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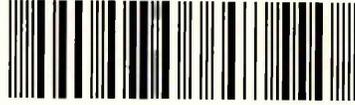
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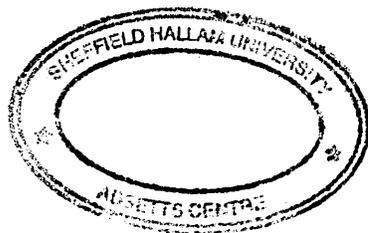
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Gas exchange kinetics in elite runners

Andrew Mark Edwards

A thesis submitted in partial fulfilment of the requirements of Sheffield Hallam University for
the degree of Master of Philosophy

November 1999



Abstract

Oxygen uptake ($\dot{V}O_2$) kinetics measured in the frequency domain are known to be faster in subjects of greater aerobic fitness (Essfeld *et al.* 1987). Carbon dioxide output ($\dot{V}CO_2$) kinetics have been shown to be delayed following 6 months sprint and strength training in footballers (Fukuoka *et al.* 1997) and might also, therefore, be expected to differentiate subjects according to their aerobic fitness.

The purpose of this study was to examine whether an established technique for measuring gas exchange kinetics could be applied as a test to differentiate between elite athletic groups. The subject groups selected for this study were 12 elite male sprinters (ST) and 12 elite male endurance runners (ET).

A 300s pseudo random binary sequence (PRBS) exercise protocol was selected to investigate differences between ST and ET. Oxygen uptake ($ml \cdot min^{-1}$) and $\dot{V}CO_2$ ($ml \cdot min^{-1}$) were measured on a breath-by-breath basis using a Marquette MGA 1100 respiratory mass spectrometer system. The test/retest reliability of the selected test procedure was examined by the limits of agreement technique. Ten healthy male subjects agreed to participate in the reliability study and all subjects completed 3 consecutive 300s PRBS cycles with 20 s work rate changes between 25 and 85 W on an electrically-braked cycle ergometer at a pedal cadence of 1 Hz. Fourier analysis was computed for frequencies 3.3, 6.7 and 10 mHz. The limits of agreement between test and retest were closer at each measured frequency of $\dot{V}O_2$ kinetics than $\dot{V}CO_2$ kinetics. The measurement error for $\dot{V}O_2$ kinetics ranged from 18-35%, while $\dot{V}CO_2$ kinetics displayed greater variability, ranging from 39 - 108%. The results of the reliability study suggest that, using the selected test procedure, $\dot{V}O_2$ kinetics are more likely to detect physiological differences between ET and ST than $\dot{V}CO_2$ kinetics.

In the study of elite athletes, twelve elite male sprinters and twelve elite endurance runners completed the selected PRBS exercise test. Blood lactate concentrations taken pre and post testing remained below 2mM.

Statistical analysis by two way ANOVA with repeated measures and tukey honest significant difference post hoc test showed significantly greater amplitude ratios for ET than ST at frequencies 6.7mHz (6.71 ± 1.09 and 5.47 ± 0.95 $ml \cdot min^{-1} \cdot W^{-1}$ respectively) ($P < 0.05$) and 10 mHz (4.97 ± 0.98 and 3.56 ± 0.69 $ml \cdot min^{-1} \cdot W^{-1}$ respectively) ($P < 0.01$) for $\dot{V}O_2$ kinetics but not for $\dot{V}CO_2$ kinetics. Significantly shorter phase shifts were observed for ET than ST at frequency 3.3mHz (-35.45 ± 4.31 and -41.26 ± 5.82 degrees respectively) ($P < 0.05$) for $\dot{V}O_2$ kinetics but not for $\dot{V}CO_2$ kinetics, although a trend for faster $\dot{V}CO_2$ kinetics was evident in ET.

The findings of this study show that $\dot{V}O_2$ kinetics and not $\dot{V}CO_2$ kinetics, as measured by the selected PRBS test, differentiate between elite sprinters and endurance runners. This supports the further development of the $\dot{V}O_2$ kinetics test as a practical measure to differentiate between sports groups.

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Abbreviations

ATP	adenosine triphosphate
CHO	carbohydrate
CS	citrate synthase
CK	creatine kinase
CO ₂	carbon dioxide
CoA	coenzyme A
ET	elite endurance trained runners
ETS	electron transport system
H ⁺	hydrogen ion
HCO ₃ ⁻	bicarbonate
HK	hexokinase
Hz	unit of frequency
LDH	lactate dehydrogenase
mM	unit of concentration: millimolar
NAD ⁺	nicotinamide-adenine dinucleotide (oxidized form)
NADH	nicotinamide-adenine dinucleotide (reduced form)
O ₂	oxygen molecule
PCr	phosphocreatine
PFK	phosphofructokinase
pH	a measure of acidity
RER	respiratory exchange ratio
SDH	succinate dehydrogenase
ST	elite sprint trained runners
TCA	tricarboxylic acid
$\dot{V}CO_2$	rate of carbon dioxide production
$\dot{V}O_2$	rate of oxygen uptake
$\dot{V}O_2 \text{ max}$	maximal oxygen uptake

Publications relevant to this thesis

Edwards, A.M., Claxton, D.B, Challis, N.V., Chapman, J.H and Fysh, M.F. $\dot{V}O_2$ kinetics determined by PRBS techniques differentiate elite endurance runners from elite sprinters. *International Journal of Sports Medicine*: 1999; 20: pp 1-6.

Edwards, A.M., Chapman, J.H and Fysh, M.F. Gas exchange kinetics of elite runners. 4th *Annual Congress of the European College of Sport Science, Rome 1999* (Abstract).

Fysh, M, Chapman, J.H, Edwards, A.M and Paggiosi, M.A. Relationships between oxygen uptake kinetics and maximal oxygen uptake. 4th *Annual Congress of the European College of Sport Science, Rome 1999* (Abstract).

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1.0 Introduction and review of literature

Sprint and endurance athletes have been used as comparative groups in a number of research studies (Bernus *et al.* 1993, Fukuoka *et al.* 1995, Granier *et al.* 1995, McCully *et al.* 1992, Paavolainen *et al.* 1994, Olesen *et al.* 1994, Ozyener *et al.* 1996, Torok *et al.* 1995). The selection of these groups has been based on the assumption that a number of physiological differences exist between sprint and endurance athletes and thus researchers have been confident of obtaining a differing physiological test response from the groups to an appropriate stimulus.

The comparison between groups presupposes that the successful performance of sprint and endurance activities relies on the availability of sufficient energy to attain the appropriate power output for the duration of the event. This is achieved through the resynthesis of adenosine triphosphate (ATP) which releases energy at different rates depending on the metabolic pathway utilised. The particular pathway utilised effects the amount and rate of ATP produced which in turn effects the available energy and subsequent power of the performer. Sprint events are completed in seconds and thus require large amounts of energy rapidly whereas endurance events lasting some minutes utilise a lower power output and thus ATP is released at a lower rate according to the demands of the event.

The ability to successfully utilise the appropriate energy systems for exercise is dependent on a number of physiological characteristics which are known to be more or less effective in different athletic groups. For example, several characteristics are known to differentiate sprinters from endurance athletes, such as: maximum cardiac output, $\dot{V}O_2$ max, capillary

density, muscle fibre type distribution and metabolic characteristics (Andersen and Henriksson 1977, Bergh *et al.* 1978, Torok *et al.* 1995) and the existence of these differences has often been cited as the basis for subject selection in research studies examining other physiological differences between these athletic groups (Bernus *et al.* 1993, Ozyener *et al.* 1996, Torok *et al.* 1995). The characteristic differences between sprinters and endurance athletes are a combination of both specific training adaptations and heredity (Bouchard *et al.* 1984, Komi *et al.* 1977, Klissouras 1971) and consequently, athletic success relies on both these factors.

The genetic contribution to exercise capacity has been estimated at about 40% for aerobic capacity, 50% for maximum heart rate, and 70% for physical working capacity (Bouchard 1986). Heredity has a substantial influence on exercise capacity and future work might determine the upper limit of genetic contribution (Bouchard *et al.* 1984). Therefore, this study acknowledges the large contribution of inherited characteristics to exercise capacity, but focuses on the fact that further improvement in athletic performance will be through physical training and the resulting physiological adaptations.

A number of physiological tests exist which potentially differentiate elite sprinters from elite endurance runners, such as maximal oxygen uptake (Bergh *et al.* 1978), accumulated O₂ deficit during intense exercise (Bangsbo *et al.* 1993) and the Wingate test (Inbar *et al.* 1999). Direct measurement of maximal capacity has more traditionally been expected to provide a better estimation of exercise capacity than submaximal evaluation. Tests of maximal capacity do, however, present greater cardiorespiratory stress than submaximal tests and could be inconvenient for elite athletes undergoing training or in studies of wider populations due to the potential risk of undiagnosed heart disease. As such, a suitable submaximal test which can

differentiate between groups would provide an attractive alternative. A submaximal and low intensity test, such as the $\dot{V}O_2$ kinetics test by the pseudo random binary sequence (PRBS) technique that is able to reflect the oxidative process of the working muscle could, consequently, have application in the field of sports testing. Such a test would, therefore, be expected to be sufficiently sensitive to detect differences between individuals, based on their oxidative potential.

Endurance athletes are known to possess greater oxidative capacities than sprinters and it is anticipated that this should be reflected in faster $\dot{V}O_2$ kinetics. If such a difference exists, a measure of $\dot{V}O_2$ or $\dot{V}CO_2$ kinetics might provide a useful test to assess the performance characteristics of athletes and the type or degree of training undertaken.

It is known that gas exchange kinetics vary between individuals, e.g Babcock *et al.* (1994), Cunningham *et al.* (1993), and that this variation can be influenced by training (Berry and Moritani 1985, Phillips *et al.* 1995a), while subjects with higher levels of aerobic fitness have been also been shown to possess faster $\dot{V}O_2$ kinetics (Essfeld *et al.* 1987). Few studies (Fukuoka *et al.* 1995, 1997, Ozyener *et al.* 1996) have utilised $\dot{V}O_2$ or $\dot{V}CO_2$ kinetics to differentiate between athletic groups and these studies have employed protocols with work rates above LT. The study of Fukuoka *et al.* (1995) utilised a sinusoidal exercise protocol alternating between 30W and 60% of the individual's $\dot{V}O_2$ max for 40 min. Plasma lactate concentrations taken at 60% of $\dot{V}O_2$ max reached levels of 3.9 mmol L^{-1} , indicating that lactate had accumulated above resting levels through the test. This suggests that the exercise protocol employed was not truly aerobic and might have thus affected the results observed.

Few studies have reported $\dot{V}CO_2$ kinetics in spite of correlations between $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics (Cerrretelli *et al.* 1966, Whipp and Wasserman 1972, Zhang *et al.* 1991). This is surprising since it has been suggested (Fukuoka *et al.* 1997) that $\dot{V}CO_2$ kinetics are more successful at determining the effects of sprint training compared to $\dot{V}O_2$ kinetics.

A review of literature has been conducted to investigate the physiological characteristics of both sprinters and endurance runners and the adaptations made through specific training for the events. A review has also been conducted of the gas exchange kinetics literature and the application of the psuedo random binary sequence (PRBS) technique. A study of reliability has been performed prior to an experimental chapter comparing the low intensity $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics of sprinters and endurance runners.

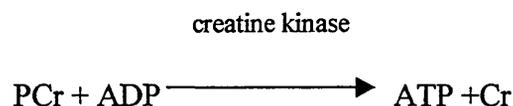
1.1 Energy systems for muscle contraction

The energy required to perform muscle contraction is provided chemically in the form of ATP. ATP is broken down enzymatically to adenosine diphosphate (ADP) and inorganic phosphate (Pi) to yield energy for muscle action (Lodish *et al.* 1995). The systems by which ATP is subsequently re-synthesised to continue muscle contraction depend on the intensity and duration of the activity undertaken (Maughan *et al.* 1997). This is facilitated by energy provision through the appropriate energy pathways.

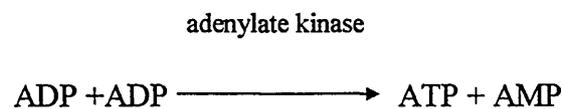
1.1.1 The immediate energy system (Phosphagen)

The phosphagen system provides the main source of energy for short and powerful activities (Fox *et al.* 1969), consequently, this system is of particular relevance to the performance of sprint events.

Human skeletal muscle contains a relatively large amount of phosphocreatine (PCr) amounting to 70-80 mmol kg dm⁻¹ at rest (Harris *et al.* 1992). PCr utilisation occurs at the immediate onset of contraction to buffer the rapid accumulation of ADP resulting from ATP hydrolysis (Margaria *et al.* 1969). The momentary rise in ADP concentration is the primary stimulus to PCr hydrolysis via the creatine kinase (CK) reaction and for each mole of PCr degraded, one mole of ATP is resynthesised. CK catalyses the breakdown of PCr with the resultant formation of ATP. This reaction is shown as:



A further reaction catalysed by the enzyme adenylate kinase (AK) results in the production of one ATP molecule from two ADP molecules. This reaction is shown as:



Adenylate kinase (also known as Myokinase) prevents ADP accumulation at the muscle fibre contraction site and, consequently, higher AK activity facilitates greater ATP production (Chi *et al.* 1983). Although the anaerobic ATP production from the AK pathway is relatively minor, it is probable that it limits sprint performance (Linossier *et al.* 1997b, Thorstensson *et al.* 1975). In addition, a high AK activity has been associated with a greater glycolytic potential (Chi *et al.* 1983).

Creatine kinase has a number of isozymes which are located at different intracellular locations (Apple and Tesch 1989, Apple and Rodgers 1986a). A number of isozymes are known to be present in skeletal muscle, for example CK-MM is located near the sites of ATP utilisation (the myofibrils) and CK-Mi is located near the site of ATP production (the mitochondria). The discovery of isozymes of CK at differing cellular locations has led to the hypothesis that PCr has a number of functions within skeletal muscle (Apple and Tesch 1989). The first, and possibly most important, relates to its cytoplasm function (shown above) whereby it maintains the cellular ATP concentration and ATP:ADP ratio. A second function is that PCr assists prolonged physical activity by acting as an energy transport system between the site of ATP production and the sites of ATP utilisation, this secondary function is known as the 'PCr shuttle' (Meyer *et al.* 1984).

Margaria *et al.* (1969) proposed that PCr degradation was the immediate and only substrate for ATP resynthesis during the early stages (<10s) of intense activity such as sprinting. Following depletion of this substrate, glycolysis was thought to be activated to provide a continued ATP supply. This theory has been used to explain how human skeletal muscle responded to the large demand for ATP during high intensity activity, however, the suggestion that anaerobic glycolysis was activated only when the PCr store became depleted in maximal activity is not supported by experimental evidence. A study of short term (<10s), maximal activity (Jacobs *et al.* 1983) revealed accumulated levels of lactate (46.1 and 25.2 mmol·kg⁻¹dm⁻¹) in males and females respectively. This study indicates that PCr degradation and anaerobic glycolysis are activated simultaneously at the onset of high intensity activity. Further evidence that anaerobic glycolysis is activated at the onset of intense exercise was provided by Hultman and Sjöholm (1983). In their study electrical stimulation of the vastus lateralis muscle for 1.28-5 s consistently raised muscle lactate indicating its presence much earlier than previously suggested. This indicates that sprinters will rely heavily on both the phosphagen system and glycolysis to provide energy for their event.

1.1.2 The short term energy system (Glycolysis)

Glycolysis facilitates energy provision for prolonged sprint (200-400m) and middle distance (800-1500m) events as a relatively high turnover of ATP is maintained in order to sustain a high intensity work rate which could not be supported by oxidative processes (Newsholme 1986). Therefore, the utilisation of this system is of importance to athletes training for events of short to medium duration.

The initial stage of glucose metabolism is glycolysis where each glucose molecule is converted to two molecules of pyruvate. When glycogen is the substrate this process is called glycogenolysis, however, except where glycogen is specifically referred to, the term glycolysis is generally used to refer to either process (Lodish *et al.* 1995). These chemical reactions take place in the cytosol and do not require oxygen. The reactions of glycolysis are shown in Figure 1.1.

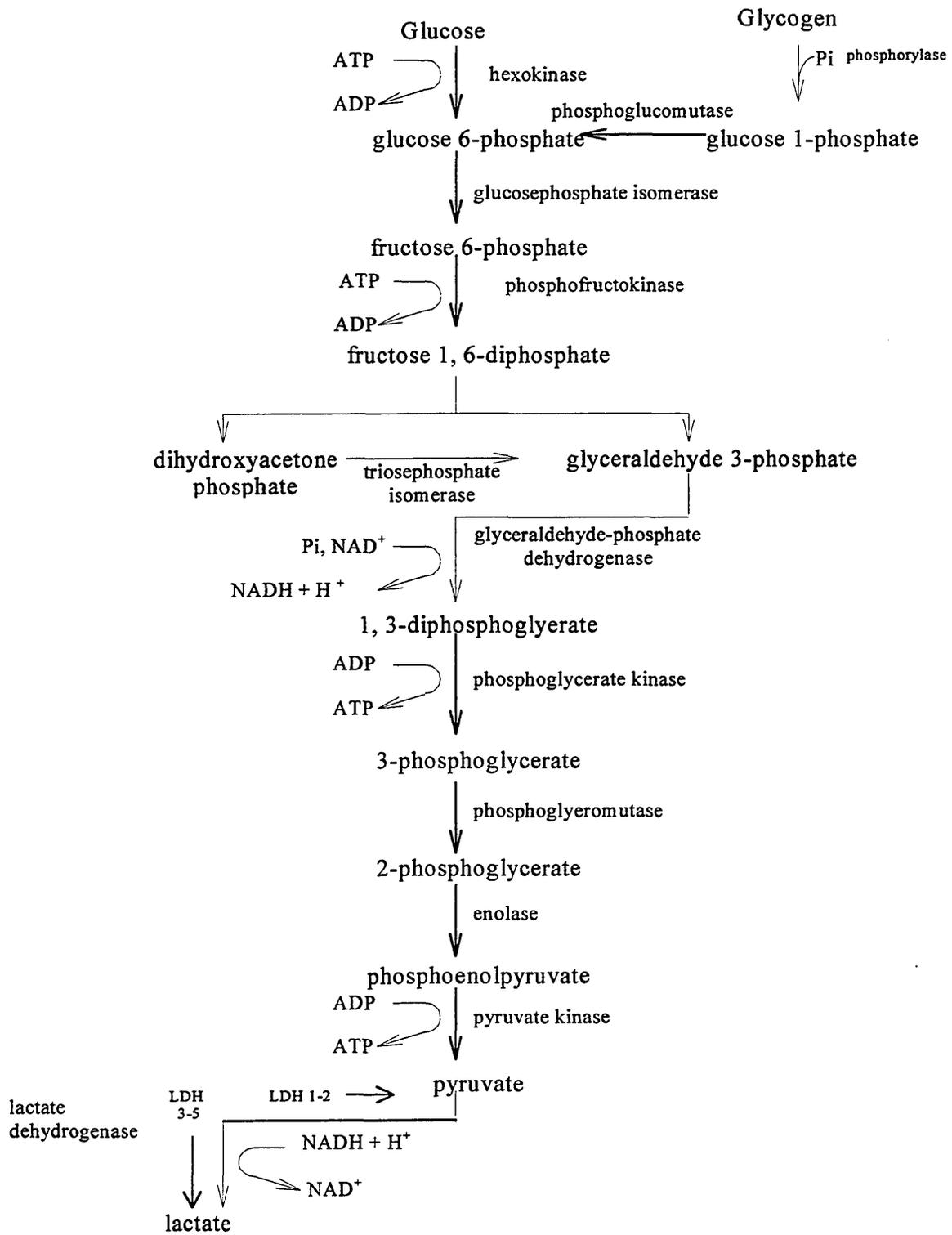


Figure 1.1. The reactions of glycolysis (Adapted from Maughan *et al.* 1997).

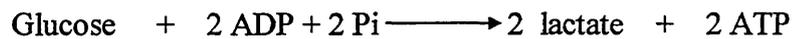
All the metabolic intermediates between the initial carbohydrate and the final product pyruvate are phosphorylated compounds. Four molecules of ATP are produced from ADP during glycolysis, two in the step catalysed by phosphoglycerate kinase and two in the step catalysed by pyruvate kinase. Two molecules are used in earlier steps of the pathway; the first by the addition of a phosphate residue to glucose in the reaction catalysed by hexokinase (HK) and the second by the addition of a second phosphate to fructose-6-phosphate in the reaction catalysed by phosphofructokinase (PFK). Therefore, there is a net gain of two ATP molecules (Lodish *et al.* 1995).

The process of ATP generation through glycolysis is substrate-level phosphorylation as the metabolites in the cytosol are chemically transformed by enzymes (membranes and ion gradients are not involved).

The activity of the glycolytic pathway is continuously regulated so that the production of ATP and pyruvate is adjusted to meet the requirements of the cell. The principal rate limiting enzyme in this pathway is PFK (Dobson *et al.* 1986). When exercise begins, ADP +Pi levels rise and enhance PFK activity, which serves to increase the rate of glycolysis. In contrast, at rest when cellular ATP levels are high, PFK activity is inhibited and glycolytic activity is slowed. PFK is inhibited by citrate which is the product of the first step of the Tricarboxylic Acid (TCA) cycle (Dobson *et al.* 1986). If citrate accumulates, its feedback inhibition of PFK reduces the production of pyruvate and acetyl CoA and so less citrate is formed via the cycle. Through its effect on PFK activity, citrate can consequently regulate the flow of acetyl CoA into the TCA cycle according to energy demand. PFK is activated by ADP and inhibited by ATP which makes the rate of glycolysis very sensitive to intracellular levels of ATP and ADP (Dobson *et al.* 1986, Ren and Hultman 1989).

The two other enzymes in the glycolytic pathway which are subject to control are hexokinase which is inhibited by its reaction product, glucose-6-phosphate and pyruvate kinase which is inhibited by ATP (Ren and Hultman 1989).

The anaerobic metabolism of glucose occurs when oxygen becomes limited and glucose can not be oxidised completely to CO₂ and H₂O, for example during the high intensity prolonged contraction of human skeletal muscle cells. Glucose is converted to two molecules of lactate with the net production of two molecules of ATP per glucose molecule.



As lactate accumulates in the muscle cell some will diffuse into the extracellular space and eventually begin to accumulate in the blood. Some lactate passes into the liver, where it is reoxidised to pyruvate and either further oxidised to CO₂ + H₂O or converted to glucose. In addition, some lactate is oxidised by cardiac muscle.

At exercise intensities above the lactate threshold (LT) the accumulation of lactic acid has the effect of causing the pH to fall to a point where it interferes with cell function. At rest, the normal pH is ~7.1, but this can fall to ~6.5 in high intensity activity when large amounts of lactate are formed (Maughan *et al.* 1997). At pH of 6.5, contraction begins to fail and some inhibition of glycolytic enzymes, such as phosphorylase (PHOS) and PFK will occur.

Consequently, the regeneration of ATP cannot keep pace with its utilisation through the glycolytic pathway and fatigue occurs. Therefore, fatigue in high intensity activity is probably

mediated in part by increased acidity that inactivates various enzymes involved in energy transfer (Mainwood *et al.* 1985). The presence of elevated levels of lactate in a low intensity exercise test would, consequently, indicate that relative work rates were inappropriately high and that the interference of lactate on cell function would undermine test responses.

Summary

The capacity of the glycolytic pathway for producing energy in the form of ATP is large in comparison with the phosphagen system (Table 1.1.), however, although the total capacity of the glycolytic pathway is greater than that of the phosphagen system, the rate at which it can produce ATP is lower (Table 1.1). As such, the power output that can be sustained through this system is reduced and consequently the attainable running speeds must be lower than those attained via the phosphagen system.

Table 1.1. Capacity and power of anaerobic systems for the production of ATP*.

	Capacity (mmol ATP kg dm ⁻¹)	Power (mmol ATP kg dm ⁻¹ s ⁻¹)
Phosphagen system	55-95	9
Glycolytic system	190-300	4.5
Combined	250-370	11

* Values are expressed per kg dry mass (dm) of muscle, and are based on estimates of ATP provision during high intensity exercise of human lateralis muscle (Maughan *et al.* 1997).

1.1.3 The aerobic energy systems

The Tricarboxylic acid (TCA) cycle

Endurance events such as long distance running rely heavily on aerobic processes. The phosphagen system and glycolysis are unable to supply sufficient quantities of ATP to sustain high intensity work for more than a few minutes (Newsholme 1986). Consequently, the intensity of the work being performed must gradually drop as the distance or exercise time increases and more reliance is placed on the aerobic energy systems to supplement and eventually take over energy provision (Newsholme 1992).

In the presence of sufficient O_2 , the pyruvate formed during glycolysis passes into the mitochondria and is oxidised completely to CO_2 . The oxidation reactions in the mitochondria generate most of the ATP produced from the conversion of glucose to CO_2 (Lehninger 1978).

The pyruvate generated in the cytosol during glycolysis is transported across the mitochondrial membranes to the Tricarboxylic acid (TCA) cycle, where it immediately reacts with coenzyme A to form CO_2 and the intermediate acetyl CoA. This reaction is catalysed by the enzyme pyruvate dehydrogenase. Acetyl CoA then enters the TCA cycle.

Stored fat is utilised as an energy source and is broken down to fatty acids and glycerol via lipolysis. The glycerol is metabolised by the glycolytic pathway while the fatty acids combine with coenzyme A before being broken down via β -oxidation. This results in the formation of acetyl CoA, NADH and $FADH_2$. The acetyl CoA is oxidized via the TCA cycle and the NADH and $FADH_2$ are oxidized via the electron transport system (ETS). The TCA cycle and ETS are summarised in Figure 1.2.

