

The Interaction Between Genetic and Lifestyle Factors in Athletes' Cardiovascular Health

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ABSTRACT

Cardiovascular health plays an important role in athletic performance because it affects how efficiently oxygen and nutrients are delivered to working muscles during exercise. This paper will take a look at how genetics and lifestyle factors influence cardiovascular health and function in athletes by looking at heart structure, blood flow, blood pressure regulation, oxygen transport, and energy production. Genetic factors such as ACE, ACTN3, NOS3, MYH7, and TTN are connected to cardiovascular adaptations and risks, including stroke volume, vascular function, muscle metabolism, endurance capacity, and inherited heart conditions. While lifestyle factors like exercise training, nutrition, sleep, and hydration all support cardiovascular system health as they increase vasodilation, angiogenesis, mitochondrial biogenesis, healthy blood pressure, and efficient cardiac output. These adaptations can lead to structural and functional changes in the heart, such as increased left ventricular volume and improved stroke volume, which allow more blood and oxygen to reach working muscles. Differences between endurance and power athletes show that genetic predispositions and training styles interact to influence performance. The paper also explains why these factors should be considered together rather than as separate influences on cardiovascular health. Overall, genetics may provide an athlete's biological starting point, while consistent lifestyle habits are well established to improve cardiovascular efficiency and performance-related physiology. However, evidence that lifestyle behaviors directly offset specific inherited genetic risks is still limited and is often associative rather than causal.

Keywords: cardiovascular health, athletic performance, genetics, lifestyle factors, oxygen delivery, cardiovascular disease

INTRODUCTION

Cardiovascular health plays a key role in athletic performance because it determines how efficiently oxygen and nutrients are delivered to working muscles. Strong heart function, healthy blood vessels, and effective oxygen use allow athletes to generate power, have endurance, and recover efficiently. While training and lifestyle habits are known to improve cardiovascular health, genetic differences also influence heart structure, blood pressure regulation, and muscle metabolism. These genetic variations may give some athletes natural advantages, while others may carry inherited risks related to cardiovascular disease. Therefore, this paper addresses the gap in understanding how inherited genetic differences and controllable lifestyle habits work together to influence cardiovascular health, disease risk,

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and performance in athletes.

METHODS

This paper was completed as a narrative literature review of peer-reviewed scientific articles, reviews, and selected medical or health-organization sources related to genetics, cardiovascular health, and athletic performance. Searches were conducted using credible sources such as Google Scholar, PubMed, and other medical journals, together with targeted searches of journal and publisher websites, during approximately December 2025 to April 2026. No formal publication-date restriction was applied because several foundational genetic studies predate recent exercise-physiology literature. Search terms were combined, such as “cardiovascular health” and athletes, ACE and athletic performance, ACTN3 and endurance or power.

Sources were included when they directly addressed cardiovascular function, genetic variation, exercise adaptations, oxygen transport, vascular physiology, or cardiovascular disease risk relevant to athletes. Sources were excluded when they were not relevant to athletes or cardiovascular physiology, lacked sufficient scientific or methodological detail, or were primarily non-scientific commentary. Peer-reviewed primary studies and systematic reviews were prioritized for causal claims; and clinical websites were retained only for basic background definitions when appropriate. Study quality was considered qualitatively by prioritizing peer-reviewed designs with clearly described methods, larger samples or replicated findings, and systematic reviews/meta-analyses when available. A formal risk-of-bias scoring tool was not applied. Findings were organized into genetic influences, lifestyle factors, cardiovascular adaptations, and gene-environment interactions, and then compared across themes to evaluate the relative strength and limitations of the evidence.

