David Marlin and Kathryn Nankervis

Equine Exercise Physiology
## Contents

*Foreword*  
v

*Acknowledgements*  
vii

### Part I  The Raw Materials  

1 Introduction  
2 Energetics of exercise  
3 Muscles  
4 Connective tissue  
5 The respiratory system  
6 The cardiovascular system  

### Part II  Exercise and Training Responses  

7 Muscular responses  
8 Skeletal responses  
9 Respiratory responses  
10 Cardiovascular responses  
11 Aspects of physiological stress and fatigue  
12 Thermoregulation  
13 Introduction to biomechanics  

### Part III  Applications of Exercise Physiology  

14 The demands of equestrian sport  
15 Training principles  
16 Training facilities  
17 Practical training  
18 Exercise testing  
19 Indicators of performance  
20 Feeding performance horses  
21 Transport  

*References*  
285

*Further reading*  
290

*Index*  
291
The training of competition horses has changed a great deal over the last few decades, for example with the introduction of the all weather gallop and the ‘interval’ method of training for staying horses.

This book explains the scientific reasoning behind the training of horses for competition in a manner that those working with horses will comprehend. It explains why training methods succeed and, just as importantly, if your horse is over stressed why those methods might fail.

The trainer of today’s horse is presented with a different set of problems from those of yesteryear. Arguably the competition is now tougher, which puts a horse under greater stress, and trainers are presented with ever more information about the condition of their horses – blood tests, food analysis and so on. We need to have at the very least a basic understanding of these factors if we are to make rational decisions about what is best for our horses. This book will help you understand how to manage your competition horse in today’s environment.

_Equine Exercise Physiology_ is a readable, up-to-date account of how to achieve the highest standards in your competition horses. It will suit all horse enthusiasts and students, as well as experienced trainers.

_Peter Scudamore_
The authors would like to thank the following friends and colleagues for comments on various chapters within this book: Rachel Neville (Chapters 2 and 20), Dr Stephanie Valberg (Chapters 3 and 7), Dr Rachel Murray (Chapters 4 and 8), Dr Bob Colborne (Chapters 4, 8 and 13), Dr Colin Roberts (Chapters 5, 9 and 18), Dr Lesley Young (Chapters 6 and 10), John Robertson and Rod Fisher (Chapter 14), Dr Catherine Dunnett (Chapter 20), Matthew French at Hartpury College for assistance in the production of the figures and Dr David Evans, Equine Performance Laboratory, University of Sydney, for providing Fig. 18.10.

Cover photo: Courtesy of Dr D.J. Marlin.

To Roma and George (DM)

To Tom (KN)
Part I

The Raw Materials
Chapter 1

Introduction

**Why train?**

In theory and in practice, the horse must surely be considered the best all round athlete of the animal kingdom. Horses are not great thinkers or fighters, they are runners. Whatever the breed or type of horse, they are all blessed with the same basic structure and the same basic physiological mechanisms; therefore they all have the potential to respond favourably to training. A horse’s performance, i.e. how fast it runs, how high it jumps, is largely determined by its natural ability, and to a lesser extent by its level of training. Natural ability is determined mainly by the genes the horse inherits from its parents (Fig. 1.1). We can’t do anything about the genes an individual horse has once it has been born, but we can do something about the training. To reach a horse’s genetic potential for performance, whether it is aimed at local riding club events or the Derby, it must be fit! From the unfit to fit state the horse undergoes a metamorphosis and its shape, its gaits, its looks, often even its attitude to life, are altered. Whatever we strive to achieve with our horses, there is much to be gained by making sure that they are fit enough for the task. To compete on an unfit horse puts both you and your horse at risk, quite apart from decreasing your chances of success and also the likelihood of the two of you going on to compete year after year.

Horses are always huge investments in terms of both time and money. If you aim to compete at any level, it pays to improve the horse’s chances of completing the work goals without risking mechanical breakdown and so incurring large veterinary bills, long periods of rehabilitation at best and destruction at worst. There is no doubt that training is an art, but a little understanding of the physiology of the horse can help anyone perfect their own art.

Much of what we currently understand about equine exercise physiology has been established in the last 20 to 30 years, largely as a result of an increased scientific and veterinary interest in exercise physiology, improvements in technology, and availability of equipment such as high speed treadmills. High speed treadmills enable vets and scientists to study the horse in controlled situations where the speed, distance, slope, going and environmental conditions can all be closely regulated. In addition, with a horse exercising on a treadmill it is a very simple matter to collect a blood sample from a catheter in an artery or vein, or to measure how much oxygen the horse is using. Procedures such as these are either difficult or at present not possible to undertake in the field. Inevitably, running on treadmills is not the same as running round a racetrack or a cross-country course, but it has enabled scientists to make great advances in the study of the horse’s responses to exercise and training. Many of these advances can now easily be applied to the management and training programmes of our own competition horses with a good degree of success. Whilst science cannot guarantee a winner, it may well shorten the odds in our favour.

**What are the aims of a training programme?**

What exactly are we trying to achieve as a result of training? The fundamental purposes of any training programme are to:

1. Increase the horse’s exercise capacity
2. Increase the time to the onset of fatigue
3. Improve overall performance, by increasing:
   - Skill
   - Strength
(4) Decrease the risk of injury.

By analysing the adaptations the horse makes in the short term (during exercise) and in the long term (throughout training) we can begin to understand how to design the horse’s work programme to achieve these aims (Fig. 1.2).

Exercise, work, training, fitness and performance

First of all, let’s tackle the vocabulary of ‘exercise physiology’. Being associated with horses entitles you to become a member of a club that has its own language, a language which is only understood by those ‘in the know’. Consider some of our expressions: we talk about grey when we mean white; we ‘break’ horses when we are introducing them to being ridden; a three-day event can be held over 4 days. What chance do outsiders have? Scientists also have their own language, one that is universal amongst all sorts of them, such as biochemists, geneticists, physiologists, etc. To be able to translate the results of scientific studies and apply them to real-life training situations we need to become familiar with the scientific vocabulary associated with equine exercise physiology.

