

RELATIONSHIP OF EXCESS POST-EXERCISE OXYGEN CONSUMPTION
TO VO₂MAX AND RECOVERY RATE

by

ANITA THERESA GITTO
B.Phed., Brock University, 1992

A THESIS SUBMITTED IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
MASTER OF HUMAN KINETICS

in

THE FACULTY OF GRADUATE STUDIES
School of Human Kinetics

We accept this thesis as conforming
to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

October 1995

© Anita Theresa Gitto, 1995

In presenting this thesis in partial fulfilment of the requirements for an advanced degree at the University of British Columbia, I agree that the Library shall make it freely available for reference and study. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by the head of my department or by his or her representatives. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Department of HUMAN KINETICS

The University of British Columbia
Vancouver, Canada

Date APRIL 26 1996

ABSTRACT

The purpose of this study was to examine (i) the relationship between Excess Post-Exercise Oxygen Consumption (EPOC), peak blood lactate [BLa], and a measure of the fast and slow components of recovery (τ_1 and τ_2) and aerobic capacity ($\text{VO}_{2\text{max}}$) using three different supramaximal treadmill tests, and (ii) the effects of varying intensity and duration of supramaximal work on EPOC. Twelve males (mean: age=23.9 y, ht=183.7 cm, wt=82.2 kg) performed a $\text{VO}_{2\text{max}}$ and three anaerobic speed tests (ASTs). The ASTs represented a controlled intensity test (20% grade), a fixed duration test (2 min.), and a fixed intensity and duration test (15%-1 min.). No significant relationships were found between $\text{VO}_{2\text{max}}$, EPOC, rate (τ_1 and τ_2), or peak blood lactate. However, significant relationships were evident between anaerobic capacity and EPOC 20% ($r=.74$, $p<.01$) and EPOC 2 min ($r=.62$, $p<.05$). ANOVA revealed a significant difference for EPOC (15%- 1 min; 7.56 l) with EPOC (2 min; 9.29 l) and EPOC (20%; 9.04 l). [BLa] for EPOC (2 min; 15.74 $\text{mmol}\cdot\text{l}^{-1}$) was significantly different ($p<.05$) from EPOC (20%; 13.62 $\text{mmol}\cdot\text{l}^{-1}$) and EPOC (15%- 1 min; 13.01 $\text{mmol}\cdot\text{l}^{-1}$). No significant differences were evident between τ_1 and τ_2 across the 3 ASTs. These findings suggest that the rate and magnitude of recovery from supramaximal work are independent of $\text{VO}_{2\text{max}}$, however, magnitude was dependent on anaerobic capacity. Recovery rates were similar for the same subject across varying degrees of anaerobic work, indicating a fixed rate of recovery despite changes in exercise condition of a supramaximal nature. This demonstrates the effects of both intensity and duration on EPOC. Finally, the absence of a lactate-EPOC relationship does not lend support for lactate as a major contributor to the presence of an elevated oxygen consumption post-exercise.

TABLE OF CONTENTS

Abstract	ii
Table of Contents	iii
List of Tables	v
List of Figures	vi
Acknowledgements	vii
INTRODUCTION	1
1.1 Statement of the Problem	2
1.2 General Hypotheses	2
1.3 Rationale	3
1.4 Significance of the Study	3
1.5 Definitions	4
1.6 Delimitations	4
1.7 Limitations	5
REVIEW OF LITERATURE	6
2.1 The Fate of Lactate	6
2.2 The Importance of Lactate Removal	10
2.3 Ability to Remove Lactate	11
2.4 Metabolic Basis of EPOC	13
2.5 Effects of Intensity and Duration on EPOC	16
2.6 Oxygen Uptake During Exercise and Recovery	18
METHODS	20
3.1 Subjects	20
3.2 Pre-Experimental Protocol	20
3.3 Research Design	20
3.4 Experimental Protocol and Procedures	20
3.4.1 Overview of Procedures	20
3.4.2 VO ₂ max Test	21
3.4.3 Anaerobic Speed Tests	21
3.5 Data Collection	22
3.5.1 Oxygen Consumption	22
3.5.2 Resting Metabolic Rate	22
3.5.3 Maximum Oxygen Consumption	22
3.5.4 Anaerobic Capacity	22
3.5.5 EPOC	23
3.5.6 Maximum EPOC	23
3.5.7 Skinfolds	23
3.5.8 Girths	24
3.5.9 Heart Rate	24
3.5.10 Peak Blood Lactate	24
3.6 Data Analysis	25
3.6.1 Recovery Rate	25
3.6.2 EPOC	29
3.7 Statistical Analysis	29
3.7.1 ANOVA	29
3.7.2 Correlation	29
3.8 Specific Hypotheses	30
RESULTS	31
4.1 Descriptive Data	31
4.2 Recovery Data	32
4.2.1 EPOC	32

TABLE OF CONTENTS

4.2.2	Blood lactate	33
4.2.3	Rate	33
4.3	Relationships	39
4.3.1	EPOC	39
4.3.2	Recovery Rate	39
4.3.3	Blood Lactate	40
4.3.4	AST Performance	41
4.4	Results of Hypotheses	44
DISCUSSION		45
5.1	Subjects	45
5.2	Metabolic Basis of EPOC	46
5.3	Comparison of EPOC	48
5.4	Blood Lactate	52
5.5	Rate of Recovery	54
5.6	The Recovery-Training Status Relationship	55
CONCLUSIONS		60
RECOMMENDATIONS		61
APPENDIX		62
REFERENCES		67

LIST OF TABLES

Table 1:	Physical and Physiological Data
Table 2:	Means and Standard Deviations of AST Data
Table 3:	Post Hoc Comparison of EPOC Data
Table 4:	Post Hoc Comparison of the Slow Component EPOC Data
Table 5:	Post Hoc Comparison of Blood Lactate Data
Table 6:	Correlation Matrix
Table 7:	Individual and Mean EPOC Data
Table 8:	Individual and Mean EPOC Data for the Fast and Slow Components
Table 9:	Individual and Mean Peak Blood Lactate Data
Table 10:	Individual and Mean τ_1 Data
Table 11:	Individual and Mean τ_2 Data

LIST OF FIGURES

- Figure 1: Endpoints of Lactate Metabolism
- Figure 2: The "Lactate Shuttle"
- Figure 3: Scatter Plot of a Recovery Curve
- Figure 4: Scatter Plot of the Fast Component of a Recovery Curve
- Figure 5: Scatter Plot of the Slow Component of a Recovery Curve
- Figure 6: Bar Graph of Mean EPOC and Peak Blood Lactate
- Figure 7: Bar Graph of Mean Slow and Fast Components of EPOC
- Figure 8: Area Graph of Mean Post-Exercise Oxygen Consumption
- Figure 9: Scatter Plot of VO_2max scores
- Figure 10: Bar Graph of AST Performance

ACKNOWLEDGEMENTS

The author wishes to extend sincere gratitude to her committee members Dr. A. Martin, Dr. E.C. Rhodes, Dr. D.C. McKenzie, and Dr. J. Taunton for devoting valuable time and effort to this research, while exhibiting tremendous patience and understanding. Each of them overstepped their responsibilities and obligations to help a desperate graduate student meet her deadlines. Further appreciation is extended to Dusan Benicky for lending his time, knowledge, and technical expertise not only during this research, but over the last three years. The author would also like to thank the twelve athletes who allowed themselves to be subjected to numerous bouts of intense exercise, all in the name of science. Most importantly, the author would like to thank her friends and family for their motivation and support during the time of this research.

INTRODUCTION

Hill and Lupton first examined recovery oxygen consumption in the 1920's, and proposed their "Oxygen Debt" hypothesis. They hypothesized that the elevated oxygen consumption following exercise was necessary for the repayment of the deficit in oxygen consumption incurred at the start of exercise. They concluded that approximately 80% of lactate formed during contraction, was returned to glycogen during recovery with the remaining lactate being oxidized. In 1933, Margaria et al. added to the hypothesis by splitting the debt into two components: a fast (alactacid) component and a slow (lactacid) component. The fast portion of the debt was supposedly representative of the restoration of ATP and CP stores, whereas the slow portion was involved in the oxidation of lactate. This prompted more research in the area and subsequently more confusion.

Finally, Gaesser and Brooks (1984), provided the first extensive review paper on the phenomenon. Here they provided evidence that indicated a different fate of lactate. It is now evident that the majority of the lactate is oxidized, whereas glycogen may be the result of <20% of the lactate formed during contraction. Gaesser and Brooks (1984) also introduced the term "Excess Post-Exercise Oxygen Consumption", or EPOC. This term is most appropriate as, unlike "oxygen debt" and "recovery O₂", EPOC does not imply any causality. Thus, the term EPOC has been adopted and the quest for its causative mechanisms continues. Despite the debate surrounding the metabolic basis of EPOC, there is little dispute over the existence of EPOC and its validity as a measure of homeostatic disturbance. Thus, EPOC serves as a sound measurement tool of overall post-exercise recovery metabolism.

EPOC has been shown to be influenced by training status, exercise intensity, exercise duration, and the thermic effect of food. This manipulation of EPOC is of special interest to researchers looking for ways to control body mass, though whether the extra energy expenditure has any significant impact on energy balance has yet to be determined. Research investigating the effects of training status on the recovery rate of EPOC have produced inconclusive results and need clarification. The various protocols used in these studies may be partly responsible for these discrepancies.

1.1 Statement of the Problem

The purpose of this study was to determine if aerobic power is significantly correlated with the rate and magnitude of recovery for athletes involved in intermittent type sports following separate conditions of short duration, high intensity exercise.