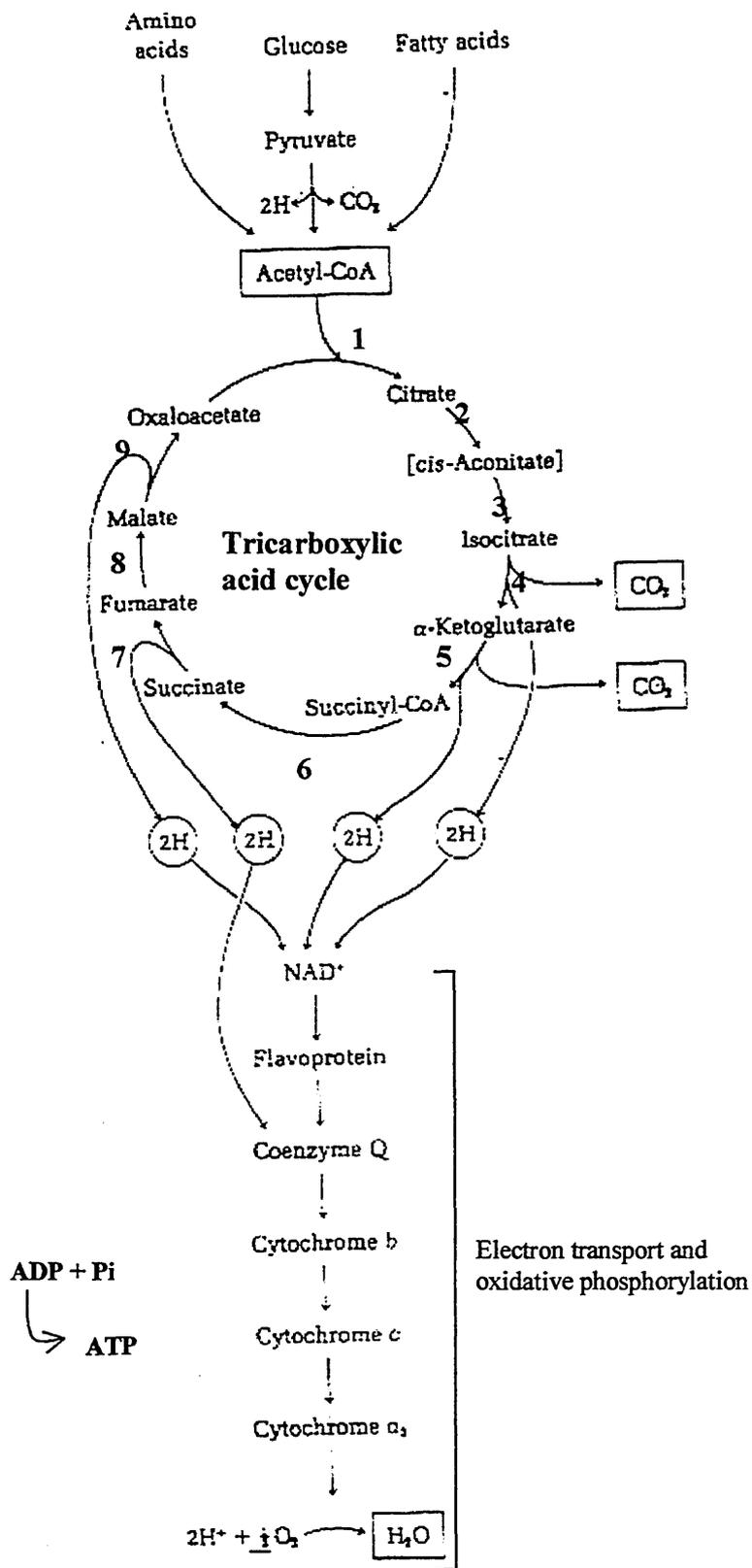


Figure 1.2. The flow sheet of respiration. Reactions 1-9 of the TCA cycle are illustrated.

Taken from Lehninger (1978).

The cycle begins with the joining of the two carbon acetyl group from acetyl CoA with the four carbon molecule oxaloacetate. The product of reaction (1) is the six-carbon citric acid, for which the cycle is often named. In reactions (2) and (3) citrate is isomerised to the six carbon molecule isocitrate by the single enzyme aconitase. Isocitrate is oxidised to the five carbon α -ketoglutarate (4) generating one CO_2 molecule and reducing one molecule of NAD^+ to NADH. In reaction (5), the α -ketoglutarate is oxidised to the four carbon molecule succinyl CoA, generating the second CO_2 molecule formed during each turn of the cycle and reducing another NAD^+ to NADH. In reactions 6-9 succinyl CoA is oxidised to oxaloacetate, regenerating the molecule that was initially condensed with acetyl CoA. Succinyl CoA is converted to succinate (6) and is subsequently oxidised to form fumarate by succinate dehydrogenase (7). Fumarate then undergoes a hydration reaction catalysed by fumarase to form malate (8). The enzyme malate dehydrogenase then oxidises the malate to form oxaloacetate (9) (Leninger 1978).

A complete turn of the TCA cycle is achieved once oxaloacetate has been reformed and one molecule of ATP, three molecules of NADH and one molecule of FADH_2 have been formed (Leninger 1978, Lodish *et al.* 1995).

The Electron Transport System (ETS)

NADH which is also generated in the cytosol during glycolysis is not transported directly to the mitochondrial TCA cycle as the inner membrane of the mitochondria is impermeable to NAD^+ or NADH. Consequently, a shuttle system (the malate shuttle) transports electrons from cytosolic NADH to the electron transport system (ETS) (Figure 1.2.).

At four different sites in the TCA cycle, H^+ ions are removed and passed through the ETS. The H^+ and electrons enter the ETS via the carriers FADH₂ and NADH and are transferred by electron carriers in a series of enzymatic reactions. The electron carriers are often referred to as the cytochromes. Electrons are passed through a series of cytochromes on to cytochrome a and a₃ which exist as a complex called cytochrome oxidase. Cytochrome a₃ finally transfers the electrons to molecular oxygen, whereby an ATP molecule is synthesised. It is this energy which is released via a number of steps that eventually is used to resynthesise ADP and Pi back to ATP (oxidative phosphorylation) (Leninger 1978, Lodish *et al.* 1995).

The rate of ATP resynthesis via the oxidative pathways is much slower than via the phosphagen and glycolytic pathways. Although the rate of ATP turnover is much reduced, the oxidative pathways are able to continue with ATP resynthesis for long periods. This invariably means that due to the reduced turnover of ATP, the attainable power output via these systems will be reduced and consequent running speed would be at a moderate level (Fox *et al.* 1969 & 1975).

1.1.4 Muscle fibre type and energy metabolism

Human skeletal muscles are composed of different populations of cell types that can be classified into three categories: Slow twitch oxidative (Type I), fast twitch oxidative-glycolytic (Type IIa), and fast twitch glycolytic (Type IIb). These cells differ in their mechanical and metabolic characteristics, which reflect their differing mitochondrial content and isoforms of enzymes and contractile proteins (Pette 1985).

Type I fibres are red cells that contain the relatively slow acting myosin ATPase and hence contract relatively slowly. The red colour is due to the presence of myoglobin, an intracellular respiratory pigment capable of binding oxygen and only releasing it at very low partial pressures i.e in the proximity of the mitochondria. Type I fibres have numerous mitochondria, mostly located close to the periphery of the fibre, near to the blood capillaries which provide a rich supply of oxygen and nutrients. These fibres possess a high capacity for oxidative metabolism, they resist fatigue and are suited for the performance of prolonged physical activity, such as endurance running (Apple and Tesch 1989, Essen *et al.* 1975).

During exercise the blood vessels in the working muscle dilate which increases capillary density up to five-fold compared with rest. Greater capillary density surrounding individual muscle fibres mean that when a fibre is recruited it becomes more effectively exposed to the flow of blood delivered to the muscle during exercise. Thus, greater capillary density potentially allows an improved rate of transfer of oxygen at high perfusion rates by presenting a greater surface area for diffusion. (Brodal *et al.* 1977, Kiens *et al.* 1993, Klausen *et al.* 1981, Hermansen and Wachlova 1971).

In comparison, type IIb fibres are much paler because they contain little myoglobin. They possess rapidly acting myosin ATPase, so their contraction and relaxation time is relatively fast and consequently they have a greater potential maximum power output than type I fibres. They also have few mitochondria and a poorer capillary density, but greater glycogen and phosphocreatine stores compared with type I fibres (Costill *et al.* 1976, Greenhaff *et al.* 1993). A high activity of glycolytic enzymes endows type IIb fibres with a high capacity for rapid (but short lived) ATP production when energy has to be released at rates in excess of

that available from oxidative phosphorylation. Consequently, type IIb fibres possess a high anaerobic capacity whereby energy is derived from anaerobic respiration without the use of oxygen. As such, type IIb fibres are best suited for rapid, forceful actions, such as sprinting but for brief periods as they are known to fatigue rapidly.

Type IIa fibres are red cells with metabolic and physiological characteristics which lie between the extreme properties of the other two fibre types. They contain fast acting myosin ATPase like that of the type IIb fibres, but have an oxidative capacity similar to that of the type I fibres.

1.1.5 Energy systems in relation to athletic performance.

The energy sources utilised during exercise depend on the intensity and duration of the activity undertaken. As indicated previously, the energy required to attain high power outputs is derived from different sources from those which sustain moderate power for prolonged periods. Energy supply is, therefore, not merely a matter of utilising one system or another, but in most cases, their integration. This can be observed in Table 1.2. which shows the estimated contribution of different fuels during exercise.

Table 1.2. The estimated contribution of different fuels to ATP generation in various running events . Taken from Newsholme (1992).

Percentage Contribution to ATP Generation					
Event	PCr	Glycogen		Blood Glucose (Liver Glycogen)	Triglyceride (Fatty Acids)
		Anaerobic	Aerobic		
100m	50	50	-	-	-
200m	25	65	10	-	-
400m	12.5	62.5	25	-	-
800m	6	50	44	-	-
1500m	*	25	75	-	-
5000m	*	12.5	87.5	-	-
10000m	*	3	97	-	-
Marathon	*	-	75	5	20
Ultra marathon	*	-	35	5	60

* In such events PCr will be used for the first few seconds and, if it has been resynthesised during the race, in the sprint finish.

It is evident from Table 1.2. that a contrast in energy requirement exists between short sprints and extreme endurance running. Ideally, studies investigating physiological differences between sprinters and endurance runners would utilise groups which primarily represent either aerobic or anaerobic energy utilisation. However, the body stores O₂ through hemoglobin and myoglobin which inevitably means that O₂ is available at the onset of exercise and consequently even short sprints could not be described as entirely anaerobic. Substantial difference can be seen in the ATP provision from aerobic and anaerobic sources

for the groups selected in this study, illustrating their reliance on different biochemical pathways to achieve their performance.

The ability to sustain prolonged exercise is derived from aerobic processes. This has the effect of providing energy for prolonged periods, but inevitably at a lower intensity due to the slower rate of production.

Unlike maximal intensity exercise, the rate of ATP production required during endurance exercise is relatively low and, therefore, PCr, CHO and fat can all contribute to ATP resynthesis. CHO is the most important fuel source during this type of exercise as it provides the greatest amount of ATP compared with the oxidation of any other substrate (Newsholme 1992). Nevertheless, intramuscular stores of CHO are relatively low and as a consequence would be depleted rapidly if energy provision was not supplemented by abundant intramuscular fat stores. Fat oxidation can only supply very limited ATP when exercising at an intensity of about 50-60% $\dot{V}O_2$ max. Therefore, it is impossible for energy derived via oxidative pathways to support the resynthesis of ATP at a sufficient rate for maximal/high intensity exercise (Galbo 1992, Maughan *et al.* 1997).

The energy requirements for endurance running, as shown in Table 1.2, differ from those of sprinting and show the expected preference for the utilisation of oxidative pathways. The major physiological requirement of endurance running (>3000m) can, therefore, be summarised as the ability to sustain a high rate of oxygen consumption for prolonged periods. This can be achieved by having a high maximum oxygen uptake ($\dot{V}O_2$ max) and by the ability to exercise at a high proportion of the $\dot{V}O_2$ max for the duration of the event (Bergh *et al.* 1978, Saltin *et al.* 1976). Many studies have confirmed the existence of a high $\dot{V}O_2$ max in

successful endurance runners and values of 70 and 80 ml·kg⁻¹·min⁻¹ are often observed in elite male runners (Bergh *et al.* 1978, Costill *et al.* 1976, Gollnick *et al.* 1972, Saltin *et al.* 1976). In addition, endurance trained runners are known to be able to sustain higher work intensities within the aerobic range at which sprinters would require energy supplementation from anaerobic sources. This is supported by a significantly greater anaerobic threshold in endurance athletes in comparison with sprinters (Paavolainen *et al.* 1994).

In contrast to endurance running, the relatively slow activation and rate of energy delivery of oxidative phosphorylation cannot meet the energy requirements of contraction for sprinting. In this situation, the ability to perform high intensity exercise is dependent on anaerobic energy production (Komi 1977b). It is known that brief periods of strenuous exercise greatly exceed the respiratory capacity of skeletal muscle. The additional energy needed for such a burst of activity is derived from anaerobic processes which can contribute approximately four times as much energy as does respiration during maximal exercise of 10s duration (Medbo and Burgers 1990). This energy is supplied from high energy phosphates and anaerobic glycolysis.

Based on the decrease in muscle PCr and ATP, as well as accumulation of metabolites like pyruvate and lactate, the anaerobic energy production of the working muscle has been quantified. Estimates of anaerobic ATP turnover rates in humans during high intensity sprint exercise are shown in Table 1.3:

Table 1.3. Estimation of anaerobic ATP turnover in sprint exercise (Adapted from Bangsbo 1998).

References	Type of Exercise	Duration of Exercise (s)	Anaerobic Energy Production (mmol ATP·kg ⁻¹ d.w)	% Contribution		
				ATP	PCr	Glycolysis
Boobis <i>et al.</i> (1982)	Cycling, max	30	123	6	30	64
Jones <i>et al.</i> (1985)	Cycling, max	10	173	4	31	64
Cheetham <i>et al.</i> (1986)	Running, max	30	183	6	32	63
McCartney <i>et al.</i> (1986)	Cycling, max	30	228	4	19	77
Nevill <i>et al.</i> (1989)	Running, max	30	186	3	30	67
Gaitanos <i>et al.</i> (1993)	Cycling, max	6	89	6	50	44

Max= Maximal effort

The capacity for anaerobic ATP provision is limited. This is consistent with the ability of humans to attain power outputs that are 2 to 4 times that required to elicit $\dot{V}O_2$ max, but only maintain them for short periods of time. A large anaerobic capacity is, therefore, of great importance for success in sprint events where a short burst of intense power is required.

1.1.6 Physiological profiles of sprinters and endurance runners.

Many physiological characteristics differentiate sprint and endurance athletes, for example maximum cardiac output, $\dot{V}O_2$ max, capillary density, muscle fibre type distribution and metabolic characteristics (Andersen and Henriksson 1977, Bergh *et al.* 1978, Bernus *et al.* 1993, Cox, 1986, Klausen *et al.* 1981, Spina *et al.* 1992, Torok *et al.* 1995).

Endurance runners possess a high oxidative capacity whereas sprinters are characterised by a high phosphagen and glycolytic capacity. These characteristics are derived from heredity and specific training adaptations. As the characteristics attributed to heredity are unmodifiable, the adaptations by sprint and endurance training have, therefore, been investigated.

1.2 Training methods

A number of physiological and biochemical adaptations are known to occur as a result of training over several days, weeks or months. The magnitude of adaptation is dependent on the manipulation of intensity and duration of exercise, the mode of training and the frequency of repetition of the activity, genetic limitations, and the level of prior activity of the individual (Maughan *et al.* 1997).

To bring about effective adaptation, a specific and repeated exercise overload must be applied. Adaptation to training will only occur if the individual exercises at a level above their normal habitual activity on a frequent basis. The appropriate overload for any individual can be achieved by manipulating combinations of training intensity, duration, frequency and mode (Fox *et al.* 1975). Physiological and metabolic adaptations to training are generally specific to the nature of exercise overload, for example, sprint training will induce different adaptations to endurance training (Fournier *et al.* 1982, Gollnick *et al.* 1973, Hickson *et al.* 1975).

Training methods have, consequently, involved differing programmes for improving both aerobic (endurance) and anaerobic (sprint) energy capacities and performances.

Of the many training procedures, the principal methods for enhancing aerobic and anaerobic processes are interval and continuous training. Although both methods have practical

applications, unique and important responses to continuous and interval training have been observed (Gorostiaga *et al.* 1991).

Continuous training regimes are predominantly used by endurance athletes where total mileage or distance covered per session and per week is an important overload factor. In all cases, the aerobic systems are the predominant source of energy, and therefore continuous training programmes facilitate the development of endurance capacity (MacDougal *et al.* 1981).

The theoretical basis for all interval training programmes is that short rest periods interspersed between periods of heavy exercise increases the total accumulated exercise time before fatigue (MacDougal *et al.* 1981). By interspersing exercise with recovery periods, blood lactate does not accumulate in the exercising muscles to the same extent as observed in continuous exercise at the same exercise intensity (Fox *et al.* 1969, Gorostiaga *et al.* 1991) and consequently the exercise load and duration can be extended.

The reduced lactate accumulation during interval training can be explained physiologically by the interaction between the phosphagen (ATP-PCr) system and anaerobic glycolysis.

Although the ATP-PCr stores are depleted within the first few seconds of exercise, during interval work they are replenished rapidly during the recovery periods (Hultman *et al.* 1967, Margaria *et al.* 1969, Saltin *et al.* 1971). Thus, during each interval run that follows a recovery period the replenished energy sources will again be available, therefore, the contribution of anaerobic glycolysis to energy production will be reduced, as will the accumulation of associated lactate. Consequently, manipulation of the duration and intensity

of interval training can achieve the required training overload by both endurance and sprint athletes.

A number of studies have compared the application of these training methods (Fox *et al.* 1969, Saltin *et al.* 1976, Fournier *et al.* 1982) in connection with athletic performance improvement, however, these studies utilised differing exercise intensities for the exercise modes making accurate comparison problematic. This issue was addressed in the study by Gorostiaga *et al.* (1991) in which the response to interval and continuous training was evaluated from the same exercise intensity. This study found differing training responses although both methods utilised an exercise intensity of 69% $\dot{V}O_2$ max for 30min of physical work. The study found the oxidative enzyme citrate synthase (CS) significantly increased by 25% ($P < 0.05$) following continuous training but did not increase after interval training. The high energy enzyme adenylate kinase increased 25% following both continuous and interval training, however, this adaptation was only significant after interval training. Interestingly, the study did not note an increase in $\dot{V}O_2$ max for the continuous trained group, while the interval trained subjects significantly increased $\dot{V}O_2$ max by ~16%. Continuous training was, nevertheless, found to be effective at increasing muscle oxidative capacity and delaying the accumulation of lactate during continuous exercise and thus specifically enable endurance athletes to sustain continuous work for prolonged periods, as required in their events. These findings suggest that both training methods have practical application for athletes, although continuous training appears less appropriate for sprinters and might also be less effective than interval training for improving $\dot{V}O_2$ max.

Further training methods utilised by sprinters and endurance athletes in addition to their event specific training are resistance, circuit and flexibility training. Resistance training utilises

bursts of anaerobic energy and has consequently been more commonly associated with the training methods of sprinters and other strength/power athletes. While strength training does not seem to attenuate cardiorespiratory capacity (Hickson 1980), there is some evidence to suggest that simultaneous training for strength with training for endurance interferes with strength development (Hickson 1980) which places doubt on its practical use as a training method for endurance athletes.

The majority of sprint training studies have investigated adaptations to just one training stimulus but in practice sprint trained athletes utilise additional training techniques in addition to specific sprint work (Ward 1988). Delecluse (1997) identified that sprint training involved the integration of a number of elements to improve performance. It has been shown that training which involves resistance and flexibility programs in addition to sprint running improve sprint performance compared to performance achieved through sprint running alone (Dintiman 1964). It is now generally accepted that sprint running speed can be improved considerably by using strength training (Delecluse 1997).

There is a limited amount of published literature which examine the effect of strength training programmes on sprint performance. Wilson *et al.* (1993) found non significant improvements in sprint performance following 10 weeks of weight training, while Smith *et al.* (1981) found a 10% improvement in sprint performance time following 6 weeks of isokinetic training.

These findings might be due to the relatively short duration of the studies as initial muscular strength adaptations over this period are known to be facilitated by improved neural activation rather than longer term muscle hypertrophy adaptation. A number of studies have found no direct impact of neuronal activation training on sprint performance (Delecluse *et al.* 1995, Dintiman 1964, Fry *et al.* 1991, Wilson *et al.* 1993) and apparently no studies have examined

the effect of hypertrophy training on sprint performance. Until more experimental evidence is available, the relative importance of weight training alone to sprint performance is unclear.

1.2.1 Specific training adaptations for sprint performance

Training that involves brief periods of sprinting causes specific changes in the immediate (ATP and PCr) and short term (glycolysis) energy delivery systems and an improvement in sprint performance. This process is shown in Table 1.4.

Table 1.4. Effects of sprint training on muscle enzyme activity and sprint performance in men

(Adapted from MacDougal *et al.* 1998).

Reference	Training Regime	Enzymatic Changes	Change in Sprint Performance
Costill <i>et al.</i> (1979)	30s max isokinetic exercise, 4x/wk for 7 wk	PHOS↑, PFK↑, CK↑, SDH↑, MDH↑	14 % ↑
Roberts <i>et al.</i> (1982)	20 to 30s sprints, 3x/wk for 5wk	PHOS↑, PFK↑, LDH↑, MDH→, SDH→	20% ↑
Jacobs <i>et al.</i> (1987)	15 and 30s sprints, 2-3x/wk for 6wk	PFK↑, CS↑	→
Cadefau <i>et al.</i> (1990)	30-80m sprints and 100-to 500m runs	PHOS↑, PFK↑, LDH→, SDH↑, CK→	5% ↑
Linossier <i>et al.</i> (1993)	5s sprints, 4x/wk for 7wk	PFK↑, LDH↑, CS→	25% ↑
Linossier <i>et al.</i> (1997)	5s sprints, 4x/wk for 9wk	PFK↑, PHOS↑, LDH↑	28% ↑
MacDougal <i>et al.</i> (1998)	30s sprints, 3x/wk for 7wk	HK↑, PFK↑, CS↑, SDH↑, MDH↑	19% ↑

PFK, phosphofructokinase; LDH, Lactate dehydrogenase; SDH, succinate dehydrogenase; CK, creatine kinase; PK, PHOS, phosphorylase; MDH, malate dehydrogenase; CS, citrate synthase. ↑, Increase; →, no change.

Sprint performance has been measured by an increase in the maximum power output (McKenna *et al.* 1997, Linossier *et al.* 1997), an increase in the performance time for a fixed distance (Roberts *et al.* 1982, Nevill *et al.* 1989) and an increase in exercise duration at high intensities (Saltin *et al.* 1976).

The overall capacity of the ATP-PCr system is enhanced in individuals possessing greater muscular stores of ATP and PCr and increased activities of key enzymes involved in their

resynthesis (Karlsson *et al.* 1972, Thornstenson *et al.* 1975). Muscular stores of ATP and PCr have been shown to increase following training programmes (Wilmore *et al.* 1975) and are known to exist in greater proportions of type II muscle fibres commonly found in elite sprinters (Gollnick *et al.* 1972, Costill *et al.* 1976, Bernus *et al.* 1993). Stored phosphates represent the most rapidly available source of energy for the muscle and therefore their increased availability would assist in improved sprint performance.

It has been suggested that training not only increases the storage capacity of ATP and PCr, but also enhances their rate of turnover. This could be facilitated by increased activities of the enzymes involved in this process. Sprint training is known to effect several key enzymes of the ATP-PCr system (Thornstenson *et al.* 1975, Staude *et al.* 1973, Karlsson *et al.* 1972, Linossier *et al.* 1997b) in which the resynthesis of ATP is facilitated by the enzymes creatine kinase (CK) and adenylate kinase (AK).

The effect of sprint training on CK is equivocal. While early work has shown increased levels of activity (Karlsson *et al.* 1972, Staude *et al.* 1973) more recent studies have demonstrated that total CK activities are not modified by training (Simoneau *et al.* 1987, Linossier *et al.* 1997b) or detraining (Chi *et al.* 1983). This can perhaps be explained by the natural abundance of CK in skeletal muscle. In untrained men, the CK isozymes in the cytoplasm which participate in the regeneration of ATP from PCr represent 97% of total CK activity (Apple and Rogers 1986, Apple and Tesch 1989). As such, CK activity would not normally represent a rate limiting step in PCr depletion (Meyer *et al.* 1984) and should not effect exercise which uses this metabolic pathway.

When sprint performance is improved as a result of sprint training, the rate of ATP turnover is further increased by an increased contribution from anaerobic glycolysis to the ATP supply. Studies have indicated that several of the key enzymes that control glycolysis are significantly effected by sprint training and it has been demonstrated that the activities of important glycolytic enzymes are much higher in sprinters than in endurance runners (Costill *et al.* 1976). The activity of phosphofructokinase (PFK) and other glycolytic enzymes such as phosphorylase (PHOS) and lactate dehydrogenase (LDH) have been shown to increase following sprint training (Table 1.4), with the most dramatic changes occurring in the type II fibres (Linossier *et al.* 1997b, Linossier *et al.* 1993, Costill *et al.* 1979, Staudte *et al.* 1973).

The increase in glycolytic enzyme activities is significant in that they accelerate the rate and quantity of glycogen broken down to lactic acid. Therefore, the ATP energy derived from anaerobic glycolysis is increased and thus contributes to the improved performance of activities that depend on this system for energy. Evidence for an increased glycolytic capacity following training is also demonstrated by the ability to accumulate significantly greater quantities of lactic acid following maximal exercise (Sharp *et al.* 1986).