Physiology is the study of the function of cells, tissues, organs and whole systems. Exercise physiology is thus the study of all systems involved in exercise. Exercise is a good example of a word commonly used in completely different contexts by scientists and horse people. Horse people often differentiate between lungeing a horse either for exercise or for work. The horse person’s interpretation of this is that by lungeing for exercise you are merely allowing the horse to ‘stretch its legs’ on the end of the lunge line, but it is not asked to do anything too taxing. Lungeing a horse for work means that it would probably wear side reins or maybe a ‘gadget’, e.g. a pessoa, and would be asked to engage its hindquarters and carry itself in a correct outline. To a scientist, work refers to the energy used up when an object moves a known or fixed distance; the amount of work done is described in terms of the energy used and is commonly measured in units such as joules (J) or kilojoules (kJ), or calories (cal) or kilocalories (kcal). A force has to be applied in order to perform work, and this force requires energy to be expended. Therefore the horse is doing work simply by moving from A to B. In scientific circles, ‘work’ does not infer any-
thing about the quality of the movement (i.e. speed, distance or direction), simply that something has moved. In fact, in strict scientific terms, it takes the same amount of energy to move a horse from A to B at a walk as it does at the gallop: the difference is in the rate at which energy is used. The same amount of energy is required to move from A to B, regardless of the speed, but when the horse moves at the gallop, the rate of energy utilisation must be greater. The rate of energy usage is referred to in terms of power. Power is measured in terms of the rate of work done (units of energy per unit time), e.g. in joules per second or watts. In galloping from A to B, the horse must generate more power than if he walks from A to B. Exercise refers to any movement or activity, so as soon as the horse moves off from a standstill, it is performing exercise. To use our scientific terms, if the horse is exercising, work is being done.

Training is another term that may have different interpretations depending upon the context in which it is used. To horse people, ‘training’ often implies that the horse is learning: its ‘basic training’ is its basic education. To an exercise physiologist, training is a long-term process of repeated bouts of exercise, which results in an improvement in fitness, where fitness refers to a certain capacity for exercise. Training for improvement in fitness is sometimes also referred to as ‘conditioning’, particularly in the USA.

Throughout exercise and training, the horse’s body should make certain physiological adjustments, adaptations or responses. An exercise response is any short-term physiological adaptation that is made as a result of an increase in the level of muscular activity, whilst a training response is a long-term physiological adaptation to repeated bouts of increased muscular activity. Exercise responses tend to return to baseline levels after the work is done. For example, during exercise there is an increase in heart rate, corresponding to the intensity of the work done. When the horse stops exercising, the heart rate will gradually return to resting levels. Training responses are more long lasting and are maintained as long as the horse continues to regularly undertake a certain volume of work. For example, a training response may be an increase in heart mass (weight) or an increase in the number of capillaries (small blood vessels) around each muscle fibre. Changes such as these occur over a period of time as the horse responds to a gradual increase in workload, but they do not change throughout the course of an exercise bout. Training changes are mainly mediated through activation of genes that may then ‘code’ for greater production of an enzyme in an aerobic energy pathway, for example.

The type of work undertaken is particularly important in determining whether or not a training response is induced. For example, walking a horse 16km (10 miles) a day, 3 days a week for a month may produce a noticeable loss of body mass (body-weight) as the horse will be using up a considerable amount of energy on each of these walks. However, it may do very little in terms of increasing fitness, i.e. producing a training response. When we think about training it is therefore not simply how much energy we use, i.e. the volume of work, that counts but the way in which that work is done, i.e. the quality of work, to induce appropriate training responses.

In summary, if we want to improve our horse’s performance, we would do well to increase their fitness (Fig. 1.3). This can be done by carrying out regular exercise, of progressively increasing workloads, to bring about the necessary training responses. The key is knowing what is the right work for what sport and when to settle for less than 100% fitness to reduce the risk of injury that might come from training very hard for a long time.
Horses are natural athletes.

Probably the biggest impact we can have on how well a horse performs is through training.

The aims of training are to increase the time to the onset of fatigue, improve performance and decrease the risk of injury.

Physiology is the study of the function of cells, tissues, organs or whole systems.

Exercise means that work is done and so by definition energy is used.

Exercise responses, e.g. an increase in heart rate, are short term.

Training is a longer process of many repeated bouts of exercise that brings about an increase in fitness.

Training responses, e.g. an increase in heart size, occur over a relatively long period of time.
Chapter 2

Energetics of exercise

Introduction

A little knowledge of biochemistry is a powerful thing! When you first become aware of the cellular processes involved in the conversion of nutrients into mechanical energy for muscle contraction it is like an enormous penny dropping, making so much of what the nutritionists tell us fall into place. For performance horses, one of the most important considerations is the energy content of the diet. To make sure our horse has enough energy for exercise, it helps to understand something about the way the horse’s muscles obtain energy from nutrients within the diet, and this forms the basis of the study of the energetics of exercise. Awareness of the energetics of exercise can help us formulate a diet to achieve a specific result. As far as interpretation of energetics is concerned, the horse is not dissimilar to a car: we input fuel at great expense and we expect a certain performance in terms of mechanical output. The horse, like the petrol engine in a car, is required to perform mechanical work. Unlike cars, however, the horse can run on a variety of fuels, and we can expect to see a difference in performance depending upon the type of fuel we put in.

Horse diets usually vary from being 100% forage-based to about 80% cereal-based. We should never feed a 100% cereal-based diet to horses, because they need a certain minimum amount of forage for effective functioning of the digestive tract. Consequently, most horses are fed a combination of forage and cereals which has to be broken down by a combination of mechanical, chemical and microbial digestive processes. The products of digestion are then absorbed into the bloodstream mainly from the small and large intestine. Some of these products may be used immediately to supply energy for muscular contraction, but the majority are more likely to be converted to fuel stores within the liver, muscle and adipose tissue (fat) to be used at a later date. Regardless of the type of feed we put into the horse, all the nutrients capable of releasing energy for work (glucose, fatty acids and amino acids) are ultimately converted to just one vital ingredient – ATP or adenosine triphosphate. ATP is our energy ‘currency’ that is required for normal functioning of all cells both at rest and during exercise.

The resting horse

A certain amount of fuel must be provided within the diet to support the horse’s energy requirements at rest and to do so whilst maintaining its body mass. Within 1 or 2 hours of a meal, particularly a cereal meal, the levels of glucose in the horse’s blood rise from about 5 millimoles per litre (mmol/l) of blood to about 7 mmol/l of blood. In response to this increase, the pancreas increases the secretion of insulin, a hormone that acts to decrease blood glucose, and several hours later the blood glucose levels are restored to 5 mmol/l. Insulin brings about a lowering of blood glucose by increasing the uptake of glucose into the muscle and liver. In other words, in times of plenty the emphasis is on accumulation of potential fuel stores within the liver and muscle. This ensures that muscle has sufficient fuel stores should there be an increase in muscle activity, and also that the liver has sufficient fuel stores to ‘buffer’ fluctuations in blood glucose arising as a result of exercise. Whilst glucose has a very important role in providing energy for muscular contraction, it is far more important from a physiological ‘housekeeping’ perspective to ensure that the brain and the heart are provided with glucose, because glucose is the primary fuel source for these vital organs. One of the most important
functions of the liver, aided by a number of hormones, is to act as a 'glucostat', i.e. a regulator of blood glucose, ensuring that blood glucose does not significantly decrease or increase, thereby guaranteeing a constant supply of glucose for the brain and heart, regardless of whether the horse is fed, starved, exercised or rested.

