Aerobic energy systems

Mike Murray explains what you need to know about energy transfer during long-duration, low-intensity exercise

EXAM LINKS

AQA Energy systems.

Edexcel Energy systems: fatigue and recovery.

OCR Energy for exercise.

During long-duration, low-intensity exercise such as walking or jogging, the body will use the aerobic system to resynthesise the adenosine triphosphate (ATP) needed to provide energy for muscle contractions.

In the presence of oxygen, this system breaks down glucose into carbon dioxide and water, which is much more efficient than anaerobic systems. The complete oxidation of glucose can produce up to 38 molecules of ATP.

The aerobic system

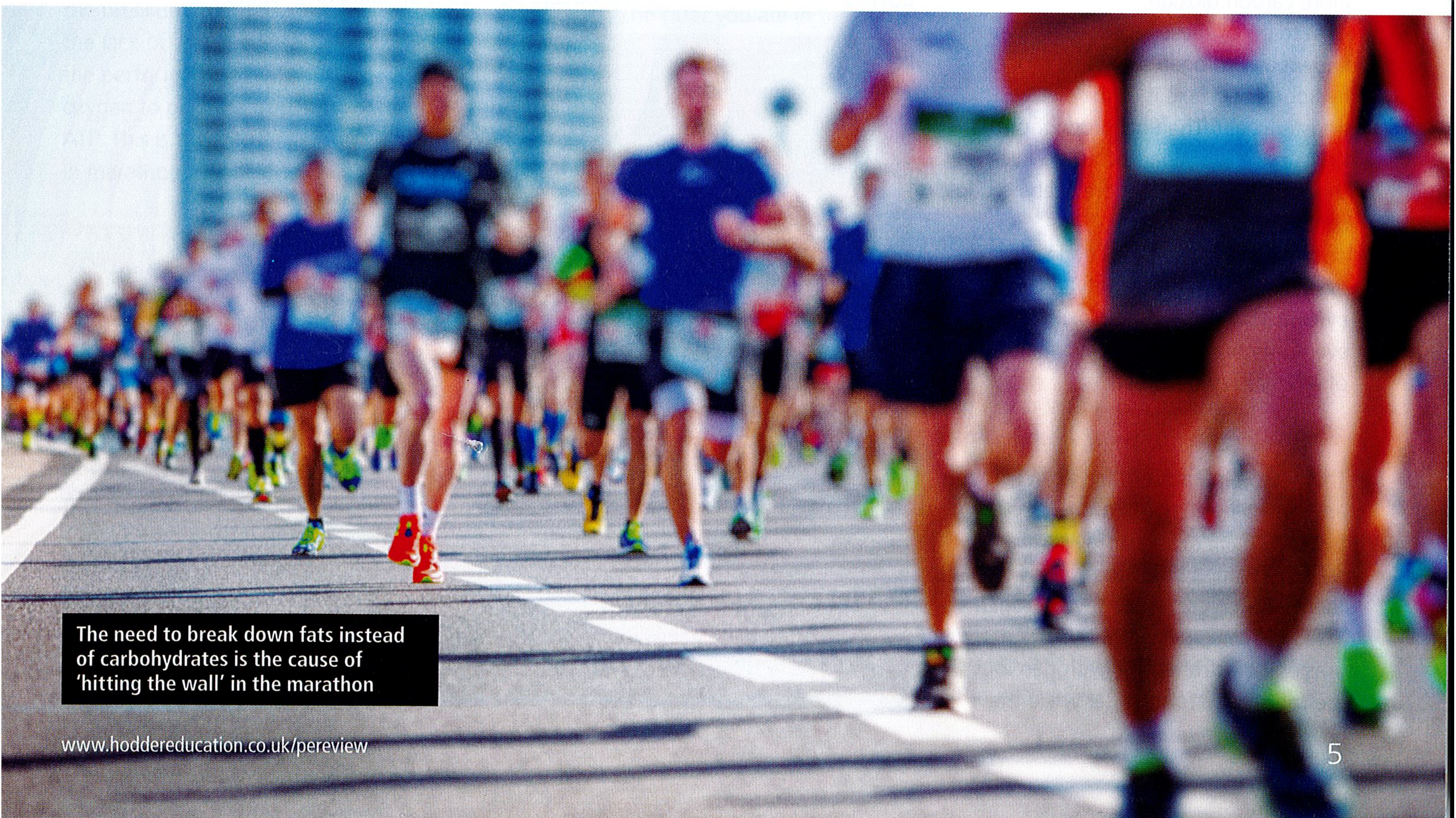
The aerobic system has three stages (see pp. 16–17):

Glycolysis

The glycolysis stage is anaerobic and takes place in the sarcoplasm of a muscle cell. It involves the breakdown of glucose through a series of ten enzyme-controlled reactions, into two molecules of pyruvic acid. Stored muscle glycogen can be broken down to glucose. For every molecule of glucose undergoing glycolysis, two molecules of ATP are resynthesised.

The Krebs cycle

At the end of glycolysis, the pyruvic acid moves from the sarcoplasm into the mitochondria (Figure 1), where the rest of the aerobic processes occur. Inside the mitochondria, each pyruvic acid molecule is broken down to form an acetyl group, releasing carbon dioxide. The acetyl group combines with coenzyme A and is then carried into the Krebs cycle as acetyl coenzyme A. This process is known as the *link reaction*.



