PHYSIOLOGY OF SPORTS

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Preface

Recent decades have witnessed a remarkable expansion of the applications of scientific principles to sport and exercise. This has been associated with the emergence of sports science as a recognized academic discipline. Developments are such that most international teams now have a systematized scientific back-up as they prepare for major competitions. Applications of science to sport are especially evident in the field of physiology; indeed sports practitioners are quick to realize the importance of acquiring basic physiological knowledge that can be put to good effect.

Exercise physiology has for many years been a respected field in its own right. Exercise has conventionally been used as a medium for perturbing physiological systems to ascertain how they behaved under stress. Thus much information has been acquired in mainstream human physiology about acute physiological responses to exercise. Exercise physiologists have taken this further in establishing the ceilings in human physiological responses and in attempting to identify those factors that limit performance in various conditions. There has also been progress in understanding how the upper limits of physiological function can be pushed further by proper diet and nutritional manipulations. This information is continually being integrated and updated in textbooks of exercise physiology. These books have formed basic references not just for students of physiology and sports science but also for sports practitioners eager for literature about the physiological aspects of exercise and training.

The present text is unique in that it tries to spell out physiological implications for a number of sports, taken in turn. Thus, far from being another book on exercise physiology, it fits information about acute and chronic adaptations to exercise to the peculiarities of each sport. The individual requirements of each sport are first outlined before the demands of the sport, the fitness profiles of top performers and training regimens are considered.

Part One has two chapters that provide a general physiological background from which the physiology of particular sports may be approached. The principles outlined can be applied broadly to exercise and sports physiology. The opening chapter presents a detailed account of metabolic aspects of exercise. The other provides an analysis of muscular adaptations to strength training, a topic of importance to a wide range of sports. Part Two considers locomotive sports, starting with short-term exercise—in the form of sprinting—and progressing to middle distance running, then to sustained endurance exercise. This is illustrated in the form of marathon running and competitive race-walking. Both shortterm and endurance exercise apply to cycling and their considerations are integrated in the final chapter of the series of five.

In Part Three the physiology of sport on water and on ice is covered. The requirements of the various swimming events are compared in the opening chapter as is the specificity of exercise in the water. The sailor has entirely different demands (as has the rower) and

hopefully with good craftsmanship stays clear of the water whilst propelling the vessel through it. The main sports conducted on ice are selected for treatment and discussed in full in the final chapter in this section.

Part Four is concerned with the physiology of games. An attempt is made to group games with features in common. Thus the various codes of football are examined together which serves to highlight differences between them as well as the similarities. The same applies to the racquet sports. The court games of basketball and volleyball are dealt with together in another chapter.

P*hysiology of Sports* applies physiological knowledge to specific sports and represents these applications within a single text. Inevitably not all sports could be accommodated. Indeed a comprehensive text providing detailed coverage of say all the sports under the Olympic Games umbrella would itself be subject to criticism. Awesome in size, it would still omit major sports not included in the Olympic Games, some of which (for example, Rugby, squash) are in fact covered here. The final chapter is an attempt to present an overall perspective from which some sports not included in the core of the book might be viewed.

The contributors to this text are characterized not only for their scientific aptitudes but also for their practical insights into their specialist sports. This combination allows interpretations that go beyond those of the sports scientist operating in broad terms. In some chapters expertise is pooled so that the sport in question is treated in a comprehensive manner.

P*hysiology of Sports* is designed for both the academic and the practitioner. Its content is of interest to lecturers and students of physiology, sports science, movement studies, physical education and coaching science. Additionally it will provide an educational resource for coaches and physical trainers as well as consultants in fitness and recreational centres that prescribe physical activities and sports for fitness purposes. It will also be of value to specialists in sports medicine and physical therapy in providing insights into sport-specific physiological stresses. For this range of readers it will help to interpret the significance of physiology for the sports that are analysed.

Part One
Exercise

1
Metabolic aspects of exercise

Clyde Williams

1.1 INTRODUCTION

In most sports the limitation to performance is the premature onset of fatigue. Training improves performance in a number of ways and not least, of course, through improvements in skill and greater experience. However, training delays the premature onset of fatigue and this in itself contributes to a significant improvement in performance. Fatigue is not a single phenomenon but the end result of a number of events within the closely coupled chain of reactions which follow the conscious decision to exercise. Inability to maintain a prescribed work task or level of exercise is a common expression of fatigue. The failure of metabolism to provide sufficient energy at the rate required by working muscles, to cover their energy demands, is the most common underlying mechanism for fatigue during dynamic physical activity. This 'energy crisis' in working muscles has different aetiologies as one might expect when one considers the range of physical activities which fall under the general heading of 'Sport'. In order to develop a broad picture of the metabolic support, and of course failure, underlying the performance of dynamic physical activity it is helpful to divide these activities into two general categories, namely the 'multiple sprint' sports and the 'endurance sports' (Williams, 1987). During participation in the multiple sprint sports, fatigue is associated with the accumulation of the end products of metabolism whereas during endurance sports, fatigue is associated with the depletion of the limited stores of carbohydrate in skeletal muscles. Of course many sports involve an unpredictable mixture of sprint and endurance activities and so localizing the cause of fatigue presents the sports scientist with a more complex set of problems. Therefore, the aim of this introductory chapter is to provide the reader with an overview of some of the metabolic responses to exercise, focusing, where appropriate, on mechanisms which attempt to explain the fatigue process and also on metabolic methods of assessing adaptations to training.

1.2 ENERGY BALANCE

The failure of metabolism to provide energy as rapidly as the working muscles require it is a very localized event and can be traced to individual motor units; however it is worthwhile setting these events against a much broader metabolic background. The energy balance equation summarizes, in a simple way, the relationship between food intake, energy expenditure and the fuel stores of the body as follows:

Energy intake = Energy expenditure ± Energy stored

Energy intake is difficult to assess without the complete co-operation of the individual or group under study because it requires an accurate record of all the food and drink consumed over a minimum period of seven days. Useful as this information is, it only provides a snapshot of the energy intake and the composition of the diet over the period of observation. It does not give a comprehensive description of the habitual diet of the individual nor take into account, for example, seasonal variations. Nevertheless without this information it is virtually impossible to assess whether or not individuals involved in sport are matching their energy expenditures with adequate energy intakes. Energy intake and energy expenditure are expressed in terms of heat units, namely kilocalories or more correctly kilojoules (4.18×kilocalories). These units reflect the way in which energy expenditure was, and to a certain extent still is, measured. The heat energy released as a result of metabolic processes can be measured in whole body calorimeters either directly or indirectly by determining the amount of oxygen consumed and carbon dioxide produced over a given period of time. The assessment of energy expenditure during free living is difficult even when using portable systems for measuring oxygen consumption. A compromise approach is one which uses heart rate monitoring or even small accelerometers to derive an estimate of the daily energy expenditure of the individual. Pre-calibration of the individual performing a range of normal activities, in the laboratory, while heart rate and oxygen consumption are measured is a necessary pre-requisite for any study using heart rate as a method of estimating energy expenditure during free living. Only when this preliminary biological calibration procedure is used do the results obtained begin to approach those found in whole body calorimetry studies. The recently developed doubly labelled water ($^2H_2{}^{18}O$) technique has the potential of providing a more accurate method of assessing energy expenditure during free living than the traditional methods (James, Haggarty and McGaw, 1988).

When considering the energy demands of exercise and the nutritional adequacy of the diet for active sportsmen and women it is worthwhile remembering that there is a significant amount of metabolic energy needed simply to fulfil the domestic requirements of the body. Maintenance energy expenditure can be assessed from the basal metabolic rate (BMR); however, in practice it is so difficult to fulfil the requirements necessary to obtain a truly basal condition that resting metabolic rate is measured as a close approximate (RMR). The RMR of someone sitting quietly at rest prior to the start of exercise can be assessed from a knowledge of the oxygen consumption and the respiratory exchange ratio (R) (i.e., the ratio of carbon dioxide production to oxygen consumption). The R value provides an estimate of the relative proportion of fat and carbohydrate being metabolized in order to provide the maintenance energy for normal bodily function. From the R value and the oxygen consumption the quantity of energy released per litre of oxygen consumed can be calculated (Consolazio and Johnson, 1963). It should be recognized, however, that the conversion of the metabolizable energy in food stuffs (which is about 95% of the absolute energy content of the food) captures only about 40% in the form of biochemically usable energy, namely ATP. The remaining 60% is lost as heat and it is this heat which maintains resting body temperature at 37°C. The

resting or maintenance metabolic rate accounts for about two-thirds of the daily energy expenditure and can be estimated from equations based on the age and weight of the individual. For example, for the age range 18–30 years the BMR is estimated from the following equation (World Health Organization, 1985):