BACKGROUND INFORMATION

Cardiovascular Health

Cardiovascular health is defined as the health of key components in the circulatory system, such as the heart and blood vessels [1]. The circulatory system's role in the human body is to transfer oxygen and nutrients through blood to different organs, tissues, and muscles throughout the body. The heart is responsible for pumping blood. With each heartbeat, blood is being pumped throughout the body with the help of connected blood vessels like veins, capillaries, and arteries, to deliver oxygen and nutrients to vital organs [2]. There are various ways to measure cardiovascular health, such as heart rate, blood pressure, and VO2 max. Heart rate is measured by how many times the heart beats per minute. A resting heart rate is the heart rate of a resting body; a normal resting heart rate is anywhere from 60 to 100 beats per minute. A heart rate that is anywhere over 100 beats per minute can highlight the heart overworking to pump blood throughout the body, while a lower resting heart rate can indicate efficient use of oxygen in the blood [3]. Blood pressure is the measure of the force of the blood pushing on blood vessel walls. A normal blood pressure is 120/80 mm Hg; if blood pressure is consistently high or low, cardiovascular disease is a major risk [4]. VO2 max is a way of measuring physical fitness. It measures how much oxygen your body is taking in during exercise. A higher VO2 max indicates that the heart and lungs are efficiently using oxygen throughout the body, and organs and muscles are using it [5].

Why Cardiovascular Health Matters in Athletic Performance

Athletic performance can be described as how capable an athlete is to perform physical activities effectively, by using factors such as strength, speed, endurance, and skill to succeed in a sport activity or physical performance [6]. All these factors involve various muscle contractions to allow the body to execute athletic movements like jumping and running. Muscle contractions are caused by myosin pulling on actin in muscle fibers, creating force and movement. ATP (adenosine triphosphate) is the energy the body uses to cause the pulling of the myosin. In order for the body to consistently create ATP, cells' mitochondria use aerobic respiration, which turns oxygen and glucose into ATP [7]. To have rapid muscle contractions, like what you would need when running, lots of oxygen is needed. Oxygen is supplied throughout the body using blood. The heart pumps blood throughout the body with the help of other cardiovascular organs in order to supply muscle fibers with oxygen. When oxygen levels are reduced in the body, muscle fatigue occurs faster. In contrast, when oxygen supply is increased, muscle fatigue occurs much more slowly [8]. Hence, good cardiovascular health, where blood carries oxygen effectively to muscle fibers, permits muscles to rapidly contract, allowing athletic movements to be performed longer.

Genetic Basics

Genes are defined as strands of DNA that are blueprints of the human body. DNA is the molecule that makes up your genes and gives instructions to the body. Every cell has DNA that is stored in the chromosomes located in the nucleus of the cell. Genes not only express physical traits such as height, but also encode other non-visible functions of the body, like muscle fiber type or heart size [9]. Specific genes can influence physiological traits in different ways, such as the ACE (angiotensin converting enzyme) gene, which affects blood-pressure and vascular regulation [12]. One frequently studied performance-related gene is ACTN3, whose R577X polymorphism has been associated with sprint/power phenotypes, although it is not a deterministic predictor of athletic ability [11,15].

GENETIC FACTORS' EFFECT ON CARDIOVASCULAR HEALTH

Variations in genes play a crucial role in cardiovascular structure, such as how the heart develops, how it pumps blood, and the regulation of blood vessels. The differences explain how individuals respond to exercise and how some athletes are naturally inclined to endurance or power-based exercises.

The angiotensin-converting enzyme or ACE gene regulates blood pressure and cardiovascular function through the renin-angiotensin-aldosterone system or RAAS [12]. ACE converts angiotensin I into angiotensin II [12]. The hormone angiotensin II causes blood vessels to narrow, also known as vasoconstriction, which causes a rise in blood pressure [12]. Angiotensin II also stimulates aldosterone release, increasing sodium and water retention, contributing to high blood pressure [12]. Variations of this gene are seen in the Insertion polymorphism allele (I) or the deletion polymorphism allele (D), which influence circulating ACE levels [13]. People with the D allele have higher ACE levels compared to people with the I allele [13]. More ACE activity can lead to more production of angiotensin II, which can increase cardiac workload over time [12]. In athletes, the I allele has been observed to be more common in endurance athletes, whereas the D allele is more commonly seen in power and sprint athletes. These findings can suggest that ACE variation can influence blood pressure and cardiovascular adaptation to different types of training [10,14].