The need to break down fats instead of carbohydrates is the cause of 'hitting the wall' in the marathon

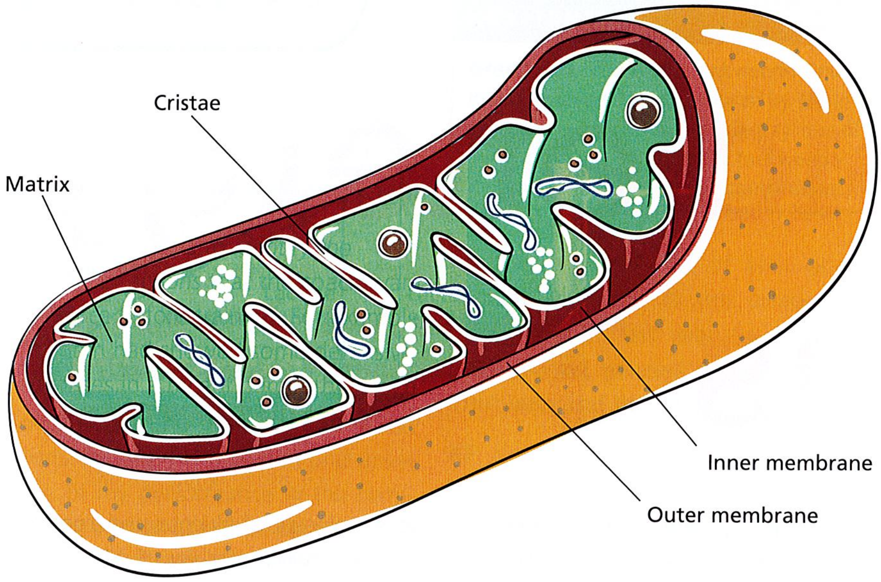


Figure 1 Structure of a mitochondrion

The two acetyl groups leave coenzyme A and diffuse into the matrix of the mitochondria. A complex cycle of reactions occurs. Each acetyl group combines with oxaloacetic acid, to form citric acid. The citric acid then undergoes a series of ten enzyme-controlled reactions. Enough energy is released in these reactions to synthesise one molecule of ATP for each acetyl molecule involved. Also, the carbon in the acetyl groups is oxidised to form more carbon dioxide.

In many of these reactions, enzymes known as hydrogen acceptors pick up hydrogen and take it to the electron transfer chain. The final product of the Krebs cycle is oxaloacetic acid, which is then ready to pick up more acetyl groups, continuing the cycle.

In summary, the Krebs cycle uses oxygen to convert the acetyl group from pyruvic acid into carbon dioxide, which is eventually breathed out. Two molecules of ATP are resynthesised.

Electron transfer chain

The hydrogen atoms in the acetyl group are carried by enzymes to the electron transfer chain. In the cristae of the mitochondria, a series of proteins transfer the hydrogen carried by the hydrogen acceptors and split it into hydrogen ions and electrons. The hydrogen ions are oxidised to form water while the hydrogen electrons provide the energy to resynthesise ATP. Throughout this process 34 molecules of ATP are formed.

In total, the aerobic breakdown of glucose produces carbon dioxide and water as waste products and resynthesises 38 molecules of ATP: two from glycolysis, two from the Krebs cycle and 34 from the electron transfer chain.

Fats and proteins

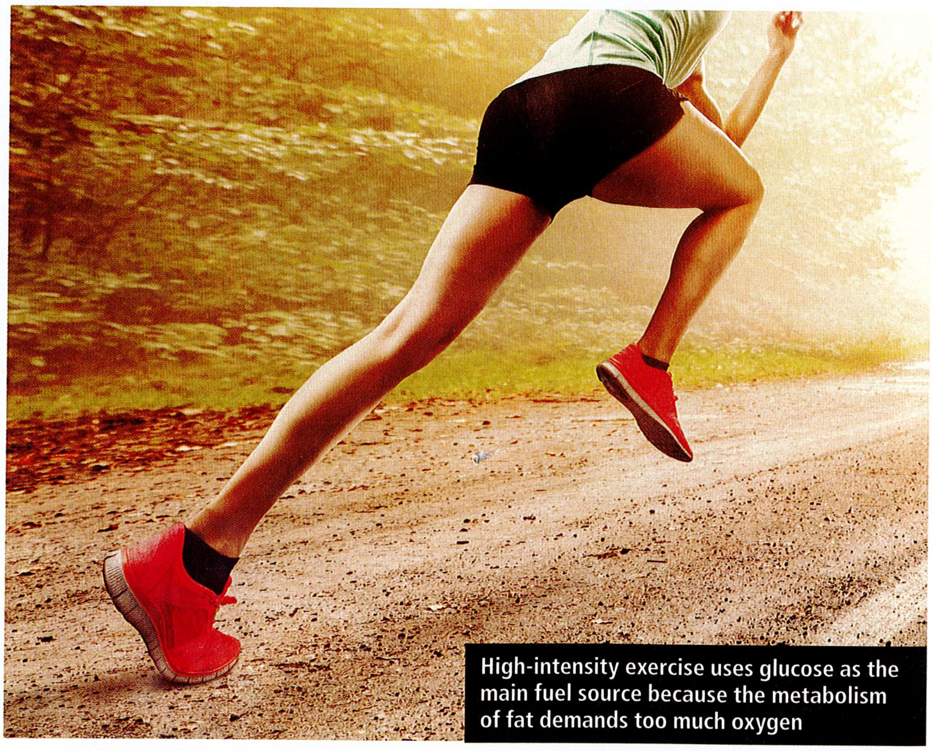
Fats and proteins can also be broken down aerobically. The various products of fat and protein metabolism are reduced to acetyl coenzyme A, which then enters the Krebs cycle.

Generally, the body uses fats and carbohydrates for energy and prefers not to use protein. At rest, approximately half of our energy requirements come from fat metabolism. During exercise, the reliance on fats reduces dramatically in explosive sports, but in endurance events the mixed use of fats and carbohydrates becomes important. The 'mix' of these food fuels depends upon the intensity and the duration of the exercise, the athlete's level of conditioning, and the athlete's diet and nutritional status.

