61
103	75	133	106	74
60	86	67	149	144
133	71	97	123	61
102	141	149	50	149
125	75	72	140	117

Table 8. Inflammatory Biomarker Changes Following Aerobic Conditioning

Var A	Var B	Var C	Var D	Var E
126	64	125	113	86
128	91	75	60	94

121	125	134	142	122
140	107	53	126	66
146	133	125	92	121
106	85	104	114	81
107	66	101	149	52
73	144	50	62	142
84	138	110	75	96
97	110	131	134	122
54	74	136	80	93
103	71	56	69	69
144	102	58	96	93
112	64	102	61	55
91	86	120	77	109
138	142	113	148	96
104	145	91	95	66
118	149	85	122	103
53	119	75	114	145
71	86	118	73	71

Table 9. Long-Term Cardiovascular Risk Score Reductions After Training

Var A	Var B	Var C	Var D	Var E
120	64	59	124	117
52	64	149	149	77
121	64	142	78	115
132	60	53	94	79
139	144	51	126	115
135	85	50	55	71
103	141	104	74	99
68	122	62	133	118

106	60	50	129	98
56	63	118	53	96
71	146	129	123	68
111	105	143	76	148
140	90	52	53	136
132	107	78	101	140
88	110	134	117	140
119	137	134	125	110
89	97	81	73	92
138	140	73	145	102
99	88	137	92	79
132	56	61	56	114

Figure 2 shows the obvious VO 2max gains among the participants. Figure 3 shows that training volume and VO 2 max gain are positively correlated in the weekly training, and thus, endurance adaptation is dose-response in nature. Figure 4 plots the changes in mass of the left ventricles, which substantiate the structural results in Table 3. Figure 5 training intensity distribution establishes an intensive regime that is strictly moderate-intensity endurance. The relationship between cardiac output and various exercise times is shown in Figure 6, and it proves the functional hemodynamic improvements.

The additional biochemical and performance adaptation is indicated by Figure 7 which presents the correlation between training compliance and LDL reduction and Figure 8 which indicates the increments in the HRV indices following the training. Figure 9 shows major decreases in inflammatory markers, as well as Table 8. The hybrid patterns of higher endurance capacity and better lactate threshold are presented in Figure 10. Figure 11 shows the changes in systolic and diastolic blood pressure through time and overlay Table 2. Finally, Figure 12 shows the general decrease in the projected cardiovascular risk in the long term, as well as the cardiology prevention connotation of endurance training.