A second purpose of this study was to determine whether changes in the intensity and duration of the exercise conditions will elicit significant differences in the rate and magnitude of each recovery period.

1.2 General Hypotheses

It is hypothesized that subjects with a greater VO_2max will demonstrate a more rapid rate of recovery than less aerobically fit subjects. It is also hypothesized that EPOC will be dependent on the intensity and duration of the work bouts.

1.3 Rationale

Presently the literature pertaining to the relationship between aerobic power and recovery is sparse and inconclusive. While some studies have shown a relationship between aerobic power and recovery (Henry and Berg, 1950; Hagberg et al., 1980; Elliot et al., 1988; Frey et al., 1993), other studies have not (Freedman-Akabas et al., 1985; Brehm and Gutin, 1986; Kaminsky et al., 1987; Chad and Quigley, 1991). This discrepancy may be attributed to the different research designs and protocols used. VO_2max was only correlated with duration of EPOC (Elliot et al., 1988), rather than the actual rate of recovery in determining time to homeostasis. Consequently, a sound research design with a more sophisticated analysis of results is needed in order to determine the true nature of the relationship.

Finally, the investigation of recovery from supramaximal exercise is limited. In fact, most studies have involved intensities less than 75% of VO_2max . More research using supramaximal protocols would provide more information to the athletes for which such intense work bouts are required. Since recovery for athletes involved in intermittent sport is important for maintaining performance and delaying fatigue, any relationship to VO_2max would provide support for intense aerobic training.

1.4 Significance of the Study

Intermittent sports require that athletes recover during rest intervals in order to continue performing. If a greater aerobic base increases the rate of recovery, this could provide

useful information to the coach and trainers. Better trained athletes may have a more significant role in the later stages of the game when fatigue is approaching.

1.5 Definitions

O₂ Deficit: the oxygen uptake debt incurred during exercise by the disparity between accumulated O₂ demand and accumulated O₂ uptake.

Supramaximal Exercise: exercise requiring a rate of energy release exceeding the maximal O₂ uptake (Medbo et al., 1988).

Anaerobic Speed Test (AST): a graded treadmill run to exhaustion.

Excess Post-Exercise Oxygen Consumption (EPOC): the summed volume of all O₂ derived processes in excess of resting VO₂ values that restore metabolic homeostasis from disturbances not only in the working muscles but all organs and tissues of the body (Roth et al., 1988). EPOC is more than mere repayment of the O₂ deficit (Gore and Withers, 1989).

1.6 Delimitations

This study is delimited to:

- 1) male athletes trained for intermittent sports between the ages of 20-34 yrs
- 2) the methodology and work bouts applied to determine and produce the EPOC and blood lactate concentrations.

1.7 Limitations

This study is limited by:

- 1) the present understanding of EPOC
- 2) the interpretation of recovery variables
- 3) the perception of fatigue experienced by each subject

REVIEW OF LITERATURE

2.1 The Fate of Lactate After Exercise

In earlier years, muscle glycogen was identified by Meyerhof (1920), as the precursor of lactic acid (Gaesser and Brooks, 1984). It was then concluded that the majority of lactate formed during contraction was returned to glycogen in recovery (Gaesser and Brooks, 1984). In recent years however, this has been proven untrue. Tracer studies performed on animals indicate oxidation to be the major fate of lactate during exercise and the subsequent recovery period (Gaesser and Brooks, 1984; Brooks, 1986; Mazzeo et al., 1986; Stainsby and Brooks, 1990). While under resting conditions, tracer-measured blood lactate disposal underestimates 'true' lactate turnover by approximately 20%. During exercise, when cardiac output and arterial lactate concentrations rise, the error in underestimating blood lactate turnover by tracer methods becomes only a few percent (Stainsby and Brooks, 1990). Figure 1 depicts the endpoints of lactate metabolism. According to Gaesser and Brooks (1984), and Brooks (1986), 55-70% of lactate is oxidized.

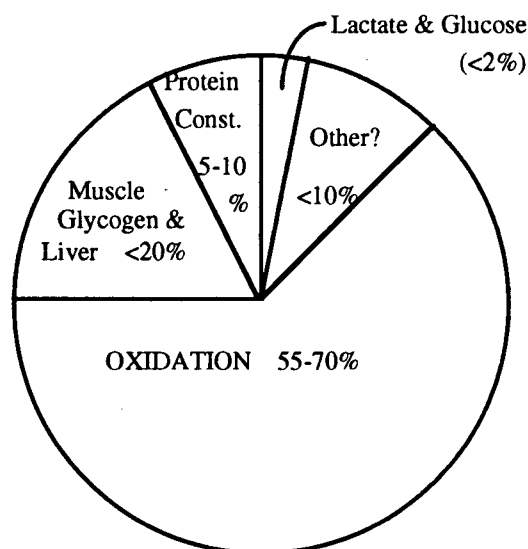


Figure 1: Endpoints of lactate metabolism following prolonged exercise to exhaustion in rats (Brooks, 1986)

The production of lactic acid occurs in working skeletal muscles when the energy demands cannot be supplied totally via oxidative processes, while the removal of lactic acid occurs in the liver, heart, skeletal muscle, brain, and kidney (Cortes et al., 1988). Lactate metabolism seems to be geared to the metabolic rate of muscle since the lactic acid removal is slowest at rest and most rapid during exercise (Bonen and Belcastro, 1976; Mazzeo et al., 1986). It was shown that while ~50% of lactate was oxidized during rest recovery, 80% was oxidized during active recovery which appears as CO₂ in the venous blood (Mazzeo et al., 1986). In fact, the results of Brooks (1986) indicate greater rates of turnover and oxidation for lactate than for glucose during exercise, and may play as great a role, or greater, in providing oxidizable substrate during exercise. During steady state cycling at 40% VO₂max, lactate turnover has been shown to exceed glucose turnover by 32% (Stanley et al., 1984).

During exercise, lactic acid removal appears to be primarily accomplished through organs in which blood flow increases (i.e. the heart and skeletal muscle) (Boileau et al., 1983; McLoughlin et al., 1991; Stanley, 1991). Without adequate flow there would be an insufficient delivery of oxygen and substrate, and inadequate removal of the products of metabolism. Thus, an increase in blood flow in excess of metabolic requirements would speed the return of all processes to homeostatic levels (Cafarelli et al., 1990). Mazzeo et al. (1986) showed that a decline in lactate metabolic clearance rate during hard exercise was due to blood flow redistribution away from lactate removing areas (liver, kidney, skeletal muscle) toward areas of lactate production. Their results also allowed them to assume that an increased fraction of the muscle mass was utilized and that recruitment of fast-twitch glycolytic (white) skeletal fibers occurred. They go further on to suggest that during exercise within a specific muscle, lactate is produced to a greater extent by the fast-twitch glycolytic fibers, with local oxidation by the slow-twitch oxidative (red) fibers, without

lactate ever being released in the blood. This ability of human muscle to simultaneously produce and utilize lactate has been demonstrated by Jorfeldt (1970), through the injection of [U- ^{14}C]lactate into the brachial artery of human subjects and collecting forearm venous blood during forearm exercise.

As for the heart, it has been shown to take up lactate in proportion to the rate of lactate delivery to the myocardium both at rest and during exercise. Further, lactate may become the major substrate for myocardial oxidative metabolism in heavy exercise with elevated lactate levels ($>3\text{--}4\text{mmol}$) (Stanley, 1991). In fact, lactate has been shown to compete with free fatty acids as an energy substrate in the heart. It seems the presence of the heart-specific lactate dehydrogenase isozyme makes conditions favourable for the conversion of lactate to pyruvate and subsequent entry into the TCA cycle (Mazzeo, 1986). However, most research has shown contracting slow-twitch striated muscle tissue to possess the only respiratory capacity sufficient to oxidize large quantities of lactate formed (Bonen and Belcastro, 1976; Brooks, 1986; Mazzeo et al., 1986; Stanley, 1991). These studies indicate that slow-twitch striated muscle is the predominant site of lactate removal where lactate is metabolized in the Krebs Cycle via reversion to pyruvate. This is a most likely site in that the H form of LDH predominates in slow-twitch oxidative fibers (Bonen and Belcastro, 1976). While during exercise, 60-70% of the lactate entering the TCA cycle is directly oxidized to CO_2 (Gaesser and Brooks, 1984; Mazzeo et al., 1986), at rest, only about 33% of lactate entering the TCA cycle is completely oxidized while, the remainder is probably incorporated into other compounds with slower turnover times (Mazzeo et al., 1986). It has also been shown that inactive skeletal muscle takes up circulating lactate during exercise of other muscle groups (Brooks, 1986; McLoughlin et al., 1991). The passive muscle acts as a sink, returning lactate to the circulation as the arterial concentration falls (McLoughlin et al., 1991).

Lactate produced as a result of recruitment of type IIb fibers or because of the mechanics of the contractile process, diffuses toward and is transported into type I or IIa fibers, where lactate is oxidized (Baldwin et al., 1977). The more glycolytic fibers within a working muscle bed shuttle oxidizable substrate to the neighbouring cells with higher respiratory rates. This is part of the 'lactate shuttle' hypothesis proposed by Brooks (1986). Approximately half the lactate formed in a working muscle bed is released into venous circulation. This, together with a significant quantity of lactate removed from arterial circulation, is combusted within the muscle and appears as CO_2 in the venous blood as shown in Figure 2 (Brooks, 1986).

