# A METABOLIC COMPARISON OF ISOKINETIC AND FREE SPRINTING

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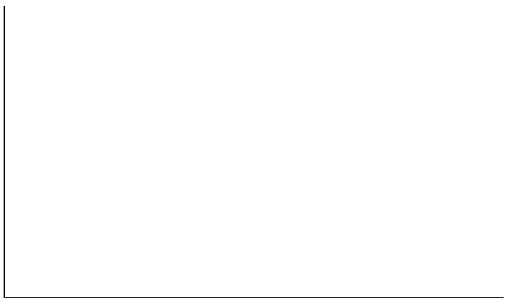

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## ABSTRACT

The purpose of this study was to examine the Excess Post-Exercise Oxygen Consumption (EPOC) and peak blood lactate responses of sprinters following exhaustive treadmill running, maximal isokinetic and free sprinting. Eight university sprinters (mean: age = 24.8 yrs., ht. = 178.9 cm., wt. = 74.9 kg.) performed a 2 minute anaerobic speed test (AST) and tethered isokinetic and free sprinting protocols consisting of five second maximal repetitions separated by ten second active recoveries. A five repetition isokinetic set was compared to five and ten repetition free sprint sets.

A correlation ($r = +0.87$) was calculated between the EPOC and peak blood lactate values over the four experimental protocols. Tukey's post hoc comparisons determined significantly different corrected EPOC (HSD = 2.04, $\alpha < 0.05$) and peak blood lactate (HSD = 1.72, $\alpha < 0.05$) cell means between the 2 minute AST (15.16 $\pm$ 2.59 Litres; 14.83 $\pm$ 1.21 mmol/L) and the other three protocols: the 5 repetition anaerobic power master (APM) (11.38 $\pm$ 2.72 Litres; 12.77 $\pm$ 1.97 mmol/L), the 10 repetition free sprint (9.88 $\pm$ 2.80 Litres; 11.25$\pm$ 2.15 mmol/L) and the 5 repetition free sprint (9.09 $\pm$ 2.51 Litres; 9.83 $\pm$ 3.09 mmol/L). Additionally, significance was between the 5 repetition APM condition and the 5 repetition free sprint condition. These findings suggest that five, 5 second isokinetic sprinting repetitions require more work in less time and thereby produce a metabolic demand similar to ten, 5 second free sprinting repetitions.

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# INTRODUCTION

To date anaerobic performance has primarily been researched and quantified using single exhausting bouts of exercise. With the development of portable oxygen analyzers and in the field ergometry, research into interval performance and training is now a viable prospect. Research into intermittent supramaximal exercise may further the understanding of the bioenergetic and recovery processes functioning during intervals of anaerobic exertion.

Shortcomings exist in the literature concerning the recovery of the adenosine triphosphate-creatine phosphate (ATP-CP) cycle and the role glycolysis plays in short bursts of high intensity intermittent anaerobic work. Medbo et al (1988) contend that creatine phosphate is broken down very rapidly and contributes little to the accumulated $O_2$ deficit with an increased exercise duration beyond 15 seconds. Subsequently, the main process that accounts for further anaerobic formation of ATP is glycolysis. However, the glycolysis dominance may not apply to the same degree to interval work which has a five second exercise duration and ten second recovery interval thereby, allowing partial restoration of the CP concentrations. Harris et al (1976) found that CP resynthesized biphasically demonstrating a fast and a slow component. The components were found to have half-times of 21 - 22 seconds and over 170 seconds respectively after 6 minutes of exhaustive exercise. However, it has been contended that the conditions of the preceeding exercise may affect the replenishment rate of CP. The scene is further complicated by the findings of Medbo and Tabata (1989) which claim that oxygen consumption can attain 85% of maximum 15 seconds from the commencement of exercise. Therefore, the contribution of aerobic metabolism to supramaximal exercises may be greater than previously thought.

Further probing raises the question of, what limits anaerobic performance and causes fatigue? Medbo et al (1988) suggest that the maximal rate of glycogen degradation may be a limiting factor for the rate of anaerobic ATP formation. This is supported by the observation that, independent of the absolute magnitude of the anaerobic capacity, a leveling off of the accumulated $O_2$ deficit occurs after 2 minutes of exhaustive exercise. Similarly, Weyland et al (1993) found that the $O_2$ deficit plateaued after only 70 seconds of exhaustive stationary bicycle exercise. Hence subjects with a large anaerobic capacity are able to produce ATP at a much higher rate than subjects with a low anaerobic capacity. Accordingly, it has been shown that sprint-trained subjects have a higher accumulated $O_2$ deficit and accumulate more lactate in the blood than endurance-trained subjects during 1 min of exhausting exercise (Medbo et al 1988).

However, despite the maximal rate of glycogen degradation it is interesting that fatigue is experienced during short exercise bouts, even though the blood and muscle lactate accumulations are far from maximal. Therefore, factors other than acidosis, produced from glycogen metabolism, may inhibit high intensity exercise (Medbo et al 1988).

The development of two new modalities, the APM, an isokinetic sprint ergometer and the Cosmed K2, a telemetered portable self-contained oxygen consumption analyzer, make it technically possible to analyze the performance of interval sprinting. The combination of these two instruments facilitates the comparison of resisted (isokinetic) sprinting and unresisted (free) sprinting via measurement of Excess Post-exercise Oxygen Consumption.

Elevated oxygen consumption after exercise recently termed "Excess Post-exercise Oyxgen Consumption" (EPOC) is a well established phenomenon (Bahr et al 1992, Gore and Withers 1990; Maehlum et al 1986; Stainsby and Barclay 1970). The volume of EPOC has been applied to quantify the homeostatic disturbance produced by exercise (Sedlock et al 1989). Thereby, the volume of EPOC, unlike the accumulated $O_2$ deficit, is viewed, not as an indication of anaerobic capacity, but as an indicator of recovery and adaptive energy employed by the entire body.

*1.1 Statement of the Problem*

This study was designed to compare EPOC and peak lactate responses to performances on the isokinetic APM and free sprinting; as well as, a comparison relative to the performance of a 2 minute AST.

*1.2 Hypotheses*

It was hypothesized that the EPOC and peak blood lactate responses to 5 and 10 free repetitions and 5 APM repetitions would produce relationships of the following:

A) $5\ \text{Rep APM}_{EPOC} > 10\ \text{Rep FreeSprint}_{EPOC} > 5\ \text{Rep FreeSprint}_{EPOC}$

B) $5\ \text{Rep APM}_{BLa} > 10\ \text{Rep FreeSprint}_{BLa} > 5\ \text{Rep FreeSprint}_{BLa}$

C) $5\ \text{Rep APM}_{EPOC} = \text{AST}_{EPOC}$

*1.3 Significance of the Study*

This study serves to validate isokinetic sprinting as a valuable sprint training supplement. This form of sprinting demonstrates the ability to perform more work in less time. This study is also significant in that it applies EPOC as an indicator of homeostatic disturbance rather than analyzing the processes which produce EPOC. Using the power of a repeated measures design both EPOC and peak blood lactate are able to metabolically compare different forms of supramaximal exercise.

*1.4 Definition of Terms*

**ANAEROBIC SPEED TEST (AST)**: A graded treadmill run to exhaustion.

**EXCESS POST-EXERCISE OXYGEN CONSUMPTION (EPOC)**: The summed volume of all $O_2$ derived processes in excess of resting $VO_2$ values that restore metabolic homeostasis in response to exercise. These processes include not only disturbances in the working muscles but all organs and tissues of the body (Roth et al 1988). Consequently, EPOC is more than mere repayment of the $O_2$ deficit (Gore and Withers 1989).

**FREE SPRINTING**: Unihibited sprinting performed on a horizontal surface.

**ISOKINETIC SPRINTING**: Sprinting performed on a horizontal surface with resistance provided by an isokinetic tethering device.

**$O_2$ DEFICIT**: The difference between the total oxygen consumed during exercise and the amount of oxygen required to produce the ATP to perform the exercise.

**SUPRAMAXIMAL EXERCISE**: An intensity of exercise requiring a rate of ATP production that exceeds the maximal power of the aerobic system.

*1.5 Delimitations*

This study was delimited by the following:

A) a sample of male university varsity sprinters between the ages of 19 and 30 years.

B) the methodology and work bouts performed to determine and produce the EPOC's and peak blood lactate concentrations.

C) a respiratory gas sampling rate set at 15 second intervals.

*1.6 Limitations*

This study was limited by the following:

A) the individuals metabolic response to the exercise protocols.

B) the data collection capabilities of the Cosmed K2, the APM and the Kontron Medical lactate analyzer 640.

C) the assumption of a maximal effort by each subject for each test.

D) the present understanding of EPOC.

E) the sensitivity each subjects' oxygen consumption to the discomfort of conditions imposed on them other than the intended experimental exercise.

# LITERATURE REVIEW

Post-exercise oxygen consumption was first examined by Hill and associates in the 1920's. The "oxygen debt" hypothesis stated that lactate metabolism was linked to post-exercise oxygen consumption. They concluded that the excess oxygen metabolized 1/5 of the lactate produced to provide energy for the conversion of the remaining 4/5 of the lactate to glycogen. In the 1930's Margaria et al elaborated the hypothesis by discriminating a fast ("alactacid") and a slow ("lactacid") recovery oxygen curve components. This hypothesis states that the fast phase represents the restoration of ATP and CP stores, while the slow phase reflects the oxidation of lactate. Since Margaria et al numerous studies have demonstrated a discrepancy between the post-exercise lactate concentrations and $VO_2$ (Gaesser and Brooks 1984).

It is apparent that the hypotheses of Hill et al and Margaria et al are overly simplistic. As no complete account of the post-exercise metabolism exists, the term EPOC has been deemed to describe the set of phenomena that occur during recovery from exercise. It is an appropriate term as it avoids implication of causality in describing the elevation of metabolic rate above resting levels after exercise (Gaesser and Brooks 1984, Stainsby and Barclay 1970).

## *2.1 Anaerobiosis*

Energy requirements for supramaximal efforts are met by phosphagen-splitting (hydrolysis of ATP and CP), glycolysis (hydrolysis of glycogen to lactic acid) and oxidative mechanisms (changes in blood and muscle oxygen saturations) respectively in a sequentially contributing and overlapping fashion (Davies 1971; Evans 1981, Wenger and

Reed 1976). Due to the delayed increase of oxygen uptake at the onset of exercise the proportional contribution of aerobic metabolism to total energy turnover increases with time (Medbo and Tabata 1989). The dependence on anaerobic versus aerobic metabolism is associated with the intensity and duration of the exercise (Gollnick et al 1986).