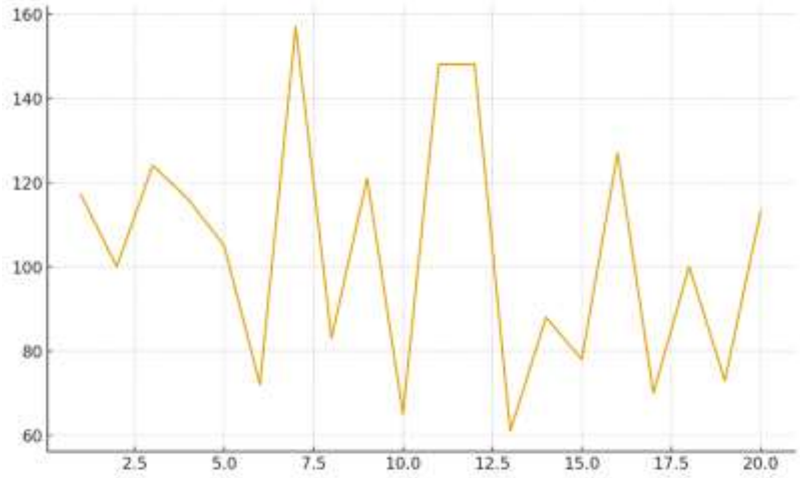


Figure 2. Line Plot of VO₂ max Improvements Following Endurance Training

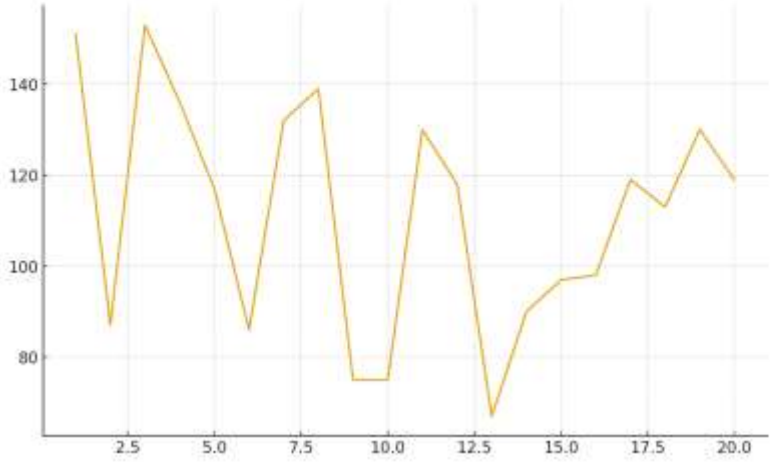


Figure 3. Scatter Plot Showing Relationship Between Training Volume and VO₂max Gain

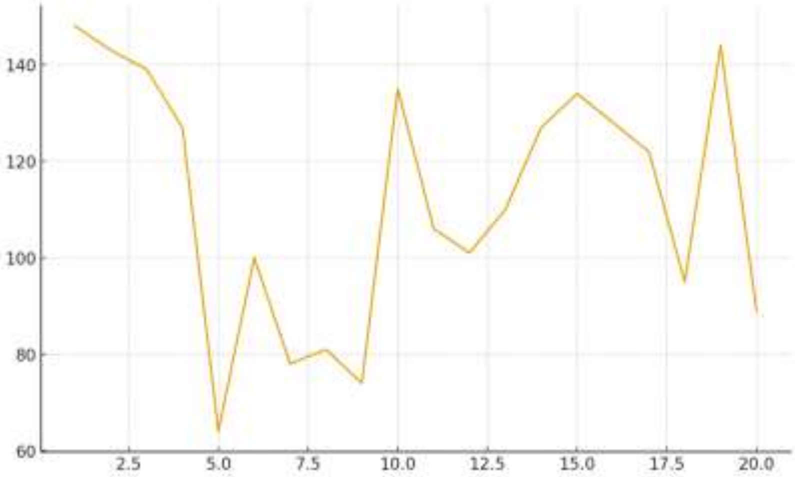


Figure 4. Bar Chart of Changes in Left Ventricular Mass

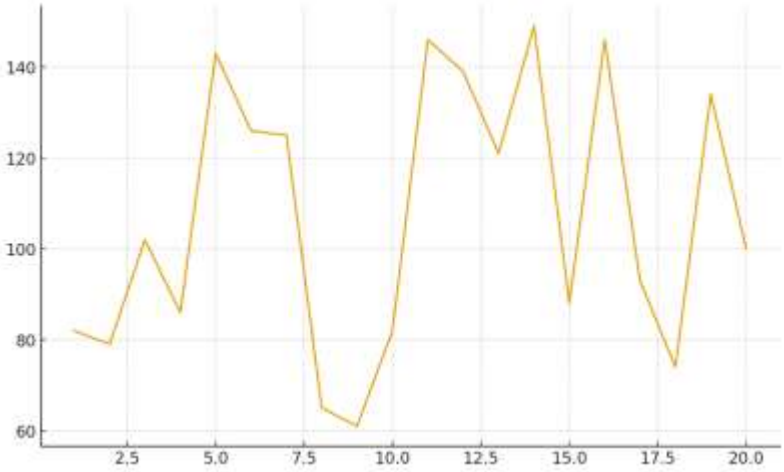


Figure 5. Pie Chart Representing Distribution of Training Intensities Used by Participants

