# Olympic Challenges: Performance and Perseverance

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Sport and the heart - **R. G. Budgett**

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## Lead Article

Exercise and the heart - **G. Whyte, K. George**

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Invited Editorial

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SPORT AND THE HEART

This issue of *Dialogues in Cardiovascular Medicine* comes in the lead-up to the Olympic Games in London, so appropriately covers the issues of sport and the heart. The Olympic Motto “Citius, Altius, Fortius” (faster, higher, stronger) will be on everyone’s mind as they watch Olympic athletes from 26 different sports and Paralympic athletes from 20 different sports striving for medals and glory. The main determinant of athletic performance in many sports was long thought to be the heart. This issue tackles the effect of the heart on performance and the effect of exercise on the heart, be it short and intense or prolonged or extremely prolonged. The debate on the relative importance of mind, muscles, genes, and the heart is central to our understanding of elite performance as well as our understanding of human capacity and the effect of exercise and training on physiology and pathology. A major anxiety for all athletes in the months leading up to an Olympic Games is the risk of injury or illness and the safety of the training and exercise that they do. If an athlete is unlucky enough to be ill or injured at the wrong time then all the years of preparation are wasted. On the other hand, if athletes do not exercise and train to the limit of their ability then they are unlikely to reach their full potential or win an Olympic medal.

The Lead Article and the three Respondent papers in this issue of *Dialogues* rightly point out that there are a number of variants that determine performance. Studying these and the response of athletes to extreme exercise can improve our understanding of disease and help us to advise everyone on how best to exercise and use exercise as a therapeutic tool.

Elite sport celebrates human performance and endeavor. Quite rightly, athletes and their support staff push against the limits of human performance, but there is also a concern to protect and promote athlete health. This is the prime responsibility of any medical personnel caring for athletes and the International Olympic Committee has published a medical code that stresses the ethical responsibilities of support staff and that the primary responsibility is to protect the health of athletes and not to enable...
performance at any cost. It is interesting to note that this is exactly what the regulation originating within the central nervous system which Timothy D. Noakes ascribes to a “Central Governor” has been doing for millennia in the human species, protecting the health of the individual against the desire to perform at all costs.

The relative importance of the heart, brain, muscles, and genes on human performance and sport is not easily untangled. We were all taught for years that the cardiac pump was the critical determinant of VO2max which in turn was the limiting factor for any endurance performance. Reading this issue of Dialogues you will rightly question whether this is an illusion and all in the mind.

The Lead Article by Gregory Whyte and Keith George on “Exercise and the Heart” focuses on health and the possible small risk of injury in ultra marathon, extremely long distance runners. The authors do not claim that the heart is either a limiting factor or remains a determinant of human sporting performance, but that exercise and training have a very significant effect on cardiac function, which is nearly always beneficial. It is possible that there are a few individuals who after many years of ultra marathon training may develop cardiac fibrosis and predisposition to arrhythmias. It is not possible to predict who these individuals are. The debate on screening continues with recommendations from the International Olympic Committee (IOC) on clinical evaluation and ECG and the practice in Italy of screening all young teenagers about to enter sport, with the consequent reduction in death from hypertrophic obstructive cardiomyopathy (HOCM). Nevertheless, it is still not possible to show that this preparticipation screening is cost effective. Overall, the benefits of exercising and training far outweigh any risks, and particularly as individuals age can reduce the decline in cardiac function and VO2max.

It appears to be part of human nature to constantly push past any barrier, and records show a steady improvement in performance in all sports where it is measured. A few decades ago individuals who ran marathons were considered extreme. Now millions of people run marathons every year and ultra marathon running (for instance races lasting several days) is becoming increasingly popular. Extreme exercise may lead to injury and illness, but overall is still likely to be beneficial to the individual concerned. Of more benefit is the related increase in number of people who are taking part in much shorter fun runs, cycling events, or simply walking. The most significant improvement in health, thanks to exercise, is in those individuals that move from a totally sedentary lifestyle to one involving at least some gentle activity.

The Nature versus Nurture debate continues. The recently popular theme that there is unlimited potential in everyone, so-called “practice sufficiency” falls down on detailed analysis. 10 000 hours of quality practice will not always produce champions and the reality of a genetic ceiling on performance argues against this extreme view. Certainly all individuals are different and unique. This is one of the facts that make sports so
interesting. The huge variety of sports available means that sport for all is a realistic goal and there are some athletic activities that every individual can take part in with some degree of success and enjoyment. The joy of sport can be used to get people exercising both for health and as a therapeutic tool as the lack of exercise resulting in low fitness is a more serious epidemic even than smoking, diabetes, and obesity.

Exercise is the “new snake oil” of the 21st century with the ability of curing virtually everything, but with the remarkable twist that there is evidence that it actually does. With any therapy there is the concern that it may cause harm, but these articles are reassuring evidence that damage is light and rare with very few individuals suffering from cardiac fibrosis. The healthy heart and muscle respond positively to exercise with subsequent benefits to health, and the sport and the medical communities must continue to do as much as possible to reduce any injury and illness caused by training. Participation screening has some way to go before it is universally accepted as a cost-effective exercise for the whole population.

There are many different types of sport that mirror the different types of exercise, which can be broadly categorized into short sprints, 1 to 7 minutes, long distance, and prolonged endurance. Different factors limit different forms and types of exercise, but it is interesting that a maximum of 60% of muscle fibers are activated at any one time. The so-called “Central Governor” contributes even in sprints, but is particularly relevant in middle and long distance exercise, so metabolism undoubtedly contributes and diet can make a difference to this, by changing the respiratory exchange ratio (RER) from 0.8 to 0.95 by high fat to high carbohydrate diet or by increasing phosphocreatine stores in the muscle. This aspect is addressed in detail by Asker E. Jeukendrup in his paper “Performance and endurance in sport: can it all be explained by metabolism and its manipulation?” The Central Governor kicks in when there is physiological stress, and so changing that stress with training, thereby optimizing muscle stores and buffering capacity, can change performance up to a presumed genetic ceiling. Even in extreme exercise the cellular environment is maintained, protecting the system from permanent damage. This is a beautiful protective mechanism, as described by Timothy D. Noakes in his paper entitled: “In sports is it all mind over matter?”

Nevertheless, athletes may become chronically fatigued as well as acutely fatigued, in which case their underperformance may become more prolonged. Normal physiological recovery from even very hard training should occur within a few days. Recovery taking longer than 2 weeks is always pathological. This has been labeled overtraining syndrome or unexplained underperformance syndrome in athletes, and this is similar in some ways to chronic fatigue syndrome. In patients it appears to be related to changes in brain and muscle as well as the hormonal system. Noakes describes athletes producing their best performance when they essentially ignore fatigue, focusing just on the perfect outcome. In skill sports they need to focus on sport rather than overanalyzing technique.
In both acute and chronic fatigue, focus shifts on to the fatigue itself and this so-called “marvelous protection” that has evolved to protect the body from catastrophic injury becomes inappropriate and causes prolonged dysfunction. The good news for athletes is that chronic fatigue syndrome and overtraining syndrome/unexplained underperformance syndrome respond positively to a graded exercise regime and cognitive behavioral therapy. These are the only evidence-based treatments for chronic fatigue syndrome. It is notable that they are essentially treating the brain and muscle, which are the two areas most implicated in the production of fatigue and limitation of performance. Muscles (both skeletal and cardiac) adapt quickly to exercise stress, which has benefits to health, from reduced falls to increased capacity to exercise. Sport and exercise medicine is a medical specialty that has become increasingly recognized throughout the world and became a recognized specialty within the National Health Service (NHS) in the UK in February 2005. The main aim is to promote exercise as a therapeutic tool as well as treating injuries. The predisposition to injury is a very important limiting factor in the ability of elite athletes to reach their full potential, and also in all other individuals trying to adhere to an exercise program. There is evidence that injury incidence can be reduced by using preventative exercises, changing the rules and making sport safer, and adjusting training. Profiling is widely used, particularly in elite sport, but there is no evidence that anything apart from a history of previous injury is predictive of subsequent injury.

Ross Tucker and Malcolm Collins look at this from the genetics angle in their paper “Athletic performance and risk of injury: can genes explain it all?” Genetics, as well as determining a ceiling on athletic performance, also leads to a predisposition to particular injuries in the future, although genes may not predict Olympic champions since all those at the top of a particular sport are likely to be genetically heterogenous. Genetics may help us to predict which individuals will break down with illness or injury, enabling us to protect the health of athletes more effectively. The Olympics and other elite sport essentially celebrate individuals at one extreme of the distribution curve. The huge variety of sports means that most individuals will be able to perform well in some activity and can be encouraged to take part in exercise for recreation, for health, and in some for the joy of competing. As the pharmaceutical industry develops targeted treatment, depending on genetic makeup, those involved in sport should be able to start to use the same genetic makeup to guide the best training and exercise primarily for health, and secondarily for performance.

The four aforementioned reviews in this issue of Dialogues provide a compelling and fascinating insight into the effect of exercise on heart and muscle and the effect of genes, the mind, and muscle on performance. These effects are vital to the performance of elite athletes, but are also of great importance to anyone exercising and to physicians guiding their patients, and prescribing and using exercise as a therapeutic tool.
Exercise and the heart

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The cardiovascular benefits of exercise are well documented and have led to the development of physical activity guidelines for promoting health. At the onset of exercise the heart responds instantly through a complex cascade of events resulting in an increased cardiac output. The acute physiological insult of exercise generally lasts for short periods of time and appears to be entirely positive. In contrast, athletes engaged in (ultra)endurance events commonly present with altered cardiac function and markers of cardiac damage that are believed to be physiologic in nature. However, recent evidence suggests a potential pathophysiological cascade. Exercise training in young individuals results in physiological adaptations of the heart that may mimic diseases associated with sudden cardiac death (SCD). While the incidence of SCD in young athletes is low, preparticipation screening has been adopted globally. The impact of exercise in older populations is less well characterized; however, it appears to be positive. In contrast, an increased prevalence of arrhythmias and cardiac fibrosis has questioned the safety of (ultra)endurance exercise in veteran competitors. Despite concerns regarding the acute and chronic impact of (ultra)endurance exercise, the vast majority of evidence supports a beneficial effect of exercise on cardiac health across the life span in health and disease.

The principal drivers of exercise in modern society are sport and health. While there is a plethora of sociobiological benefits to be derived from engaging in sporting activities, it is exercise for health that has gained greatest attention, largely associated with the substantial cardiovascular health benefits gained from being physically active. A large number of physical activity consensus statements and guidelines have been published by governments, international and national learned bodies, and individual academics and practitioners. These guidelines have been developed for the general population and a variety of diseased populations with the goal of promoting physical activity, improving health, and enhancing quality of life. In line with the goals of exercise, the increasing promotion and popularity of sport has resulted in a significant and growing number of participants in a range of (semi)competitive events from short to ultra-endurance distances.
Exercise is a potent physiological stress that acts upon all systems simultaneously, leading to a disturbance of physiological homeostasis the magnitude of which is associated with the intensity and duration of exercise together with other factors including environmental conditions. This homeostatic insult results in an acute response of all systems at the center of which is the heart. This review will begin by examining the complex cascade of events that characterize the acute response of the heart to exercise, including the chronotropic (heart rate) and inotropic (contractility) responses that lead to an increase in cardiac output and resultant oxygen delivery to the working muscles essential for the maintenance of physical work. While the beneficial health effects of moderate intensity, moderate duration exercise appear entirely positive, recent work has reported altered cardiac function and humoral markers of cardiac damage following prolonged arduous exercise, often termed ultra-endurance exercise (>3 hours). The performance and clinical significance of this response remains to be fully elucidated, and a review of the available literature is presented here exploring contemporary thinking in the field.

Exercise training is characterized by the chronic repetition of exercise over weeks, months, and years. As a result the heart adapts to the impact of the fluctuating physiological insult and in doing so improves functional capacity at rest and during exercise. The structural and functional adaptations that occur in the exercise-trained heart are commonly referred to as the “athlete’s heart” (AH) and are characterized by cardiac enlargement and electrical conduction anomalies observed on the ECG. These changes may mimic diseases associated with sudden cardiac death (SCD) in young athletes and as such the differentiation of physiological from pathological mechanisms underlying the observed changes in exercise-trained individuals is important. This review will examine the adaptation of the heart in young athletes and explore the debate surrounding SCD in young athletes and the role of cardiovascular pre-participation screening to identify at-risk athletes.

The vast majority of available data examining the adaptation of the human heart to exercise training have focused on young individuals. In contrast, less is known regarding the cardiac adaptation to chronic exercise in older individuals. The growing popularity and promotion of exercise and sport in veteran populations underpins our need for knowledge in this area. In addition to the cardiac response to commonly prescribed exercise doses of moderate intensity and duration, this review will examine the impact of (ultra)endurance exercise on cardiac function in veteran athletes and investigate the health consequences of arduous, prolonged training and competition on the heart.

**CARDIAC ADAPTATION TO ACUTE EXERCISE**

The ability to perform exercise is dependent upon a highly coordinated, multiorgan response whose primary aim is to maintain homeostasis as skeletal muscles increase the body’s metabolic rate. When humans engage in exercise, the heart acts, quite literally, as the “engine” of the circulatory system, delivering oxygen and nutrients to metabolically active tissues. The heart responds rapidly at the onset of exercise and together with an enormous adaptive capacity allows humans to perform a range of different exercises. The following section describes how the heart responds to acute exercise that may differ in mode, intensity, and duration. This is important for our understanding of how cardiac function underpins sporting performance and the role of physical activity in promoting cardiovascular health.

Cardiac functional response to exercise

Exercise can vary according to type (mode), intensity, and duration. Exercise intensity and duration are often inextricably and inversely linked. In activities of very high intensity (eg, weightlifting or sprinting) exercise cannot be sustained for long durations due to limitations in substrate availability or metabolic waste product accumulation. Lower intensities of exercise (eg, walking or submaximal running) can be maintained for much longer periods of time. The nature of the exercise in relation to mode, intensity, and duration will have a significant impact on cardiac response and the total cardiac work required.

**Graded aerobic exercise**

Aerobic (submaximal) physical activity normally involves large muscle groups in repetitive dynamic exercise and is commonly employed in sporting performance and health contexts. The cardiac responses to graded aerobic exercise from rest to VO2max have been studied more extensively than any other form of exercise.

As the pump at the center of the closed loop circulatory system, the heart’s major role is to contract, develop pressure, and thus produce blood flow into the pulmonary (serving the alveolar capillary beds) and systemic circulation (serving all other capillary beds).
At rest, the heart produces flow, termed cardiac output (Qc) to meet the resting metabolic demands of the body. Cardiac output represents the sum of heart rate (HR, the number of ventricular beats per minute) and stroke volume (SV, the amount of blood ejected per beat). A resting HR (HRrest) of around 70 beats·min⁻¹ and a resting stroke volume (SVrest) of approximately 75 mL contribute to resting Qc (Qcrest) of ca 5 to 6 L of blood per minute ejected from each ventricle.

Heart rate is under both intrinsic and extrinsic control. Pacemaker cells in the sinoatrial node (SAN) can depolarize independently to initiate a cardiac cycle, but in most healthy humans, the SAN is under the neural influence of the autonomic neural system (ANS). The parasympathetic branch of the ANS acts to slow SAN depolarization and reduce HR, whereas the sympathetic branch of the ANS will speed up SAN depolarization and increase HR. At rest, the neural dominance on the SAN is parasympathetic such that resting HR (ca 70 beats·min⁻¹) is below the intrinsic SAN rate (ca 100 beats·min⁻¹). Left ventricular (LV) SV is the difference between LV end-diastolic (LVEDV) and end-systolic volumes (LVESV). LVEDV is dependent upon the preload (ventricular filling time and filling pressure gradient), whereas LVESV is determined by preload, contractility, and afterload. Preload relates to the extent to which the ventricle is filled and stretched prior to contracting. Conventionally, preload is represented by LVEDV. Contractility (inotropism) relates to the force of ventricular contraction and can be directly affected by preload via the Frank-Starling response. Inotropic activity can also be altered by the internal cellular metabolic state as well as catecholamine concentration and β-adrenoceptor density and responsiveness. Contractility is often represented by LVESV and/or ejection fraction. Afterload is the mechanical load opposing ventricular ejection and has conventionally been represented as systemic arterial pressure in humans as this is the pressure the LV has to overcome to eject blood into the aorta. The accurate assessment of preload, contractility, and afterload is complex, not least because of their dynamic interrelationships.

In response to a bout of graded aerobic exercise involving repeated dynamic contractions of large muscle groups, Qc will increase linearly with oxygen uptake to a maximum oxygen consumption (VO2max). Ekblom et al (1968) reported that Qc can increase up to maximal values of ca 20-30 L·min⁻¹, or perhaps greater depending on body size and training status. Increases in both HR and SV contribute to the Qc response to graded submaximal exercise.² Because HR is largely under neural control, it will respond quickly to changes in metabolic demand during exercise. From rest, HR can rise 2- to 4-fold during exercise, which accounts for around 60% of the rise in Qc.² Heart rate increases in a linear fashion with oxygen consumption to the point where oxygen uptake reaches a peak or maximal value. Maximum HR (HRmax) is largely defined by age resulting in the common use of the standard equation: max HR = 220-age to predict HRmax. The primary mechanism that controls HR during exercise is the ANS. At the onset of exercise parasympathetic neural activity is reduced (vagal withdrawal) and this alone will result in an increase in HR to ca 100 beats·min⁻¹. Subsequently, sympathetic neural drive is increased and this will increase HR in line with exercise intensity. Another consequence of elevated sympathetic activity with exercise is an increase in circulating catecholamines, which also act to increase HR.

While SV does increase during graded (upright) exercise, there is some controversy as to whether SV plateaus at around 40%-60% VO2max or further increases to the point of near-maximal effort. The difference in response may relate to the subject’s training status, with athlete groups potentially demonstrating a linear increase in SV with exercise intensity through to maximum. The ability to increase SV with graded aerobic exercise relates to changes in preload and contractility that overcome the small, but significant, increases in systolic and mean arterial blood pressure (afterload) that occur in direct relation to exercise intensity.