However, the energy requirements for 4-5 seconds for all-out efforts of short duration are mainly met by phosphagen-splitting (Kaczkowski et al 1982; Maehlum et al 1986) which proceeds at a rate independent of $O_2$ supply and is limited only by the amount of ATP and CP in the muscle (Davies 1971) and the magnitude of CNS recruitment (Radford 1984). In activities that demand high tension and/or velocity of movement fast twitch units must be recruited to supplement the aerobically produced energy. Accordingly, the leg muscles of successful athletes involved in muscle strength and power activities possess a predominance of fast twitch muscle fibre area (Kaczkowski 1982; Radford 1984).

Decreases in ATP and CP concentrations in skeletal muscle during exercise vary with the relative work rate. Training results in a lower decline of these muscle high energy phosphate concentrations in the same exercising individual. Two factors that may account for a lower decrease in high energy phosphate concentrations, a smaller increase in lactate concentration and a smaller EPOC are the following: 1) a more rapid rise in $O_2$ delivery, resulting in the development of less muscle hypoxia at the onset of exercise 2) an improvement in oxygen availability to the contractile fibres made possible by the exercise training-induced adaptations in the muscle resulting in the increased extraction of $O_2$ from the blood (Hagberg et al 1980) and 3) an increase in the magnitude of the recovery enzyme profile (Astrand et al 1986).

Hagberg et al (1980) further reported that under submaximal conditions trained individuals were capable of increasing their $VO_2$ more rapidly than untrained individuals. More importantly, it was found that $VO_2$ adjusted to energy need more rapidly after training even at the same relative work rate. It seems clear that in addition to the relative work rate, the individual's level of training plays an important role in determining the time course of the adjustment to exercise and the time course of recovery. Such adaptations are magnified under supramaximal conditions and explain the ability of aerobic metabolism to supply upwards of 40% of the total ATP required for 30 seconds of exhaustive exercise (Medbo and Tabata 1989). The major weakness of Kreb's cycle lies in its compartmentalization in the mitochondria, thus limiting the rate of transportation of the ATP to the cross bridges. Conceptually, the intramitochondrially produced ATP are being transported to the cytosol more rapidly and in greater magnitude (Wenger and Reed 1976). Functionally, the adaptation of the aerobic energy system to supramaximal exercise increases the duration that the ATP-CP and glycolytic systems can function at a given intensity before anaerobic arrest.

*2.2 Anaerobic Muscular Fatigue*

Anaerobic muscular fatigue primarily occurs as a consequence of lactate accumulation which lowers muscle and blood pH and in turn inhibits (PFK). These effects serve to reduce the glycolytic flux of ATP. The increased acidity has been suggested to affect the permeability of membranes to $Na^+$ and $K^+$ effectively hyperpolarizing the cell and decreasing contractility. This effect is even more pronounced in type IIB (-85mv) over type I (-70mv) fibres as their inherent resting potential is lower. Additional inhibitory effects have been implicated by the accumulation of $H^+$ which compete with Ca+ for binding sites on actomyosin effectively rendering these cross bridges disfunctional (Wenger and Reed 1976).

*2.3 Effect of Exercise Intensity and Duration on EPOC*

When exercise ceases abruptly, oxygen uptake does not return immediately to the pre-exercise level. Oxygen uptake decreases exponentially, approaching the pre-exercise level almost asymptotically. During the period of time in which the oxygen deficit is accumulating, the metabolizing tissues are using stored oxygen and energy sources such as free ATP, CP and glycogen for energy production. These sources of energy are "borrowed" and presumably must be restored via $O_2$ uptake, hence the term "EPOC". The existence of EPOC is well established but the magnitude, duration and physiological basis after various intensities and durations of exercise is still debated (A. Gore and Withers 1990; Maehlum et al 1986; Stainsby and Barclay 1970).

Exercise intensity affects both the magnitude and duration of EPOC, whereas the exercise duration affects only the duration of EPOC (Bahr and Sejersted 1991). The duration of EPOC is not necessarily related to the amount of postexercise energy expenditure as recovery metabolism is affected by the magnitude of the homeostatic disturbance such that exercise intensities exceeding approximately 50% $VO_2max$ will have an increasingly greater impact on EPOC. It seems that when exercise intensity is greater, the caloric expenditure at the onset of recovery is greater due to the elevated metabolic rate during exercise which is carried over into the EPOC. The magnitude and duration of EPOC are not necessarily related and both should be assessed when examining the postexercise response (Sedlock et al 1989).

Consensus has been achieved that $O_2$ deficit reaches a maximum magnitude for exhaustive bouts of running lasting 2 min or more which is consistent with peak blood lactate concentrations achieved after 2 minutes of exhaustive exercise. Estimations of EPOC volumes, which are closely associated with $O_2$ deficit, vary from 30 minutes to 72 hours in

duration (Maehlum et al 1986; Medbo 1988). It is likely that the varied reports of EPOC duration are due to the lack of standardized methods for, determining $O_2$ consumption baselines and producing exercise protocols.

Gore and Withers (1990) reported that the magnitude of EPOC increases with exercise intensity linearly for exercise bouts between 20% and 80% $VO_2$max whereas it has been shown to increase exponentially once exercise intensity approaches 100% $VO_2$max. In a repeated measures factorial design exercise intensity was the major determinant of EPOC since it explained five times more of the EPOC variance than either exercise duration or the intensity duration interaction (Gore and Withers 1990).

*2.4 Mechanisms and Metabolic Components of EPOC*

Because the mitochondrion is the site of $O_2$ consumption in the cell, the explanation of the elevated post-exercise $VO_2$ may be found at the level of this cellular organelle. Direct control of mitochondrial respiration may be exerted by concentrations of primarily ADP in addition to ATP, Pi and CP. Mahler and Homsher (1982) proposed that the rate-limiting step is the intramitochondrial production of ADP by creatine kinase. Indirect control of mitochondrial respiration may include a variety of factors, including catecholamines, thyroxine, glucocorticoids, fatty acids, calcium ions and temperature (Gaesser and Brooks 1984; Roth et al 1988).

It may be stated that metabolic rate will return to control levels when all the factors that influence mitochondrial respiration have returned to control levels. Without careful consideration of all the factors influencing mitochondrial respiration, measurements of the elevated post-exercise $VO_2$ may be of limited value (Gaesser and Brooks 1984).

Numerous factors have been associated with the elevated postexercise metabolic rate. However, the relative contribution of each factor remains unknown (Sedlock et al 1989). Exercise triggers a multitude of processes that must return to a basal turnover rate during the recovery period (Bahr and Sejersted 1991). Therefore, other tissues as well as muscle must be involved since whole body EPOC is much greater than can be accounted for by local muscle events (Bahr et al 1992; Bahr and Sejersted 1991; Bangsbo et al 1990; Stainsby 1970). The components of the EPOC are believed to include the following:1) replenishment of $O_2$ stores in blood and muscle 2) resynthesis of ATP and CP in the exercising muscles 3) metabolism of lactic acid 4) repletion of glycogen 5) response to catecholamine release 6) oxidation of fat 7) turnover of substrate cycles 8) elevation of body temperature 9) compensatory increased protein synthesis 10) restoration of ionic homeostasis 11) thermic effect of food and 12) elevated physiological functioning (Bahr et al 1987; B. Bahr and Sejersted 1991; Bangsbo et al 1990; Bangsbo et al 1991; Bielinski et al 1985; Medbo et al 1988; Roth et al 1988; Stainsby and Barclay 1970).

Bangsbo et al (1991) concluded the metabolism of lactate, ADP, inorganic phosphate and creatine could account for only 47% of recovery $O_2$ from 0 to 10 min. Consequently the classical lactate theory of EPOC has been strongly refuted and a large body of literature now contends that EPOC may be explained in terms of the numerous factors listed above (Gore and Withers 1990).

*2.5 Lactate*

Lactic acid, the endproduct of glycolysis, increases in concentration in the blood exponentially with increasing exercise intensity (Gollnick et al 1986). Lactic acid is a strong organic acid (pK=3.8). At physiological pH values it will dissociate to a proton ($H^+$) and an anion ($C_3H_6O_3^-$). Isolated mitochondria oxidize exogenous lactate at the same or greater rate than pyruvate. Thereby, it is impossible to estimate lactic acid production rates during exercise from measurements of blood lactate levels or to interpret the blood lactate inflection point solely as a sudden increase in production. Lactic acidosis occurs to some degree at all exercise intensities and is due to the difference between its net release to and clearance from the blood. Therefore, measurement of blood lactate concentration only allows speculation on its underlying mechanisms (Gollnick et al 1986, Stainsby and Brooks 1990).

Any ATP produced from glycolysis leading to lactate formation will not have required the use of oxygen. Lactate production appears to be large under some circumstances and apparently contributes a savings in oxygen uptake which is measureable but lactate production is transient, being large only early in activity (Stainsby and Barclay 1970).

Another beneficial aspect of lactate production concerns the extrememly high rate of energy derivation associated with glycolysis or glycogenolysis. The degradation of 1g of carbohydrate to lactate does not yield as much energy as the combustion of 1g of carbohydrate or fat combusted to water and carbon dioxide; however, the rate at which the energy is produced surpasses by far the speed with which energy can be derived aerobically (Jacobs 1986).

Traditionally, lactate has been considered to be a metabolic end product whose appearance in muscle and blood during exercise was thought to indicate anaerobic metabolism (Brooks and Gaesser 1980). In fact, non-oxidative metabolism must supplement aerobic metabolism in order to meet ATP requirements of working muscle but lactate production, in contracting muscle, can occur even when the muscle is well oxygenated. Therefore, lactate levels in the blood can certainly be influenced by factors other than the degree of oxygenation (Gaesser and Brooks 1984).

### *2.5.1 Blood Lactate Kinetics*

The activity of lactate dehydrogenase in skeletal muscle is far greater than the combined activities of enzymes providing alternate pathways for pyruvate metabolism and the rate limiting enzyme for the Kreb's cycle. In fact, lactate is produced at rest at approximately 100 $mg \cdot kg^{-1} \cdot h^{-1}$. Therefore, lactate production in muscle is an inevitable consequence of glycolytic carbon flux (Brooks 1986, Gaesser and Brooks 1984).