While sprint training studies have typically demonstrated significant increases in the activity of glycolytic enzymes, the effect on oxidative enzymes is less clear. A number of reports have shown that compared to endurance training, sprint training has either no effect (Linossier *et al.* 1993) or a lesser effect (Roberts *et al.* 1982) on mitochondrial enzyme activity. The study by Roberts *et al.* (1982) (Figure 1.3.) reported significant increases in glycolytic but not oxidative enzymes in response to sprint training. A limited number of studies have shown that sprint training can lead to improvements in $\dot{V}O_2$ max (Saltin *et al.* 1976) indicating a beneficial cross-training effect of sprint activity on endurance performance. These adaptive

responses could be due to the different training stimuli employed by researchers in terms of exercise duration and chosen protocol.

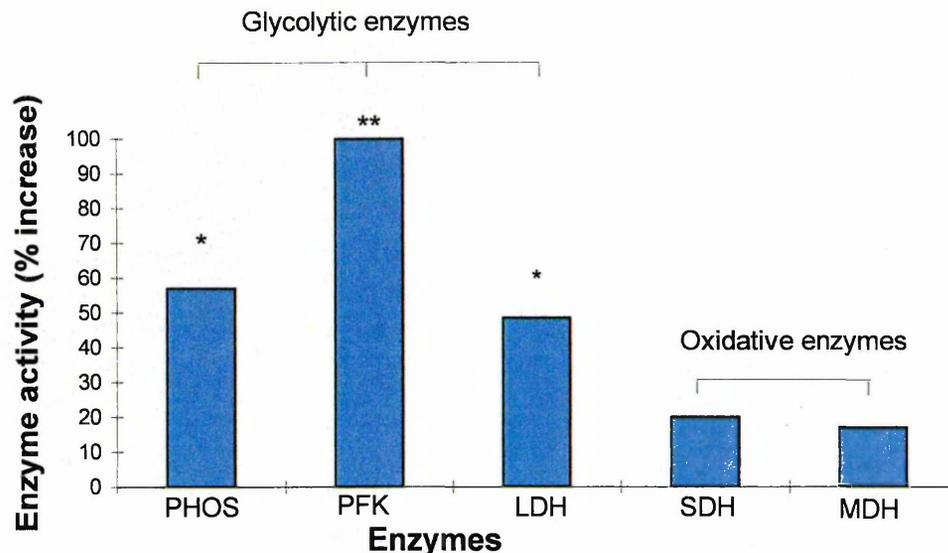


Figure 1.3. Changes in enzyme activities following sprint training. Significant change with training, *, $P < 0.05$ and ** $P < 0.01$ (Adapted from Roberts, A.D *et al.* 1982).

After a regime of sprint training, higher concentrations of blood lactate can be observed during maximal exercise (Boobis *et al* 1983, Nevill *et al* 1989, Sharp *et al* 1986) and this might be due to the higher levels of intramuscular glycogen and glycolytic enzymes present. Sprint training requires considerable motivation and increased pain tolerance to the metabolic acidosis could contribute to the higher levels of blood lactate observed after sprint exercise in the trained state. Improvements in the capacity of muscle to buffer the protons associated with lactate accumulation could also be of importance, as training is known to lead to reduced lactate accumulation through greater clearance potential (Phillips *et al.* 1995b). Muscle H^+ regulation is enhanced after sprint training, with an increased *in vivo* muscle buffering capacity in type II muscle fibres of humans (Sharp *et al.* 1986) and an increased muscle

lactate transport capacity in rats (Pilegaard *et al.* 1993). Consequently, sprinters with higher levels of type II fibres would experience reduced fatigue during maximal exercise due to a greater muscle buffering capacity.

In summary, the rapid release of energy by the muscle cell can be altered through short term, high intensity sprint training. Relatively brief sprint interval training can bring about an increase in glycolytic and oxidative enzyme activity with the effect of increasing both short term sprint performance and $\dot{V}O_2$ max. The increase in the $\dot{V}O_2$ max of sprint trained subjects indicates that sprint training has, to some extent, a beneficial effect on endurance characteristics above that of non-endurance trained groups.

1.2.2 Specific training adaptations for endurance performance

Following endurance training, oxygen consumption at a given submaximal work load remains largely unchanged or slightly decreased compared with pre training levels (Hickson *et al.* 1978). This might be interpreted as an expression of a more effective oxygen transport system, leading to a diminished contribution of anaerobic processes. The most pronounced difference in O_2 consumption at a given submaximal work rate exists between highly trained and nontrained subjects (Fukuoka *et al.* 1995), however, endurance runners are also known to require lower levels of O_2 consumption than power athletes (Fukuoka *et al.* 1995). In addition, at a given submaximal O_2 uptake, the level of lactic acid in the blood of endurance athletes is lower compared with power athletes (Torok *et al.* 1995, Fukuoka *et al.* 1995).

Cardiovascular and respiratory adaptations to training influence the delivery of oxygen to the working muscles. Several researchers believe that exercise involving a large muscle mass,

such as endurance running, is limited by oxygen delivery to the working muscles rather than by the capacity of the muscles to extract and utilise oxygen (Cochrane and Hughson 1992, Hughson 1990b). The central adaptations resulting from endurance training include an increased heart size, stroke volume, blood volume, haemoglobin levels and capillary density in conjunction with a decreased heart rate (Andersen and Henriksson 1977, Kiessling *et al.* 1971, Ekblom and Hermansen 1968, Bevegard 1963, Klausen *et al.* 1981).

The heart size of athletes are generally greater than healthy nonathletes (Kiessling *et al.* 1971). Cardiac hypertrophy can be viewed as a fundamental biological adaptation of muscle to an increased work load (Goldberg 1975) and heredity (Bouchard 1986). The degree to which the relatively large heart volumes of endurance athletes reflect genetic endowment or training adaptations has yet to be determined.

Effective endurance training requires prolonged periods of time during which the cardiac output is sustained at high levels (Fox *et al.* 1972) and has been known to reach levels near $40 \text{ L}\cdot\text{min}^{-1}$ in endurance runners (Ekblom *et al.* 1968). The training response (volume stress) causes cardiac hypertrophy through an increase in the size of the left ventricular cavity. Power trained athletes are not subjected to volume stress but rather intermittently elevated arterial blood pressure. The cardiac hypertrophy in response to this stimulus is a thickening of the left ventricular wall. Therefore, even though the magnitude of cardiac hypertrophy is similar in power athletes to endurance runners, their stroke volume capacities are no different from those of nonathletes.

The stroke volume of the heart during constant load submaximal exercise is increased following endurance training (Bevegard 1963, Fox *et al.* 1975). This effect is related to the

increased size of the ventricular cavity and to an increased myocardial contractility also promoted by training.

The blood flow in trained subjects is lower than in untrained subjects at the same submaximal workload (Klasson *et al.* 1970). The decrease in blood flow is compensated for by a decreased blood flow to the non exercising areas and a greater redistribution to the working muscles (Klasson *et al.* 1970, Miyachi *et al.* 1998).

Endurance training is known to increase capillary density and capillary to muscle fibre ratio (Klausen *et al.* 1981, Andersen and Henriksson 1977) and trained endurance runners are known to possess significantly greater capillary density than sprinters (Torok *et al.* 1995). The number of capillaries surrounding each skeletal muscle fibre is related to the size and type of the muscle fibre (Brodal *et al.* 1977) and the number of mitochondria per muscle fibre (Andersen 1975). Increased capillarization could be an explanation for the reduced total blood flow rate through the capillaries as this could increase the time available for diffusion of oxygen to occur from the red blood cells.

For many years, the metabolic adaptations to endurance training were believed to be due simply to improvements in central cardiac function. In 1967, Holloszy *et al.* reported that an endurance training program doubled the capacity of the mitochondrial fraction of rat muscle to oxidise pyruvate. Similar increases were observed in cytochrome c content and succinate dehydrogenase activities. These findings have subsequently been confirmed in humans (Gollnick *et al.* 1972, Morgan *et al.* 1971).

These adaptations are primarily the result of increases in the size and number of mitochondria (Morgan *et al.* 1971). Mitochondria from trained muscle have a greatly increased capacity to generate ATP aerobically by oxidative phosphorylation. Kiessling *et al.* (1971) demonstrated a 120% increase in the number of mitochondria in the vastus lateralis muscle of humans following a 28 week endurance training program. Several studies have shown increases in the number, size and membrane surface area of mitochondria (Costill *et al.* 1976, Holloszy *et al.* 1967) and an increase in the level of activity of the enzymes involved in the TCA cycle and electron transport system (Benzi *et al.* 1975, Holloszy *et al.* 1967).

Many metabolic reactions involved in the TCA cycle and electron transport system are controlled by the presence of specific enzymes. Training results in an increased level of activity of these enzymes and therefore more ATP can be produced in the presence of oxygen. Changes in the activity of specific enzymes within the mitochondria appear to occur at individual levels. Holloszy *et al.* (1970) reported the cytochrome c content of the gastrocnemius muscle of rats was elevated above control values by 102% in response to endurance training, whereas the activity of TCA enzymes: citrate synthase, succinate dehydrogenase and malate dehydrogenase increased by 34-101%. The level of activity of these enzymes in rat skeletal muscle have been shown to double in the course of 12 weeks endurance training (Holloszy 1967). In humans, the increase in just 8 weeks of training is ~27% (Henriksson 1977a), but this is a specific response occurring only in those muscles involved in the exercise training. A summary of oxidative enzyme change in humans following endurance training is shown in Table 1.5.

Table 1.5. Effects of endurance training on muscle enzyme activity and endurance

performance.

Reference	Training regime	Enzymatic change	Performance change
Gollnick <i>et al.</i> (1973)	1hr cycle@75-90% $\dot{V}O_2$ max, 4x/wk for 5 months	PFK \uparrow , SDH \uparrow	$\dot{V}O_2$ max (15% \uparrow)
Andersen and Henriksson (1977)	40min cycle@80% $\dot{V}O_2$ max, 4x/wk for 8wk	Cy \uparrow , SDH \uparrow	$\dot{V}O_2$ max (16% \uparrow)
Henriksson <i>et al.</i> (1977a)	45min cycle@70% $\dot{V}O_2$ max, 3x/wk for 8wk	SDH \uparrow ,	$\dot{V}O_2$ max (11% \uparrow)
Klausen <i>et al.</i> (1981)	30min cycle @ HR ~170 b.min, 3x/wk for 8wk	Cy \rightarrow , LDH \uparrow , PFK \rightarrow , SDH \uparrow ,	$\dot{V}O_2$ max (15% \uparrow)
Green <i>et al.</i> (1992)	2hrs cycle@67% $\dot{V}O_2$ max for 5-7 days	CS \rightarrow , SDH \rightarrow ,	$\dot{V}O_2$ max \rightarrow
Phillips <i>et al.</i> (1995a)	2hrs cycle@60% $\dot{V}O_2$ peak, 5-6x/wk for 30days	CS \uparrow	$\dot{V}O_2$ peak (11% \uparrow) $\dot{V}O_2$ kinetics (26% \uparrow)
Phillips <i>et al.</i> (1996)	2hrs cycle@59% $\dot{V}O_2$ peak, 5-6x/wk for 31days	HK \uparrow , LDH \uparrow , MDH \uparrow , PFK \rightarrow , PHOS \rightarrow , SDH \uparrow	$\dot{V}O_2$ peak (10% \uparrow)

CS, citrate synthase; Cy, cytochrome oxidase, HK, hexokinase; LDH, lactate dehydrogenase; MDH, malate dehydrogenase; PFK, phosphofruccokinase; PHOS, phosphorylase; SDH, succinate dehydrogenase. \uparrow , significant increase in performance ($P < 0.05$); \rightarrow , no change.

The glycolytic enzymes of endurance athletes appear to have the same or a slightly reduced activity compared with untrained subjects (Costill *et al.* 1976). The activities of the LDH isozymes 1 and 2 are exceptions and both of these enzymes appear to follow the profile of the oxidative enzymes (Karlsson *et al.* 1975).

The isozymes LDH₁ and LDH₂ are bound to mitochondria and favour the oxidation of lactate to pyruvate during aerobic metabolism while the isozymes LDH 3-5 favour the conversion of pyruvate to lactate (Apple and Rogers 1986a and 1986b). Karlsson *et al.* (1974) found that the LDH isozyme pattern in human skeletal muscle was related to fibre type, with slow twitch muscle fibres containing a higher percentage of the LDH₁ and LDH₂ isozymes than fast twitch muscle. Total LDH activity is lower in endurance trained athletes than in power athletes, however, the activity of isozymes LDH₁ and LDH₂ is significantly higher in endurance trained athletes (Karlsson *et al.* 1975). This suggests that endurance training assists in improved lactate clearance through the increased activities of the oxidative isoforms of LDH.

The oxidative capacity of both Type I and Type II fibres can be enhanced by endurance training as the increase in mitochondrial density is not specific to the Type I fibres (Gollnick *et al.* 1973). The oxidative adaptation appears to occur in similar proportions in both the main fibre types and thus the Type I fibres maintain a higher oxidative capacity than Type II fibres. Interestingly, the oxidative capacity of Type II fibres of elite endurance runners have been shown to exceed the oxidative capacity of the Type I fibres of untrained individuals (Essen *et al.* 1975).

Endurance training increases the capacity of trained skeletal muscle to oxidize carbohydrate. There is also an increased glycogen storage capacity in the muscle following training (Gollnick *et al.* 1972, 1973). It has also been demonstrated (Bergstrom *et al.* 1967) that the initial level of muscle glycogen is directly related to endurance capacity. This increase in glycogen storage is due, in part, to the training induced increased activity of the enzymes responsible for glycogen synthesis and breakdown. Consequently, large amounts of pyruvate can be converted to acetyl-CoA and moved through the TCA cycle.

Endurance training has also been shown to improve the oxidation of fat (Mole *et al.* 1971) in addition to pyruvate (Holloszy *et al.* 1967). The increase in the capacity of skeletal muscle to oxidize fat following endurance training is related to three factors: an increase in the intramuscular stores of triglycerides, an increased release of free fatty acids from adipose tissue and an increase in the activation, transport and breakdown of fatty acids (Hoppeler *et al.* 1973, Benzi *et al.* 1975). The increased oxidation of lipid is probably a consequence of an increase in the potential for oxidation of substrates relative to endurance training and therefore, trained endurance athletes are likely to use more fat and less carbohydrate.

The respiratory exchange ratio (RER i.e. the ratio of $\dot{V}CO_2$ [CO₂ release] to $\dot{V}O_2$) gives a reasonable estimate of the proportions of carbohydrate and lipid being oxidised in steady state exercise. An RER value of 1.0 indicates that only carbohydrate is being oxidised, whereas an RER of 0.7 indicates that fat is the sole substrate being oxidised. As exercise intensity increases, the level of fats being oxidised decreases with a concurrent increase in carbohydrate oxidation. During moderate exercise, RER values of ~0.85 are average for healthy subjects suggesting that a combination of carbohydrate and fat is being oxidised. Numerous studies (Coggan *et al.* 1993, Green *et al.* 1991, Hurley *et al.* 1986, Gollnick *et al.* 1973) have confirmed that endurance training lowers the respiratory exchange ratio (RER) during exercise indicating an improved effectiveness of O₂ utilisation through the increased proportion of fats being oxidised.

Associated with the decreased rate of carbohydrate oxidation during exercise in the trained state is a decreased rate of lactate accumulation in the blood. During exercise with heavy but submaximal work loads, greater fat oxidation would lead to reduced glycogen depletion, less

lactic acid accumulation and less muscular fatigue (Davies *et al.* 1981). In addition, an enhanced ability to clear excess lactate from the blood following endurance training (Phillips *et al.* 1995b) could also contribute to reduced blood lactate accumulation during exercise. A decreased rate of carbohydrate oxidation and a decreased rate of lactate accumulation result in a sparing of the body's limited carbohydrate reserves. Therefore, the decreased rate of glycogen utilisation is a major factor in improving performance in endurance events.

1.2.3 The time course of adaptations to training in relation to improved oxidative potential.

While it is known that physiological adaptations occur to enhance endurance performance, it is unclear whether there is a sequential order in which adaptations occur over the period of training. From this, it is difficult to relate which mechanisms control, and thus limit, endurance performance over a course of endurance training. A study by Phillips *et al.* (1995a) observed that $\dot{V}O_2$ kinetics were improved after only 4 days of endurance training which appears a more rapid adaptation than traditionally associated with maximal tests of aerobic capacity. This indicates that different physiological mechanisms could be responsible for test performances between $\dot{V}O_2$ kinetics and $\dot{V}O_2$ max.

Many studies have been conducted on the time course of endurance adaptation and although these studies can be compared one should be aware of the different training regimes applied by the various researchers. A schematic summary of the main endurance training adaptations is shown in Figure 1.4.

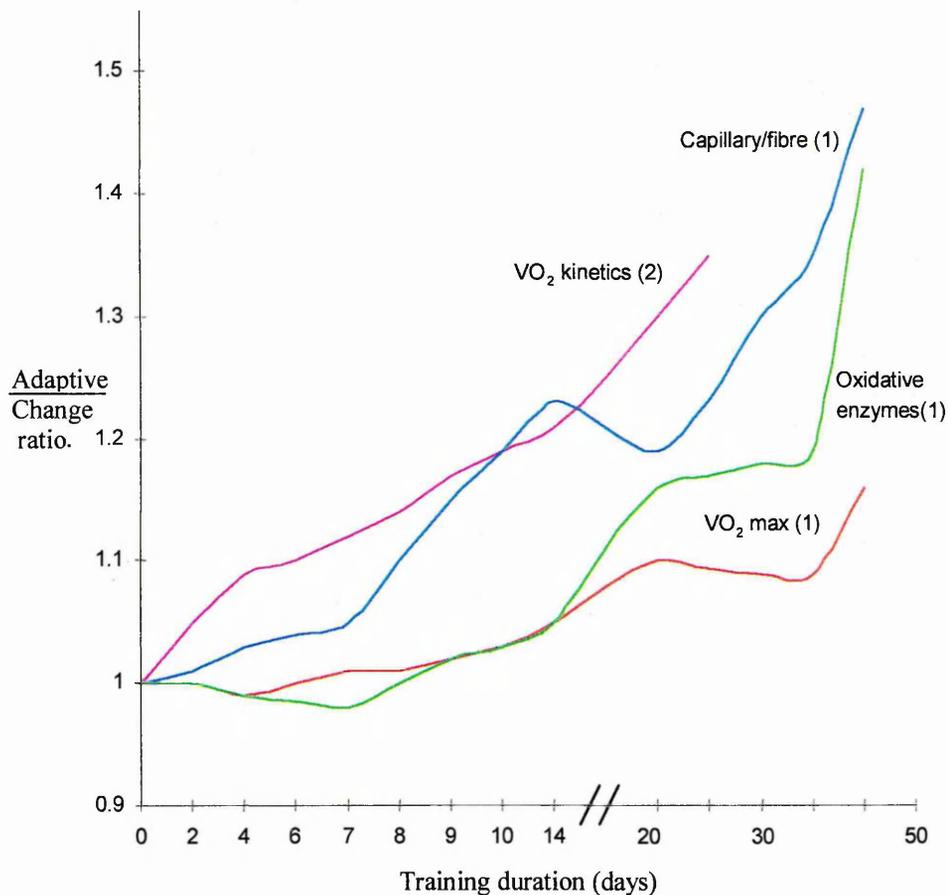


Figure 1.4. A schematic summary of some of the adaptations taking place with endurance training. The graph is based on the endurance training studies of (1) Andersen and Henriksson (1977) (capillary/fibre ratio, oxidative enzymes and $\dot{V}O_2$ max) and (2) Phillips *et al.* (1995a) ($\dot{V}O_2$ kinetics). Further details of the training studies are shown in Table 1.5.

$\dot{V}O_2$ max has been considered an indicator of the capacity to deliver O_2 to the working muscle, however, short term endurance training studies (<7 days) have not shown improvements in $\dot{V}O_2$ max (Green *et al.* 1992, Andersen and Henriksson 1977). For many years it was thought that the increase in the capacity for endurance exercise was exclusively the result of the cardiovascular adaptations to endurance training, which by increasing the capacity of O_2 delivery to the working muscles were primarily responsible for the large increase in $\dot{V}O_2$ max. Central to this concept is the theory that the working muscles become

hypoxic during exercise and that as a result of training they become less hypoxic at the same relative exercise intensity. Many longitudinal endurance training studies have shown $\dot{V}O_2$ max to be a relatively long term adaptation with no significant improvements made before 8 weeks of training (Klausen *et al.* 1981, Henriksson 1976). Although, Henriksson *et al.* (1977b) later reported a significant ($P < 0.001$) improvement (11.1%) in $\dot{V}O_2$ max after 3 weeks of endurance training, however, $\dot{V}O_2$ max is known to typically increase by ~15 - 30% during the first 2-3 months of training, while a further improvement of ~40 - 50% occurs over the next 9-24 months (Saltin *et al.* 1977).

Phillips *et al.* (1995a) has shown that $\dot{V}O_2$ kinetics are improved by 4 days of endurance training, indicating that the ability to respond to change in work at a sub maximal level might depend on differing mechanisms than those regulating $\dot{V}O_2$ max. The possible mechanisms responsible for this rapid improvement in endurance performance are as yet unclear.

At the muscle level, it is known that human skeletal muscle undergoes an adaptive increase in oxidative enzymes in response to endurance training (Gollnick *et al.* 1973, Morgan *et al.* 1971) and that elite endurance trained runners have increased levels of these enzymes (Costill *et al.* 1976, Gollnick *et al.* 1972). More recent studies (Green *et al.* 1991, Phillips *et al.* 1995a) have been unable to detect changes in mitochondrial enzyme activities or oxidative capacity after 5-10 days of training suggesting that O_2 utilisation does not significantly influence the rapid $\dot{V}O_2$ kinetics improvement. Based on these findings, the early stages of training could be primarily influenced by factors other than mitochondrial metabolic capacity.

Although many of the 'structural' cardiovascular adaptations such as heart size and volume occur over a prolonged period, it is known that regional blood flow is rapidly increased early

in endurance training (Shoemaker *et al.* 1996). It is probable that as the blood flow to the working muscle during exercise is increased, adaptations occur to utilise this improvement. It has been proposed (Henriksson *et al.* 1977b) that the increase in blood flow acts to stimulate new capillary growth around the fibre, thus providing it with greater surface area for diffusion to the muscle cell.

Rapid capillary growth has been shown in studies of rabbit fast twitch muscle. It has been reported that prolonged electrical stimulation at a frequency naturally occurring in a nerve to a slow twitch muscle has resulted in growth of new capillaries after 2 days (Skorjanc *et al.* 1998) and 4 days (Hansen-Smith *et al.* 1996, Brown *et al.* 1973) preceding the changes in the activity of oxidative enzymes (Brown *et al.* 1973, Cotter *et al.* 1972, Skorjanc *et al.* 1998). That increases in capillarization and mitochondrial enzyme activities seem to follow different time courses is most evident during the first days of electrical stimulation at a frequency of 10 Hz 12 hours a day, 1 hour on followed by 1 hour off and is shown in Figure 1.5:

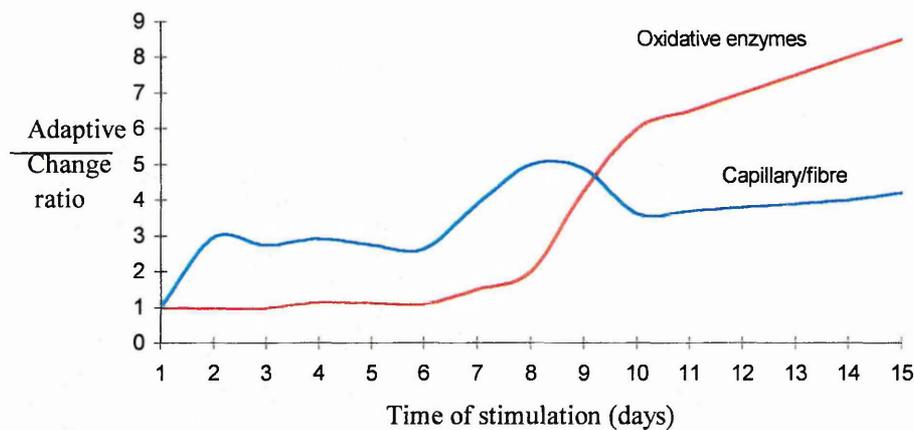


Figure 1.5. Adaptation of rabbit fast twitch muscle to chronic low frequency electrical stimulation (10Hz, 12h/day, 1h on-1h off). Adapted from the data of Skorjanc *et al.* (1998).

Significant changes in the capillary to fibre ratio were observed in 2 days, whereas, increases in enzyme activities become evident only several days later (Skorjanc *et al.* 1998). Significant elevation of SDH activity was only recorded after 8 days.

Few studies of humans have examined capillary changes over such a short time period, although, Andersen (1975) found that increases in capillary to fibre ratio preceded oxidative enzyme change in response to endurance training (Figure 1.4). This suggests that enhanced blood supply and thus oxygen delivery to the muscle is required for a stimulation of the synthesis of oxidative enzymes.

Green *et al.* (1991) have argued that capillarization is unlikely to contribute to the early <10 days improvement in endurance performance as increased capillarization has been observed only at 10-12 days (Green *et al.* 1991). However, this contention seems questionable as capillary measurements were not actually taken prior to days 10-12 by which time significant adaptation might have occurred. Consequently, it is likely that an early increase in the network of capillaries to fibre might facilitate improved O₂ supply to the mitochondria in the early stages of training.

These observations indicate that elevations in the capacity of mitochondrial oxidation follow, but do not precede, enhanced capillarization. This points to sequential inductions of firstly improved oxygen supply and secondly mitochondrial induction. This could explain the findings of Phillips *et al.* (1995a) whereby improved $\dot{V}O_2$ kinetics were observed following 4 days training and preceded oxidative enzyme change.

In summary, training brings about specific metabolic and physiological adaptations that involve cardiovascular and intra muscular changes. Physiological changes that occur as a consequence of endurance training include increases in mitochondrial size and number, increased activity of aerobic enzymes, increased capillarization of the trained muscle, and enhanced oxidation of fat and carbohydrate. Sprint training has been shown to enhance the rate of turnover of the glycolytic pathway through increases in specific enzyme activities, however, the effect of sprint training on the oxidative processes remain equivocal.