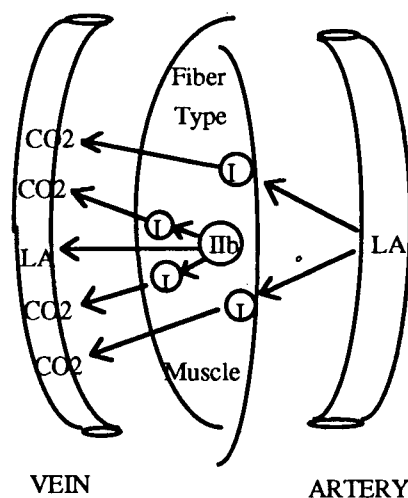


Figure 2: Illustration of the proposed "Lactate Shuttle"

Support for the lactate shuttle hypothesis comes from several sources. Combined, these sources support the following: lactate can be exchanged between muscle and blood, between blood and muscle, between inactive and active muscles, between active and inactive muscles, between active and active muscles, between blood and heart, between active muscle and liver, between liver and other tissues such as exercising muscle, between skin and blood, between intestine and portal blood, between portal blood and liver, and

along pH and concentration gradients within muscle tissue (Stainsby and Brooks, 1990; Brooks, 1991).

2.2 The Importance of Lactate Removal

It has been shown that the accumulation of lactic acid in the muscle during strenuous exercise may play a role in the development of fatigue since increased H^+ may interfere with the formation of cross bridges in the muscle cell by competing with calcium on the binding sites of troponin (Bonen and Belcastro, 1976; Tesch and Wright, 1983) thus, hindering muscle contraction. Lactic acid was also thought to retard the rate of glycolysis by inhibiting the activity of lactate dehydrogenase and phosphofructokinase (Belcastro and Bonen, 1975; Bonen and Belcastro, 1976). Research presently underway, however, is investigating the build up of inorganic phosphate (P_i) and the unavailability of glycogen as a substrate due to increased lactate concentrations, as promoters of fatigue as a result of intense exercise (Belcastro, 1992).

Much research in the past has also linked fatigue to decreased pH levels (Bond et al., 1991). Contrary to this belief, Sahlin et al. (1978) found that 20 minutes after exercise, muscle pH was not depressed ($pH=7.2$) at the same time intramuscular lactate was elevated. It has also recently been shown that decreased pH actually increases PFK activity, thus speeding up glycolysis (Belcastro, 1992). Therefore, whether pH is decreased or not, it does not seem to be associated with fatigue.

Fast twitch fibers are recruited to a large extent during intense exercise. Since lactate accumulates at a higher rate in fast twitch over slow twitch muscle fibers of exercised

muscles (Belcastro and Bonen, 1975; Tesch and Wright, 1983; Brooks, 1991) lactate formed in fast-twitch fibers may result in a greater inhibitory effect on the contractile machinery and a greater decrement in muscle force if the muscle is made up of a high percentage of fast-twitch fibers than a muscle with a high percentage of slow-twitch fibers (Tesch and Wright, 1983). A higher rate of blood lactate release to the blood stream may be expected from slow-twitch fibers due to a more developed capillary network, thus making them more efficient in oxidizing lactate. Tesch and Wright (1983) found that both fatigue and recovery of knee extensor muscles were found to correlate with the capillary density of that muscle. These results allowed them to suggest that recovery processes are influenced by the rate of lactate disappearance from the muscle. They do note however, that muscle force recovers at a faster rate than lactate is eliminated. Perhaps, this may explain why certain studies have shown no detriment in performance with increased lactate concentrations (Gisolfi et al., 1966; Weltman et al., 1982). These studies investigated isokinetic muscle function, and found no difference in maximal effort exercise following 20 minutes of recovery with a blood lactate concentration varying between 5-12mmol prior to the second work bout. There have been reports that increased blood lactate concentrations have had no effect on subsequent swimming performance (Seibers and McMurray, 1981). However, studies involving cycle ergometer work did show a detrimental effect on subsequent performance. In fact, Karlsson et al. (1975) reported a trend for decreased work output with elevated lactate concentrations.

2.3 Ability to Remove Lactate

Many researchers have determined that active recovery is more efficient than passive recovery in returning the body to homeostasis, especially in the removal of blood lactate

(Belcastro and Bonen, 1975; Bonen and Belcastro, 1976; 1977; McLellan and Skinner, 1982; Milesis et al., 1982; Boileau et al., 1983; Evans and Cureton, 1983; Watson and Hanley, 1986; Cortes et al., 1988). It is believed that exercise recovery aids circulation, thus preventing pooling of the blood in the working muscles and speeding recovery (Gisolfi et al., 1966; Bonen and Belcastro, 1976). This increased blood flow may also facilitate the transport of lactic acid to the removal sites (Bonen and Belcastro, 1976; Evans and Cureton, 1983; Mazzeo et al., 1986; McLoughlin et al., 1991). Also, since the oxidation of lactate is greater in slow twitch muscle fibers (Tesch and Wright, 1983), the recruitment of these fibers during active recovery would greatly increase the disappearance of lactate in comparison with passive recovery during which slow-twitch muscle fibers are less involved (Katch et al., 1978).

With regard to passive uptake of lactate from exercising muscle to inactive muscle, Buckley et al. (1993), have shown that the rates of lactate removal were not different in untrained and trained forearms. While it was calculated that the untrained and trained forearms disposed of approximately 83% and 91% respectively, these values were not significantly different. Thus, it would seem that inactive muscle, regardless of training status, has the ability to remove a significant amount of lactate. Buckley et al. (1993), calculated the amount to represent 11% of the disposed lactate. Given that skeletal muscle constitutes approximately 45% of body mass in non-obese humans (Poortmans et al., 1978), it can be suggested that resting skeletal muscle plays an important role in blood lactate removal from high intensity exercise.

A number of investigators have suggested that differences among individuals in the rate of blood lactate disappearance during active recovery may be related to their level of physical condition (Strom, 1949; Gisolfi et al., 1966; Davies et al., 1970; Bonen and Belcastro,

1976). Gisolfi et al. (1966), found a higher rate of blood lactate disappearance following a bout of exhaustive treadmill running in a distance runner than by a sprinter. They hypothesized that the greater lactate disappearance by the distance runner may have been due to his higher level of physical condition and to his ability to carry out a higher rate of recovery work. Davies et al. (1970, as cited by Evans and Cureton, 1983), reported a considerably greater rate of blood lactate disappearance following a heavy bout of cycle ergometer exercise in one subject with a high VO_2max compared with three other subjects who had much lower VO_2max values. Interestingly, Bonen and Belcastro (1976), noted that rates of blood lactate disappearance were significantly higher in a study having trained subjects compared with the other study in which subjects were less trained.

The rate of lactate removal has been associated with the individual's higher level of fitness due to increased metabolic efficiency (Gollnick et al., 1986). In support of this, it has been recently found that subjects with a greater ability to remove lactate during the recovery, increased their exercise blood lactate concentrations later and to a lesser extent. Also, the exercise intensities required to attain the same blood lactate were higher for trained than untrained subjects. Their results indicated that training causes a shift of the increase in blood lactate concentrations to higher work rates. However, it has been reported that physical conditioning does not affect the rate of lactate disappearance from the blood during passive recovery, or active recovery at a given absolute intensity that is considerably below the anaerobic threshold (Evans and Cureton, 1983).

2.4 Metabolic Basis of EPOC

Some of the metabolic processes believed to be responsible for EPOC are: replenishment of oxygen stores in the blood and muscle; resynthesis of ATP and creatine phosphate; lactate

removal; increased ventilation, circulation, and body temperature (Gaesser and Brooks, 1984; Bahr et al., 1992). These processes are believed to subside within 1-2 hours after exercise (Bahr and Sejersted, 1991). While lactate removal in EPOC is questionable, there has been a significant correlation found between the two: $r=.86$, $p<.0005$ (Bahr et al., 1992) and $r=.90$, $p<.05$ (Frey et al., 1993). Despite the close relationship between EPOC and the amount of lactate available for glycogen synthesis, some researchers conclude that a large part of total EPOC cannot be accounted for by lactate removal (Bahr et al., 1992). Since rectal temperatures and blood lactate levels have been shown to return to control levels in the presence of a prolonged EPOC component, Bahr and Sejersted (1992), suggest that processes other than those traditionally associated with the rapid components of EPOC are responsible for the prolonged increase in resting oxygen uptake. This prolonged EPOC component that can be detected for as much as 8-48 hours following certain types of exercise (Bahr et al., 1987; Maehlum et al., 1986; Gore and Withers, 1990). Increased catecholamine concentrations have been associated with this prolonged component. Mean increase in plasma norepinephrine over the first hour of recovery show a correlation with EPOC of $r=.70$, $p<.005$ (Bahr et al., 1992) and $r=.78$, $p<.05$ (Frey et al., 1993). Thus, some other mechanisms must come into play with these types of exercises causing an EPOC >1 hour.

Factors that can affect recovery metabolism are the subjects' state of training, food consumption, and the intensity and duration of exercise (Kaminsky et al., 1987). These factors may elicit certain processes which have been implicated in the prolonged EPOC component: increased rates of triglyceride-fatty acid (TG-FA) cycling, a potentiation of thermic effect of food (TEF), and the energy cost of increased glycogen resynthesis in muscle and liver (Bahr and Sejersted, 1991).

A more trained state of fitness has been known to increase recovery metabolism (Hagberg et al., 1980). Since poorer trained subjects have most often served as subjects in those studies documenting EPOC to occur in <1 hour, this is therefore not likely a cause of prolonged EPOC.