A small fraction of lactate moves across cell membranes via simple diffusion. However, most lactate appears to move across cell membranes in conjunction with a cation (Na+ or H+) via facilitated transport. Consequently, the blood lactate increases and peaks approximately 5 minutes after exercise because the active muscle intracellular lactate concentration is not in equilibrium with the extracellar space and the blood. Additionally, decreasing pH inhibits key glycolytic enzymes and only a minor fraction of the lactic acid formed may diffuse to the cell membrane for transport to the extracellular space near the cell membrane. Thus the exchange of lactate between cells and blood is affected by lactate concentration, proton gradients, intracellular pH and intracellular metabolism; thereby, not solely reflecting the rate of glycolysis. It appears transport is restricted, equilibrium is not

achieved and significant cell-to-blood gradients do exist and the net output may be delayed significantly relative to net production (Gollnick et al 1986, Stainsby and Brooks 1990).

Training produces effects on the hormonal response to exercise which could effect the lactate production. Untrained compared to trained individuals demonstrate a stronger adrenergic response to exercise, which has been linked to an excessive rate of glycogenolysis. The elevated glycogenolysis produces an excess of pyruvate which is converted to lactate via lactate dehydrogenase resulting in a functionally falsely elevated blood lactate (Brooks 1986, Gollnick et al 1986). Meanwhile, interval sprint training produces enzymatic adaptations of increased capacity to derive ATP from both glycolytic and oxidative processes (Jacobs et al 1987).

The reasoning for the production and distribution of lactate is explained by the lactate shuttle theory. It hypothesizes that lactate serves as: 1) a substrate to maintain blood glucose via hepatic gluconeogenesis and more importantly as 2) an oxidizable substrate from active muscle (areas of production) to many diverse tissues (areas of net removal), thereby functionally distributing substrate and removing metabolic "waste". Fittingly, the rate of these two fates of lactate are linearly related to arterial lactate concentration and exponentially related to $VO_2$ (Brooks 1986, Stainsby and Brooks 1990).

### *2.5.2 Supramaximal Exercise*

Muscle lactate production and accumulation will occur almost immediately with the onset of exercise which demands more energy than can be provided aerobically. Adaptive responses to training with this type of exercise include an enhanced ability to 'pump out' lactate quickly from the muscle to the circulation (Jacobs 1986).

Consequently, the main difference between trained and untrained individuals during heavy exercise is the greater metabolic clearance rate (MCR) imparted by training. This difference may be partly attributable to a lesser autonomic response in the trained. Higher blood glucose levels in the trained during heavy exercise are consistent with the maintenance of splanchnic blood flow. In untrained individuals the stress of heavy exercise may trigger an autonomic response that shunts blood away from the gluconeogenic organs and toward contracting muscle, thus limiting the capacity for release of glucose from the liver (Donovan and Brooks 1983).

The lactate response to supramaximal exercise is a sensitive indicator of adaptation to "sprint training" and is correlated with supramaximal exercise performance. Although the lactate response to exercise is reproducible under standardized conditions blood lactate concentration can be influenced by the site of blood sampling, ambient temperature, changes in the body's acid-base balance prior to exercise, prior exercise and dietary manipulation (Jacobs 1986).

The highest lactate concentrations after single bouts of maximal exercise are induced by exercise corresponding in duration to a 400m or 800m run. Such lactate levels can reach 25 mmol/L or 15 to 20 times normal resting concentrations. These peak blood lactate values after exhaustive exercise are reliable if activity following exercise is standardized. The rate at which lactate leaves the muscle cell will influence the rate of recovery from local muscular fatigue when supramaximal exercise is subsequently resumed (Jacobs 1986).

Interestingly, anaerobic capacity according to Schnabel and Kindermann (1983) can be reliably estimated by the increase in arterial lactate concentration over the pre-exercise

value of a 40 second submaximal test ($^L_{40}$) and the maximal arterial lactate level in the maximal test ($L_{max}$) which explains 30.8% and 57.2% of the variability in maximum time respectively. Additionally, if the run is performed at the same speed in all subjects, time to exhaustion is a measure for inter-subject comparisons of anaerobic capacity.(Schnabel and Kindermann 1983)

### *2.5.3 Blood Flow*

Other compounding factors include the competition for blood flow. As exercise intensity and the mass of active muscle increases, central commmand in concert with assorted peripheral chemoreceptor and mechanoreceptor afferent inputs increases sympathetic vasoconstrictor activity to muscle and other tissues. In the active muscles this constrictor activity is opposed by local vasodilator mechanisms, which are linked by unestablished mechanisms to the metabolic rate of the muscle. As would be expected for proportional control systems, the system does not fully compensate. As a result, flow does not rise as much as $VO_2$ and extration of $O_2$ increases to aid in the elevation of $VO_2$ (Stainsby and Brooks 1990).

Immediately after a sudden modest (20-30%) reduction in flow the force of contraction decreases with little increase in extraction. As a result, $VO_2$ decreases almost as much as the flow. Indications are that contracting muscle performance and $VO_2$ are very sensitive to changes in blood flow and suggest that the effect is not mediated via $O_2$ limitation in the mitochondria. Whatever the mechanism, flow is a significant effector of $VO_2$ and when it is changed $VO_2$ may be changed. Because of competition for flow, the maximal $VO_2$ of a given muscle is reduced, compared to that when working alone, when a large

mass of muscle is active at the same time. But at no time is the muscle so hypoxic that $O_2$ transport limits oxidative phosphorylation in the muscles (Stainsby and Brooks 1990).

*2.5.4 Role of O2 in Lactic Acid Production*

The rate of lactate production may not always be a suitable indicator of oxygen lack even when hypoxia is severe. Stainsby and Brooks (1990) demonstrated that raising arterial $Po_2$ by breathing 100% $O_2$ had no effect on $VO_2$, contraction performance or lactate production during repetitive twitches. Therefore, during severe hypoxia oxygen lack may be present but metabolic arrest may reduce substrate availability and preclude production of lactic acid. Measurements of muscle myoglobin saturation during repetitive twitch contractions have shown $O_2$ to be low but adequate in muscle mitochondria and directly unrelated to lactate production under free flow, normoxic conditions at any level (Stainsby and Brooks 1990).

Breathing hypoxic gas causes muscle blood flow to increase and $(a\text{-}v)O_2$ to decrease during submaximal exercise so that muscle $VO_2$ is maintained during hypoxemia. Muscle net efficiency remains at 23% during hypoxic exercise as during normoxic exercise. In contrast to the constancy of $VO_2$ and muscular efficiency, net lactate release increases dramatically during hypoxia. A 3-4 fold increase in arterial epinephrine levels are observed during hypoxic exercise. Accordingly, it is possible to suggest that the increased muscle lactate outflow is, in part, due to a Beta adrenergic stimulation of muscle glycogenolysis (Stainsby and Brooks 1990).

*2.6 VO2 Baselines*

Recovery oxygen volumes would be expected to be different when different recovery baselines are used. Traditionally, three baselines have been used in studies of recovery oxygen. These are 1) basal metabolic rate (BMR) 2) resting metabolic rate (RMR) and 3) light work metabolic rate (Stainsby and Barclay 1970).

BMR is probably the proper baseline for measurement of recovery oxygen but because of the tedium necessary to achieve BMR it is impractical and almost never used. The most commonly used baseline which seems to yield reproducable results is RMR. Use of RMR assumes that whatever created the RMR continues unchanged through the exercise period and throughout recovery. However, this assumption ignores the anticipatory response to exercise which is present pre-exercise but non-existent post-exercise. Consequently, one must raise the issue as to what recovery includes and what parts of it one wishes to study (Stainsby and Barclay 1970).

Often pre-exercise resting baseline volumes are significantly greater than recovery baseline volumes. This disparity may be largely explained by the anticipatory responses to exercise. As a result, by convention, metabolic adjustments made before and after exercise are evaluated by means of pre- and post-exercise baseline substractions (Roth et al 1988). Additionally, resting oxygen consumption may vary diurnally; therefore, control values should be taken at the same time of day as the postexercise values (Maehlum et al 1986).

*2.7 Methodologically Induced Individual Variation*

Determination of an individuals performed work may be obscured solely by the measure of external work. Since energy is expended and oxidizable substrate formed whether muscular contractions perform external work or not, due to imperfect biomechanics or maintainence of posture, the metabolic cost of the work is the proper measure of performed work. With the exception of limited blood flow, isometric work and pathological conditions, internal work (oxygen metabolism) occurs at a set efficiency. Alternately, external work occurs at a variable efficiency because of variation in such factors as neuromuscular coordination, biomechanics and anatomical anamolies. Consequently, external work ultimately impacts internal work creating metabolic individual variations (Henry 1951).

Additionally, subjects with greater maximal aerobic power have a more rapid time course of early recovery but similar total EPOC possibly reflecting the influence of increased metabolic heat load produced by performing greater absolute, albeit equal relative workload. As a result, variability in the degree of aerobic training will influence the EPOC profile but not the total volume (Kaminsky et al 1987).

Above all, the experience of fatigue is the single most important source of methodological error. Fatigue is a subjective experience that is influenced by motivation and is therefore difficult to assess objectively. Surprisingly Medbo et al (1988) found the values for the acccumulated $O_2$ deficit to be highly reproducible for all durations. For exhausting bouts lasting 2 min the precision (standard deviation) was 3 ml/kg, corresponding to a relative error of 4% of the maximal accumulated $O_2$ deficit. Apparently, motivated subjects well accustomed to strictly standardized procedures produce small methodological errors (Medbo et al 1988).

## 2.8 Isokinetic Dynamometry

Isokinetic contraction is the muscular contraction that involves constant velocity limb movements around a joint. The velocity of movement is maintained constant by a special dynamometer such that the resistance produced is equal to the muscular forces applied throughout the range of movement (Baltzopoulos and Brodie 1989). In this manner, the controlled variable is the velocity of muscular shortening and not the resistance as is normally the case in muscle strength testing where the velocity becomes a consequence of the load applied on the muscle (Thorstensson et al 1976). This method provides the opportunity to manipulate the speed so as to establish conditions specific to exercise with regard to contraction speed. Once an isokinetic device is set at a specific operating speed, it permits and demands muscular contractions at that speed (Baltzopoulos and Brodie 1989; Hislop and Perrine 1967).