Endurance training causes functional and dimensional changes in the cardiorespiratory system which include decreases in resting and submaximal exercise heart rate, enhanced stroke volume and maximum cardiac output and greater O₂ extraction. These adaptations contribute to a greater aerobic production of ATP and thus enhanced oxidative capability. It is, therefore, likely that the adaptive changes to endurance training will result in an improved aerobic exercise test performance in comparison with sprinters.

1.3 Gas exchange kinetics

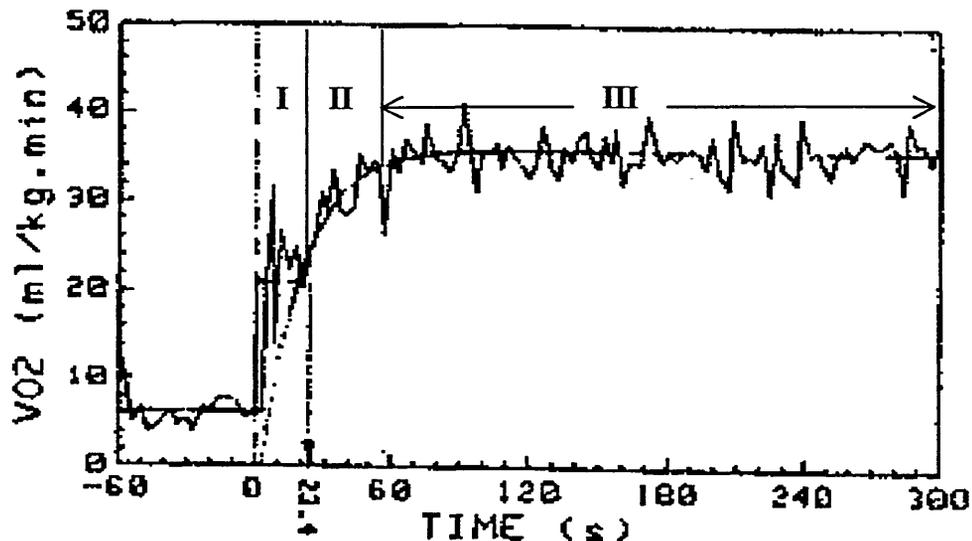
Gas exchange kinetics describe the rate of change of $\dot{V}O_2$ and $\dot{V}CO_2$ in response to the onset of exercise or to a change in work rate (Whipp and Wasserman 1972, Whipp *et al.* 1982). In response to exercise, O_2 uptake at the lungs ($\dot{V}O_2$) largely reflects the O_2 consumed by the cells. CO_2 output ($\dot{V}CO_2$) is known to closely resemble the response of $\dot{V}O_2$ to exercise, although influenced by the intervening body CO_2 stores (Whipp *et al.* 1982). Consequently, individual variations in either $\dot{V}O_2$ or $\dot{V}CO_2$ kinetics may potentially describe aerobic fitness.

1.3.1 The time course of gas exchange kinetics

Both $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics are characterised by three phases (Whipp *et al.* 1982). The time course of these alterations are shown in Figure 1.6.

Figure 1.6. The three time phases of gas exchange in response to a step change in work.

Taken from Hamar, D (1991).



Phase I: The cardiodynamic phase.

Phase I is the initial period, characterised by the immediate increase in gas exchange at the start of exercise (Whipp *et al.* 1982). This rapid response lasts ~15 to 20 seconds covering the period in which venous blood from the active muscle has not yet reached the lungs (Whipp and Wasserman 1972). The increase in venous return and cardiac output that result from the neural responses cause an increased pulmonary blood flow and therefore an increase in $\dot{V}O_2$ and $\dot{V}CO_2$ which is not temporally related to an increased muscle oxygen uptake. Since the magnitude of the phase I $\dot{V}O_2$ and $\dot{V}CO_2$ responses are dependant on the magnitude of the change in work rate and since it does not reflect muscle oxygen uptake, most protocols are designed to minimise phase I by employing work to work transitions rather than rest to work (Hughson & Morrissey 1982, Whipp *et al.* 1982). Work to work exercise protocols minimise the abrupt transition and cardiodynamic adjustment often observed in rest to work protocols.

Phase II: Metabolic phase

During this phase, venous blood from the active muscle arrives at the lungs (Whipp and Wasserman 1972, Whipp *et al.* 1982). This blood has a lower oxygen content and higher CO_2 content than the blood arriving at the lungs during phase I. The resulting increased $\dot{V}O_2$ and $\dot{V}CO_2$ reflect, respectively, the increased muscle O_2 uptake and CO_2 production and continues to rise until a steady state is reached (Phase III). The time constant for the phase II $\dot{V}O_2$ response is ~45secs, corresponding to a half time of ~30 seconds but will vary according to the physical conditioning of the individual.

Phase III: Steady state phase

Provided the work rate is below the lactate threshold (LT), $\dot{V}O_2$ reaches this phase at ~ 3 minutes after the start of exercise (Whipp *et al.* 1982). $\dot{V}CO_2$ is slower to reach steady state, due to the enhanced storage capacity of CO_2 in the tissues and is consequently reached at ~ 4 min (Whipp *et al.* 1982). During the steady state phase, as O_2 supply matches O_2 demand in the cell, blood lactate levels remain below 2mM (Essfeld *et al.* 1987).

1.3.2 The slow component of O_2 uptake

In exercise above the lactate threshold, phase III may not be reached, rather the $\dot{V}O_2$ continues to rise slowly while $\dot{V}CO_2$ increases more rapidly than $\dot{V}O_2$ (Ceretelli *et al.* 1966, Whipp and Wasserman 1972). The slow rise in $\dot{V}O_2$ is known as ‘the slow component’ and does not display first order exponential kinetics (Whipp and Wasserman 1972), an indicator of which would be an increase in blood lactate levels above resting levels (Essfeld *et al.* 1987). It has been shown that phase II $\dot{V}O_2$ kinetics are slowed by the accumulation of lactic acid (Paterson and Whipp 1991). However, $\dot{V}CO_2$ while slower to reach steady state in phase III, speeds up once above LT. This is due to the additional CO_2 supply from HCO_3^- buffering of lactate at high work rates. Therefore, owing to these factors, it is important that a protocol designed to elicit phase II responses has an upper work rate which is below the lactate threshold.

1.3.3 Techniques for measuring gas exchange kinetics

Gas exchange kinetics have been measured in the time domain (Hughson *et al.* 1991, Phillips *et al.* 1995a) and in the frequency domain (Essfeld *et al.* 1987, Hoffmann *et al.* 1992 & 1994a). Step tests have typically been used to analyse $\dot{V}O_2$ kinetics in the time domain (Hughson and Inman 1986, Phillips *et al.* 1995a, Whipp *et al.* 1982) whereas sinusoidal or multifrequent changes in work rate have been used to analyse $\dot{V}O_2$ kinetics in the frequency domain (Hoffmann 1992 & 1994a, 1994b). Frequency response results have been seen as more convenient because their interpretation is calculated without the need for mathematical modelling (Essfeld *et al.* 1987).

The rate of increase in $\dot{V}O_2$ is well characterised in time domain analysis by description of the time constant of the response (Hughson *et al.* 1988, Whipp *et al.* 1982). In step tests, a mathematical model is fitted to the test data and the time taken to reach a particular point (~63% of the overall response) is measured (Hughson *et al.* 1990c). The time taken to reach this point has variously been described as a total lag time (TLT) (Hughson *et al.* 1990c) or mean response time (MRT) (Phillips *et al.* 1995a) depending on the method of calculation and the point measured. A limitation of this method as a practical test is that it requires multiple repeats of the step protocol to generate sufficient data for analysis (Hughson *et al.* 1988, Stegemann *et al.* 1985). Multifrequent tests, for example the pseudo random binary sequence (PRBS) test, have the advantage of being able to gain information about gas exchange kinetics from a single test.

In the PRBS test, work rate is switched between two levels over a predefined period of time according to the output of a computer algorithm known as a shift register with feedback (Bennett *et al.* 1981, Kerlin 1974). Several identical sequences can be placed in series over the duration of the exercise test and consequently considerable test data can be gained from the response to identical cycles of the PRBS. In truly random signals, the work rate transitions from high to low or low to high could occur at any time. In a PRBS, the changes in work rate must occur at specific times and are, therefore, controlled by a computer system (Hampton 1965). The externally controlled changes in work rate allows the test to appear random to the subject who cannot predict where these changes will occur and consequently cannot prepare for them.

The PRBS exercise test potentially provides a sensitive method to measure aerobic fitness (Essfeld *et al.* 1987). Because it is a sub maximal test, it can be applied repeatedly with minimal imposition to the subjects (Hughson *et al.* 1990a) and can also provide a description of $\dot{V}O_2$ kinetics for a range of input frequencies in a single submaximal assessment (Hoffmann *et al.* 1994). This is advantageous for the testing of large population groups as physiological assessment would only require minimal laboratory contact.

A standard method of analysis for multifrequent tests, such as the PRBS test is in the frequency domain by application of Fourier analysis. An advantage of using the PRBS test by frequency analysis is that a properly defined and designed PRBS cycle contains a range of frequencies which include the fundamental (a defined) and integer multiples of the fundamental frequency. A more regular multifrequent sequence such as a regular square wave work rate would be missing certain frequencies according to Fourier analysis theory and so would not provide the same coverage of frequencies as the PRBS. The PRBS can measure

response to a range of sinusoidal inputs in one test, provided the assumption of system linearity can be made.

In this technique the length of the PRBS cycle is treated as a sine wave with different harmonic frequencies. The lowest frequency that completes a full sine wave in the length of the PRBS cycle is known as the fundamental frequency and displays the highest power output. The frequencies of the harmonics are determined according to how many times they occur over the test sequence, for example the frequency of a sine wave occurring once over a 300s test (fundamental) is calculated as $1(\text{signal})$ divided by 300s (test length), corresponding to 3.3mHz. The higher frequencies of the sine waves are then determined according to their occurrence over the same 300s period. As the frequencies become progressively higher, their power becomes dissipated leading to progressively lower levels of power being retained at each frequency and thus the underlying signal becomes less discernible from non physiological background noise. Consequently, only the first few frequencies are considered valid for further analysis.

Analysis by the Fourier method examines the relationship between the work rate input signal and the gas exchange output signal of the subject. This method of analysis assumes that the work rate input will be the same for all subjects, while individual variations in gas exchange will reflect the physiological difference in response between subjects. Therefore, the difference between the input and output signals will indicate the physiological response of subjects to exercise.

Fourier analysis yields estimates of the response to a sinusoidal work rate at each harmonic frequency. From the Fourier analysis amplitude ratios and phase shift angle are calculated

(Jenkins and Watts 1968). The amplitude ratio indicates the magnitude of the response made by the subject, while the phase shift angle describes the delay in the response. The relationship between input work rate and $\dot{V}O_2$ output signals is shown schematically for the fundamental frequency in Figure 1.7. Higher harmonic frequencies are also compared in terms of their magnitude (amplitude ratio) and delay (phase shift) between input work rate and $\dot{V}O_2$ output signals.

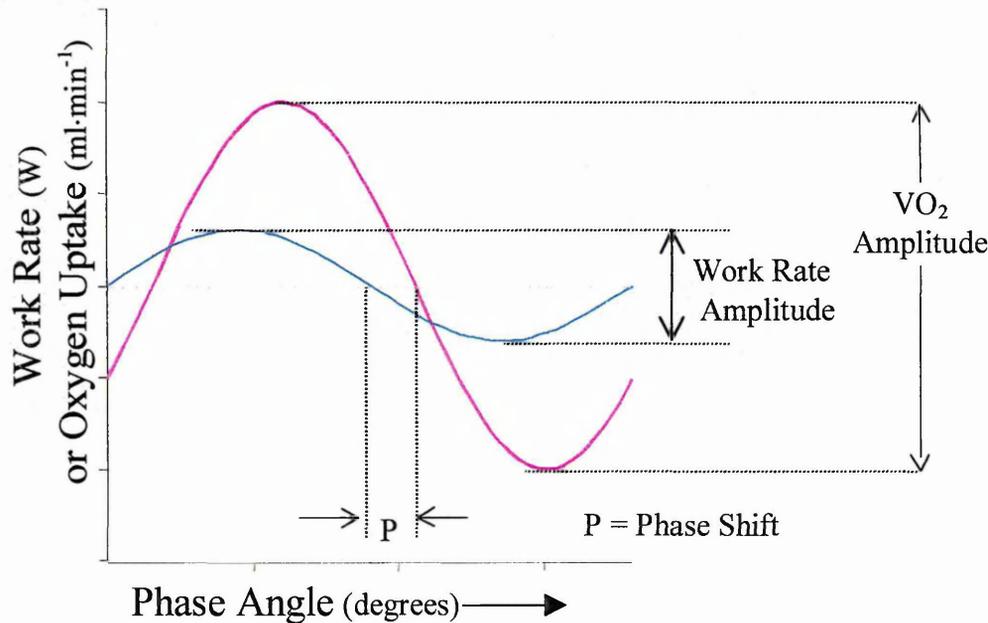


Figure 1.7. Frequency analysis demonstrated for a single sine wave. The period of the sine wave is the length of the PRBS cycle. The ratio between the amplitudes of oxygen uptake (amplitude of the response) and the amplitude of the work rate (amplitude of the input) and P (phase shift) between $\dot{V}O_2$ and work rate provide information about the ability of the subject to meet the oxygen demands caused by a change in exercise intensity.

$\dot{V}O_2$ kinetics have been examined by Essfeld *et al.* (1987), Stegemann *et al.* (1985) and Hoffmann *et al.* (1994) with a PRBS comprising 15 units of 30s/units for a total period for one cycle of 450s. In their protocol, work rates alternated between 20 W and 80 W with a

decision made every 30s according to an algorithm whether to leave the work rate at its existing level or to change it.

Other investigators (Bennett *et al.* 1981 & Hughson 1990a) have used a sequence of 63 units, with 5s/unit for a total period of 315s. These investigators employed this particular sequence because of its greater ability to examine the high frequency 'neural' components of the ventilatory and heart rate responses.

Further investigators (Hoffmann *et al.* 1992, 1994a, 1994b, Kusenbach *et al.* 1999) have applied PRBS comprising 15 units of 20s/unit for a total period of 300s. This sequence represents a shorter potential test length while also ensuring that, like the protocol of Essfeld *et al.* (1987), Stegemann *et al.* (1985) and Hoffmann *et al.* (1994b) it could not exclusively represent the phase I response at any of the work rate forcings. A summary of the PRBS cycles used by various investigators is shown in Table 1.6:

Table 1.6. A selection of PRBS cycles used by various investigators in the study of gas exchange kinetics.

Reference	Unit length (s)	Period (s)	No. of cycles	Work rates (W)	Upper work limit	Analysis (T/F)
Bennett <i>et al.</i> (1981)	5	315	5-6	25-125	AT	T
Stegemann <i>et al.</i> (1985)	30	450	3	20-80	<2mM	F
Essfeld <i>et al.</i> (1987)	30	450	3	20-80	<2mM	F
Hughson <i>et al.</i> (1990a)	5	315	5	25-105	Tvent	F
Kowalchuk <i>et al.</i> (1990)	15	225	6	25-125	Tvent	T
Hughson <i>et al.</i> (1991)	30	450	4	25-105	Tvent	T
Hughson <i>et al.</i> (1991)	5	315	5	25-105	Tvent	T
Hoffmann <i>et al.</i> (1992)	30	450	2	20-80	<2mM·L ⁻¹	F
Hoffmann <i>et al.</i> (1992)	20	300	3	20-80	<2mM·L ⁻¹	F
Hoffmann <i>et al.</i> (1994)	20	300	3	20-80	<2mM·L ⁻¹	F
Kusenbach <i>et al.</i> (1999)	20	300	2	20-80 or 40% of PIWC	40% of PIWC	F

AT= Anaerobic threshold, T=Time domain analysis, F=Frequency domain analysis,

Tvent= Ventilatory threshold, PIWC= Predicted individual working capacity.

As shown in Table 1.6, a number of investigators have applied time domain analysis (Bennett *et al.* 1981, Hughson *et al.* 1991, Kowalchuk 1990) of gas exchange to PRBS exercise tests. Time domain analysis involves the application of auto and cross correlation techniques in order to fit a mathematical model to the breath-by-breath data. As the auto correlation of the PRBS work rate input appears in the form of a triangular shaped pulse, Hughson *et al.* (1991) decided to view this as a ramp increase followed by a ramp decrease in work rate and thus

applied ramp forms of exponential equations to the data. Hughson *et al.* (1991) found agreement between time domain analysis by PRBS and step tests.

Following examination of the PRBS cycles outlined in Table 1.6, a 15 unit protocol of 300s was selected with a base unit of 20s (Hoffmann *et al.* 1992, Kusenbach *et al.* 1999). Exercise intensities were set at 25 W and 85 W. By selecting a 20s unit PRBS it is possible to examine a range of responses in multiples of 20s but not less than 20s. By reducing the high frequency content of a PRBS to concentrate on phase II, the workload becomes much easier for subjects to perform and consequently reduces the effects of fatigue and the likelihood of illness or injury. The exercise intensities of the PRBS test should remain at a sustainable aerobic level as higher intensity exercise would introduce a further slower rise in $\dot{V}O_2$ (slow component) (Whipp *et al.* 1982, Whipp and Ward 1990, Barstow *et al.* 1994).

1.3.4 Factors limiting $\dot{V}O_2$ kinetics

The study of $\dot{V}O_2$ kinetics during single leg cycle ergometry suggests that the limiting factor for phase II $\dot{V}O_2$ kinetics lies within the exercising muscle (Yoshida *et al.* 1995). In this study a submaximal square wave test of $\dot{V}O_2$ kinetics was employed during one legged cycle ergometry. This was followed, after a five minute rest, by an identical test using the same leg. The $\dot{V}O_2$ kinetics were significantly faster after the prior exercise, while heart rate and cardiac output kinetics remained the same. Therefore, if there is no change in heart rate or function, there is no change in the delivery process of O_2 . In contrast, when the alternate leg was used in a third identical test, $\dot{V}O_2$ kinetics were significantly slower when compared to the previous test. Again heart rate and cardiac output kinetics were not effected. This suggests that it is local factors within the exercising muscle that are controlling $\dot{V}O_2$ kinetics and these factors could either be related to blood flow distribution or to metabolic conditions in muscle.

Other studies have compared $\dot{V}O_2$ kinetics to the simultaneous measurement of the kinetics of phosphocreatine (PCr) degradation in the active muscle (Barstow *et al.* 1994 and McCreary *et al.* 1996). It has been proposed that $\dot{V}O_2$ kinetics of the muscle and therefore $\dot{V}O_2$ kinetics measured at the lung, reflect the metabolic processes that control mitochondrial respiration in the exercising muscle (Meyer, 1988, Whipp and Ward, 1990). The rate of PCr degradation has been implicated as a step that reflects the rate of mitochondrial respiration (Mahler 1985). Phosphocreatine breakdown can be estimated by nuclear magnetic resonance imaging (^{31}P NMR) and by muscle biopsy techniques, both in isolated muscle preparations and *in situ* during voluntary muscle contractions in humans (Coggan *et al.* 1993). Barstow *et al.* (1994) measured time constants during two different exercise modes which provided indirect evidence of this link since the time constants for phase II $\dot{V}O_2$ kinetics were similar to those for PCr degradation. In a more recent study, McCreary *et al.* (1996) measured $\dot{V}O_2$ kinetics and PCr kinetics during plantar flexion and found time constants of 44.5 s for $\dot{V}O_2$ kinetics and 47 s for PCr kinetics. The similarity between these time constants supports the contention that $\dot{V}O_2$ kinetics are controlled by muscle oxidative function with PCr acting as the transducer, linking ATP hydrolysis with mitochondrial ATP production.

The sequence of metabolic events in the muscle fibre at the onset of exercise has been shown as a flow diagram in Figure 1.8:

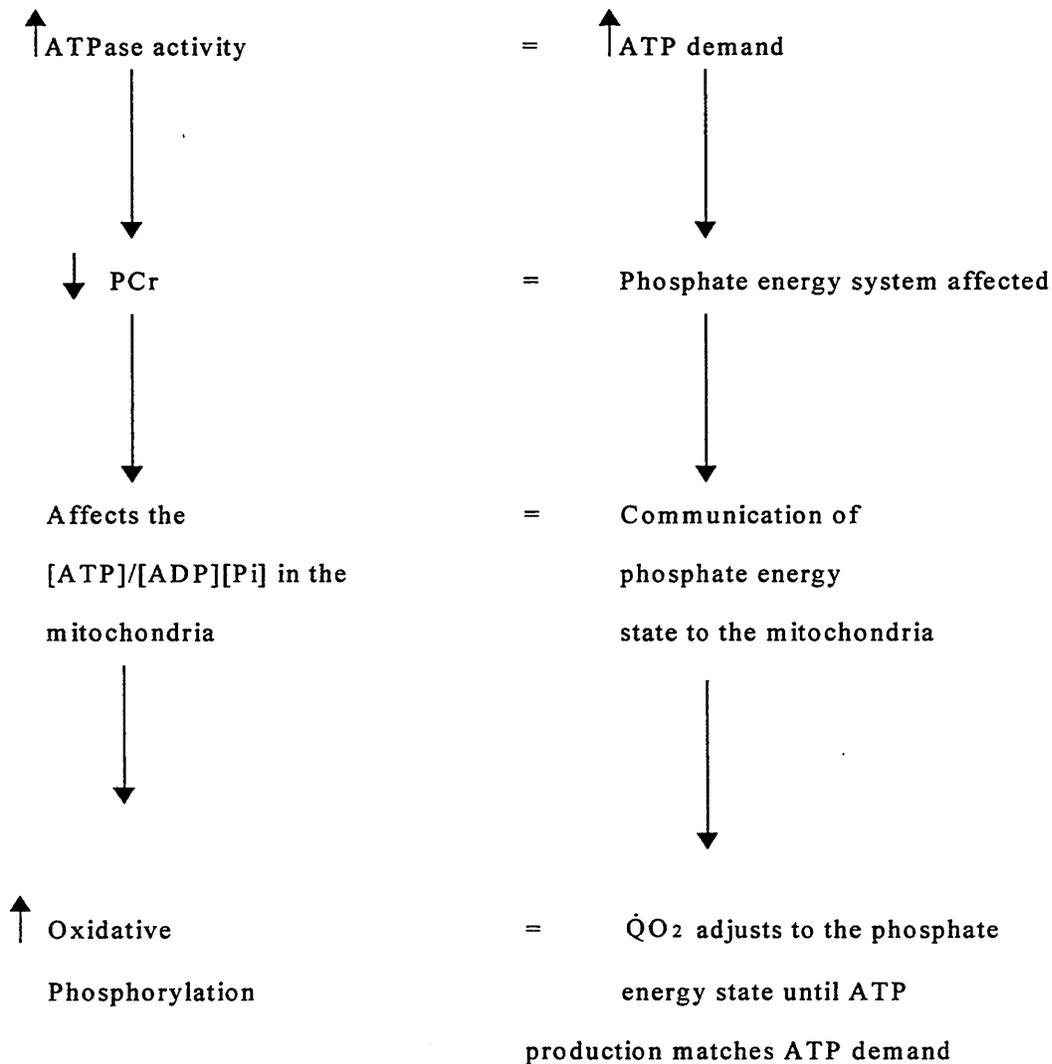


Figure 1.8. Sequence of events at the onset of exercise (Taken from Tschakovsky and Hughson 1999). Increased ATP demand effects phosphate energy system of the cell, which, in turn, directs the change in respiratory rate of mitochondria such that muscle $\dot{Q}O_2$ adjusts in response to phosphate energy state until aerobic ATP production matches ATP demand.

The increased ATP demand effects the phosphate energy system of the cell as shown by a decrease in PCr. The phosphate energy state of the cell is the main controller of oxidative phosphorylation (Meyer and Foley 1996) providing the link between decreased PCr and increased oxidative ATP synthesis and therefore increased muscle oxygen uptake.

Mitochondrial PO_2 and mitochondrial enzyme activation modulate oxidative phosphorylation.