In regards to eating, a meal consumed 2 hours after cessation of exercise has demonstrated a marked increase in the rate of oxygen consumption in comparison to the increase observed in response to the same meal taken by the subject when no exercise has been taken (Maehlum et al., 1986). However, it has been shown that the prolonged EPOC component can occur in the fasted state (Bahr and Sejersted, 1991). Thus, the prolonged EPOC component observed in the fasted state must be caused by processes other than increased rates of glycogen resynthesis or a potentiation of TEF.

While duration of exercise affects EPOC, it has not been the determining factor for the prolonged EPOC component. It now seems quite evident that exercise intensity is the primary factor responsible (Maresh et al., 1992). Studies involving exercise of very high intensity, yet short duration (supramaximal), have recorded a prolonged EPOC component (Bahr et al., 1992). The majority of studies that have shown a prolonged EPOC have involved exercise intensities approximately 70% of VO_2max over durations ranging from 20 minutes to 80 minutes (Maresh et al., 1992).

Elevated concentrations of catecholamines are believed to be partly responsible for EPOC (Gaesser and Brooks, 1984). Interestingly, the concentrations of catecholamines do not increase during exercise unless the intensity of the activity exceeds 70% VO_2max (Shephard, 1984). Also, Mathews (1981), has shown that the greater the work intensity, the greater the increase in epinephrine and norepinephrine. Thus, while this cannot account

for EPOC following all exercise, it may become a factor in the prolonged EPOC component. Furthermore, catecholamines have been shown to increase the rate of the TG-FA cycle in white and brown adipose tissue (Challiss et al., 1984). Hence, it is suggested that during post-exercise periods in which the plasma levels of catecholamines are increased, a stimulation of the rate of some substrate cycles occurs, therefore demanding energy expenditure (Maehlum et al., 1986). This energy expenditure may account for a significant fraction of EPOC (Astrand et al., 1986; Wolfe et al., 1990). In support of this, Bahr et al. (1990), have found evidence to suggest that the energy cost of the increased TG-FA cycling rate may account for as much as half of the delayed component of EPOC.

Unfortunately, after many years of research and speculation, the metabolic processes causing EPOC are not completely understood (Bahr et al., 1992).

2.5 Effects of Exercise Intensity and Duration on EPOC

Many researchers have found evidence to suggest exercise intensity is the primary determinant of the magnitude of EPOC (Hagberg et al., 1980; Bahr et al., 1987; Sedlock et al., 1989; Gore and Withers, 1990; Bahr and Sejersted, 1991; Bahr et al., 1992; Smith and McNaughton, 1993; Frey et al., 1993). In fact, Gore and Withers (1990), have provided solid evidence to show that exercise intensity is about five times more important than either exercise duration, or the interaction of intensity and duration, in determining the magnitude of EPOC, although not all research agrees. Some investigators have determined duration to be the primary stimulus to elevated post-exercise oxygen consumption (Knuttgen, 1970; Chad and Wenger, 1985; 1988; Sedlock, 1991). However, in their research, work performed was equated, therefore the intensity variable was not manipulated, thus showing no effect. Interestingly, in a later study, Chad and Quigley (1991), have found exercise

intensity to also be an important factor in the long-term elevation of oxygen consumption after exercise. These results contradict previous findings by the principal author which found support for duration as being the primary manipulator (Chad and Wenger, 1985; 1988).

Other studies have found no effect of either duration or intensity on EPOC (Freedman-Akabas et al., 1985; Kaminsky et al., 1987; Maresh et al., 1992) although the durations and intensities used for comparison could be criticized. It has been shown that there is a threshold before exercise intensity affects the magnitude of the EPOC, as well as a duration threshold (Gore and Withers, 1990). Intensity thresholds have been reported to be $>50\%$ $\text{VO}_{2\text{max}}$ (Gore and Withers, 1990; 1990; Bahr and Sejersted, 1991) and $>65\%$ (Hagberg et al., 1980). At these intensities, a linear relationship is then observed between exercise duration and EPOC (Hagberg et al., 1980; Bahr et al., 1987; Gore and Withers, 1990; 1990; Frey et al., 1993). Even at supramaximal intensities ($>100\%$ $\text{VO}_{2\text{max}}$) a linear relationship between exercise duration and EPOC was found (Bahr et al., 1992). By contrast, this linear relationship was not seen by Chad and Quigley (1991). Their results indicated greater EPOC response for 30 minutes of cycling at 50% $\text{VO}_{2\text{max}}$ than at 70% $\text{VO}_{2\text{max}}$ for both trained and untrained subjects. Interestingly, when a constant 50 minute run at 70% $\text{VO}_{2\text{max}}$ was compared with intermittent activity of the same intensity (25 minute run - rest - 25 minute run), EPOC was significantly increased although magnitude of increase was rather small: 13.88kcal vs 6.39kcal (Kaminsky et al., 1990). Intermittent exercise in another study produced similar findings with the magnitude of the increase somewhat larger; 38.2 kcal versus 26.6 kcal (Ziegenfuss and Sedlock, 1992). Oddly, when intensity and duration were held constant, running versus cycling resulted in no significant differences in EPOC (Sedlock, 1992). Unfortunately this only adds to the

controversy surrounding EPOC, as running usually involves a significantly higher fat-free mass than cycling.

2.6 Oxygen Uptake During Exercise and Recovery as a Result of Training

Earlier research has provided evidence that physical training decreases oxygen uptake during exercise and recovery for the same absolute workload (Robinson and Harmon, 1941; Knehr et al., 1942; Crescitelli and Taylor, 1944; Henry and Berg, 1950; Cotes and Meade, 1959; Douglas and Becklake, 1968; Ekblom et al., 1968; Cunningham and Faulkner, 1969; Girandola and Katch, 1973; Hagberg et al., 1980). Conditioning was said to produce a more significant change in the amount of 'debt' reduction than in the rate of pay-off (Henry and Berg, 1950). However, Hagberg et al. (1980), have shown an increase in recovery rate even at the same relative workload, when amount of 'debt' was somewhat larger but not significantly different, suggesting that an individual's level of training may play an important role in the time course of recovery. However, subsequent research has been sparse and inconclusive.

Following the suggestion by Hagberg et al. (1980), only a few studies have attempted to test this. Brehm and Gutin (1986) and Freedman-Akabas et al. (1985) found no significant differences in recovery VO_2 response between fit and unfit subjects. However, these investigators utilized low exercise intensities which may not have been sufficient in obtaining a substantial EPOC (Medbo et al., 1988; Kaminsky et al., 1990). Kaminsky et al. (1987), used one group of subjects on two different exercise tasks and found no correlation with $\text{VO}_{2\text{max}}$ for either task. These intensities and durations used may also have been inadequate for obtaining sufficient EPOC. More recently, Chad and Quigley (1991) have found no significant difference in recovery from 30 minutes of cycle ergometer

exercise between trained and untrained cyclists. Since this was not the focus of their research, they did not discuss these findings in detail.

Interestingly, in 1988, Elliot et al. observed a correlation between maximal oxygen uptake ($r=-.7$, $p<.1$) and recovery energy expenditure. While their findings were not the focus of their research, the results have sparked renewed interest in the problem due to the moderately high correlation. The most recent research found that training status of the individual contributed to the magnitude of EPOC (Frey et al., 1993). EPOC was larger in trained subjects due to higher oxygen consumption, yet recovery time was shorter. Their results indicated that training status affects primarily the fast component and the initial phase of the slow component of EPOC. This has also been suggested by previous studies (Girandola and Katch, 1973; Hagberg et al., 1980). Also, no significant differences were found in EPOC after 20 minutes of recovery, however, differences did exist for plasma norepinephrine level, rectal temperature and blood lactate after the 20 minutes (Frey et al., 1993). This would suggest that these variables cannot account for EPOC entirely. Unfortunately, recent research has been unable to determine the true relationship, thus, further investigation is required.

METHODS

3.1 Subjects

Twelve trained males involved in intermittent sports between the ages of 19-34 years served as the subject pool for this study. The subjects selected represented a range of fitness levels to avoid a cluster effect in the statistical analyses. Approval was obtained from the University of British Columbia's Clinical Screening Committee for Research Involving Human Subjects.

3.2 Pre-Experimental Protocol

Prior to giving consent, subjects visited the lab to become informed and familiarized with the tests to be performed, the equipment being used, and the degree of exhaustion they could expect. Subjects completed a written informed consent form and a physical activity readiness questionnaire (PAR Q).

3.3 Research Design

The study employed a three condition (20% AST, 15%- 1 min. AST, 2 min AST) repeated measures design.

3.4 Experimental Protocol and Procedures

3.4.1 Overview of Procedures

Subjects were required to perform five treadmill tests, each on a separate day with a minimum of two rest days in between. All tests were administered in the J.M. Buchanan Exercise Science Lab at UBC. Subjects were instructed to refrain from eating during the two hour period prior to the testing. Anthropometric data was collected on the first testing day prior to the VO_2max test. The remaining tests were ASTs: 20% grade, 15% grade, 15% grade- 1 min, and 2 min. EPOC was not

collected for the 15% AST. However, the performance time for this test, along with the performance time of the 20% AST was used in the extrapolation of the grade used for the 2 min AST. EPOC was collected for thirty minutes immediately following the completion of the other three ASTs. Each subject's fasting RMR was measured at the start of a randomly selected testing day.

3.4.2 VO₂max Test

Subjects approached volitional fatigue by an incremental treadmill test in which the speed of the treadmill started at 5 mph and was increased by .5 mph each minute until exhaustion (Taylor et al., 1953). Subjects were verbally encouraged to go as long as possible to ensure that VO₂max was attained.