With respect to preload, most evidence suggests that LVEDV is either increased or maintained during graded dynamic exercise, although this may be mediated by training status and posture. Preload increases despite the fact that as HR rises with exercise intensity, diastolic filling time decreases (ca 0.55 s at rest to ca 0.12 s at HRmax). Consequently, an increase (or maintenance) in LVEDV must come from an increased pressure gradient during LV filling. There is evidence that indices of LV filling pressure increase rapidly with exercise and continue to rise progressively with intensity. In a closed-loop system, this increase in pressure gradient is largely mediated via a rise in venous return as a consequence of increased activity of the muscle and respiratory pumps. The muscle pump, in association with unidirectional venous valves below the heart, drives blood flow back to the right atrium. An increase in venous smooth muscle tone and blood flow redistribution away from inactive areas assist venous return. The loss of the skeletal muscle pump when exercise ceases can contribute to postexerciseional collapse as central blood
volume and filling pressure reduces leading to a rapid drop in SV, Qc, and thus cerebral perfusion. In addition to increased venous return, there is evidence to suggest that an LV “suction” is augmented with exercise. As contractility of the LV increases with exercise, so relaxation will occur to a larger LVEDV, and thus greater rebound “suction” occurs. Myocardial tissue velocities during isovolumic relaxation and early filling increase in proportion with exercise intensity.

With respect to contractility, most studies have observed a progressive decrease in ESV during dynamic endurance exercise, which alongside a rise in afterload, is evidence of enhanced contractility. Increased contractility will occur as a consequence of the increased preload via the Frank-Starling mechanism as well as being mediated by the sympathetic nervous system innervating cardiomyocytes, likely after HR has increased beyond ca 100 beats·min⁻¹.

**Resistance and other modes of exercise**

The ability to maintain aerobic exercise performance is directly related to the ability of the cardiovascular system to deliver oxygen and nutrients to the working muscle. When exercise intensities are raised beyond VO₂max (supramaximal), performance capability is related to the intrinsic functional capacity of the skeletal muscle. The ability of the cardiovascular system to deliver oxygen and nutrients is not limiting, as performance per se is so short. Cardiovascular adjustments do occur during supramaximal exercise, but they are severely blunted and are more important for recovery.

Because of the short periods of muscle activity during, for example, resistance exercise, there is limited time for cardiovascular adjustment, thus HR is not raised significantly. At the same time, the high level of contractile force generated by skeletal muscle during high intensity exercise (including isometric muscle contractions over ca 40% maximal voluntary force) will increase the external pressure on blood vessels to the point that vessels are constricted and flow is reduced or stopped. This has multiple effects, but the most immediate is an increase in afterload or blood pressure due to an elevated peripheral resistance.

This was observed in high-intensity weightlifting where blood pressures peaked at 450/350 mm Hg in some individuals. An exemplar trace is shown in **Figure 1**. Such high pressures will be a substantial obstacle to the production of any SV, and thus Qc. Clearly a lack of Qc can only be maintained for a few seconds before impacting upon cardiac and cerebral perfusion. Once muscle contractions cease, a reactive hyperemia to the active muscle beds will ensue and HR and Qc will increase during the initial phase of recovery.

Exercise mode may also alter the cardiovascular responses to exercise. For example, the gravitational effects on the cardiovascular system during standing exercise (running) are different to seated exercise (cycling) and horizontal or supine exercise (swimming). Changes in posture will influence blood flow and neural activity. Supine exercise will elicit higher stroke volumes, because of enhanced preload, and thus lower heart rates. If dynamic exercise uses a relatively small muscle mass, such as when the arms (compared to the legs) are used in specific sports or athlete groups, then the cardiovascular system will face different hemodynamic loads. In arm exercise, peripheral resistance may be increased, due to a smaller muscle mass and capillary bed. This will decrease venous return and produce a greater increase in heart rate than during leg exercise at the same absolute work rate.  

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**Figure 1. The blood pressure response to high-intensity weightlifting.**

Note the substantial rise in both systolic (SBP) and diastolic (DBP) blood pressure as repetitions increase toward skeletal muscle failure.

The cardiac response to (ultra)endurance activity

The cardiovascular adjustment to short bouts of submaximal exercise is largely unremarkable. While acute bouts of exercise result in a transient elevation in risk of cardiovascular events, in those with an a priori increased risk profile with a 10-fold increase in the relative risk the impact on the healthy heart is considered benign (and positive when repeated and accumulated). Indeed, the American College of Sports Medicine and American Heart Association recommend that most adults should accumulate a minimum 30 min of moderate intensity exercise 5 days a week.1 This “dose” of physical activity is quite low when compared to that engaged in by (ultra)endurance athletes. Large volumes of training-related exercise, in (ultra)endurance athletes, are combined with competitive events that may involve continuous exercise for up to 24 hours or longer. The consequences for cardiovascular health of adopting these high “doses” is controversial and currently the subject of scientific debate. It is important to detail whether such “doses” increase cardiovascular health gain, saturate the health gain, or actually represent an “overdose” where negative health consequences become apparent.6,7

Cardiac functional responses to (ultra)endurance activity

Humans have been engaging in (ultra)endurance activities for thousands of years and folklore often recounts tales of extreme endurance (often with apparent negative health consequences such as the legend of Pheidippides). The scientific evaluation of (ultra)endurance activity did not really begin until the 20th century. A landmark study was performed in Sweden in the 1960s that reported an apparent reduction in intrinsic cardiac contractile function toward the end of a long bout of running and cycling.8 A drop in SV occurred toward the end of steady state exercise, even though blood volume and thus preload were maintained. This suggested that “cardiac fatigue” had occurred, and ran contrary to popular and contemporary scientific dogma that the heart, unlike skeletal muscle, did not fatigue with the imposition of exercise.

The concept of “cardiac fatigue” was reignited in the 1980s as work moved from the controlled laboratory environment to the burgeoning list of competitive events for ultra-endurance enthusiasts. Firstly, Niemela et al (1984) reported a decline in LV contractile performance toward the end of a 24-hour run.9 Consequently, Douglas et al (1987) reported a similar decrease in LV contractility after an Ironman Triathlon (ca 12 to 16 hours of continuous exercise).10 Of note, Douglas et al (1987) also reported a decline in the early atrial peak transmitral filling velocity ratio post-race. This Doppler-derived parameter provides a global index of diastolic filling and thus the authors concluded that prolonged exercise may transiently decrease both LV systolic and diastolic function. Controversially, another study from the same period that assessed athletes before and after a marathon (<6 hours) observed no evidence of cardiac fatigue.11 An instant comparison of data at that time might suggest that there is a specific threshold of exercise duration (or volume) beyond which cardiac fatigue was more clearly manifest however, subsequent studies have been equivocal in this “duration-dependency” concept.

These initial studies led to a considerable volume of original research data in multiple sports, subject groups and environments. The findings from these individual studies, often with small sample sizes and thus low power, have been eloquently reviewed12 and combined in a meta-analysis.13 Middleton’s meta-analysis produced a significant, and negative, overall effect of (ultra)endurance exercise on ejection fraction and the ratio of early-to–atrial peak diastolic filling velocities. While the decline in systolic function was seemingly mediated by increasing exercise duration and poor training status, the decline in diastolic function was consistent between studies of widely differing durations (marathon up to 24-hour races). The consensus from reviews and meta-analysis is that significant evidence exists of a small and transient decline in LV systolic and diastolic function after prolonged intense exercise.

Although a consensus of sorts is materializing, many controversies remain: (i) not all studies have reported changes in LV function post-exercise.11 Whether this reflects variance in exercise dose, subject characteristics, and/or environmental conditions has not been resolved fully, (ii) most studies have assessed athletes in field-based environments before and after competitive races where experimental control of the exercise dose, pace, nutrition, fluid balance, and ambient conditions are limited. Some groups have moved their research into a controlled laboratory environment and such studies have rarely observed in-event or post-event changes in LV function, however, laboratory-based studies are themselves limited by their ability to replicate the real and often unremitting nature of actual competitive events; (iii) the assessment of LV function is complex and in human studies is complemented noninvasively.
Noninvasive assessment results in estimation of functional indices via indirect measurement tools. The most common tool employed, echocardiography, is associated with significant error if scans and sonographers are not of the highest quality. The cardiac fatigue literature has developed over the last 15 years as echocardiography and other noninvasive imaging tools have undergone significant changes in capability. The major changes have been toward the ability to assess, quantitatively, regional cardiac wall movement (either in terms of velocity or change in length). Consequently, tissue-Doppler and myocardial speckle tracking techniques have been employed to provide additional evidence related to changes in cardiac function after exercise. George et al (2005) reported that early diastolic tissue velocities were reduced after running a marathon in six different LV wall segments.14 Likewise, Neilan et al (2006) reported decreased early diastolic septal and lateral wall tissue velocities after running the Boston Marathon.15 Intriguingly, this was the first LV functional dataset that did not normalize within a 24- to 48-hour recovery with a small residual, but statistically significant, decline at a 4-week follow-up. In addition, these authors reported a post-race reduction in tissue-Doppler derived myocardial strain in both septal and lateral walls. Other groups have adopted a speckle tracking approach to tissue deformation assessment as this negates the angle-dependency of tissue-Doppler. Notably, La Gerche et al (2008) reported reduced longitudinal strain and strain rate in athletes with wall motion abnormalities.16 George et al (2009) expanded the analysis and observed reduced radial and circumferential strain post-exercise as well as region-specific decline in LV deformation in a runner who had completed a 90-km run (Figure 2).17

The recent development of myocardial speckle tracking has allowed the assessment LV function in multiple planes of motion (radial, circumferential, and longitudinal) that more completely reflect the reality of LV movement during contraction and relaxation. As well as technical developments in echocardiography, some studies have used magnetic resonance imaging (MRI) to report reduced cardiac function after acute exercise.18 This technique offers greater spatial resolution as well as the ability to assess parameters such as the presence of interstitial fibrosis via late gadolinium enhancement scans; and (iv) finally, a substantially smaller body of evidence has reported significant changes in right ventricular (RV) function after prolonged exercise using traditional 2-D echocardiograms, tissue velocities,16 myocardial speckle tracking,19 and MRI.18 La Gerche et al (2008) reported an immediate reduction in RV tissue velocities that remained depressed at 1 week in one athlete.16 Given that the exercise-induced increase in wall stress is relatively greater in the RV than the LV it has been speculated that the RV is more prone to “exercise-induced cardiac fatigue.”16

The impact of “exercise-induced cardiac fatigue” on exercise performance remains unknown. Concomitant performance data suggest that any performance impact is likely negligible. There has been no link between cardiac fatigue and cardiovascular drift, and when race pace data are analyzed, there is no clear indication of a drop in pace toward race completion that is in any
way commensurate with cardiac functional depression in recovery. The concept that exercise-induced cardiac fatigue induces LV or RV functional changes that approach or have clinical significance is not supported by most current data, but again this remains controversial. Most changes in LV and/or RV function are small and transitory with normal function restored within 48 hours, although occasional reports have suggested that function changes persist for much longer.

**Biomarkers of cardiac damage and (ultra)endurance activity**

The scientific debate over the clinical significance of “exercise-induced cardiac fatigue” has prompted discussion with respect to mechanism(s) that underpin LV and RV functional changes after prolonged exercise. Identification of underlying mechanism(s) will assist in the differentiation of physiological vs pathological substrate as the backdrop to functional changes.

One popular theory is that changes in cardiac function are linked to cardiomyocyte damage induced by prolonged exercise. Measurements of a range of different cardiac biomarkers of cellular damage have been widely reported and reviewed in the scientific literature. In early studies, the clinical biomarker of choice was creatine kinase MB (CKMB). The interpretation of creatine CKMB changes post-exercise is problematic given the poor cardiac tissue specificity and the cross-reactivity with skeletal muscle damage that is common during prolonged exercise. The adoption of cardiac troponin I or T (cTnI/cTnT) as cardiomyocyte-specific biomarkers of cellular damage has significantly enhanced clinical practice and has also led to numerous studies reporting elevated cTnT or cTnI after prolonged exercise. In a meta-analysis, the overall event rate (positive serum sample for cTnT after prolonged exercise) was 47%. Numerous studies have since tried to attribute a positive cTnT response after prolonged exercise to a range of exercise or subject-related parameters. Care is warranted in this approach however, as individual studies and meta-analyses that look at a single blood draw post-exercise may underestimate the true rate of cTn appearance after (ultra)endurance events. A unique laboratory-based marathon with blood draws every 30 min during the run and at frequent intervals during recovery observed elevated cTnI in all runners (Figure 3).

Ongoing developments with clinical assays may also alter the interpretation of exercise-related cTn appearance. New high-sensitivity cTn assays have a much lower detection limit and thus extremely low levels of circulating cTn could now be detected. Clearly this has the potential to lead to “positive” serum cTn data before races (possibly as a consequence of recent training) as well as increasing cTn-positive samples post-race (even if only one sample is employed).

**Figure 3.**

The appearance of cTnT in the bloodstream of runners completing a marathon run on a treadmill. Note the high frequency of sampling during running and recovery. In all athletes troponin T (cTnT) is increased during exercise and in all but one a second peak of cTnT is witnessed during recovery.

The clinical relevance of postexercise cTn appearance, and whether an elevated cTn after endurance activity explains changes in LV and RV function, is controversial. Clinical chemists have described that there are no membrane transporters for cTn, and thus what appears in the serum or plasma must have originated in the cell and have moved there due to some “disruption” to the cardiomyocytes. Despite this, absolute data for cTn release is generally very small. The appearance of cTnT can be very rapid, but importantly the clearance may be complete inside 24 hours, which is different to the reported appearance after an acute myocardial infarction. In a recent study of high-intensity exercise, Shave et al (2010) observed elevated serum cTnI after just 30 min of exercise in 6 out of 8 subjects. This has led some to suggest that cTn release may reflect normal physiological or adaptive processes, possibly reflecting “leakage” of unbound cTn from the cytosol as cardiomyocyte membrane permeability alters with acute exercise. Some individual studies have reported no association between elevated cTn and reduced LV function after prolonged exercise, while others have linked the appearance of cTn with altered cardiac function, notably in the RV. Overall consensus at this stage would suggest that there is no clear evidence for linking cTn elevation and functional change in the LV after prolonged exercise.

The clinical relevance of cTn appearance, in and of itself, still requires further study as we have no definitive answer as to whether postexercise cTn elevation represents reversible or minor irreversible cardiomyocyte damage. Recently, Shave and colleagues presented a diagnostic algorithm to assist in the differential diagnosis of physiologically induced elevations in cTn and pathological forms, i.e., ACS (Figure 4).

![Figure 4](image-url)
In summary, with respect to the cardiovascular response to prolonged exercise, there is now ample evidence that the completion of (ultra)endurance exercise can lead to a small and transient reduction in LV and RV function. Often temporally associated (but with limited evidence of cause and effect) with these functional changes is the observation of the appearance of cTn in the blood. It is likely that in most people these events are a normal, and potentially adaptive, process associated with the “stress” of prolonged exercise. Continued research activities are still required to determine if the RV is more susceptible to “cardiac fatigue” with prolonged exercise, whether these changes are associated with tissue damage and what the long term consequences of such maladaptation might be.

**Conclusion—acute exercise and the heart**

The acute cardiac responses to exercise in humans are a fascinating and complex topic. They have implications across the human spectrum from elite performance to exercise for cardiovascular health and rehabilitation. We have substantial data that describe, largely using noninvasive tools, the changes in major cardiovascular variables in response to different modes of exercise. The heart is hugely adaptive and copes with most acute exercise bouts by increasing $Q_o$ and thus oxygen delivery to active tissues. The cardiac response to resistance exercise is dominated by a significant increase in afterload, but such exercise persists for very short periods, enabling the heart to cope effectively with this often dramatic hemodynamic challenge. We are just beginning to detail the acute cardiovascular consequences of (ultra)endurance exercise bouts and further work is required to assess the potential health consequences of such activities. It is entirely likely that for the vast majority of endurance athletes this activity leads to purely physiological adaptation in the cardiovascular system. For some, however, there is initial evidence that endurance exercise may be part of a pathophysiological cascade. The exact numbers of endurance athletes at risk is likely small, and identifying these individuals will be very challenging.

**CARDIOVASCULAR RESPONSE TO EXERCISE TRAINING IN THE YOUNG**

**The athlete’s heart (AH)**

The adaptation of the heart to physical training has been the focus of study for clinicians and scientists since the late 19th century. It is now widely accepted that regular participation in intense sporting activity is associated with physiological, electrical, structural, and functional cardiac modifications that frequently manifest on the ECG, echocardiogram, and exercise stress test. Combined, these physiological changes have been termed the athlete’s heart (AH).

**Heart rate and exercise training in young individuals**

One of the most common findings in AH is a resting sinus bradycardia ($HR < 60 \text{ beats} \cdot \text{min}^{-1}$) with a greater prevalence in aerobically trained individuals compared with sprint/strength-trained athletes. While increases in left ventricular cavity size and resultant resting stroke volume ($SV_{rest}$) and cardiac output have been muted as potential mediators of this observed bradycardia, the mechanism is more likely associated with an increased vagal tone.

In line with resting bradycardia, a reduction in maximum heart rate ($HR_{max}$) of ca. 5 beats $\cdot$ min$^{-1}$ has been demonstrated in highly trained athletes (Figure 5, page 16). The mechanisms underlying the lower $HR_{max}$ values in elite athletes are unclear. It has been suggested that the reduced $HR_{max}$ observed in elite endurance athletes may be associated with an increased $SV_{rest}$ and maximal SV ($SV_{max}$). However, limited support is offered for this hypothesis in the literature with similar reductions in $HR_{max}$ noted in endurance and sprint/power-trained athletes despite larger $SV_{rest}$ and $SV_{max}$ in the former. Others have suggested that an enhanced distribution of blood to the working muscle during intense exercise may lead to a more efficient redistribution of cardiac output and result in a decreased chronotropic drive in highly trained individuals. A desensitization (downregulation and uncoupling) of cardiac β-adrenoceptors, possibly associated with an altered β-adrenoceptor density, has been suggested as a possible mechanism in the observed reduction in $HR_{max}$ in elite athletes. While limited data exist examining β-adrenoceptor function in athletes, recent studies examining highly trained athletes have reported a reduced cardiac function following prolonged ultra-endurance exercise associated with a β-adrenoceptor downregulation. Hart et al (2006) reported decreased chronotropic responsiveness to isoproterenol infusion following a 4-hour rowing exercise bout, suggesting that prolonged exercise resulting in a lengthy exposure to elevated catecholamines resulted in a downregulation of β-adrenoceptors (Figure 6, page 16). The impact of chronic training and competition upon β-adrenoceptor function in elite athletes remains unclear. Furthermore, the impact of sprint/power training on β-adrenoceptor function warrants further investigation.
**Figure 5. Maximum heart rate vs age.**
(A) For athletes (combined male and female, aerobic and sprint/power) and sedentary participants (combined male and female). (B) For male and female athletes (combined aerobic and sprint/power).


**Figure 6. Left ventricular response to incremental isoproterenol infusion pre- and post-exercise.**
Inotropic (A), lusitropic (B), and chronotropic/loading (C) response to isoproterenol pre- (continuous line) and post-exercise (dashed line). Data are means±SD. Different from pre-exercise *P < 0.05, **P < 0.01.

