

Physiology of sport

The elite athlete represents the extreme of the human gene pool, where genetic endowment is developed by an intensive training programme. Sport encompasses many different activities, calling for different physical and mental attributes. Understanding the physiology of exercise provides insights into normal physiological function.

For those used to dealing with the 'normal' population, encountering an elite athlete offers a different perspective on human physiology. The elite endurance athlete may have a resting heart rate of less than 30 beats per minute and a maximum cardiac output of more than 40 litres/min. Many late middle aged people are disabled by the consequences of inactivity, but their active contemporaries achieve feats of strength, speed or endurance that are little diminished from those of their youth.

Most factors that contribute to athletic success are determined by the individual's genetic inheritance. Of these, some are readily amenable to change in response to an appropriate training stimulus, while others are relatively immutable. Indeed, the ability to respond to a given training stimulus with an improvement in performance – so-called 'trainability' – may itself be genetically determined. Intrinsic characteristics, including psychological attributes, training load and nutritional practices, will influence all aspects of both training and competition. The external environment can also influence athletic performance, with factors such as heat and altitude having effects on different events and also affecting different athletes to varying degrees. At lower levels of performance, participation is open to all, and most people compete in sport without expectation of success, if success is defined by winning.

The major factors contributing to performance can be listed as: size, shape, strength, speed, stamina, suppleness, skill, and (p)sychology. All bodily organs and tissues are involved to some degree in every sporting event, but different events place very different demands on these tissues. This review summarizes the main characteristics of the different tissues that distinguish the successful athlete.

Strength

Human skeletal muscle is a heterogeneous tissue, consisting of different fibre types with varying contractile characteristics and differing metabolic profiles (Table 1). Most muscles contain a mixture of different fibre types, allowing them to meet the varying demands placed upon them. Two key elements dictate the contractile and metabolic responses to exercise: characteristics of the individual muscle fibres and the pattern of fibre recruitment. Only as many fibres are activated as are needed to carry out a given task. In normal day-to-day activities, only type 1 (slow twitch, fatigue resistant) fibres are

active, but high forces require activation of type 2 (fast contracting, easily fatiguable) fibres.

Since muscle fibre adapts to a training stimulus only if it is activated, a large part of the muscle is not stimulated by low intensity, short duration activities. Increasing training intensity will activate more fibres and induce adaptive responses: likewise, very long exposures to submaximal stimuli will result in fatigue of fibres with the lowest activation thresholds and recruitment of others to replace them.

Training has little effect on the contractile properties of muscle, but can dramatically alter functional capacity and metabolic characteristics. Endurance training is characterized by an increased mitochondrial content and an increased capacity for oxidative metabolism, especially oxidation of fat (Snow, 2003). Strength training, in contrast, increases the muscle content of the contractile proteins, actin and myosin, leading to increases in muscle size and strength without any change in the oxidative capacity of the muscles. These adaptations occur as a result of selective stimulation of protein breakdown and synthesis within the muscle fibres. This in turn depends on the application of an appropriate training stimulus and on the metabolic and hormonal environment within the cell.

Maximum strength is little affected by muscle fibre composition, as major fibre types generate similar maximum forces. Where fast movements have to be performed at high forces, however, type 2 muscle fibres perform better than type 1 fibres (Snow, 2003). Most strength athletes compete in weight categories, so training aims to increase strength without increasing muscle mass. Trained athletes are better able to maximally activate all muscle fibres.

Speed

Moving the body mass at speed requires the ability to generate high power outputs, and all elite sprinters have a high proportion of type 2 muscle fibres. The high energy

Table 1. Characteristics of the main skeletal muscle fibre types in humans

	Type 1	Type 2a	Type 2x
Contractile speed	Slow	Fast	Fast
Glycolytic capacity	Low	High	High
Capillary density	High	Moderate	Low
Oxidative capacity	High	High	Low
Recruitment threshold	Low	Moderate	High
Fatigue resistance	High	Moderate	Low