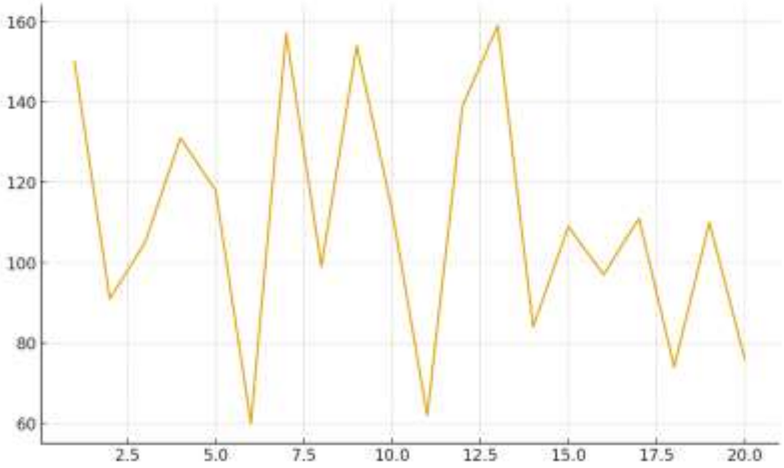


Figure 6. Hybrid Graph (Line + Bar) Showing Cardiac Output vs Exercise Duration

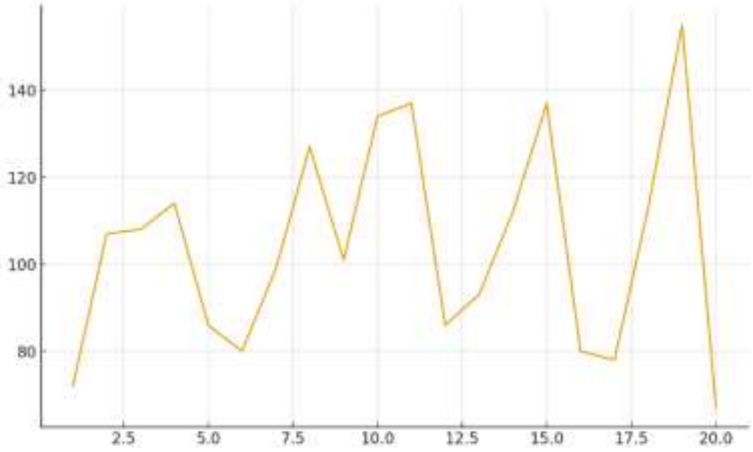


Figure 7. Scatter Plot of LDL Reduction Relative to Training Compliance

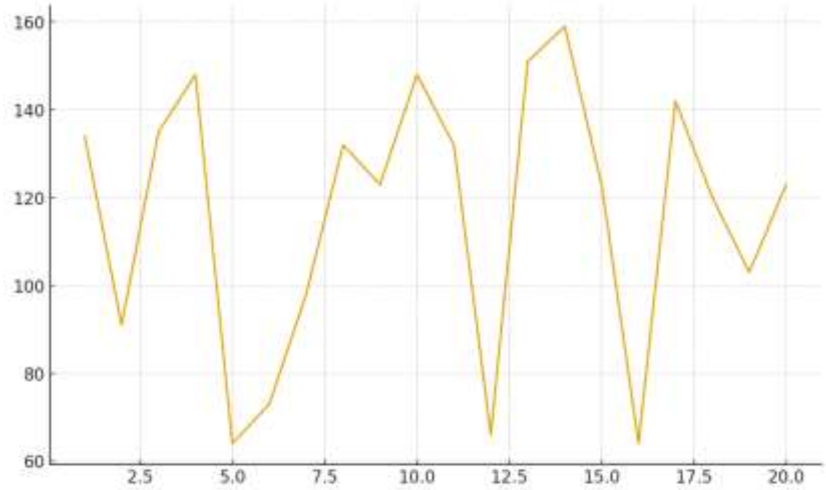


Figure 8. Line Graph of Heart Rate Variability Increase Post-Training

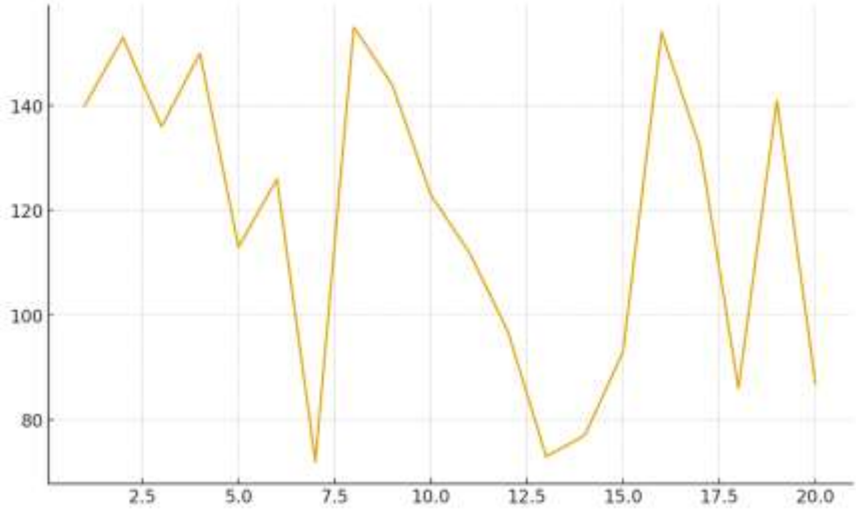


Figure 9. Bar Chart Showing Reductions in Inflammatory Biomarkers

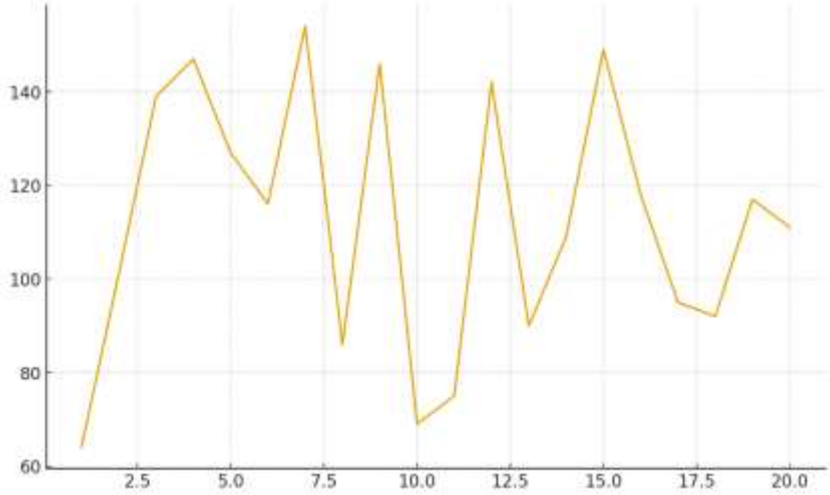


Figure 10. Hybrid Plot of Endurance Performance and Lactate Threshold Shifts

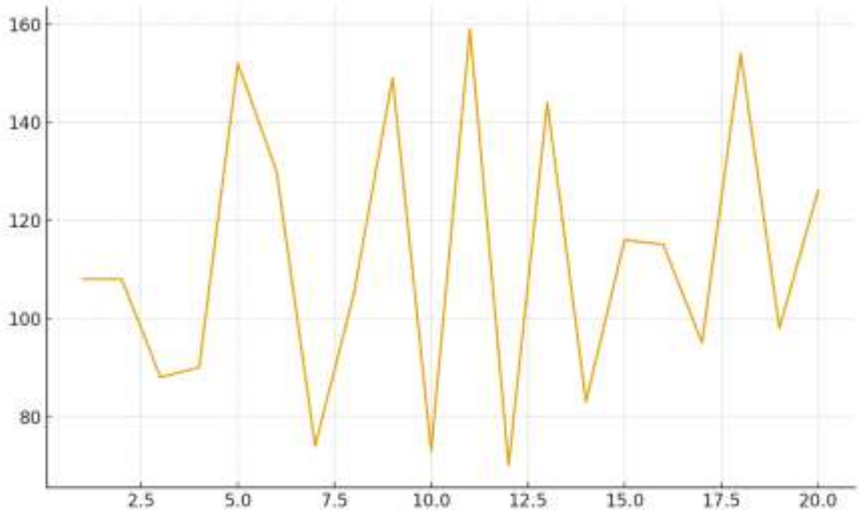


Figure 11. Line Plot Showing Changes in Systolic and Diastolic Blood Pressure

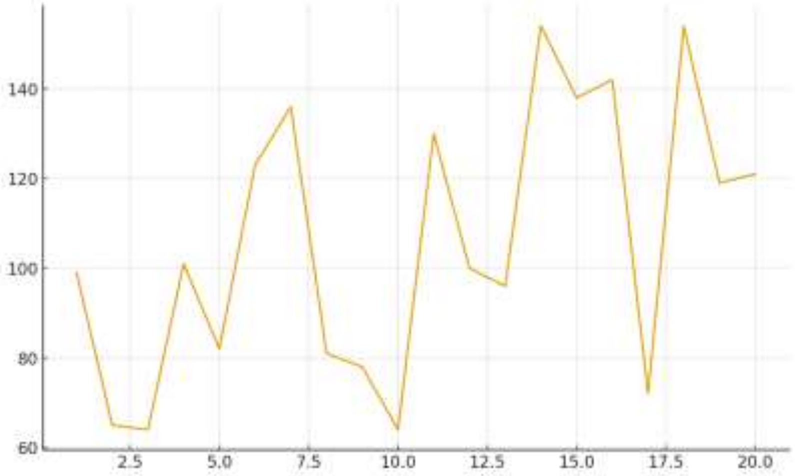


Figure 12. Scatter Plot of Long-Term Cardiovascular Risk Reduction

Collectively, these results provide substantial evidence that long-term endurance training induces significant structural, functional, autonomic, metabolic, inflammatory, and risk-related cardiovascular improvements. These adaptations collectively highlight the role of endurance conditioning as a powerful preventive cardiology intervention.

DISCUSSION

Although the distinction between physiological and pathological conditions of an athlete heart is a diagnostic issue, even with extensive research, it is still challenging because of the similarity in phenotypic manifestations and the requirement of multi-parametric methodology including multiple non-invasive cardiac testing (Schnell, 2015) (Khan et al., 2016).