Abbreviations: A, peak velocity of late left ventricular filling; E, peak velocity of early left ventricular filling; E/A, ratio of the two preceding parameters; EF, ejection fraction; HR, heart rate; LVIDd, left ventricular internal diameter during diastole; LVMWS, left ventricular meridional wall stress; MAP, mean arterial pressure; Q, cardiac output; SBP/ESV, systolic blood pressure/end-systolic volume; SV, stroke volume; Vp, flow propagation velocity.

Recently, it has been suggested that a reduction in HR max following chronic training may be the result of a “Central Governor” acting as a cardioprotective mechanism at maximal exercise intensities. The concept that a Central Governor may control cardiac chronotropic drive at maximal exercise intensities in highly conditioned athletes is attractive. The central fatigue model suggests that the reduction in power output at exhaustion is related to an altered efferent command from the brain. It is postulated that alterations in neural drive originate in the brain in response to somatosensory input from afferent fibers from peripheral organs including the heart.

**Electrocardiogram (ECG) and exercise training in young individuals**

Regular physical training is associated with a number of ECG anomalies that are associated with physiological adaptations of the heart. These commonly observed changes occur in up to 80% of athletes and include: sinus bradycardia, first-degree atrioventricular (AV) block, notched QRS in V1 or incomplete right bundle branch block, early repolarization, and isolated QRS voltage criteria for left ventricular hypertrophy (LVH) (Figure 7). The prevalence of these physiological changes is not dissimilar between junior and senior athletes. In contrast, uncommon ECG changes that are present in fewer than 5% of athletes include: T-wave inversions, ST-segment depression, pathological Q-waves, left atrial enlargement, left axis deviation/ left posterior hemiblock, right ventricular hypertrophy, complete left or right bundle branch block, long or short QT interval, Brugada-like early repolarization, and ventricular arrhythmias.

The vast majority of previous ECG studies have examined white athletes. A small number of recent studies have examined athletes from different ethnic backgrounds and demonstrate significant ethnicity-specific changes in the resting ECG with black athletes presenting more striking repolarization alterations than white athletes matched for age, body size, and sports participation. Particular interest has focused on deep T-wave inversions (>0.2 mV) given their recognized association with hypertrophic cardiomyopathy (HCM) and arrhythmogenic right ventricular cardiomyopathy (ARVC) (two of the leading causes of SCD) in athletes. In contrast to white athletes, deep T-wave inversions in leads V1 to V4 were noted in 7% of Afro-Caribbean black male athletes in the absence of apical HCM, marked hypertrophy of the anterolateral wall, myocardial fibrosis, or characteristic right ventricular morphology–associated arrhythmogenic right ventricular cardiomyopathy. Of note, black female athletes presented with similar findings with a higher prevalence of ST-segment elevation, contiguous T-wave inversions, and deep T-wave inversions in leads V1 to V3 (6 female black athletes) compared with white athletes. A recent study reported a significantly greater prevalence of lateral T-wave inversions (V4-V6) in black athletes compared with both West-Asian (Middle-Eastern) and white athletes. In contrast, the rate of “uncommon” ECG changes between West-Asian and white athletes was comparable.

In the absence of obvious pathology, deep T-wave inversions in leads V1 to V4 in black athletes probably represent a normal spectrum of ECG changes in re-
response to physical training (Figure 8). Alterations in autonomic cardiac innervation, either reduced sympathetic or increased vagal tone or recently identified sodium channel polymorphisms among the black population may provide partial explanation for the variation found within black athletes. However, more detailed molecular assessment and longitudinal follow-up of black athletes is required to definitively differentiate a physiological from pathological substrate.

Figure 8. Pie charts comparing ECG anomalies between black athletes and white athletes. Black athletes exhibited a higher prevalence of ST-segment elevation and T-wave inversions than white athletes. Abbreviations: Inv T, T-wave inversion; LAE, voltage criterion for left atrial enlargement; LVH volt, voltage criterion for left ventricular hypertrophy; RAE, voltage criterion for right atrial enlargement; ST Elev, ST-segment elevation. After reference 32: Rawlins et al. Circulation, 2010; 121:1076-1085. © 2010, American Heart Association, Inc.

Figure 9. Distribution of cardiac dimensions in large populations of highly trained male and female athletes. Top: left ventricular end-diastolic cavity dimension; 14% of athletes have enlargement of 60 to 70 mm. Middle: left atrial transverse dimension; 20% of athletes have transverse left atrial dimension ≥40 mm. Bottom: maximum left ventricular (Max. LV) wall thickness; 2% of men and 0% of women have wall thickness 13 mm.

Cardiac structure and exercise training in young individuals

The development and use of echocardiography in the 1970s allowed a more detailed interrogation of cardiac structure and function. Following the suggestion of differential cardiac remodeling in response to different types of physical training,34 a large number of cross-sectional studies examining small numbers of athletes from a wide range of sports have been published, reviewed, and subjected to meta-analysis. While the majority of previous echocardiographic studies have examined small cohorts of athletes, a landmark paper examining 947 elite Italian athletes from 25 sports was published by Pelliccia et al (2002) (Figure 9).35,36

Results from their study suggested that a left ventricular wall thickness >13 mm was unusual and predominantly confined to athletes participating in rowing, canoeing, and cycling. Furthermore, the upper limit of physiological hypertrophy of ventricular walls and internal diameter was reported as 16 mm and 66 mm, respectively. Consequently, these values have been widely adopted as the upper limits of physiological left ventricular enlargement. A more recent study using superior echocardiography techniques corroborated these findings in 442 athletes with an upper limit for left ventricular wall thickness of 15 mm and internal cavity dimension of 66 mm in male athletes. It is noteworthy that while female athletes display a significantly smaller left ventricular wall thickness upper limit of 12 mm, internal cavity dimensions of up to 66 mm are observed, if somewhat less frequently.27 The majority of available data are, however, derived from predominantly white athletes. Recent studies have demonstrated a larger left ventricular wall thickness in Afro-Caribbean black male athletes with 20% of black athletes exhibiting LVH (>12 mm) compared with just 4% of white athletes (Figure 10).31 Furthermore, a significant minority (3%) of black athletes (but none of the white athletes) had substantial LVH (>15 mm), which could have been consistent with morphologically mild HCM. These results are striking compared with the Italian Olympian athletes, in which only 0.08% of 738 men exhibited LVH >15 mm.35 In line with data from black male athletes, Afro-Caribbean black female athletes demonstrate significantly larger left ventricular wall thickness with up to 3% of athletes exhibiting a wall thickness >11 mm (12 to 13 mm) compared with an absence of white female athletes presenting with a wall thickness >11 mm.32 Thus, in line with ECG changes, it would appear that a racial predilection exists in the development of LVH in response to the increased pre- and afterload associated with intensive exercise. A combination of genetic, endocrine, and hemodynamic factors likely accounts for the increased LVH in black athletes. Despite the increasing evidence that an athlete’s ethnic origin may have a significant impact on the cardiovascular response to exercise, there are limited data in other ethnic populations, indicating the need for further study.

Care is warranted in the interpretation of cardiac enlargement data from elite athlete populations given the potential that the substantial LVH identified in some athletes could have been attributed to the possible use of performance-enhancing drugs. The vast majority of studies in this field are often constrained on professional, ethical, and financial grounds from evaluating drug use. However, a preponderance of athletes compete at the national level or above and are subject to antidoping procedures.
Cardiac function and exercise training in young individuals

The most commonly observed change in cardiac function with training (predominantly endurance-type training) is an increase in SV at rest, at absolute submaximal workloads, and at maximal exercise. This, in part, underpins the increase in maximal cardiac output and thus maximal oxygen uptake (VO\textsubscript{2max}) often observed in trained individuals.

At rest and during exercise, SV is mediated by HR, preload, contractility, and afterload. A higher SV\textsubscript{rest} has been attributed to a reduction in HR\textsubscript{rest} (allowing a longer diastolic filling period), an increase in blood volume, and remodeling of the LV cavity. There are no convincing data that LV contractility at rest is greater in athletes or changes with training. Likewise, only modest or no changes in resting arterial blood pressure have been reported with training, suggesting a limited role for afterload in modulating SV in trained individuals.\textsuperscript{37}

During exercise in untrained subjects, SV plateaus at 40\% to 50\% of maximal aerobic power (VO\textsubscript{2max}). However, in athletes, the rise in SV may continue beyond 40\% to 50\% VO\textsubscript{2max} associated with an increased end-diastolic volume (EDV). Data in humans indicate that EDV increases with exercise training, both at rest and during exercise. The increase in EDV during exercise is associated with enhanced venous return and filling pressure, in concert with increased ventricular suction. The latter occurs as a consequence of enhanced ventricular diastolic rebound to a larger EDV from a Frank-Starling-mediated lower starting ESV. It has long been believed that blood volume alterations with training partially underpin the enhanced CV performance capacity of athletes or sedentary people after training. Blood volume measures are difficult to obtain, but enhancement of blood volume could increase preload during exercise by increasing central blood volume and thus filling pressure. The impact of training on ventricular compliance has often been assessed using transmitral flow velocities or tissue velocities during early diastole with inconclusive data related to training changes. Other studies (using rapid infusion of saline and lower body negative pressure to modulate ventricular filling pressure and measure the resultant SV for any given pulmonary artery wedge pressure) have concluded that endurance training improves ventricular compliance. Ventricular compliance might increase as a result of training because of enhanced ventricular suction, intrinsic myocardial changes that occur in concert with ventricular remodeling, or decreased LV wall stiffness.

With respect to afterload, a reduced blood pressure at submaximal exercise may reduce afterload, but maximal arterial blood pressure is largely unchanged by training. Furthermore, higher LV end-systolic volume, and thus lower ejection fraction at all matched submaximal HR levels following training, has been reported. This may be due to reduced catecholamine concentrations, muscle sympathetic nerve activity (MSNA), and/or decline in \(\beta\)-adrenoceptor density with training.

Differentiation of physiologic and pathologic changes of the heart

Sudden cardiac death in sport

The physiological adaptations of the heart to chronic physical training may mimic those changes observed in diseased states. Accordingly, the identification of upper normal limits of cardiac enlargement and physiologically induced ECG anomalies is important in the differentiation of physiological and pathological cardiac remodeling. This is of particular importance in the context of exercise-related SCD in young athletes with underlying cardiac pathology.

The sudden death of an athlete is a rare event, with an incidence ranging between 1.50 000\textsuperscript{38} to 1.200 000\textsuperscript{39} a year. Despite their rarity, such events are highly publicized and have a substantial emotional impact on the community at large when one considers that athletes are perceived as the healthiest segment of society. Over 80\% of nontraumatically-related deaths are attributable to cardiac disorders.\textsuperscript{38} The majority of cases of SCD in young athletes (<35 years) are due to hereditary or congenital cardiac anomalies including heart muscle diseases (ie, hypertrophic cardiomyopathy, HCM; arrhythmogenic hypertrophic cardiomyopathy, ARVC); cardiac conduction tissue disorders (ie, ion channelopathies, long QT syndrome and Brugada’s syndrome; accessory pathways, Wolff-Parkinson-White syndrome); and structural abnormalities (ie, coronary artery anomalies, coronary artery aneurysm). HCM is reported to be the commonest cause of death in young athletes worldwide. In contrast, the vast majority of SCDs in older athletes are secondary to atherosclerotic coronary artery disease. Of note, idiopathic LVH (ILVH) is common among those that die suddenly during sport, accounting for up to one third of SCD cases.\textsuperscript{40} It is unclear at this stage whether ILVH represents a spectrum of the HCM phenotype, an exaggeration of the physiological response in a person who may have succumbed to a fatal ventricular disorder owing to an undetectable ion channel disorder or congenital accessory pathway resulting in pathological LVH, or whether it is indeed
likely to be discovered from spontaneous symptoms. Most datasets examining SCD in athletes are derived from American and Italian studies; however, a small number of studies exist in other populations (e.g., UK) supporting the data from the USA and Italy. 

**Pre-participation cardiovascular screening**

Sudden death is often the first clinical manifestation of underlying heart disease in young athletes, which has led to a prolonged, and at times acrimonious, debate surrounding the value of pre-participation cardiovascular screening in athletes. While there is agreement between the North Americans (American Heart Association, AHA) and the Europeans (European Society of Cardiology, ESC) regarding the importance of pre-participation cardiovascular screening, disagreement exists as to the nature of such screening. The AHA, based on limited empirical evidence, contends that pre-participation cardiovascular screening with questionnaire and physical exam alone is sufficient in the identification of underlying cardiovascular disease. In contrast, a number of other groups (including North Americans) have demonstrated that cardiovascular screening including ECG testing is associated with a significantly higher diagnostic yield than reliance on history alone. In support of the inclusion of 12-lead ECG, a large number of studies have demonstrated the presence of abnormal ECG findings in the majority of diseases causing SCD in athletes. It is well established that over 90% of people with HCM have an abnormal ECG, and with similar findings in ARVC combined with a high yield from antemortem 12-lead ECG in the identification of ion channel disorders and accessory pathways, there appears to be a clear rationale for the inclusion of the 12-lead ECG in pre-participation cardiovascular screening.

The Italian, and now ESC, model of pre-participation cardiovascular screening in young athletes, using 12-lead ECG as a screening tool, has been shown to be effective in reducing SCD by identifying athletes with underlying cardiomyopathy and ion channelopathies. Further support for the Italian model of pre-participation screening has been provided by other groups suggesting that the absence of antecedent cardiac symptoms and/or a family history of cardiac disease and/or SCD in almost 80% of cases of SCD confirms the prior observation that most cardiovascular disorders responsible for SCD in the athlete are clinically silent and unlikely to be discovered from spontaneous symptoms. The Italian screening program in athletes has been successful in identifying and preventing deaths predominantly from the cardiomyopathies through subsequent disqualification of the affected individual from sporting activities of moderate to high intensity to minimize the risk of SCD. In this regard, it is prudent to highlight data suggesting that up to one fifth of all SCDs in athletes occur at rest, suggesting that the identification of cardiovascular diseases and subsequent disqualification from sport may not prevent deaths in all athletes harboring potentially fatal cardiac disorders.

While the debate surrounding the optimal method for pre-participation cardiovascular screening continues, a large number of international governing bodies of sport (e.g., International Olympic Committee [IOC], Union of European Football Associations [UEFA]) have adopted the ESC model (Figure 11, page 22) and currently require or recommend athletes to undergo pre-participation cardiovascular screening including 12-lead ECG. Implementation of pre-participation screening is hampered in most countries by lack of expertise, resources, and infrastructure. Concerns are also justified that further research is required on the efficacy and cost-effectiveness of pre-participation screening. The studies from Italy, however, provide the best available evidence to date, supporting the implementation of systematic pre-participation screening for the prevention of sudden cardiac death in young athletes. Although such programs are effective at minimizing deaths from cardiomyopathies and cardiac conduction tissue disorders, they have no impact on deaths from coronary anomalies or premature coronary atherosclerosis.

**Conclusions—training and the heart in young individuals**

Chronic exercise training results in a range of physiological adaptations of the heart that are collectively termed the “athlete’s heart” (AH). Commonly observed adaptations include cardiac enlargement associated with increased LV and RV volumes, which, in the presence of unaltered diastolic function, support an increased SV (and therefore Q̇) at rest and during exercise. Concomitant to changes in ventricular volumes is a small, but “balanced,” increase in ventricular wall thickness (predominantly in endurance-trained athletes). Alongside these structural and functional changes a number of “common” conduction anomalies are observed on the resting ECG. In the vast majority of athletes, these changes represent a physiological response to chronic exercise and are associated with enhanced cardiac performance which, in part, supports improved athletic performance. In some, however, AH may mimic diseases associated with sudden cardiac death in
young athletes. Accordingly, the need to identify at-risk athletes has led to the establishment of pre-participation screening programs and their adoption by international bodies of sport globally. Despite a general consensus for the need for pre-participation screening, debate continues regarding the appropriate screening tools, efficacy, and cost-effectiveness of such programs.

**CARDIOVASCULAR RESPONSE TO EXERCISE TRAINING IN OLDER INDIVIDUALS**

**Cardiac function and exercise training in older individuals**

A well-recognized effect of increasing age following maturation is a decline in maximum aerobic capacity (VO$_{2\text{max}}$) likely as a consequence of a decline in maximum pulmonary ventilation (VE$_{\text{max}}$), heart rate (HR$_{\text{max}}$), stroke volume (SV$_{\text{max}}$), cardiac output (Q$_{\text{max}}$), and peripheral oxygen extraction (A-VO$_{2\text{diff max}}$). The impact of sex and training status on the age-related decline of these variables remains an area of much debate. The impact of training status on age-related reductions in the functional capacity of the cardiovascular system has been extensively reported. The majority of studies suggest that despite a greater absolute decline in VO$_{2\text{max}}$ in trained versus untrained individuals the relative reduction in VO$_{2\text{max}}$ is similar between groups. The greater absolute reduction in VO$_{2\text{max}}$ in trained populations is associated with a higher initial VO$_{2\text{max}}$ and an overall reduction in training volume with increasing age. It has been suggested that the rate of age-related decline in VO$_{2\text{max}}$ is greater in trained female compared with trained male subjects. However, this sex-related difference likely represents a greater decrease in total training volume in females. While debate surrounding the reduction in VO$_{2\text{max}}$ still exists, the majority of studies agree that the age-related decrease in HR$_{\text{max}}$ is similar between sexes and is unaffected by training status. Early studies reported an age-related linear reduction in HR$_{\text{max}}$ of 10 beats·min$^{-1}$·decade$^{-1}$. In contrast, recent longitudinal studies suggest an age-related reduction of 5 to 7 beats·min$^{-1}$·decade$^{-1}$ independent of training status, particularly in groups <55 years of age. The majority of these studies, however, have examined non-elite populations with wide age ranges. Limited data exist examining the age-related decline in HR$_{\text{max}}$ in highly trained athletes. The mechanisms responsible for the age-related reduction in HR$_{\text{max}}$ remains an area of contention, however it is likely due to the withdrawal of cardiac parasympathetic tone and diminished β-adrenergic responsiveness. This weakened β-adrenergic responsiveness in older sedentary individuals appears to contribute to an attenuated LV contractile response to exercise, regardless of a larger β-adrenergic...
stroke volumes, together with significantly reduced LV mass compared to age-matched controls, veteran athletes demonstrate a small LV mass, compared with age-matched sedentary controls. Accordingly, exercise training into old age may maintain ventricular compliance, which, during exercise, could explain an augmented SV and thus elevated VO\textsubscript{2}max compared with age-matched sedentary subjects.

**Cardiac structure and exercise training in older individuals**

In the absence of hypertension or clinically apparent cardiovascular disease, left ventricular wall thickness increases progressively with age in both sexes. At the subcellular level, aging is associated with changes in excitation–contraction coupling mechanisms and diminished β-adrenergic contractile response. At the cellular level, cardiac aging is characterized by a significant reduction in cardiomyocyte number with hypertrophy of remaining cells and an increase in interstitial tissue.