The ACTN3 gene gives the body instructions to make alpha-actinin-3, a protein expressed in fast twitch

muscle fibers that contribute to high-force, high-velocity movements [15,16]. The R577X polymorphism can produce a premature stop codon; individuals with the XX genotype do not produce functional alpha-actinin-3 in these fibers [15,16]. The functional R allele is more common among sprint and power athletes in several cohorts, although ACTN3 genotype is not a reliable stand-alone predictor of endurance or power status [10,15]. Experimental evidence also links ACTN3 deficiency with a shift toward more oxidative muscle metabolism [16]. Because muscle fiber type affects oxygen use and ATP production, ACTN3 may indirectly influence cardiovascular demand and fatigue resistance, but its effects are probabilistic rather than deterministic [15,16].

The endothelial nitric oxide synthase gene (NOS3) codes for an enzyme that contributes to nitric oxide production in blood vessels. Nitric oxide promotes vasodilation by relaxing vascular smooth muscle and is important for endothelial function and blood-flow regulation [33]. However, associations between NOS3 polymorphisms and elite athletic status have been inconsistent: some studies report associations with power-oriented phenotypes, whereas a large endurance-athlete study found no difference for several common NOS3 variants between elite endurance athletes and controls [34,35]. Therefore, NOS3 is biologically relevant to vascular physiology, but its value as a stand-alone predictor of athletic performance remains uncertain.

In addition to performance-related traits, inherited variants can increase cardiovascular disease susceptibility. Pathogenic variants involving sarcomeric genes such as MYH7 are well established causes of hypertrophic cardiomyopathy [36]. Hypertrophic cardiomyopathy is an important cause of sudden cardiac death in young people and athletes, although contemporary evidence shows that the causes of sudden cardiac death in athletes are heterogeneous and vary by population [37]. Truncating variants in TTN are a major genetic cause of dilated cardiomyopathy, but penetrance and clinical severity vary among carriers [38]. These examples show that genetic effects range from modest performance associations to clinically important disease-causing variants, and those categories should not be treated as equivalent.

LIFESTYLE FACTORS THAT AFFECT CARDIOVASCULAR HEALTH

Since cardiovascular health is linked to athletic performance, what can athletes do to keep their cardiovascular health in the best condition possible? There are multiple factors that people can control in their day-to-day lives that can improve cardiovascular health by increasing heart efficiency, helping blood flow, and decreasing the risk of cardiovascular disease.

The first lifestyle factor that can improve cardiovascular health and avoid disease is participating in physical activities or training. Physical activity improves the functions of blood vessels and increases oxygen delivery. Repeated exercise promotes vascular adaptations including improved vasodilator function and angiogenesis [17,22]. Vasodilation increases vessel diameter and can improve blood flow, while angiogenesis increases capillary supply to active tissue [17,22]. Angiogenesis is when new capillaries are formed by endothelial cells. Endothelial cells are the cells that make up blood vessels. When cells of a specific organ are not receiving enough oxygen, they signal for angiogenesis, where endothelial cells form new capillaries. With more capillaries, as well as previously existing expanded ones, more oxygen and nutrients can reach the organs and tissues that require them.

Exercise also increases mitochondrial function. Mitochondria are central to ATP production during

athletic performance. Exercise promotes mitochondrial biogenesis and broader mitochondrial remodeling, which can improve oxidative capacity in skeletal muscle [17,18]. PGC-1 α is an important regulator of these adaptations, although it is not the only pathway involved; experimental evidence indicates that some exercise-induced mitochondrial changes can still occur when PGC-1 α signaling is impaired [19]. This illustrates why single-gene or single-protein explanations should be interpreted cautiously.

Additionally, endurance exercise promotes physiological cardiac remodeling and improvements in cardiac output. Meta-analytic and longitudinal evidence shows that endurance training can increase left-ventricular dimensions and mass within a physiological range, while trained endurance athletes can maintain or increase stroke volume at higher exercise intensities [20,21]. These adaptations are consistent with increased diastolic filling and improved pumping capacity, although the magnitude of remodeling differs by training type, sex, age, and training history. Stroke volume is the amount of blood ejected from the left ventricle with each contraction; a higher stroke volume can contribute to greater cardiac output and oxygen delivery during exercise [20].