For men:	BMR (kcal/24 h)=17.5 W+651
For women:-	BMR (kcal/24 h)=14.7 W+496

From these equations the BMR can be calculated per minute or per hour and then the contribution to energy expenditure of the daily round of activities can be estimated. For example, the energy expenditure during sleeping is calculated as 1.0×BMR whereas the waking resting energy expenditure is calculated as 1.4×BMR. Timing all activities throughout the day and then calculating their energy expenditure from tables of energy expenditure constants provides a useful first approximation of the overall energy expenditure of the individual (WHO, 1985; Durnin and Passmore; 1967). When considering the energy balance equation, factors which influence the resting metabolic rate are probably more important for weight loss, in sedentary individuals, than the increase in energy expenditure through physical activity. While an increase in habitual physical activity, through fitness training programmes, has been shown to decrease body fat in male subjects, the available evidence suggests that similar changes in body composition may not occur so readily in females unless accompanied by a reduced energy intake. It has been suggested that there is an increased efficiency with which food is metabolized in females on restricted dietary intakes, which may be a mechanism to protect limited, yet essential, fuel stores (for review see Brownell, Steen and Wilmore, 1987). It is also important to remember that a matching of energy intake and energy expenditure does not appear to occur on a day by day basis. Extensive studies completed in the late 1960s on military conscripts showed that an apparent energy balance only begins to appear, at least in arithmetical terms, when the period of observation is greater than seven days (Edholm *et al.,* 1970). Endurance runners appear to be able to maintain their energy balance on fairly modest intakes of approximately 2000–3500 kcal per day even though they cover considerable distances in training (Figure 1.1 and Figure 1.2). There is, of course, always the problem of the ‘observer effect’ when collecting information on energy intake when using even the 7 day weighed intake method (Marr, 1971). Nevertheless, it is surprising and encouraging that similar results for energy intake and food composition are obtained for similar groups even when the surveys are carried out by different observers (Short and Short, 1983). In very demanding endurance competitions, such as the Tour de France, energy balance appears to be maintained in spite of large energy expenditures of approximately 6000 kcal per day (25 MJ). The energy intakes required to match energy expenditure of the professional cyclists can

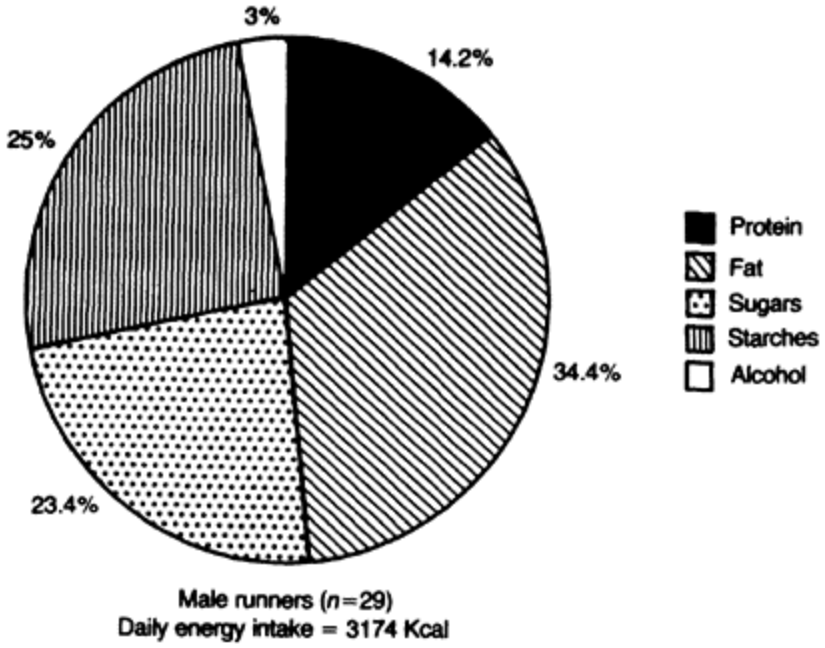

Figure 1.1 Energy intake and composition of male runners.

only be achieved by daily supplementing their habitual diets with concentrated carbohydrate solutions (Saris, van Erp-Baart and Brouns, 1989). Should these cyclists be unable to consume high-energy diets on a daily basis then they may not be able to complete the 22 days of competition because of the lack of opportunity to make up the deficits incurred. While failure to maintain an energy balance, especially in terms of the carbohydrate intake, will inevitably lead to a reduction in physical performance, it is only on rare occasions that it is a life-threatening event. One such dramatic and tragic example was the loss of the famous Scott expedition in 1913 during its attempt to be the first to reach the South Pole. The daily energy expenditures of these explorers has recently been estimated to have been between 5000 kcal (21 MJ) and 7000 kcal (29 MJ)/day which was far in excess of their energy intakes of approximately 4300 kcal (18 MJ)/day. It has been suggested that this energy deficit was mainly responsible for the inability of Scott and his four companions to complete successfully the return journey from the South Pole rather than vitamin C deficiency (scurvy) (Stroud, 1987).

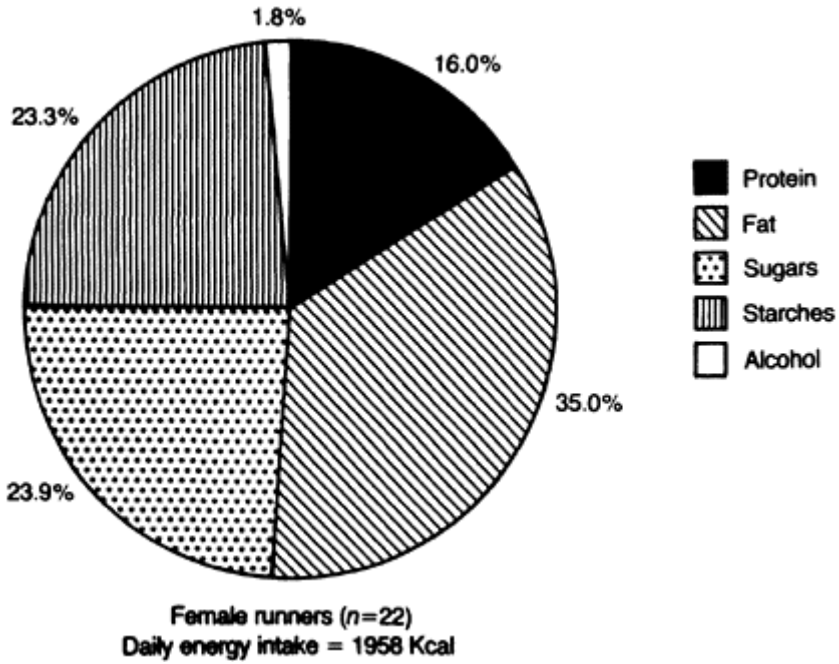

Figure 1.2 Energy intake and composition of female runners.

1.3 ENERGY STORES

The fuel for energy production is stored in the form of carbohydrate and fat whereas protein, which is the principal constituent of muscle, tends to be used as a fuel only when carbohydrate stores are particularly low (Lemon and Mullen, 1980; Callow, Morton and Guppy, 1986). Carbohydrate is stored in skeletal muscles and liver as a polymer of glucose called glycogen. Expressed in terms of glucose (glucosyl) units the glycogen concentration of human skeletal muscle is in the range 60–150 mmol kg^{-1} wet weight (w.w.) or 258–645 mmol kg^{-1} dry weight (d.w.). The metabolic intermediates in the stepwise degradation of glycogen are shown in Figure 1.3 together with the points in the pathway at which the rate of glycolysis can be controlled. The degradation of glycogen to form the phosphorylated glucose, glucose 6-phosphate, is controlled by the enzyme phosphorylase, namely phosphorylase *a*. The activation of phosphorylase, and hence glycogenolysis, occurs as a result of an increase in the sarcoplasmic concentration of Ca^{2+} and also as a result of an increase in circulating adrenaline. Once formed,