The load acting in isokinetic exercise is the result of the mechanical process of energy absorption which an isokinetic device performs in order to keep the exercise speed constant. Energy cannot be dissipated by acceleration because this is mechanically prevented by the device. Because the energy is not dissipated anywhere in the process, it completely converts to a resisting force which is always proportional to the magnitude of the input (muscular force). Thus it varies in relation to the efficiency of the skeletal leverage (Hislop and Perrine 1967).

### 2.8.1 Research Value of Isokinetics

Isokinetic dynamometry is popular because it avoids invasive technique, is simple to administer and short in duration. Statistically it is repeatable, reliable, valid and sensitive to changes in anaerobic fitness. Isokinetic endurance tests represent valid laboratory tests

for evaluating high-intensity, short-term exercise in which the muscle is primarily dependent upon anaerobic processes for energy release (Patton and Duggan 1987). Peak power is assumed to reflect maximal power generation by the breakdown of phosphagens during the initial repetition; whereas, mean power represents energy production from combined phosphagen utilization and glycogenolysis over the entire duration of the test (Smith 1987).

### *2.8.2 Effect of Gravity and Inertia*

Isokinetic dynamometers like the cybex measure the muscle moment, which is the muscle force application times the length of the radius of the lever arm from the axis to the line of muscle pull, which is usually labelled the "muscle torque." The primary limitations of the Cybex dynamometer include torque overshoot during acceleration of the limb to the regulated speed (inertial effects) and a lack of gravity compensation (Alexander 1990; Baltzopoulos and Brodie 1989). This has been demonstrated by Osternig (1975) who found the maximum isokinetic torque values tended to shift to more extended joint positions as the speed of knee extension increased. This shift may have been due to the momentum of the leg during the faster isokinetic speeds overcoming some of the inertia of the weight of the leg as it was extended (Osternig 1975).

The underlying weakness in the cybex mechanism lies in its analysis of vertical plane movements; therefore, the limbs are not only working against the dynamometer but are alternately aided and opposed by gravity throughout the range of motion. Often these gravitational forces have not been taken into account, and the error involved can be quite large (Winter 1981). A key feature of the APM is the fact that it analyzes movements in the horizontal plane which involves no gravitational forces (Rhodes and Roberts 1992).

Although the Kin/Com has largely overcome the limitations plaguing the Cybex (Alexander 1989) it is still a laboratory test and is not feasible as an in the field testing modality.

*2.9 Modalities*

Until recently, direct metabolic and ergometric analyses were not possible on interval sprinting. Previous technology permitted the analysis of sprinting exercise solely by single exhausting exercise bouts such as the AST. The development of a new modality, the APM and a telemetered portable self-contained $O_2$ consumption analyzer (Cosmed K2) now make it technologically possible to ergometrically and metabolically analyze the performance of interval sprinting.

The APM is a new isokinetic tool that has been engineered to calculate the linear mean and peak power output for up to a twenty-four metre distance. This unit consists of a recoiling drum with 24 metres of cord wrapped around it. The unit allows cord to be reeled off the drum at a preprogrammed rate, of up to 5 m/s, which is regulated through a microprocessor that controls an electromagnetic brake installed on the drum. The functioning module and its accompanying supportive frame is extremely portable, easy to assemble and simple to use. The athlete fitted with a waist belt, that is harnessed to the cord of the APM with a hand clasp, sprints away from the device. Once the athlete has accelerated to a pre-set velocity, the unit adaptively controls the athlete for a preselected time and then stops the forward motion (Rhodes et al 1991).

The power output is calculated every 1/100th of a second by measuring the cord tension and unwound length. The LCD displayed output is the average power generated during all completed strides during the designated time. The power and length of each complete

stride is also calculated and can be diplayed on a PC optionally attached to the ergometer. Reliability coefficients ranged from .81 to .88 using human subjects while coefficients reached .99 dropping known weights from a suspended APM (Rhodes et al 1991).

Similar to ergometry the field of metabolic physiology has been broadened with the development of, the Cosmed K2, a telemetered portable self-contained $O_2$ consumption analyzer. The K2 consists of a portable analyzer unit and a receiver unit. The analyzer unit is firmly but not restrictively straped to the subject's chest and consists of a face mask, an expiratory gas sampling pump, a dynamic microchamber, an $O_2$ analyzer, a cardiac frequency transmitter and a radio transmitter. The unit processes and displays the data on the receiver unit with an option of 15, 30, and 60 second interval printouts. Most importantly the Cosmed K2 allows the absolute freedom of movement during performance of exercise. The Cosmed K2 and the 2001 exercise system demonstrate strong correlation co-efficients for $VO_2$ (r=0.95), VE (r=0.96) and HR (r=0.97) using stationary bicycle exercise . However, a significant difference was found in VE at higher workloads (Ienna et al 1992).

Lothian et al (1993) found that the variability between trials with the Cosmed K2 and the Quinton on-line oxygen analysing system were 3.0 - 11.4% and 1.1 -3.9% respectively. Findings also demonstrated that at workloads in excess of 1.10 L/min the Cosmed K2 underestimated the $VO_2$. Similarly, Peel and Utsey (1993) found the Cosmed K2 produced $VO_2$'s 2 - 3 ml·min-1·kg-1 lower than a Gould 9000PC metabolic system. However, these results were based on treadmill exercise which produces slippage of the face mask with the motion of the head and subsequent air leakage leading to the findings of lower $VO_2$'s compared to conventional metabolic systems. Alternately Dal Monte et al (1989) found no significant difference between the Cosmed K2 and the Jaeger on-line

system for different workloads using stationary bicycling. Lothian et al (1993) attributes the latter finding to the type of exercise used in which head movement is minimal thus reducing the air leakage to a minimum.

The combination of these two instruments facilitates a novel comparison of the energy requirements between resisted (isokinetic) sprinting and unresisted free sprinting. Rhodes and Roberts (1992) yielded APM power outputs of up to 1428 watts. The APM demonstrates the capacity to generate unparalleled exercise intensities under accurately simulated performance conditions with the exception of speed.

# PROCEDURES

## *3.1 Subjects*

Ten male U.B.C. varsity sprinter and middle distance track athletes served as the subject pool. The experimentation was conducted during the Canadian Interuniversity Athletic Union (C.I.A.U.) competitive season to ensure all subjects would be in a highly trained condition. Subjects were requested to be 2 hours postabsorptive before each test.

## *3.2 Pre-Experimental Protocol*

Prior to granting written consent subjects were fully informed and familarized with the equipment, the tests to be performed and the degree of exhaustion which would be experienced during the experimentation. Interested individuals completed a written informed consent form and a physical activity readiness questionnaire (PAR Q).

## *3.3 Research design*

The study employed a four condition (2 Min AST, 5 Rep APM, 5 Free, 10 Free) repeated measures design.

## *3.4 Experimental Protocol and Procedures*

### *3.4.1 Overview of Procedures*

Subjects were required to perform eight tests in all, each on separate days. The eight tests consisted of three sets. Set one involved lying supine for 10 minutes fitted with a K2 oxygen analyzer to measure fasting RMR. Tests in the second and third sets were

performed randomly and not necessarily in the order described. The second set, tests two to four, consisted of three 8 mph AST's: one at 15% grade, one at 20% grade and one at 25% grade. The purpose of the three AST's was to establish an intensity vs. duration performance curve for each individual and to extrapolate a treadmill angle which would exhaust that subject after two minutes of running at 8 mph.

The third set of tests was the experimental trials. The fifth test was a 2 minute AST, performed at the angle extrapolated from the set two data, which additionally entailed measuring peak blood lactate and fitting the athlete with a Cosmed K2 system prior to the test to measure EPOC. The two minute AST was designed to assess maximum attainable EPOC. The remaining three tests contrasted in that they were not exhaustive but still required maximal efforts and like test five necessitated fitting of the Cosmed K2 system prior to the test. Two sprinting conditions, an isokinetic protocol and an unresisted (free) protocol, were also employed. Test number six, the isokinetic condition, required subjects to perform one set of five, five second, three metres per second repetitions, each separated by a 10 second fifteen metre return jog. Tests seven and eight, the free sprinting conditions, entailed the performance of one set of 10 repetitions and one set of 5 repetitions respectively. Each repetition, in both tests, was five seconds in duration and was separated by a ten second, fifteen metre jogging recovery.

### *3.4.2 Anaerobic Speed Tests*

All four AST's were performed in the J.M. Buchanan Exercise Science Laboratory at the U.B.C. Aquatic Centre. The treadmill was elevated to a specified angle and set at 8 mph. Subjects were instructed to hold on to the hand rails and by their own volition to step on to the moving treadmill. Subjects ran until exhaustion, at which time they straddled the

treadmill and grabbed the handrails. Performance was measured by duration in seconds maintained on the treadmill. Timing of the run commenced once the subject had hopped on to the treadmill and released his grip from the handrails and stopped once the subject had departed the treadmill or had grasped the handrails once again.

### *3.4.3 Isokinetic Sprints*

Isokinetic sprints (test six) were conducted on an APM in the U.B.C. Thunderbird stadium concourse. Subjects were fitted with a waist belt which was clasped on to a cord which was wound on to the recoiling drum of the APM. The APM protocol involved 5 repetitions of 5 seconds at 3 metres per second with a 10 second recovery. The subject was harnessed to the APM and the test was started as the subject initiated the first repetition. The APM allowed the sprinter to accelerate up to the programmed velocity (3 m/s) after which time the uncoiling drum was isokinetically braked, making acceleration impossible. The five second timer was automatically activated once the isokinetic brake had engaged. Once the five second interval was over the machine braked the sprinter to a rapid stop and activated the 10 second recovery timer. The subject was cued of the end of the ten second recovery and the simultaneous start of the next repetition by an electronic beeper. During the recovery subjects were required to jog back to the APM to allow it to recoil the cord for the next interval. At the end of the fifth repetition the subject was detached from the APM and lead into the training room, which was directly adjacent from the finishing position, and made to lie supine on a plinthe for collection of blood and EPOC.