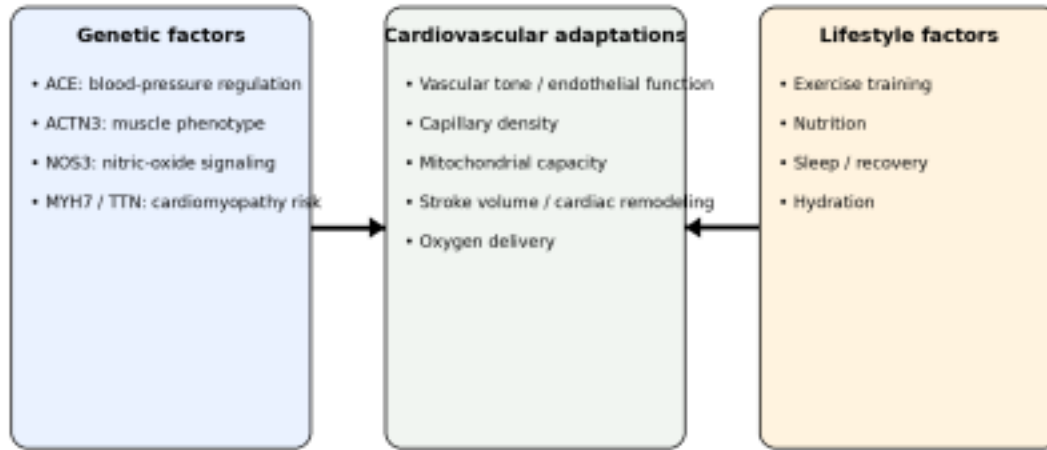
NON-ACTIVITY RELATED CONDITIONS THAT AFFECT CARDIOVASCULAR HEALTH

Proper nutrition supports cardiovascular health in athletes through oxygen transport, vascular function, and energy metabolism. Iron is required for hemoglobin and therefore for oxygen transport; low iron status can impair aerobic performance, particularly when deficiency progresses toward anemia [23]. Dietary nitrate from foods such as leafy greens can be converted through the nitrate nitrite-nitric oxide pathway, increasing nitric-oxide availability and supporting vasodilation [24,25]. Controlled studies and meta-analyses suggest that nitrate can improve exercise efficiency or tolerance in some populations, although effects vary with training status, dose, and exercise type [26]. Dietary patterns also influence vascular function: evidence suggests that high saturated-fat intake can adversely affect endothelial function and arterial stiffness, whereas replacing saturated fat with unsaturated fats may be more favorable [27]. These findings support an overall nutrient-rich diet, but they do not show that a single nutrient can override inherited cardiovascular risk.

Along with nutrition, sleep is another lifestyle factor that can influence cardiovascular regulation. Sleep and sleep deprivation affect autonomic balance, blood pressure, and vascular function [28]. Systematic-review evidence links sleep deprivation with impaired endothelial function, although study designs and measured outcomes vary [29]. A separate systematic review found that total sleep deprivation can alter autonomic and cortisol responses to acute stressors, but the direction of these responses was not consistent across studies [30]. For athletes, adequate sleep therefore supports recovery and cardiovascular regulation, but the available literature does not establish that sleep directly modifies the effect of a specific cardiovascular genotype.

Hydration is important for maintaining blood volume and cardiovascular function during exercise. As dehydration progresses, reductions in blood volume are associated with lower stroke volume and a compensatory increase in heart rate [31]. Experimental work indicates that the fall in stroke volume during dehydration is explained largely by impaired venous return and cardiac filling rather than a loss of intrinsic left-ventricular contractile function [32]. Thus, hydration can preserve cardiovascular performance during exercise, but it should be viewed as a modifiable physiological factor rather than a mechanism that removes inherited cardiovascular risk.

