

# Effects of Excessive Endurance Activity on the Heart

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## Abstract

Regular moderate exercise confers many cardiovascular and health benefits. Because of this, endurance sports events have become very popular with participation increasing tremendously over the past few years. In conjunction with this increase in popularity and participation, people also have increased the amount that they exercise with many training for and competing in ultraendurance events such as ultradistance running events, iron distance triathlons, or multiday races. This excess endurance activity may appear to increase the risk of cardiac abnormalities, which may increase the risk for long-term morbidity or mortality. While it is known that moderate exercise has benefits to cardiovascular health, ultimately, the long-term cardiac effects of excessive endurance activity are unclear. What is clear, however, is that moderate exercise is beneficial, and to date, the evidence does not support recommending against physical activity.

## Introduction

It has long been known that regular moderate exercise has been shown to have many benefits. Exercise reduces the risk of heart disease, hypertension, heart failure, depression, and diabetes (7). Those who perform regular exercise live longer and are more functional for the duration of their lives (5). The American College of Sports Medicine, as part of its Exercise is Medicine® initiative, recommends 150 min·wk<sup>-1</sup> of moderate physical activity (1). A recent study in Taiwan demonstrated a small but measureable benefit from exercising as little as 90 min·wk<sup>-1</sup> with only minimal additional health benefits from exercising longer than 1 h·d<sup>-1</sup> (37). Similarly, in a study of runners, those that ran less, up to 20 miles·wk<sup>-1</sup>, received a mortality benefit while those that ran more than 20 miles·wk<sup>-1</sup> did not reap significant additional health benefits (14). It would appear that a U-shaped relationship between exercise and benefit gained exists, where initially there is benefit gained, but more exercise may not always be better.

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The running boom of the 1980s created a culture of marathoners as endurance junkies, and those that completed a marathon were considered extreme. However, in the last 20 years, the number of people participating in marathon races has increased 20-fold (15). With marathons becoming more “main-stream” and less extreme, endurance aficionados have moved toward longer, more challenging events such as long-distance triathlons (140.6 miles) and ultramarathons to distinguish themselves as hardcore endurance athletes. These ultradistance events require many hours of training per week, and the competitions last hours to even days. Multisport or ultraendurance athletes

indeed may be exercise addicts who obsessively engage in a pattern of compulsive and excessive daily exercise, which may lead to physical health consequences (40) to include cardiac abnormalities.

## Athlete's Heart

Long-term endurance training leads to adaptations termed the “athlete's heart.” These adaptations include increased left and right ventricular (RV) volumes as well as increased left ventricular (LV) wall thickness and increased left atrial size (25,26). Because in endurance athletes, the LV ejection fraction is maintained, these adaptations have long been viewed as benign (16,25). However current events may suggest that excessive endurance training actually may lead to cardiac damage.

The recent deaths of Olympic Trials marathoner Ryan Shay and ultramarathon runner Micah True (17), both of whom were elite level or veteran endurance athletes, revealed that even high-level athletes are not immune from sudden cardiac death. The autopsies on both revealed some degree of myocardial fibrosis or scarring, which furthered this idea that too much endurance activity may promote cardiac abnormalities. In addition, Whyte et al. (38) published results of a post-mortem examination of a highly trained athlete with a 20-year running history that experienced sudden cardiac arrest while running a marathon who exhibited similar findings of idiopathic LV hypertrophy and

interstitial myocardial fibrosis. It would appear then that long-term participation in endurance activity leads to cardiac abnormalities, which in fact may lead to sudden death. But is this the case?

### Imaging

Many of these endurance adaptations have become apparent due to the improvement in cardiac imaging technology. Echocardiography is among the most commonly available technique; however it contains inherent variability that makes reproducibility difficult. Such problems include user variance and the inability to quantify small changes in cardiac function due to complex 3-D geometry of the chambers visualized on a 2-D screen. But echocardiography has been around since the late 1960s, has widespread availability, and therefore has more data associated with it. Furthermore the convenience and portability of echo allows researchers to examine participants immediately posttrace, which have allowed the amount of echo-based information to fill the literature. However newer technologies have allowed more precise measurements of cardiac structure and function than what echo can evaluate. To date, cardiac magnetic resonance (CMR) has been shown to characterize best the athlete's heart as demonstrated by La Gerche et al. (10). CMR has been demonstrated to be the reference standard for the assessment of ventricular dimensions, function, and mass in terms of accuracy and reproducibility (41). Furthermore the advent of diffusion tensor weight magnetic resonance imaging has allowed the ability to visualize the orientation of the myocardial fibers, which allows for recognition of more potential abnormalities. Thus it is unclear if there are actually more cardiac abnormalities or if advances in imaging have allowed us to detect more subtle findings than before.