All fat in our diet is primarily carried in the blood as 'free' fatty acids. The first stage of fat breakdown 'prepares' the fatty acid for entry into the mitochondria. This preparation is called *beta oxidation*. Once in the mitochondria, the final result is the same as for glucose: after the Krebs cycle and the electron transfer chain, water and carbon dioxide are produced, and energy is liberated for the resynthesis of ATP.

Breaking down fats

Fats cannot be broken down anaerobically. The use of fats for energy relies on adequate supplies of oxygen.



High-intensity exercise uses glucose as the main fuel source because the metabolism of fat demands too much oxygen

If exercise is aerobic, involving slow continuous activity (lasting at least an hour), then fat can be the main substrate used, and the majority of energy is derived from fats.

Fat breakdown requires more oxygen than is needed for glucose breakdown. Therefore, when oxygen supply becomes a limiting factor, as occurs during intensive exercise, fat use is restricted. Exercise at high intensity uses glucose as the main fuel source because the metabolism of fat demands too much oxygen.

Appropriate training increases the body's ability to get oxygen to the mitochondria in muscles, and thus increases the ability to use fat for energy. Endurance training increases the body's ability to release free fatty acids from their fat stores, and this is one of the reasons that trained performers can use more fats for energy than the untrained.

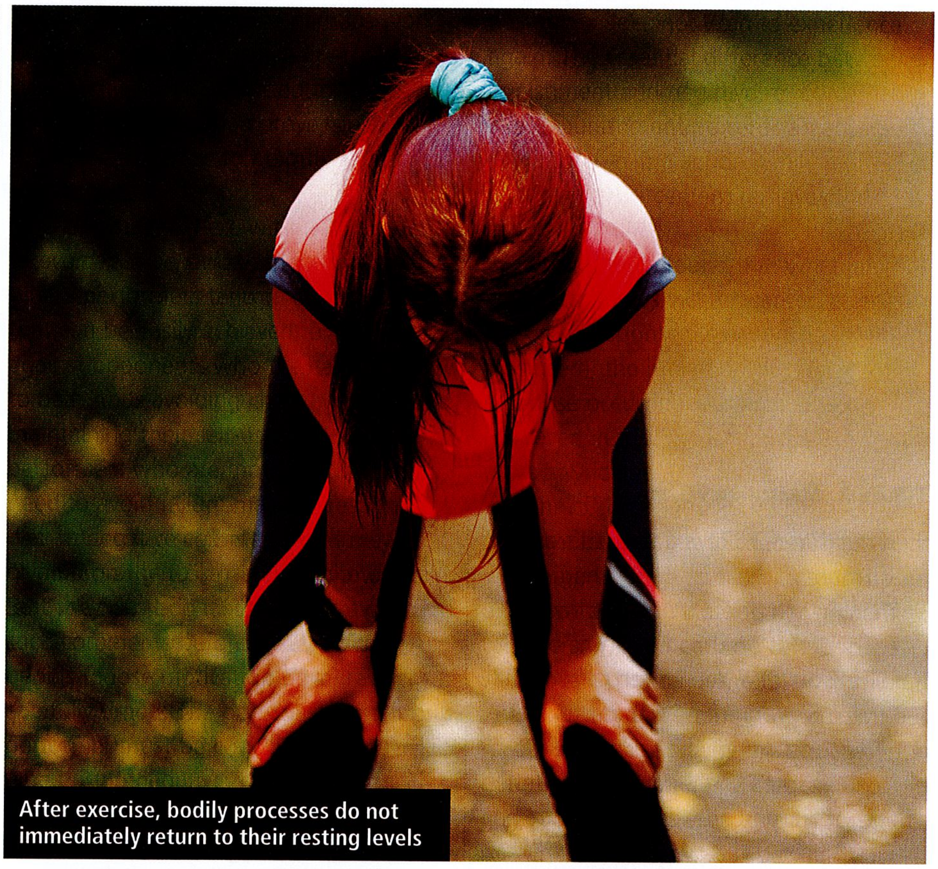
The breakdown of fats requires the presence of carbohydrates, because without carbohydrates, the Krebs cycle will not operate. Therefore, if energy demand remains high during prolonged, continuous exercise, carbohydrate (glycogen) stores become depleted, and the breakdown of fats starts because of the lack of carbohydrates. This will cause the performer to slow, as fat needs more oxygen to produce the same amounts of ATP. This is the cause of 'hitting the wall' in marathon running.

Oxygen and exercise

VO_2 max

When we exercise, we take in oxygen to supply muscles so that they can release energy aerobically. If the level of exercise intensity increases, so does the level of oxygen uptake. This relationship continues until, during extreme exercise, we reach a level of maximum oxygen consumption (VO_2 max). Maximum oxygen consumption is therefore defined as:

> The greatest amount of oxygen uptake that an individual can achieve during exercise.



After exercise, bodily processes do not immediately return to their resting levels

VO_2 max is usually regarded as an accurate indication of an athlete's aerobic fitness or stamina, since it directly estimates how much oxygen the athlete has available for aerobic energy production. VO_2 max varies with training and fitness. The fitter you are in terms of aerobic fitness, the higher your VO_2 max.

When you exercise, you tend to work at a level of oxygen consumption that is some percentage of your VO_2 max, the value being dependent on your level of fitness and the intensity of the exercise.

Lactate threshold

The concept of lactate threshold is linked to VO_2 max. The harder you exercise, the more likely you are to generate lactic acid as a waste product of anaerobic metabolism.

The lactic acid diffuses into your blood. When levels of lactic acid become 2 millimoles of lactate per litre of blood above normal resting levels, the lactate threshold is reached. *Onset of blood lactate accumulation (OBLA)* is just a different way of measuring the same principle (increasing blood lactic acid levels). OBLA is when levels of lactic acid reach 4 millimoles of lactic acid per litre of blood.