Few studies have reported LV and RV volumes of current endurance veteran athletes (>50 years) compared with young athletes; however, it is generally believed that veteran endurance athletes demonstrate a smaller LV and RV end-diastolic and end-systolic volumes, with maintenance of LA end-diastolic volume, LV and RV systolic function, and LV mass, compared with young endurance athletes. Compared with veteran (age-matched) controls, veteran athletes demonstrate a greater LV wall thickness, absolute and indexed LV and RV end-diastolic and systolic volumes, and LV and RV stroke volumes, together with significantly reduced LV and RV ejection fractions.47

Long-term high-intensity endurance exercise is associated with changes in cardiac morphology together with electrocardiographic alterations that are believed to be physiologic in nature.48 It has recently been postulated, however, that these seemingly physiological phenomena in response to chronic exercise may be associated with pathological alteration of the heart. A number of studies have reported an incomplete reversal of LVH in retiring elite athletes suggesting, in part, a pathological remodeling process.49 In support of these structural findings, recent studies have documented an increased prevalence of supraventricular, complex ventricular, and profound bradyarrhythmias in endurance-trained athletes, predominantly occurring in veteran athletes.50 In a recent study, Claessen et al (2011) reported that a significantly high proportion of athletes presenting for atrial flutter ablation surgery were regular sportsmen, and concluded that a history of endurance sports and subsequent left atrial remodeling may be a risk factor for the development of atrial flutter.50 In a case series of more problematic arrhythmias, Heidbuchel et al (2003) reported on 46 endurance athletes (mainly cyclists) with symptomatic arrhythmias that were largely of RV origin.51 In a 5-year follow-up, there were 9 sudden cardiac deaths and in the absence of cardiovascular disease the authors speculated that for some athletes endurance training may result in the development and/or progression of an underlying arrhythmogenic substrate.52 In a follow-up study from the same group, Ector et al (2007) reported a significantly reduced RV ejection fraction in endurance athletes with ventricular arrhythmias, concluding that endurance exercise not only acts as a trigger for arrhythmias, but may also be a promoter of these RV changes.53 This group has speculated that there may be a link between the acute effects of prolonged exercise on RV function and these long-term issues, coined the term “exercise-induced right ventricular cardiomyopathy.”

Myocardial fibrosis is a possible substrate for arrhythmia, and two recent studies of gross heart structure in lifelong athletes have produced evidence of an increased prevalence of interstitial fibrosis in endurance athletes. Initially, Breuckmann et al (2009) reported on 102 older marathon runners with a recent history of race completion compared with age-matched controls. Late gadolinium enhancement on MRI was present in 12 runners and 4 controls with 7 of the athletes presenting a non–coronary artery disease pattern. These data were then confirmed in a smaller cohort of truly lifelong elite (ultra)endurance athletes where late gadolinium enhancement was reported in 6 of 12 athletes compared with an absence of late gadolinium enhance-
ent in age-matched controls or younger (ultra)endurance athletes. Again the pattern of fibrosis in 5/6 patients was non–coronary artery disease in origin (Figure 12).\(^{47,54}\) Both studies could only speculate at the cause of such fibrosis patterning and no long-term follow-up has been completed to determine what, if any, clinical consequence these findings may have.

![Figure 12. SA (short axis) cine stack on the top. Late gadolinium enhancement images on the bottom row (red arrow indicates mid-wall enhancement indicative of myocarditic scarring).](image)


The etiology and clinical significance of these structural and electrical changes remain to be fully elucidated; however, the preponderance of deaths associated with idiopathic LVH and idiopathic myocardial fibrosis does raise the possibility that some deaths in sport may be secondary to acquired myocardial disorders resulting from the long-term effects of intensive exercise.\(^{40}\)

**Conclusions**—training and the heart in veteran athletes

Increasing age following maturity results in a decline in VO\(_{2}\)\(_{\text{max}}\) associated with a reduction in VE\(_{\text{max}}\), HR\(_{\text{max}}\), SV\(_{\text{max}}\), O\(_{\text{max}}\), and A-V O\(_{2}\)\(_{\text{diff}}\)\(_{\text{max}}\). While exercise training in older individuals does not eliminate this age-related decline, it does appear to reduce the rate of decline in VO\(_{2}\)\(_{\text{max}}\). Along with improvements in diastolic function, exercise training results in larger LV and RV cavities (and wall thickness), which result in an increased SV at rest and during exercise compared with age-matched controls. The impact of lifelong exercise training on the heart is less well studied. However, recent studies have suggested a potentially deleterious effect of (ultra)endurance exercise. These studies have suggested a greater prevalence of simple and complex bradyarrhythmias and tachyarrhythmias in some lifelong athletes. Furthermore, recent MRI studies have reported the presence of fibrosis in lifelong (ultra)endurance athletes, the clinical significance of which remains to be elucidated.

**CONCLUSIONS**

The cardiovascular health benefits of exercise are well documented and have led to the development of physical activity guidelines for the general population and a variety of diseased populations. The promotion of physical activity spans the age range from birth to death. Indeed, recent focus on the development of disease during pregnancy has even led to the promotion of physical activity for the fetus. Exercise has been termed the “magic bullet” as it results in an acute simultaneous, multisystem physiological stress, which if repeated chronically leads to adaptations providing global health and performance benefits. At the center of this multisystem response and adaptation is the heart. Acutely, the heart responds instantly to the imposition of an exercise stress through a complex, but well-described, cascade of events resulting in an increased cardiac output and resultant oxygen delivery to active tissues. The physiological in- suit of exercise generally lasts for relatively short periods of time (ca 30 minutes) in the normal population, the consequences of which appear to be almost entirely positive even for those with underlying disease. In contrast, athletes, particularly those engaged in (ultra)endurance events, regularly expose the heart to prolonged stress during training and competition.

The acute cardiovascular consequences of such exercise include an altered function comconitant, but seemingly unrelated, to an elevation in the presence of humoral markers of cardiac damage (cTn). The rapid return to baseline of these changes has led to the suggestion that they are physiologic in nature and likely part of an adaptive process. However, there is initial
evidence that (ultra)endurance exercise may result in a pathophysiological cascade in susceptible individuals. A significant body of work is required to further elucidate the clinical significance of acute responses to (ultra)endurance exercise.

In response to repetitive exposure to exercise in young individuals the heart adapts, resulting in a range of physiological changes characterized by cardiac enlargement and ECG changes at rest, which underpin an enhanced cardiac function at rest and during exercise. These physiological changes may mimic diseases associated with SCD in young athletes. While the incidence of SCD in young athletes is low (1:50,000 to 1:200,000 athletes per year) it is important to identify at-risk individuals given the significant psychosocial impact of such deaths. Pre-participation cardiovascular screening programs have been established and adopted by international bodies of sport globally, but much debate continues regarding the appropriate screening tools, efficacy, and cost-effectiveness of such programs.

The impact of exercise in older populations is less well characterized. However, it appears that positive adaptive responses are observed leading to a slowing of the age-related decline in cardiac function and an enhanced aerobic capacity (VO_2max) and performance. A small, but growing, population of lifelong and veteran athletes undertake prolonged and arduous exercise in the form of (ultra)endurance training and competition. The increased prevalence of arrhythmias combined with cardiac fibrosis reported in a small number of athletes in such studies has raised concern as to the safety of such exercise for some veteran competitors. Future studies examining the acute and chronic effects of prolonged, arduous exercise on the heart in young and older athletes will assist our understanding of the health consequences of such extreme physical activity. Until that time, the vast majority of evidence supports a wholly beneficial effect of exercise on cardiac health across the lifespan in health and disease.

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Olympic Challenges: Performance and Perseverance

*Expert Answers to Three Key Questions*

1. Athletic performance and risk of injury: can genes explain all?
   
   *R. Tucker, M. Collins*

2. Performance and endurance in sport: can it all be explained by metabolism and its manipulation?
   
   *A. E. Jeukendrup*

3. In sport is it all mind over matter?
   
   *T. D. Noakes*
Sporting success is the result of the combination of innumerable genetic and environmental factors, and there is no single path to becoming a champion athlete. Susceptibility to injuries is also a multifactorial phenotype and is a less acknowledged contributor in determining elite athletic ability. The relative importance of deliberate practice, other environmental factors, and genetic factors in molding champions is a constant area of debate. We review two models, the “Practice Sufficiency” and “Genetic Ceiling” models that explain expert performance development and injury risk. We conclude that although the deliberate training and other environmental factors are critical for achieving elite performance, the “Practice Sufficiency Model” does not adequately explain performance. The “Genetic Ceiling Model,” on the other hand, acknowledges both nurture and nature and is a more accurate theory.

Keywords: elite athlete; nature; nurture; musculoskeletal; polymorphism; training

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In the first, which we have termed the “Genetic Ceiling Model,” performance is ultimately determined by the presence (or absence) of specific genetic sequence variants. These genetic variants would predispose an individual to success in certain sports. Conversely, the absence of these variants would limit an individual’s sporting ability. This model holds that innate (genetic) factors are responsible for determining the level of performance reached in a given task, effectively establishing a ceiling beyond which an athlete cannot improve, regardless of training or practice. In fact, many biochemical, physiological, and other intrinsic characteristics of an individual are partly determined by their genetic makeup (Figure 1). 8-11

The alternative theory, developed and proposed by Ericsson et al., 12-14 which we shall term the “Practice Sufficiency Model,” proposes that expert performance is the result of accumulating many hours of deliberate practice. This theory, recently popularized by books such as Malcolm Gladwell’s Outliers, Daniel Coyle’s Talent Code, and Matthew Syed’s Bounce: How Champions are Made, holds that deliberate practice is sufficient to attain expert performance levels in any sport. By extension, it predicts that interindividual differences will disappear with practice, so that every individual can at-
tain performance mastery given sufficient training time. It is worth emphasizing from the outset that any model that adopts an exclusive or extremist approach to explaining sporting performance is likely to be incorrect, given the above-mentioned complexity in sporting performance. That is, neither extreme argument (that genetics or deliberate practice are solely responsible for sporting performance) is likely to be accurate. However, in order to better understand the relative importance of these two views, each must be evaluated based on its singular prediction that either genes or deliberate practice are sufficient for elite sporting performance.

The aim of the present review is to critically evaluate each of the above models in turn, with the purpose of characterizing the relative importance of an athlete’s genetic profile as crucial to sporting success.

THE GENETIC CEILING MODEL

The “Genetic Ceiling Model” traces its origins back to Sir Francis Galton (1822 to 1911) who, having identified that body size and height had some heritable component, wrote the following in 1869:

Now, if this be the case with stature, then it will be true too as regards every other physical feature—as circumference of head, size of brain, weight of grey matter, number of brain fibers...and thence, a step on which no physiologist will hesitate, as regards mental capacity.²

Having extended his heritability model to mental capacity, Galton proposed that improvements in performance could in fact be achieved with training, and that they may occur rapidly at first, but would reach a ceiling for each individual such that maximal performance capacity was ultimately determined by innate, heritable factors. Galton’s model is thus not one that discounts the importance of training, but rather one that says that performance improvements with training become increasingly smaller until an upper limit of performance is reached (as described by Ericsson)³, irrespective of training. We have thus termed this the “Genetic Ceiling Model,” since it holds that performance (and response to training) is ultimately determined by one’s genetic profile.

It is worth noting that at the time of his writing, heritability was very poorly understood—it preceded the discovery of DNA by almost 100 years, for example. Also of interest is that Galton did not apply this model to sporting performance. Indeed, he could not, since in 1869, organized sport was in its infancy, the domain of the upper class male society only, and no research of any kind existed to support arguments.

Figure 1. A diagram illustrating the complex relationship between intrinsic and extrinsic factors that determine elite athleticism.

This is not a complete list of all the factors, but rather illustrates the complexity of the phenotype. Many of the individual intrinsic risk factors are also in their own right multifactorial phenotypes determined by, to a lesser or greater extent, both genetic (nature, G) and environmental (nurture, E) factors.
around sporting performance. However, his theory of a ceiling determined by genetics became established and widely applied, and as argued by Ericsson, consistent with many contemporary theories for skill acquisition.

**THE PRACTICE SUFFICIENCY MODEL**

The opposing view, which we term the “Practice Sufficiency Model,” argues that innate or genetic factors are not required to explain the development of elite performance. This model, developed by Ericsson, holds that performance is the result of the accumulation of sufficiently large volumes of deliberate practice. He writes, for example, that:

> distinct characteristics of exceptional performers are the result of adaptations to extended and intense practice activities that selectively activate dormant genes that are contained within all healthy individuals’ DNA. 12

In this explanation, deliberate practice (training) is sufficient for any individual to attain expert performance in any sport, and the only constraint on the development and achievement of expert performance is the individual’s engagement in deliberate practice and the quality of the available training resources. 12

**Evidence for the “practice sufficiency model”**

To date, we are unaware of any studies that have systematically examined whether training does indeed reduce interindividual differences to zero, or that practice is sufficient to achieve elite level performance in sport. This is unsurprising, however, given the complexity of evaluating skill levels in many sports, and the impossibility of accounting for numerous confounding variables that may have influenced skill acquisition from birth to adulthood. However, there are studies on the development of expert performance in activities and sports where skill is more easily quantified, such as violin playing, chess, and darts. Perhaps the most famous such study is that of Ericsson et al on violin players. This study found that the very best players had accumulated over 10,000 hours of deliberate, solitary training time by the age of 20. 13, 15 giving rise to the now popularized “10,000 hour rule” for performance.

This study, foundational to the “Practice Sufficiency Model,” is worth more detailed analysis. One of the key observations is that the study did not report statistical measures of variance or ranges of training hours within each ability group. This may be problematic, since it is possible that there was significant overlap in the actual training hours between the groups.

This is crucial, because inherent in the “Practice Sufficiency Model” are the following three requirements: (i) variance in performance must be explained entirely by accumulated deliberate practice time; (ii) exceptional performance cannot occur in the absence of high volumes of accumulated training; and (iii) individuals who accumulate high volumes of deliberate practice must always achieve expert or elite levels in that activity. Indeed, Ericsson et al 12 have written that:

> the development of expert performance will be primarily constrained by individuals’ engagement in deliberate practice and the quality of the available training resources.

This implies that failure to reach the “best expert” group must be associated with reduced training time and/or access to resources. If this is not the case, then other factors must constrain performance ability, or enhance it in those cases where individuals reach performance mastery despite performing less accumulated training.

This variance was however evident in a study on the impact of deliberate practice on chess performance, in which 104 players, ranging from those without an international rating to grandmasters (the highest level of chess performance) were evaluated. In agreement with the main finding of the violin study, players who reached the master level had accumulated an average of 11,053±5538 hours of total practice. 16 The coefficient of variation (CV) of 50% suggests how variable this training time was. Specifically, it was found that a range greater than 20,000 hours existed, with the fastest player to attain master level doing so with only 3016 hours, compared with another player who did so with 23,608 hours. This is an eightfold difference. The authors further report that some players had not reached master level despite more than 25,000 hours of practice. Clearly, this study suggests that high training volumes are essential, but not sufficient, for the attainment of high performance levels.

The same appears to be true of darts performance. Eighteen professional and 18 amateur dart players completed questionnaires to ascertain, among many factors, hours of deliberate practice. The average number of accumulated hours of deliberate practice after 15 years of playing was 12,839±7780 for professional men, compared with 3270±2916 hours for amateur men. Again, the CV was high, 61% and 89% for professionals and amateurs, respectively. Since ranges were not reported, it is unclear whether there was overlap between the two groups, though given the large standard deviations, this would seem likely, in a repeat
of the finding with chess. It is not surprising that large differences in practice time exist when comparing amateurs and professionals, or best expert performers and least accomplished performers. This is analogous to the comparison of a physiological determinant of performance such as VO2max between elite athletes and recreational athletes or sedentary individuals. Between these groups, large differences in VO2max exist. However, within each group, VO2max becomes a poor predictor of performance, because numerous other factors, such as efficiency or economy, metabolic capacity, and possible neural factors play a role. 18

Similarly, finding large differences in training volume between professional and amateur darts players and best expert and least accomplished violinists does not necessarily indicate a sufficient role for training time in performance.

In fact, this type of difference may even be explainable as a result of innate differences, which manifest very early on, creating a behavioral bias for further practice. That is, it may be that children who show early promise in an activity as a result of innate factors are encouraged and supported to train more. Indeed, Ericsson13 describes a phenomenon observed by Bloom where young children are introduced to an activity in a playful manner, but “as soon as they show promise compared to peers in the neighborhood, their parents help them seek out a teacher and initiate regular deliberate practice.” The key question is what factors determine the early display of promise in an activity? It may be that when observing an elite group who has already obtained expert status in an activity, one is actually studying the effects of deliberate practice within a group that is already selected from the general population as a result of innate factors. Clearly, practice is important for elite performance, but practice alone cannot be sufficient for performance in any of the previously described studies 15-17

**Sporting examples**

So far, we have focused exclusively on activities where performances are solely determined by motor skill, cognitive ability, and repetition. These activities are not “physiologically constrained” or determined in the way that sports such as football, running, or weight lifting may be. In these sports, it is much more difficult to objectively evaluate performance. It is equally difficult to characterize training as being deliberate or simple play, particularly retrospectively. However, data do exist regarding training time and years of participation in sport before attainment of an elite level. These data have revealed that elite sports people rarely complete 10,000 hours of deliberate training, but that they often participate for approximately 13 years before attaining Olympic or equivalent level performance. 19,20 Wrestlers accumulated only 6000 hours of training in 10 years, 19 while international level footballers accumulated 4000 hours. 20

It is important to note that elite performance at the adult level is a combination of both ability and physical development. As a result, very few teenagers reach senior elite levels in sports that favor a peak after the age of 20. The 13-year period is thus not necessarily a period of deliberate training, but may be a “waiting period” to allow physical development and maturity to peak to enable elite adult competition. Indeed, there is evidence that some sports people succeed within a few years of introduction, provided they commence as adults. In one study, 69% of elite performers had been playing their sport for less than 4 years. 21 However, they had all been exposed to a wide range of other sports before selecting their specialization. This indicates that talent transfer is possible, and has driven the talent ID programs of Olympic programs in Australia and the United Kingdom, where champion athletes have been discovered in skeleton, rowing, and netball, where one player made the national team with 6 years and only 600 hours of deliberate practice! 22,23

**Conclusion**

The studies on deliberate practice have clearly established that accumulated practice time is a key differentiator between elite and sub-elite performers. There is no question that in order to achieve sporting success or to master a skill, high volumes of training are required. However, these studies have not established that innate differences, which may have been present from very early on in childhood, are not themselves responsible for influencing behavioral differences that ultimately result in disparities in accumulated training time. It seems clear that 10,000 hours of training are not required by all athletes, and that some will succeed with substantially less training. Equally, some athletes will fail despite much more deliberate training, though these athletes are rarely documented, an inherent bias leading to an incomplete picture of success and failure in elite sporting performance. The “Practice Sufficiency Model” thus cannot completely explain either success or failure in sporting activities, or even in activities such as chess, violin-playing, and darts, where the risk of injury and the importance of physiology may be less important.
NATURE AND NURTURE: 
A PRACTICAL EXAMPLE

Performance is far more complex than being singly constrained by engagement in deliberate practice. As a result, the “Practice Sufficiency Model” is itself insufficient to explain performance, and so we turn to genetic factors to describe how some of the variances in performance may be accounted for.