### Short- and Long-Term Effects

These cardiac effects of endurance activity can be recognized both acutely and chronically. Multiple studies have evaluated cardiac activity at numerous time intervals (minutes, hours, weeks, or months) following high endurance activity and competition in attempts to gain data as to cardiac compensation and decompensation following extreme cardiac demand. Most findings, including systolic and diastolic ventricular dysfunction, increased serum cardiac biomarkers, and decreased RV ejection fractions, were shown to persist for no more than 1 wk (10,11,20,34). La Gerche et al. (13) term this phenomenon "cardiac fatigue." It is unclear, however, what the effects of completing another hard endurance effort before the heart has had time to recover are. Although seen primarily as a transient phenomenon, little is known about the long-term sequelae such repetitive effects may have on cardiac myocytes, the coronary vasculature, and the arrhythmogenic potential this may cause in the future.

Chronically the physiologic adaptations the RV displays following the conclusion of ultraendurance activities have been well studied. Whether maladaptive or beneficial, the RV has displayed an increased size, increased pressure, increased volume, and a reduction in function (23,24). According to the Frank-Starling mechanism, the aforementioned stretch of the RV should augment contractility. However it has been proposed that the increased RV volume likely represents a true impairment (10). Support for this finding is seen by the dose-dependent reduction in RV function as observed more

prominently in those athletes whose endurance was tested with a longer duration and subsequently found to have a finite functional end point (10,12). It has been speculated that the RV is less compliant and not as adaptive as the LV, and therefore, excessive loads may lead to maladaptation or disruption of myocyte junctions, which may be the mechanism that leads to fibrosis (11). What may confound the issue is that many of these studies were conducted on professional cyclists, many of whom may have been using performance enhancing drugs (3); therefore it is unclear if these findings are generalizable to amateur or recreational athletes. However the majority of the evidence would support that this cardiac adaptation is more of a beneficial physiologic response to exercise than a true impairment (2).

### Biomarker Analysis

Studies demonstrate the improvements in imaging modalities to evaluate cardiac structure and function, while the laboratory analysis has remained relatively unchanged over the years. Although the studies have differed in their choice of cardiac biomarker (myoglobin, creatine kinase, creatine kinase MB, troponin T, troponin I, or b-type natriuretic peptide), the results have been consistent in finding increased levels of biomarkers after an acute bout of prolonged endurance exercise when compared with control subjects, which could potentially indicate myocardial damage (6,10,11,20–22,27,28,32–34,36,41). However, troponin rise following endurance exercise normalizes within 36 h, which is unlike the troponin rise after myocardial infarction, which may stay elevated for as long as 10 d. This difference may indicate a different stimulus for biomarker increase, and perhaps strenuous endurance activity is more of a mechanism for an enzyme leak than true muscle damage (36).

Technological advances in imaging as mentioned allow researchers to look more specifically at cardiac muscle architecture for myocardial injury or necrosis following strenuous activity. As mentioned, elevated cardiac biomarkers have been routinely found to be elevated in ultraendurance athletes following competition. Different imaging modalities, most often echocardiography, have been used to correlate simultaneously findings of elevated cardiac enzymes with reduced cardiac function. Given the previously described limitations of echocardiography, the results have been inconsistent, and while many studies correlated elevated enzymes with reduced ventricular contractility, a true causal relationship has been hard to prove. Initially it was speculated that these post-exercise cardiac enzyme elevations were linked to myocardial tissue damage; however emerging CMR data may contradict those findings as no evidence of myocardial edema or true myocardial necrosis is present on CMR imaging following endurance activity even with a biomarker increase (20).

It seems that with improved imaging technology, recent research has determined that the increase in cardiac troponin after a marathon or endurance activity may not be due to myocyte damage and the release of other cardiac biomarkers may not appear to be linked with ischemic injury, but some other mechanism for enzyme release may exist (36). Furthermore it has been noted that exercise-induced cardiac damage is the only recognized stimulus of cardiac troponin release that is not associated with poor clinical outcome as most people recover quickly without permanent cardiac effects

(32). Therefore the rise in troponin after endurance exercise is probably better described as a sign of adaptive heart remodeling than true heart damage.

With supportive evidence against myocardial cell necrosis as a cause of cardiac biomarker elevation, it has been proposed that an increase in membrane permeability is a factor in its release (32). The increased mechanical stress on the myocytes may lead to transient disruptions of the plasma membrane, making it possible that biomarkers may leak out into extracellular compartments (34). Although the acute release of cardiac biomarkers does not appear to be pathologic in nature, 47% of participants who have been studied have been found to have troponin-positive elevations (34). Finally little is known about the long-term cardiac implications of repeated release of intracellular cardiac biomarkers after repetitive endurance events; however current evidence indicates it is more adaptive than pathologic.

### **Accelerated Atherosclerosis**

While exercise does not appear to have an ischemic or necrotic effect on cardiac muscle, several studies of veteran marathon runners demonstrated an increase in coronary artery plaque (measured with coronary artery calcium score) compared with nonrunners (18,31). However, what clouds this finding is that increased LV mass may contribute to elevated coronary calcium scores (18) and endurance athletes who possess physiologic cardiac changes will have an enlarged LV when compared with nonathletes, which may contribute to this higher calcium score. It has been noted, however, that in many of the cardiac exercise studies looking at atherosclerosis markers in exercising adults, there was no record of the runners health before the study (35). It is therefore possible that the runners previously demonstrated poor health habits or already had developed some degree of heart disease before they became runners, which then contributed to their increased calcium scores (29). Thus, the significance of the elevated coronary calcium score in endurance athletes is unknown.