3.4.3 Anaerobic Speed Tests (ASTs)

Four ASTs were performed. The treadmill was elevated to a specified angle and the speed was consistently set at 8 mph. Subjects, wearing a safety belt, were instructed to hold on the hand rails and step on to the moving treadmill. As soon as the subjects felt secure with the treadmill speed, they were instructed to let go of the hand rails at which point timing of the AST began. Subjects ran until exhaustion or to the desired time, at which time they straddled the treadmill and grabbed the handrails. A technician was also on hand to ensure this occurred safely and that the mouthpiece remained in the subject's mouth. Performance was measured by duration in seconds maintained on the treadmill from two of the four ASTs. The remaining two ASTs had fixed durations.

3.5 Data Collection

3.5.1 Oxygen Consumption (VO_2)

Oxygen consumption was monitored every 15 seconds during the VO_2max , the RMR, and throughout the recovery from the three ASTs, via the Beckman Metabolic Cart and Hewlett-Packard 3052A data acquisition system.

3.5.2 Resting Metabolic Rate (RMR)

RMR was measured for 20 minutes prior to the exercise. Subjects rested supine while remaining quiet and still throughout the collection. RMR was calculated as the mean rate (l/min) of oxygen consumption for the last five minutes of the twenty minute session.

3.5.3 Maximum Oxygen Consumption (VO_2max)

Three criteria were met for each subject to ensure maximum oxygen uptake: 1) heart rate within 10 bpm of age predicted maximum; 2) RER greater than 1; and 3) a leveling off of VO_2 with an increase in workload. VO_2max was represented in $\text{ml}\cdot\text{kg}\cdot\text{min}$.

3.5.4 Anaerobic Capacity (20% AST)

The performance (duration in seconds) of the 20% AST was used as a measure of anaerobic capacity. The AST protocol for this has been standardized (Cunningham and Faulkner, 1969) and widely used among athletic populations.

3.5.5 EPOC (20% AST and 15%-1 min AST)

The ASTs were run at 8 mph on a 20% and 15% grade. The 20% AST was run to exhaustion, while the 15% AST was run for one minute. The 20% AST represents the standard AST commonly performed as a measure of anaerobic power and capacity with many athletes. It has a fixed intensity, but duration is varied because of individual anaerobic differences. The 15%- 1 min AST represents a fixed intensity and fixed duration exercise condition, therefore presenting each athlete with the same absolute workload. These two exercise conditions were provided to examine the intensity/duration issue and how it manipulates EPOC. EPOC was measured for thirty minutes beginning immediately from cessation of the exercise. EPOC was represented in litres for all of the analyses except for the correlations between VO_2max for which EPOC was represented in ml·kg.

3.5.6 Maximum EPOC (2 min AST)

Maximum EPOC was measured for thirty minutes following the cessation of the 2 min AST. The inclination was extrapolated from the performance (durations) of the 20% and 15% ASTs to be a grade in which running could be sustained for a two minute duration. Two minutes of exhaustive treadmill running has been documented as sufficient time in which to achieve maximum EPOC (Medbo et al., 1988).

3.5.7 Skinfolds

The sum of 6 skinfolds (mm) was recorded using Harpenden calipers, from measurements taken at the following sites: bicep, tricep, subscapularis, suprailiac, anterior thigh, medial calf. Two measurements were taken for each site. A third

and/or fourth were taken when there was a discrepancy of measurements by more than 2mm. The skinfolds were used to calculate percent fat.

3.5.8 Girths

Four individual limb girths (cm) were measured using a standard tape measure at the following locations: mid-arm, maximum forearm, mid-thigh, maximum calf. Two measurements were taken with a third and/or fourth taken in the event of a discrepancy greater than .1cm. The girths along with two of the skinfold measurements were used to estimate muscle mass for each subject.

3.5.9 Heart Rate

Heart rate was monitored using a Sport Tester 3000 heart rate monitor and recorded every minute during the VO_2max test and during recovery from the ASTs. During the ASTs heart rate was recorded every 15 seconds as an indicator of intensity.

3.5.10 Peak Blood Lactate

Five cutaneous finger tip blood samples were collected and immediately hemolyzed. The samples were analyzed with a Kontron 640 lactate analyzer to determine the blood lactate concentration. Samples were taken prior to the exercise, and at minutes one, two, three, and four post-exercise.

3.6 Data Analysis

3.6.1 Recovery Rate

Subjects' minute EPOC values in log form were plotted against time in a scatter plot. Each individual's recovery data were split into two components (fast and slow) and fitted to separate exponentials of the form:

$$\begin{array}{ll} y_1 = A_1 e^{-t/\tau_1} & y_2 = A_2 e^{-t/\tau_2} \\ \text{(fast)} & \text{(slow)} \end{array}$$

The strategy for determining the point at which the recovery curve would be split involved careful examination of the total curve. The first data point in which there was a significant lateral movement as opposed to an obvious vertical decay became the start of the slow component. The constants were determined by carrying out linear regressions of the log-log data:

$$\begin{array}{ll} \text{If} & y = Ae^{-t/\tau} \\ \text{Then} & \log y = \log A + \log e^{-t/\tau} \\ & = \log A - \frac{t}{\tau} (\log e) \\ \text{Therefore} & \log y = \log A - \frac{t(.434)}{\tau} \end{array} \quad (1)$$

If $\log y$ is plotted against t , the result is a straight line of slope $-1/\tau$ and intercept $\log A$. These values were obtained by regression analysis of the semi-log data using Cricket Graph (Mac SE). If m and b are the linear regression constants, i.e.

$$y = mx + b \quad (2)$$

then, comparing equation (1) and (2) gives:

$$m = -\frac{.434}{\tau} \quad \text{and} \quad b = \log A$$

giving $\tau = -\frac{.434}{m}$ and $A = 10^b$ (3) (4)

The correlation coefficient was used to check whether or not the best fit was found for the curve. Figure 3, 4, and 5 illustrate how this was done. If the curve did not produce a very high r value (particularly for the fast component) the cut point was changed and used in the analyses if it produced a better correlation, thus, indicating a better fit.