The energy for muscle contraction

Energy cannot be created or destroyed; it is merely converted from one form into another. All animals convert chemical energy from food into mechanical energy of work and heat is given off as a by-product. No process of converting stored or potential energy into work or movement is 100% efficient. In fact, animals (including humans) are rather inefficient energy converters, with only around 20% of the energy obtainable from food being converted into useful work, i.e. used for movement by muscle, and the rest (about 80%) being released as heat. To put this in context of mechanical engines, modern car engines would be able to convert around 20–30% of the potential energy in petrol into movement.

Adenosine triphosphate (ATP) is the universal fuel source: it has to be produced and stored within all cells in the body, whether muscle cells or any other cell, because it cannot be transported around the body. ATP is stored throughout the muscle cell. The structure of ATP is shown in Fig. 2.1. It is made up of adenosine attached to ribose and three phosphate groups. Only a certain amount of ATP can exist within the muscle: this is approximately 6mmol/kg wet muscle (equivalent to 24mmol/kg dry muscle) or approximately 700 g throughout all the skeletal muscle in the body of a 500 kg horse.

ATP provides a chemical energy source that is used by all cells, in all animals. Muscles cannot contract or even relax without ATP being present. When muscle cells contract, ATP is broken down into adenosine diphosphate (ADP) and phosphate (see Fig. 2.1), a reaction that is triggered by an enzyme within the muscle cell called adenosine triphosphatase (ATPase). The breakdown of ATP to ADP releases a fixed amount of energy – exactly 1.8 kJ per mole of ATP. To give some idea of the enormous rate of ATP regeneration, consider that the average person turns over (breaks down and restores) more than half their own bodyweight in ATP in a day, just at rest. An active horse may actually turn over four times their own bodyweight in ATP per day. The chemical breakdown of fuel stored within the muscle ensures that this enormous demand for ATP is met.

The main fuels used to provide energy are glucose, glycogen (both glucose and glycogen are forms of carbohydrate) and fatty acids (fat). Protein is only used to provide energy in cases of extreme exhaustion, starvation or disease. Glucose and fatty acids both circulate in the bloodstream and can also be easily taken up or released by muscles. Glycogen is the animal equivalent of starch in plants and is simply a long string of glucose units joined together. Because of its structure and size, glycogen in cells cannot leave and enter the bloodstream. The main sites of glycogen storage are within liver and muscle.

The conversion of food into useful energy for exercise

Muscle contractions can only be produced using ATP. Because only a small amount of ATP exists within the muscle and this is rapidly used up during
exercise (in fact within one or two muscle contractions), to continue working the muscle must constantly regenerate ATP by phosphorylating ADP to ensure a constant release of energy. The phosphorylation of ADP is achieved by several different biochemical processes or energy pathways within the muscle cell, all of which require the input of nutrients or fuels. One way to look at this would be to think about electricity and gas which are two different forms of energy or fuel. Electricity could be produced from a gas turbine generating station, but we can only light a bulb using electricity not gas. ATP is the equivalent of electricity, whereas gas is the equivalent of all the other potential fuels such as glucose, glycogen and fat. You may wonder why we cannot simply stick the phosphate back on to the ADP to regenerate ATP. To do so would be like trying to send the reaction uphill against an energy gradient: if it were possible to recycle ATP in this fashion, there would be no need to obtain energy from our diet and exercise could continue indefinitely. Simple recycling is not an option, and if we do not want to deplete our stores of ATP, we must utilise fuel supplies. Ideally, we should aim to regenerate ATP as fast as it is being used up by muscular contractions. The faster an animal travels, the greater the rate of ATP consumption by the muscles and the more quickly it needs to regenerate ATP from ADP to match demand to supply. Whilst regeneration of ATP from ADP is important to maintain a high ATP concentration, it is also important to keep the ADP concentration low because an increase in free ADP may contribute to muscle fatigue.

The energy demands of a single bout of exercise can significantly deplete a horse’s fuel stores; however, on a day-to-day basis, the food provided in the horse’s diet should keep the main carbohydrate and fat stores stocked up. Carbohydrates and fats are stored within the liver, skeletal muscle and adipose tissue. Glucose (a carbohydrate) is stored as glycogen within the liver and skeletal muscle, whilst fatty acids (fat) are stored as triglycerides within liver, muscle and adipose tissue, e.g. around the withers, crest, loins and around internal organs. A certain amount of fuel is available within the bloodstream, in the form of glucose and free fatty acids. The primary fuel supplies for any given piece of exercise are normally provided by glycogen within the muscle and free fatty acids from the bloodstream. Fuel stores within the liver and adipose tissue are used to top up the muscle stores when demand for energy is substantially increased as a result of exercise of either high intensity or long duration.

Energy pathways

There are several biochemical routes for phosphorylation of ADP, otherwise known as ‘energy pathways’, and one or more of these pathways will be automatically selected within any particular period of exercise. However, it is important to understand that the pathways available are not used on an all or nothing basis and that a number of different pathways may be used simultaneously for generating energy. The different pathways vary in their fuel economy, i.e. in how much ATP is released per gram of fuel broken down, and also in their ‘performance’, i.e. how quickly ATP is made available for contraction. There is no one energy pathway that has both a high ATP yield, i.e. is economic, and a high rate of ATP production, i.e. a high performance. The horse will therefore select a particular combination of energy pathways depending on the nature of the exercise and the state of its fuel stores. There are four basic energy pathways, two requiring oxygen (aerobic energy pathways) and two that do not require oxygen (anaerobic energy pathways). It is important to understand that the two anaerobic pathways are not only used in situations when there is no oxygen around. They are called anaerobic because they do not need oxygen, but they may be used when there is a plentiful supply of oxygen to the muscle.