The PO_2 can limit oxidative phosphorylation if it falls below a critical value in the cell. The mitochondrial PO_2 depends on the balance between oxygen consumption and oxygen delivery into the cell. The processes linking ATP demand to aerobic ATP production are shown in

Figure 1.9:

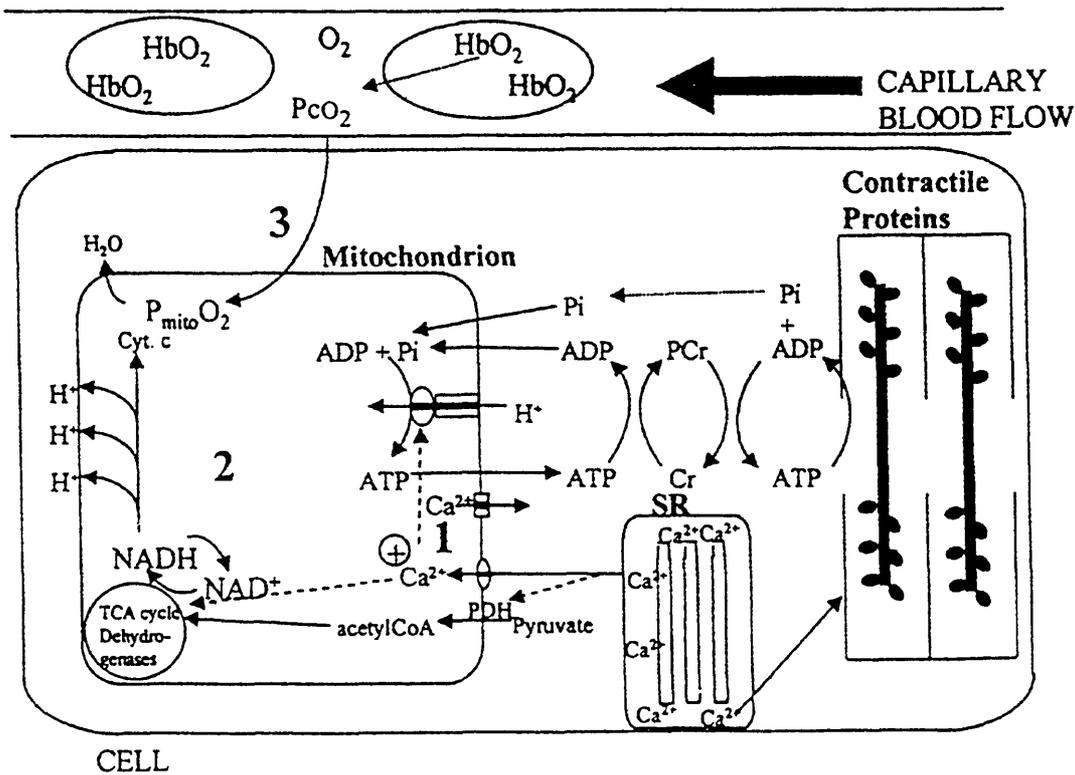


Figure 1.9. Local factors that might interact to determine muscle \dot{V}_{O_2} kinetics (Taken from Tschakovsky and Hughson 1999). 1: $\mu\text{M Ca}^{2+}$ levels in mitochondrial matrix activate dehydrogenases and ATP synthase, which effect mitochondrial resistance and redox potential. Flux kinetics of separate import and export transporters will determine mitochondrial matrix $[\text{Ca}^{2+}]$. 2: $[\text{ATP}]/[\text{ADP}] \cdot [\text{Pi}]$ and $[\text{NAD}^+]/[\text{NADH}]$ contribute to net electron transport system (ETS) flux rate, where the former is likely the main controller, and the latter a modulator of respiratory rate. 3: mitochondrial PO_2 (P_{mitoO_2}) interacts with $[\text{ATP}]/[\text{ADP}] \cdot [\text{Pi}]$ and $[\text{NAD}^+]/[\text{NADH}]$ to determine ETS flux via its effects on mitochondrial resistance. P_{mitoO_2} is dependent on balance between O_2 consumption and O_2 flux in cell [a product of capillary PO_2 (PcO_2), which is, in turn, dependent on local capillary blood flow and Hb affinity for O_2]. Cr, creatine; Cyt. C, cytochrome-c; PDH, pyruvate dehydrogenase; SR, sarcoplasmic reticulum.

PCr is the transducer between ATP demand at the site of the contractile proteins and the ATP/ADP ratio in the mitochondrion. The regulation of biochemical pathways involved in oxidative phosphorylation are complex but Tschakovsky and Hughson (1999) suggest that 3 different local factors might interact to determine muscle $\dot{V}O_2$ kinetics.

1) Activation of mitochondrial enzymes and the role of Ca^{++}

Pyruvate dehydrogenase (PDH) determines the availability of substrates for the TCA cycle and subsequently the electron transport chain. Increases in mitochondrial Ca^{++} concentration converts PDH to its active, dephosphorylated form and increases the activity of some of the TCA enzymes. A number of studies suggest that under these circumstances of increased enzyme activity, increasing the supply of substrate increases oxidative phosphorylation.

2) Control of ETC flux rate by the ratios of $[ATP]/[ADP] \cdot [Pi]$ concentrations and $[NAD^+]/[NADH]$ concentrations.

When mitochondrial PO_2 is adequate, the two factors determining oxidative phosphorylation are the redox potential ($[NAD^+]/[NADH]$ concentration ratio) and the phosphorylation potential ($[ATP]/[ADP] \cdot [Pi]$ concentrations). It is the latter that is the main controller and the former that is the modulator of oxidative phosphorylation.

The limitation of oxidative phosphorylation and therefore $\dot{V}O_2$ kinetics by either of these two mechanisms has been termed intrinsic metabolic inertia (Tschakovsky and Hughson 1999)

which means that the rate of adaptation is determined solely by metabolic controllers and /or mitochondrial activation. It implies that mitochondrial PO_2 is adequate.

3) Modulation of mitochondrial respiratory rate by mitochondrial PO_2 -

Oxygen supply to the mitochondria, and therefore mitochondrial PO_2 might become limiting when capillary PO_2 falls to 15 - 20 mmHg (Whittenberg 1989) because of the physical factors limiting oxygen diffusion. Between a capillary PO_2 of 20 - 30 mmHg, although mitochondrial PO_2 falls into a region where it can modulate oxidative phosphorylation, this is compensated for by changes in the redox and phosphorylation potentials and ATP production is maintained at the required rate. If, during a step change in work rate, mitochondrial PO_2 remains adequate then $\dot{V}O_2$ kinetics are controlled by intrinsic metabolic inertia whereas if mitochondrial PO_2 is not adequate then $\dot{V}O_2$ kinetics are controlled by the inertia of the oxygen supply systems to the mitochondria i.e. extrinsic mechanisms (Tschakovsky and Hughson 1999).

Studies which have attempted to determine the effect of manipulating oxygen delivery on $\dot{V}O_2$ kinetics (Hughson and Kowalchuk 1995, Kowalchuk and Hughson 1990, Smyth *et al.* 1984) have not shown a consistent effect.

Another approach has been to compare the $\dot{V}O_2$ response characteristics with those of heart rate (Hughson and Morrissey 1983, Linnarsson 1974) cardiac output (Cerretelli *et al.* 1966, De Cort *et al.* 1991, Yoshida and Whipp 1994) and blood flow kinetics (Erikson *et al.* 1990, Grassi *et al.* 1996, Walloe and Wesche 1988). Although these studies suggest that neither cardiac function nor muscle blood flow limit $\dot{V}O_2$ kinetics, until it is possible to measure the

blood flow distribution to the active muscle fibres, the role of blood flow limiting $\dot{V}O_2$ kinetics will remain controversial.

Endurance training results in improvements in cardiovascular function and in muscle oxidative capacity (Ekblom *et al.* 1968, Saltin *et al.* 1976, Spina *et al.* 1992, Coggan *et al.* 1993, and Phillips *et al.* 1995a). Since these extrinsic and intrinsic factors are implicated in the control of $\dot{V}O_2$ kinetics, it would be expected that improvements in oxidative enzyme potential and blood flow redistribution would result in faster $\dot{V}O_2$ kinetics in elite endurance runners. Endurance trained athletes have also been demonstrated to attain a higher maximum cardiac output (Torok *et al.* 1995) and greater capillary to fibre ratio (Anderson and Henriksson 1977, Bergh *et al.* 1978) than sprinters. The greater capillary density of the endurance athletes is facilitated by a high proportion of type I muscle fibres which are known to have a greater capillary to fibre ratio than type II muscle fibres. It is possible that through endurance training, increased blood flow to the exercising muscle stimulates capillarization and thus facilitates improved oxidative potential. Growth in the capillary to fibre ratio has been shown to occur rapidly as a consequence of endurance training in men (Anderson and Henriksson 1977) and via a change of stimulation frequency in the fast twitch muscle of rabbit (Brown *et al.* 1973, Hansen-Smith *et al.* 1996, Skorjanc *et al.* 1998). Significantly increased capillary density has been demonstrated as early as 2 days in the fast twitch muscle of rabbit (Skorjanc *et al.* 1998), however, further studies of short term capillary growth in men are needed to confirm this observation.

Endurance training programmes have all resulted in faster $\dot{V}O_2$ kinetics (Hagberg *et al.* 1980, Berry and Moritani 1985, Yoshida *et al.* 1992, Babcock *et al.* 1994, and Phillips *et al.*

1995a). Studies involving younger subjects have shown changes in $\dot{V}O_2$ kinetics of between 6% (Berry and Moritani 1985) and 26% (Phillips *et al.* 1995a) after training. Greater improvements have been shown in much older subjects (Babcock *et al.* 1994) and in studies where step changes in work rate might have introduced a slow component (Hagberg *et al.* 1980, and Yoshida *et al.* 1992) into the resultant $\dot{V}O_2$ kinetics.

In contrast, a combination of sprint training and weight training regimes on the $\dot{V}O_2$ kinetics of football players (Fukouka *et al.* 1997) resulted in unchanged $\dot{V}O_2$ kinetics even though both $\dot{V}O_2$ max and ventilatory threshold were improved over the 9 month period.

It is likely therefore that given the different physiological and biochemical profiles of sprint and endurance runners and the contrasting effects of different training programmes, that tests of phase II $\dot{V}O_2$ kinetics would differentiate between the two groups of athletes.

1.3.5 Factors limiting $\dot{V}CO_2$ kinetics

Although it is known that carbon dioxide output ($\dot{V}CO_2$) kinetics differentiate fit subjects from less fit subjects (Zhang *et al.* 1991) it is an area that has not been extensively investigated. This could be attributed to the role of $\dot{V}CO_2$ as a waste product of energy metabolism and not as a source of energy provision. Nevertheless, both $\dot{V}O_2$ and $\dot{V}CO_2$ display very similar time courses at moderate exercise intensities and both potentially describe elements of aerobic fitness.

As described previously in section 1.3.1, $\dot{V}CO_2$ and $\dot{V}O_2$ kinetics follow distinct time phases, although $\dot{V}CO_2$ kinetics are known to be slower than $\dot{V}O_2$ kinetics (Whipp *et al.* 1982). While

the phase II for $\dot{V}CO_2$ kinetics displays single exponential kinetics, the $\dot{V}CO_2$ time constant is considerably longer than that of $\dot{V}O_2$ (~50s and ~30s respectively) (Hughson and Morrissey 1982, Linnarsson 1974, Whipp *et al.* 1982). In addition, the time taken for $\dot{V}CO_2$ to reach steady state is ~4 min compared to $\dot{V}O_2$ at ~3min. The reasons for the longer response in $\dot{V}CO_2$ could be attributed to a number of processes between initial O_2 uptake and CO_2 output but are most likely caused by i) CO_2 storage and ii) CO_2 produced from buffering mechanisms.

CO_2 storage

At the onset of exercise, it is known that CO_2 produced from muscle metabolism increases relatively slowly compared to O_2 . This is attributable to the relatively high solubility of CO_2 in tissues compared to O_2 . Storage of CO_2 increases during the first minute of exercise (Zhang *et al.* 1991) and the presence of sizeable intervening stores of CO_2 stores serves to dissociate $\dot{V}CO_2$ from CO_2 production (Whipp and Ward 1990). While a proportion of CO_2 production is being stored, the $\dot{V}CO_2$ kinetics measured at the mouth will not directly reflect CO_2 kinetics at the muscle. The effect of CO_2 storage on the $\dot{V}CO_2$ response to square wave exercise is shown in Figure 1.10:

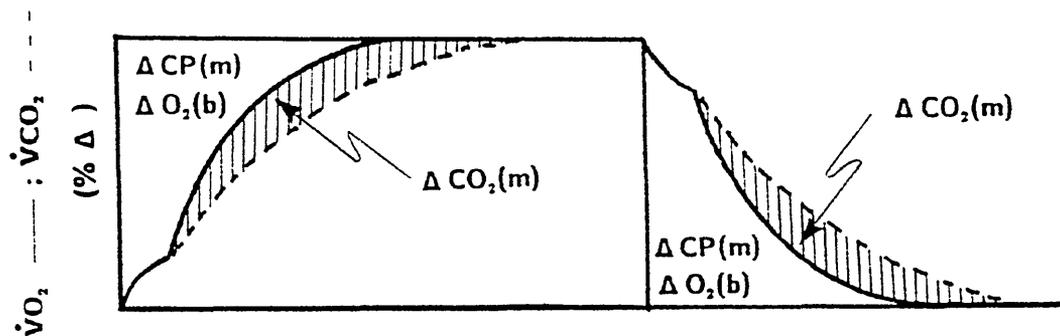


Figure 1.10. Schematised time course of pulmonary gas exchange ($\dot{V}O_2$, $\dot{V}CO_2$) response to, and recovery from square wave exercise (Taken from Whipp and Ward 1990). The time course of muscle O_2 consumption and CO_2 production are assumed to be similar and, allowing for vascular transit delays, are well approximated by the $\dot{V}O_2$ time course (solid line). However, owing to the significant change in muscle CO_2 storage capacity ($\Delta CO_2(m)$), the $\dot{V}CO_2$ response (dashed line) is appreciably slower than the $\dot{V}O_2$ response.

Several investigators have examined the effect of CO_2 storage on gas exchange during exercise (Ward *et al.* 1983, Hughson and Inman 1985, Jones and Jurkowski 1979). In the study by Ward *et al.* (1983) body CO_2 stores were depleted prior to exercise by controlled volitional hyperventilation. Following volitional hyperventilation, $\dot{V}CO_2$ dynamics were observed to be slower when compared with control exercise values. Jones and Jurkowski (1979) found that CO_2 storage studied by hyperventilation in the steady state of exercise decreased at higher work rates. These studies indicate that CO_2 storage might slow $\dot{V}CO_2$ kinetics where subjects have low body CO_2 stores prior to testing. A study by Hughson and Inman (1985) supports the contention that greater CO_2 is stored at lower exercise intensities. In their study CO_2 storage was shown to decrease with metabolic acidosis. This finding offers a possible explanation of the faster $\dot{V}CO_2$ kinetics commonly observed at higher work rates.

A number of studies (Hughson and Morrissey 1982, 1983, Whipp *et al.* 1982) have shown that the kinetics of $\dot{V}CO_2$ and $\dot{V}O_2$ are dependent on the exercise transitions studied. From rest to work, the time constants for $\dot{V}O_2$ are faster than those for $\dot{V}CO_2$, however, when exercise proceeds from light to moderate or heavy work rates, the $\dot{V}O_2$ kinetics become slower while the $\dot{V}CO_2$ kinetics are either unchanged or become faster (Hughson and Morrissey 1982, 1983). Thus the pattern changes such that less CO_2 must be stored at higher work rates. During high intensity work, the body's capacity to store CO_2 decreases (Jones and Jurkowski 1979) and subsequently a progressive increase is evident in pulmonary CO_2 output.

Despite the complications associated with the increased storage of CO_2 , there is a close relationship between $\dot{V}CO_2$ and $\dot{V}O_2$ during moderate exercise. At moderate exercise intensities, both $\dot{V}CO_2$ and $\dot{V}O_2$ display single exponential kinetics (Whipp and Ward 1990) indicating that either $\dot{V}CO_2$ or $\dot{V}O_2$ kinetics provide a description of gas exchange kinetics.

CO_2 produced from buffering lactic acid

$\dot{V}O_2$ kinetics are known to display a slow component rise above the lactate threshold with a consequent slowing of $\dot{V}O_2$ kinetics at high work rates compared to low level intensities (Whipp and Ward 1990, Zhang 1991). $\dot{V}CO_2$ kinetics do not slow at high work rates as a supplementary supply of CO_2 is produced in the buffering of lactic acid (Zhang *et al.* 1991). The presence of elevated lactate levels during aerobic exercise would be indicative of inappropriately high work rate intensities as the buffering system would then have come into effect.

Bicarbonate ions (HCO_3^-) acts to free oxygen for tissue respiration and aids the removal of metabolites such as lactic acid by buffering H^+ (Beaver *et al.* 1986). Therefore, at work rates above LT, the HCO_3^- buffering system of H^+ will remove the H^+ associated with lactate (although blood lactate will continue to rise) and reduce the instability of the pH of the muscle and enable exercise to be continued for a prolonged period. Consequently, this effect should not be observed in an appropriately designed aerobic test.

In summary, although $\dot{V}\text{CO}_2$ display similar characteristics to $\dot{V}\text{O}_2$ during moderate work, $\dot{V}\text{O}_2$ has more commonly been utilised by investigators as it is considered the gas of energy provision. $\dot{V}\text{CO}_2$ kinetics have, however, been investigated at higher work rates (Casaburi *et al.* 1989, Zhang *et al.* 1991) whereby differences between $\dot{V}\text{O}_2$ and $\dot{V}\text{CO}_2$ kinetics might provide useful mechanistic information.

1.3.6 Gas exchange kinetics as a test to differentiate athletic groups

An individual's dynamic O_2 response to exercise depends on various factors such as work rate (Jones *et al.* 1968, Casaburi *et al.* 1989), state of fitness (Hagberg *et al.* 1978, Hickson *et al.* 1978), age (Chillibeck *et al.* 1996) and specificity of exercise (Hamar and Komadel 1984). An improved response would reflect a more effective aerobic system with less reliance on anaerobic energy contribution at the onset of exercise. In terms of athletic performance, this would have the beneficial effect of enabling athletes to have more energy available for a competitive event and would also be of benefit to athletes requiring a change of pace during a race, as they would avoid incurring premature fatigue.

Although gas exchange kinetics describe the rate at which the aerobic energy systems adapt to meet increases in metabolic requirements during exercise (Essfeld *et al.* 1987), there are only a limited number of studies where they have been applied to sporting situations (Fukuoka *et al.* 1995 and 1997, Faina *et al.* 1998). This is surprising since pulmonary $\dot{V}O_2$ kinetics provide a useful non invasive estimate of muscle $\dot{V}O_2$ kinetics (Essfeld *et al.* 1991, Hoffmann *et al.* 1992).

Ozyener *et al.* (1996) measured $\dot{V}O_2$ kinetics at high intensity work rates in sprint and endurance trained humans. That workloads utilised were above the lactate threshold in order to investigate the slow phase of O_2 uptake, and as such are not directly comparable with this study. Nevertheless, the subjects were chosen for that study on a similar basis to this study in so far as their athletic performance characteristics were consistent with predominantly fast or slow twitch fibre types. Accordingly, a physiological difference in O_2 uptake would be expected to exist between groups.

A study by Fukuoka *et al.* (1995) utilised a sinusoidal protocol with the higher work set at 60% of $\dot{V}O_2$ max. That study investigated the $\dot{V}O_2$ dynamics of American football players (AFPs), distance runners (DRs) and untrained men (UTM). The AFPs had trained in a sprint-type fashion and thus comparison with the DRs bears similarities with this study. In their study, the AFPs and UTM displayed significantly slower $\dot{V}O_2$ dynamics than the DRs indicating that athletes participating in different sports have characteristic dynamic $\dot{V}O_2$ responses. The protocol used in that study elicited elevated plasma lactate levels in both the AFPs (3.3 mmol L^{-1}) and UTM (3.9 mmol L^{-1}) which might have slowed their $\dot{V}O_2$ responses and thus effected the linearity of the testing system.

A further study conducted by Fukuoka *et al.* (1997) investigated the effect of a combination of sprint and strength training on $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics on six footballers in response to a sinusoidal protocol. That study utilised a sinusoidal exercise protocol, ranging from 30 watts to 60% of the subject's $\dot{V}O_2$ max. It was found that $\dot{V}O_2$ kinetics were unaffected by sprint type training over the nine month period, while $\dot{V}CO_2$ kinetics were significantly slowed after six and nine months. This finding suggests that $\dot{V}CO_2$ kinetics could prove a more sensitive measure of training and/or fitness levels than $\dot{V}O_2$ kinetics.

A further sport specific study examining $\dot{V}O_2$ kinetics was conducted by Faina *et al.* (1998) who compared a laboratory measure of $\dot{V}O_2$ kinetics on a Concept II rowing machine with an actual rowing field test. No significant differences were found between laboratory and field test in the study, although it should be noted that few subjects were used (6) and thus further investigation would be required to verify this result.

$\dot{V}O_2$ and $\dot{V}CO_2$ kinetics (Essfeld *et al.* 1987, Zhang *et al.* 1991) have been shown to be faster in individuals possessing higher levels of aerobic fitness and when applied to monitoring an athlete's aerobic fitness over a training period, this could provide a useful guide to the athlete's improvement (Essfeld *et al.* 1987, Hagberg *et al.* 1980, Hickson *et al.* 1978, Powers *et al.* 1985, Zhang *et al.* 1991).

A possible explanation for the lack of practical application of gas exchange kinetics as a sports test could lie in the methodology of the technique. As previously indicated, several repetitions of the step protocol are required to gain adequate data and are thus unsuitable as a quick means of assessing aerobic fitness in a single laboratory visit, however, the

PRBS technique has a potential advantage in that it enables the investigator to gain sufficient test data for analysis in a single test. Investigators have employed a number of PRBS cycles of varying length (Table 1.6) the appropriateness of which depends on the experimental aims of the investigator.

1.4 Experimental aims

In this study, a prominent consideration was to achieve credible results with the minimum imposition to the elite athletes training routines and limited available time. Therefore, a PRBS cycle of 300s was selected and the athletes all performed three repetitions of this in order to assist the ensemble averaging process which is known to reduce non physiological breath-by-breath noise.

The style of PRBS protocol chosen for exercise testing depends on the time phase of gas exchange kinetics of interest to the investigator. As gas exchange is known to increase in distinct time phases, the protocol can be largely manipulated to concentrate on the particular time phase of interest. As the phase I (cardiodynamic) response represents the time before blood from the exercising muscles has appeared at the lungs, investigators have employed protocols to minimise its effect (Hughson and Morrissey 1983), such as the PRBS exercise test (Hughson *et al.* 1990a). This can be achieved by using a PRBS test as the PRBS utilises work to work transitions which minimise the abrupt transition from rest to work employed in many single step protocols (Hughson and Morrissey 1982, Hughson *et al.* 1990b). In addition, a PRBS with a minimum base unit of 20s duration would further safeguard from any of the units exclusively measuring phase I.

Few studies (Fukuoka *et al.* 1995 and 1997, Faina *et al.* 1998) of gas exchange kinetics have applied the technique in a sporting context (Jakeman 1994). It is likely that a measure of $\dot{V}O_2$ kinetics will differentiate between sporting groups due to differences in oxidative processes. In addition, the study of Fukuoka *et al.* (1997) indicates that measurement of $\dot{V}CO_2$ kinetics will also differentiate between the two groups. This might result in the development of a convenient and low intensity test capable of distinguishing between a wide range of sport performers.

The aims of the study are to:

- assess the test/retest reliability of the chosen PRBS exercise protocol
- use the PRBS exercise test to distinguish between athletic groups of elite sprinters and elite endurance runners

2.0 Materials and Methods

2.1 Protocol design.

The PRBS protocol chosen for this study was based on the 300s model of Hoffmann *et al.* (1992), (1994a). The base unit of the PRBS is 20s with work rates alternating between 25 and 85 W. The time sequence and work rate changes for a single 300s PRBS cycle are shown in Table 2.1:

Table 2.1. The time sequence and work rates for one 300s PRBS cycle.

Time (s)	0-80	80-100	100-120	120-140	140-180	180-220	220-240	240-300
Work rate (W)	85	25	85	25	85	25	85	25

Three consecutive PRBS cycles were performed by all subjects and were preceded by a warm up. The 300s PRBS cycle is shown in Figure 2.1:

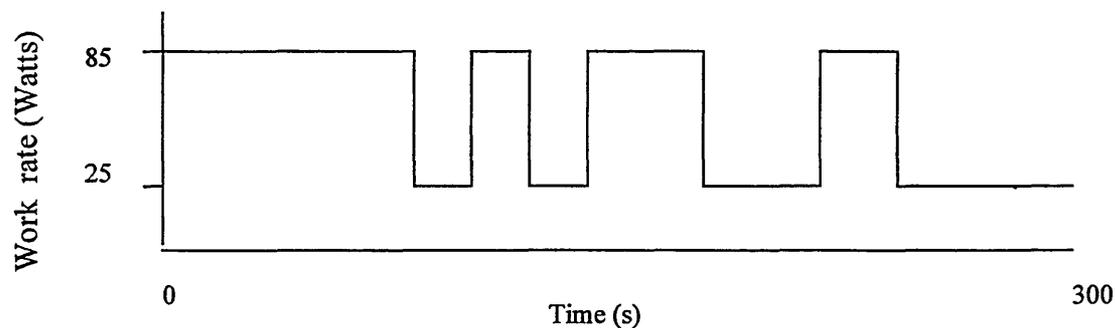


Figure 2.1. The 300s pseudo random binary sequence (PRBS) exercise protocol. Three complete cycles of the PRBS are performed and are preceded by constant load work at 25 W and followed by constant load work at 85 W. The 300s sequence is derived from 15 units of 20s.

2.2 Work rate control system.

All exercise tests were performed on an electrically braked cycle ergometer (550 ERG, Bosch, Berlin Germany) at a constant pedalling frequency of 1 Hz. By linking the ergometer to a work rate control system, the alternating exercise intensities were automatically adjusted by computer (First Breath Software version 2.0 1992). The exercise cycle was calibrated (Claxton 1999) prior to testing and checked following testing in order to ensure that consistent power outputs were achieved for the procedure. The computer control system consequently accounted for small differences and thus to achieve an actual power of 25W it was programmed to generate 25.4W and for 85W it was programmed to generate 89.5W.

2.3 Subjects

All subjects were asked to complete a medical questionnaire prior to any form of testing (Appendix 1). The subjects were also informed that they should refrain from heavy exercise, alcohol and caffeine in the 24 hours prior to testing.

2.4 Overall mean average cardiorespiratory responses to the PRBS test

Heart rate traces were taken for all subjects over the test duration. Heart rates were calculated at 5s intervals by Polar Sports Tester equipment over the PRBS exercise test. Overall average $\dot{V}O_2$ and Respiratory exchange ratio (RER) were calculated from the breath-by-breath data over the full duration of the exercise test.

2.5 Measurement of $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics.

$\dot{V}O_2$ and $\dot{V}CO_2$ were measured breath-by-breath using a computer system (Marquette Electronics Inc, Milwaukee, WI) incorporating a respiratory mass spectrometer (Marquette MGA 1100) and a volume turbine (Alpha Technologies YMM-110). The calibration of the mass spectrometer was performed immediately before each test with standard calibration gases and the system was further checked following testing. A 3 litre syringe was used to calibrate the volume turbine using flow rates similar to subject ventilation. The required tolerance of repeated calibration was $\pm 1\%$. Gas volumes were corrected to standard temperature pressure dry (STPD). An algorithm (Beaver *et al.* 1981) was used to calculate gas exchange at the alveolar level with corrections for changes in lung volume and lung gas composition. In order to reduce the influence of non physiological noise, a computer software (First Breath Software version 2.0 1992) filter was used to eliminate outlying data. A typical oxygen uptake response is shown in Figure 2.2.