Performers must control the intensity of their exercise so as not to accumulate too much lactic acid within their blood, while at the same time working at a level close to their VO_2 max. The fitter the athlete, the higher their VO_2 max, and the closer to their VO_2 max that their lactate threshold occurs. Elite performers will be able to exercise at a lactate threshold that is 90% of their VO_2 max. Less fit individuals may have to be satisfied with a workload nearer 60–70% of their VO_2 max.

Aerobic capacity

During a treadmill run, or any static aerobic activity where the workload can be increased every 3 minutes or so, oxygen consumption will rise during each stage of the test until the runner is unable to continue due to exhaustion. At this point in time, the runner is said to have reached his or her *aerobic capacity*. The amount of oxygen being consumed in these final few seconds of exercise is

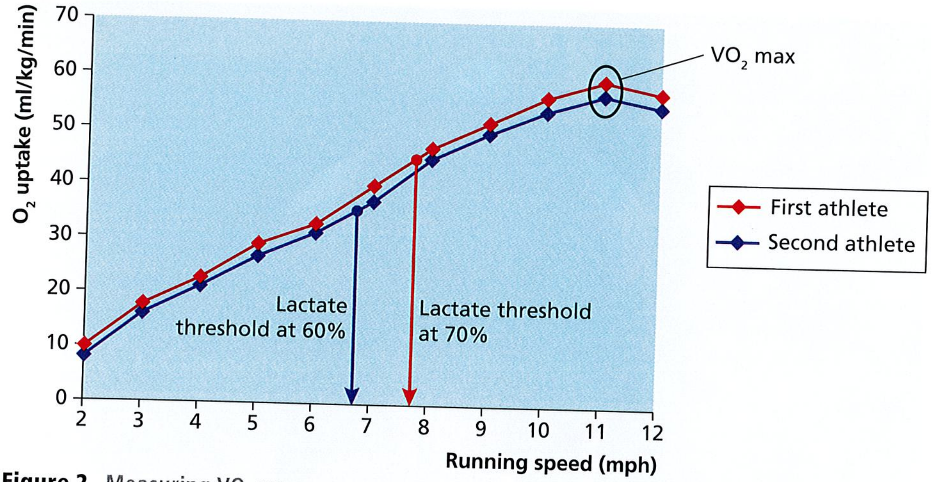


Figure 2 Measuring VO_2 max

then measured, to give a value for VO_2 max (Figure 2).

Training can affect VO_2 max. The effect depends on the intensity of the training workload, the length of time that the training programme is undertaken, and the suitability of the performer to become adapted to training. Usually, the effects of training are to increase VO_2 max by 5–20%. The main causes of these increases are changes to the blood, heart and muscles.

Oxygen deficit

Whenever we suddenly start to exercise, it takes some time before our oxygen transport system (heart, lungs and blood) are fully efficient. This delay in getting our oxygen consumption high enough to match our energy demands at the beginning of exercise is called the *oxygen deficit*. During this period of time, some of the energy we require has to be supplied by our anaerobic systems. This oxygen deficit occurs during all types of exercise, both exercises at maximum intensity such as sprinting and sub-maximal exercises such as a slow jog.

Physiologists tend to compare these two levels of exercise intensity and therefore describe two types of oxygen deficit:

- The **sub-maximal oxygen deficit** occurs during low-intensity exercise.
- The **maximal oxygen deficit** (also called the maximal accumulated oxygen deficit) occurs during maximum-intensity exercise.

Maximal oxygen deficit is larger than sub-maximal oxygen deficit because there is a greater demand for anaerobic energy supply at the beginning of exercise (Figure 3). Maximal oxygen deficit can be measured in the laboratory and is often used as an indicator of maximal anaerobic capacity.

Oxygen and recovery

After exercise, bodily processes do not immediately return to their resting levels, especially if the exercise has been stressful. During recovery, our energy demand is much lower than while we are exercising, but our oxygen demand is still quite high for a period of time, the length of which depends on the intensity of exercise.

This higher than resting level of oxygen consumption used to be called our oxygen debt, but is now more properly called *excess post-exercise oxygen consumption (EPOC)* (Figure 4).

EPOC

Because we use different energy systems for exercise (anaerobic and aerobic), there are two components to EPOC: the *fast (alactacid) component* and the *slow (lactacid) component*.

The fast component is concerned with getting the anaerobic phosphocreatine energy system back to normal. During

> **EXAM-STYLE QUESTIONS**
>
> 1 Analyse the use of the Krebs cycle during a team game such as basketball or netball. (8 marks)
>
> 2 Why would a 100m sprinter not use fats as an energy source during their race? (3 marks)
>
> 3 At the end of a 1500m race an athlete will be out of breath and will continue to breathe heavily even though they have finished the race and come to a complete rest. Explain why this breathlessness occurs. (4 marks)
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> Check your answers at www.hoddereducation.co.uk/pereviewextras