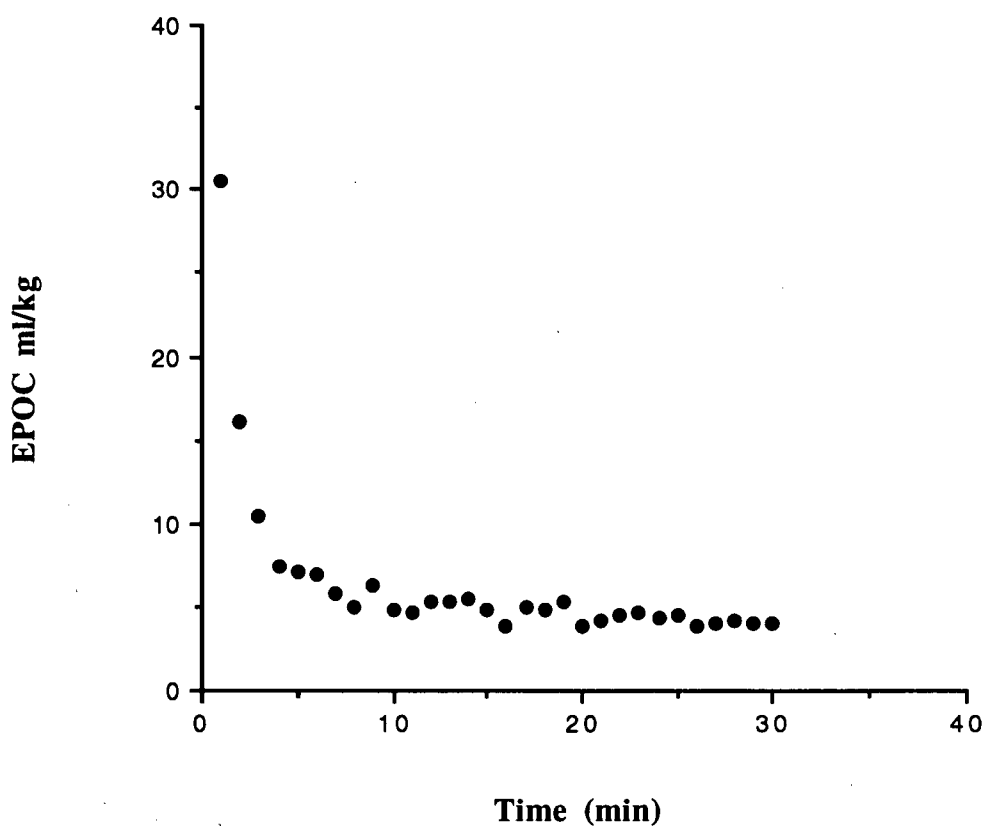


Figure 3: Recovery curve for subject #1

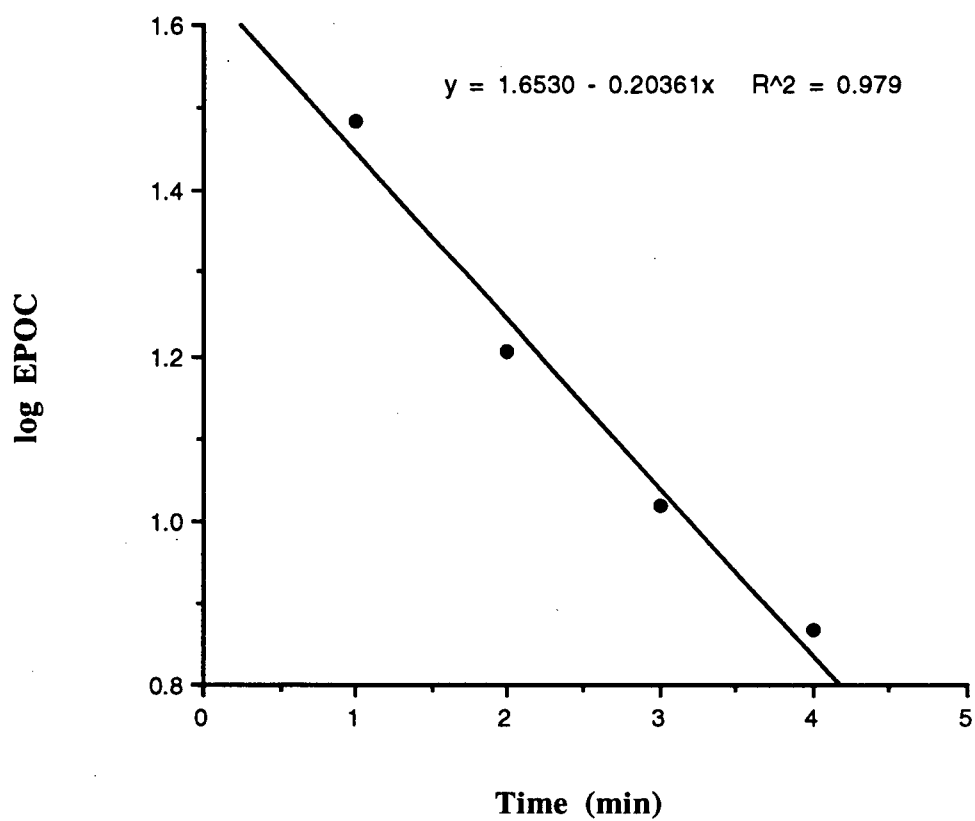


Figure 4: Fast component of the recovery curve for subject #1

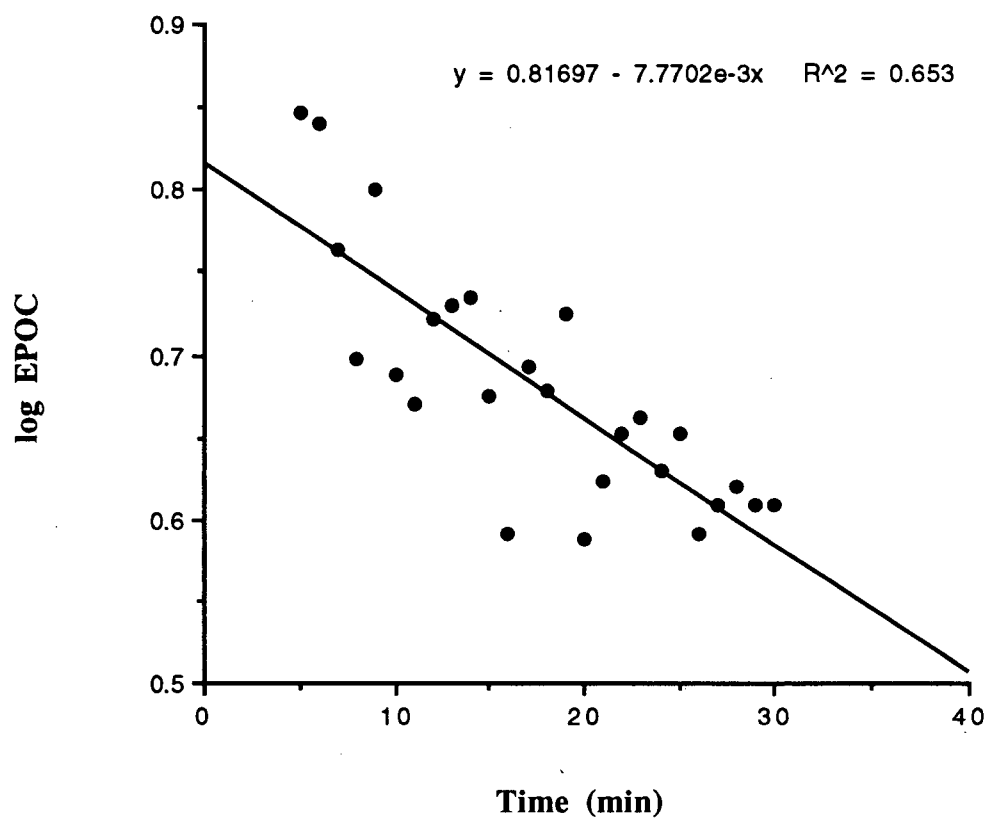


Figure 5: Slow component of the recovery curve for subject #1

3.6.2 EPOC

The area under the recovery curve represents the magnitude of EPOC. EPOC was obtained by summing the net energy expenditure for each minute value of the EPOC period (Sedlock, 1991).

$$\sum_{j=1}^{30} X_j \quad \text{where } X \text{ represents the net EPOC value for minute } j$$

The area under the fast and slow components were also analyzed and referred to as EPOCfast and EPOCslow to allow for possible identification of where the significant differences in the total EPOCs took place.

3.7 Statistical Analysis

3.7.1 ANOVA

A three condition repeated measures Analysis of Variance was used to determine significant differences within the dependent variables EPOC, peak blood lactate, τ_1 and τ_2 . When appropriate, pairwise comparisons were made using Tukey's post hoc analysis.

3.7.2 Correlation

Relationships between $VO_{2\max}$, τ_1 , τ_2 , EPOC, and peak blood lactate were identified using Pearson Correlation (r). $VO_{2\max}$ was analyzed expressed as $\text{ml}\cdot\text{kg}\cdot\text{min}$.

3.8 Specific Hypotheses

- 1) there will be a significant negative correlation between $\text{VO}_{2\text{max}}$ and EPOC for the 15%- 1 min AST.
- 2) there will be no significant correlation between $\text{VO}_{2\text{max}}$ and EPOC for the 20% and 2 min ASTs.
- 3) the magnitude of EPOC 2 min > EPOC 20% > EPOC 15%- 1min.
- 4) there will be a significant negative correlation between the time constants (τ_1, τ_2) and $\text{VO}_{2\text{max}}$ such that the greater the $\text{VO}_{2\text{max}}$, the faster the rate of recovery.
- 5) there will be no significant difference in τ_1 across the three exercise conditions
- 6) there will be no significant difference in τ_2 across the three exercise conditions
- 7) there will be a significant positive correlation between peak blood lactate and EPOC.
- 8) blood lactate 2 min > blood lactate 20% > blood lactate 15%- 1min.

RESULTS

4.1 Descriptive Data

Fifteen male athletes began this study, however, three subjects dropped out due to injury. Of the twelve subjects who completed the study, all were in good health and injury free throughout the duration of testing. Individual and mean physical and physiological data for the twelve subjects are presented in Table 1.

Table 1: Physical and Physiological Data

Subject (#)	Age (years)	Height (cm)	Weight (kg)	Muscle Mass(kg)	VO ₂ max (ml·kg·min ⁻¹)	RMR (l·min ⁻¹)
1	20	184.0	75.6	45.31	57.83	.29
2	26	180.0	75.9	43.55	55.84	.27
3	32	187.3	91.8	53.03	55.02	.27
4	27	181.7	84.2	47.58	50.98	.27
5	23	181.1	86.5	47.36	44.55	.31
6	21	186.7	83.9	51.03	57.40	.28
7	24	182.0	85.0	49.92	54.71	.26
8	21	173.3	67.0	41.64	55.26	.23
9	28	181.5	83.5	50.44	53.61	.22
10	23	188.1	82.7	44.12	45.27	.26
11	23	186.5	86.6	51.67	49.14	.28
12	19	192.7	84.2	51.02	54.08	.24
Mean	23.92	183.74	82.24	48.06	53.53	.27
SD	3.75	4.96	6.50	3.69	4.54	.03

4.2 Recovery Data

Each recovery variable is presented in table 2 for ASTs 20%, 15%- 1 min and 2 min respectively, along with duration and percent grade where applicable.

4.2.1 EPOC

Mean EPOC values are presented in Table 2. The 2 min AST produced the highest average EPOC (9.29 l or 112.97 ml·kg) followed by the 20% AST (9.04 l or 110.79 ml·kg) and then the 15%-1 min AST (7.56 l or 92.53 ml·kg). Analysis of variance indicated significant differences in the magnitude of EPOC between exercise conditions ($p<.01$). Tukey's post-hoc comparisons of the EPOC means (Table 3) revealed the significant differences ($HSD= 1.48$ l, $p<.05$) were between the 20% and the 15%- 1 min EPOCs (1.48 l) and between the 2 min and the 15%- 1 min EPOCs (1.73 l). The magnitude of the EPOCs for each AST are illustrated in Figure 6. Individual EPOC values for each AST are listed in Table 7 of the APPENDIX. When magnitude of EPOC was investigated according to its two components, fast and slow, significant differences were observed in the slow component only ($p<.01$). EPOC for the slow component was 3.94 l, 2.71 l, and 4.23 l, for the 20%, 15%- 1 min, and 2 min ASTs, respectively. Post-hoc comparisons revealed the significant differences ($HSD=1.45$ l, $p<.05$) were between the 2min and the 15%- 1 min ASTs (1.74 l) and the 20% and the 15%- 1 min ASTs (1.51 l). Figure 7 illustrates the magnitudes of these separate components for each AST. Individual fast and slow magnitudes for each AST are listed in Table 8 in the APPENDIX.