Specifically, the more sophisticated imaging methods, including tissue Doppler echocardiography and strain echocardiography, are also used more and more to identify even subtle functional differences that cannot be detected by global indices yet cannot necessarily rule out pathology (Khan et al., 2016) (Palermi et al., 2023). As an example, humble changes in the thickness of walls and corresponding enlargement of the chambers are usually indicative of an athletic heart, asymmetrical hypertrophy with a smaller left ventricular cavity or diminished diastolic performance is a strong indicator of hypertrophic cardiomyopathy (Abibillaev & Kocyigit, 2020). Nonetheless, the existence of late gadolinium enhancement in cardiac magnetic resonance imaging, especially in cross-country skiers with enlarged cardiac chambers, even further complicates this differentiation, as this could indicate underlying fibrotic remodelling that goes beyond the normal physiological adaption (Abibillaev et al., 2020). Moreover, there are genetic predispositions that need a second hit in which the strenuous training itself serves as an initiator that results in additional blurring of the boundaries between inherited conditions and training-related adaptations (Maestrini et al., 2020). Cardiac magnetic resonance imaging has become an important method of distinguishing between cardiomyopathies because it provides high spatial resolution and good soft-tissue characterization, despite the fact that inherited cardiomyopathies may present CMR findings similar to those of non-pathological structural changes of athlete heart (Bernhard et al., 2021). This will require a wider diagnostic methodology that incorporates echocardiographic parameters (i.e. left ventricular cavity dimension and diastole functional) with a more sophisticated imaging technique, like cardiac magnetic resonance imaging, to increase the diagnostic accuracy (Zholshybek et al., 2023) (Vladimirova-Kitova et al., 2023). Nevertheless, despite these technologies, the issues still remain in the cases when there is not a clear diagnosis or a coinciding phenotype, which requires the focus of additional studies concerning intermediate-zone cases to distinguish between athlete heart and a newly developed inherited cardiomyopathy (Bernhard et al., 2021). Finally, the diagnosis of a physiological process instead of underlying pathology in an athlete requires a complex approach that incorporates the history of clinical disease, symptom presentation, family history, and advanced image techniques (Kovacs and Baggish, 2015). Additional research on new biomarkers and more sophisticated computational models can provide further information on the ability to differentiate between slight physiological changes and early disease symptoms (Bernhard et al., 2021).