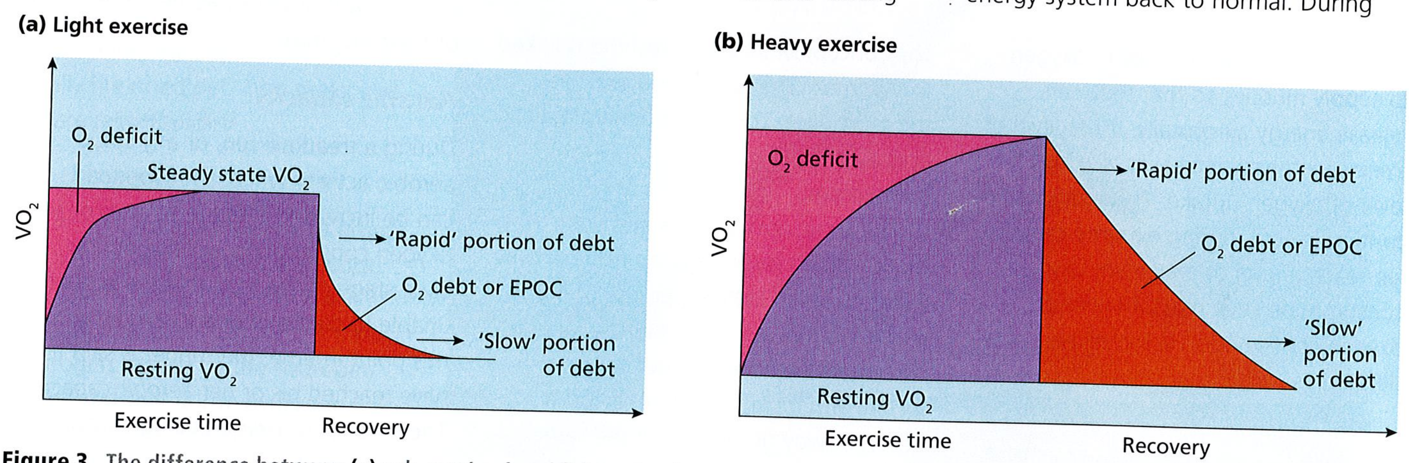

Figure 3 The difference between **(a)** sub-maximal and **(b)** maximal oxygen deficit

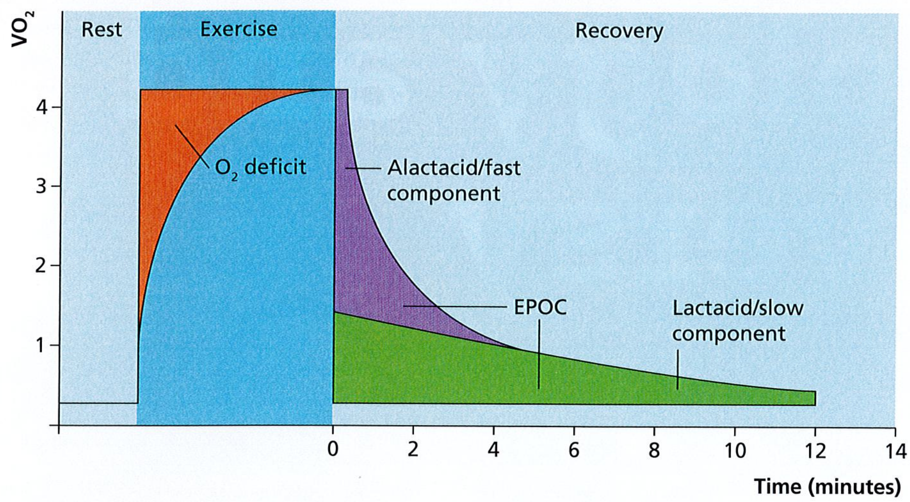

Figure 4 The process of excess post-exercise oxygen consumption (EPOC)

the fast component, which lasts no more than a few minutes, muscle phosphocreatine stores are built up to resting levels, thus we talk about *phosphocreatine restoration*. At the same time, oxygen stores in the muscles are restored, so we talk about re-saturating myoglobin with oxygen. This component is replaced within a few seconds if exercise is highly aerobic.

During strenuous exercise, we produce lactic acid. Removing this and returning the body systems to normal following strenuous exercise may take several hours. This is the slow component of EPOC. This slower phase of the recovery process is also used for other processes apart from removing lactic acid, such as supplying extra oxygen to the breathing and heart muscles that are still recovering.

Lactic acid is mainly removed from the blood by its conversion back into pyruvic acid and subsequent oxidation to carbon dioxide and water in mitochondria, which resynthesises ATP. This occurs in muscles that have sufficient oxygen, or can happen in other organs where oxygen is available, such as the liver, which can also convert the pyruvic acid into glucose for later use in respiration. A negligible amount of lactic acid is excreted in urine and sweat.

In exams

You might be expecting an extended question on aerobic energy systems in the summer exams. I think it is likely that any question on this topic would need to reduce its knowledge (AO1) content, and is therefore more likely to be a question on glycolysis, the Krebs cycle or the electron transfer chain, as show in the 'Exam-style questions' box.