### 3.4.4 Free Sprints

The two free sprinting conditions were also performed in the U.B.C. Thunderbird stadium concourse. The free sprint protocols mimicked the isokinetic condition to facilitate valid comparisons of the free and isokinetic conditions. The 5 repetition and 10 repetition free sprint protocols (tests seven and eight) required subjects to perform maximal sprinting repetitions of 5 seconds separated by 10 second, 15 metre jogging recoveries. Signalling was performed manually using a whistle in response to a continously running stopwatch and each test was designed so that the subject would complete the last repetition adjacent to the training room to minimize any further activity post-experimental. Upon completion of the fifth and tenth repetitions in their respective tests, blood lactate and EPOC were measured.

## 3.5 Data Collection

### 3.5.1 Oxygen Consumption

All oxygen consumption analysis was performed with a portable K2 oxygen analyzer, which was set to sample every 15 seconds. Oxygen consumption analysis was performed for RMR determination (test one), the 2 minute AST (test five), the 5 repetition isokinetic sprint protocol (test six), the 5 repetition free sprint protocol (test seven) and the 10 repetition free sprint protocol (test eight).

### 3.5.2 Resting Metabolic Rate

Each subject's fasting resting oxygen consumption was measured for 10 minutes in the supine position to assess RMR. The assessment took place at the subject's residence just

after waking up but prior to eating breakfast. RMR was calculated as the mean rate (L/min) of oxygen consumption for the last five minutes of the ten minute session.

### *3.5.3 Excess Post-exercise Oxygen Consumption*

Subject's were layed to rest supine on a plinthe and thirty minute EPOC volumes (litres) were measured immediately after each test in set three. The Cosmed K2 oxygen consumption analyzer was activated and continuously run from three minutes prior to the test to thirty minutes post-exercise. Oxygen consumption was averaged for each minute from the four 15 second sampling intervals; consequently, absolute EPOC was calculated as the sum of the thirty one minute averages and corrected EPOC was calculated as absolute EPOC minus 30 minutes of RMR conditions extrapolated from the 5 minute RMR measurement.

### *3.5.4 Maximum EPOC*

Maximum EPOC was determined for each subject in response to, test five, an eight mph AST at an angle designed to last 2 minutes for each subject. The 2 minute value was based on the findings of Medbo et al (1988) who documented a plateauing of the attainable $O_2$ deficit after 2 minutes of exhaustive treadmill running. This recognized value circumvents the assignment of an arbitrary AST duration to elicit a maximum EPOC.

### *3.5.5 Power Outputs*

Power outputs for each isokinetic repetition were measured in watts directly by the APM strain gauges. The power output is the average for all the strides in the five second interval. Alternately, AST power outputs had to be calculated using trigonometry. Direct power measures were not obtainable for the free sprinting protocol.

### *3.5.6 Blood Lactate*

Cutaneous finger tip 20 microlitre blood samples were collected one, three and five minutes post-exercise after each set three test while the subject lay on a plinthe. The samples were immediately haemolyzed and analyzed with a Kontron Medical lactate analyzer 640 to determine the blood lactate concentration in millimoles per litre.

### *3.5.7 Free Sprint Distances*

The free sprint repetition and total distances were assessed using a marker dropping system. Ten sets of two markers were made. Marker sets 1 to 5 were used for the 5 repetition protocol while all ten marker sets, 1 to 10, were used for the 10 repetition protocol. Each set of two markers were numbered 1 to 10 to indicate the respective repetition. One set of markers were handed to the subject prior to the start of each repetition. One marker was dropped at the starting postition while the other marker was dropped when the five second interval whistle was heard. After the test was over all distances between matching markers were measured in metres using a tape measure.

*3.6 Statistical Analysis*

An analysis of variance was used to compare the dependent variables EPOC (litres), and peak blood lactate (mmol/L) over the four experimental conditions (tests 5 to 8). A statistical significance level of ($p < 0.001$) was used in the analyses. Significant differences between EPOC and peak blood lactate cell means were elucidated using Tukey's post hoc comparisons. Statistical significance level of ($\alpha = 0.05$) was used in the analyses. A correlation was then performed to measure the relationship of EPOC to peak blood lactate.

# RESULTS

*4.1 Subjects Descriptive Data*

Ten anaerobically trained university track athletes participated in this study. Two subjects were not included in the data analysis because they did not perform their greatest EPOC after the 2 min AST protocol. All subjects were in good health and injury free for each test. Descriptive data (age, height, weight, RMR and RMR 30 minute volume) for the eight subjects are presented in Table 1.

**Table 1: Subjects Mean Descriptive Data**

| | Age (years) | Height (cm) | Weight (kg) | RMR (litres/min) | RMR 30 Minute Volume (litres) |
|---|---|---|---|---|---|
| **Mean** | 24.8 | 178.9 | 74.9 | 0.25 | 7.39 |
| **St.Dev.** | 3.7 | 6.0 | 6.6 | 0.02 | 0.60 |

*4.2 Performance Data*

The variables of $EPOC_{30Min}$ (litres) and peak blood lactate (mmol/L) were repeatedly measured over the four experimental protocols (tests 5 to 8) and correlated ($r = +0.87$). An ANOVA for each variable, shown in Table 7, over the four protocols revealed significant differences ($p<.001$) between the cell means. Tukey's post hoc comparison of

$EPOC_{30Min}$ means, displayed by Table 8, demonstrates that the significant differences (HSD = 2.02; $\alpha$ = .05) were between the 2 minute AST and 5 free sprinting repetitions (6.07), the 2 minute AST and 10 free sprinting repetitions (5.28), the 2 minute AST and 5 APM repetitions (3.78) and 5 APM repetitions and 5 free sprinting repetitions (2.29). Similarily, Tukey's post hoc comparison of peak blood lactate means shown by Table 9, demonstrates that the significant differences (HSD = 1.72; $\alpha$ = .05) were between the 2 minute AST and 5 free sprinting repetitions (5.00), the 2 minute AST and 10 free sprinting repetitions (3.58), the 2 minute AST and 5 APM repetitions (2.06) and 5 APM repetitions and 5 free sprinting repetitions (2.94).

Tables 2 thru 5 also display post-exercise oxygen consumption (POC) for 1 minute ($POC_{1Min}$) and other means further relevant to each described test. Expanded tables of individual data are provided in the Appendix. Table 6 expresses the $EPOC_{30Min}$ and peak blood lactate means for 5 APM, 10 free and 5 free repetitions as a percentage of the 2 Min AST $EPOC_{30Min}$ and peak blood lactate means.

Figures 1, 2 and 3 graphically represent the POC over time, the volume of EPOC and the peak concentrations of blood lactate respectively for the four experimental protocols.

**Table 2: 2 Minute AST Means; Test 5**

| | % Grade | Duration (seconds) | Watts | Watts/Kg | 1 MIN POC (litres) | 30 MIN POC (litres) | 30 MIN EPOC (litres) | Peak La (mmol/L) |
|---|---|---|---|---|---|---|---|---|
| **Mean** | 15.4 | 122 | 398.8 | 5.3 | 3.28 | 22.55 | 15.16 | 14.83 |
| **St.Dev.** | 1.9 | 5.24 | 52.8 | 0.65 | 0.29 | 2.37 | 2.59 | 1.21 |

**Table 3: 5 Repetition APM Sprint Means; Test 6**

| | Total Watts | Watts/Kg | 1 MIN POC (litres) | 30 MIN POC (litres) | 30 MIN EPOC (litres) | Peak La mmol/L |
|---|---|---|---|---|---|---|
| **Mean** | 4362.9 | 58.2 | 2.68 | 18.77 | 11.38 | 12.77 |
| **St.Dev.** | 508.6 | 4.00 | 0.43 | 2.84 | 2.72 | 1.97 |

**Table 4: 10 Repetition Free Sprint Means; Test 7**

| | Total Distance (metres) | 1 MIN POC (litres) | 30 MIN POC (litres) | 30 MIN EPOC (litres) | Peak La mmol/L |
|---|---|---|---|---|---|
| **Mean** | 347.85 | 2.52 | 17.27 | 9.88 | 11.25 |
| **St.Dev.** | 18.28 | 0.32 | 2.96 | 2.80 | 2.15 |

**Table 5: 5 Repetition Free Sprint Means; Test 8**

| | Total Distance (metres) | 1 MIN POC (litres) | 30 MIN POC (litres) | 30 MIN EPOC (litres) | Peak La mmol/L |
|---|---|---|---|---|---|
| **Mean** | 183.87 | 2.55 | 16.48 | 9.09 | 9.83 |
| **St.Dev.** | 14.11 | 0.38 | 2.48 | 2.51 | 3.09 |

**Table 6: Proportion of AST Peak La and $EPOC_{30Min}$ Means**

| | 5 Rep APM | 10 Rep Free | 5 Rep Free |
|---|---|---|---|
| **EPOC 30 Min** | 75.1%±11.9 | 65.2%±15.2 | 60.0%±11.0 |
| **Peak La** | 86.1%±9.3 | 75.9%±9.8 | 66.3%±15.7 |