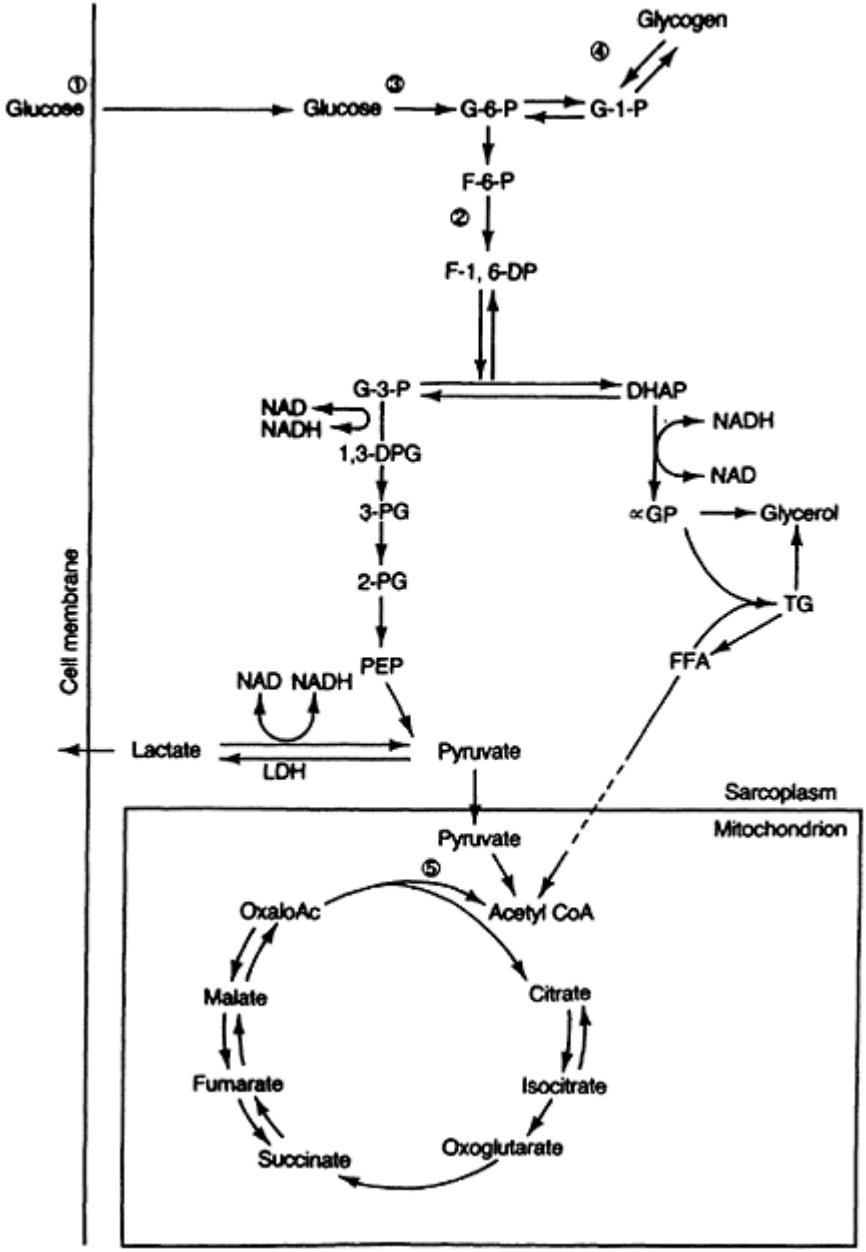

Figure 1.3 Metabolic pathways of glycogenolysis and glycolysis in muscle. The numbers indicate the various points at which the rates of glycogenolysis and glycolysis can be controlled. Entry of blood glucose into the cell; (2) conversion of fructose-6-phosphate to fructose 1, 6-diphosphate by the enzyme phosphofructokinase; (3)

phosphorylation of glucose by the enzyme hexokinase; (4) glycogen degradation by the enzyme phosphorylase; (5) conversion of pyruvate to acetyl CoA by the enzyme pyruvate dehydrogenase.

Abbreviations: G-6-P, glucose-6-phosphate; G-1-P, glucose-1-phosphate; F-6-P, fructose 6-phosphate; F-1, 6DP, Fructose 1,6 diphosphate; G-3-P, glyceraldehyde-3-phosphate; 2-PG, 2-phosphoglycerate; PEP, phosphoenolpyruvate; DHAP, dihydroxyacetone phosphate; alpha GP, alpha-glycerophosphate; NAD, nicotinamide-adenine dinucleotide; NADH, nicotinamide-adenine dinucleotide (reduced form); LDH, lactate dehydrogenase; TG, triglyceride.

glucose 6-phosphate is indistinguishable from the glucose 6-phosphate which is produced from the entry, into muscle, of blood glucose. The phosphorylation of glucose in this case is the result of the activity of the enzyme hexokinase, which is thought to be located on the inner membrane of the sarcolemma and to be involved in the control of glucose uptake by muscle. An increase in the glucose 6-phosphate concentration exerts an inhibitory influence over the activity of hexokinase. Thus, during the early part of exercise, when the glycogen and glucose 6-phosphate concentrations are high, the rate of phosphorylation of glucose is low, as is the uptake of glucose from the blood. However, during the later stages of prolonged exercise, when the glycogen concentration is low, the inhibition of hexokinase is lifted in the presence of a reduced concentration of glucose 6-phosphate producing intracellular conditions which are conducive to an increased influx of blood glucose (Wahren, 1973; Bonen *et al.,* 1981).

The rate of stepwise degradation of glucose 6-phosphate, called glycolysis, can be changed by modifying the activity of the control enzyme phosphofructokinase (PFK). Thereafter the degradation process proceeds to the formation of pyruvate which has a number of potential fates. The main one, after entering the mitochondria, is aerobic metabolism which results in the production of carbon dioxide, water and, of course, the oxidative phosphorylation of ADP to form ATP. When the rate of pyruvate formation exceeds the capacity of the available mitochondria to accept this glycolytic product it is converted into lactate or even alanine. The glycogen to pyruvate part of the pathway, or more correctly the Embden-Meyerhof pathway, normally has a greater capacity for pyruvate formation than has muscle to oxidize the pyruvate formed (Keul, Doll and Keppler, 1967). The fact that the Embden-Meyerhof pathway is a non-oxidative pathway or more commonly called an anaerobic pathway has sometimes caused confusion. The increased activity of this anaerobic pathway does not mean that it is proceeding in an anaerobic environment nor necessarily in a hypoxic environment. Notwithstanding this, however, a decrease in the available oxygen in the muscle cell will lead to an increase in the activity of this pathway (orginally known as the Pasteur effect). During glycolysis, glucose 6-phosphate, which is a six carbon molecule, is converted into two three carbon pyruvate molecules. Thus this increased glycolytic activity during exercise increases the number of molecules and as such would appear to change the osmotic balance within muscle cells. However, there are approximately 3–4 g of water stored with every gram of glycogen and so the degradation of glycogen may not, therefore, cause disruptive osmotic changes in muscle during exercise.

The size of the store of glycogen in the liver depends on the nutritional state of the individual. For example, in the fed state the adult liver weighing about 1.8 kg, contains approximately 90 g or 550 mmol of glucosyl units, whereas after an overnight fast the concentration of glycogen falls to about 200 mmol but after a number of days on a high carbohydrate diet it can increase to as much as 1000 mmol (Nilsson and Hultman, 1973). Interestingly, however, an overnight fast does not appear to lower muscle glycogen concentration as it does with liver glycogen (Maughan and Williams, 1981). Liver glycogen is the reservoir from which glucose is released in order to maintain blood glucose concentrations within a fairly narrow range of values and it is under the control of glucagon, a hormone which is released from the alpha cells of the Islets of Langerhans, in the pancreas, when blood glucose concentrations decrease (Newsholme, 1976). The central nervous system uses approximately 120 g of blood glucose a day as its main, but not exclusive, substrate for energy metabolism and so a reduction in blood glucose concentrations to low levels, i.e. hypoglycaemia, is frequently accompanied by dizziness and headaches. The passage of glucose into the liver, as a result of the digestion and absorption of carbohydrate foods, is not under hormonal control as is the entry of glucose into adipose tissue and muscle cells. Insulin, released from the beta cells of the Islets of Langerhans, regulates the uptake of glucose into muscle and fat cells but during exercise there is a decrease in insulin concentration which is inversely related to the circulating noradrenaline concentration (Pruett, 1970). The increase in noradrenaline concentration is the result of the outflow of this neurotransmitter from the sympathetic nerves, and also from the adrenal medulla. It is the increase in the concentration of this catecholamine during exercise which suppresses the release of insulin from the pancreas (Porte and Williamson, 1966). Carbohydrate metabolism in skeletal muscle during prolonged submaximal exercise, such as endurance running, can be sustained at a rate of 2.5–3.0 g min^{-1} (Williams, Brewer and Patton, 1984); therefore, if muscle had free access to blood glucose then there would be a rapid onset of severe hypoglycaemia. Thus the reduction in plasma insulin concentration during exercise does, in part, prevent the flow of blood glucose into working muscles.

Fat is stored in adipose tissue of which there are two types, namely white adipose tissue cells (WAT) and brown adipose tissue cells (BAT). Fat is stored as triacylglycerol (triglycerides) in these cells and also in skeletal muscles. White adipose tissue cells are the long-term storage sites for fat and it is from these cells that fatty acids are mobilized for use as a metabolic fuel for energy metabolism. Brown adipose tissue has a more specialist function in that it is apparently involved in the regulation of energy balance and also in cold-induced thermogenesis. Whereas WAT is innervated by relatively few sympathetic nerves and relies mainly on circulating catecholamines to stimulate the mobilization of fatty acids, BAT is richly supplied by sympathetic nerves and capillaries (Trayhurn and Ashwell, 1987). Unlike WAT, which has relatively few mitochondria, the mitochondrial density of BAT is very large and it is this characteristic which gives BAT its colour and more importantly, its capacity to increase its metabolic rate severalfold. Whereas in most cells the aerobic degradation of substrate is closely coupled to the formation of ATP by the process of oxidative phosphorylation, in BAT cells metabolism is able to proceed at a high rate in the presence of uncoupling of oxidative phosphorylation. The BAT cells are therefore, uneconomical in the conversion of fatty

acids into ATP but, as a result of this energy-wasting activity, they are very good heat generators. Most of the studies on the physiological roles of BAT have been conducted on laboratory animals and little work has been carried out on human subjects. Therefore the contribution of BAT to the regulation of energy balance in response to overfeeding, and its contribution to increased heat production in response to cold exposure, in adult man has yet to be clearly established (Nicholls and Locke, 1984).