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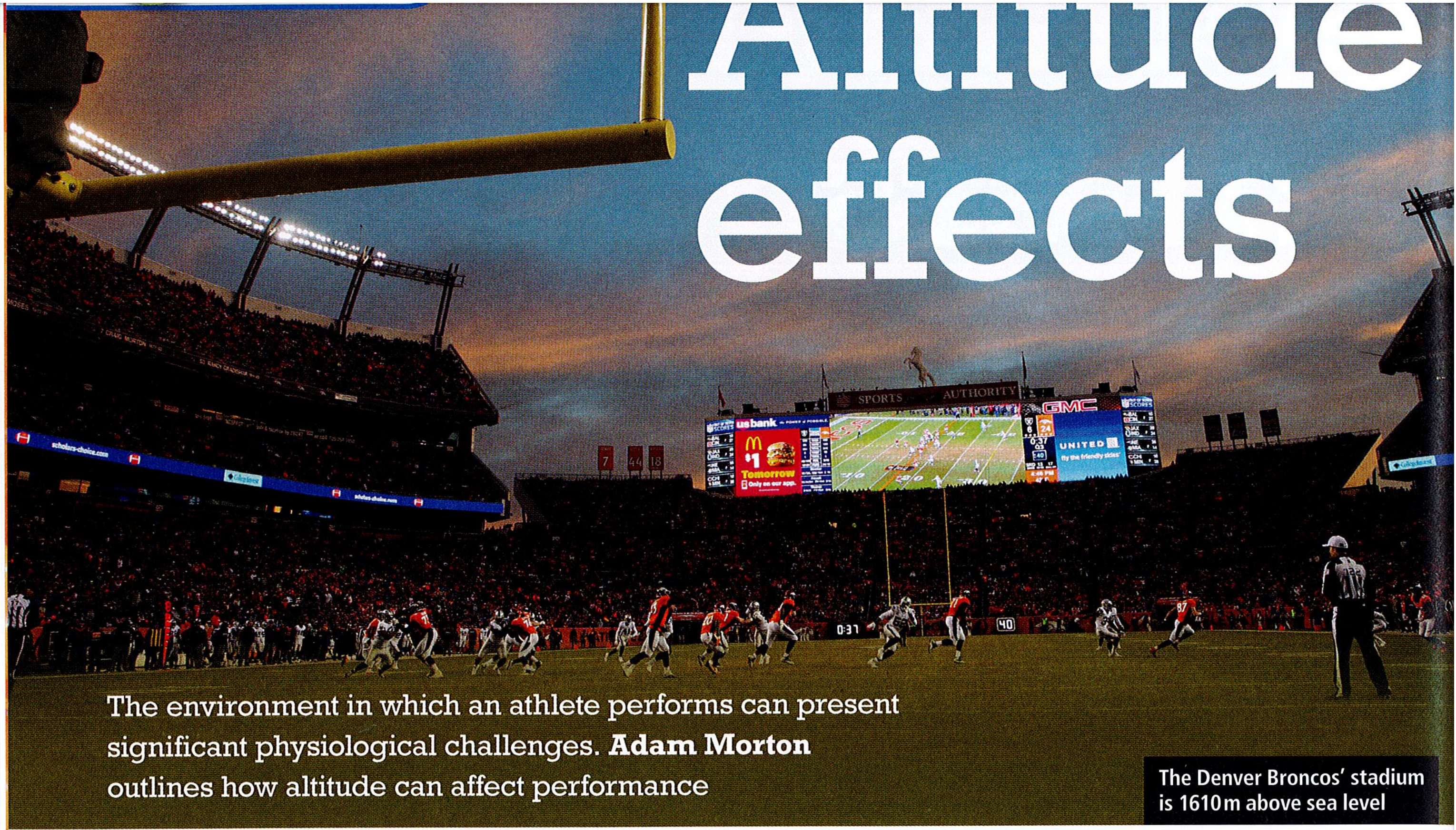
Altitude effects



The environment in which an athlete performs can present significant physiological challenges. **Adam Morton** outlines how altitude can affect performance

The Denver Broncos' stadium is 1610 m above sea level

> **EXAM LINKS**
>
> **AQA**, **Edexcel** and **OCR** all require an understanding of the physiological effects of altitude, and altitude training as a method of preparing for performance.

Athletic performance in many sports is affected by altitude. In track and field, altitudes as low as 150 m above sea level have been shown to impair performance in middle- and long-distance running events. Conversely, higher altitudes can improve performances in events such as the 100 m and long jump. In this 'Exam focus' we will look at the problems posed by altitude, how the body responds in the short and long term (acclimatisation) and some of the effects of altitude on sporting performance.

Altitude effects

You are probably familiar with images of mountaineers using supplementary oxygen when climbing some of the world's tallest peaks. While few people perform at such heights, the effect of altitude on performance is a significant consideration for many athletes and their coaches. Understanding the effects of altitude on the cardiovascular and respiratory systems in the short and long term is an important consideration for those looking to optimise sporting performance, both at high altitude and at sea level.

The difficulty with performing at high altitude is the reduced availability of oxygen in the atmosphere. This leads to **hypoxia**, a lack of oxygen in the body, and impairs the body's ability to produce energy aerobically. Performance in aerobic events is negatively affected as a result.

Atmospheric pressure and the density of air both have an inverse relationship with altitude. As height above sea level increases, atmospheric pressure and density decrease, and vice versa.

Partial pressure of oxygen

Atmospheric pressure (also referred to as barometric pressure) is measured in millimetres of mercury (mmHg) (Box 1).

> **KEY TERM**
>
> **Hypoxia** An insufficient supply of oxygen to the body.

While changes in atmospheric pressure do not affect the concentration of the different gases that constitute air (i.e. oxygen still makes up 21%), the lower density of air at higher altitudes does reduce the total amount of oxygen present. Consequently, with less oxygen molecules per unit of volume (e.g. metres cubed, m^3), oxygen molecules exert less force per unit of volume, referred to as a lower *partial pressure of oxygen* (ppO_2).

A lower partial pressure of oxygen leads to a reduced diffusion gradient between the air in the alveoli and blood in the network of alveolar capillaries. This reduces the rate of diffusion of oxygen across the alveolar and capillary membranes into the blood, leading to a decrease in oxygen saturation levels of the blood. Blood oxygen saturation is expressed as a percentage, with normal