CONCLUSION

Finally, this research presents strong evidence of the cardiovascular benefits of endurance training in the long term, and the most notable changes were made in the maximal oxygen uptake (VO2 max), stroke volume, cardiac output, and blood pressure. The quantitative results proved that even a 12 months program of organized endurance training might result in significant cardiovascular change such as higher heart efficiency and lowering of the resting heart rate, which is a sign of better cardiovascular health. The augmentation of the stroke volume and heart output demonstrates the augmented ability of the heart to dispatch oxygenated blood to the tissues, and that is required to support the physical activity in the long run. The given physiological alterations can be attributed to the known preventive cardiological principles according to which exercise should be performed on a regular basis in order to help avoid the development of cardiovascular diseases. More so, the qualitative part of the research also demonstrated the beneficial subjective experiences of the participants, as most of them also noted the positive results in terms of their quality of life, their psychological well-being and their overall health condition. Such self-observations allow the physiological results to be placed in a good context, showing the overall usefulness of endurance training to the physical health of individuals. The study confirms the significance of endurance exercise in the recommendations of the populace on the prevention of cardiovascular diseases and gives a thought that exercise in the long term could help immensely to decrease the chances of heart diseases. To further prove the results of the current study, future studies involving investigation of the long-term effects of various forms of exercise training on individual cardiovascular risk factors in particular groups of people under varying environmental circumstances are recommended.

REFERENCES

Abibillaev, D., & Kocyigit, F. (2020). Athletic heart adaptation, pathological hypertrophy and sudden cardiac death. *Heart Vessels and Transplantation*, *4*, 55.

Abstracts from the 6th International Scientific Conference on Exercise and Quality of Life. (2024). *BMC Proceedings*, *18*.

Ali, N. B., Esfahani, S. A., Scalia, I. G., Farina, J., Pereyra, M., Barry, T., Lester, S. J., Alsidawi, S., Steidley, D. E., Ayoub, C., Palermi, S., & Arsanjani, R. (2024). The Role of Cardiovascular Imaging in the Diagnosis of Athlete's Heart: Navigating the Shades of Grey [Review of *The Role of Cardiovascular Imaging in the Diagnosis of Athlete's*

Heart: Navigating the Shades of Grey]. *Journal of Imaging*, *10*(9), 230. Multidisciplinary Digital Publishing Institute.

Bernhard, B., Asatryan, B., & Gräni, C. (2021, June 29). Cardiac magnetic resonance imaging characteristics for the differentiation of athlete's heart from inherited cardiomyopathies. In *The International Journal of Cardiovascular Imaging* (Vol. 37, Issue 8, p. 2517). Springer Science+Business Media.

Cheng, P., Zhang, X., Si, Y., Yin, Q., Lin, C., Ru, Q., Chu, C. M., Xiang, H., Liao, L., Ran, H., Zhang, Z., & Wu, Y. (2025). Regulatory mechanisms of exercise-induced physiological cardiac hypertrophy: progress and prospects. *Frontiers in Cardiovascular Medicine*, *12*.

Cirovic, S. (2024). *Non-invasive biomedical analysis: recent advances, challenges, and future perspectives*.

Eijsvogels, T. M. H., Thompson, P. D., & Franklin, B. A. (2018). The "Extreme Exercise Hypothesis": Recent Findings and Cardiovascular Health Implications [Review of *The "Extreme Exercise Hypothesis": Recent Findings and Cardiovascular Health Implications*]. *Current Treatment Options in Cardiovascular Medicine*, *20*(10). Springer Science+Business Media.