4.2.2 Blood Lactate

Mean peak blood lactate values for each AST are listed in Table 2. The 2 min, 20% and 15%- 1 min ASTs produced mean peak lactate values of 15.74 mmol·l, 13.62 mmol·l and 13.01 mmol·l, respectively. Analysis of variance showed significant differences in blood lactate between exercise conditions ($p < .05$). Post hoc analysis further revealed that the significant differences ($HSD = 2.20 \text{ mmol·l}$ @ $\alpha = .05$) were between the 2 min and the 15%- 1 min AST (2.73 mmol·l). The 20% and 15%- 1 min lactate values were not statistically different (.61 mmol·l), nor were the 20% and 2 min lactate values (2.12 mmol·l) as shown in Table 5. Figure 6 illustrates the peak blood lactate concentrations for each exercise condition. Individual peak blood lactate values are listed in Table 9 of the APPENDIX.

4.2.3 Recovery Rate

Mean τ values are presented in Table 2. τ_1 represents the rate of the fast component of the recovery curve and τ_2 , the rate of the slow component. Analysis of variance showed no significant difference τ_1 across all three ASTs ($p = .37$). Likewise, no significant difference was found in τ_2 across exercise conditions ($p = .83$). Figure 8 illustrates the mean recovery curves for the three ASTs. Individual τ_1 and τ_2 values are listed in Tables 10 and 11, respectively, in the APPENDIX.

Table 2: Means and Standard Deviations of ASTs

AST	Grade (%)	Duration (seconds)	EPOC (litres)	EPOC fast (litres)	EPOC slow (litres)	Peak BLa (mmol l)	τ_1 (minutes)	τ_2 (minutes)
20%	20.0	48.83 ± 13.13	9.04* ± 2.03	5.00 $\pm .77$	3.94* ± 1.44	13.62 ± 2.33	2.60 .42	49.38 19.90
15%- 1min	15.0	60.0	7.56 ± 1.13	5.07 ± 1.16	2.71 $\pm .97$	13.01 ± 1.99	2.35 .36	44.86 12.11
2min	11.8 ± 2.65	129.08 ± 16.01	9.29* ± 2.22	5.02 $\pm .58$	4.23* ± 1.76	15.74* ± 2.60	2.40 .29	45.40 8.78

*statistically significant

Table 3: Post Hoc Comparison of EPOC Data

		15%- 1 min	20%	2 min
		7.56 l	9.04 l	9.29 l
15%- 1 min	7.56 l	0	1.48 l *	1.73 l *
20%	9.04 l		0	.25 l
2 min	9.29 l			0

*HSD=1.48 litres; $\alpha=.05$

Table 4: Post Hoc Comparison of the slow component EPOC Data

		15%- 1 min	20%	2 min
		2.53 l	4.04 l	4.27 l
15%- 1 min	2.53 l	0	1.51 l *	1.74 l *
20%	4.04 l		0	.23 l
2 min	4.27 l			0

*HSD=1.45 litres; $\alpha=.05$

Table 5: Post Hoc Comparison of Blood Lactate Data

		15%- 1 min	20%	2 min
		13.01 mmol·l	13.62mmol·l	15.74 mmol·l
15%- 1 min	13.01 mmol·l	0	.61 mmol·l	2.73 mmol·l *
20%	13.62 mmol·l		0	2.12 mmol·l *
2 min	15.74 mmol·l			0

*HSD=2.20 mmol·l; $\alpha=.05$

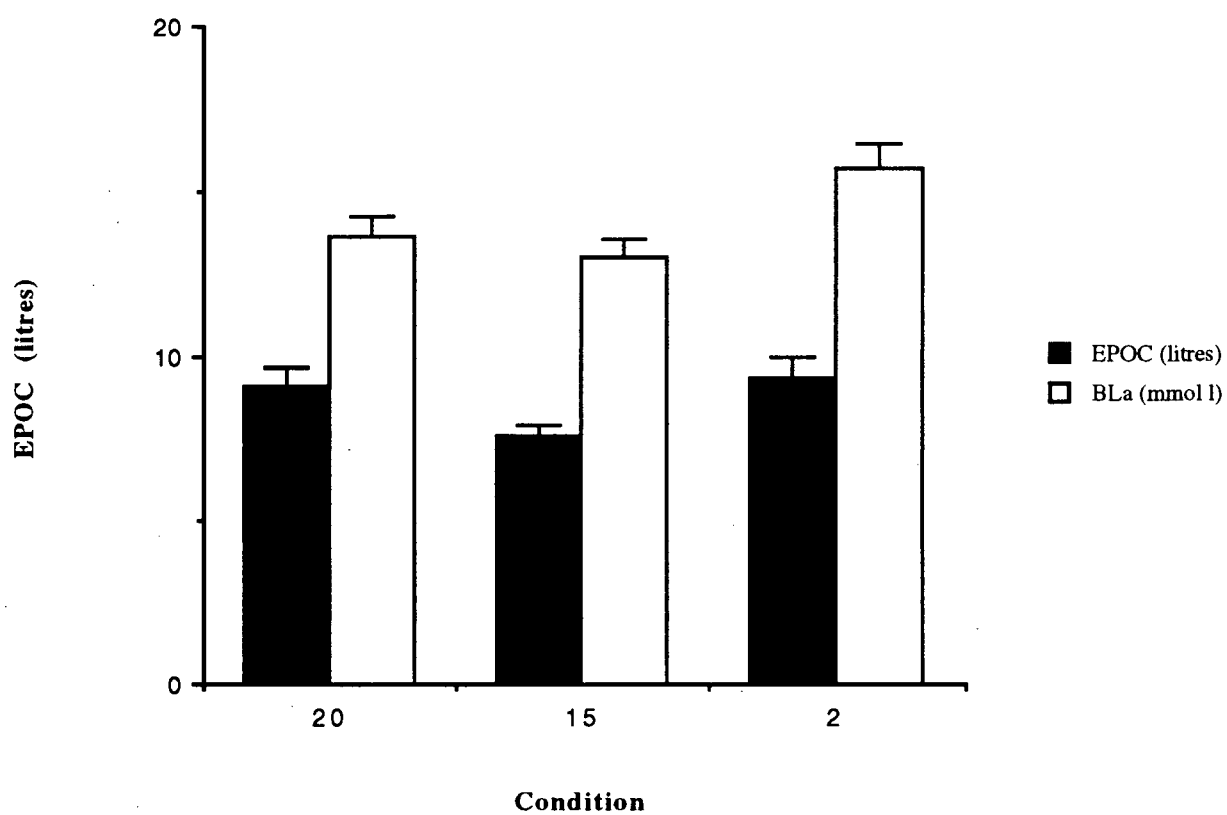


Figure 6: EPOC and Blood Lactate Response to Exercise Condition. 20=20% AST; 15=15%- 1 min AST; 2=2min AST

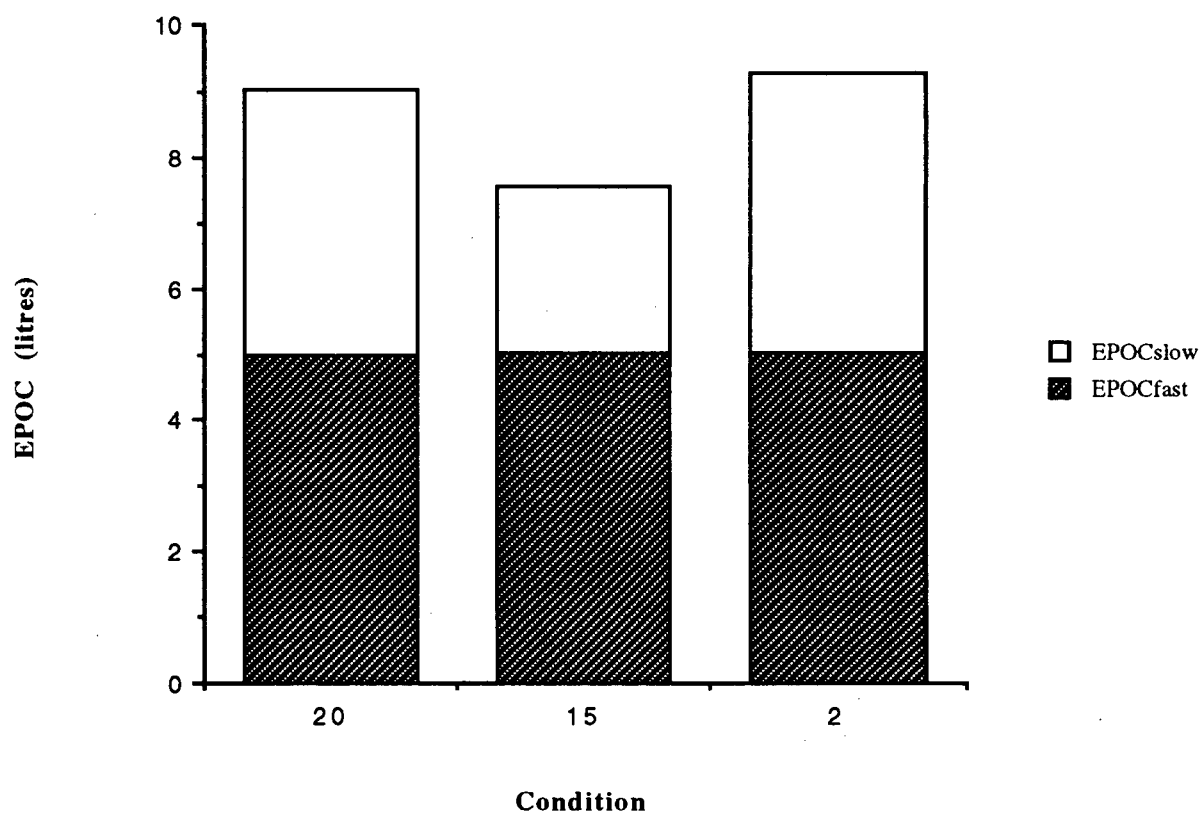


Figure 7: Mean magnitude of EPOCfast and EPOCslow between exercise condition. 20=20% AST; 15=15%- 1min AST; 2=2min AST