Pathway 1: anaerobic phosphorylation of ADP using high energy phosphate stores in muscle

High energy phosphates include molecules such as phosphocreatine (PCr) that have high energy phosphate bonds. In other words, the energy is bound up in their structure. If these molecules can be broken down, the energy stored within their bonds becomes available for the regeneration of ATP from ADP. The production of ATP from ADP using
PCr is catalysed by creatine phosphokinase (CK or CPK), and is described by the chemical equation

$$
\text{PCr} + \text{ADP} \rightarrow \text{Cr} + \text{ATP}
$$

Phosphocreatine thus provides a quick method of regenerating ATP for use by the muscle. By ‘stealing’ a phosphate from PCr, ATP is very quickly regenerated within the muscle cell. Phosphocreatine stores can be used in this way to regenerate enough ATP rapidly, but there is only enough stored PCr to last for several seconds’ exercise. Earlier we learned that the concentration of ATP in the horse’s muscle is around 6 mmol/kg wet muscle, but the amount of stored PCr is around 15–20 mmol/kg wet muscle. However, it is important to emphasise again that the muscle cells cannot use the energy bound in the phosphate bond in PCr directly, but only after it has been transferred to ATP.

In certain circumstances, another reaction known as the myokinase reaction (named after the enzyme catalysing the reaction) may take place. This reaction occurs when the rate of breakdown of ATP is very fast, such as during acceleration or galloping, and the concentration of free ADP within the muscle fibres (cells) starts to increase. In this instance, ADP is the high energy phosphate, but also like PCr, it cannot be used directly by the muscle cells. However, when two ADP molecules are combined, one ADP effectively loses a phosphate (producing a molecule of adenosine monophosphate, AMP) whilst the other ADP gains a phosphate to become ATP. The chemical equation for this reaction is

$$
\text{ADP} + \text{ADP} \rightarrow \text{ATP} + \text{AMP}
$$

The myokinase reaction normally only occurs during high intensity exercise and then primarily in those muscle fibres which are recruited during high speed exercise, acceleration and jumping. Reactions such as this are often self-limiting in that if there is a build up of, in this case, AMP, the reaction from left to right will slow down. Because increases in ADP may be related to the fatigue process in high intensity exercise, the aim would be to try and keep the ADP low by removing it as fast as it appears. To do this, the muscle also needs a way to remove the AMP: this is carried out by the enzyme AMP deaminase. AMP deaminase converts AMP to inosine monophosphate (IMP) and ammonia.

All these reactions regenerate ATP quickly and without the use of oxygen. High energy phosphates such as PCr are used at the onset of exercise or for an explosive effort such as in a jump, or whenever speed of ATP regeneration is the primary requirement. The stores of ATP and PCr are small; therefore to sustain exercise for more than merely a few seconds, the body must switch to other energy pathways to regenerate ATP for muscle contraction. Once other forms of energy production have taken over, the high energy phosphate stores will themselves be replenished if exercise is of low to medium intensity. During higher intensity exercise the muscle ATP and PCr concentrations may be reduced by 50–70% by the end of the exercise bout as a result of decreases in muscle pH consequent to lactic acid production (see later).

The following two energy pathways (see pathways 2 and 3) involve the breakdown of fuel stores to support exercise lasting from several minutes to many hours. Fuel stored in the form of fat and carbohydrate can be broken down aerobically, i.e. in the presence of and requiring oxygen, to produce significant amounts of energy in the form of ATP. Glycogen (a carbohydrate) is a very large polymer (many strings of glucose molecules) of glucose residues and is the animal storage form of glucose (equivalent to starch in plants). By storing glucose in the form of glycogen, energy from glucose is available between meals. The glycogen molecules themselves vary enormously in size, existing in cells in the form of glycogen granules that cannot pass out of the cell into the bloodstream.

### Pathway 2: aerobic (oxidative) phosphorylation of ADP using carbohydrate stores

The breakdown of glycogen within the muscle using oxygen involves several stages, with oxygen playing a part only in the final stage. The first stage of the breakdown involves the conversion of glycogen to pyruvate, which occurs in the cytoplasm of the muscle cell and without the involvement of oxygen. The conversion of glycogen to pyruvate involves a specific sequence of phosphorylating reactions known as glycolysis (see Fig. 2.2). Glycolysis itself takes place rapidly but only yields a small amount...
of ATP directly (three ATP molecules per glucose unit broken down from carbohydrate stored within the muscle, i.e. from glycogen). However, more importantly, glycolysis produces two molecules of pyruvate that are used to feed into the next stage of the aerobic energy pathway, ultimately yielding considerably more ATP. Up to this stage, all reactions have taken place in the cytoplasm of the muscle cell.

The next stage in the aerobic breakdown of glycogen is the conversion of pyruvate to another three-carbon structure known as acetyl coenzyme A (acetyl CoA). This reaction only occurs inside mitochondria and is catalysed by an enzyme called pyruvate dehydrogenase (PDH). Mitochondria are found throughout the muscle cell, but particularly around the myofibrils (see Chapter 3). Mitochondria are specialised structures with an outer membrane and an inner folded membrane on which the enzymes of oxidative phosphorylation are located. The folding of the membrane increases the surface area available for reactions to take place. The number of mitochondria within a cell or tissue is an indication of its activity. Hence, the density of mitochondria is high in both locomotory muscle and cardiac muscle cells. Acetyl CoA then enters the third stage of the aerobic breakdown of glycogen that occurs within the mitochondria. Acetyl CoA initiates a series of reactions known as the tricarboxylic acid (TCA) cycle (see Fig. 2.3), also sometimes referred to as the Krebs cycle.

The net result of the TCA cycle is the production of two molecules of ATP and two hydrogen ions. Two hydrogen ions are also produced by glycolysis and these hydrogen ions combine with the two coenzymes nicotinamide adenine dinucleotide
(NAD) and flavin adenine dinucleotide (FAD) to produce NADH and FADH₂. NADH and FADH₂ enter the ‘electron transport chain’ (see Fig. 2.4) on the inner mitochondrial membrane.

The hydrogen ions are then split into electrons and protons, and through a series of chemical reactions (‘electron transport’) ADP is regenerated to yield 34 molecules of ATP and the hydrogen ions are eventually combined with oxygen to produce water. Because this process requires oxygen it can be termed aerobic or oxidative phosphorylation. The production of water at the end of the chain has the advantage of ‘removing’ hydrogen ions from the cell because these would make the inside of the cell acidic. Both glycogen and glucose can be used in glycolysis. Glycogen would be obtained from the muscles’ own glycogen stores whilst glucose would be obtained from the bloodstream.