The average adult male has about 15% of his body weight as fat whereas lean females have about 20% of their body weight as fat. Even the very lean, almost emaciated-looking, male distance runners have a body fat content of approximately 5–10%; the very lean female distance runner has about 10% of her body weight as fat though lower values have been reported (Chapter 5). A relatively large proportion of this stored fat is available as a fuel during exercise and the more of the fatty acids used, the greater the sparing of the body's limited glycogen stores. Whereas glycogen is stored in association with water, fat is stored in the anhydrous form and so weight for weight it can provide more energy than carbohydrate. More importantly, however, the complete oxidative metabolism of one gram of fat yields approximately 39 kJ (9.3 kcal) whereas the oxidative metabolism of one gram of carbohydrate (glucose) yields only 16.7 kJ (3.75 kcal). Therefore, if a 70 kg man were required to store the glycogen equivalent of fat, instead of fat, then his body weight would increase by about 55 kg. This calculation, together with the knowledge that endurance training increases the capacity of working muscles for fat oxidation, helps explain why fat rather than carbohydrate is the ideal fuel for prolonged activity whether it be for the endurance runner or nature's remarkable endurance athletes, namely birds during their annual migration.

The triacylglycerol in adipose tissue and in muscle, is made up of free fatty acids and glycerol and in WAT it is stored almost entirely as a single droplet whereas in BAT it is stored as a number of droplets. Hydrolysis of triacylglycerol results in the release of free fatty acids (FFA) and glycerol in a ratio of approximately three to one. The FFA are released from the white adipose tissue cells located around the body and are transported in loose combination with plasma albumin in the systemic circulation. The amount of FFA taken up by working muscles is dictated by their plasma concentration and the blood flow (Gollnick, 1977). Therefore it is important to recognize that an increase in perfusion rate without an accompanying increase in plasma concentration will increase FFA uptake by working muscles. However, the metabolism of FFA is not directly proportional to their uptake by active muscles because their oxidation is determined by the number of mitochondria in muscle (Gollnick and Saltin, 1982). Those free fatty acids which are not immediately oxidized in the mitochondria are stored as intramuscular triglycerides which increase with training and decrease during prolonged exercise (Jansson and Kaijser, 1987). While there is a greater amount of energy released as a result of the aerobic metabolism of FFA than following the aerobic metabolism of an equal amount of carbohydrate, the rate of production is slower from fatty acids than from glycogen (McGilvery, 1975). The limiting factor is not the mobilization or transport of FFA to the working muscles, nor, during submaximal exercise the supply of oxygen, but the limited number of mitochondria available to take advantage of this high-energy substrate (Gollnick and Saltin, 1982).

Glycerol and those fatty acids not taken up by muscle pass into the

Table 1.1 ATP yield from aerobic and anaerobic metabolism

Reaction		*ATP per mole of substrate*	*Respiratory exchange ratio R*
Glvcoeen	. . . . . lactate	3	—
Glvcoeen	. . . . . CO_2, H_2O	37	1.00
Glucose	. . . . . lactate	2	—
Glucose	. . . . . CO_2, H_2O	36	1.00
Fatty acids	. . . . . CO_2, H_2O	138	0.70
Acetoacetate	. . . . . CO_2, H_2O	23	1.00
3-OH buterate	. . . . . CO_2, H_2O	26	0.80

(After McGilvery, 1975.)

liver where they are either converted back into triglycerides and then packaged in a protein coat, before being turned out into the general circulation as chylomicrons or the glycerol contributes to the regeneration of liver glycogen by the process known as gluconeogenesis. Fatty acids entering the liver are also oxidized as an energy source, however, when the liver glycogen concentration is low, such as during starvation or very prolonged submaximal exercise, only partial oxidation of fatty acids occurs resulting in the production of ketones. Ketones, namely acetoacetate and 3-hydroxybutyric acid can be used as a substrate for brain but, apparently, not for muscle metabolism (Robinson and Williamson, 1980). Thus the particular fates of glycerol and fatty acids in the liver depend on the nutritional status of the individual in general, and the concentration of liver glycogen in particular. In contrast to the mobilization of triglycerides into fatty acids and glycerol, which is almost entirely under the control of the catecholamines, the degradation of muscle glycogen is mainly initiated by the contractile activity of the muscle itself.

In addition to the two metabolic fuels, glycogen and fatty acids, the muscle has a small amount of ATP and a relatively small store of another high-energy compound called phosphocreatine (PCr) which is four to five times the size of the ATP store. The energy yield, in terms of ATP, from the main metabolic pathways are summarized in Table 1.1.

1.4 MUSCLE MORPHOLOGY

The aerobic metabolism of glycogen and fatty acids occurs in muscle fibres which are characterized by their high oxidative capacities, their apparently slow speeds of contraction and their high endurance quality. Fast contracting, fast fatiguing muscle fibres rely mainly on glyco-genolysis for the resynthesis of ATP because of their lower oxidative capacities and the nature of their function (Barnard *et al.,* 1971; Burke *et al.,* 1971). These two populations of fibres are found in all muscles in different proportions and it is the presence of a greater amount of one or the other which dictates whether the muscle has predominantly fast or slow contractile speeds. The speed with which a muscle fibre contracts is related to the rate at which the energy-releasing conversion of ATP to ADP occurs. The splitting of the high energy phosphate, ATP, is catalysed by the enzyme myosin ATPase (myofibrillar adenosine triphosphatase) and muscles with high contractile speeds have been found to have high myosin ATPase activities (Barany, 1967).

The presence of the enzyme myosin ATPase can be located in muscle fibres by using histochemical techniques (Padykula and Herman, 1955), and so the proportion of fast and slow fibres in a muscle, can be estimated (Dubowitz and Brooke, 1973). Such techniques rely on a visual identification of reaction products from the catalytic activity of the enzyme. Factors such as temperature, pH and fixation need to be carefully controlled because they have an important influence on enzyme activity (Brooke and Kaiser, 1970).

In addition, these histochemical procedures also allow a qualitative assessment of the oxidative and glycolytic potential of the fast-contracting and the slow-contracting fibres (Essen *et al.,* 1975; Saltin *et al.,* 1977). The oxidative capacity of muscle is reflected by the activity of a number of enzymes, one of which is nicotinamide adenine dinucleotide-tetrazolium reductase (NADH-TR, also called NADH diaphorase); the enzyme used as an indicator of glycolytic potential in skeletal muscle, is α-glycerophosphate dehydrogenase (α-GPDH). By using these histochemical methods, each muscle fibre can be described not only in terms of its relative speed of contraction, e.g. fast twitch or slow twitch, but also in terms of its predominant means of producing energy, i.e., by oxidative or glycolytic metabolism. Three populations of muscle fibres are generally recognized when several sections of muscle are analysed by these procedures. They have been described as slow twitch oxidative (SO), fast twitch oxidative and glycolytic (FOG) and fast twitch glycolytic (FG) (Peter *et al.,* 1972). A more conservative nomenclature is favoured when describing human muscle, which is, Type I, Type IIa and Type IIb in place of SO, FOG and FG. The reason for the more conservative nomenclature is that whereas in animal studies both the histochemical profile and the contractile characteristics of muscle fibres can be determined directly, in studies using human subjects only the histochemical profiles of muscle fibres can be precisely described (Dubowitz and Brooke, 1973). Studies on the fibre composition of muscle from elite athletes have shown, as might be expected, that the endurance runners have a high proportion of Type I and Type IIa fibres whereas the elite sprinters have a high proportion of Type IIb fibres and only a small proportion of Type I fibres (Gollnick *et al.,* 1972; Costill *et al.,* 1976a, b).

The value of the histochemical characterization of human muscle fibres is extended by

inclusion of a staining procedure for glycogen, because it allows the exercise-induced changes in glycogen concentration to be described for each population of fibres. Thus from changes in the intensity of the glycogen stain, the recruitment pattern of each of the muscle fibre populations can be described (Kugelberg and Edstrom, 1968). The results of studies using these techniques on both animal and human muscles have shown that the recruitment pattern during exercise of increasing intensity is as follows: Type I>Type IIa>Type IIb (Kugelberg and Edstrom, 1968; Gollnick *et al.,* 1973, 1974; Edgerton *et al.,* 1975). During prolonged high-intensity exercise, such as cross-country running, glycogen depletion occurs in the Type I fibres and the inability of these fibres to maintain the desired contractile rate is probably responsible for the onset of fatigue (Costill *et al.,* 1973). While glycogen depletion is not a limiting factor during a brief period of high intensity exercise, such as sprinting, it may be a contributory factor during brief intermittent high intensity exercise such as in the multiple sprint sports of Rugby, soccer and hockey (Saltin, 1973; MacDougall *et al.,* 1977). High intensity exercise requires the contribution of both the Type I and Type II fibres and there is some evidence to suggest that the Type II, i.e., fast twitch fibres, experience glycogen depletion more rapidly than the Type I fibres (Edgerton *et al.,* 1975; Essen, 1978a). This is not unexpected, of course, because the Type IIb fibres have a low aerobic capacity and derive their energy mainly from glycogenolysis.

In studies using laboratory animals the conversion of fibre populations has been shown to occur as a result of, for example, manipulating the thyroid status of the animals (Ianuzzo *et al.,* 1977), training and even prolonged electrical stimulation of selected skeletal muscles (Petite, 1986). The evidence for the conversion of fibre types in human subjects, following prolonged training, is not so convincing, but this is not in itself a denial of the plasticity of human skeletal muscle. There is a recent study, for example, which shows that very prolonged exercise, lasting several weeks, has profound influences on human muscle fibre composition which could be interpreted as an increase in the proportion of Type I fibres at the expense of the Type IIb fibres (Sjostrom, Friden and Ekblom, 1987).