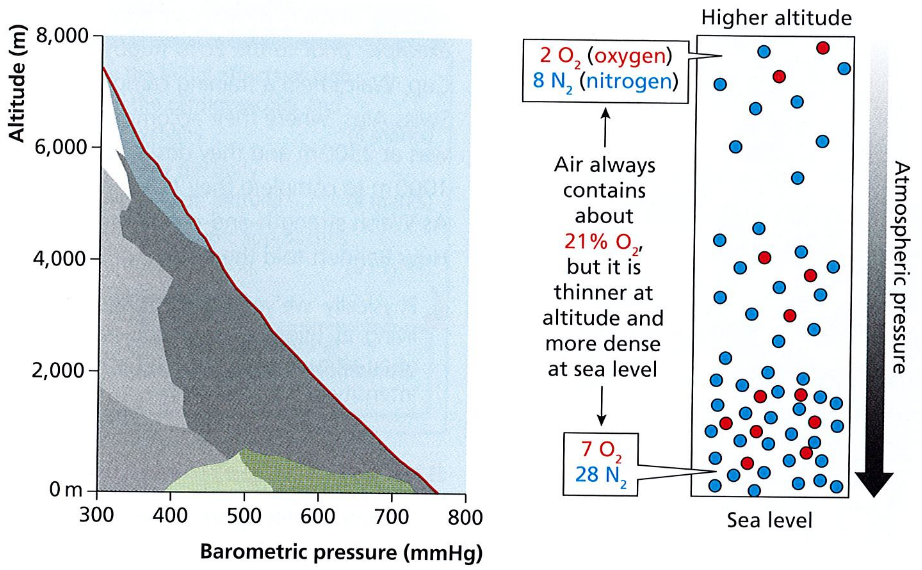


Figure 1 The impact of altitude on oxygen levels

Box I Air pressure

At low altitude there are more molecules of oxygen for any given volume of air and so oxygen exerts more force (it has a higher partial pressure) than at higher altitudes, where there are fewer molecules of oxygen and a lower partial pressure (Figure 1).

Atmospheric pressure is measured in mmHg. This is the height at which the pressure of the surrounding air raises liquid mercury inside an inverted tube. Atmospheric pressure at sea level is 760 mmHg, while at the top of Mt Everest (8848 m) it is 253 mmHg (Figure 2).

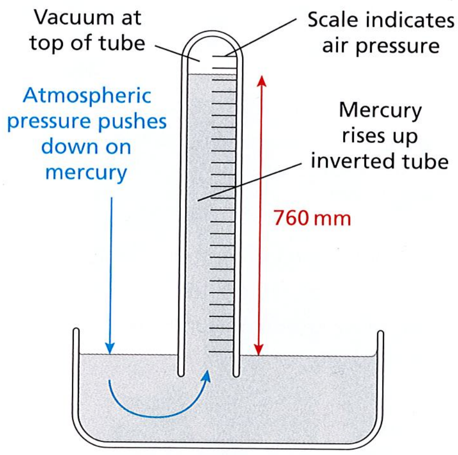


Figure 2 Measuring atmospheric pressure

readings at sea level ranging between 95% and 100%. This would drop to around 40% if you were to climb to the top of Mt Everest.

Oxygen in the blood

Blood oxygen saturation can be measured using a pulse oximeter placed over the top of a finger, which shines a light through the skin, detects the colour of the blood and calculates oxygen saturation from that. Many sports watches do a similar thing by shining red and infrared light onto your wrist to calculate *oxygen saturation of peripheral blood* (SpO_2).

With less oxygen in the blood, there is a reduction in arterial oxygen (PaO_2, the partial pressure of oxygen in arteries), known as *hypoxemia*. This decreases the oxygen diffusion gradient, and therefore the rate of diffusion, between the capillaries and muscle. As a result, the delivery of oxygen to working muscles and the capacity for aerobic energy production are reduced. Aerobic performance is thus impaired.

Changes in ppO2 are detected by the **carotid body**. Located in the neck at the bifurcation of the carotid artery (where it divides into two branches), this small organ acts as a chemoreceptor, sensing changes in the amount of oxygen (PaO_2) in the blood. Within seconds of being exposed to high altitude, the cardiovascular and respiratory systems act to compensate for the reduced availability of oxygen through the following:

- increased heart rate and cardiac output (Figure 3)
- hyperventilation (increased minute ventilation)

While heart rate and cardiac output both increase, stroke volume decreases during the first few days of exposure to high altitude. This is the result of a decrease in blood plasma volume, which increases the concentration of haemoglobin in the blood, leading to a greater diffusion gradient between capillary blood and muscle tissue.

KEY TERM

Carotid body A group of chemoreceptor cells that monitor O_2, CO_2 and pH levels of the blood and influence cardiovascular and respiratory function accordingly.

Acclimatising to altitude

While exercising at altitude is generally more challenging, over time the body responds to this environmental stress with adaptations that compensate for the lack of oxygen. This process of acclimatising to altitude has several key stages:

- The decreased oxygen saturation of the blood stimulates the production erythropoietin (EPO) in the kidneys.
- EPO stimulates the production of additional red blood cells (erythropoiesis) in the bone marrow.
- Additional red blood cells increase the oxygen carrying capacity of the blood and offset reductions in **VO2 max**.
- Additional adaptations include increases in aerobic enzymes, myoglobin, mitochondrial density and capillarisation.

The time required for these adaptations depends on the altitude. However, while these adaptations continue to develop for up to 3 months, there is relatively little improvement after an initial 2-week period. Timing the arrival of a performer prior to competing at altitude is therefore

KEY TERM

VO_2 max The maximum volume of oxygen that can be taken in and utilised by the body per minute (l/kg/min).