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demands of power events are met largely by anaerobic metabolism, in part because of the delay in full activation of the aerobic processes, and in part because aerobic energy supply cannot provide energy at a sufficient rate. All the immediate energy needs of muscle are met by the energy released when adenosine triphosphate (ATP) is hydrolysed to form adenosine diphosphate (ADP) and inorganic phosphate. The ATP content of muscle is very small and it must be regenerated as fast as it is synthesized to maintain muscle function. The muscle contains a four times higher concentration of creatine phosphate than ATP, and a high energy phosphate group can be transferred to ADP to regenerate ATP. This reaction, catalysed by creatine kinase, is extremely rapid, and makes a major contribution to energy supply in an activity such as a 100 m sprint lasting about 10 s. Power athletes are aware that ingestion of large amounts of creatine (10–20 g per day for a few days, compared to the normal dietary content of about 1 g) can increase muscle creatine phosphate stores and improve power output (Greenhaff, 2000). Creatine supplementation does not contravene doping regulations and is not known to be harmful to health.

Within a second or two of exercise, substrate level phosphorylation of ATP is activated, allowing glycolysis (degradation of glucose derived from the circulation) and glycogenolysis (degradation of glycogen stored within muscle cells) to contribute to ATP regeneration. The sequential degradation of glucose to pyruvate allows synthesis of three molecules of ATP for each molecule of glucose degraded, or four ATPs if glycogen is the starting point. This involves the conversion of the cofactor nicotinamide adenine dinucleotide (NAD) to its reduced form (NADH). Because the muscle content of NAD is very small, NADH is converted back to NAD by transfer of hydrogen atoms to pyruvate, resulting in the formation of lactate. Free hydrogen ions are also generated, resulting in a fall in intracellular pH when glycolysis proceeds at high rates (Figure 1). The formation of lactate is thus essential for the maintenance of high power outputs but is ultimately limited by the increasing muscle acidity, which interferes with the contractile process itself. The significance of this is evident from the improvements in performance of short duration, high intensity events that occur when alkalising agents such as sodium bicarbonate or sodium citrate are ingested before exercise (McNaughton, 2000).

Stamina

Stamina is the ability to sustain a given power output for a sustained period of time. This might refer to the ability of the sprinter to last for 400 metres without loss of speed over the last few metres of the race, or the ability of the endurance athlete to complete a marathon or triathlon. In any event lasting more than a few seconds, oxidative metabolism makes a major contribution to energy supply, and when high intensity exercise lasts more than 100 s, aerobic metabolism provides more than 50% of the total energy cost of the task (Maughan and Gleeson, 2004).

A high capacity for energy supply by aerobic metabolism is one of the distinguishing characteristics of the endurance athlete, and this is normally measured as the maximum oxygen uptake (VO_2max). The highest values for VO_2max are normally observed in elite middle distance athletes, whose events last not more than a few minutes. The endurance athlete can compensate to some extent by increasing the fraction of the maximum that is sustained, but the middle distance athlete needs a very high absolute power output, and this means a high anaerobic capacity and a high aerobic capacity.

There was much debate at one time as to whether VO_2max is limited by oxygen supply to the muscles or by the ability of the muscles to use the oxygen delivered to them. It seems clear that oxygen supply is the limiting factor in healthy individuals when large muscle groups are involved (Saltin and Strange, 1992). The metabolic properties of the muscles themselves should not be ignored, however, as success in endurance events requires both a high VO_2max and the ability to sustain a high fraction of this value for prolonged periods. The limitation to oxygen supply to the muscles may lie at any one of several parts of the pathway from oxygen uptake in the lung to oxygen supply to the site of use in the mitochondria (Figure 2).