1.5 SUBMAXIMAL EXERCISE

During low intensity exercise which can be sustained for more than

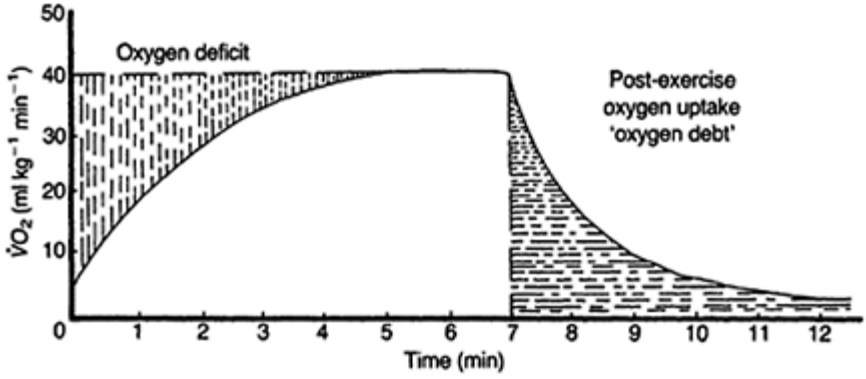

Figure 1.4 Schematic diagram of oxygen deficit/oxygen debt phenomena.

several minutes, such as walking, swimming or jogging, the energy needs of working muscles are provided by aerobic metabolism. This is easily demonstrated by measuring the oxygen uptake of an individual at different time intervals during exercise of constant intensity. At the start of exercise, however, the amount of oxygen used is less than that required and only after several minutes does the oxygen consumption reach a steady state where oxygen demand appears to be met by oxygen supply (Figure 1.4). The difference between the supply and demand for oxygen at the beginning of exercise is called the 'oxygen deficit' (Åstrand and Rodahl, 1970). The delay in achieving a steady state of oxygen consumption during submaximal exercise of constant intensity has been explained in terms of the sluggishness with which the cardiovascular system delivers oxygen at the onset of exercise (Margaria *et al.,* 1963). However, it must be appreciated that an increase in oxidative metabolism is stimulated by an increase in ADP concentration in the mitochondria. The contractile activity of muscle at the start of exercise increases the ADP concentration in the sarcoplasm and so it takes time for the translocation of the ADP from the sarcoplasm into mitochondria. Therefore, the delay in achieving a steady-state oxygen consumption is not simply the response of a sluggish cardiovascular system but is associated with the cellular events which are responsible for increasing metabolic rate. During the onset of exercise the deficit in the aerobic production of energy is covered by contributions from the following three non-oxidative or anaerobic reactions, namely,

$$PCr + ADP \rightarrow ATP + Cr$$
$$ADP + ADP \rightarrow ATP + AMP$$
$$Glycogen \rightarrow 3\ ATP + Lactate + H^+$$

Therefore, even during low intensity exercise there is an increase in muscle lactate concentration, and hence blood lactate concentrations, if only transiently, at the onset of exercise as shown over half a century ago by Bang (1936). However, as the duration of exercise progresses there is an ever-increasing contribution from aerobic metabolism to the energy requirements of the working muscles and a point will be reached where nearly

all the oxygen demand is met by the oxygen supplied, i.e. an aerobic steady-state.

Training increases the capillary and mitochondrial density in skeletal muscles (Ingjer, 1979) and so these changes would be expected to contribute to a faster rate of oxygen consumption at the onset of exercise and hence a reduction in the oxygen deficit. There is some evidence to support this proposition from studies of the oxygen transport kinetics of trained and untrained individuals at the start of exercise (Weltman and Katch, 1976; Hagberg, Nagle and Carlson, 1978; 1980). However, the greater rate of oxygen transport is not simply the result of differences in the size of an individual's $\dot{V}O_{2max}$ as has been suggested in some studies (Hagberg, Nagle and Carlson, 1978; Powers, Dodd and Beadle, 1985), but more likely to be related to the training status of the individual (Hickson, Bomze and Holloszy, 1978). For example, in a study which examined the half-time of the oxygen uptake at the onset of submaximal exercise in male and female runners of similar training status but with different $\dot{V}O_{2max}$ values, there was no significant relationship with either the $\dot{V}O_{2max}$ values or the absolute running speeds (Lake *et al.,* 1986). However, the link between improvements in performance and oxygen uptake kinetics as a result of training in already well-trained athletes has, as yet, to be fully examined.

During prolonged exercise of submaximal intensity the motor units of Type I and IIa fibre are mainly responsible for locomotion (Vollestad, Vaage and Hermansen, 1984). When the glycogen concentration in these muscle fibres is reduced below a critical value they are then unable to continue their contribution to contractile activity, at the required rate, and so fatigue occurs. The limited glycogen stores in skeletal muscle can be increased in preparation for prolonged exercise by increasing the carbohydrate content of the diet during the three days prior to competition (Hultman, 1967; Costill, 1988). Carbohydrate loading, as the dietary and exercise preparation for endurance competition is popularly known, can even be achieved by simply supplementing, rather than radically changing, the normal diet with either simple or complex carbohydrates (Roberts *et al.,* 1988). Both types of carbohydrate supplementation have been shown to produce a significant improvement in endurance running capacity (Brewer, Williams and Patton,

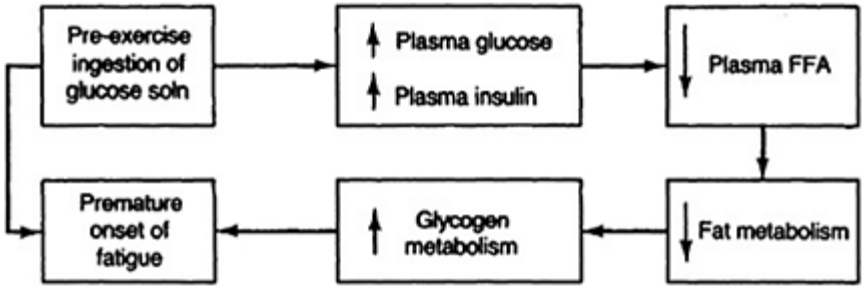

Figure 1.5 Diagram describing paradoxical onset of fatigue following pre-exercise ingestion of glucose solutions.

1988). Supplementing the carbohydrate stores of the body in the hour before the start of exercise by consuming carbohydrate-containing solutions does not appear to be of benefit

to the endurance capacity of the individual. One study has shown that ingesting a concentrated glucose solution 45 minutes before 30 minutes of treadmill running produces hypoglycaemia, a reduction in plasma free fatty acid concentrations and a greater glycogen utilization than occurs following the pre-exercise ingestion of water alone (Costill *et al.,* 1977). Furthermore, the supplementation of the body's carbohydrate stores in this way has been reported to reduce endurance capacity by 19% (Foster, Costill and Fink, 1979). This paradoxical early onset of fatigue is mediated through a glucose-stimulated increase in plasma insulin concentration as outlined in Figure 1.5. Reducing the availability of plasma-free fatty acids, for muscle metabolism, shifts the choice of fuel to carbohydrate and so there is an even greater demand on the limited glycogen stores. This increase in carbohydrate metabolism has also been clearly demonstrated in a series of exercise experiments in which the mobilization of fatty acids was depressed, by the pre-exercise administration of nicotinic acid (Bergstrom *et al.,* 1969; Pernow and Saltin, 1971).

Glucose solutions ingested immediately before exercise and during exercise, however, do not provoke the same degree of hyperinsulinaemia as occurs during rest. The explanation for this difference is that the rise in noradrenaline concentration, which accompanies the onset of exercise, inhibits the release of insulin (Porte and Williamson, 1966; Pruett, 1970), Fructose solutions have been used as a carbohydrate supplement immediately before and during exercise in an attempt to avoid the hyperinsulinaemia and to spare the limited glycogen stores, however, this has not yet proven to be as successful as initially predicted (Williams, 1989). The most notable study to demonstrate the benefits of ingesting concentrated carbohydrate-containing solutions immediately before and during exercise reported that trained cyclists extended their performance times by an hour beyond the time they were able to achieve while consuming only water. In this study there was no evidence, however, of a glycogen-sparing effect as a result of ingesting the carbohydrate solution. After three hours of cycling at 77% $\dot{V}O_{2max}$ the muscle glycogen concentrations were the same for both the water and the carbohydrate experiments. However, during the carbohydrate experiments the subjects continued cycling for a further hour suggesting that the exogenous carbohydrate was making a significant contribution to energy metabolism during the later stages of exercise (Coyle *et al.,* 1986). An increased fat metabolism during exercise has been shown to exert a glycogen-sparing effect and produce an improvement in endurance capacity (Rennie, Winder and Holloszy, 1976). Caffeine in amounts equivalent to three cups of coffe has been reported to increase mobilization of fatty acids and to increase the endurance capacity of trained cyclists when taken prior to exercise (Ivy *et al.,* 1979). By way of contrast it is interesting to note that the carbohydrate loading procedure will reduce the fasting plasma FFA concentrations below normal and yet this procedure results in an improvement in endurance performance (Brewer, Williams and Patton, 1988). The decreased plasma FFA concentrations, following carbohydrate loading, are accompained by an incresed carbohydrate loading, are aacomplished by an increased carbohydrate metabolism even at exercise intensities which would normally be covered by a greater amount of fat metabolism (Maughan *et al.,* 1978). Whether this is a result of a decreased plasma FFA concentration *per se* or a change in the kinetics of glycogenolysis as a result of the increased glycogen concentrations remains to be established (Richter and Galbo, 1986). Nevertheless,

carbohydrate loading combined with the pre-exercise ingestion of caffeine would appear to be the ideal nutritional perparations for prolonged exercise. However, the increased fatty acid mobilization and metabolism induced by the ingestion of caffeine does not appear to be effective in subjects who have undergone carbohydrate loading (Weir *et al.,* 1987).