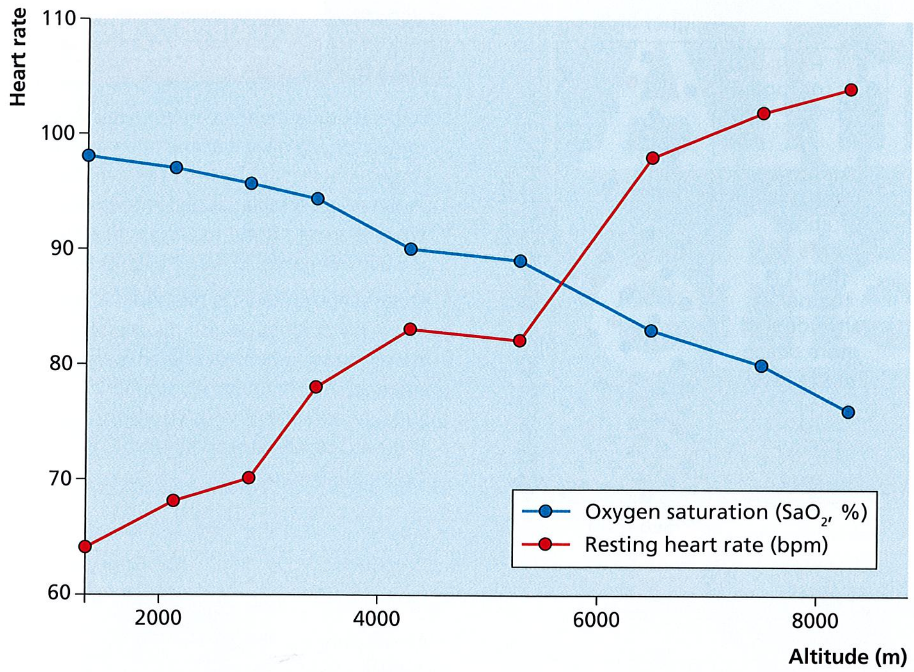


Figure 3 The relationship between altitude, blood oxygen saturation and heart rate. As altitude increases, oxygen saturation decreases and heart rate increases to compensate for this

an important consideration. Other environmental factors the athlete must plan for include time zones, temperature and humidity.

High or low?

As well as training at altitude while preparing to compete at altitude, many athletes in aerobic events and multiple-sprint sports (e.g. team games such as rugby) seek the performance-enhancing benefits of the body's adaptations to altitude. They will usually employ one of the following methods of altitude training to stimulate these physiological changes. Bear in mind that high altitude can be simulated using hypoxic (low-oxygen) tents or chambers in which athletes may sleep or train:

Live high, train high

British athlete Laura Muir regularly takes part in training camps at Font Romeu, 1850 m up in the French Pyrenees. This method increases exposure to high altitude, but athletes often experience a detraining effect, as they are unable to work at the same intensity.

Live high, train low

With this method, living at high altitude stimulates required adaptations, while training intensity is not compromised. For example, prior to the 2019 Rugby World Cup, Wales held a training camp in the Swiss Alps, where their accommodation was at 2300 m and they descended 1000 m to complete their training sessions. As Welsh strength-and-conditioning coach Huw Bennett told the BBC:

> 'Physically we've got the benefits of living at [high] altitude and we train at an altitude which doesn't affect the intensity of our sessions.'

Altitude and performance

A number of elite sporting venues around the world are at altitudes that pose significant difficulties for performers. At 1610 m above sea level, the home of the National Football League team the Denver Broncos is known as the 'Mile High Stadium'. In South Africa, Ellis Park in Johannesburg regularly troubles unacclimatised visiting rugby teams at 1724 m. The Kenyan city of Iten, famed as a high-altitude training camp for distance runners, is 2400 m above sea level. Higher still, several stadiums in South America are located more than 3000 m above sea level. To put this into perspective, the highest stadium in the English Football League is The Hawthorns, the home of West Bromwich Albion, at 168 m.

Negative effects

Generally speaking, most people experience limited effects up to 1500 m, when VO2 max is reduced by approximately 10%. However, research has shown that performance in endurance events can be affected by altitudes as low as 150 m. For example, women's 5000 m times are typically 0.6% slower when the race is run between 150 m and 299 m compared to at sea level. For an athlete like Laura Muir, whose personal best time for the 5000 m is 14:49, this would see her run more than 5 seconds slower.

British athlete Laura Muir regularly attends high-altitude training camps

RESOURCE

Hamlin, M. J. et al. (2015) 'Effects of altitude on performance of elite track-and-field athletes', **www.tinyurl.com/y3hyslgz**

EXAM-STYLE QUESTIONS

1 Identify the initial effects of high altitude on the cardiovascular and respiratory systems. (4 marks)

2 Describe how the body acclimatises to high altitude. (4 marks)

3 Discuss the different approaches taken to altitude training. (6 marks)

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Above 1500 m the impact is much greater. At this altitude women's 5000 m times are, on average, 3.2% slower. For someone running the distance in 15 minutes at sea level, this would result in running almost half a minute slower.

Positive effects

While high altitude has a negative impact on aerobic endurance events (e.g. middle- and long-distance running), it can benefit some performers. With air resistance dependent on air density, among other environmental factors (e.g. temperature, humidity, wind), some events benefit from being performed at higher altitudes where the air is less dense. In the men's 100 m sprint, for example, an average improvement of 0.7% was seen above 1500 m in comparison to sea level.

Small improvements were also seen over 200 m and 400 m. However, because air resistance is proportional to velocity, as the average velocity decreases and the contribution of the aerobic system to energy production increases, athletes in longer-distance events are increasingly disadvantaged by the reduced availability of oxygen.

Performances are also enhanced in the long jump and triple jump at altitudes above 1500 m, as the lower density of air reduces air resistance. An average improvement of 1% translates to a gain of 18 cm for an 18 m triple jumper and 8 cm for an 8 m long jumper. As these athletes do not rely on the aerobic system for energy production, their performance is not impaired by a lower ppO_2.

Bob Beamon's long jump world record of 8.90 m was performed at 2240 m during the 1968 Mexico City Olympics and stood for 23 years

Performances in the throwing events differ according to the nature of the projectile:

- In the javelin and discus, where lift is such a significant consideration, performance is impaired, as any benefit gained due to a decrease in air resistance is offset by the reduced lift force offered by less dense air.
- For the shot put, the slower velocity of the shot renders changes in air resistance negligible.
- The hammer throw is the event for which performance is most notably improved. Here the high velocity and relative mass of the projectile (7.26 kg for men and 4 kg for women) mean that a reduction in air resistance has greater impact on its flight than the reduction in lift force.

Summary

Exercising at high altitude presents a significant physiological stress on the body due to the lower partial pressure of oxygen. This triggers a series of immediate responses and longer-term adaptations that partially compensate for the reduced availability of oxygen. Athletes competing at altitude seek to maximise these benefits by acclimatising in advance of their events, while some performers competing at sea level may also benefit from altitude training.

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