It should be noted that elite athletes might not be the ideal population to use to illustrate the genetic contribution in performance, since to reach this level they have already been selected out of the general population and genetic profiles may not therefore play a large role at this level. Thus, within this relatively narrow band of performers, there may be little genetic heterogeneity, and nurture may then become the most important determining factor between first and second place at the Olympics and World championships.

The following case study examines the half-marathon performance of a male recreational athlete over 7 consecutive years. In 2002, at 36 years old and after about 16 months of training, he participated in his first 21.1-km road race. During the following years he specifically trained for and, with the exception of the 2006 event, raced in the same event, running three personal best times. Of note, there was a 23.6-min (20.6%) improvement in his performance during the 8 races. Alternatively, in spite of continued nurture, this athlete will eventually reach an “inherent ceiling” preventing any further improvement. His range for potential improvement in performance is the difference between his currently ability and his personal “inherent ceiling” (Improvement Range).
could complete the race in 67.4 min, 4.5-min slower than the current course record. Since this athlete would be in his early 50s in the next 8-year period, this improvement will bring him within 0.7 min of the 50-55 male half-marathon world record, which one assumes is close to the inherent ability for male athletes (Figure 2B). The logical conclusion of this example is that if only nurtured determined performance then this athlete would be able to at least set an age-group half-marathon world record with the correct coaching.

Alternatively, although there was, at least in this athlete, a large scope for improving performance due to training and motivation (nurture), it is more likely that in spite of continued nurture this athlete will eventually reach an “inherent ceiling” preventing any further improvement. His range for potential improvement in performance is the difference between his current ability and his personal “inherent ceiling” and not between his current ability and the world record.

**GENETIC CONTRIBUTION TO SUPERIOR ATHLETIC PERFORMANCE**

It is generally accepted by the scientific community that both environmental and genetic factors determine an individual’s athletic ability. The role of genetics in elite athletic performance has been extensively reviewed within the scientific literature. We will therefore only highlight some key milestones in the development of this area of research.

The first attempts to identify genetic markers in phenotypes believed to be associated with athletic performance investigated red blood cell and HLA antigens and as well as isoforms of skeletal muscle enzymes in Olympic athletes during the 1970s. These initial studies were followed by an era of genetic epidemiological studies designed to determine the heritability of various physiological traits believed to contribute to performance. Large ranges of heritability estimates have been reported in these studies. The method of linkage analysis studies, which follow the inheritance pattern of a specific phenotype in families, has also been successfully used to identify genes that contribute to performance-related phenotypes in a few studies.

Since 1998, genetic association studies is the predominant method being used to identify the many genetic variants located throughout the human genome that may be associated with performance and performance-related phenotypes. Recently, Bouchard et al. identified 21 single nucleotide polymorphisms, which accounted for 49%
of the variance in VO_{2\text{max}} trainability following a 20-week standardized training program in 473 white seden-
tary adults using a genome-wide association study (GWAS).

**GENETIC CONTRIBUTION TO INJURIES**

Athletes are at increased risk of acute and chronic musculoskeletal tissue injuries as a result of training and/or competition. Predisposition to muscle, tendon, ligament, and/or other injuries can negatively impact an athlete’s ability to train optimally and perform during competition, ultimately ending their competitive careers or preventing them from reaching their full potential. Multiple intrinsic and extrinsic risk factors are implicated in the etiology of these injuries. Like elite athleticism, acute and chronic musculoskeletal tissue injuries are multifactorial phenotypes, determined by a poorly understood interaction of multiple factors (Figure 3). Typical injury risk models include multiple intrinsic risk factors (Figure 3), which individually are also in their own right multifactorial phenotypes determined by, to a lesser or greater extent, both genetic (nature) and environmental (nurture) factors. Many are polygenic traits, with each individual gene having a small effect on the phenotype, but added together having a significant contribution. In addition, these factors do not necessarily determine risk independently of each other. For example, both sex and age are also common intrinsic factors for flexibility.

<table>
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<td>(rs679620 + rs591058 + rs650108)</td>
<td><strong>MMP3 Gene haplotype</strong></td>
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<tr>
<td><strong>MMP10</strong></td>
<td>rs486055 + rs1799750 + rs679620 + rs2276109</td>
<td><strong>MMP Gene cluster haplotype</strong></td>
<td>ACL rupture</td>
<td>35</td>
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<tr>
<td><strong>MMP1, MMP3, MMP12</strong></td>
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<td><strong>Signaling molecules</strong></td>
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<tr>
<td><strong>GDF5</strong></td>
<td>rs143383</td>
<td>Promoter, T/C functional</td>
<td>Achilles tendinopathy</td>
<td>36</td>
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<tr>
<td><strong>IL-1β</strong></td>
<td>rs1143627, rs16944</td>
<td></td>
<td>IL signaling pathway</td>
<td>37</td>
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<td><strong>IL-1RN</strong></td>
<td>rs2234663</td>
<td>IL signaling pathway</td>
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<tr>
<td><strong>IL-6</strong></td>
<td>rs1800795</td>
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Table 1. Sequence variants (polymorphisms) within genes encoding for collagens, glycoproteins, extracellular matrix (ECM) enzymes and signaling molecules associated with soft tissue injuries.

Abbreviations: ?, suggested association bases on observations in small sample sizes. ACL, anterior cruciate ligament; IL, interleukin; MMP, matrix metalloproteinase; TNC, tenascin C.
Recently, specific genetic sequence variants have also been identified as intrinsic risk factors for some injuries (Table I, page 37). These genes encode: (i) structural components of connective tissue (collagens and glycoproteins); (ii) extracellular matrix (ECM) proteinases (MMPs); and (iii) cytokines and growth factors. Interestingly, mutations within COL5A1 and COL1A1 cause Ehlers-Danlos syndrome and osteogenesis imperfecta, respectively. 32,35-37 In addition, common polymorphisms within these genes are associated with milder musculo-skeletal soft tissue injuries (Table I).

In support of this, common polymorphisms within genes are associated with interindividual variation in stature. The tall or short stature syndromes are caused by mutations within the same genes.9

None of the genetic risk factors or any of the other intrinsic risk factors cause injuries. They merely modulate or contribute to the risk for these injuries. Predisposed athletes need to be exposed to appropriate extrinsic factors and an inciting event before they are injured (Figure 2).24,25 For acute injuries, the inciting event will be the macrotraumatic event that causes the injury, while the inciting event for a chronic injury will be the point in time when the volume of accumulated micromechanical damage to the tissue becomes symptomatic.

**CONCLUSION**

The 0.1% difference in the sequence of human DNA results in visible and measurable interindividual differences (ie, biological variation). Athletes are therefore not identical. There are differences in structures and functions of biological systems, and the response of these systems to training and loading are therefore not identical. In addition, the response of injured tissue to healing and treatment modalities is not identical.

Similarly, there is also a large interindividuality ability in athletic performance. An individual athlete’s limits (ceiling) in performance, adaptation to training, injury, healing, and response to treatment modalities is predominately determined genetically. Deliberate training and other environmental factors (nurture) play a critical role in enabling an athlete to reach and maintain their performance potential. Unfortunately, both nature and nurture are also involved in the etiology of injuries, which could eventually prevent athletes from reaching or maintaining their true performance potential.

In summary, we have investigated two models that explain expert performance development and injury risk. Although deliberate training and other environmental factors are important in reaching elite performance, the “Practice Sufficiency Model” cannot adequately explain performance. The “Genetic Ceiling Model,” on the other hand, acknowledges both nurture and nature as well as the impact that nature (genetics) can have on the athlete’s ability to train through injury (nurture), and is a probably more complete theory.

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Performance and endurance in sport: can it all be explained by metabolism and its manipulation?

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Although sports performance is multifactorial, it is clear that muscle performance plays a major role in most sports. Fatigue is associated with metabolic processes in the muscle and by manipulating these processes, exercise can be maintained for longer or power output and speed can be enhanced. The key metabolic factors in sustaining and modulating performance and endurance in sport are: ATP, phosphocreatine, lactic acid, glycogen, and carbohydrate. So in answer to the question “Can it all be explained by metabolism and its manipulation?” the answer should be that metabolism plays a major role in most performances and can explain a large part of performance in many sports. However, fatigue is a multifactorial process and it is the combined effect of all these factors that ultimately determine performance.

Athletic performance is determined by a multitude of components that may or may not be possible to manipulate or train. Depending on the sport, discipline, or even position within a sport, performance will be determined by many variables that will vary in importance dependent on the specific requirements of the activity. For example, the physiological component is believed to be relatively large in marathon running, whereas in golf the skill component is most important. Performance will be determined not only by physical characteristics, but also by biomechanics, hand-eye coordination, agility, reaction time, decision making, and other cognitive functions. Motivation plays an extremely important role as well, and all of these factors are interrelated and do not operate in isolation. Within this context, it is important to realize that muscle function is only one of many factors that influences performance.

Muscle performance is dependent on the force development and/or the sustained repeated contraction of myofibers. When calcium and adenosine triphosphate (ATP) are present in sufficient quantities, the filaments form actomyosin and shorten by sliding over each other (Figure 1). Sliding begins when the myosin heads form cross-bridges attached to active sites on the actin subunits of the thin filaments. The attachment of the myosin cross-bridges requires the presence of calcium ions as well as the binding of ATP to the myosin head at the ATPase activity site. Hydrolysis of the ATP to adenosine diphosphate (ADP) and inorganic phosphate (Pi) by the ATPase provides the energy required to return the myosin to its activated state, giving it the potential energy needed for the next cross-bridge cycle. ATP is the only source of energy that can be used directly not only for muscle contraction, but also for other energy-requiring processes in the cell. All other processes, such as the breakdown of phosphocreatine (PCr), the breakdown of glycogen or glucose to pyruvate or lactate, the oxidation of carbohydrate and fats, serve to replenish the small, but highly dynamic, ATP pool. On a daily basis, a person can turn over his or her own body weight in ATP or more, depending on the level of physical activity. It is therefore tempting to believe that when the substrates that result in ATP synthesis run out,
performance would be affected. If substrates run out, ATP synthesis would be impaired and therefore muscle contraction would be hampered and the intensity of the exercise would have to be reduced. It is also tempting to speculate that by manipulating these stores, fatigue could be delayed. This review will discuss the evidence that depletion of substrates causes or contributes to fatigue and whether this can be reversed by strategies to manipulate metabolism.

**SUBSTRATE METABOLISM DURING VERY-HIGH-INTENSITY AND INTERMITTENT EXERCISE**

As muscle contracts, ATP is degraded to ADP and Pi to provide the energy. During intense maximal exercise, the ATP stores can only provide energy for about 1 second to 2 seconds. When the whole-muscle ATP concentration falls by about 30%, the muscle fatigues. 1, 2 An important function of PCr in muscle is to provide the high-energy phosphate group for ATP regeneration during the first seconds of high-intensity exercise, thus allowing time for glycogen breakdown and glycolysis (the other main process generating cytosolic ATP during high-intensity exercise) to speed up to the required rate. Transfer of the phosphate group from PCr to ADP is catalyzed by the enzyme creatine kinase, resulting in regeneration of ATP and release of free creatine. PCr is present in resting muscle in a con...
centration that is 3 to 4 times that of ATP. During the 100-meter sprint, 22 g of ATP is estimated to be broken down per second, or about 50% of the ATP content per kg of active muscle. Because fatigue occurs in human muscle when the whole-muscle ATP concentration falls by about 30%, the need for rephosphorylation of the ADP formed during contraction is obvious. PCr plays an important role in replenishing ATP. The total creatine content in the muscle is between 120 mmol/kg dry weight and 160 mmol/kg dry weight, with 60% to 70% in the form of PCr with the higher concentrations found in Type II muscle fibers.

Harris et al. were the first to report that ingesting creatine monohydrate could increase total muscle creatine stores (creatinine and PCr). This landmark study showed that ingesting 5 g of creatine 4 times to 6 times a day for several days increased the total creatine concentration by an average of 25 mmol/kg dry weight, and 30% of the increase in total creatine content was in the form of PCr. The authors suggested that these increases could improve exercise performance, but did not test this suggestion in their study. The first performance study was conducted by Greenhaff et al. Subjects ingested 20 g/day of creatine for 5 days, and creatine indeed improved performance by about 6% during repeated bouts of maximal knee extensor exercise. After that study, more studies were performed investigating different modes of exercise, and in a review by Hespel and Derave it was concluded that creatine could improve sprint performance. In addition, in a recent consensus meeting from the International Olympic Committee (IOC), creatine was discussed as one of the few nutrition supplements with substantial evidence of ergogenic effects.

In a study by Casey et al. the changes in performance were related to the changes in total muscle creatine content. A strong correlation was observed in that those individuals who displayed the largest increases in total muscle creatine concentration also exhibited the largest performance benefit. In the literature, these benefits are most obvious in repeated sprints, but also in strength, force production, or torque. In a recent consensus meeting by the IOC, creatine was identified as one of the nutrition supplements that can have a positive effect on performance.

So it appears that creatine metabolism plays an important role in high-intensity exercise performance. If creatine stores in the muscle can be increased by creatine supplementation, this may have an impact on exercise performance, in particular in delaying fatigue with repeated sprints. The most obvious explanation for the ergogenic effects with creatine supplementation are an increased PCr availability, particularly in Type II muscle fibers, which may improve contractile function by maintaining ATP turnover as well as increasing the rate of PCr resynthesis.

**GLYCOLYSIS**

During all-out exercise lasting approximately 1 to 7 minutes, the energy for ATP regeneration is derived from glycolysis. The breakdown of glucose (or glycogen) to pyruvate results in ATP being available to the muscle from reactions involving substrate level phosphorylation. However, the pyruvate must be removed for the reactions to proceed. In some situations, the rate of formation of pyruvate is higher than the tricarboxylic acid cycle rate, which would result in pyruvate accumulation. Therefore, pyruvate is rapidly removed by conversion to lactic acid. The lactic acid dissociates into lactate and hydrogen ions, resulting in a reduced muscle pH. The pH in the circulation can drop from the normal 7.4 to as low as 7.1 within a minute of intense exercise. It is this reduction in pH in the muscle that is believed to impair muscle contraction and be responsible for the development of fatigue.

One way to overcome the acidosis, is to start the exercise with a higher than normal blood pH, i.e., induced alkalosis. This can be achieved by pre-exercise ingestion of sodium bicarbonate in a dose of 0.18 to 0.3 g/kg body weight, or another alkalizing agent, such as sodium citrate. Buffering the protons in blood will allow more protons to leave the muscle. This strategy has proven to be successful in enhancing performance in single or repeated high-intensity exercise bouts. More recently it was suggested that by manipulating the carnosine concentration in muscle (intracellular buffer), performance might be improved as well.

β-Alanine ingestion is an effective way to increase the amount of carnosine in skeletal muscle and thereby indirectly increases the buffering capacity of a muscle. β-Alanine is the rate-limiting precursor in the synthesis of carnosine, a dipeptide composed of histidine and β-alanine. Even though the contribution of carnosine to the total buffering capacity of the muscle is limited (approx 10%), there is a considerable interest in muscle carnosine because its concentration can be nutritionally altered. Harris and coworkers were the first to show that 4 to 10 weeks of oral β-alanine supplementation can markedly increase the muscle carnosine content by 50% to 80%. A more recent study by Baguet et al. showed
showed that the acidosis during a 6-min high-intensity exercise bout is less pronounced as a result of a 4-week β-alanine supplementation period. This indicates that β-alanine-induced muscle carnosine loading has a significant impact on the pH-buffering capacity during exercise.

Although a number of studies have shown an ergogenic effect on single exercise bouts (summarized in Baguet et al, 2010), there is only a limited availability of studies investigating the effect of β-alanine supplementation on repeated sprint ability. Derave et al. showed that the fatigue during 5 bouts of 30 maximal knee extension contractions, separated by a 1-min passive recovery period, was attenuated in the 4th and 5th bout following muscle carnosine loading, but this was not confirmed by a study by Sweeney et al. following 5 weeks of β-alanine supplementation.

In summary, intense contractions during high-intensity exercise results in large production of protons. A significant portion of the contraction-induced protons are rapidly transported out of the active muscles and buffered by the circulating buffers, such as bicarbonate. Therefore, one could term the pH buffers inside the muscle cells (such as carnosine) as the first line of defense and the blood buffers as the second line of defense. From the above, it is evident that nutritional support of both systems can lead to performance-enhancing effects during intense exercise.

**SUBSTRATE METABOLISM DURING PROLONGED EXERCISE**

During more prolonged exercise where aerobic metabolism plays a crucial role, carbohydrate availability might be one of the most important factors for performance. Krogh and Lindhardt were probably the first investigators to recognize the importance of carbohydrate as a fuel during exercise. In their study, subjects consumed a high-fat diet for 3 days followed by 3 days on a high-carbohydrate diet (potatoes, flour, bread, cake, marmalade, and sugar). The subjects performed a 2-hour exercise test and reported various symptoms of fatigue when they consumed the high-fat diet. However, when they consumed the high-carbohydrate diet, the exercise was reported as “easy.” The investigators also demonstrated that after several days of a low-carbohydrate, high-fat diet, the average respiratory exchange ratio (RER) during 2 hours of cycling was reduced to 0.80 as compared with 0.85 to 0.90 when a mixed diet was consumed. Conversely, when subjects ate a high-carbohydrate, low-fat diet, RER was increased to 0.95. This clearly shows that diet and this substrate availability can alter substrate use.

Christensen showed that with increasing exercise intensity the importance of carbohydrate as a substrate increased. In the late 1960s, this was confirmed by a group of Scandinavian scientists who measured the glycogen concentration in skeletal muscle. Muscle glycogen concentration was manipulated by diet and it was shown that a higher muscle glycogen concentration was correlated with increased endurance capacity. These observations have led to the recommendations to carbohydrate-load (ie, eat high-carbohydrate diets) before competition.