The most effective method of achieving glycogen sparing and an improvement in endurance capacity *is,* of course, training (Karlsson, Nordesjo and Saltin, 1974; Jansson and Kaijser, 1987). A clear illustration of glycogen sparing and thus improvement in endurance capacity are seen in a study in which subjects undertook six weeks of endurance training using one leg cycling in the trained leg improved by approximately 500% after training whereas $\dot{V}O_{2max}$ improved by 22%. The glycogen utilization and lactate production of the quadriceps muscles after approximately 30 minutes of cycling were significantly less after training compared with the values obtained before training (Table 1.2). The reduction in glycogenolysis and the lower muscle lactate concentrations, in the trained leg during exercise, does not support the proposition that low blood lactate concentrations after training are mainly the result of a more rapid lactate clearance after training are mainly the result of a more rapid lactate clearance rather than a decreased lactate production (Donovan and Brooks, 1983). The restoration of muscle glycogen stores may take as long as two days, especially

Table 1.2 Changes in muscle metabolism during single-leg submaximal exercise following endurance training ($mmol^{-1}$ $kg^{-1}dw$)

	Trained Leg	
	Pre-training	*Post-training*
Glycogen	–250.0	−91.7*
PCr	−27.1	−9.1
ATP	−2.0	−0.4
ADP	+0.46	+0.19
Glu-1-P	+1.02	+1.02
Fru-1-P	+0.11	−0.01
Fru-1,6-DP	−0.02	+0.20
Pyruvate	+0.59	+0.56
Lactate	+48.70	+11.10*

*Significantly different from pre-training values ($p<0.05$).

in well-trained individuals who have high pre-exercise concentrations (Piehl, 1974). In the immediate post-exercise period, however, the permeability of skeletal muscle membranes to glucose appears to be greater than exists prior to exercise and so in conjunction with an increased activity of glycogen synthase the conditions appear to be

conducive for rapid glycogen resynthesis (Holloszy and Narahara, 1965; Fell *et al.,* 1982). To take maximal advantage of these optimal conditions for glycogen resynthesis carbohydrate should be consumed immediately after exercise because to delay the provision of exogenous glucose appears to lead to an incomplete restoration of muscle glycogen (Ivy *et al.,* 1988).

1.6
ANAEROBIC THRESHOLD

During exercise of increasing intensity there is a rise in blood lactic acid concentration and this response was first reported over half a century ago (Owles, 1930; Bang, 1936). While the appearance of lactate in blood during exercise is the result of an increased glycogenolysis, it is important to recognize that its concentration is, at any time, the result of a balance between the rates of production and removal (Brooks, 1986). Nevertheless during exercise of increasing intensity, the rise in blood lactate concentration is an indication of increased glycogen metabolism (Saltin and Karlsson, 1971). This increase in blood lactate concentration has been interpreted as a reflection of the onset of hypoxia in skeletal muscles and the exercise intensity at which anaerobic metabolism complements the regeneration of ATP by aerobic metabolism has been called the 'anaerobic threshold' (Wasserman and Mcllroy, 1964; Wasserman *et al.,* 1973; Davis, 1985; Katz and Sahlin, 1988). The identification of the anaerobic threshold from an examination of the non-linear increases in pulmonary ventilation rates is based on the simple premise that hypoxia in working skeletal muscles leads to the formation of lactic acid which leaves the muscle and stimulates respiration (Wasserman and Mcllroy, 1964; Wasserman *et al.,* 1973). The translocation of the lactic acid from the skeletal muscle to the venous circulation probably occurs as lactate and hydrogen ions independently of each other (Mainwood and Renaud, 1985). The hydrogen ions are buffered by the plasma bicarbonate resulting in an increase in carbon dioxide production and an accompanying increase in pulmonary ventilation. This is known as respiratory compensation for metabolic acidosis and is one of the first lines of defence against the development of acidosis. More recently the terms 'ventilatory threshold' and 'lactate threshold' have been used when attempting to define the anaerobic threshold from changes in respiratory and blood lactate responses to exercise. However, the premise that an increase in blood lactate concentration during exercise of increasing intensity reflects hypoxia in working muscles is not a widely accepted one (Gollnick and Hermansen, 1973; Brooks, 1985). There is persuasive evidence from animal studies to suggest that lactate production can occur under aerobic conditions (Connett, Gaueski and Honig, 1984) and that mitochondrial function is only impaired at extremely low partial pressures of oxygen (Chance and Quistorff, 1978). Although there is apparently not the same evidence available from human studies, there are studies which show that blood lactate concentrations can be increased during submaximal exercise (Jansson, Hjemdahl and Kaijser, 1986) and during maximal exercise (Spriet, Ren and Hultman, 1988) by stimulating glycogenolysis through the infusion of adrenaline. This limited circumstantial evidence suggests that lactate formation during submaximal exercise may not be the

result of hypoxia but there may be other reasons for an increase in lactate concentration. For example, the transient increase in blood lactate concentration at the start of low-intensity exercise may simply be the consequence of fibre recruitment because when muscle fibres begin to contract there is an increase in glycogenolysis and as a result the production of lactate. Therefore, it is not unreasonable to expect that at the onset of exercise newly recruited muscle fibres, or more correctly motor units, will contribute some lactate and hydrogen ions to the general circulation. At higher exercise intensities, however, blood lactate does not decrease but increases as exercise continues, suggesting that the rate of production exceeds the rate of removal. Another explanation for the increase in blood lactate concentration during exercise of increasing intensity could be the net effect of differential metabolic rates of the Embden-Meyerhof and mitochondrial oxidative pathways (Keul, Doll and Keppler, 1967).

Nevertheless the concept of an 'anaerobic threshold' is a very attractive one because it offers a method of identifying the exercise intensity at which anaerobic metabolism makes a significant contribution to the provision of ATP. In theory, the anaerobic threshold provides, in a non-invasive way, a picture of what may be happening in working muscles, albeit however, not in sharp focus. It is now well established that endurance training increases the exercise intensity at which there is a significant rise in blood lactic acid concentration (Williams *et al.,* 1967; Hurley *et al.,* 1984). This improvement in aerobic capacity is a consequence of a training-induced increase in the number of capillaries surrounding the Type I and Type IIa fibres along with an increase in the number of mitochondria within these populations of fibres (Ingjer, 1979; Gollnick and Saltin, 1982). These changes should be detectable as changes in the anaerobic threshold whether it is measured as the lactate or the ventilatory threshold.

Thus in theory, the anaerobic threshold concept offers a submaximal method of assessing responses to training and also a way of describing the 'aerobic capacity' of an individual in terms of the $\%\dot{V}O_{2max}$ (Williams *et al.,* 1967; Lafontaine, Londeree and Spath, 1981; Williams, Brewer and Patton, 1984). This particular definition of aerobic fitness allows individuals with different $\dot{V}O_{2max}$ values to be compared because the anaerobic threshold responds to endurance training and is independent of $\dot{V}O_{2max}$ *per se*. For example, the aerobic fitness of males and females can be compared on a basis which reflects their training background rather than simply having separate scales for males and another for females, as presently exists when $\dot{V}O_{2max}$ alone is used as a measure of fitness (Ramsbottom *et al.,* 1989). Furthermore, the concept of an anaerobic threshold is also appealing because it may be more sensitive to training-induced adaptations than $\dot{V}O_{2max}$ alone. This is especially useful for assessing the adaptations to training of well-trained individuals who often show little additional improvements in $\dot{V}O_{2max}$ with further training, but significant improvements in endurance capacity (Daniels, Yarborough and Foster, 1978; Williams and Nute, 1986).