The process of glycogen breakdown is known as glycogenolysis and results in the formation of glucose-1-phosphate. The breakdown of glycogen is controlled by an enzyme called glycogen phosphorylase which has both inactive and active forms, termed α and β. Whilst glucose units from glycogen breakdown end up as glucose-1-phosphate and after conversion to glucose-6-phosphate can enter directly into the glycolytic pathway, glucose taken up into the muscle from the blood must first be phosphorylated, i.e. have a phosphate added to make it into glucose-6-phosphate. This reaction is catalysed by an enzyme called hexokinase and requires a molecule of ATP to ‘donate’ a phosphate. The fact that glucose-1-phosphate is produced directly from glycogen breakdown saves an ATP being expended in the initial stages of glycolysis, and prevents leakage out of the muscle cell, because phosphorylated compounds, including ATP, ADP and AMP, cannot normally cross cell membranes unless these have been damaged. Whether the glucose units come from blood glucose or muscle glycogen, the first stage of glycolysis is therefore considered to be the production of glucose-6-phosphate.

Complete aerobic metabolism (breakdown) of one glucose unit from glycogen to water and carbon dioxide (formed from the TCA cycle) yields 39 molecules of ATP (three ATP from glycolysis, two ATP from the TCA cycle and 34 ATP from the electron transport chain). The complete aerobic breakdown of glucose taken up by the muscle cell from the

---

**Fig. 2.3** Tricarboxylic acid (TCA) cycle.

**Fig. 2.4** Electron transport chain.
bloodstream yields one less ATP because one ATP molecule is required to convert glucose to glucose-6-phosphate in the first stage of glycolysis. Therefore the net ATP yield from the aerobic breakdown of glucose is $39 - 1 = 38$.

### Pathway 3: aerobic phosphorylation of ADP using fatty acids

The aerobic breakdown of fat in the form of fatty acids begins with the conversion of 2-carbon chunks of fatty acid being converted to acetyl CoA by a process called beta-oxidation. In the breakdown of fat, acetyl CoA is therefore not produced by glycolysis but by beta-oxidation. The mitochondrial stages of the breakdown of fat, i.e. the TCA (Krebs) cycle and the electron transport chain, are thus identical to those of glycogen, but the steps leading to acetyl CoA formation are different. A schematic overview of the stages of glycogen and fat breakdown is shown in Fig. 2.5.

The individual stages involved in the aerobic breakdown of fat (as for carbohydrate) are also known collectively as oxidative phosphorylation. The yield of ATP from fat is always higher than for the same mass of carbohydrate (whether glucose or glycogen) but also varies between different types of fat source. The fats stored within the body are in a form known as triglycerides that consist of one molecule of glycerol and three fatty acid molecules. The
Triglycerides are broken down by enzymes known as lipases and the process is referred to as lipolysis. Once the fatty acids have been separated from their glycerol ‘backbone’, they are free to move into the bloodstream and be taken up by muscle. In this state they are known as free fatty acids (FFA). Muscle itself also contains small triglyceride stores that can also be broken down to liberate FFA that can be used inside the muscle cell. There are a number of different FFAs found in the body which differ primarily in the number of carbon atoms they contain. Volatile fatty acids (VFAs) are another very important source of fuel, being produced from the fermentation of carbohydrates in the large intestine. Once in the circulation VFAs can be taken up and used immediately to fuel muscle contraction after conversion to ATP; if not, they are stored in adipose tissue as triglycerides.

The complete aerobic metabolism of palmitic acid (a typical 16-carbon fatty acid) with the molecular formula \( \text{C}_{16}\text{H}_{32}\text{O}_{2} \) yields 129 molecules of ATP net per molecule of fatty acid. The total production is 131 molecules of ATP, but two ATPs are used to ‘activate’ (prepare) the FFA before they can enter the TCA cycle. Activation of FFA takes place on the outer mitochondrial membrane before oxidative phosphorylation within the mitochondria that results in a total of 35 ATP. Every time the fatty acid chain is shortened by two carbons, one FADH\(_2\) and one NADH are formed, resulting in the production of five ATP molecules by oxidative phosphorylation. In a 16-carbon fatty acid, the chain is shortened by two carbons seven times to leave eight two-carbon pieces; hence \( 5 \times 7 = 35 \) ATP. The TCA cycle yields eight ATP molecules directly and 88 by oxidative phosphorylation, totalling 131 ATP minus two ATP for fatty acid activation, giving 129 ATPs.

The fact that oxidative phosphorylation of fat yields about three times as much ATP as the oxidative phosphorylation of carbohydrate explains why fat is referred to as an ‘energy-dense’ food source. With one molecule of fat yielding three times as much energy as one molecule of carbohydrate, you can begin to see why exercising to lose fat often seems like an uphill struggle as you have to expend considerable amounts of energy at a relatively low intensity, i.e. exercise over a longer time in order to break down excess adipose tissue. On the positive side, the fact that fat is energy-dense is great news for endurance athletes, as a little bit of fat goes a long way. Even a thin horse will only use a small proportion of stored body fat to complete a 100-mile endurance race.

On a mass for mass basis, 1 gram of fat is better than 1 gram of carbohydrate when it comes to ATP yield, but the disadvantages of fat as a fuel are that, firstly, it requires far more oxygen to break down one molecule of fatty acid than it does to break down one molecule of glycogen. Secondly, the speed (rate) of energy release from fat is much slower than from carbohydrate (see Table 2.1). Exercise using fat as the main fuel source is therefore limited to trotting and slow–medium speed.

### Table 2.1

<table>
<thead>
<tr>
<th>Source of Energy</th>
<th>Maximum power (mmol ATP/kg/s)</th>
<th>Time to reach maximum power</th>
<th>( \text{O}_2 ) requirement (mmol ( \text{O}_2/\text{ATP} ))</th>
<th>Work time to fatigue</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaerobic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ATP(^a)</td>
<td>11.2</td>
<td>(&lt;1\text{s})</td>
<td>0</td>
<td>seconds</td>
</tr>
<tr>
<td>PCr(^b)</td>
<td>8.6</td>
<td>(&lt;1\text{s})</td>
<td>0</td>
<td>seconds</td>
</tr>
<tr>
<td>(\text{CHO}^+ \to \text{lactate})</td>
<td>5.2</td>
<td>(&lt;5\text{s})</td>
<td>0</td>
<td>minutes</td>
</tr>
<tr>
<td>Aerobic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\text{CHO}^+ \to \text{CO}_2 + \text{H}_2\text{O})</td>
<td>2.7</td>
<td>2–3\text{min}</td>
<td>0.167</td>
<td>hours</td>
</tr>
<tr>
<td>(\text{FFA}^d \to \text{CO}_2 + \text{H}_2\text{O})</td>
<td>1.4</td>
<td>30\text{min}</td>
<td>0.177</td>
<td>days!!</td>
</tr>
</tbody>
</table>

\(^a\) Adenosine triphosphate.  
\(^b\) Phosphocreatine.  
\(^c\) Carbohydrate.  
\(^d\) Free fatty acid.
cantering. At speeds above this the body must gradually switch to using more and more carbohydrate to match the increased rate of ATP usage by the muscles with the rate of rephosphorylation of ADP. The faster a horse runs the less it is able to use fat as an energy source.