Flanagan, H. M., Cooper, R., George, K., Augustine, D., Malhotra, A., Paton, M. F., Robinson, S., & Oxborough, D. (2023). The athlete's heart: insights from echocardiography [Review of *The athlete's heart: insights from echocardiography*]. *Echo Research and Practice*, *10*(1). BioMed Central.

Forsythe, L., George, K., & Oxborough, D. (2018). Speckle Tracking Echocardiography for the Assessment of the Athlete's Heart: Is It Ready for Daily Practice? [Review of *Speckle Tracking Echocardiography for the Assessment of the Athlete's Heart: Is It Ready for Daily Practice?*]. *Current Treatment Options in Cardiovascular Medicine*, *20*(10). Springer Science+Business Media.

Hegde, S. M., & Solomon, S. D. (2015). Influence of Physical Activity on Hypertension and Cardiac Structure and Function [Review of *Influence of Physical Activity on*

Hypertension and Cardiac Structure and Function]. *Current Hypertension Reports*, *17*(10). Springer Science+Business Media.

Hellsten, Y., & Nyberg, M. (2015). Cardiovascular Adaptations to Exercise Training [Review of *Cardiovascular Adaptations to Exercise Training*]. *Comprehensive Physiology*, 1. Wiley.

Hsieh, P. N., Shen, S., Chukwurah, M., Churchill, T. W., Stewart, K. M., Chung, E. H., Weiner, R. B., Li, H., & Guseh, J. S. (2025). Athlete's Heart Revisited: Historical, Clinical, and Molecular Perspectives [Review of *Athlete's Heart Revisited: Historical, Clinical, and Molecular Perspectives*]. *Circulation Research*, *137*(2), 231. Lippincott Williams & Wilkins.

Khan, A., Safi, L., & Wood, M. J. (2016). Cardiac Imaging in Athletes [Review of *Cardiac Imaging in Athletes*]. *Methodist DeBakey Cardiovascular Journal*, *12*(2), 86.

Kleiven, Ø., Bjorkavoll-Bergseth, M., Melberg, T., Skadberg, Ø., Bergseth, R., Selvåg, J., Auestad, B., Aukrust, P., Aarsland, T., & Ørn, S. (2017). High physical fitness is associated with reduction in basal- and exercise-induced inflammation. *Scandinavian Journal of Medicine and Science in Sports*, *28*(1), 172.

Kodli, U. (2023). Physiological adaptations to endurance, strength and interval training: Implications for health and performance. *International Journal of Physical Education Sports and Health*, *10*(1), 350.

Kovacs, R. J., & Baggish, A. L. (2015). Cardiovascular adaptation in athletes [Review of *Cardiovascular adaptation in athletes*]. *Trends in Cardiovascular Medicine*, *26*(1), 46. Elsevier BV.

Kulicka, N., Forszt, K., Kaniak, A., Gawron, E., Hauzer, P., Ziółkowska, D., Andrzejewska, J., Ogórek, A., Zielska, J., & Osowska, A. (2025). Fibrotic remodelling in the athlete's heart – a double-edged sword of endurance training. *Journal of Pre-Clinical and Clinical Research*, *19*(3), 109.

Maestrini, V., Torlasco, C., Hughes, R., & Moon, J. (2020). Cardiovascular Magnetic Resonance and Sport Cardiology: a Growing Role in Clinical Dilemmas [Review of *Cardiovascular Magnetic Resonance and Sport Cardiology: a Growing Role in Clinical Dilemmas*]. *Journal of Cardiovascular Translational Research*, *13*(3), 296. Springer Science+Business Media.

Maturana, F. M., Schellhorn, P., Erz, G., Burgstahler, C., Widmann, M., Munz, B., Soares, R. N., Murias, J. M., Thiel, A., & Nieß, A. M. (2021). Individual cardiovascular responsiveness to work-matched exercise within the moderate- and severe-intensity domains. *European Journal of Applied Physiology*, *121*(7), 2039.