**Figure 1: Line Graph of Mean Post-Exercise Oxygen Consumption**

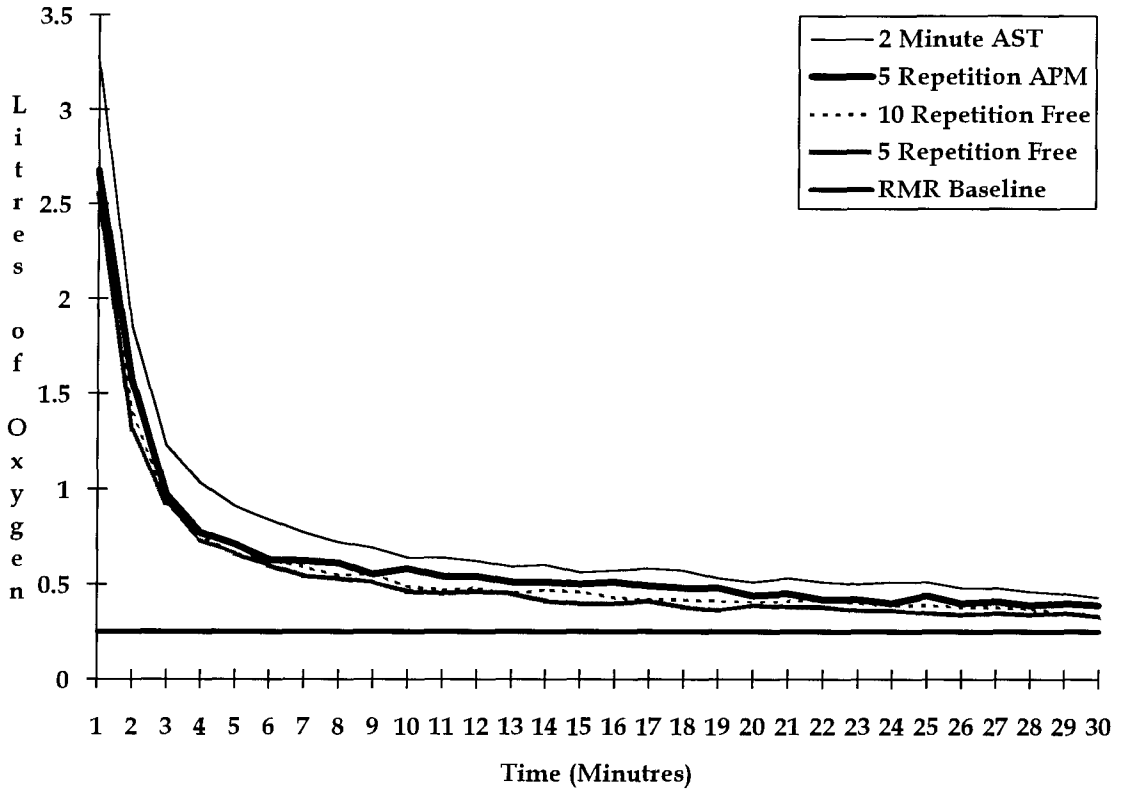

**Figure 2: Bar Graph of Mean Excess Post-Exercise Oxygen Consumption**

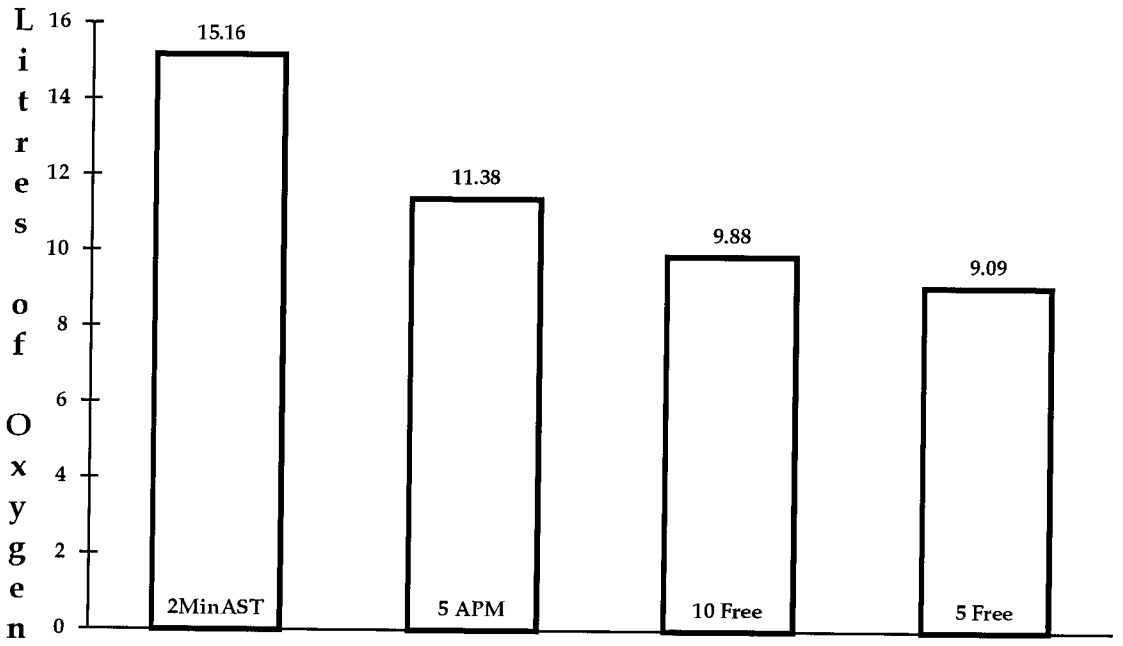

**Figure 3: Bar Graph of Mean Post-Exercise Peak Blood Lactate**

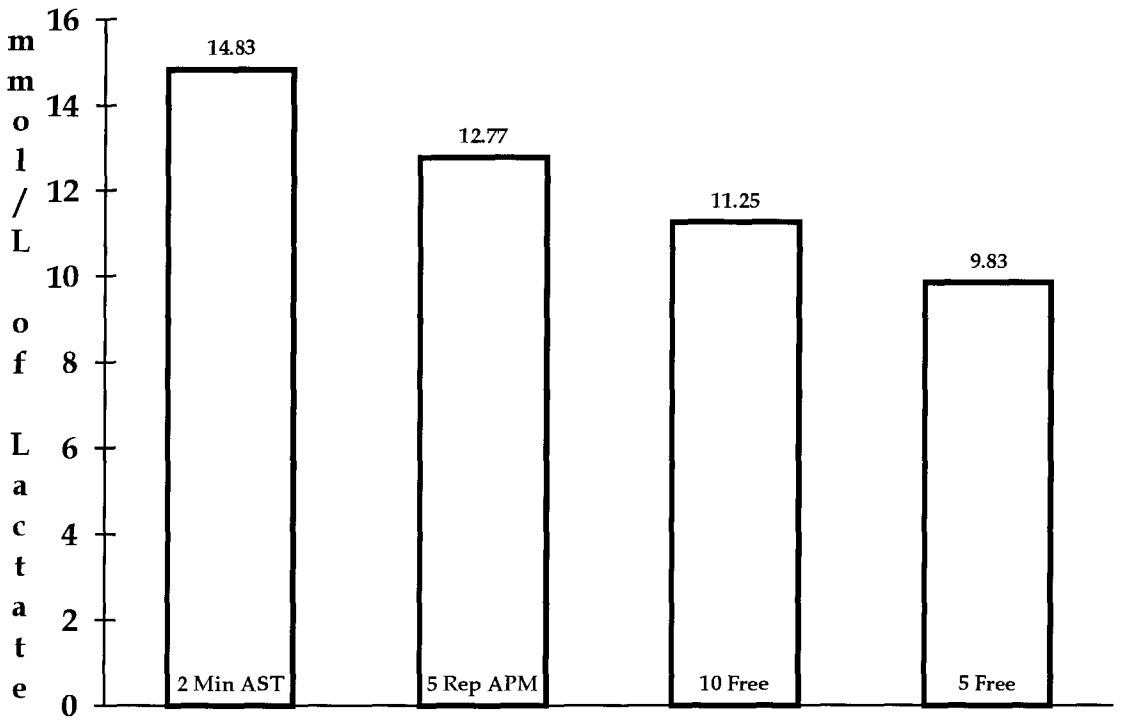

**Table 7: Repeated Measures 1 x 4 Anova**

| Variable | Sprinting Protocol (df, p) | Significance (Yes/No) |
|---|---|---|
| **Peak Lactate** | (18, .000)* | Yes |
| **EPOC 30 Min** | (21, .000) | Yes |

* Note degrees of freedom are lower for peak lactate due to the inability to extract blood from subject 2.

**Table 8: Tukey's Post Hoc Comparison of EPOC Data**

**HSD= 2.02; $\alpha$= .05**

| | | 5FREE | 10FREE | 5 Rep APM | 2 Min AST |
|---|---|---|---|---|---|
| | | **9.09** | **9.88** | **11.38** | **15.16** |
| **5FREE** | **9.09** | 0 | 0.79 | 2.29* | 6.07* |
| **10FREE** | **9.88** | | 0 | 1.5 | 5.28* |
| **5 Rep APM** | **11.38** | | | 0 | 3.78* |
| **2 Min AST** | **15.16** | | | | 0 |

*** = Significant difference ($\alpha$ = .05)**

**Table 9: Tukey's Post Hoc Comparison of Peak Blood Lactate Data**

**HSD= 1.72; $\alpha$= .05**

| | | 5FREE | 10FREE | 5 Rep APM | 2 Min AST |
|---|---|---|---|---|---|
| | | **9.83** | **11.25** | **12.77** | **14.83** |
| **5FREE** | **9.83** | 0 | 1.42 | 2.94* | 5.00* |
| **10FREE** | **11.25** | | 0 | 1.52 | 3.58* |
| **5 RepAPM** | **12.77** | | | 0 | 2.06* |
| **2 Min AST** | **14.83** | | | | 0 |

*** = Significant difference ($\alpha$ =.05)**

## 4.3 Results of Hypotheses

### 4.3.1 EPOC Means

A) 5 Rep $APM_{EPOC}$ = 2 Min $AST_{EPOC}$ Reject

B) 5 Rep $APM_{EPOC}$ > 10 Rep $FreeSprint_{EPOC}$ Reject

C) 5 Rep $APM_{EPOC}$ > 5 Rep $FreeSprint_{EPOC}$ Accept

### 4.3.2 Peak Blood Lactate Means

A) 5 Rep $APM_{BLa}$ = 2 Min $AST_{BLa}$ Reject

B) 5 Rep $APM_{BLa}$ > 10 Rep $FreeSprint_{BLa}$ Reject

C) 5 Rep $APM_{BLA}$ > 5 Rep $FreeSprint_{BLa}$ Accept

# DISCUSSION

The uniqueness of the exercise protocols, modalities and methodologies used in this study, make it difficult to compare previous findings with those presented in this paper. The variables analyzed have complicated underlying mechanisms and have not yet been decisively quantified during exercise; therefore, the discussion will be somewhat general. Ten subjects participated in this study to investigate if $EPOC_{30MIN}$ and peak blood lactate responses are significantly different between maximal isokinetic and free sprinting interval workbouts.

## 5.1 Subjects

The subject pool used in this study appears to be descriptively typical of other studies which used trained athletes. The fasting RMR (0.25 L $\pm$ 0.02) falls well within the range of other findings (Bahr et al 1987, Bahr et al 1991). Similarly, the other descriptive characteristics of height, weight and age are undistinguished from one other supramaximal EPOC study (Bahr et al 1992).