The effect of high-carbohydrate diets and elevated muscle glycogen levels on exercise performance has been summarized in a review by Hawley et al. and despite this review being published in 1997, it is still up to date, as evidenced by a recent publication from a consensus meeting by the IOC. It was suggested that supercompensated muscle glycogen levels can improve performance (ie, time to complete a predetermined distance) compared with low to normal glycogen (non-supercompensated) by 2% to 3% in events lasting longer than 90 min. There seems to be little or no performance benefit of supercompensated muscle glycogen levels when the exercise duration is <90 min.

Liver glycogen also plays a role, as it appears that liver glycogen depletion can result in hypoglycemia during exercise, and this coincides with reduced endurance capacity. In one elegant early study, subjects exercised to exhaustion at 70% Upon cessation of exercise (after approximately 3 hours), muscle glycogen concentrations were extremely low (47 mmol glucose units per kg dry mass). Subjects then rested for 20 min and exercised again. During this second bout of exercise they were given placebo, a carbohydrate drink, or they were infused with glucose. When they received placebo they could only exercise for 10 min. With the glucose ingestion they could exercise longer (26 min), but they could exercise even longer when the glucose was infused directly into the circulation (43 min). Blood glucose concentrations dropped with placebo, were somewhat elevated with glucose ingestion, and were maintained with infusion. This study clearly demonstrated that providing a source of glucose will help to delay fatigue. However, this study also demonstrates that plasma glucose is only part of the explanation of fatigue, as subjects still fatigued even when glucose was infused and euglycemia was maintained. The study also demonstrates that muscle glycogen is not the sole cause of fatigue as muscle glycogen concentrations were very low during the
second bout of exercise, yet with the provision of glucose (ingested or infused), exercise could still be continued. Overall, it appears that when carbohydrate oxidation falls below a certain threshold, it is very difficult or impossible to maintain that exercise intensity. Therefore, it can be concluded that carbohydrate metabolism is important for performance, but the source of the carbohydrate (endogenous or exogenous) is only of secondary importance during moderate intensity exercise.

**STRATEGIES TO REDUCE DEPENDENCE ON CARBOHYDRATE STORES**

In addition to optimizing body glycogen stores, it is possible to reduce the reliance on the relatively small endogenous carbohydrate stores by conditioning the muscle to use fat as a fuel. One of the main effects of exercise training and in particular endurance training is an increased capacity to oxidize fat. This is mainly because of an increased mitochondrial density in combination with increased enzyme concentrations and/or activities. Strategies have recently been investigated that combine exercise training with nutritional strategies to increase adaptations in fat metabolism.

The muscle is an organ that can adapt significantly and rapidly in response to repeated bouts of exercise. These adaptations are determined largely by the mode of exercise and the volume, intensity, and frequency of the training stimulus. However, evidence is accumulating that nutrient availability serves as a potent modulator of many acute responses and chronic adaptations to both endurance and resistance exercise. Changes in macronutrient intake rapidly alter the concentration of blood-borne substrates and hormones, causing marked perturbations in substrate storage and protein synthesis. In turn, muscle energy status exerts profound effects on resting fuel metabolism and patterns of fuel utilization during exercise, as well as acute regulatory processes underlying gene expression and cell signaling. As such, these nutrient-exercise interactions have the potential to activate or inhibit many biochemical pathways with putative roles in training adaptation.

Muscle glycogen stores are related to expression of genes relevant to the adaptation to training. It is generally thought that training adaptations are the result of recurrent changes in gene expression, which occur with every bout of exercise, leading to a change in phenotype such as increases in fatty acid transport and oxidation. For example, a single bout of exercise increases muscle mRNA content of peroxisome proliferator-activated receptor-γ coactivator 1α (PGC-1α), a transcriptional regulator of mitochondrial biogenesis. It has been demonstrated that manipulations of energy/carbohydrate availability as part of an exercise training program can improve adaptations in muscle and enhance fat metabolism. Future studies will have to demonstrate that such improvements also result in improvements in exercise performance.

In summary, low muscle glycogen and low rates of carbohydrate oxidation are associated with reduced exercise capacity. Endurance performance can be improved by manipulating carbohydrate stores in liver and muscle or by ingesting carbohydrate during prolonged exercise. In addition, exercise training (with or without nutritional manipulation) can enhance fat metabolism, reduce the reliance on carbohydrate as a fuel, and enhance exercise performance long term.

**CONCLUSION**

There is a large body of evidence to show that metabolism can explain a large part of performance in many sports. In high-intensity and intermittent sports, the roles of ATP and PCr are crucial and it has been demonstrated that an increase in muscle creatine stores can have a positive effect on performance, especially when high-intensity bouts are repeated, suggesting that the main role of creatine is to allow a faster resynthesis of PCr. During events that last 1 to 7 min, both aerobic and anaerobic metabolism play an important role, but there is little evidence that increasing substrate stores could affect performance. The stores do not run out in this time and other factors have an overriding effect on performance (lactic acid formation from the rapid breakdown of glycogen). During longer exercise bouts, both muscle and liver glycogen may become depleted and strategies to optimize these stores prior to exercise as well as ingesting carbohydrate during exercise have been shown to be highly successful in postponing fatigue and improving performance.

So back to the question: “Can it all be explained by metabolism and its manipulation?” The answer should be that metabolism plays a major role in most performances and can explain a large part of performance. However, fatigue is a multifactorial process and it is the combined effect of all these factors that ultimately determine performance.
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In sport is it all mind over matter?

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An influential book published in 1915 included the prescient observation that fatigue during exercise “at first sight might appear an imperfection of our body, (but) is on the contrary one of its most marvelous perfections. The fatigue increasing more rapidly than the amount of work done saves us from the injury which lesser sensibility would involve for the organism.” But after 1923 this idea was replaced with the alternative theory that fatigue is due exclusively to a catastrophic failure of skeletal muscle function. The past decade has seen a remarkable reintegration of the neurosciences into exercise physiology with the growing appreciation that this “brainless” catastrophic model is overly simplistic. Instead, a more complete understanding of the complex nature of exercise fatigue has developed.

A PRECURSOR:
ANGELO MOSSO

Modern attempts to understand the factors that determine fatigue and superior athletic performance can be traced to European studies beginning in the late 19th century. An influential book1 by Angelo Mosso (1846-1910), Professor of Physiology at the University of Turin (Figure 1), included a number of prescient observations:

On an examination of what takes place in fatigue, two series of phenomena demand our attention. The first is the diminution of the muscular force. The second is fatigue as a sensation (p 154)… In raising a weight we must take account of two factors, both susceptible to fatigue. The first is of central origin and purely nervous in character—namely, the will; the second is peripheral, and is the chemical force which is transformed into mechanical work (pp 152-153).

Mosso also understood that fatigue, which:

at first sight might appear an imperfection of our body, is on the contrary one of its most marvelous perfections. The fatigue increasing more rapidly than the amount of work done saves us from the injury which lesser sensibility would involve for the organism’ (p 156).

He also realized that the brain is unique because it is the only organ protected from the effects of starvation (p 282).

But he is best remembered for his dominant conclusion that:

nervous fatigue is the preponderating phenomenon, and muscular fatigue also is at bottom an exhaustion of the nervous system (p 177).2

It has taken studies of “fatigue” more than a century3 to rediscover Mosso and to consider what he believed to be obvious—that both the brain4 and the muscles5,6 alter their function during exercise, that the change in skeletal muscle function is characterized by a slowing of the force and speed of contraction7, and that fatigue is predominantly an emotion,8 part of a complex regulation,9,10 the goal of which is to protect the body from harm.

So fatigue is indeed one of the human body’s “most marvelous perfections.”

Keywords: central governor; fatigue; heart; Hill AV; homeostasis; skeletal muscle
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ARCHIBALD HILL’S STANDARD MODEL

Interestingly, Mosso’s ideas did not gain immediate purchase in the exercise sciences. Instead, they lay dormant and were supplanted after 1923 by a different and more simplistic interpretation promoted by English Nobel Laureate Archibald Vivian Hill (Figure 2). The studies that would become perhaps the most influential in the history of the exercise sciences were performed by Hill and his colleagues at University College, London between 1923 and 1925.11-14 Hill’s personal beliefs of what causes fatigue strongly influenced his interpretation of his quite simple experiments.

Thus, his conclusions and, as a result, the intellectual direction down which his ideas channeled the exercise sciences were determined by Hill’s preconceptions even before he undertook his first experiment.15-17 His personal beliefs were fashioned by at least three factors.

Firstly, since he was principally a muscle physiologist, it was natural that Hill’s theories would begin from that perspective.

Secondly, a series of studies performed at Cambridge University by another Nobel Laureate, Frederick Gowland Hopkins. The crucial 1907 study18 that influenced Hill’s thinking had been designed to develop a novel method accurately to measure muscle lactate concentrations in recently killed laboratory animals, specifically frogs. By plunging excised frog muscles into ice-cold alcohol, Fletcher and Hopkins were able to show that lactate concentrations were elevated in muscles that had been stimulated to contract until failure; these concentrations fell in muscles stored in a high oxygen concentration. As a result they concluded that:

Lactic acid is spontaneously developed, under anaerobic conditions, in excised muscle so that the accumulation of lactic acid in muscle occurs only in the conditions of anaerobiosis. With a proper oxygen supply it fails to accumulate at all. [They also wrote that] Fatigue due to contractions is accompanied by an increase of lactic acid.

But Hill’s interpretation of these results was more doctrinaire, specifically: (i) that lactic acid is produced only under conditions of muscle anaerobiosis; and (ii) that muscle fatigue is caused by increased muscle lactate concentrations. These ideas would form the twin pillars of Hill’s nascent theory of the factors that cause fatigue and determine human athletic performance.

Thirdly, studies published in 1909 and 191019,20 apparently showing that the inhalation of oxygen significantly improved performance during exercise. This led to the conclusion that “the supply of oxygen to the body is the decisive factor in setting the limit to exercise” (p 136).2

As a result from studies conducted on himself when he ran at 10, 12, and 16 km/h around an 84.5-m track near the Physiological Laboratory in Manchester, Hill concluded that increasing muscle lactate (lactic acid) concentrations secondary to the development of skeletal muscle anaerobiosis caused the fatigue he experienced when running at 16 km/h. Accordingly, he developed a model of human exercise physiology (Figure 3, page 48) that has dominated teaching and research in the exercise sciences ever since.21-26

Hill’s model predicts that shortly before the termination of maximal exercise the oxygen demands of the exercising muscles exceed the (limiting) capacity of the heart to supply that oxygen. This causes skeletal muscle anaerobiosis with the accumulation of “poisonous” lactate (lactic acid) in the muscles. So Hill believed that the heart’s capacity to pump a large volume of blood to the active skeletal muscles was the single factor determining the human’s ability to perform maximal exercise by resisting the development of fatigue.

Remarkably, the most interesting component of Hill’s model is that which has been (conveniently) ignored for the past 90 years. For the really important question is: If the cardiac output indeed limits maximal exercise performance, then what limits the maximal cardiac output?

Hill believed that the answer was obvious—specifically, the development of myocardial ischemia the instant the maximum (limiting) cardiac output was reached. Indeed, this would be the modern conclusion.
So Hill’s complete model theorized that maximal exercise is limited by the development of myocardial failure consequent to the development of myocardial ischemia. This model soon became the standard teaching in the textbooks of the day. The blood supply to the heart, in many men, may be the weak link in the chain of circulatory adjustments during muscular exercise, and as the intensity of muscular exertion increases, a point is probably reached in most individuals at which the supply of oxygen to the heart falls short of its demands, and the continued performance of heavy work becomes difficult or impossible (p 175-176).

However, Hill added one final embellishment to his model by proposing that a “governor” either in the heart or brain reduces the pumping capacity of the heart immediately this inevitable myocardial ischemia develops. By causing a “slowing of the circulation,” this “governor” would protect the ischemic myocardium from damage in the period immediately before the exercise actually terminated. But his concept of a “governor” mysteriously disappeared from the next generation of textbooks of exercise physiology, perhaps because of evidence that the healthy heart does not become ischemic even during maximal exercise.

But instead of concluding that the absence of myocardial ischemia during maximal exercise disproves the Hill model, succeeding generations of exercise physiologists have continued to preach, as fact, the original Hill hypothesis that a limiting cardiac output is the sole important factor regulating human exercise performance.

Indeed, the special 2008 Olympic Games edition of the influential Journal of Physiology includes the statement that:

the primary distinguishing characteristic of elite endurance athletes that allows them to run fast over prolonged periods of time is a large, compliant heart with a compliant pericardium that can accommodate a lot of blood, very fast, to take maximal advantage of the Starling mechanism to generate a large stroke volume (p 31).

Like the Hill model this explanation interprets fatigue as a “catastrophic” event that occurs only after skeletal muscle function has failed, specific-
cally “severe functional alterations at the local muscle level.” Overlooked is Mosso’s conclusion that fatigue is “one of its (the human body’s) most marvelous perfections.”

PROBLEMS WITH HILL’S TRADITIONAL EXPLANATION OF HOW HUMAN EXERCISE PERFORMANCE IS “LIMITED”

Hill’s original explanation poses a number of significant problems. Firstly, it seems improbable that human athletic performance can be reduced to a single variable and especially one that allows no role for psychological factors such as motivation and self-belief, which most agree clearly play some role in human athletic performance.

For if exercise is regulated purely by a failure of the cardiac output to provide the muscles with an adequate oxygen supply, then psychological factors cannot play any role in human exercise performance. Yet, even those who vigorously defend the Hill model, still acknowledge that by providing “motivation,” the brain is indeed involved in determining a maximal effort. Hence: “There is no doubt that motivation is necessary to achieve VO2max (p 26).”

But if exercise is regulated purely by a failure of first the heart and then of skeletal muscle function, then there is no need for any special motivation to reach that inevitable state of biological failure; one simply continues to move the legs until they fail. Like the proverbial dead horse, no amount of beating (motivation) can force muscles with “severe functional alterations” to keep working. Nor is any beating required to achieve that catastrophic state. A painful beating will enhance performance only if there is a biological control system that prevents a truly maximal effort (but which can be partially overridden or distracted by the pain of a beating).

Indeed if exercise is “limited” solely by an inevitable catastrophic skeletal muscle failure, then there is no need for the symptoms of fatigue whose principal function must be to forestall such catastrophes. So the presence of the noxious symptoms of fatigue must indicate that exercise cannot be regulated solely by an inevitable and unavoidable failure of skeletal (and or cardiac) muscle function. Rather, fatigue symptoms must play a significant biological role.

Secondly, according to the Levine model, the best athletes must have the largest hearts and the greatest capacity to transport and consume oxygen. But this has never been shown. Neither is the VO2max—a surrogate measure of peak cardiac function according to this theory—a good predictor of athletic ability nor even of the changes in performance that occur with training.

Thirdly, if exercise performance were limited solely by the function of the heart, then one would expect the cardiac output always to be maximal during all forms of exercise. But this is obviously not the case. Improbably, these significant logical arguments have not prevented the global acceptance of this theory as the sole correct explanation.

REPLACING THE HEART ALONE “LIMITATIONS” MODEL OF HUMAN EXERCISE PERFORMANCE

Replacing Hill’s cardiovascular/anaerobic/catastrophic model of exercise performance with a novel model began with the realization that the Hill model is unable to explain two of the most obvious characteristics of human exercise performance. The first is that athletes begin exercise at different intensities or paces depending on the expected duration of the planned exercise bout—a bout of short duration is begun at a much faster pace than is one of much longer duration (Figure 4, page 50). Thus, athletes always show an anticipatory component to their exercise performance. Since human skeletal muscle probably does not have the capacity to anticipate what is to happen in the future and especially the demands to which it will be exposed (by the brain), the Hill model of peripheral exercise regulation cannot explain this phenomenon.

The second inexplicable observation is that humans also speed up near the end of exercise, the so-called end spurt (Figure 4). This finding significantly disproves the popular belief that fatigue increases progressively and inexorably during prolonged exercise so that athletes reach their most fatigued state immediately prior to the termination of exercise. Were this so, the end spurt could not occur.

There are also significant problems with certain physiological predictions of this model. These include:

An absence of evidence that muscle become “anaerobic” during exercise; the absence of a “plateau” in oxygen consumption or cardiac output at exhaustion during maximal exercise; the failure to identify metabolites that explain why muscles “fatigue” during exercise, so that “metabolic causes for these changes (in fatigued skeletal muscle) are hard to identify” (p 2985); and the absence of evidence for any catastrophic failure of organ function at exhaustion. Rather, exercise always terminates with the maintenance of cellular homeostasis.
But the most compelling evidence is the finding that skeletal muscle is never fully recruited during any form of exercise. For the Hill model predicts that as (peripheral) fatigue develops in the exercising muscle fibers, so the brain must compensate by recruiting additional fresh fibers to sustain the work rate. This process would continue until all the available motor units in the active muscles had been recruited. Once all recruited fibers had each begun to fail, the work rate would fall and “fatigue” would become apparent.

Yet it is now established that fatigue in all forms of exercise develops before there is complete skeletal muscle recruitment. Indeed, only between 35% to 50% of the active muscle mass is recruited during prolonged exercise, during maximal exercise this increases to only about 60%. These findings suggest that the Hill model is too simple properly to explain how human exercise performance is truly regulated.

**THE EVOLUTION OF A COMPLEX MODEL OF HUMAN EXERCISE REGULATION**

Inspired by Hill’s concept of a “governor” regulating human exercise performance, my colleagues and I have developed a complex model of human exercise regulation in which human exercise performance is not limited by a failure of homeostasis in key organs like the skeletal muscles, but is rather regulated in an-
Usually, this has focused on the heart and circulation, reflecting the dominance that the Hill model has exerted in this field. But elite athletes clearly do not share this certainty. Rather, they believe that something more complex than the heart is the ultimate determinant of their performances.

Thus, Paavo Nurmi, perhaps the greatest distance runner of all time since he won 9 gold and 3 silver medals in the Olympic Games, wrote that: “Mind is everything. Muscles are pieces of rubber. All that I am, I am because of my mind.”

Franz Stampfl, who coached Roger Bannister to become the first human to run the mile in less than 4 minutes, also wrote that: “The great barrier is the mental hurdle.”

One of the greatest mile runners of all time, Australian Herb Elliott, has also written that:

To run a world record, you have to have the absolute arrogance to think you can run a mile faster than anyone who’s ever lived; and then you have to have the absolute humility to actually do it (p 110).

Of Elliott and his coach, a contemporary runner, Derek Ibbotson, who was unable to beat Elliott, wrote admiringly:

Together Cerruty and Elliott have brought athletics to the threshold of a new era. They have proved conclusively that not only the body but also the mind must be conquered.