When it comes to the methods of assessing the anaerobic threshold, however, there is less than universal agreement about how it should or can be measured. There is considerable support for the idea that the anaerobic threshold can be determined from the ventilatory responses to exercise of increasing intensity. The exercise of choice is cycling

and the protocol favoured is one which involves an increase in exercise intensity every minute (Wasserman, 1986). The respiratory changes used to detect the anaerobic threshold tend to be the $\dot{V}_E/\dot{V}O_2$ rather than $\dot{V}_E$ alone as originally suggested by Wasserman *et al.,* (1973) (Caizzo *et al.,* 1982; Yoshida *et al.,* 1981). However, it is common practice to use a number of respiratory responses to exercise in an attempt to confirm the inflection or break points used to identify the anaerobic threshold or more correctly the 'ventilatory threshold' (Yoshida *et al.,* 1981). Although the appearance of break points at different exercise intensities for different respiratory responses has been suggested as a means of identifying the anaerobic threshold, the lack of clarity surrounding the identification of the 'Ventilatory threshold' is a cause for concern. For example, it is common practice for authors to report that the ventilatory threshold was identified by two or more independent observers in order to introduce objectivity into the process (Buchfuhrer *et al.,* 1983; Farrell and Ivy, 1987). Some authors report that a mathematical treatment of the appropriate respiratory variable should be used to locate the 'threshold' in an attempt to achieve objectivity and reliability (Orr *et al.,* 1982). Other authors, using this latter approach, question the existence of any real abrupt changes in the respiratory responses to exercise of increasing intensity (Shorten and Williams, 1982). To add to this debate only some (Caizzo *et al.,* 1982; Yoshida *et al.,* 1981; Ivy *et al.,* 1980) but not all investigators find a high correlation between the ventilatory and the lactate thresholds. There is also some evidence which suggests that these two thresholds can be manipulated independently of each other by using different exercise protocols (Hughson and Green, 1982), or lowering muscle glycogen concentrations prior to exercise (Hughes, Turner and Brooks, 1982) or even using subjects who are unable to produce lactic acid, namely patients with McArdle's disease (Hagberg *et al.,* 1982).

The lack of general agreement about how best to identify the elusive 'anaerobic threshold' may of course be an indication that the signals are too weak and too readily masked by metabolic and respiratory 'noise' to provide reproducible and precise indications of changes in muscle metabolism. It is then surprising that such good agreement has been found between changes in blood lactate concentrations and the ventilatory threshold during exercise of increasing intensity (Ivy *et al.,* 1980). Nevertheless while the physiological mechanism underlying the anaerobic threshold and even its existence has (Jones and Ehrsham, 1982; Davis, 1985; Brooks, 1985) and continues to be debated (McLellan, 1987), many authors continue to use it to describe the aerobic fitness of their subjects as an adjunct to $\dot{V}O_{2max}$ (Bunc *et al.,* 1987; Vago *et al.,* 1987).

Irrespective of how close the link is between the changes in blood lactate concentrations and the ventilatory threshold, blood lactate values during submaximal exercise have been shown to have good correlations with running performance and in some cases they are stronger than those obtained between $\dot{V}O_{2max}$ and performance (Jacobs, 1986; Ramsbottom, Nute and Williams, 1987). Some authors have chosen to examine the relationship between lactate thresholds and performance (Farrell *et al.,* 1979; Tanaka and Matsuura, 1984) whereas others have used reference lactate concentrations (Kindermann, Simon and Keul, 1979; Lafontaine, Londeree and Spath, 1981; Kumagai *et al.,* 1982; Sjodin and Jacobs, 1981; Williams and Nute, 1983). The detection of the

lactate threshold requires an excessive amount of blood sampling which is unacceptable for routine purposes and so a compromise is the use of reference concentrations; the most popular of which is 4 mmol l^{-1}, referred to as the 'onset of blood lactate accumulation' (OBLA). However, a running speed equivalent to a blood lactate concentration of 2 mmol l^{-1} is closer to the speeds freely chosen by endurance athletes during marathon running than the speeds equivalent to 4 mmol l^{-1} (Tanaka and Matsuura, 1984; Williams, Brewer and Patton, 1984). Therefore, in an assessment of aerobic fitness the use of a reference blood lactate concentration of 2 mmol l^{-1} rather than 4 mmol l^{-1} would appear to be more appropriate. Although the physiological and metabolic bases for OBLA have been proposed (Sjodin *et al.,* 1982), it is quite clear from a consideration of the relationship between blood lactate concentration and exercise intensity that significant accumulation occurs before a concentration of 4 mmol l^{-1} is reached. Therefore, 'OBLA' set at this particular concentration is something of a misnomer and really no more than a convenient reference lactate concentration. It should not be interpreted as 'the lactate or anaerobic threshold' because this confuses rather than clarifies the description of the metabolic responses to exercise. For example, a change in the carbohydrate content of the diet (Yoshida, 1986) or an exercise-induced decrease in muscle glycogen concentration (Sjodin and Jacobs, 1981; Farrell and Ivy, 1987; Fric *et al.,* 1988) will alter the concentration of blood lactate but this may not necessarily change the exercise intensity at which lactate begins to increase, i.e., the lactate threshold. Therefore while accepting these limitations, blood lactate concentrations of 2 mmol l^{-1} and 4 mmol l^{-1} are useful reference concentrations for following the responses to training, but they are not, as has been proposed, the equivalent of the aerobic and anaerobic thresholds respectively (Kindermann, Simon and Keul, 1979).

It has been suggested that training at an exercise intensity equivalent to a blood lactate concentration of 4 mmol l^{-1} produces optimum adaptations (Kindermann, Simon and Keul, 1979: Hollman *et al.,* 1981). Prescribing training intensities in relation to a fixed blood lactate concentration is based on the idea that an exercise intensity equivalent to a concentration of 4 mmol l^{-1} represents the highest lactate steady state an individual can sustain. At an exercise intensity above the lactate steady state it has been suggested that there is an abrupt increase in blood lactate concentration and an early onset of fatigue. An intensity equivalent to the lactate steady state allows the individual to exercise long enough at a sufficiently provocative work-load, to achieve both the necessary duration and intensity for optimum adaptations to occur. Recognizing that setting a fixed blood lactate concentration as a universal lactate steady state is a sweeping generalization, Stegman, Kindermann and Schnabel (1981) described a method by which the lactate threshold of the individual could be calculated and confirmed that this individual anaerobic threshold was closer to the lactate steady state than was the fixed lactate value of 4 mmol l^{-1} (Stegman and Kindermann, 1982). Exercise intensities equivalent to individually calculated lactate thresholds could be tolerated for approximately an hour whereas at exercise intensities corresponding to a blood lactate concentration of 4 mmol l^{-1} fatigue occurred in less than half this time (Stegman and Kindermann, 1982). The accessibility of portable automated blood lactate analysers has led to an increased interest, by coaches, in using this metabolic approach to the prescription of training intensities. Although some studies have reported the benefits of training at an exercise

intensity equivalent to 4 mmol l^{-1} (Sjodin, Jacobs and Svedenhag, 1982; Yoshida, Suda and Takeuchi, 1982) more training studies are required in order to obtain a clearer picture of the advantages of this method over traditional methods. The traditional approach to training is, of course, based on empirical information which has been gathered over many years and it is characterized, irrespective of the sport, by the application of a wide range of exercise intensities and durations. Therefore the prediction of a training intensity from laboratory measurements may only contribute to one aspect of what is, of necessity, a comprehensive programme of fitness training.

A further development has been the suggestion that the anaerobic threshold can be detected from measurements of heart rates during exercise of increasing intensity. The point at which the heart rate departs from its linear relationship with exercise intensity has been termed the 'deflection point' and reported to be coincident with the lactate threshold (Conconi *et al.,* 1982). However, in the studies designed to show the coincidence in the changes of these two physiological variables, the authors first established the exercise intensity at which the heart rate deflection point occurred and then, in a separate experiment, they measured blood lactate concentrations at exercise intensities around the previously determined heart rate deflection point for each of their subjects. The lactate measurements were made on blood samples obtained after exercise rather than during exercise, which is quite a different approach to the question of whether or not there is coincidence between the lactate and ventilatory thresholds! While the heart rate deflection point may, in itself, be of some value, the attempt to link it with the anaerobic threshold through the lactate threshold by this method appears somewhat contrived. Nevertheless the measurement of heart rate in this way may offer a very useful practical method of monitoring responses to exercise and training, however, it does not shed greater light on the enigma of the anaerobic threshold.

1.7 MAXIMAL EXERCISE

The numbers of participants in the multiple sprint sports which include, for example, football, hockey, the racquet sports, etc. far outweigh the numbers taking part in distance running, even when it was at its most popular. Brief periods of maximal exercise interspersed with periods of lower intensity activity are characteristic of the multiple sprint sports. The level of activity and the recovery between periods of high intensity exercise varies between sports and within the sport itself. While recognizing that brief periods of high intensity exercise are part of the common experience, irrespective of the arena in which they are performed, this aspect of human performance has not received as much attention as, for example, prolonged submaximal exercise. There has, however, been some confusion of terms which may have given rise to the impression that there is a large body of literature on the metabolic responses to maximal exercise. Exercise physiologists have used the term maximal exercise to describe the exercise intensity at which an individual reaches maximal oxygen uptake (Essen, 1978b). However, during brief maximal exercise of 5–6 s duration, the period of maximal continuous activity frequently observed during multiple sprint sports, the power outputs achieved are two to three times higher than those recorded as 'maximal' during a maximal oxygen uptake test

(Lakomy, 1984, 1986).