## 5.2 EPOC

Considerable debate surrounds the accounting of the processes which produce EPOC; however, there is little dispute over the existence of EPOC (Bahr et al 1992, Gore and Withers 1990; Maehlum et al 1986; Stainsby and Barclay 1970) and its validity as a quantifier of homeostatic disturbance (Sedlock et al 1989). However, until all the processes which influence EPOC are traceable and measureable, EPOC research will be technologically limited to interpreting the volume of EPOC simply as an index of overall

post-exercise recovery metabolism (Gaesser and Brooks 1984). Most EPOC research has been confined to submaximal exercise and some low level supramaximal exercise.

EPOC is a multifactorial process which is accounted for by more than just the events occuring in the active muscle bed and other required cellular metabolism (Bahr et al 1992). Processess reported to contribute to EPOC are the following: 1) replenishment of $O_2$ stores in blood and muscle 2) resynthesis of ATP and CP in the exercising muscles 3) conversion of lactate to glycogen 4) repletion of glycogen 5) response to catecholamine release 6) oxidation of fat 7) turnover of substrate cycles 8) elevation of body temperature 9) compensatory increased protein synthesis 10) restoration of ionic homeostasis 11) thermic effect of food and 12) elevated physiological functioning (Bahr et al 1987; B. Bahr and Sejersted 1991; Bangsbo et al 1990; Bangsbo et al 1991; Bielinski et al 1985; Medbo et al 1988; Roth et al 1988; Stainsby and Barclay 1970). EPOC estimates the total energy required to restore homeostasis. Even when controlling for catecholamines and temperature, less than one third of the first three minutes of EPOC can be traced and accounted for by the major oxygen requiring processes, which are the following 1) ATP and CP resynthesis 2) reloading of haemoglobin and myoglobin and 3) glycogen resynthesis (Bangsbo et al 1989). Therefore, EPOC is technologically limited to estimating the magnitude of homeostatic disturbance (Sedlock et al 1989).

Non-steady workrate supramaximal exercises do not allow a linear extrapolation of $O_2$ demand and subsequent calculation of $O_2$ deficit. Therefore, EPOC must be used as a measure of recovery from metabolic stress. Although EPOC reflects more than anaerobic catabolism (Bangsbo et al 1989; Gaesser and Brooks 1984; Medbo et al 1988; Stainsby and Barclay 1970) it is a sensitive measure of performance improvements and of the capacity to perform exhaustive exercise of short duration (Hermansen 1969). Hermansen

was able, using $O_2$ debt, to distinguish a highly trained athlete's 100M, 200M and 400M performances and was able to rank highly trained, trained, and untrained subjects according to a standardized short duration performance. Since Hermansen's study, the term $O_2$ debt has been replaced with the term EPOC to avoid any implication of causality when explaining the elevated oxygen consumption. The change is purely in nomenclature and not in method of analysis (Gaesser and Brooks 1984). Thus, the value of the EPOC measurement lies in its ability to metabolically compare non-steady state exercises such as isokinetic and free sprinting.

The metabolic rate during the terminal stage of exercise carries over into recovery. Sedlock et al (1989) who used aerobic exercise, suggests that the initial metabolic rate in recovery is an index of the intensity of the exercise. However, the metabolic rate as measured by $O_2$ consumption reaches a ceiling at the $VO_{2Max}$. As a result, supramaximal efforts of differing intensities will produce similar initial recovery $VO_2$'s. $EPOC_{1MIN}$ after isokinetic and both free sprints were the same, demonstrating only that the three conditions maximally taxed the aerobic system for the given intermittent protocol. Therefore, the three forms of sprinting used in this experiment can not be metabolically classified by $EPOC_{1MIN}$.

Gore and Withers (1990) using a repeated measures 3 X 3 factorial (30%, 50%, 70% $VO_{2MAX}$ * 20, 50, 80 minutes), intensity by duration, design found that intensity explained five times the variance in EPOC volume that duration or the intensity duration interaction did. The $EPOC_{30MIN}$ volume in this study predominantly reflects the intensity of each condition given the small duration range (25 and 50 seconds). The EPOC differences found between 5 and 10 free sprints are surprisingly small, 9.09 and 9.88 litres respectively; however, closer inspection reveals strikingly similar intensities

demonstrated by the average distance of each repetition, 36.77 and 34.79 metres respectively. These results support the findings of Gore and Withers that exercise intensity has an overwhelming influence over duration.

The total distances attained by the free sprint protocols most closely reflect a 200 and 400 metre sprint, not taking the recovery periods into account. Hermansen (1969) in a repeated $O_2$ debt comparison of a 200 (22.2 seconds) and 400 metre (48.1 seconds) sprint found an $EPOC_{8MIN}$ differential of 1 litre. The $EPOC_{8MIN}$ differential of the 5 and 10 free sprints of this study was 0.20 litres. The slightly higher intensity of 5 repetitions reduces the effect of the exercise duration of 10 repetitions.

5.3 Blood Lactate

Blood lactate is the product of one process, albeit a net resultant of many factors. Generally, the net release of lactate to the blood is a function of the mass of muscle recruited, its fiber type composition and its intensity of activation (Stainsby and Brooks 1990). Long duration submaximal exercise bouts eventually produce an equilibrium between lactate production and removal, resulting in a large total flux of lactate which is not accurately reflected by the blood lactate concentration. Alternately, short duration supramaximal exercise bouts produce increased blood lactate concentrations due to the type IIB muscle fiber recruitment but also due to the cessation of exercise and subsequent restriction of lactate clearance mechanisms by the reduction in blood flow and $VO_2$ (Jacobs 1986; Stainsby and Brooks 1990).

The supramaximal blood lactate concentration reflects the production of lactate whereas the submaximal blood lactate concentration reflects the capacity of the blood lactate clearance mechanisms (Astrand et al 1986; Gollnick et al 1986; Jacobs 1986; Stainsby and

Brooks 1990). Under the protocols used in this study, the factors which usually obscure the findings based on peak blood lactates have largely been removed using a repeated measures supramaximal protocol. Subsequently, the supramaximal peak blood lactates will predominantly indicate the degree of glycolysis that occured in the active musculature. The larger lactate response of isokinetic sprinting over free sprinting as indicated by the peak blood lactate values (Table 6) illustrates that the greater intensity demanded by isokinetic sprinting stimulates a greater turnover of glycolysis.

Similar to EPOC, peak blood lactate data limits researchers to speculate on its underlying mechanisms. Lactate appearance in the blood is the net result of its production, metabolism and transport from the cells and removal from the blood. Consequently the concentration of lactate in the blood does not necessarily accurately reflect the intracellular glycolytic activity (Stainsby and Brooks 1990) However, the volume of lactate has been estimated from its concentration in the blood. This requires estimating the distribution volume for lactate and the exercising muscle mass which invokes considerable controversy (Astrand et al 1986).

Despite the reported limitations of using blood lactate concentrations it has been shown that, the repeated measures design, the exclusive use of sprinting, and the short duration and range of exercise (25 - 50 seconds) used in this study, minimizes the fluxing of lactate. Thus the concentration of blood lactate is indicative of the actual volume of lactate released to the blood. It is assumed from the similarity of all the exercise protocols that the same accumulation and clearance mechanisms are at work; thereby, facilitating a direct comparison of conditions using the peak blood lactate concentrations. In a literature review Jacobs (1986) concluded that the lactate response to supramaximal exercise was a sensitive indicator of adaptation to sprint training and went on to state that it correlated to

supramaximal performance. Consequently, peak lactate concentration after supramaximal exercise of 1 minute duration is often used as a reliable index of glycolytic capacity. This is provided that the conditions following the exercise are standardized (Jacobs 1986).

5.4 EPOC and Blood Lactate Relationship

Since Hermansen's study in 1969, there has been a paucity of research literature pertaining to the measurement of EPOC and peak blood lactate after short duration, maximal and exhaustive exercises. Bahr et al (1992) studied intensities at 108% of $VO_{2MAX}$ which elicited an $EPOC_{1h}$ of 7.8 ± 0.7 l and a peak blood lactate value of 9.98 ± 1.10 $mmol \cdot l^{-1}$ after 3 x 2min bouts of stationary cycling. Comparatively, the present study demonstrates the overwhelming influence of intensity over duration on EPOC and peak blood lactate. The 2 Min AST produced an $EPOC_{30MIN}$ of 15.16 ± 2.59 l and a peak blood lactate of 14.83 ± 1.21 $mmol \cdot l^{-1}$. The least taxing (5 Free) condition evoked an $EPOC_{30MIN}$ of 9.09 ± 2.5 l and a peak blood lactate of 9.83 ± 3.09 $mmol \cdot l^{-1}$.

The correlation (r = +0.87) between $EPOC_{30MIN}$ and peak blood lactate parallels the finding of Bahr et al (1992). Bahr found a significant relationship (r = +0.86) between the mean increase in blood lactate concentration over the first hour post-exercise and $EPOC_{1h}$. The correlations by no means imply that the amount of lactate produced directly dictates the EPOC volume, rather these variables are spuriously related by the effect of intensity. Both EPOC and peak blood lactate concentration are products of the effect of intensity of exercise. Correspondingly, induced lactacidemia via circulatory occlusion, which does not reflect the intensity of exercise, does not result in an elevation of EPOC. The predominant removal of lactate during exercise recovery through oxidation does not allow lactate to critically contribute to EPOC (Roth et al 1988). Despite the

close relationship, lactate contributes to only a small part of EPOC (Bangsbo et al 1989; Bangsbo et al 1990).

Five free sprint repetitions demand a larger proportion of ATP from the ATP-CP cycle than 10 free sprints. The supply of CP gets progressively more depleted with the performance of each repetition (Kaczkowski 1982). Subsequently, glycolytically derived ATP will be relied on. This helps to explain the larger lactate concentration produced by 10 free sprints, while the findings of Bangsbo et al (1989, 1990) serve to explain the non-compensatory increase in EPOC.

Maintainenance of the same free sprinting intensity of exercise requires a compensatory increase in the cycling of the glycolytic and Kreb's cycles. The increased glycolytic activity results in a greater but not significantly different peak blood lactate response to 10 free repetitions over 5 free repetitions. The predominantly oxidative fate of lactate results in an insignificantly larger 10 free repetition EPOC. Alternately, 5 isokinetic repetitions are more intense and produce a greater homeostatic disturbance, resulting in both a larger peak lactate and EPOC response.