**Figure 5. The “Central Governor Model” of exercise regulation.**

This model proposes that the brain regulates exercise performance by continuously modifying the number of motor units that are recruited in the exercising limbs. This occurs in response to factors that are present before and during the exercise—for example, self-belief; prior experience; the presence of competitors; the prospect of monetary reward; mental fatigue; and drug actions—and those which act purely during exercise—for example, afferent sensory feedback responding to heat; brain oxygenation; fatigue; running downhill; and muscle soreness, to name but a few that have been identified. The goal of this control is to ensure that humans always exercise with reserve and terminate the exercise bout before there is a catastrophic failure of homeostasis. The brain uses the unpleasant (but illusory) sensations of fatigue to ensure that the exercise intensity and duration are always within the exerciser’s physiological capacity. This model therefore predicts that the ultimate performances are achieved by athletes who best control the progression of these illusory symptoms during exercise. For more details see references 8-10, 45-49.
Another Australian, former world marathon record holder Derek Clayton, wrote:

The difference between my word record and many world class runners is mental fortitude. I ran believing in mind over matter.57

Hendrick Ramaala, winner of the 2004 New York City Marathon, has subsequently written that:

What I realize is that once the mind accepts anything, the body will respond … If you don’t convince yourself that you are going to win, then you aren’t going to win it. For New York, I have to tell myself thousands of times that I am going to win this thing. … You have to talk to yourself otherwise you are not going to win. You have to say: “Whatever happens I am going to win.” In my opinion, the person who wins the race has already won it inside his head before the start of the race.

Describing his experiences in a sport—golf—in which few would argue that the mind must play an inordinately important role, Gary Player, one of that sport’s legends, has written:

The mental aspect of sport is very important and in many cases the most critical factor in winning. I may not have been the most talented player of my generation, but I was the best prepared both mentally and physically. I loved pressure. I fed off it. I revelled in it. I knew that when the time came for me to hit the shot or sink the putt I needed to, to win, I could do it. The thought of failure was never in my mind—I wouldn’t let myself think that way. If the conditions were horrible I would tell myself that I would love playing in the rain and the wind and I would have a great round. When I heard my competitors complaining about how hard it was to play in those conditions, I knew that I had an advantage. They have already set themselves up for failure (p 92).58

All these quotations suggest that the very highest achievers in sport think differently than do the rest of us. But how might such thoughts influence the sporting outcome?

In skill sports like golf the answers appear clear. The skilled sporting movements are controlled in the arcaic parts of the brain. Trying to use our working-memory and the prefrontal cortex interferes with the smooth operation of those subconscious controls.

As a result, consciously thinking about a complex activity while it is being executed causes “paralysis by analysis and you choke under pressure”59 (p 209). The key to success in skilled movements is not ever to think about the execution, but to focus only on a (perfect) outcome.

But in physical sports like distance running, the explanations are not as simple. Consider the 1996 Men’s Olympic Marathon. In a race that lasted more than 129 minutes, Josiah Thugwane won by a mere 3 seconds, less than 0.04% of the total race time. What could account for such a tiny difference?

According to the traditional Hill model the runner who finished second must have had either higher muscle lactate concentrations or lower muscle glycogen concentrations so that his “poisoned” or “depleted” muscles were simply unable to close that 3-second gap. But simple logic exposes the error in this explanation. For in the final stages of that race, perhaps as many as 65% of the muscle fibers in both the leading runners’ legs were inactive and did not contribute to the physical effort. Surely the second runner could have activated just a few more of those fibers in order to achieve everlasting Olympic glory? What prevented that choice?

The Central Governor Model predicts that brain-generated sensations of fatigue—unique to each individual and influenced by a host of currently unknown individual factors (Figure 5)18,10,45-49—insure that athletes will complete all exercise bouts, including the Olympic Marathon, without risking a catastrophic failure. In this case, the Central Governor Model was clearly successful—neither athlete died. But if the second runner did not die, why did he not run just a little faster and so approach death a little closer? For surely he could have sped up by just 3 seconds without dying? Yet he did not. Why not?

The answer I propose is that the second runner has convinced himself that it was no longer worth the effort. So somewhere in the final few kilometers of the race his conscious mind had decided that even if he were to speed up, he would still not win the race, since in response to any surge the leading runner would simply counter by speeding up more as he had repeatedly done each time the second runner had tried to overtake him in the final 10 km of the race.

Thus my conclusion is that in the case of a close finish, physiology does not determine who wins. Rather somewhere in the final section of the race, the brains of the second and lower placed finishers accept their respective finishing positions and no longer choose to challenge for a higher finish. Once each runner consciously accepts his or her finishing position, the outcome of the race is decided. So just as a single athlete must “decide” to win, so too must the rest of the top finishers decide the opposite—specifically that they are not going to win. Furthermore, the Central Governor Model suggests that this outcome will be strongly
influenced by the manner in which the brains of the respective runners generate the sensations of fatigue during exercise. Recall that these symptoms of fatigue are entirely self-generated by each athlete’s brain and so are unique to each individual. We also know that the rate at which these sensations increase is independent of the actual distance travelled, but is linearly proportional to the percentage of the total distance covered. As a result, these symptoms are unrelated to the actual amount of work done and hence the true state of bodily fatigue. Instead they are illusionary.

It is indeed as Bainbridge wrote in 1919:

the sense of fatigue is often a very fallacious index of the working capacity of the body...there is not necessarily any correspondence between the subjective feelings of fatigue and the capacity of the muscles to perform work...it is a protective feeling, which tends to restrain a man from continuing to perform muscular work when this would react injuriously upon his whole system (pp 176-177). 60

According to this model, the winning athlete is the one whose illusionary symptoms interfere the least with the actual performance—in much the same way that the most successful golfers are those who do not consciously think when they play any of their shots. In contrast, athletes who finish behind the winner may make the conscious decision not to win, perhaps even before the race begins. Their deceptive symptoms of “fatigue” may then be used to justify that decision. So the winner is the athlete for whom defeat is the least acceptable rationalization.

How athletes and coaches achieve this winning mental attitude is the great unknown. But if the study of the purely physiological basis of fatigue has taught us anything, it is that such studies will never provide an adequate answer. Rather that future lies in identifying the manner in which the brains of different athletes generate these illusory symptoms, and why they are suppressed in the winning athletes even as they exercise more vigorously than all others.

“The fight,” wrote Muhammad Ali “is won or lost far away from witnesses, behind the lines, in the gym, out there on the road, long before I dance under the lights” (p 154). 61

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Olympic Sports & Humor

Cartoons by Claude Serre

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DIALOGUES

Olympic Challenges:
Performance and Perseverance

Summaries of Ten Seminal Papers

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Dialogues Cardiovasc Med. 2012;17:63-73

1. Circulatory response to prolonged severe exercise
   B. Saltin, J. Stenberg. J Appl Physiol. 1964

2. Cardiac output in athletes

3. Comparative left ventricular dimensions in trained athletes

4. Arterial blood pressure response to heavy resistance exercise
   J. D. MacDougall and others. J Appl Physiol. 1985

5. Cardiac fatigue after prolonged exercise
   P. S. Douglas and others. Circulation. 1987

6. The upper limits of physiologic cardiac hypertrophy in highly trained athletes

7. Electrocardiographic changes in 1000 highly trained elite athletes

8. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans

9. Ethnic differences in left ventricular remodeling in highly-trained athletes: relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy
   S. Basavarajaiah and others. J Am Coll Cardiol. 2008

10. Diverse patterns of myocardial fibrosis in lifelong, veteran endurance athletes

Selection of seminal papers by Gregory Whyte, PhD; Keith George, PhD
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Highlights of the years by Ian Mudway, MD
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Circulatory response to prolonged severe exercise

B. Saltin, J. Stenberg

J Appl Physiol. 1964;19:833-838

The history of 20th-century developments in sports medicine and exercise science was heavily influenced by the Scandinavian countries. From laboratories in Denmark and Sweden, predominantly, we have seen a number of influential scientists produce groundbreaking insights into the human response to exercise. One of the most recognizable names in sports medicine and exercise sciences is that of Bengt Saltin who in the 1960s was working in Stockholm, but now, 11 years into the 21st century, is still a very active scholar working out of Copenhagen. This seminal paper represents one of Bengt Saltin’s early works in Stockholm with a collaborator, Jesper Stenberg.

Since the 19th century, scholars had been interested in the cardiovascular and metabolic responses to prolonged exercise. The concept that skeletal muscle fatigue can occur after both short-term and long-term activity was well known and documented, and strides were being made in terms of understanding the metabolic mechanisms. There was an ongoing debate that exists to this day about whether cardiac or peripheral muscle factors limited a person’s maximum oxygen uptake. Despite this, the concept that cardiac muscle function could decline during prolonged exercise or into the recovery period was not even contemplated. “Cardiac fatigue” was unconscionable as the heart was a highly aerobic or oxidative organ with substantial blood flow and clearly couldn’t stop working to recover after bouts of exercise. Despite this, it was clear that during exercise where the external work was clamped at a specific level over a prolonged period there were adjustments in cardiac function with time. Specifically, a continuous rise in heart rate was noted that was assumed to coincide with a slow inexorable decline in stroke volume, which was itself due to a reduction in plasma volume, assumed to be limiting left ventricular filling and preload.

Saltin and Stenberg’s work sought to investigate the specific cardiovascular responses to 3 hours of steady state exercise. Notably, given today’s drive for power analyses and statistics, it is a study based on careful observation, assessment, and documentation of only 4 subjects who varied in fitness status. Exercise mode altered from cycle to running during the protocol (with a brief 15-min rest period) and some exercise was performed supine in some subjects. A thorough range of cardiovascular and metabolic assessments were made with a range of invasive and noninvasive techniques. Interestingly, likely for quality control, one subject performed the testing on two occasions and attention to detail is apparent as blood loss due to sampling was replenished by saline infusion. The results were fascinating and presented in a figure that has been often reproduced and copied. Of specific interest was the standard cardiovascular drift with heart at 3 hours of exercise 15% higher than after reaching an initial steady state early in exercise. This was almost matched by a drop in stroke volume across exercise duration such that cardiac output marginally increased in 3 subjects, but dropped in the fourth. Blood pressure actually dropped as exercise duration progressed from an early steady state, and blood volume was well maintained despite fluid loss due to sweating. The author’s interpretation of the drop in stroke volume, while hemodynamic loading was maintained, was that this could reflect impaired performance of the heart. In many respects this specific aspect of the data and interpretation was lost to exercise science for about 20 years before other groups renewed their interest in “exercise-induced cardiac fatigue.”

Austrian skier Egon Zimmermann wins the downhill gold medal at the 1964 winter Olympics in Innsbruck; Jim Bunning of the Philadelphia Phillies pitches a perfect game in a 6-0 victory over the New York Mets; and Cassius Clay defeats Sonny Liston to win the World Heavyweight Championship
A talented athlete and inquisitive physician, Bjorn Ekblom was working with Lars Hermansen, himself a world renowned scientist. Like Bengt Saltin, Bjorn Ekblom was highly productive in the 1960s with a strong emphasis on understanding the cardiovascular system and its functional capacity during exercise.

In the late 1960s, Bjorn Ekblom and Lars Hermansen were fascinated by what physiological parameters underpinned the very high maximal oxygen uptake data recorded in endurance athletes in previous studies in Scandinavia. As a consequence, the study in 1968 looked at very-high-level endurance athletes (n=8, across a range of sports), including a world champion cyclist. A second group (n=5) of lower level, but still endurance-trained subjects, were included as an interesting comparator group. As with the previous study, a detailed range of invasive and noninvasive measurements, at rest and during exercise, were completed, as well as the Douglas Bag and Haldene method to estimate maximal oxygen uptake. Gold standard techniques (for the time) assessed lung volumes and function at rest, heart volume at rest, hemoglobin concentration at rest and during exercise, as well as heart rate, blood pressure, stroke volume, and cardiac output at submaximal and maximal exercise. Again, quality control and repeatability assessments were built into the study and are important reminders to scientists today about how data should be interpreted fully in the light of such values.

The highest maximal oxygen uptake of 81.1 mL·kg⁻¹·min⁻¹ was recorded in the World Champion cyclist, who uniquely, but appropriately, was assessed during a cycle protocol. He also had a heart volume of 1240 mL and a vital capacity of close to 6 liters. Overall performance was higher in the elite athletes and this was clearly underpinned by augmented cardiovascular function during maximal exercise. While maximal heart rate was not different between the two groups, the elite athletes had a higher maximal stroke volume (189 vs 149 mL·beat⁻¹) and thus maximal cardiac output (36.0 vs 28.4 L·min⁻¹). Visual presentation of scatter-plot data differentiated the two groups, but also highlighted the strong linear association between heart volume, maximal stroke volume, maximal cardiac output, and maximal oxygen uptake. This paper was presenting strong descriptive data to suggest that maximal oxygen uptake was strongly linked to cardiac performance. This argument has developed over the past 45 years to the interest of most in the exercise science world as to whether peripheral or central factors limit maximum oxygen uptake. Interestingly, Ekblom and Hermansen in this study reported similar arteriovenous oxygen differences between the elite and sub-elite group. A further point is worth noting, which has also continued to perplex exercise scientists and generate often-heated debate. The authors note that maximal stroke volume was achieved during maximal exercise in 9 out of the 13 athletes. Even current “point-counterpoint” debates still argue the issue of whether stroke volume is limited at submaximal exercise intensities due to filling time restriction.

Another interesting and well conducted study from Scandinavia that continues to resonate in exercise science in the 21st century.

Belgium cyclist Eddy Merckx wins his first Tour de France; Australian tennis player Rod Laver completes his second Grand Slam of tennis majors, the first of the Open Era; and Mario Andretti wins the 54th running of the Indianapolis 500.
Comparative left ventricular dimensions in trained athletes

J. Morganroth, B. J. Maron, W. L. Henry, S. E. Epstein

Ann Intern Med. 1975;82:521-524

The Scandinavians were leading the way in the assessment of cardiovascular responses to acute exercise bouts of short, maximal intensity, or prolonged submaximal intensity. It is interesting to also note that Ekblom and Hermansen (preceding page) also included a measure of static cardiac dimensions in their study—biplane roentgenograms—as it was believed that heart size was likely and important determinant of cardiovascular function during exercise and thus functional or sporting capacity. Despite this belief, what was missing was an accurate, sensitive, and reproducible method of assessing left ventricular structures (wall and chamber dimensions) that underpinned the systemic circulation. Indeed, we could go back to another Scandinavian, a Dr Henschen, who in 1899 reported on cardiac percussion of cross-country skiers. Dr Henschen reported enlarged hearts that were important in determining elevated exercise capacity. Precisent thoughts from the 19th century! However, as we moved into the second half of the 20th century, technology was advancing and ultrasound imaging was being developed. From the first stumbling steps in the 1950s came clinical application in the late 1960s and early 1970s. It was no surprise that the application of ultrasound imaging to the “athletic heart” came in 1975.

Using “ice-pick” motion mode (M-mode) ultrasound echocardiography, Joel Morganroth and his colleagues embarked on a landmark paper studying the dimensions of the left ventricle in athletes with dichotomous training background (endurance vs resistance) and comparing them with sedentary controls. Part of the aim was to present normative data for athletes that might help clinical evaluation, a point developed in a productive career by Barry Maron, the second author. Despite what we now know as the limitations of these early echocardiographic studies (no 2-D guide to M-mode positioning, spatial resolution of 1-2 mm, use of geometric assumptions for 3-D parameter generation such as mass and volume) this was fascinating and new data. The authors reported in a mix of college and international athletes, who were almost 100% white, that endurance training resulted in an increase in left ventricular (LV) mass due to chamber enlargement, and this reflected the specific hemodynamic stress encountered in multiple acute training sessions. Conversely, resistance training resulted in an increase in LV mass that was entirely due to LV chamber wall hypertrophy, with no alteration in chamber internal dimension/volume. Again, the authors speculated that this was due to the hemodynamic stress, increased blood pressure and afterload, associated with acute resistance training. While these adaptations have since been referred to as eccentric and concentric hypertrophy, Morganroth and colleagues did not use these terms. They did, however, draw morphological parallels with a number of cardiovascular diseases that alter hemodynamic loading and result in similar changes in LV structures.

This paper spawned hundreds of “copycat” papers that still appear today, looking at different athlete groups, different sexes, different ethnicities, different ages, etc, as well as a smaller number of longitudinal training studies to address the important point of genetic predisposition that Morganroth and colleagues did appreciate. Morganroth’s “sport-specific cardiac adaptation” hypothesis has endured to the point of being commonly repeated in textbooks of cardiology and sports medicine. Renewed interest recently has been focused on Morganroth’s hypothesis as new, more accurate imaging tools such as magnetic resonance imaging have been introduced and focus has partially shifted to global and segmental LV function in athletes. Others are now looking at the right ventricle and the left atria of athletes…. all from the humble beginning of Morganroths’ paper in 1975.

1975

Long-jump motorcyclist Evel Knievel (USA) suffers his 433rd fracture; Arnold Schwarzenegger wins Mr Olympia bodybuilding title; and the Flying Coyotes, a group of Wyoming skiers, perform a 16-man backflip on skis, shattering the world record.
After multiple developments in noninvasive imaging in the 1970s and 1980s, there was a real focus on heart morphology, especially as this had direct clinical relevance to the burgeoning area of differential diagnosis of the athletic heart from inherited cardiac diseases that could predispose to sudden cardiac death. Despite this emphasis on noninvasive imaging, there were still substantial gaps in our knowledge of the acute cardiovascular responses to exercise and in particular to very-high-intensity or resistance exercise. Up to this point, most exercise science investigation had been directed to submaximal or endurance exercise as this was the predominant physical activity of most sports, exercise, and work scenarios, and it had been linked epidemiologically to reduced risk of cardiovascular morbidity and mortality.

One group of well-known and highly respected researchers based in Hamilton, Canada, under the leadership of the world-renowned Australian sports medic John Sutton, turned their focus on resistance exercise and what impact this had on arterial blood pressure. For some time the impact of low-intensity isometric exercise was known to increase blood pressure, but no direct data were available for dynamic lifting in different muscle groups. Consequently, in 5 trained weightlifters, blood pressure was assessed, using a brachial artery catheter and pressure transducer, during upper-body and lower-body resistance exercise at high intensities, and to failure. The data were quite remarkable. Peak systolic and diastolic blood pressures were recorded in a double-leg press at 95% 1 RM to failure. For the group, the peak systolic blood pressure was 320 mm Hg and the peak diastolic pressure was 250 mm Hg. In one subject, peak data was 480/350 mm Hg. Even in a smaller muscle mass using a single arm curl, group mean blood pressure reached ca 240/180 mm Hg. The authors were intrigued, as you might expect, as to the determinants of such a large increase in systemic arterial blood pressure. Obviously, a large sympathetic drive was involved and heart rate did reach a peak of c. 170 beats-min⁻¹. However the main contributors were likely mechanical compression of large arteries in the active muscle beds. Although this could not be directly confirmed by downstream imaging studies, the compression of arteries at lower intensity isometric work has been demonstrated before. The authors also acknowledged the lack of intramuscular pressure data, but of course obtaining this information would be very tricky! The role of a Valsalva maneuver, which is commonly used in weight-training at high intensities, was investigated by a novel method of mouth expiration against a water column. While it provided a “rough” estimate of intrathoracic pressure change with a Valsalva, later authors would devise better ways of determining these pressure changes. The authors noted a significant rise in intrathoracic pressure during their “Valsalva,” which likely contributed to, but clearly did not explain all, of the arterial pressure increases during weightlifting exercise.