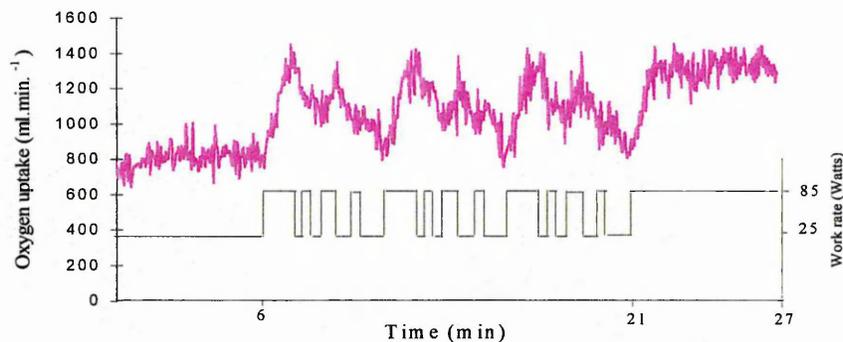


Figure 2.2. Breath-by-breath oxygen uptake ($\dot{V}O_2$) and work rate (25 W and 85 W during three consecutive 300s pseudo random binary sequences (PRBS) for a single elite endurance runner. For the study of elite runners, the PRBS cycles are preceded by six minutes at 25W and followed by six minutes at 85W for the calculation of static gain. In the limits of

agreement study, three minutes constant load work were used at 25W. The data has been linearly interpolated and filtered for outlying breaths.

2.6 Data analysis of $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics.

All breath-by-breath data from three repetitions of the 300s PRBS were linearly interpolated at 1s intervals, superimposed and ensemble averaged to yield a single data set per subject. Data gained from the PRBS exercise test was analysed within the frequency domain, Fourier analysis was performed on the PRBS input signal, in addition to the $\dot{V}O_2$ and $\dot{V}CO_2$ output signals to separate them into individual harmonic components. The relationship between the input and output signals was then compared according to the parameters of amplitude ratio and phase shift. The relationship between input and output signal is obtained by dividing the amplitude of the output by that of the test input to yield an amplitude ratio and by subtracting the phase angle of the input from that of the output to yield a phase shift parameter (Hughson *et al.* 1990a, Jenkins and Watts 1968). The frequency responses can then be represented by amplitude ratio ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) and phase shift (degrees) components for the relationship of input/output (i.e work rate/gas exchange).

It has been shown (Hoffmann *et al.* 1992 and 1994a & b) that the system controlling gas exchange kinetics during PRBS testing is at least quasi-linear over a wide range of frequencies at least down to periods of 100s. Consequently, Fourier transform was computed for frequencies 3.3, 6.7 and 10 mHz.

3.0 Reliability study

3.1 Introduction

Reliability is the ability of a measurement system or procedure to display the same results on a number of occasions (Atkinson and Nevill 1998). Although, several measurements of the same quantity on the same subject will not in general be the same. This might be due to the natural variation of the subject, variation in the measurement process, or both. Repeated measurements on the same subject will vary around the true value because of measurement error. Measurement error can be defined as the true value minus the measured value (Bland and Altman 1986). This is of particular importance when a measurement procedure involves a biological system, for example a human subject. An assessment of measurement error can also aid in the quantification of the variability inherent in any biological system (biological error), the error due to the apparatus (systemic error) and inter-operator error. Both systemic and inter-operator error can be controlled to some extent by monitoring the calibration of equipment and reducing the number of operators involved in data collection.

Measurement error can be expressed in a number of forms. The mean of the replicate test results accompanied by the standard deviation of the variance within these results, determined by means of ANOVA, can be used to express measurement error in its simplest form, i.e. $\text{mean} \pm \text{SD}_{\text{var within}}$. This approach can be extended to calculate the 95% confidence interval for the replicate results, in the form of $\text{mean} \pm 2\text{SD}_{\text{var within}}$. This confidence interval provides an indication of the range within which the difference between test results will lie with a probability of 0.95.

Bland and Altman (1986) have advocated the use of the 95% limits of agreement method to assess the extent of the repeatability between the test and retest results obtained during the performance of identical protocols. Atkinson and Nevill (1998) suggest that for a new subject from the studied population, it would be expected (with 95% probability) that the difference between any two tests should lie within the limits of agreement. This method has now been widely applied to examine the reproducibility of biological systems (Lee 1992, Shaw *et al.* 1994, Liehr *et al.* 1995).

It has been suggested (Atkinson and Nevill 1998) that to increase the practical use of the limits of agreement, a systematic bias should be calculated. A systematic bias refers to a general trend for measurements to be different in a particular direction between repeated tests as, for example, if a learning effect was present. Bias might also be due to insufficient recovery between tests which would show a worse score than in a previous test.

Currently, there is limited knowledge concerning the reproducibility of replicate measures of gas exchange kinetics. Berry and Moritani (1985) have reported the reproducibility of replicate measures of $\dot{V}O_2$ kinetics made during two identical square wave work rate forcings. They described the results of their study as satisfactory evidence for the reproducibility of $\dot{V}O_2$ kinetics. Claxton *et al.* (1996) examined the reliability of a 63 unit PRBS exercise test with 5s work rate changes between 25 and 105 W to measure $\dot{V}O_2$ kinetics. An assessment of intra-subject variability for amplitude ratio and phase shift was made using the analysis of variance technique, and limits were calculated accordingly. That study reported wide limits of agreement at all frequencies of both amplitude ratio and phase shift although there was no significant difference between the test and retest of $\dot{V}O_2$ kinetics.

In a review of literature no evidence could be found of any study which described the intra-subject variability of the test/retest results determined during a PRBS exercise test with 20s work rate changes between 25 and 85 W.

3.2 Methodology

A study was conducted to evaluate the reliability of $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics resulting from PRBS testing.

3.2.1 Subjects.

Ten healthy male subjects agreed to participate in this study [age: 25 years (± 3.89), body mass: 79.88 kg (± 13.27), stature: 183.7 cm (± 4.5)]. All subjects participated in regular physical activity and provided their informed consent (Appendix 1) prior to any testing in accordance with the Research Degree Committee regulations of the university. The test and retests were carried out on the same day and the test was repeated after 30 min for each subject.

3.2.2 Exercise protocol.

$\dot{V}O_2$ and $\dot{V}CO_2$ kinetics were measured in response to a PRBS exercise test. Three successive repetitions of a 300s PRBS pattern (Hoffmann *et al.* 1992, 1994) were performed and the protocol started at the lower intensity of 25W which acted as a warm up.

3.2.3 Data analysis

Firstly, the $\dot{V}O_2$ and $\dot{V}CO_2$ breath by breath data for the 3 PRBS cycles was 'smoothed' by a computer software filter (First Breath Software version 2.0 1992) which eliminated outlying data. The data from the 3 PRBS cycles was then linearly interpolated at 1s intervals and averaged to yield a single 300s data set per subject. Fourier transform was then performed on the input and output signals for comparison at individual harmonic frequencies.

The PRBS exercise test results were analysed within the frequency domain by Fourier techniques. The frequencies 3.3, 6.7 and 10 mHz (up to 100s from the fundamental frequency) have been included for analysis (Essfeld *et al.* 1991). Beyond this point, it is unlikely that the subject response will be discernible from non physiological breath-by-breath noise (Essfeld *et al.* 1987).

3.2.4 Statistical analysis

A two-way analysis of variance was used for the assessment of intra-subject variability of the PRBS exercise test.

Limits of agreement (95%) have been applied to the 300s PRBS exercise test. Individual differences between test and retests have been plotted against the mean difference (bias) for the two tests of both $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics (Figures 3.1.-3.4.). Upper and lower limits of agreement of ± 1.96 SD were set, within which 95% of the differences between tests should occur.

The 95% limits of agreement have additionally been represented as a measurement error percentage (Bland 1995) by applying the calculation: $100 \times (1.96 \times \text{SD diff} / \text{grand mean})$, where 'SD diff' represents standard deviation of the differences between tests and retests and 'grand mean' represents $(\text{mean of test 1} + \text{mean of test 2})/2$. This method enables the researcher to quickly assess measurement error between comparable tests by a percentage (%) in either a positive or negative way. This is of particular use in the comparison of the limits of agreement for $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics on the same set of data.

Systematic Bias was assessed as a percentage from: the average difference between tests / the mean of Test 1 and Test 2 $\times 100$.

A two way analysis of variance (ANOVA) with repeated measures was used to investigate whether differences existed between Test 1 and Test 2.

3.3 Results

A two way analysis of variance (ANOVA) with repeated measures did not reveal significant differences between Test 1 and Test 2 of either $\dot{V}O_2$ or $\dot{V}CO_2$ kinetics. The limits of agreement results for $\dot{V}O_2$ kinetics are shown in Table 3.1. and $\dot{V}CO_2$ kinetics in Table 3.2.

Table 3.1. The mean values, systematic bias, measurement error and limits of agreement for amplitude ratio and phase shift of $\dot{V}O_2$ kinetics.

	Frequency (mHz)		
	3.3	6.7	10
Amplitude: mean values ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) - Test 1	7.57 (± 0.89)	5.83 (± 0.77)	4.17 (± 0.65)
Amplitude: mean values ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) - Test 2	7.68 (± 1.04)	5.79 (± 0.87)	4.2 (± 1.06)
Amplitude: systematic bias (Percentage)	-1%	0%	0%
95% limits of agreement ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)	-1.57 to 1.79	-2.06 to 1.99	-1.47 to 1.52
Measurement error (Percentage)	$\pm 22\%$	$\pm 34\%$	$\pm 35\%$
Phase shift: mean values (degrees) - Test 1	38.99 (± 3.75)	67.54 (± 5.81)	94.31 (± 15.46)
Phase shift: mean values (degrees) - Test 2	37.31 (± 6.44)	70.5 (± 7.96)	93.11 (± 9.56)
Phase shift: systematic bias (Percentage)	-4%	4%	-1%
95% limits of agreement (degrees)	-13 to 9.64	-10 to 15.93	-26.69 to 24.29
Measurement error (Percentage)	$\pm 29\%$	$\pm 18\%$	$\pm 27\%$

Table 3.2. The mean values, systematic bias, measurement error and limits of agreement for amplitude ratio and phase shift of $\dot{V}CO_2$ kinetics.

	Frequency (mHz)		
	3.3	6.7	10
Amplitude: mean values (ml·min ⁻¹ ·W ⁻¹) - Test 1	5.34 (±1.6)	4.05 (±0.93)	2.45 (±1.52)
Amplitude: mean values (ml·min ⁻¹ ·W ⁻¹) - Test 2	5.21(±1.88)	3.91 (±1.18)	2.41 (±1.13)
Amplitude: systematic bias (Percentage)	-2 %	-4 %	-2 %
95% limits of agreement (ml·min ⁻¹ ·W ⁻¹)	-3.01 to 2.85	-2.55 to 2.25	-2.72 to 2.65
Measurement error (Percentage)	±57 %	±59 %	±108 %
Phase shift: mean values (degrees) - Test 1	-50.48 (±11.82)	-81.28 (±13.39)	116.13 (±61.45)
Phase shift: mean values (degrees) - Test 2	-48.48 (±20.66)	-85.12 (±22.99)	128.67 (±36.68)
Phase shift: systematic bias (Percentage)	-4 %	4 %	10 %
95% limits of agreement (degrees)	-35.45 to 31.38	-29.48 to 37.16	-95.77 to 120.86
Measurement error (Percentage)	±66 %	±39 %	±87 %

The systematic bias in Table 3.1. ranges from -4 to +4% for the amplitude ratios and phase shift for $\dot{V}O_2$ kinetics. The systematic bias, shown in Table 3.2. for $\dot{V}CO_2$ kinetics ranges from -4 to its highest level of 10% frequency 10mHz for the phase shift. In the $\dot{V}CO_2$ kinetics, measurement error is also at its highest at frequency 10 mHz of both the amplitude ratio and phase shift (108% and 87% respectively). It can, however, be seen in Table 3.1. that the measurement error of $\dot{V}O_2$ kinetics is of a lower level and not consistently at its highest at the higher frequency of 10mHz.

The 95% confidence levels for the amplitude ratio and phase shift of $\dot{V}O_2$ are shown graphically in Figures 3.1. and 3.2. respectively. The $\dot{V}CO_2$ kinetics results are shown in Figures 3.3. and 3.4.

Figure 3.1. The 95% limits of agreement and bias for the amplitude ratios of $\dot{V}O_2$ kinetics.

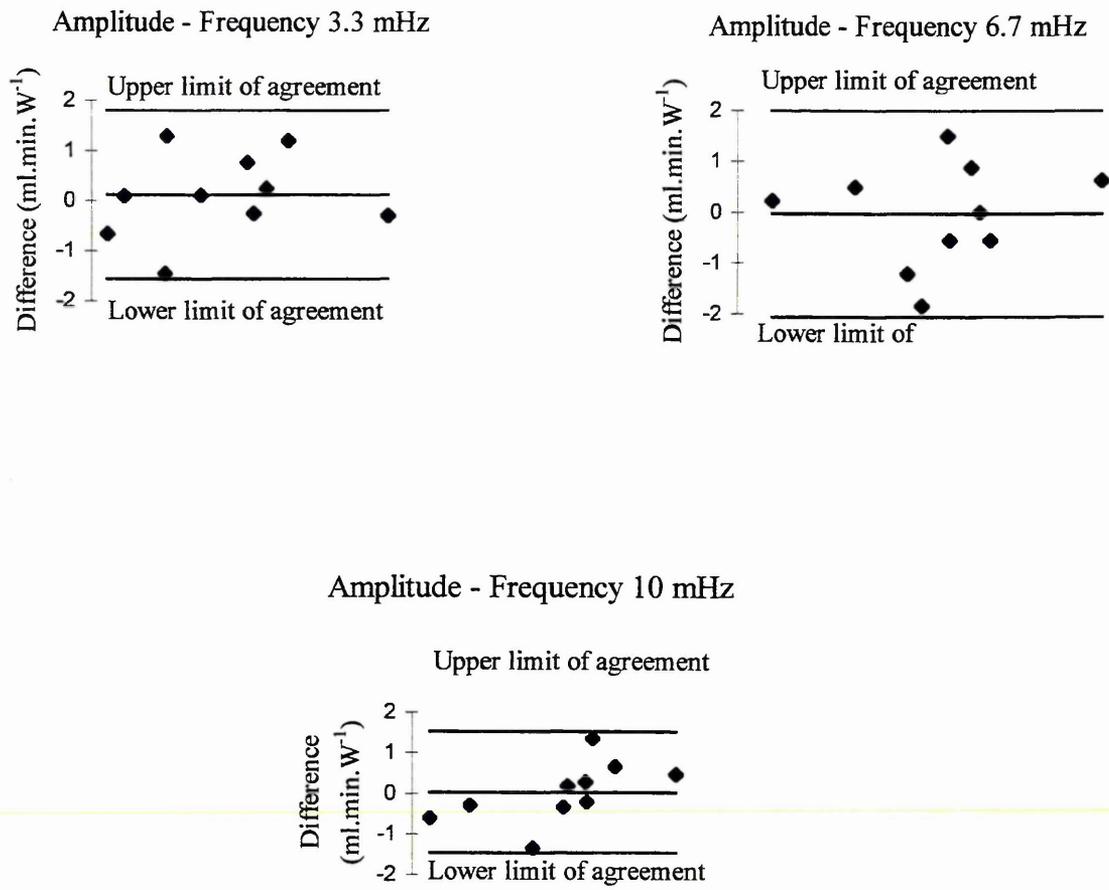
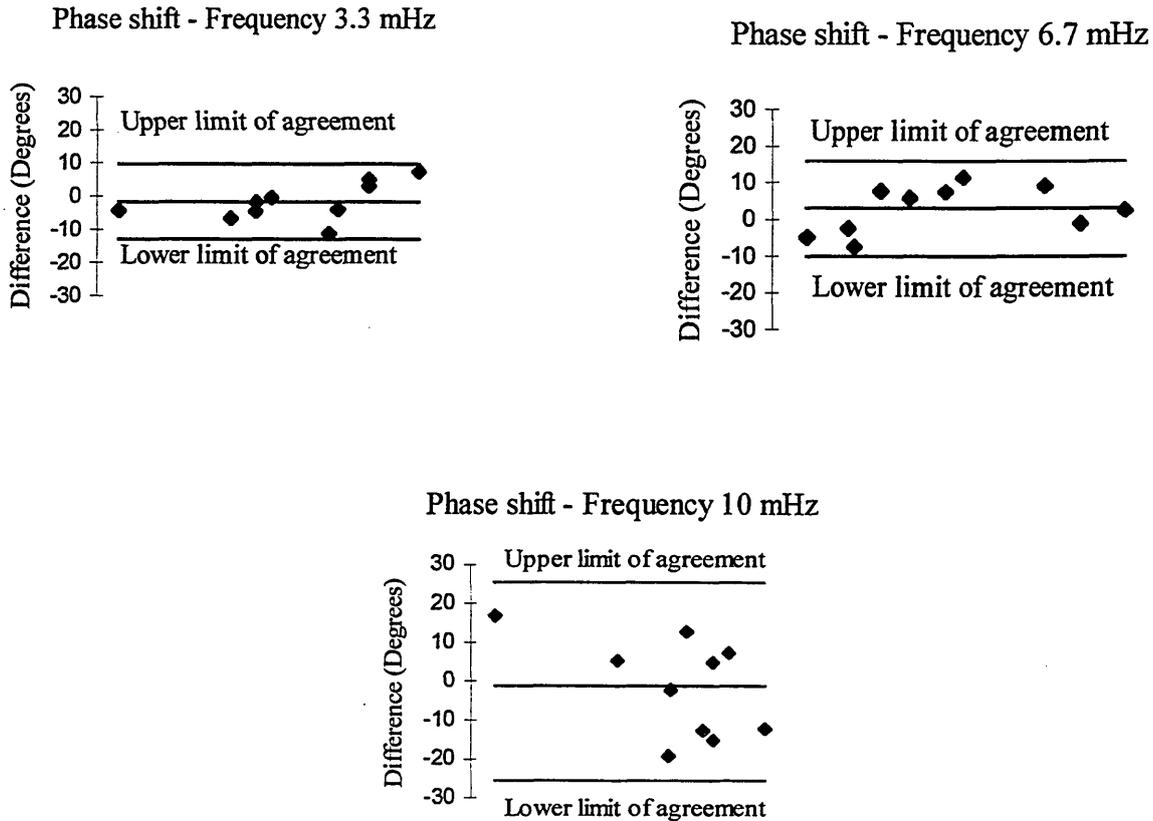


Figure 3.2. The 95% limits of agreement and bias for the phase shift parameters of $\dot{V}O_2$ kinetics.



From Figure 3.2 it is possible to see that wider 95% limits of agreement displayed at frequency 10mHz of the phase shift of $\dot{V}O_2$ kinetics (-26.69 to 24.29). Despite the wider limits of agreement at this frequency, the measurement error is lower (27%) than for frequencies 6.7 and 10mHz of the amplitude ratio of $\dot{V}O_2$ kinetics (34 and 35% respectively).

No trend for faster or slower $\dot{V}O_2$ kinetics was detected in the study of 95% limits of agreement.

Figure 3.3. The 95% limits of agreement for amplitude ratios of $\dot{V}CO_2$ kinetics.

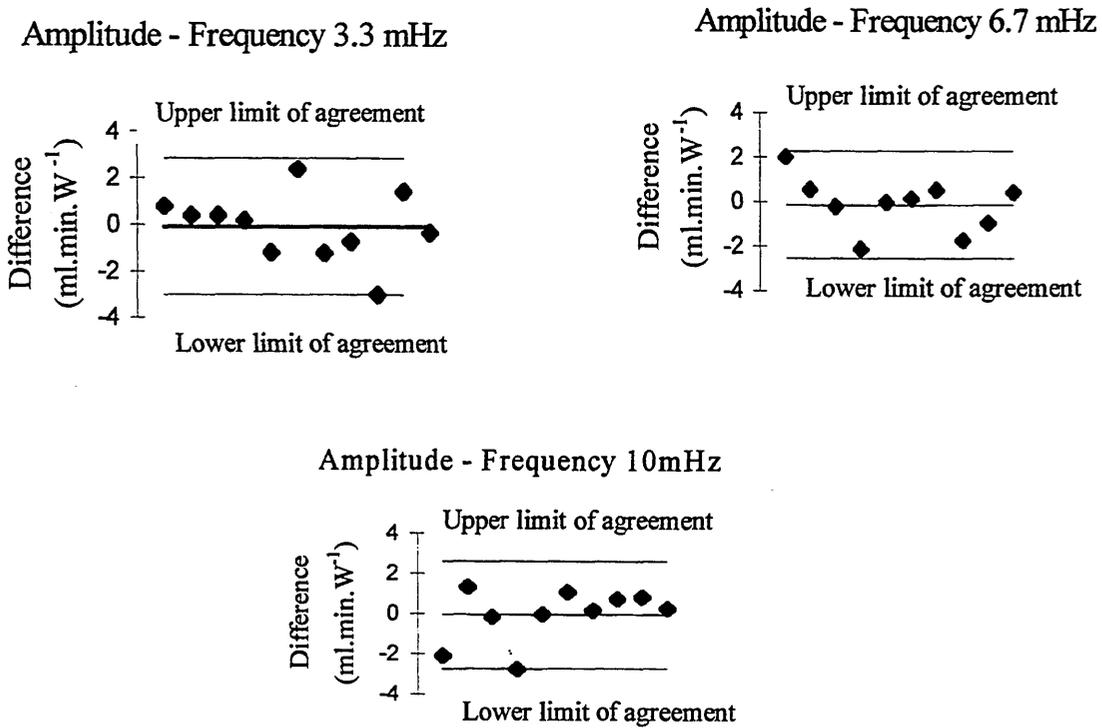
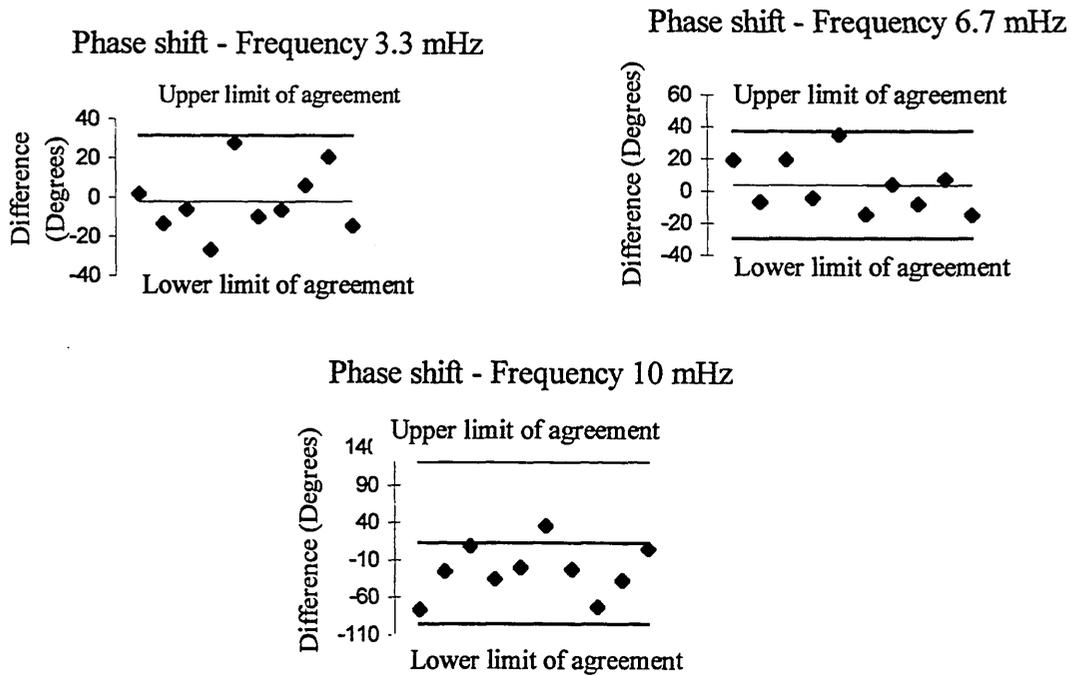


Figure 3.4. The 95% limits of agreement and for the phase shift of $\dot{V}CO_2$ kinetics.



The 95% limits of agreement for $\dot{V}CO_2$ kinetics are at their widest at the higher frequencies. This is, however, most likely a function of the increased magnitude of the measured value as measurement error (Tables 8 and 9) show a lower positive or negative percentage for $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics than at frequency 3.3 mHz. Further examination of both individual and mean differences did not identify any particular upward or downward trend, except at frequency 10 mHz of kinetics where the prevalence appears to be for longer phase shifts from test 1 to test 2 (116.13 ± 61.45 and 128.67 degrees ± 36.38).

3.4 Discussion

The results of a two way ANOVA analysis with repeated measures did not reveal significant differences between the test and retest of either $\dot{V}O_2$ or $\dot{V}CO_2$ kinetics. This supports the further development of the technique as a differential test of sports groups.

The results of the limits of agreement study indicate that physiological assessment by $\dot{V}O_2$ kinetics is likely to be more reliable than by $\dot{V}CO_2$ kinetics. This is evident from the measurement error values for $\dot{V}O_2$ kinetics which range from ± 18 - $\pm 35\%$, compared with $\dot{V}CO_2$ kinetics which range from ± 39 - $\pm 108\%$ of all parameters. The larger variability between the $\dot{V}CO_2$ kinetics tests suggests that this test is less likely to detect physiological differences between groups. It is further apparent that any difference between groups (i.e sprinters and endurance runners) would have to be of a disproportionately high level to prove statistically significant and thus, if used exclusively as an aerobic fitness indicator, $\dot{V}CO_2$ kinetics might

not reveal physiological characteristics that could be identified through a more accurate measurement tool.