**Pathway 4: anaerobic phosphorylation of ADP using carbohydrate**

Technically, the conversion of ADP back to ATP using phosphocreatine (pathway 1, described above) is an anaerobic energy pathway, but the overall contribution of this energy pathway to the total energy cost of a bout of exercise is not usually significant because most exercise bouts last more than a few seconds. The most significant anaerobic energy pathway involves the conversion of glycogen or glucose to lactic acid to yield ATP. Only glycogen or glucose can be used to produce energy anaerobically via the glycolytic pathway. The glycolytic pathway involves the production of pyruvate from glucose or glycogen as in aerobic energy production, but this time, instead of the pyruvate being converted to acetyl CoA and entering the mitochondria, the pyruvate is converted to lactic acid by the enzyme lactate dehydrogenase (LDH). Lactic acid immediately dissociates into a free hydrogen ion (with a positive charge) and a lactate ion (with a negative charge). The two terms lactic acid and lactate are often used interchangeably, for example when referring to blood or plasma concentrations. Thus, the reactions involved in the aerobic and anaerobic productions of ATP are identical up to the point at which pyruvate is formed. The net result of the anaerobic breakdown of carbohydrate is the production of a small amount of ATP (three ATP molecules if glycogen is the glucose source and only two ATP molecules if blood glucose is used) and the conversion of NAD to NADH. NADH is an important intermediary in glycolysis and one that would normally be regenerated to NAD following the completion of the electron transport chain in oxidative phosphorylation. Because there is no electron transport chain in the anaerobic pathway, the only way of regenerating NAD from NADH is as a by-product of the conversion of pyruvate to lactic acid. If all the NAD in a muscle cell were converted to NADH then glycolysis would stop. The production of lactic acid enables NADH to be regenerated to NAD and allows glycolysis to proceed beyond glyceraldehyde 3-phosphate.

$$\text{Pyruvate} + \text{NADH} \rightarrow \text{Lactate} + \text{H}^+ + \text{NAD}$$

The anaerobic energy yield from one molecule of ‘glucose’ from glycogen is three ATP molecules, whereas it is two ATP molecules for one molecule of glucose from blood. Two molecules of lactate are also formed which can be converted back to pyruvate and eventually to glucose by a process known as the Cori cycle. Because the anaerobic production of energy involves the conversion of pyruvate to lactate, it can be seen that it would be impossible to break down fat anaerobically.

**Anaerobic energy production is inefficient but fast**

When you need energy fast, such as during acceleration, when galloping or jumping, glycogen is broken down anaerobically to lactic acid. A major disadvantage of anaerobic energy production is the low ATP yield per molecule of glycogen or glucose; therefore substantial reliance on anaerobic energy production leads to significant depletion of muscle glycogen stores (Fig. 2.6). Resting muscle glycogen concentrations in the horse are in the region of 100 mmol/kg wet muscle, and up to around 150 mmol/kg wet muscle (600 mmol/kg dry muscle) or more in trained horses. Muscle glycogen con-
centrations can be reduced by one-third after just a single bout of high intensity exercise. Fine! Go fast, use lots of glycogen, but if you want to do it again in a few hours’ time, or tomorrow, and the day after, and the day after that, you may run into trouble. The muscle needs a certain amount of time to restore the glycogen levels to those before exercise. In man, it has been shown that by manipulating the diet it is possible to both increase glycogen storage before exercise (glycogen loading) and to speed up glycogen repletion after exercise. So far, no one has managed to achieve the same in horses and it doesn’t matter if after exercise you feed your horse hay, or hay and cereals, or even pure glucose powder, the rate of glycogen repletion appears to be the same.

The duration of exercise that you can undertake when using glycogen alone as the energy source is limited because fatigue is partly related to the acidification of the muscle cells by the free hydrogen ions produced in the conversion of pyruvate to lactic acid. At the sort of rates of glycogen breakdown seen during maximal all-out sprints, muscle glycogen stores can be reduced by around 50%. Even more glycogen can be used by carrying out repeated bouts of short, fast exercise with recovery periods in between (often termed intermittent, high intensity exercise or intermittent, maximal exercise). However, as more and more lactic acid is produced and the muscle pH becomes lower (more acid), a feedback mechanism takes over to prevent complete exhaustion of muscle glycogen stores. The rate of glycolysis slows and therefore so does the rate of glycogen breakdown and lactic acid production. This process of fatigue is actually protective. Whilst it is possible to completely use up all the muscle glycogen present in those cells used the most during high intensity exercise, it is not possible to deplete all cells in a muscle. Glycogen depletion can also occur in endurance exercise, affecting those cells used predominantly during low to medium intensity exercise (see Chapter 3).

**Energy partitioning**

At the onset of maximal exercise the demand for energy is high, but there is a lag time in reaching maximal aerobic energy production. In other words, anaerobic pathways are often necessary to supply energy for the early stages of exercise (even though there may be no shortage of oxygen in the muscle) whilst the aerobic pathways get up to speed. It can take up to at least a minute to reach maximal aerobic energy production at the start of maximal exercise. Thus the intensity of the exercise and the nature of the onset of the high intensity exercise (gradual increase in speed to maximum or flat out from a standing start) have a bearing on the extent to which aerobic and anaerobic pathways contribute to the overall energy requirement.

During low intensity exercise of long duration (exercise producing heart rates up to around 160 beats/min), energy is provided largely by aerobic pathways because these can produce ATP at a sufficient rate and offer the greatest fuel economy. During high intensity exercise of short duration energy is provided by anaerobic pathways offering high rates of ATP production but low fuel economy. However, there is not a point at which a direct switch from one source to the other occurs. At any point in time, some muscle fibres will be functioning aerobically and some anaerobically, but there is a general increase in the reliance on anaerobic pathways as speed increases. At speeds greater than 8–10 m/s, i.e. 500–600 m/min (around 20 mph), the mitochondria tend to back-up with substrate and there is neither sufficient area of mitochondrial membrane nor aerobic enzymes available to cope with demand. Latterly it was thought that the switch was due to lack of oxygen availability, but this is not usually the case. Oxygen delivery to the working muscles is usually sufficient; the main drive to begin to recruit anaerobic pathways is the point at which the aerobic energy pathways are working at maximum, but demand for ATP is still rising, i.e. the horse is being asked to go faster. The resultant shortfall in ATP supply must be addressed using anaerobic pathways resulting in an increase in blood lactate levels. The point at which lactate levels start to rise in often called the ‘anaerobic threshold’ (AT). This term is widely used among lay people to describe the point at which anaerobic pathways begin to contribute significantly to total energy requirements, but it is a slightly misleading term in that it implies there is a switch from one form of energy pathway to the other, which is not true, as described above.