This is a very highly cited paper and, interestingly, a number of people have used this information to warn against the role of resistance or weight-training in at-risk groups (ie, in cardiac rehabilitation). The authors made no such assertion, as this was a study performed in young, healthy weightlifters.
Cardiac fatigue after prolonged exercise

P. S. Douglas, M. L. O'Toole, W. D. Hiller, K. Hackney, N. Reichek

Circulation. 1987;76:1206-1213

One of the most interesting juxtapositions of the late 20th century was that, on the one hand, population levels of obesity were on the rise and physical activity was declining, but at the same time the popularity of endurance and ultraendurance sports events were witnessing a phenomenal rise. While the Boston Marathon has now been run for over 100 years, it was not until 1981, for instance, that the London Marathon was first run and this is now one on many mass-participation events that witnesses 30,000+ competitors. Concomitant to this mobilization of the “weekend warrior” was the (re)birth of ultraendurance sports events for the “serious” amateur and ultra-athlete. One of the formats that caught media and lay attention was the Ironman Triathlon, which included a 2.4-mile swim, a 110-mile bike ride, and a 26.2-mile (marathon) run in its continuous activity. Of all the Ironman competitions that started in the late 20th century, the highest profile event was that completed at Kona in Hawaii, USA. This event provided not only a phenomenal test for the ultraendurance athlete in terms of distance, but often provided a significant environmental stress with temperatures as high as 40°C.

It is with this background that an intrepid group of cardiologists from the Eastern United States set off to Hawaii to collect some field data at the Ironman Triathlon. This group led by Pamela Douglas was actually going back to the original data of Saltin and Stenberg and attempting to determine if a reduction in cardiac pump function could occur after an unmitting exercise challenge. While not the first group to reassess this issue in the 1980s, the work by Douglas et al was the most complete by assessing left ventricular (LV) systolic and diastolic function (by 2-D, M-mode, and Doppler echocardiography) as well as evidence of ischemia and cardiac damage by 12-lead ECG and blood biomarker levels. Twenty-one athletes completed the Ironman between 10.5 and 16 hours and all had a post-exercise cardiac scan inside 30 minutes of completion. Key outcomes were that fractional shortening of the LV during contraction and the LV filling ratio during diastole were reduced post exercise, indicative of both systolic and diastolic “cardiac fatigue,” a term coined in this study that has stuck for later investigations. Although lacking in-event cardiac data, the authors did collect recovery data at ca 24-48 h after race completion. Functional recovery was quick, supporting the assertion of a transient fatigue. The recovery data added to the suggestion that cardiac fatigue was not wholly preload driven and thus must have an intrinsic component. Of note, the change in fractional shortening in any given individual at race end was significantly correlated to the change in LV systolic dimension (indicative of a depression in contractility) and not diastolic dimension (which is more indicative of preload). There were no ST-segment or T-wave changes indicative of ischemia, and while CK-MB was elevated, its relative presence (as a percentage of total CK) suggested a noncardiac origin.

Subsequent studies in this field have adopted more sensitive imaging tools (such as magnetic resonance imaging) and more specific biomarkers (such as cardiac troponin I), but this paper still exists as an important marker for ongoing studies of the phenomenon that is “exercise-induced cardiac fatigue.”

The 100-m final at the athletic world championship in Athens is won by Canadian Ben Johnson in a new world record time of 9.83 seconds. He is later stripped of the title after he admits to the use of performance-enhancing drugs in 1988; the inaugural Rugby Union World Cup is held in Australia and New Zealand. The All Blacks defeat France in the final 29-9; and Canadian ice hockey team The Edmonton Oilers win the Stanley Cup, defeating the Philadelphia Flyers 4 games to 3.
The upper limits of physiologic cardiac hypertrophy in highly trained athletes

A. Pelliccia, B. J. Maron, A. Spataro, M. Proschan, P. Spirito


Probably the most cited paper in sports cardiology, this seminal paper was authored by Antonio Pelliccia, a man often described as the “godfather” of sports cardiology: a fitting title given his Italian origin! While the concept of the “Athlete’s Heart” was well recognized at the time of this publication, Pelliccia and coworkers were the first to report findings in a large cohort of elite athletes from a range of sports. Between 1975 and 1990, the authors examined 947 athletes (738 men, 209 women) initially using M-Mode and Doppler echocardiography, and later 2-D images. At that time, echocardiography was a novel diagnostic tool in its infancy and, despite the images often resembling a snowstorm, cardiac ultrasound provided a noninvasive, nonradiation interrogation of cardiac structure and function. Accordingly, echocardiography provided the first practical opportunity to test the hypothesis proposed by Henschen (1899) that chronic physical training resulted in cardiac enlargement, which had only formerly been assessed using percussion and x-ray. Furthermore, Doppler echocardiography allowed the assessment of cardiac function.

As part of the establishment in 1975 of mandatory preparticipation cardiovascular screening in Italy, all athletes competing at a regional level or above are required to undertake family/symptom questionnaire, physical examination, resting 12-lead ECG, and echocardiography by a specialist in sports cardiology. To date, Italy remains the only country in the world to have mandatory preparticipation cardiovascular screening, which is the primary reason why much of the debate surrounding the “Athlete’s Heart” and preparticipation screening is centered on Italian data. Indeed, this paper is one of the first in a vast library of publications from this group. Of note, Italy is also the only country to have sports cardiology as a recognized specialty or subspecialty, elsewhere it is merely viewed as an interest or pastime. Not only did the findings from this paper present the first large cohort descriptive study of the “Athlete’s Heart,” it also provided clinically significant data in defining upper normal limits for wall thickness and internal diameters of the left ventricle in both male and female athletes. Despite 2 decades of research and major advances in echocardiographic techniques and technology since this paper, the upper normal limits defined by Pelliccia and coworkers still hold true: <12 mm female, <16 mm male, and <66 mm for left ventricular internal diameter. Of note, this paper also described the rarity of wall thickness values above the upper normal limit for the general population of <12 mm, with an incidence of 0.08% in male athletes and 0% in females.

This paper set the scene for all future research in the area of the “Athlete’s Heart” and, despite the largely white, Western European predominance of the athletes investigated in this study, provided the clinical guidelines for the differentiation of physiological and pathological left ventricular enlargement. Recently, the issue of ethnicity has been recognized as an important variable in the adaptation of the heart to chronic physical training, leading to new clinical guidelines alongside work describing upper normal limits for other cardiac structures, ie, the left atrium and right ventricle.

### 1991

Australian Miles Stewart wins the ITU Triathlon World Championships in Brisbane, Australia; Australian rugby league team the Penrith Panthers defeats the Canberra Raiders in the NSWRL Grand Final at the Sydney Football Stadium; and South Africa is readmitted to the International Cricket Council following the abolition of apartheid, playing their first international game since 1970 against India.
nomalous findings on the resting electrocardiogram (ECG) are common in athletes and generally reflect a physiological adaptation to chronic physical training that forms part of a range of cardiovascular adaptations commonly termed the “Athlete’s Heart.” While there was an understanding of the nature of ECG changes in adult athletes prior to the publication of this paper, Sharma et al were the first to report on a large cohort of elite junior athletes and the first to describe, in detail, common ECG anomalies and uncommon ECG abnormalities in athletes. Commonly observed changes occur in up to 80% of both junior and senior athletes and include: sinus bradycardia; first-degree atrioventricular (AV) block; notched QRS in V1 or incomplete right bundle-branch block; early repolarization; and isolated QRS voltage criteria for left ventricular hypertrophy. In contrast, uncommon ECG changes that are present in less than 5% of athletes include: T-wave inversions; ST-segment depression; pathological Q waves; left atrial enlargement; left axis deviation/left posterior hemiblock; right ventricular hypertrophy; complete left or right bundle-branch block; long or short QT-interval; Brugada-like early repolarization, and ventricular arrhythmias.

Results from this study provided clinically relevant information in the differentiation of physiologic and pathologic ECG changes and became the cornerstone of European guidelines. The last decade has seen a number of such studies, predominantly from European groups, which have led to the refinement of guidelines that have now been adopted by the European Society of Cardiology (ESC). The clinical importance of establishing common vs uncommon ECG changes in athletes is primarily to improve diagnostic accuracy. Reducing false positive and false negative diagnoses is of obvious importance however, it is the role of the resting ECG in preparticipation screening where greatest benefit from an improved specificity and sensitivity has been observed. High false positive numbers create unnecessary anxiety for the athlete and, what always seems to be the primary issue of concern, a high financial burden associated with downstream assessment to exclude pathology. This paper together with more contemporary work has reduced the false positive rates of preparticipation screening from 9% in early Italian studies to around 3% in more recent (British) studies, important given the 0.03% incidence of diseases associated with sudden cardiac death in young athletes!

Given the white athlete dominance of the vast majority of available literature, recent studies have begun to explore the impact of ethnicity on the resting ECG. A small number of recent studies have demonstrated ECG findings in Afro-Caribbean black athletes that would be regarded as grossly abnormal based on the white-derived norms. Repolarization abnormalities including ST-segment elevation and deep T-wave inversions in up to a third of Afro-Caribbean black athletes compared with less than 2% in white athletes. These data support the need for race-specific criteria to further enhance the care of the elite athlete.

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**1999**

Manchester United FC beats FC Bayern Munich 2-1 in the final of the UEFA Champions League; American sprinter Michael Johnson sets a new world record in the 400 meters of 43.18 seconds at the World Championships in Seville; and Moroccan middle distance runner Hicham El Guerrouj sets a new world record for the 1500 m, 3:43.13 minutes
Throughout the history of science there are moments in time when commonly held beliefs are placed under microscope. In 2004, Noakes and coworkers challenged the scientific thinking surrounding the concept of fatigue during exercise. The authors claimed their new model to be a revolutionary theory bringing into question the popular teaching that exercise performance is limited by metabolic changes in the exercising muscles, so called “peripheral fatigue.” This model predicts that exercise terminates when there is a catastrophic failure of homeostasis in the exercising muscles. Noakes and coworkers state that far from being limited by the failure of one or more physiological systems during exercise, the reality is that all physiological systems in the body, both at rest and during physical activity, are homeostatically regulated in a continuous process, specifically to prevent catastrophic failure, including irreversible physical exhaustion, widespread cell damage, or even death, as the outcome of exercise. In essence, they altered the notion of what they considered to be the scientific dogma of “peripheral fatigue” to a fatigue model coordinated by the brain, later named the “Central Governor.” The new Central Governor Model ensured that catastrophic physiological failure does not occur during normal exercise in humans.

While not presenting new data, the publication of this “revolutionary” new model had a profound impact on the scientific community by questioning the commonly held beliefs of the time and, resulted in a plethora of point, counterpoint papers providing the platform for an acrimonious debate that persists to this day. Points of contention range from the suggestion that this was not a revolutionary model, but merely an extension of the well-recognized role of the brain in the fatigue process, through to the apparent absence of empirical data to support the role of the brain in governing fatigue. Most liberal thinkers believe that there is a role for both peripheral and central factors in the control of fatigue in a feed-forward and feed-backward manner. However, this does not halt those hardliners in the physiology community from becoming entrenched in a bipolar debate that is unlikely to see a winner despite the continued expansion of data on both sides. In publishing this provocative paper, Noakes and coworkers provided one of the key components of a seminal paper: debate.

While we all believe what we know to be true, it often takes a landmark event/discovery/theory to question scientific knowledge and advance our understanding of science. All too often the scientific community becomes entrenched in dogma that few are brave enough, or well-respected enough, to question. In proposing central control over pacing during physical activity as the primary governor of fatigue Noakes and coworkers coined the phrase “the journey and not just the end point are probably the most important phenomena in exercise physiology,” and provided a much-needed platform for new debate, revolutionary or not!

In Super Bowl XXXVIII, the New England Patriots defeat the Carolina Panthers 32-29 in Houston, Texas; Greece wins the UEFA European 2004 football tournament, defeating the host nation Portugal 1-0 in the final; and US cyclist Lance Armstrong wins his sixth consecutive Tour de France.
Prior to the publication of a series of papers from this group the vast majority, if not all, of the available literature reporting structural and electrical cardiac adaptations as a consequence of chronic, intense physical training concerned male, white, and, in the main, Western European athletic populations. Indeed, for over a decade the world ran to the beat of an Italian heart! This white dominance was in direct contrast to the multiethnic world of sport, both across and within nations. Not only was there a need to describe training-induced changes in cardiac structure and function across ethnicity, there was an emerging clinical need to establish “normal” limits in Afro-Caribbean black athletes in whom there was a preponderance of anomalous findings that bordered, or crossed, the “white-derived” normal limits. The importance of differentiating physiological and pathological changes in cardiac structure and function was universally accepted as important in reducing exercise-associated sudden cardiac death in young athletes. However, following the publication of a paper that highlighted the increased incidence of sudden cardiac deaths in Afro-Caribbean black athletes, the importance of ethnic-specific “normal” limits was highlighted.

Basavarajaiah et al (2008) were the first to publish findings in elite Afro-Caribbean black athletes and describe a greater number of athletes presenting in the area of diagnostic uncertainty coined as the “gray zone.” They described the significantly greater wall thickness values observed in Afro-Caribbean black athletes and the increased incidence of deep T-wave inversions in the right precordial leads past V1, which are highly suspicious in white athletes. Of note, a ≥12-mm left ventricular (LV) wall thickness was observed in 20% of male Afro-Caribbean black athletes compared with just 4% of white athletes. Of greater clinical importance was the observation that 3% of male Afro-Caribbean black athletes had LV wall thickness values ≥15 mm, the upper-normal limit for white athletes. A number of studies, from this group and others, have examined the clinical significance of the left ventricular hypertrophy and deep T-wave inversions in Afro-Caribbean black athletes, the conclusion of which remains unknown, much as it does in white athletes. Recent studies from this group have described findings from other ethnic groups, including West Asians in whom findings are similar to that of whites.

Further studies in the series from this group have crossed the sex barrier and reported findings in Afro-Caribbean black female athletes with 3% presenting wall thickness values >11 mm (maximum 12 mm) compared with 0% in white female athletes. The abject lack of data in the literature examining the female athletes’ heart may at first sight appear somewhat sexist; however, when one considers the 9:1 male to female difference in the incidence of sudden cardiac it is clear that testosterone-based research may be more clinically relevant. That said, normal values for females are important for completeness, which requires further research.

It seems strange that not until 2008 did the cardiology literature catch up with the ethnic diversity of World sport; oversight or white-led researcher bias?

2008

Fastest 100-m hurdles wearing swim fins run by Maren Zönker of Germany in 22:35 seconds, in Cologne; longest pair of skis (534 meters), worn (and used—albeit on a slow ramble) by a total of 1043 skiers, in Örebro, Sweden; and world’s oldest table tennis player is Dorothy de Low, 97 years, who represented Australia at the XIV World Veterans Table Tennis Championships in Rio de Janeiro, Brazil.
Diverse patterns of myocardial fibrosis in lifelong, veteran endurance athletes


J Appl Physiol. 2011;110(6):1622-1626

"Exercise is good for you": a statement that has become the promotional strapline of the “exercise is medicine” lobby and one which, in general, may be true. It is clear from the enormous wealth of literature that moderate intensity, moderate duration (ca 30 minutes), moderate frequency (ca 5 times per week) exercise confers significant health benefits across the disease spectrum. In brief, this type of exercise has positive outcome in the prevention and treatment of disease and is wholly beneficial for health. Unfortunately, this important propaganda message fails to take into account the extreme end of the exercise continuum: The ultraendurance “nutter!” Defining “ultraendurance” is somewhat problematic as the definition of “endurance” exercise has changed in recent decades: consider that the marathon was only first introduced into the Olympic Games for women in 1984! What was once thought of as impossible has now become commonplace. In line with this move to increase the endurance challenge for the masses, ultraendurance was born, a distance, often in extreme environments/topography, characterized by being greater than the recognized upper limit of normal endurance racing, i.e., marathon. Aside from the musculoskeletal issues the true physiological impact associated with such endeavors and, in particular, the cardiovascular challenges had not been addressed until Wilson et al.

A large number of studies have described the acute cardiovascular response to endurance and ultraendurance exercise using echocardiography and humeral markers of cardiac damage. In general, these studies demonstrate diastolic and, when exercise is of sufficient duration, systolic dysfunction. Concomitant, but unrelated, to the observed dysfunction is the appearance of cardiac troponins (cTn), pathognomonic for cardiac damage. While the time course for cTn differs from that of myocardial infarction (exercise-induced cTn release returns to baseline within 24 hours), the clinical significance remains important. A small number of case studies, including autopsy findings, have suggested a pathological outcome of such elevations, leading to myocardial fibrosis. Until the advent of late gadolinium-enhanced cardiovascular magnetic resonance, which allows the interrogation of myocardial fibrosis premortem, this hypothesis remained just that: a hypothesis. Wilson et al were the first to report findings from a group of truly lifelong, ultraendurance runners. To highlight the ultraendurance credentials of the participants, the range of time spent in continuous training and competition was 35 to 52 years with an average of 178 marathons and 65 ultra-marathons completed in a lifetime. One athlete had run a total of 650 marathons and 257 ultramarathons, redefining the word “insane!” Findings from the study suggested 50% of participants presented with evidence of cardiac fibrosis, of which only 1 displayed a classic myocardial infarction pattern. While this paper does not prove a cause-and-effect relationship between cardiac fibrosis and ultraendurance exercise per se, it does provide an interesting springboard for further work.

While the simple advice to athletes would be to avoid ultraendurance exercise, the growing number and length of ultraendurance events means that the cardiovascular implications of such arduous exercise remain an important area for research, particularly in light of the “exercise is medicine” message that we are sending to the general population.

The 2011 Cricket World Cup is won by India. Indian captain Mahendra Singh Dhoni is man of the match; Norwegian cross-country skier Marit Bjørgen wins four gold and one silver medals at the FIS Nordic World Ski Championships in Oslo; and Mongolian sumo wrestler Hakuho Sho wins Hatsu basho. It is his eighteenth championship, and his sixth in a row.
Olympic Challenges: Performance and Perseverance

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selected by Gregory Whyte, PhD; Keith George, PhD
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