Figure 1. Conceptual interaction of genetic and lifestyle factors in athletic cardiovascular health.



The figure summarizes pathways discussed in the review and does not imply that lifestyle factors directly reverse pathogenic genetic variants.

DISCUSSION

Taken together, the literature supports a stronger causal evidence base for many physiological effects of training than for most single common genetic variants associated with athletic performance. Exercise training consistently produces measurable adaptations in vascular function, mitochondrial capacity, cardiac structure, and stroke volume [17,18,20,21,22]. By comparison, associations between common variants such as ACE, ACTN3, and NOS3 and elite performance are generally smaller, population-dependent, and not uniformly replicated [10,15,34,35]. Genetics therefore appears to influence predisposition and response variability rather than determining a fixed cardiovascular or performance outcome.

The ACE and ACTN3 literature illustrates this difference in evidence strength. ACE insertion/ deletion variants have been associated with endurance or power phenotypes in several cohorts, but these associations do not mean that an allele directly determines training response or cardiovascular health [10,13,14]. Likewise, ACTN3 R577X is repeatedly associated with muscle-performance phenotype, yet training can still produce substantial metabolic adaptation across genotypes [10,15,16]. These findings are more consistent with genetic influence on probability or phenotype than with genetic determinism.

There is also conflicting mechanistic evidence within the exercise-adaptation literature itself. PGC-1 α is widely described as a major regulator of mitochondrial biogenesis, and multiple studies support its role in exercise-induced mitochondrial and angiogenic adaptation [17,18]. However, knockout-model evidence demonstrates that mitochondrial biogenesis can still occur without normal skeletal-muscle PGC-1 α signaling [19]. This does not eliminate the importance of PGC-1 α ; instead, it shows that exercise adaptation is controlled by overlapping biological pathways rather than a single molecular switch.

For inherited cardiomyopathy variants, the interpretation must be even more cautious. Lifestyle behaviors can improve general vascular health, blood-pressure control, hydration status, sleep, and exercise capacity, but they do not remove a pathogenic MYH7 or TTN variant [36,38]. It is therefore more accurate to say that healthy behaviors may reduce additional cardiovascular stress or improve modifiable risk factors than to claim that they “compensate for” or directly modify the causal genetic defect. Direct evidence for gene-by-lifestyle modification of cardiomyopathy risk in athletes remains limited and requires longitudinal and mechanistic study.

Overall, the strongest conclusion is that cardiovascular health in athletes reflects both inherited biology and environmental exposure, but the two forms of evidence are not equally established for every proposed interaction. Training and other lifestyle factors clearly alter cardiovascular physiology; common performance-associated variants may influence baseline traits or responsiveness; and pathogenic variants can create disease risk that lifestyle alone cannot erase. Future research should use prospective athlete cohorts, repeated physiological measurements, genotype-specific training analyses, and mechanistic studies to determine which gene-environment interactions are clinically meaningful.

LIMITATIONS

This review has several limitations. First, it is a narrative review rather than a systematic review, so study selection may be more vulnerable to selection and publication bias. Second, a formal risk-of-bias or evidence-grading tool was not applied, which limits direct comparison of study quality. Third, the included literature spans different athlete populations, training backgrounds, ages, sports, and study designs, making direct comparison difficult. Finally, genetic effects are challenging to separate from training history, diet, sleep, socioeconomic factors, and other environmental exposures. These limitations are particularly important when interpreting reported gene-performance associations or proposed gene-environment interactions as causal.

CONCLUSION

Current evidence supports a model in which genetics contributes to baseline cardiovascular traits and disease susceptibility, while training, nutrition, sleep, and hydration produce well-established physiological effects that shape cardiovascular function and athletic performance. However, evidence that lifestyle factors directly offset specific genetic predispositions is less certain and is often associative. Athletes can meaningfully improve modifiable cardiovascular factors through healthy behaviors, but inherited pathogenic variants remain clinically important. More longitudinal, genotype stratified, and mechanistic research is needed to define when gene-environment interactions meaningfully alter cardiovascular outcomes in athletes.

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