The contribution of each energy pathway to the total energy requirement for exercise is known as
energy partitioning. Scientists have been able to estimate how much each pathway contributes to the total energy requirements by measuring oxygen uptake and carbon dioxide and lactic acid production at various speeds of exercise. In the UK, the shortest distance raced is 5 furlongs or 1000 metres. In the USA, Quarter horses run over 2 furlongs or 400m: these are the true sprinters of the horse world, reaching speeds of around 40 m.p.h. During exercise of this intensity and duration, the horse will obtain approximately 60% of its energy anaerobically and 40% aerobically. Compare this to human sprinters who run 100 metres near 100% anaerobically; only taking one or two breaths in 10 seconds. In contrast, the true equine sprinters, the Quarter horses, are running for at least double this time. In a middle distance Thoroughbred horse race such as the Derby run over 1\(\frac{1}{2}\) miles (2.4 km), the energy partitioning would be approximately 80% aerobic and 20% anaerobic. The anaerobic portion is mainly required for the acceleration at the start and for the last furlong or so.

The true endurance athletes of the horse world are able to complete 100 miles (160 km) in a day, travelling at speeds of 10 m.p.h. (16 km/h). True endurance horses will work about 96% aerobically at this speed. Even the speed and endurance day of a three-day event is predominantly aerobic (about 90%).

Normally, anaerobic energy production begins at a heart rate of around 150–180 beats/min, but there is great individual variation. A heart rate of 150–180 beats/min is the equivalent of a ‘good’ or three-quarter speed canter. In other words, as soon as the horse starts to really open up the canter stride, it is likely that it is getting some of its energy by anaerobic means, with the resultant appearance of lactic acid in the bloodstream.

Although it is possible to give general guidelines concerning the onset of anaerobic processes in muscle, a number of factors can affect the speed at which anaerobic energy production starts. For example, a horse will start anaerobic energy production at a lower speed when unfit compared to when fit. A horse that has a high proportion of fast-twitch high glycolytic (‘sprinting’ or type IIB fibres) in its muscles will produce lactate at a lower speed than a horse with few of this type of muscle fibre. Health problems that interfere with oxygen transfer from atmosphere to mitochondria, such as upper airway obstruction, cardiovascular disease or lower airway disease will also tend to decrease the speed at which anaerobic energy production starts. Even excitement, pain, amount and type of warm-up exercise, time of feeding and environmental conditions can affect the point at which the anaerobic metabolism becomes significant and the concentration of lactic acid in blood begins to increase.

How can we tell what fuels are being used for energy generation? The amounts of oxygen used up and carbon dioxide produced vary according to the energy substrates being used at any point in time. The respiratory exchange ratio (RER) is the ratio of carbon dioxide production ($\dot{V}_{\text{CO}_2}$, litres/min) to oxygen consumption ($\dot{V}_{\text{O}_2}$, litres/min) measured at the nostril (p. 94). The ratio of carbon dioxide production to oxygen consumption or RER is 1.0 when carbohydrate is the only fuel being used and is being fully oxidised. However, when fat is the sole fuel source a relatively greater amount of oxygen is required and the RER is 0.7. Any RER value between 0.7 and 1.0 either at rest or during exercise therefore indicates that both fat and carbohydrate are being used simultaneously (see Table 2.2). During moderate to intense exercise, the production of lactic acid within muscles causes an increase in the hydrogen ion concentration of blood (blood pH decreases). As a consequence of the mechanism within the blood for buffering hydrogen ions, there is an increase in the amount of exhaled carbon dioxide. Thus, an RER above 1.0 indicates that some of the energy is coming from anaerobic lactic acid production. The higher the RER the greater the contribution from lactic acid to total energy production, with values as high as 1.4 being reached in intense exercise. However, once the RER has risen above 1.0 it is not possible to estimate the

<table>
<thead>
<tr>
<th>RER</th>
<th>% Energy from carbohydrate</th>
<th>% Energy from fat</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.70</td>
<td>0</td>
<td>100</td>
</tr>
<tr>
<td>0.80</td>
<td>33</td>
<td>66</td>
</tr>
<tr>
<td>0.90</td>
<td>66</td>
<td>33</td>
</tr>
<tr>
<td>1.0</td>
<td>100</td>
<td>0</td>
</tr>
<tr>
<td>&gt;1.0</td>
<td>100</td>
<td>0</td>
</tr>
</tbody>
</table>
relative contributions of fat and aerobic and anaerobic carbohydrate metabolism.

RER can be used to determine responses to dietary manipulation, exercise and training, and may also reflect the muscle fibre type composition in horses with very different muscle types. RER in humans can be around 0.7–0.8 at rest but is generally around 0.9 in horses. In a study of Standardbred horses fed different diets for 4 weeks each, RER was about 0.9 on a typical hay and concentrate diet, but was around 0.75 on a high fat diet (containing 15% soyabean oil), indicating that the horses were actually using more fat for energy at rest on the high fat compared to the normal hay–concentrate diet (Pagan et al. 1987).

Table 2.3  Estimated times to exhaust each of the main body energy stores of a 500kg horse if each fuel was used as the only energy source at 60% (endurance), 90% (four-star three-day event steeplechase speed) and 120% $\dot{V}_O_{2,max}$ (1 mile (1.6km) flat race)

<table>
<thead>
<tr>
<th>Total body stores$^a$ (kJ)</th>
<th>Exercise time at 60% $\dot{V}<em>O</em>{2,max}$</th>
<th>90% $\dot{V}<em>O</em>{2,max}$</th>
<th>120% $\dot{V}<em>O</em>{2,max}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>ATP 38</td>
<td>3.3 s</td>
<td>1.8 s</td>
<td>1.1 s</td>
</tr>
<tr>
<td>PCr 188</td>
<td>16.3 s</td>
<td>9.0 s</td>
<td>5.7 s</td>
</tr>
<tr>
<td>Glycogen 75 300</td>
<td>109 min</td>
<td>60 min</td>
<td>38 min</td>
</tr>
<tr>
<td>Fat 640 000</td>
<td>15.4 h</td>
<td>8.5 h</td>
<td><em>b</em></td>
</tr>
</tbody>
</table>

$^a$From McMiken (1983).