### **Arrhythmias**

Finally, rhythm abnormalities are a growing concern for ultraendurance athletes. Middle-aged endurance athletes possess a fivefold increase in the risk of atrial fibrillation when compared with nonathletes (19,39). The mechanism has been speculated to be secondary to the atrial remodeling and the autonomic changes that occur following long periods of endurance activity (4,39). Heidbuchel et al. (9) coined the phrase exercise-induced arrhythmogenic RV cardiomyopathy (ARVC) to describe the outcome of repetitive RV strain experienced by intense endurance athletes. A majority of ventricular arrhythmias in endurance athletes stem from the RV origin. Knowledge that the RV is insulted repetitively with high-intensity training may be speculated as a potential cause for this ARVC (9). Although the exact dose of cardiac stress that exceeds the ability to accommodate is not known, it has been demonstrated that the prolonged hemodynamic demands placed upon it from excessive endurance activity may lead to maladaptations; however further research is needed.

### **Fibrosis**

Perhaps a more concerning finding for veteran endurance athletes is the presence of late gadolinium enhancement

visualized on CMR imaging suggestive of myocardial fibrosis as was seen in Ryan Shay and in Micah True following their exercise-related deaths. Studies demonstrate that fibrosis was found to be associated with the number of years training and the number of competitive marathons completed with more cumulative years and competitions resulting in a higher likelihood of scarring (42). This fibrotic structural heart disease then is hypothesized to be a potential trigger for arrhythmias in athletes, which then may proliferate to fibrillation and ultimately death. However, the studies that evaluate this fibrosis (38,42) have limited numbers of participants, which makes it unclear whether this fibrosis is truly pathologic or the product of the increased sensitivity of improved imaging technology.

Why do these potentially dangerous changes to the heart occur? It is thought that individuals who do long-term endurance training over prolonged distances and maintain an elevated heart rate, blood pressure, and cardiac output for long periods may generate myocyte inflammation and free radicals, which over a long-term repeated basis may lead to these adverse adaptations (10). Furthermore this repetitive cycle may stimulate the immune system to secrete signals that cause adverse changes in the cardiac muscle contributing to fibrosis (8). It has been demonstrated by previous research that the acute structural changes caused by endurance activity resolve within a week (10), and perhaps allowing for a recovery period between intense endurance activities may mitigate the risk of developing maladaptations. However, the ultimate causal insult is not known, and further research into the long-term cardiac implications of endurance activity is needed.

### **Exercise Advice**

How then should we advise people who desire to pursue endurance sport? It is clear from many studies that regular exercise reduces overall risk of death, and therefore we should be recommending exercise to our patients. It is paradoxical however that the risk of sudden death rises during vigorous exercise in susceptible individuals (35), but we know that the cumulative effect of regular exercise reduces the baseline risk and there is much evidence to support the benefits of exercise (35). Furthermore those who choose to participate in long-term endurance exercise may have existing or congenital risk factors despite regular aerobic exercise. Although exercise is beneficial, it may not overcome existing cardiovascular risk factors such as high blood pressure, high cholesterol, diabetes, smoking history, or strong family history of cardiovascular disease. Good advice for people older than 40 years, or those with existing risk factors, is to consult with a physician for risk stratification prior to beginning an endurance training program. As standard exercise stress testing has not been found to be helpful in screening extreme endurance athletes, it is unclear what the best method of risk stratification is in this population (30). If exercise testing is to be used, it should be as specific to the endurance activity as possible. Although not cost-effective at this time, coronary calcium scoring may have a role as a method of risk stratification. If the calcium score is greater than 100, the athlete may be at higher risk for a cardiac event during exercise and therefore may warrant further or more invasive evaluation (29). The current uncertainty of the significance of an elevated calcium score in endurance athletes does not preclude exercise in those with

a high score, but elevated calcium, especially in light of other existing risk factors, may necessitate advising athletes to moderate physical activity or to further consider the risk that excessive endurance activity may pose.

## Conclusion

Although we have more information regarding the potential long-term effects of excessive endurance activity on the heart, there is still much to learn. Further research is needed to determine the exact nature of the long-term risk to endurance athletes. It seems that some individuals may be more susceptible to long-term damage, whereas others have no long-term risk, but it is unclear exactly what increases susceptibility. The evidence to date would suggest that extreme or obsessive exercisers, as well as those with existing risk factors, are at more risk for cardiac issues than those who practice moderation. Finally, as the cardiac benefits of exercise do not seem to increase after  $1 \text{ h} \cdot \text{d}^{-1}$  and the risk level may indeed rise after this threshold, susceptible individuals should be advised to adhere to moderate exercise, as there is plenty of evidence to support the benefits of moderate physical activity.

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