Cardiorespiratory function

It is generally accepted that the lung does not limit oxygen uptake in normal individuals exercising at sea level, but there is some evidence to challenge that view. Boutellier et al (1992) showed that a few weeks of specific training of the respiratory muscles appeared to result in significant improvements in exercise performance. At altitude, where the oxygen content of the inspired air is reduced, desaturation of arterial blood is commonly observed, suggesting that insufficient oxygen is available to the blood passing through the lungs to allow complete saturation of haemoglobin (Hb). There is also evidence, however, that highly trained athletes show arterial desaturation during maximal exercise at sea level (Powers et al, 1988); in these athletes, breathing hyperoxic mixtures can increase VO_2max , while no effect on VO_2max is seen in less fit subjects (Powers et



Figure 1. In the later stages of a 400 m sprint, metabolic acidosis is a major cause of fatigue.

al, 1989). This may be explained by the relatively limited adaptations in lung function that are possible: when all other parts of the oxygen transport chain are increased, the lung may become the limiting factor.

The athlete's heart has attracted much attention from cardiologists over the years. It is now recognized that the extreme left ventricular hypertrophy, and consequent bradycardia, of the elite endurance athlete is not generally harmful to health. The elite endurance athlete shows a number of characteristics that allow high rates of oxygen delivery to working muscles (Table 2). The ability to achieve a high cardiac output is probably the most important attribute of the elite endurance athlete. As maximum heart rate is not affected by training, and, if anything, is slightly decreased, this must be the result of a high stroke volume. There is good evidence of a linear relationship between VO_2 max and maximum cardiac output. The relative slowness of changes in VO_2 max in response to training or detraining is largely a result of the time course of changes in cardiac dimensions. One easily observable consequence of a high stroke volume is the endurance athlete's low resting heart rate: the typical resting cardiac output of about 5 litres/min is achieved by the sedentary

individual with a heart rate of about 70 b/min and a stroke volume of about 70 ml, but the best endurance athletes may have a resting heart rate of 30–40 b/min, indicating a stroke volume at rest of about 120–150 ml.

Oxygen is transported in the circulation bound to Hb within the red blood cells. Each gram of Hb can bind about 1.34 ml of oxygen, so a high blood Hb concentration means an increased oxygen content of each litre of arterial blood. In response to endurance training, the total amount of Hb in the circulation increases, but there is usually a disproportionate expansion of the plasma volume, leading to a reduction in the concentration of Hb in the blood. Nonetheless, VO_2 max is normally seen to be proportional to the circulating Hb concentration. Infusion of red blood cells to elevate the Hb concentration therefore results in an increase in VO_2 max. Although most of the effective methods of increasing Hb concentration, such as reinfusion of homologous or autologous red cells and the administration of recombinant erythropoietin, are prohibited by the doping regulations, they have nonetheless been used by many athletes. Altitude training, which is less effective but is not prohibited, is also widely practised, and many athletes adopt a strategy of training at sea level but living at altitude. This is achieved by travelling between venues at different altitudes, or by using artificial environments that provide either normobaric or hypobaric hypoxia (Levine and Stray-Gundersen, 1992).

Within the skeletal muscles, there is an increase in capillary density in response to endurance training. This decreases the diffusion distance for oxygen from the capillary to the mitochondria. Type 1 muscle fibres are generally smaller than type 2 fibres and have a higher capillary: fibre ratio, so are better suited to aerobic metabolism.



Figure 2. The endurance athlete is characterized by an effective cardiovascular system and a high capacity for oxidative metabolism.

Table 2. Typical cardiovascular dimensions of 'normal' adult males and of the elite endurance athlete

	Normal	Athlete
Resting heart rate (b/min)	70	30–40
Maximum heart rate (b/min)	220 minus age	220 minus age
Resting stroke volume (ml)	70–75	120–150
Maximum exercise stroke volume (ml)	100	200
Resting cardiac output (litres/min)	5	5
Maximum exercise cardiac output (litres/min)	20	40