$^b$No figure has been calculated for 120% $\dot{V}_O_{2,max}$ because at this intensity no fat would be used.

Size of the fuel stores

How much fuel can a 500kg horse carry? The sum of fat, muscle and liver comes to just over 230kg, with muscle weighing about 200kg, liver about 6.5kg and fat about 25kg.

The horse has around 95% of its total body glycogen stored in muscle and around 5% in the liver (although the actual concentration of glycogen in the liver is greater than in muscle). In contrast, around 95% of the body’s fat is stored in adipose tissue, with only around 5% stored within the muscles.

Approximately ten times as much energy (either in terms of kilojoules or kilocalories) is stored as fat compared with glycogen (see Table 2.3). This means that if you were to oxidise or simply burn (literally set alight) all the available fuel in the horse’s body, the fat stores would give off ten times as much heat energy as glycogen. However, if all the available fuel is respired aerobically, approximately 30 times as much ATP is produced from the oxidative phosphorylation of fat as from the oxidative phosphorylation of glycogen. Remember, in terms of ATP yield, fat is better than glycogen, with over three times as much ATP produced per gram of fat compared with a gram of carbohydrate. Fat is an energy-dense source; literally, a little bit of fat goes a long way. If you were setting out on a day’s walking or trekking and you had to carry all your food for the day, you would look to carry those foods which gave you lots of energy, but did not weigh very much; in other words, you would look for energy-dense foods. If you were a horse embarking upon a 100-mile endurance ride, it would pay you to use fat as your fuel source as far as possible because it is so energy-dense. Exercise is rarely, if ever, limited by running out of fat.

Fat is ideal for exercise where the body requires:

- Slow release of energy, i.e. for exercise at low speed
- A large energy reserve, i.e. for exercise of long distance or duration.

Training of low speed and long duration increases the number of enzymes involved in the oxidative phosphorylation of fat, so that the body becomes better at utilising fat, and will tend to rely more on fat as an energy source. This is good news for those of us thinking of training to get rid of unwanted fat: the more you train, the better you are at burning
fat, but you must keep the speed and intensity of the exercise sessions low. If, as a rider you want to lose weight, i.e. reduce your body mass, there is no better way to start than by doing plenty of walking and slow jogging. Start to run faster and you will start using more and more carbohydrate up to a point where you won’t be using any fat. You can get fit but won’t necessarily weigh any less. It is alarming how many people still think that the best way to reduce a horse’s waistline is to gallop and ‘get a sweat on him’. If you gallop a fat horse, all you will do is:

1. Risk breakdown of musculoskeletal structures
2. Make it sweat and lose body mass (due to loss of fluid) in the short term which it will put back on as soon as it can drink
3. Use up muscle glycogen
4. Give it an appetite!

You will not encourage utilisation of fat stores. It is also worth being aware that when you train your horse, although its shape and appearance may change, its body mass may not change dramatically. This doesn’t mean that you aren’t working it hard enough. Fat is less dense than muscle. The density of fat is 0.9007 g/cm³ (1 cm³ = 1 ml), whilst the density of muscle is 1.065 g/cm³, i.e. 20% more dense than fat. That means that the same volume of fat weighs less than the same volume of muscle. Therefore, if you replace 1 kg of body fat by 1 kg of muscle, it will take up less space but you will have the same body mass. This is why dieting combined with exercise may mean your shape changes as you lose fat but your body mass may stay the same or even increase as a result of muscle development from the exercise.

Running out of energy

Fatigue during exercise is almost never caused by running out of fat because the fat stores throughout the body are so plentiful, but is commonly due to running out of muscle and/or liver glycogen. Depletion of muscular glycogen leads to muscular fatigue. Depletion of liver glycogen and as a consequence low blood glucose may make you feel light headed and tired. Human athletes competing in long duration events use a technique called glycogen loading or carbohydrate loading to try and offset fatigue. It involves either exercising to fatigue or fasting in order to deplete the existing glycogen stores, and then eating large quantities of a high carbohydrate meal. Depletion before loading seems to increase the amount of glycogen that it is possible to store. Glycogen loading is not recommended in horses because it would require the feeding of large, high carbohydrate meals, when we know that high energy feeds should be split up into several small meals to avoid conditions such as colic or azoturia (tying-up).

Whilst we should not attempt to strategically glycogen load our horses, it is likely that a horse in hard regular work that is not provided with sufficient energy in its diet will be at a disadvantage. Following a hard piece of work that significantly reduces muscle glycogen, such as a short fast gallop, it may take 2 days for the horse to fully replenish the glycogen stores. This should be remembered when planning a training programme that involves fast work. You cannot expect a horse to perform intense work, e.g. maximal sprint or interval work, well more than two or three times a week or on consecutive days, because glycogen stores will not be fully replenished before the next bout of intense work. In addition, the rate at which glycogen can be broken down in glycolysis to yield ATP has been shown to be dependent on its concentration. If the concentration is high, the rate of breakdown will be high and vice versa. This is likely to be an advantage in high intensity sprint or jumping events.

---

**KEY POINTS**

- Energy that is used each day for exercise must be replaced by energy obtained from the diet.

- Glycogen (the animal form of starch) and fat represent the two main sources and body stores of energy.
• Cells cannot use glucose, glycogen or fat directly, only the energy released from the breakdown of ATP to ADP.
• At rest, food is converted via digestion to stores of glycogen (liver and muscle) and fat (adipose tissue).
• Energy cannot be created or destroyed, only changed from one form to another.
• The efficiency of conversion of energy into useful, mechanical work is only around 20%.
• Stores of ATP within the body are only sufficient for several seconds of exercise; for continued exercise ADP must be regenerated to ATP by two anaerobic (PCr and glycolysis to lactic acid) or aerobic pathways (oxidative phosphorylation of glucose or glycogen or fatty acids).

• PCr and metabolism of glucose or glycogen to lactic acid regenerate ADP fast without the need for oxygen but inefficiently, i.e. only a small amount of ADP is regenerated.
• Aerobic oxidation of carbohydrate and fat require oxygen and are much more efficient but regenerate ADP to ATP more slowly.
• Energy partitioning describes the relative contribution of different pathways of ADP regeneration at different stages of exercise and during different types of activity.
• The respiratory exchange ratio (RER) indicates what fuels are being used at any point in time.
• Fat stores almost never limit exercise capacity, but carbohydrate (glycogen) stores can become significantly depleted by repeated bouts of high speed exercise or prolonged endurance exercise.