### 5.5 Anaerobic Power Master

Medbo and Tabata (1989) found that anaerobic ATP generation is highest during the initial 5 - 10 seconds of a Wingate test; therefore, tests of peak power should last for 10 seconds or less. These findings present the first repetition of the APM test as an ideal index of peak power generation. Particularly appealing is the fact that the test is performed by sprinting and not cycling like the Wingate test. This form of peak power assessment could be applicable to a wider range of sports. The APM can be easily adapted to the environment of any linear motion sports performance. Besides sprinting, Rhodes et al (1991) used the APM in the analysis of power output for ice skating. The

mean power output found by Rhodes and Roberts (1992) on the APM agrees with the mean power (1001.3 ± 128.6 watts) produced in the first repetition of the present study.

*5.6 Work*

The comparison of the 5 and 10 free sprinting protocols via distance sprinted is a valid one by virtue of the similarity of the two conditions. However, they do not compare to isokinetic or treadmill sprinting by similar performance variables. Also the isokinetic and treadmill tests do not compare to each other by wattage. The wattage produced running on a graded treadmill is a function of the subjects mass and the vertical displacement. The wattage is based solely on the physical work performed; therefore, the metabolic cost is only partially reflected. Alternately, the APM produces a strict horizontal resisting force, the wattage of which wholly reflects the energy output of the individual. Consequently, the wattage calculated by the 2 minute AST protocol (398.8 ± 52.8) is much smaller than that of the 5 repetition APM protocol (4362.9 ± 508.6). Therefore, to compare the non-steady state conditions, a common objective measure of metabolic demand (EPOC and peak blood lactate) must be employed.

*5.7 Conclusions*

In conclusion, this study demonstrates that there is a significant $EPOC_{30MIN}$ and peak blood lactate response between 5 isokinetic sprint repetitions and 5 free sprint repetitions and no significant $EPOC_{30MIN}$ and peak blood lactate response between 5 isokinetic sprint repetitions and 10 free sprint repetitions.

Essentially the study shows the metabolic value of isokinetic tethered sprinting. More physical work is performed in less time compared to free sprinting. The isokinetic device

is specific to the acceleration of non-resisted performance; however, it is not applicable to training maximal non-resisted velocities. Therefore, the value of the Anaerobic Power Master is as a supplement to sprint training acceleration and training the glycolytic pathways. However, to distinguish the true value of isokinetic sprinting a controlled longitudinal training study using non-sprint trained individuals must be employed.

Also the study has proven the value of employing a portable oxygen analyzer to measure EPOC and to compare non-steady state exercises. This improved technology facilitates the use of data collection from "field" performances. The applicability of research to actual sport performance is thereby enhanced.

## RECOMENDATIONS

An interesting hypothesis based on the rationale of this study is that the effect of intensity, duration, and the intensity duration interaction on EPOC may be maximal for exhaustive exercise which elicits a peak $O_2$ deficit as described by Medbo (1988). It would be interesting to test the hypothesis that the percentage of maximal $O_2$ deficit performed for an exhaustive exercise may correlate to the subsequent percentage of maximal EPOC.

Much of the literature on EPOC focuses on acounting for the processes which produce post-exercise oxygen consumption in excess of RMR. The findings are consistently inconclusive and based on widely differing exercise protocols. The direction that EPOC research has taken, appears to have reached a deadlock with the state of technology. Further, attempts to account for the occurrence of EPOC do not appear to be advancing the body of knowledge. Until further technological advances facilitate objective tracing of post-exercise oxygen consuming processes, EPOC research in the mean time would yield greater returns by applying the concept in its present state of understanding to establish its value as a research variable. It has been demonstrated that significance of EPOC to weight loss is negligible (Bahr and Sejersted 1991); therefore, it may prove valuable in researching the vastly untouched realm of non-steady workrate ultra-supramaximal exercise, which does not lend itself to $O_2$ deficit determination.

# APPENDIX

**Table 10: Individual Subject Descriptive Data**

| Subject | Age (years) | Height (cm) | Weight (kg) | RMR (litres/min) | RMR 30 minute volume (litres) |
|---|---|---|---|---|---|
| **1** | 26 | 180.80 | 83.30 | 0.22 | 6.60 |
| **2** | 27 | 186.70 | 82.10 | 0.22 | 6.60 |
| **3** | 20 | 180.30 | 81.40 | 0.25 | 7.50 |
| **4** | 22 | 178.70 | 70.60 | 0.28 | 8.40 |
| **5** | 26 | 183.90 | 74.80 | 0.24 | 7.20 |
| **6** | 30 | 171.50 | 66.00 | 0.25 | 7.50 |
| **7** | 27 | 168.90 | 69.10 | 0.25 | 7.50 |
| **8** | 20 | 180.70 | 71.70 | 0.26 | 7.80 |

**Table 11: Individual 2 Minute AST Data; Test 5**

| Subject | % Grade | Duration (seconds) | Total Watts | Watts /Kg | 1 Min POC (litres) | 30 Min POC (litres) | 30 Min EPOC (litres) | Peak La (mmol/L) |
|---|---|---|---|---|---|---|---|---|
| **1** | 16.00 | 119 | 459.88 | 5.54 | 2.94 | 22.72 | 16.12 | 14.56 |
| **2** | 13.00 | 130 | 369.68 | 4.51 | 3.61 | 26.16 | 19.56 | N/A |
| **3** | 15.25 | 120 | 427.92 | 5.28 | 3.35 | 24.65 | 17.15 | 16.70 |
| **4** | 16.50 | 122 | 405.57 | 5.71 | 3.43 | 22.92 | 14.52 | 15.22 |
| **5** | 16.50 | 117 | 428.42 | 5.71 | 3.18 | 21.73 | 14.53 | 14.54 |
| **6** | 17.00 | 117 | 388.33 | 5.88 | 3.47 | 20.38 | 12.88 | 13.76 |
| 7 | 12.00 | 130 | 287.26 | 4.16 | 2.78 | 18.57 | 11.07 | 13.18 |
| **8** | 17.00 | 121 | 423.63 | 5.88 | 3.45 | 23.28 | 15.48 | 15.86 |

**Table 12: Individual 5 Repetition APM Data; Test 6**

| Subject | Total Watts | Watts/Kg | 1 Min POC (litres) | 30 Min POC (litres) | 30 Min EPOC (litres) | Peak La (mmol/L) |
|---|---|---|---|---|---|---|
| **1** | 5152.00 | 62.07 | 2.71 | 18.28 | 11.68 | 12.66 |
| **2** | 4447.00 | 54.23 | 3.48 | 20.44 | 13.84 | N/A |
| **3** | 4956.00 | 61.19 | 2.53 | 21.72 | 14.22 | 16.00 |
| **4** | 3755.00 | 52.89 | 2.79 | 22.46 | 14.06 | 12.56 |
| **5** | 4075.00 | 54.33 | 2.41 | 15.40 | 8.20 | 9.78 |
| **6** | 3799.00 | 57.56 | 2.53 | 16.96 | 9.46 | 12.68 |
| 7 | 4188.00 | 60.70 | 2.04 | 14.89 | 7.39 | 11.46 |
| **8** | 4531.00 | 62.93 | 2.98 | 19.97 | 12.17 | 14.27 |

**Table 13: Individual 10 Repetition Free Sprint Data; Test 7**

| Subject | Total Distance (metres) | 1 Min POC (litres) | 30 Min POC (litres) | 30 Min EPOC (litres) | Peak La (mmol/L) |
|---|---|---|---|---|---|
| **1** | 361.20 | 2.64 | 14.33 | 7.73 | 9.28 |
| **2** | 340.15 | 2.60 | 17.06 | 10.46 | N/A |
| **3** | 356.50 | 3.10 | 22.78 | 15.28 | 14.88 |
| **4** | 331.40 | 2.35 | 17.50 | 9.10 | 12.06 |
| **5** | 341.35 | 2.16 | 14.57 | 7.37 | 9.12 |
| **6** | 369.30 | 2.53 | 17.85 | 10.35 | 11.24 |
| **7** | 317.25 | 2.09 | 14.34 | 6.84 | 9.42 |
| **8** | 365.65 | 2.68 | 19.73 | 11.93 | 12.72 |

**Table 14: Individual 5 Repetition Free Sprint Data; Test 8**

| Subject | Total Distance (metres) | 1 Min POC (litres) | 30 Min POC (litres) | 30 Min EPOC (litres) | Peak La (mmol/L) |
|---|---|---|---|---|---|
| **1** | 200.65 | 2.95 | 15.47 | 8.87 | 8.00 |
| **2** | 164.75 | 3.04 | 18.49 | 11.89 | N/A |
| **3** | 185.20 | 2.56 | 18.85 | 11.35 | 16.38 |
| **4** | 172.30 | 1.86 | 16.27 | 7.87 | 8.00 |
| **5** | 175.55 | 2.35 | 13.58 | 6.38 | 8.04 |
| **6** | 187.40 | 2.35 | 16.13 | 8.63 | 9.28 |
| 7 | 178.80 | 2.51 | 13.06 | 5.56 | 8.14 |
| **8** | 206.30 | 2.79 | 19.96 | 12.16 | 10.96 |

**Table 15: Individual Proportion of AST Peak La and $EPOC_{30Min}$ Means**

| Subject | 5 Rep APM | | 10 Rep Free | | 5 Rep Free | |
|---|---|---|---|---|---|---|
| | $EPOC_{30Min}$ | Peak La | $EPOC_{30Min}$ | Peak La | $EPOC_{30Min}$ | Peak La |
| **1** | 72.5% | 87.0% | 50.0% | 63.7% | 55.0% | 54.9% |
| **2** | 70.8% | N/A | 53.5% | N/A | 60.8% | N/A |
| **3** | 82.9% | 95.8% | 90.8% | 89.1% | 66.2% | 98.1% |
| **4** | 96.8% | 82.5% | 62.7% | 79.2% | 54.2% | 52.6% |
| **5** | 56.4% | 67.3% | 50.7% | 62.7% | 43.9% | 55.3% |
| **6** | 73.4% | 92.2% | 80.4% | 81.7% | 67.0% | 67.4% |
| **7** | 66.8% | 86.9% | 61.8% | 71.5% | 50.2% | 61.8% |
| **8** | 78.6% | 90.0% | 77.1% | 80.2% | 78.6% | 69.1% |

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