The limits of agreement for $\dot{V}O_2$ kinetics appear tighter than for $\dot{V}CO_2$ kinetics, although similarly they appear widest at the highest frequencies of the phase shift, indicating that group comparison data would be unlikely to recognise physiological differences at those frequencies. However, the percentage measurement error indicates that the widest positive or negative difference between tests exists at frequencies 6.7 and 10 mHz of the amplitude ratio which is somewhat surprising given the smaller values of the amplitude ratio.

Since the variability between Test 1 and 2 of the $\dot{V}O_2$ kinetics is relatively high, caution should be taken in the assessment of individual results, however, group comparison data could reasonably be expected to attain statistical significance with sufficient subjects.

The systematic bias results of $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics (Tables 8 and 9) were negligible for all measured parameters, except at frequency 10 mHz of the phase shift for $\dot{V}CO_2$ kinetics. A bias would indicate that either a learning effect had occurred or that fatigue had influenced the results. Although not statistically significant, the predominance of a longer phase shift in test 2 at frequency 10mHz of $\dot{V}CO_2$ kinetics suggests that results for that parameter might not be reliable. As no trend for faster or slower $\dot{V}O_2$ or $\dot{V}CO_2$ kinetics was observed in the other parameters, confidence can be maintained in the test procedure. Therefore, it is concluded that no habituation or learning effect was associated with this PRBS exercise test protocol.

The relatively wide variability of the limits of agreement are largely consistent with other single session laboratory exercise tests which have often been reported to fluctuate (Hughson

and Inman 1986, Claxton *et al.* 1996). Laboratory test confidence would undoubtedly be enhanced by repeated testing as an averaged test response would minimise the effect of variations and further reduce the effect of non physiological breath-by-breath noise which is a feature in tests of this nature (Lamarra *et al.* 1987). The relatively high variability in the $\dot{V}CO_2$ kinetics results is probably due to changes in CO_2 storage due to hyperventilation at such relatively low work intensities. The purpose of this is to incur minimal imposition to the subjects while providing them with a useful description of their fitness. According to this criteria, the proposed PRBS exercise test appears to have potential as a practical exercise test for group comparison.

4.0 Gas exchange kinetics in elite runners

4.1 Introduction

In this study, the PRBS exercise test has been applied to elite sprinters and elite endurance runners. Elite sprinters and endurance runners were selected on the basis of their acknowledged (Chapter 1) physiological differences. It is anticipated that due to the differing energy requirements of sprint and endurance performance, the analysis of gas exchange kinetics in response to the PRBS exercise test will differentiate between these two athletic groups. This would identify a practical application for the test and would support the development of the test to further differentiate between other sporting groups.

Few studies have applied this technique in a sporting context, although, Fukuoka *et al.* (1995) demonstrated the $\dot{V}O_2$ kinetics of Distance Runners were significantly improved compared with anaerobically trained American Football Players. Other studies have identified that endurance training improves $\dot{V}O_2$ kinetics (Berry and Moritani 1985, Phillips *et al.* 1995a), however, one study (Fukuoka *et al.* 1997) has shown that sprint training does not effect $\dot{V}O_2$ kinetics. That study also showed that $\dot{V}CO_2$ kinetics were slowed as a consequence of sprint training which suggests that $\dot{V}CO_2$ kinetics might be a more sensitive indicator of training status and/or fitness levels than $\dot{V}O_2$ kinetics.

4.2 Methodology

4.2.1 Subjects.

A group of elite male endurance trained (ET) (n=12) and elite male sprint trained (ST) (n=12) runners were selected for this study based on their proven athletic ability at elite status. The physical characteristics of the subjects are shown in Table 4.1.

Table 4.1. Mean age, stature and mass (\pm SD) of sprint and endurance athletes.

	Stature (cm)	Mass (kg)	Age (years)
Sprint (n=12)	182.1 (\pm 4.5)*	83.2 (\pm 7.8)*	24.4 (\pm 3.8)
Endurance (n=12)	177.1 (\pm 4.3)	66.8 (\pm 4.94)	24.3 (\pm 3.6)

* Significant difference between the two groups $P < 0.01$

The criteria for elite status was based on the British Amateur Athletics Association (AAA) National Championship qualifying times (1997) for the participating event of the athlete. All subjects satisfied the criteria by achieving the standard in the three months prior to testing (Table 4.2). All subjects were informed of the intent and procedures of the study and a signed written informed consent (Appendix 1) was obtained prior to any data collection. This study was approved by the Research Degrees Committee of the university. All subjects were requested to refrain from any form of heavy exercise and the consumption of alcohol or caffeine in the 24 hours prior to testing.

Table 4.2. Event and mean performance time (range) of elite sprint and elite endurance athletes in relation to qualifying time for each event.

Subjects	N	Event (m)	Mean Performance Time	Qualifying Time*
Sprint	7	200	21.30s (20.83-21.50)	21.5s
	5	400	46.99s (45.90-47.80)	48.0s
Endurance	2	3000S/C	8:48 min (8:26-9:00)	9:00min
	6	5000	13:56 min (13:43-14:09)	14:20 min
	4	10000	30:05 min (29:35-30.37)	30:40 min

*1997 AAA British National Championship qualifying times. 3000S/C - 3000m steeplechase

4.2.2 System linearity.

An important consideration in the study of gas exchange kinetics, is that of system linearity (Hughson *et al.* 1990b). Investigations have shown that system linearity during PRBS testing could be compromised following a significant increase in blood lactate concentrations above resting levels. One criterion for system linearity is that lactate concentrations should remain below 2 mM and show no significant increase during the test (Essfeld *et al.* 1987, Hoffmann *et al.* 1992, 1994a&b). Immediately preceding and following the exercise test blood samples were taken from each subject's finger. Blood lactate concentrations were measured with a Analox GM7 lactate analyser (Analox Instruments, London, UK). A further criterion of system linearity is that no significant increase in $\dot{V}O_2$ between minutes 3 and 6 of constant load work at the higher intensity should occur. A significant increase in $\dot{V}O_2$ would indicate the presence of a slow component which would affect system linearity (Barstow *et al.* 1994, Whipp and Ward 1990).

In the absence of a slow component rise in $\dot{V}O_2$ static gain values can be obtained. The static gain values for $\dot{V}O_2$ and also $\dot{V}CO_2$ are derived from the steady state values at 25W and

85W preceding and following the PRBS cycles. In a truly aerobic test, the difference between the steady state values (25 W and 85 W) should not differ between subjects (Essfeld *et al.* 1987). A significant difference in static gain between ET and ST would indicate the test being more exerting for one or other of the groups and that the exercise intensities had been set inappropriately high.

4.2.3 Exercise protocol.

The reliability study conducted earlier confirmed the application of a 300s PRBS cycle and consequently this is unchanged. The constant load exercise preceding and following the PRBS sequence was extended to 360s in order to investigate any non linearities in the system.

4.2.4 Training information.

Training logs (Appendix 2) were collected from all subjects in this study. The subjects provided details of three typical training weeks reflecting the seasonal changes in their training practices. i.e the winter season, the competitive summer season and the immediate period prior to testing. The training weeks were divided into seven exercise types to allow for the variability in training stimulus between the groups of sprinters and endurance runners. Training was analysed in terms of the number and types of sessions completed in typical weeks.

4.2.5 Statistical analysis

A two way analysis of variance (ANOVA) with repeated measures was applied to investigate differences between ET and ST. A post hoc Tukey test of honest significant difference was applied to compare the mean values where there was a difference.

Pearson Product Moment Correlations were applied to investigate the relationship between the amplitude ratio and phase shift of $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics and also between $\dot{V}O_2$ kinetics and running performance times.

Statistical significance is accepted at $P < 0.05$. Unless stated otherwise, results are expressed as means \pm SD.

4.3 Results

4.3.1 Physical characteristics.

As shown in Table 4.1, significantly greater values for both stature (cm) ($P < 0.05$) and body mass (kg) ($P < 0.001$) were observed in ST. The age of the athletes was closely matched and the criteria for elite athletic status ensured this parameter was comparable.

4.3.2 System linearity.

The criteria for system linearity was met by all subjects (n=24). Mean pre and post test blood lactate concentrations are shown in Table 4.3. No sample of blood lactate concentration from either group exceeded 2 mM. The pre test lactate concentrations of the sprint group were significantly greater than the endurance group ($P<0.05$), but there was no significant elevation in blood lactate levels from pre to post test in either group. No significant difference was observed between the post test samples of ET and ST. No slow components were observed in the $\dot{V}O_2$ responses to the constant work load at 85 W. This was assessed by comparing the $\dot{V}O_2$ at 3min and at 6 min of the constant load work at 85W for ST (1490 ± 72.0 and 1500 ± 65.5 ml·min⁻¹) and ET (1400 ± 83.1 and 1400 ± 77.4 ml·min⁻¹) respectively. No significant change in $\dot{V}O_2$ was detected from minute 3 to 6 in either group.

Table 4.3. Mean blood lactate concentrations (\pm SD), pre and post pseudo random binary sequence (PRBS) test.

	Blood lactate (mM)	
	Pre test	Post test
Sprint (n=12)	1.47* (± 0.4)	1.52 (± 0.5)
Endurance (n=12)	1.12 (± 0.4)	1.22 (± 0.4)

* significant difference between sprint and endurance groups, $P<0.05$

$\dot{V}O_2$ static gain values of $8.9 (\pm 0.6)$ and $9.2 (\pm 0.9)$ ml·min⁻¹·W⁻¹ were derived for ST and ET respectively, $\dot{V}CO_2$ static gain values for ST and ET (8.9 ± 1.2 and 8.7 ± 0.9 ml·min⁻¹·W⁻¹ respectively) were also obtained. Neither the $\dot{V}O_2$ or $\dot{V}CO_2$ static gain values between ST and ET were significantly different.

4.3.3 $\dot{V}O_2$ kinetics.

The $\dot{V}O_2$ kinetics results of the two groups are shown in Table 4.4. Both groups showed the expected change in amplitude ratio and phase shift in relation to frequency (Figure 4.1).

Significantly greater amplitude ratios were observed in ET than ST at frequencies 6.7 mHz ($P<0.05$) and 10 mHz ($P<0.01$) as shown in Table 4.4. Significantly shorter phase shifts were identified in ET at frequency 3.3 mHz ($P<0.01$).

Table 4.4. $\dot{V}O_2$ kinetics (amplitude ratio and phase shift) of endurance and sprint runners to the PRBS exercise test. Mean \pm SD.

	Amplitude ratio ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$)			Phase shift (degrees)		
	3.3 mHz	6.7 mHz	10 mHz	3.3 mHz	6.7 mHz	10 mHz
Sprint	8.02	5.47*	3.56**	-41.26*	-73.74	-99.53
(n=12)	(± 0.70)	(± 0.95)	(± 0.69)	(± 5.82)	(± 7.93)	(± 9.29)
Endurance	8.38	6.71	4.97	-35.46	-68.72	-95.79
(n=12)	(± 0.90)	(± 1.09)	(± 0.98)	(± 4.31)	(± 6.57)	(± 7.24)

*Significant difference at $P<0.05$, **, $P<0.01$.

Figure 4.1.

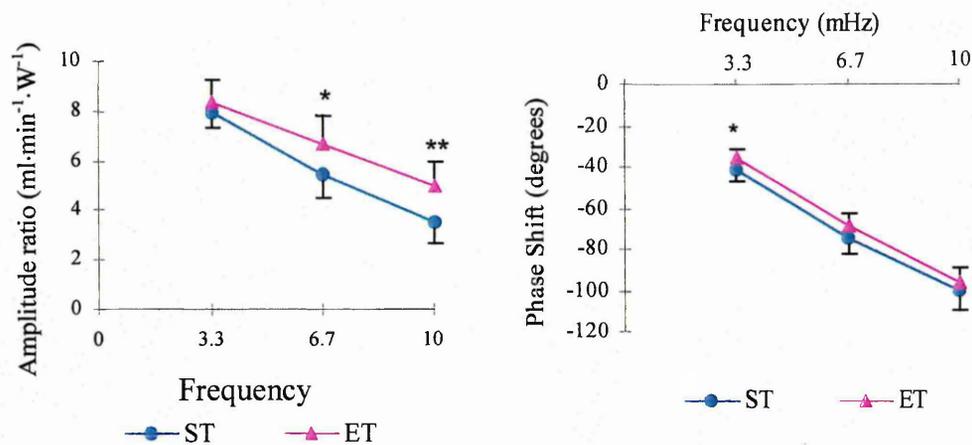


Figure 4.1. Bode plot of the mean changes (\pm SD) in frequency for $\dot{V}O_2$ kinetics of ST and ET expressed in amplitude ratio ($\text{ml}\cdot\text{min}^{-1}\cdot\text{W}^{-1}$) and phase shift (degrees) components.

Significance is shown as * $P<0.05$ and ** $P<0.01$.

Figure 4.1. illustrates the consistent differences between the amplitude ratio and phase shift of ET and ST. Although significance is only present at frequencies 6.7 and 10 mHz of the amplitude ratio ($P<0.05$ and $P<0.01$ respectively) and frequency 3.3 mHz of the phase shift ($P<0.05$), the trend for a greater amplitude ratio and shorter phase shift is evident in ST at each frequency.

4.3.4 $\dot{V}CO_2$ kinetics.

The $\dot{V}CO_2$ kinetics results for ST and ET are shown in Table 4.5. As with the $\dot{V}O_2$ kinetics results, $\dot{V}CO_2$ kinetics show the expected change in amplitude ratio and phase shift in relation to frequency (Figure 4.2.). Statistical analysis did not reveal significant differences between ST and ET at any frequency.

Table 4.5. $\dot{V}CO_2$ kinetics for amplitude ratio and phase shift of endurance and sprint runners to the PRBS exercise test. Mean \pm SD.

	Amplitude ratio ($ml \cdot min^{-1} \cdot W^{-1}$)			Phase shift (degrees)		
	3.3 mHz	6.7 mHz	10 mHz	3.3 mHz	6.7 mHz	10 mHz
Sprint	6.96	3.72	2.11	-59.84	-99.44	-125.92
(n=12)	± 1.3	± 1.03	± 0.74	± 12.71	± 22.87	± 22.41
Endurance	6.33	4.35	3.06	-49.62	-89.88	-110.38
(n=12)	± 0.6	± 1.17	± 0.84	± 7.15	± 13.6	± 14.28

No significant differences observed between ST and ET.

Although not statistically significant, a trend can be observed for a shorter phase shift in ET (Figure 4.2.). This is consistent with the results for $\dot{V}O_2$ kinetics as shown in Figure 4.1. The results for the amplitude ratio of $\dot{V}CO_2$ kinetics are less clear at frequency 3.3mHz where ST attained a higher mean response than ET (6.96 and 6.33 $ml \cdot min^{-1} \cdot W^{-1}$ respectively). The two further frequencies (6.7 and 10 mHz) follow the expected pattern of higher amplitude ratios for ET.

Figure 4.2.

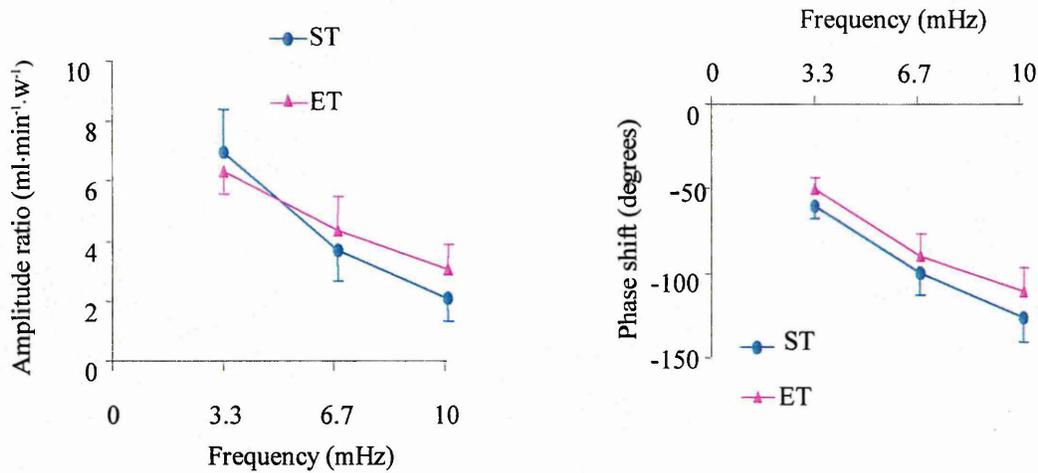


Figure 4.2. Bode plot of the mean changes (\pm SD) in frequency for $\dot{V}CO_2$ kinetics of ST and ET expressed in amplitude ratio ($ml \cdot min^{-1} \cdot W^{-1}$) and phase shift (degrees) components.

4.3.5 Overall mean average cardiorespiratory responses to the PRBS test

The average $\dot{V}O_2$ values for ST were significantly higher than those of ET (22.0 ± 1.1 and $20.4 ml \cdot min^{-1} \cdot W^{-1} \pm 1.3$) ($P < 0.01$). The average $\dot{V}CO_2$ was also elevated in ST compared with ET (19.8 ± 1.3 and $17.6 ml \cdot min^{-1} \cdot W^{-1} \pm 1.2$ respectively) ($P < 0.01$).

Respiratory Exchange Ratio (RER) and 5s average heart rates were collected over the test duration. RER values for ST were also significantly elevated in comparison with ET (0.9 ± 0.1 and 0.86 ± 0.1 respectively) ($P < 0.05$), while the heart rate ($b \cdot min^{-1}$) were also significantly higher in ST than ET (96 ± 9.1 and $80 b \cdot min^{-1} \pm 9.9$ respectively) ($P < 0.001$).

4.3.6 The relationship between $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics

A high level of agreement was, in general, found between the results of $\dot{V}O_2$ kinetics and $\dot{V}CO_2$ kinetics (Table 4.6). Significant correlations were found between $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics for ET and also for ST at all frequencies except at frequency 3.3 mHz of the amplitude ratio where $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics did not correlate for ST ($r=0.12$).

Table 4.6. Correlations of $\dot{V}O_2$ kinetics with $\dot{V}CO_2$ kinetics for the amplitude ratio and phase shift of elite sprinters and for elite endurance runners.

	Amplitude ratio			Phase shift		
	3.3 mHz	6.7 mHz	10 mHz	3.3 mHz	6.7 mHz	10 mHz
Sprint (n=12)	0.12	0.85***	0.79**	0.75**	0.94***	0.63*
Endurance (n=12)	0.53*	0.78**	0.64*	0.72**	0.63*	0.68**

* $P<0.05$, ** $P<0.01$, *** $P<0.001$.

4.3.7 Training information

The training information provided by ST and ET for winter, summer and the immediate period preceding testing is shown in Table 4.7. The data shows the expected preference in training methods between groups with the sprinters completing significantly more interval and resistance training sessions than the endurance runners during typical winter training ($P<0.05$), while ET performed significantly greater continuous sessions at all reported stages of training ($P<0.05$).

Table 4.7. Frequency of training among elite sprinters and endurance runners for three time periods of: winter, summer and the close season prior to testing.

	Interval (Track)	Interval (Road)	Technical training	Continuous training	Resistance training	Circuit training
Endurance(w)	1.53(±0.5)*	1.08(±0.8)	-	7.58(±1.9)*	0.33(±0.5)*	1.33(±0.5)
Sprint (w)	2.75(±0.6)	-	0.92(±0.7)	0.92(±1.0)	2.58(±0.8)	1.25(±0.5)
Endurance (s)	2.33(±0.7)	0.83(±0.6)	0.33(±0.8)	4.92(±0.8)*	-	0.89(±0.3)
Sprint (s)	1.75(±0.5)	-	1.1(±0.6)	0.59(±0.5)	1.42(±0.7)	0.16(±0.4)
Endurance (c)	1.89(±0.6)	1.1(±0.8)	0.33(±0.8)	4.54(±0.4)*	0.43(±0.6)*	1.19(±0.8)
Sprint (c)	1.41(±0.5)	-	0.83(±0.6)	1.14(±0.9)	2.42(±0.7)	0.75(±0.6)

Sprint (n=12), Endurance (n=12). (w), winter training, (s), summer training, (c), current training in the week preceding testing. *, Significant difference from sprinters (P<0.05).

4.3.8 Running performance and $\dot{V}O_2$ kinetics

All sprint subjects provided a 200m performance time while all endurance runners reported a 5000m time for the calculate of correlations between running and $\dot{V}O_2$ kinetics test performance. No significant correlations were found between running performance time and $\dot{V}O_2$ kinetics test for either ST or ET (Table 4.8.).

Table 4.8. The relationship between running performance (200m and 5000m) and

$\dot{V}O_2$ kinetics test for ST and ET.

	Amplitude ratio			Phase shift		
	3.3 mHz	6.7 mHz	10 mHz	3.3 mHz	6.7 mHz	10 mHz
Sprint 200m (n=12)	-0.33	-0.05	0.14	0.25	-0.25	0.02
Endurance 5000m (n=12)	0.02	0.35	0.03	-0.09	0.27	-0.34

No correlations were observed between running performance and $\dot{V}O_2$ kinetics test performance.

4.4 Discussion:

4.4.1 Validity of the test.

Blood lactate levels below 2mM and the absence of a slow component rise in $\dot{V}O_2$ indicates that valid measures of gas exchange kinetics have been obtained. The lactate values for ST and ET taken prior to testing were significantly elevated in ST relative to ET ($P < 0.05$), possibly reflecting that trained sprinters maintain elevated lactate levels even while at rest. It is well known that sprinters are able to attain higher levels of lactate during maximal exercise (Ohkuwa *et al.* 1984, Thomson and Garrie 1981) and the higher resting levels might be a function of a higher tolerance level, however, although the lactate levels were consistently greater in ST, the mean pre test levels were still below 2mM which was the criteria set as an indication of elevated levels. Interestingly, post test levels for ST and ET were not statistically different and remained below 2mM for both groups. This indicates that the test did not require contribution from anaerobic sources and, in some cases, might have acted to clear lactate.

One of the criteria for system linearity was that lactate should remain below 2mM in the post test sample. A significant rise in lactate from pre to post test would indicate that the exercise intensities were set inappropriately high thus resulting in the contribution of anaerobic processes. This was not the case in any of the subjects, and therefore, confidence in the test can be maintained according to this criterion.

A further criteria set to ensure that the test remained at an aerobic level was that there should not be an increase in the $\dot{V}O_2$ between the third and sixth minute of steady state exercise at the higher work rate. Non steady state of $\dot{V}O_2$ would indicate the presence of a slow component rise in $\dot{V}O_2$ which would consequently effect the test validity. A slow component rise in $\dot{V}O_2$ was not detected for any of the subjects and thus further confirms the test remained at appropriate intensities for the subjects.

4.4.2 Physiological profiles of elite sprinters and elite endurance runners

The average $\dot{V}O_2$, $\dot{V}CO_2$ and RER values were significantly elevated in ST ($P < 0.01$) indicating less respiratory control and substrate utilisation than ET. This was not surprising as the sprinters had undergone training to enhance their short term glycolytic systems to be able to perform at maximal efficiency for events of less than one minute duration. The endurance runners, however, have undergone training in order to adapt to perform sustained exercise for ~10 - 30min that require greater contributions from the aerobic energy systems. A more efficient respiratory system is known to delay fatigue in both short and long term exercise (Martin, B.J *et al.* 1979) and the training adaptations of elite endurance runners are consistent with this finding.

The elevated RER for group ST suggests that the endurance runners performed the test with more efficient substrate usage than the sprinters. Although the RER values for ST were significantly greater than those of ET, no RER values were greater than 1.0 in either group which indicates that the test was set at a suitable aerobic intensity, however, the higher RER of the sprinters demonstrates an increased reliance on the limited carbohydrate stores rather than the abundant fat stores. Prolonged carbohydrate rather than fat utilisation would undoubtedly lead to premature fatigue.

Substrate usage during exercise is known to be affected by endurance training (Henriksson *et al.* 1977a) with increased reliance on intramuscular lipid as an energy source during exercise. This effect, coupled with other training adaptations, decreases the rate of utilisation of muscle glycogen and blood glucose and decreases the rate of accumulation of lactate during submaximal exercise. Sprint training has not typically effected substrate utilisation, although, a greater glycolytic capacity (as demonstrated by PFK activity) has been shown in sprinters (Costill *et al.* 1976, Gollnick *et al.* 1972). Increased reliance on anaerobic glycolysis would inevitably involve greater utilisation of carbohydrate with a consequent increase in RER reflecting the contribution of this substrate to energy provision. Consequently, the difference in RER between ST and ET, most probably, reflects the type of training undertaken.

A number of other factors are known to influence the selection of substrate for exercise. Among these are: nutritional status, diet, exercise intensity, previous exercise, drugs, hormones and environmental factors such as temperature and humidity. The most important factor influencing the selection of substrate for muscular work is the relative intensity of the exercise being undertaken by the subjects. Endurance trained subjects with a larger aerobic capacity could perform the test at a low percentage of their capacity. Conversely, sprinters

and non endurance trained groups might perform the test at a higher percentage of their capacity placing increasing reliance on their non aerobic energy systems, however, this would be reflected in elevated blood lactate, which was not the case in this study. In addition, the issue of relative work rates is not relevant in the low intensity PRBS test as, below LT, the $\dot{V}O_2$ response is independent of the work rate (Hughson 1990b).

In this study, all subjects were asked to refrain from heavy exercise, alcohol and caffeine in the 24 hours prior to testing. Caffeine in particular is known to increase the mobilisation of FFA from adipose tissue (Ivy *et al.* 1979, LeBlanc *et al.* 1985) and hence elevates the plasma FFA concentration. Therefore, caffeine ingestion would potentially spare the limited glycogen stores and reduce the RER during the exercise test. While all subjects have stated that they have met these criteria, the effects of diet prior to testing were not controlled. Therefore, it is possible that variations in dietary intake of carbohydrate and fats prior to testing might have contributed to the difference observed. This is, however, unlikely as the difference observed between groups would indicate that one of the groups displayed a consistent dietary characteristic that the other group did not.