In previous studies which have declared that 'maximal exercise' of 10 s duration can be repeated indefinitely as long as there is a recovery period of 25–30 s, the exercise intensity has only been that which is required to achieve maximal oxygen uptake. The absence of a significant increase in blood lactate concentration during exercise of this intensity led to it being described as 'alactic' (Margaria *et al.,* 1969), suggesting that the resynthesis of ATP occurs only as a result of the phosphorylation of ADP by the phosphocreatine stores within the muscle. However, more recent studies have shown that during a brief period of high intensity exercise of just 6 s duration, muscle lactate concentration increases by approximately 200% (Table 1.3; Boobis, Williams and Wootton, 1982). During this brief period of high intensity exercise, phosphorylation of ADP by phosphocreatine contributes 50% to ATP resynthesis whereas glycolysis contributes the remaining 50% (Boobis,

Table 1.3 Muscle metabolites before and after 6 seconds of cycle ergometer exercise of maximal intensity (mmol kg^{-1} dw, mean ±SD)

	Pre-exercise	*Post-exercise*	*% change*
Glycogen	266.9 (±28.1)	229.0 (±42.5)	−14.2
PCr	84.3 (±2.3)	54.8 (±11.3)	−35.0
ATP	24.4 (±0.9)	22.2 (±1.1)	−8.3
ADP	3.7 (±0.3)	4.0 (±0.4)	+8.1
AMP	0.19 (±0.04)	0.23 (±0.05)	+21.1
Glucose	1.8 (±0.7)	2.7 (±0.9)	+50.0
Glu-1-P	0.08 (±0.05)	0.54 (±0.18)	+575
Glu-6-P	1.3 (±0.5)	11.0 (±4.4)	+746
Fru-6-P	0.13 (±0.07)	1.98 (±0.78)	+1423
Fru-1, 6-DP	0.13 (±0.15)	0.53 (±0.22)	+308
Triose-P	0.34 (± 0.07)	0.38 (±0.04)	+12
Pyruvate	0.17 (±0.04)	0.46 (±0.16)	+171
Lactate	9.29 (±1.82)	28.40 (±7.73)	+205

1987). Once the phosphocreatine concentrations have fallen to low values peak power output cannot be restored even though glycolysis continues to provide ATP (Wootton and Williams, 1983; Boobis, 1987; Spriet *et al.,* 1987a).

Fatigue during maximal exercise of several seconds duration may be viewed as a simple mismatching between the rate of ATP utilization, by working muscles, and the rate at which it is replaced by the various phosphorylation processes. Although there is some support for this particular explanation for the fatigue process (Gollnick, 1986) an

alternative explanation is that ATP utilization and not ATP resynthesis is inhibited, by the products of metabolism, namely hydrogen and or phosphate ions (Hultman, Spriet and Sodelund, 1987). The inhibition of mechanical activity in muscle and so a decreased ability to sustain a prescribed power output, has been attributed to a decreased availability of Ca^{2+} (Vollestad and Sejersted, 1988). Certainly an increased rate of ATP production by adrenaline-induced glycogenolysis does not appear to restore power output by human quadriceps muscles during the onset of fatigue (Spriet, Ren and Hultman, 1988). Furthermore, the decrease in muscle pH during high-intensity exercise or repeated periods of electrical stimulation does not completely inhibit glycolysis, suggesting that the muscle is still able to generate ATP even at very low pH values (Spriet *et al.,* 1987b). Therefore this evidence contributes to the argument that fatigue is not simply the result of a mismatch between the rates of regeneration and utilization of ATP but that the inhibition of ATP utilization may be a significant contributory factor to the onset of fatigue.

The idea that an individual develops 'lactate tolerance' after training has been quoted so frequently in coaching circles that it has become accepted as a general truth. The ability to tolerate high intensity exercise for longer periods of time or to generate higher power outputs after training is usually accompanied by increased concentrations of muscle and blood lactate. However, when the post-training exercise intensity and duration are the same as the pre-training conditions then the lactate concentrations are unchanged or decreased. Fatigue occurs during dynamic exercise at muscle pH values which are higher in trained than in untrained subjects (Sahlin and Henriksson, 1984) and after training compared with values before training (Cheetham *et al.,* 1989); this strongly suggests that muscle becomes 'intolerant of lactic acid' after training. The mechanisms proposed for this 'intolerance' include an increase in intracellular buffering capacity (Parkhouse and MacKenzie, 1984) and possibly an improved removal rate of hydrogen ions out of muscle (Mainwood and Renaud, 1985). There is some evidence to suggest that training increases the buffering capacity of skeletal muscles and this is offered as an explanation for the improved work capacity during high intensity exercise (Sharp *et al.,* 1986; Cheetham *et al.,* 1989). How much of the apparent improvement in buffering capacity is the result of changes within the muscle itself and how much is the result of a more rapid efflux of hydrogen ions across the cell membrane is not entirely clear because of the difficulties of making these measurements on samples of human muscle. Furthermore, the current method involves the titration of homogenates of the whole muscle sample and so changes in intracellular buffering capacity have to be relatively large to overcome the 'dilution' effect of using a sample of muscle containing all fibre types (Sahlin, 1978).

The removal of hydrogen ions from the sarcoplasm of muscle as quickly as possible is, of course, an additional method of buffering. Support for the central role of hydrogen ions in the fatigue process comes from these studies which have shown improved exercise tolerance under conditions which favour an improved removal rate. When the blood perfusing an *in situ* preparation of canine gastrocnemius muscle was made alkalotic, with sodium bicarbonate, then the endurance capacity of the muscle in response to repeated electrical stimulation was significantly increased. When, however, the blood perfusing the working muscle was made acidotic, with ammonium chloride, then fatigue

occurred earlier than under control conditions (Hirche *et al.,* 1975). In human studies the ingestion of bicarbonate solutions, up to three hours before exercise, has been shown to improve endurance capacity, during cycle ergometry (Jones *et al.,* 1977), as well as running times over 400 m (Goldfinch, McNaughton and Davies, 1988) and 1500 m (Wilkes, Gledhill and Smyth, 1983) and also improvements in swimming performance during interval training (Gao *et al.,* 1988). There is a transient rise in blood pH as a result of the ingestion of bicarbonate solutions as well, of course, as in plasma bicarbonate concentration. The clearance rate of the additional bicarbonate is relatively fast as reflected by the prompt rise in the pH of the individual's urine and this change in the normal pH is also a way of clearly detecting the use of the bicarbonate treatment as an aid to improved performance.

The quantification of high intensity exercise in physiological or metabolic terms has not been developed to the same extent as has the quantification of submaximal exercise. The gold standard or reference point for aerobic exercise is the maximal oxygen uptake of an individual. However, there is not, as yet, an equivalent gold standard for 'anaerobic exercise' which offers the same opportunities for standardization of exercise intensities. At present the anaerobic power of an individual is reported only in terms of the absolute values achieved during, for example, cycle ergometry or sprinting on a non-motorized treadmill (Lakomy, 1984, 1986; Cheetham *et al.,* 1986). Attempts have been made to describe the anaerobic capacity of an individual, based on the largest 'oxygen debt' which can be generated during dynamic exercise (Hermansen, 1969). More recently the concept of a maximal oxygen deficit has been proposed as a method of assessing the anaerobic capacity of an individual (Medbo *et al.,* 1988). Further studies are required to establish the usefulness of this particular concept before it is ready to take its place alongside maximal oxygen uptake as a cornerstone concept in exercise physiology.

Fitness for endurance sports can be best described in terms of the highest percentage of $\dot{V}O_{2max}$ an individual can sustain for prolonged periods of time irrespective of $\dot{V}O_{2max}$ *per se*. In the multiple sprint sports, fitness is reflected in the ability to repeat maximal sprints, separated by only short recovery periods, with only the minimum of fatigue. Therefore, fitness should be assessed in terms of the ability to reproduce maximal effort rather than only assessed in terms of the absolute power output or the maximal oxygen deficit achieved. Thus 'fatiguability' during repeated sprints may provide a more useful index of 'fitness' for participants in the multiple sprints sports than measurements of maximal power output (Wootton and Williams, 1983).

Unlike the adaptations to endurance training where there is clear evidence of an increase in the capacity for the aerobic production of ATP, sprint training has not been shown to produce such large changes in the key enzymes of the glycolytic pathway (Saltin and Gollnick, 1983). However, it has not been established whether or not training simply increases the number of motor units recruited during maximal exercise and therefore it is the additional 'muscle mass' which is responsible for the training-induced increase in power output (Cheetham *et al.,* 1989). An improvement in efficiency of contractile activity following high intensity training has also been suggested as an explanation for improved performance on the basis of studies on rodent muscles (Westra *et al.,* 1985). There is no reason to suggest, however, why this improvement in performance does not follow the same development as occurs during strength training,

namely the neurogenic response preceding the myogenic adaptations of muscle (Chapter 2). Therefore, to search only for metabolic explanations for improved performances following sprint training of different durations, using subjects of varied levels of fitness, may at best be somewhat short sighted and at worse, misleading.

In summary, suffice to say that when individuals successfully meet the challenge of exercise during the preparation for and the participation in sport they do so as a result of an exquisitely orchestrated collection of physiological and metabolic events. While a knowledge of the metabolic events underlying the responses to exercise will not, on their own, explain successful performances nor the failure of performance we know as fatigue, they will provide invaluable pieces of the jigsaw from which a more complete picture of human performance can be built.

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