Metabolic limitations and adaptations

All muscular activity requires input of energy to the working muscles, made available by the hydrolysis of ATP within the cells. The challenge to the muscle is to regenerate ATP as fast as it is being hydrolysed. At rest, the energy demand is low, and is met by oxidative catabolism of carbohydrate and fatty acids, requiring an oxygen consumption of about 4 ml/kg body mass, or about 250 ml for the average individual. Increasing effort involves recruitment of more muscle fibres and requires an increasing rate of energy supply. Fibres with a high oxidative capacity, and especially a high capacity for fat oxidation are first to be recruited, but as more fibres are needed, those that rely more on oxidative degradation of carbohydrate become involved. A point is reached where the fibres recruited are those that can break glycogen down to pyruvate faster than the pyruvate can enter the tricarboxylic acid cycle (TCA or Krebs cycle) for oxidative metabolism. At this point lactate begins to accumulate. In high intensity efforts, such as those of a sprinter leaving the starting blocks, the energy demand in the muscles can increase to many times the resting level, and all muscle fibres are activated. The energy demand is far in excess of the maxi-

imum rate of oxidative energy supply, and energy supply is met by catabolism of creatine phosphate, high rates of anaerobic glycolysis, and high rates of oxidative degradation of glycogen (Maughan and Gleeson, 2004).

Muscles have a limited supply of carbohydrate stored as glycogen (about 300–400 g, depending on muscle mass, prior diet and exercise) with a further 80–100 g available from liver glycogen. This can be broken down at high rates (about 3–4 g/min) in endurance exercise such as marathon running. When the glycogen store is reduced to a critical level, the running speed must be reduced to a level that can be met mainly by fat metabolism, with a small added contribution of blood glucose generated by gluconeogenesis in the liver or from ingested carbohydrates.

The role of the CNS

Although we may feel that fatigue is a phenomenon of the muscular or cardiovascular systems, it has long been recognized that this may not be so. In 1919, Bainbridge wrote that 'It has long been recognised that the main seat of fatigue after muscular exercise is the central nervous system. Mosso long ago stated that "nervous fatigue is the preponderating phenomenon and muscular fatigue is also at bottom an exhaustion of the nervous system". There appear, however, to be two types of fatigue, one arising entirely within the central nervous system, the other in which fatigue of the muscles themselves is super-added to that of the nervous system'. This remarkable insight appears to have been ignored by physiologists for almost a century, but has now been resurrected.

It is clear that performance can be altered by drugs that affect the CNS, including stimulants and narcotics, and these have been widely abused in sport, leading to their inclusion on the list of prohibited substances (World Anti-Doping Agency, 2007). A 'central fatigue hypothesis' was proposed by Newsholme et al (1987) to provide a biochemical explanation for the fatigue that accompanies prolonged exercise. This theory postulated that increased availability to tryptophan, the metabolic precursor of the neurotransmitter serotonin, would lead to increased sensations of fatigue. This has not been entirely supported by subsequent studies, but selective serotonin-reuptake inhibitors, widely used to treat depression, may alter exercise performance (Wilson and Maughan, 1992). Again the evidence is mixed. More recently, there is evidence that drugs affecting dopaminergic neurotransmission may alter performance, especially when exercise is performed in the heat (Watson et al, 2005).

Complex sports activities

Many sports, especially team games, require an ability to accomplish multiple short sprints at high speed and a highly developed endurance capacity. These activities must also be performed while maintaining a high level of fine motor control to allow precise passing of the ball as well as shooting and heading skills in a game such as football (soccer). It has been estimated that the average

elite football player covers something between about 10 and 13 km in a 90-minute game; included in that distance are about 150–250 brief, intense actions (Bangsbo et al, 2006). This complexity imposes many demands on the player and has, until recently, deterred most exercise physiologists from studying these activities. Although the cardiovascular and metabolic demands are now well characterized, an understanding of the attributes that determine the best players' skills remains elusive. **BJHM**

Conflict of interest: none.

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KEY POINTS

- The elite athlete often lies at the extremes of the human gene pool. Genetic endowment is enhanced by intensive training.
- Sprinters are characterized by high proportions of type 2 muscle fibres and a high capacity for anaerobic metabolism.
- Endurance athletes have a highly developed cardiovascular system, both central and peripheral, and a high capacity for aerobic metabolism.
- Adaptations to training are highly specific to the stimulus applied and proportional to the training load (intensity, duration and frequency).
- Pharmacological interventions that affect CNS function can influence exercise performance, so it is not clear that there is a peripheral limitation to exercise capacity.