Heart rate over the test duration were significantly higher in ST than ET ($P < 0.001$) which conforms to the characteristic differences commonly observed in sprint and endurance athletes. As indicated previously, heart rate differences are largely explainable by specific endurance training adaptations which are known to cause bradycardia due to reduced sympathetic nerve activity and a more effective stroke volume (Winder *et al.* 1978, Bevegard 1963). These factors will cause bradycardia due to the long term endurance training adaptations which the elite runners would be expected to have undergone. While the sprinters

had conducted a similar training volume to the endurance runners, the specific training methods and intensities used by endurance runners, most likely, caused the observed effect. The training logs obtained from the subjects showed the expected preference for interval training in ST and continuous training in ET. All ST subjects (n=12) participated in at least two interval sessions in a typical training week during the winter period, although the quantity of this work reduced during the competitive summer season. These sessions would be expected to result in elevated lactate levels and appear typical for those previously reported in elite sprinters (Ward 1988). Additionally, all sprinters incorporated resistance work into their training programmes which has been shown to improve sprint running performance above that experienced through sprint training alone (Dintiman 1964). The training practices observed in ST are known to result in glycolytic enzyme enhancement (Table 1.4) and improved sprint performance. Few of the sprinters reported any typical aerobic sessions in their training programmes and enhancement in aerobic fitness could be viewed as a 'by-product' of sprint training. The training logs demonstrate that elite endurance runners completed significantly more continuous sessions in their training than the sprinters ($P < 0.05$). By completing endurance training of this type, ET have gained considerable aerobic adaptation and this has undoubtedly led to their success in endurance events. This process will also have increased the oxidative efficiency of their slow twitch muscle fibres. At least five continuous runs of ~1hr were performed by all ET subjects in a typical week over the winter season in addition to interval training sessions of a higher (although still endurance) intensity. As such, they typically completed training methods training consistent with those expected to lead to the training adaptations outlined earlier in section 1.

All runners (ST and ET) had been training for their specific events for at least two years and by attaining elite status would have already have gained substantial training adaptation in addition to their genetically endowed abilities. Therefore, the training undertaken by both groups and their running performance represents typical values for elite athletes specialising in sprint and endurance events. Any absence of statistical difference in PRBS test performance between groups cannot, therefore, be attributed to their incorrect representation of either sprinters or endurance runners.

4.4.3 Relationship between $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics

Although $\dot{V}CO_2$ kinetics did not differentiate ST from ET, significant correlations were observed between the $\dot{V}O_2$ kinetics and $\dot{V}CO_2$ kinetics results (Table 4.6.). This reflects the close relationship between the measures and the use of aerobic processes by the subjects via O_2 intake and CO_2 output. Many physiological mechanisms intervene between O_2 uptake and CO_2 output and, as previously observed, some will have a substantial impact. The correlations between $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics indicate that the oxidative processes of $\dot{V}O_2$ are closely reflected in the production of $\dot{V}CO_2$. Interestingly, the amplitude ratio at frequency 3.3 mHz did not produce a correlation between the $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics of ST. There was no relationship for ST at this frequency ($r=0.12$) and lower level agreement for ET ($r=0.53$) ($P<0.05$) than at any other frequency. The physiological processes reflected at this frequency are, as yet, unclear, however, it is possible that this frequency has detected an element of hyperventilation in ST and thus effected the results.

The similarity between the values observed for $\dot{V}O_2$ and $\dot{V}CO_2$ kinetics in this study suggests that $\dot{V}CO_2$ kinetics could potentially act as a similar test variable to $\dot{V}O_2$ kinetics but as acknowledged with less sensitivity.

4.4.4 The $\dot{V}O_2$ kinetics of elite sprinters and elite endurance runners

The $\dot{V}O_2$ kinetics results of this study support earlier work (Fukuoka *et al.* 1995) which demonstrated that athletes participating in different sports could be differentiated by their dynamic $\dot{V}O_2$ responses during cycling exercise.

The limits of agreement study indicated that a test of $\dot{V}O_2$ kinetics by the PRBS technique was a more reliable measure than $\dot{V}CO_2$ kinetics to distinguish between groups. This finding has been supported in the study of ST and ET runners where ET runners have attained differentially improved $\dot{V}O_2$ kinetics at both amplitude ratio (frequencies 6.7, $P < 0.05$ and 10 mHz, $P < 0.01$) and phase shift (frequency 3.3 mHz, $P < 0.05$). All frequencies of $\dot{V}O_2$ kinetics were faster in ET than ST, however, the relatively large limits of agreement have revealed a lack of sensitivity in the testing process which reduces the confidence of making individual results assessments rather than group comparisons.

The results of this study demonstrate that elements of $\dot{V}O_2$ kinetics within the frequency domain are significantly slower in elite sprinters compared to elite endurance runners. The significantly larger amplitude ratios and shorter phase shift values observed in the endurance trained runners are consistent with the theory that endurance training adaptations interact to

accelerate the adjustment of O₂ supply to O₂ demand during submaximal exercise (Hickson *et al.* 1978). However, opinion is divided among researchers as to whether $\dot{V}O_2$ kinetics might be limited by the rate of O₂ delivery from the heart to the working muscle (Cochrane and Hughson 1992, Hughson 1990b) or by peripheral factors such as oxidative enzyme activity within the muscle mitochondria which limit the rate of O₂ utilisation by working muscles (Mahler 1985, Yoshida *et al.* 1995).

Improvements associated with endurance training include a greater concentration of oxidative enzymes and mitochondria (Gollnick *et al.* 1973, Henriksson *et al.* 1977) which should result in a more rapid $\dot{V}O_2$ adjustment to energy requirements. Sprint type training results in increased activity of glycolytic enzymes (Linossier *et al.* 1997b, Saltin *et al.* 1976) but the effects on oxidative enzyme activity are equivocal (Jacobs *et al.* 1987, Sharp *et al.* 1986). Elite endurance trained athletes have been acknowledged as possessing higher proportions of type I muscle fibres than elite sprinters (Bergh *et al.* 1978, Costill *et al.* 1976) and consequently greater oxidative potential. In addition, the training methods of the endurance runners (Table 4.7.) identifies significantly ($P < 0.05$) greater amounts of aerobic work than the sprinters in any one week which would enhance the oxidative potential of their type I fibres. This leads to the conclusion that they not only possess more type I fibres, but the type I fibres they possess are also more efficient in oxidative processes than those of the sprinters due to specific training adaptations. This might explain the difference in $\dot{V}O_2$ kinetics observed between the two groups.

Endurance training is associated with improvements in O₂ transport to the working muscle, due to greater cardiac stroke volumes (Bevegard 1963) and higher capillary density (Andersen

et al. 1977). Sprinters have been shown to possess a lower capillary density and lower vasodilatory response to dynamic exercise than endurance runners (Torok *et al.* 1995). Therefore, the faster $\dot{V}O_2$ kinetics in endurance runners could be a function of a more rapid increase in total cardiac output and improved blood flow redistribution to the working muscles (Shoemaker *et al.* 1996) which stimulate capillary growth (Hansen-Smith *et al.* 1996).

Although sprint training is more commonly associated with improvements in the anaerobic capacity of muscle, several researchers have shown that sprint type training in previously untrained subjects improves $\dot{V}O_2$ max (McKenna *et al.* 1997, Sharp *et al.* 1986). This increase in $\dot{V}O_2$ max could be attributable to an increased maximal cardiac output as has been found in rats (Hilty *et al.* 1989) suggesting that a similar adaptation might occur in humans. Therefore, sprint training potentially increases $\dot{V}O_2$ kinetics but to a lesser extent than endurance training. Further work investigating differences between the $\dot{V}O_2$ kinetics and untrained and sedentary subjects would be required to substantiate this.

Several studies have examined the effects of endurance training on $\dot{V}O_2$ kinetics (Berry and Moritani 1985, Phillips 1995a, Yoshida 1992) in different population groups using a variety of endurance training programs. These studies have all demonstrated faster $\dot{V}O_2$ kinetics following endurance training. Therefore, it is not surprising that faster $\dot{V}O_2$ kinetics were observed in the elite endurance athletes in the present study, however, as $\dot{V}O_2$ is known to display first order kinetics, it is likely that only one factor is rate limiting.

4.4.5 The $\dot{V}CO_2$ kinetics of elite sprinters and elite endurance runners

An examination of $\dot{V}CO_2$ kinetics did not reveal significant differences between ST and ET. Although statistical significance was not observed between the groups, a trend for larger amplitude ratios and shorter phase shift terms was observed for ET (Table 4.5). Statistical significance in $\dot{V}CO_2$ kinetics might be expected between ST and ET as they have been shown to differentiate fit subjects from less fit subjects (Zhang 1991) and follow a similar time course to $\dot{V}O_2$ kinetics which are also improved in subjects with greater aerobic capacities (Essfeld *et al.* 1987). The $\dot{V}CO_2$ kinetics results in this study also do not support the findings of Fukuoka *et al.* (1997) which suggested that $\dot{V}CO_2$ kinetics could prove a more sensitive measure of training and/or fitness than $\dot{V}O_2$ kinetics. As this is not the case, it could be attributable to a number of factors.

The limits of agreement study for $\dot{V}CO_2$ kinetics revealed substantial variability between test and retest. The measurement error between tests ranged from 39-108% in a positive or negative direction meaning that the detection of a physiological difference might not be possible due to the lack of reliability of the testing process. The $\dot{V}CO_2$ kinetics results were filtered for outlying breaths by using the First Breath Software package and further ensemble averaged in the same manner as the $\dot{V}O_2$ kinetics results but this did not bring the reliability of $\dot{V}CO_2$ kinetics to a similar level. It is, therefore, difficult to draw physiological conclusions from test results of higher variability. It is likely that the subjects less familiar with control of breathing during exercise testing and/or relatively long periods of time could have hyperventilated and thus adversely affected their $\dot{V}CO_2$ kinetics results. It is not possible to dismiss this option as a possibility.

A physiological consideration which might affect the $\dot{V}CO_2$ kinetics results is that of CO_2 storage during exercise. CO_2 storage is known to slow $\dot{V}CO_2$ kinetics below the ventilatory threshold (Hughson and Inman 1985), however, this in itself does not explain the similarity between the $\dot{V}CO_2$ kinetics of ST and ET. The $\dot{V}CO_2$ kinetics results of ST and ET could be a function of the high variability illustrated by the reliability study, as the storage levels of CO_2 could have differed from the pre exercise level of Test 1 to the recovery period preceding Test 2. A difference in the levels of CO_2 storage between tests might have acted to dissociate the test results.

As a physiological explanation for the differences in $\dot{V}CO_2$ kinetics between ST and ET, CO_2 storage could be expected to increase the difference already observed in $\dot{V}O_2$ kinetics between groups as the storage potential of ST should be enhanced by their greater body mass ($P < 0.01$) and stature ($P < 0.05$). That this is has not been observed, leads to the conclusion that storage of CO_2 does not effect $\dot{V}CO_2$ kinetics by PRBS exercise testing. This suggests that an alternative explanation is appropriate.

It is possible that the PRBS exercise test could have attenuated the effect of CO_2 storage through work to work transients. As less CO_2 is stored during exercise intensity increases, the accumulated effect of the 27 minute test could be to minimise storage of CO_2 . Most studies investigating $\dot{V}CO_2$ kinetics have done so with step test protocols (Zhang *et al.* 1991, Hughson and Morrissey 1982), some of which have involved rest to work transitions (Hughson and Morrissey 1982), and this abrupt alteration in state would increase CO_2 storage and more prominently exaggerate its effect. Therefore, a study of $\dot{V}CO_2$ kinetics by step test between sprinters and endurance runners might reveal differences not apparent in PRBS testing.

While CO₂ storage does not appear to explain the absence of statistical difference between ST and ET, the selected exercise intensities must be examined. Increased CO₂ is released and converted to HCO₃⁻ which acts as a buffer to the H⁺ which accompanies lactate as exercise levels become anaerobic. It is possible that if one group i.e ST, were unable to perform the test aerobically then increased CO₂ would be released to act as a buffer and that this might be reflected in closer $\dot{V}CO_2$ kinetics between ST and ET than observed in $\dot{V}O_2$ kinetics, as at high exercise intensities it is known that this process quickens $\dot{V}CO_2$ kinetics (Hughson and Inman 1985, Zhang *et al.* 1991, Whipp and Ward 1990), however, CO₂ only acts as a buffer to the H⁺ and lactate levels would continue to rise while the buffering took place. Subjects would be able to perform the test quite comfortably for some time during this process, but post test lactate testing should reveal increased lactate levels if this had occurred. As this was not the case and all post test samples remained below 2mM it is more likely that the inherent testing variability observed in the limits of agreement offers the most reasonable explanation.

4.4.6 Relationship between $\dot{V}O_2$ kinetics, $\dot{V}CO_2$ kinetics and running performance time.

No correlation was observed between PRBS exercise test variables and running performance. This could be due to the selection of subjects as the subjects groups for ST and ET were based on elite running performances. Consequently, the sprint performances and also the endurance performances were closely grouped. Therefore, the test would have to be particularly sensitive to differentiate between the individual sprinters and also between the individual endurance runners. The study was set up to differentiate between groups of elite ST and ET runners, otherwise it would have been desirable to include performers with a wide range of running

performances in order to examine the correlation potential of the test in relation to practical running performance. As previously indicated, the wide limits of agreement make the test largely impractical for assessment of individuals rather than groups and this finding confirms this.

4.4.7 Future directions and developments.

The comparison of sprinters and endurance runners in this study utilised groups of 200-400m runners and 3000m+ runners. While these groups have been differentiated by $\dot{V}O_2$ kinetics in this study, a larger difference could be expected to exist between the $\dot{V}O_2$ kinetics of shorter duration (100m) sprinters and longer distance (marathon) endurance runners. The selection of 100m and marathon runners might have produced significance between the groups at frequencies where it was not previously observed.

The study of Newsholme *et al.* (1992) identified that the energy required for the 100m sprint is primarily derived from anaerobic pathways, while conversely, the marathon utilises primarily aerobic systems. Consequently, the athletes selected in this study had trained for events for which, to some extent, contributions are required from both aerobic and anaerobic pathways. Although the relative contributions of aerobic energy to 200 metre sprinting and anaerobic energy to 3000 metre running might be minimal, they might have had some influence on the test results. Practical considerations such as subject availability must, however, also be considered and this, in part, determined the selection of subjects for this study.

Although ideally more distinct groups would have been used, it has been demonstrated, in this study, that 3000m+ elite endurance runners have differentially faster $\dot{V}O_2$ kinetics compared with 200-400m sprinters. Therefore, it is questionable whether further studies of 100m and marathon runners would add to this topic other than to reinforce the observed responses.

$\dot{V}CO_2$ kinetics did not differentiate between sprinters and endurance runners in this study, but further studies of more distinct athletic groups might demonstrate the $\dot{V}CO_2$ kinetics expected on the basis of other research (Fukuoka *et al.* 1997).

The surprising finding observed in this study was that $\dot{V}CO_2$ kinetics did not differentiate the elite sprinters from the elite endurance runners. It is possible that this finding was caused by the storage mechanisms of CO_2 in the muscle as the storage process is known to occur more readily at low intensity work rates, such as those utilised in this study. The contribution of CO_2 storage to the $\dot{V}CO_2$ kinetics results could be expected as the two groups had significantly different body types and thus different muscle mass in which to store CO_2 . The elite sprinters would be expected to have slower $\dot{V}CO_2$ kinetics as a consequence of a greater CO_2 storage potential in muscle than their significantly smaller (stature, $P < 0.05$ and body mass, $P < 0.01$) elite endurance counterparts. This was not shown to be the case here, which could be due to a number of reasons.

In the first instance, the underlying $\dot{V}CO_2$ kinetics of the subjects might be obscured by the high variability illustrated by the limits of agreement study. Secondly, the sprinters could have hyperventilated at the low intensity work rates, due to unfamiliarity with the testing process and the test length. Both of these areas provide possible directions for future work.

In order to maximise the potential for differentiation between athletic groups, a future development could be to use a larger difference between higher and lower work intensities. Although the PRBS test must remain beneath the ventilatory threshold, individual measurement of the ventilatory threshold would enable the researcher to more fully exploit the aerobic range of an individual by assessing the work intensities relative to the fitness of the subject. Recent work by Kusenbach *et al.* (1999) applied similar criteria for setting the higher work intensity of PRBS exercise and utilised either 80 W or 40% of the predicted individual working capacity for patients with cystic fibrosis. Application of a larger difference between the work intensities might lead to the difference in $\dot{V}CO_2$ kinetics expected between elite sprinters and elite endurance runners.

To investigate the effect that hyperventilation might have on $\dot{V}CO_2$ kinetics, a study could be devised whereby subjects are tested by the PRBS method outlined earlier and retested following a period of rest but are requested to increase their breathing frequency during exercise. This would have the effect of increasing the CO_2 output and differences could then be examined between $\dot{V}CO_2$ kinetics under normal conditions and during hyperventilation. It might then be possible to assess the extent to which CO_2 storage characteristics during exercise differed between the two groups (i.e sprinters and endurance runners).

The effects of sprint training alone on $\dot{V}O_2$ and/or $\dot{V}CO_2$ kinetics have not previously been investigated, therefore, further studies are required to assess the contribution of sprint training to the difference observed in this study of the $\dot{V}O_2$ kinetics between elite sprinters and elite endurance runners. The study of Fukuoka *et al.* (1997) utilised a training regime comprising both strength and speed components over a nine month period and showed $\dot{V}CO_2$ kinetics were slowed after strength/speed training while there was no effect on $\dot{V}O_2$ kinetics. A further

finding of that study was that strength and speed training improved $\dot{V}O_2$ max and not $\dot{V}O_2$ kinetics suggesting that the limiting factors for $\dot{V}O_2$ max are different from those limiting $\dot{V}O_2$ kinetics. This reinforces the need for further examination of specific sprint training effects on gas exchange kinetics in order to establish whether, as speed and strength athletes have a similar dynamic response to sedentary groups (Fukuoka *et al.* 1995), the effects seen in this study are entirely due to endurance training. If this were the case, then a PRBS test of gas exchange kinetics could be limited to discriminating between athletic groups on the basis of endurance characteristics alone.

A number of studies have employed sprint training regimes (Jacobs *et al.* 1987, Jansson *et al.* 1990, Linossier *et al.* 1993 & 1997) to investigate changes in anaerobic enzyme activity and or muscle fibre distribution. These studies have additionally demonstrated adaptive changes in sprint performance over their training periods and their format would, therefore, appear suitable for the investigation of the effect of sprint training on gas exchange kinetics. The training regimes employed in these studies utilised multiple Wingate type tests 3-4 times a week as their training tools as this method provides a scientific control over training in the laboratory and is relatively straightforward to implement and monitor. In addition, cycle training is specific to the subsequent testing on an exercise cycle, as would be the case in the gas exchange kinetics test.

A further developmental area would be to investigate the mechanisms limiting gas exchange kinetics during PRBS exercise. Traditionally, techniques of gaining information of metabolic function in the muscle have been carried out with invasive techniques. However, a new and non-invasive method of studying intracellular metabolism has been developed. Nuclear magnetic resonance (NMR) spectroscopy uses radiofrequency energy within a strong

magnetic field to probe and identify the content of chemical elements and compounds within living tissue. One such field is ^{31}P , which examines levels of high energy phosphates within the muscle (Boicelli 1989). Consequently, levels of PCr, inorganic phosphates (Pi) and ATP can be measured within the muscle. This method has recently been developed to investigate the mechanisms controlling $\dot{V}\text{O}_2$ kinetics by Whipp *et al.* (1999) and is likely to provide greater insight in this area in the future.

High energy phosphates including PCr have been found to reach their highest concentrations in sprinters, and in fast-twitch fibres, that is, in the athletes and muscle fibres that specialise in high intensity work (Bernus *et al.* 1993, McCully *et al.* 1992). A study of sprint and long distance runners (Bernus *et al.* 1993) revealed significant differences in PCr and Pi concentrations between groups.

If high energy phosphate levels in the muscle could be increased by an appropriate training stimulus, an improvement in sprint performance could be expected. For example, a previously endurance trained group would be expected to sprint faster following sprint training but conversely their oxidative capacity could be negatively affected, leading to compromised gas exchange kinetics and endurance performance. Therefore, a future study investigating the effect of sprint type training on previously endurance trained subjects and the resultant gas exchange kinetics and a ^{31}P NMR investigation would be a study of interest.

An increasing number of studies have begun to look at the relationship between $\dot{V}\text{O}_2$ and PCr kinetics (Mahler 1985, McCreary *et al.* 1996, Whipp *et al.* 1999) and an indication to a peripheral rather than central limitation to $\dot{V}\text{O}_2$ kinetics. Further development of this area could reveal more insight into the mechanisms controlling the $\dot{V}\text{O}_2$ kinetics response.

4.4.8 Summary and conclusions of gas exchange kinetics in elite runners.

The measurement of $\dot{V}O_2$ kinetics in the frequency domain using PRBS techniques has been shown in this study to discriminate between elite endurance runners and elite sprinters. CO_2 output kinetics did not differentiate between groups and thus these findings do not support the findings of Fukuoka *et al.* (1997) which indicated that $\dot{V}CO_2$ kinetics could prove a more sensitive measure of training status and/or fitness than $\dot{V}O_2$ kinetics. Further work might reveal whether sprint training enhances or compromises $\dot{V}O_2$ kinetics and further support the development of the test to distinguish between a wide range of sport performers.

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Appendices

Appendix 1. Informed Consent.

CONSENT FORM

INVESTIGATOR: ANDREW EDWARDS

Health and Fitness Laboratory, Pearson Building, 27 Broomgrove Road. Tel: 2252454

Discomforts

The protocol used in this study is sub maximal. This means that it is unlikely to cause any discomfort or pose a risk to health. However, should you experience any unusual feelings associated with the physical effort, then please stop the test.

Medical screening

You are requested to complete a simple health and exercise questionnaire prior to testing. Only current health problems indicated on this form will exclude you from the study. All records are strictly confidential.

Inquires

Any questions about the procedures used in this study are encouraged. If you have any doubts or queries then please feel free to ask.

Freedom of consent

Your permission to perform these tests is required. Your participation is strictly voluntary and you are free to deny consent and withdraw from this investigation at any time.

A) Personal details

Surname _____

First name(s) _____

Date of birth _____

Age _____

Contact address

Telephone No. _____

B) Medical details

i) Have you been told that you have any of the following health problems?

Yes No

- | | | |
|---------------------------|--------------------------|--------------------------|
| a) asthma | <input type="checkbox"/> | <input type="checkbox"/> |
| b) high blood pressure | <input type="checkbox"/> | <input type="checkbox"/> |
| c) diabetes | <input type="checkbox"/> | <input type="checkbox"/> |
| d) anaemia | <input type="checkbox"/> | <input type="checkbox"/> |
| e) cardiovascular disease | <input type="checkbox"/> | <input type="checkbox"/> |

ii) Have you had any of the following in the last 6 months?

Yes No

- | | | |
|---|--------------------------|--------------------------|
| a) viral or bacterial infections | <input type="checkbox"/> | <input type="checkbox"/> |
| b) musculoskeletal injuries | <input type="checkbox"/> | <input type="checkbox"/> |
| c) operations involving a general anaesthetic | <input type="checkbox"/> | <input type="checkbox"/> |

iii) Are you taking any medications prescribed by your G.P?

Yes No

If yes, please give details

iv) Do you have any other disabilities that may impair your exercise performance in these tests?

Yes No

If yes, please give details

Name _____ (please print)

Signature _____ Date _____

Witness _____ Date _____

Appendix 2. Training information.

Name: Age:

Main performance event:

Personal best time for 1997: Date achieved:

Personal best time for 200m (Sprinters): Date achieved:

Personal best time for 5000m (Endurance): Date achieved:

Training information

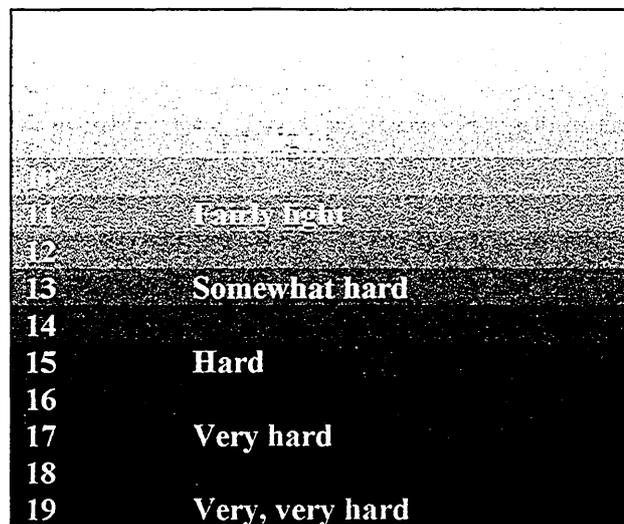
Please specify, in as much detail as possible, your typical training week during:

1) Competitive season Dates:.....

Exercise Type	Frequency (times/week)	Intensity* (RPE scale)	Duration (time)	Example session
Interval (Track)				
Interval (Road)				
Technical training				
Continuous training				
Resistance training				
Circuit training				

* See RPE scale to grade your exercise intensity (6-19)

Rating of Perceived Exertion (RPE) scale



2) Winter season**Dates:.....**

Exercise Type	Frequency (times/week)	Intensity* (RPE scale)	Duration (time)	Example session
Interval (Track)				
Interval (Road)				
Technical training				
Continuous training				
Resistance training				
Circuit training				

3) Immediate week prior to testing**Dates:.....**

Exercise Type	Frequency (times/week)	Intensity* (RPE scale)	Duration (time)	Example session
Interval (Track)				
Interval (Road)				
Technical training				
Continuous training				
Resistance training				
Circuit training				