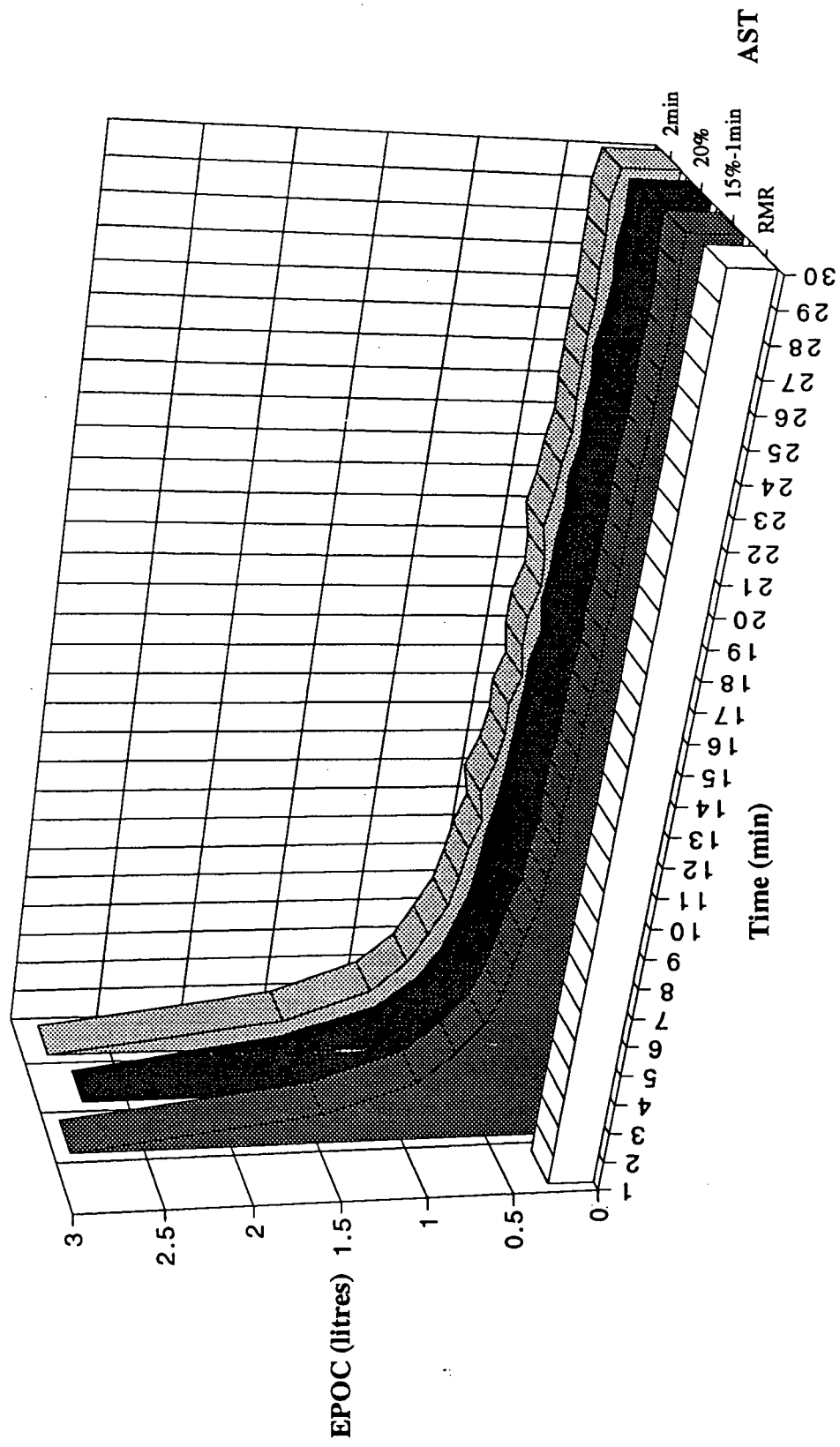


Figure 8: Area graph of Mean Post-Exercise Oxygen Consumption

4.3 Relationships

4.3.1 EPOC

A correlation matrix for the recovery variables is presented in Table 6. EPOC was expressed in ml·kg for the correlation analyses in order to provide relative comparisons. No significant relationships were found between EPOC and VO_2max . Figure 9 illustrates the range of VO_2max scores for the group. There were also no significant relationships between EPOC and peak blood lactate. There was a correlation found between VO_2max and EPOCfast for the 15%- 1 min AST ($r=.63$, $p=.03$). This would suggest the higher the VO_2max the larger the magnitude of the fast component of the recovery curve for that AST. This may be a spurious correlation. Strong correlations were found between the EPOCfast and total EPOC for all ASTs as well as between the EPOCslow and total EPOC for all ASTs (Table 6). Of the two groups, the EPOCslow set had slightly higher correlations than the EPOCfast set, which lends further support to the magnitude of the slow component of EPOC having the greater influence on total magnitude.

4.3.2 Recovery Rate

The correlations between VO_2max and rate (Table 6) did not produce any significant relationships except for a moderate correlation between VO_2max and τ_1 for the 15%-1 min AST ($r=.59$, $p=.04$). This correlation would indicate that the higher the VO_2max the slower the slow component rate of recovery for that AST. This may be another spurious correlation as there are no explanations in the literature to support such a finding.

4.3.3 Blood lactate

The only significant relationship between VO_2max and peak blood lactate (Table 6) also occurred for the 15%-1 min AST ($r=-.60$ $p=.04$). This correlation would suggest that the higher the VO_2max , the lower the blood lactate for that test.

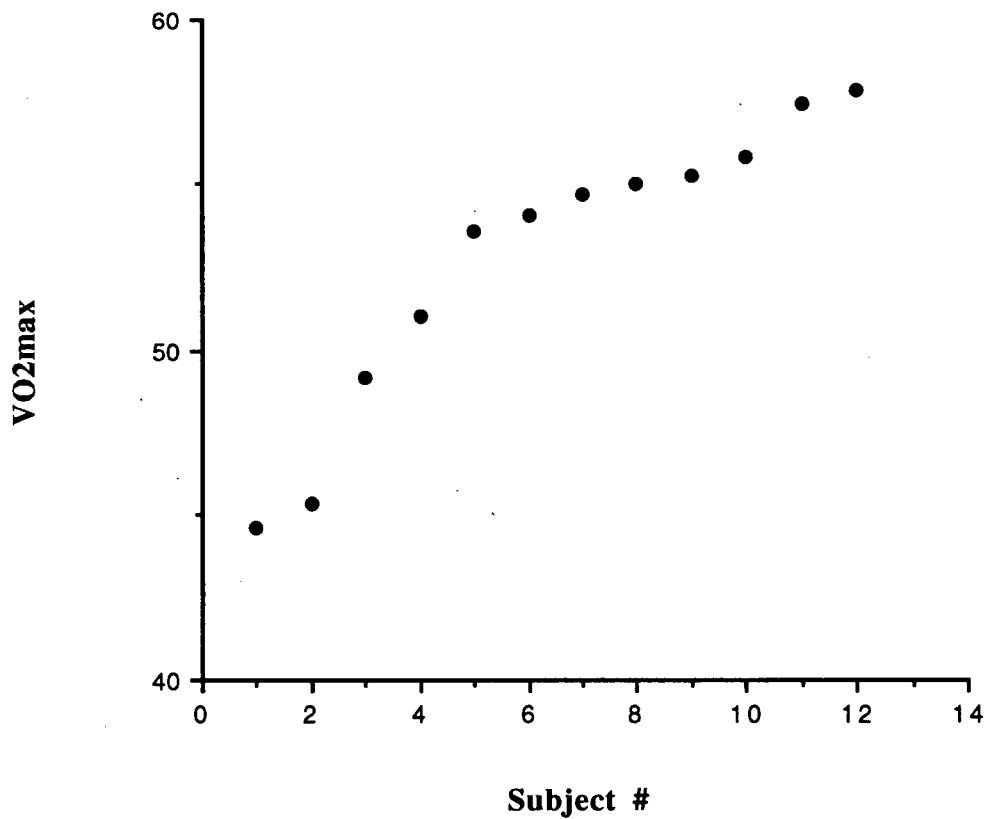


Figure 9: Scatter plot of VO_2max scores

4.3.4 AST Performance

Figure 10 illustrates the anaerobic capacity of each subject. As shown in Table 6, significant positive relationships were found between AST performance and the 20% EPOC ($r=.74$, $p=.01$), and the 2 min EPOC ($r=.62$, $p=.03$). When looking at the magnitudes of the fast and slow components, AST performance was significantly correlated with EPOCfast ($r=.69$, $p=.01$) and EPOCslow ($r=.65$, $p=.02$) for the 20% AST, EPOCfast ($r=.66$, $p=.02$) for the 15%- 1 min AST, and EPOCslow ($r=.67$, $p=.02$) for the 2min AST. There was also a significant negative correlation found between AST performance and peak blood lactate ($r=-.64$, $p=.03$) for the 15%- 1 min AST. No significant relationships were found between AST performance and rate.

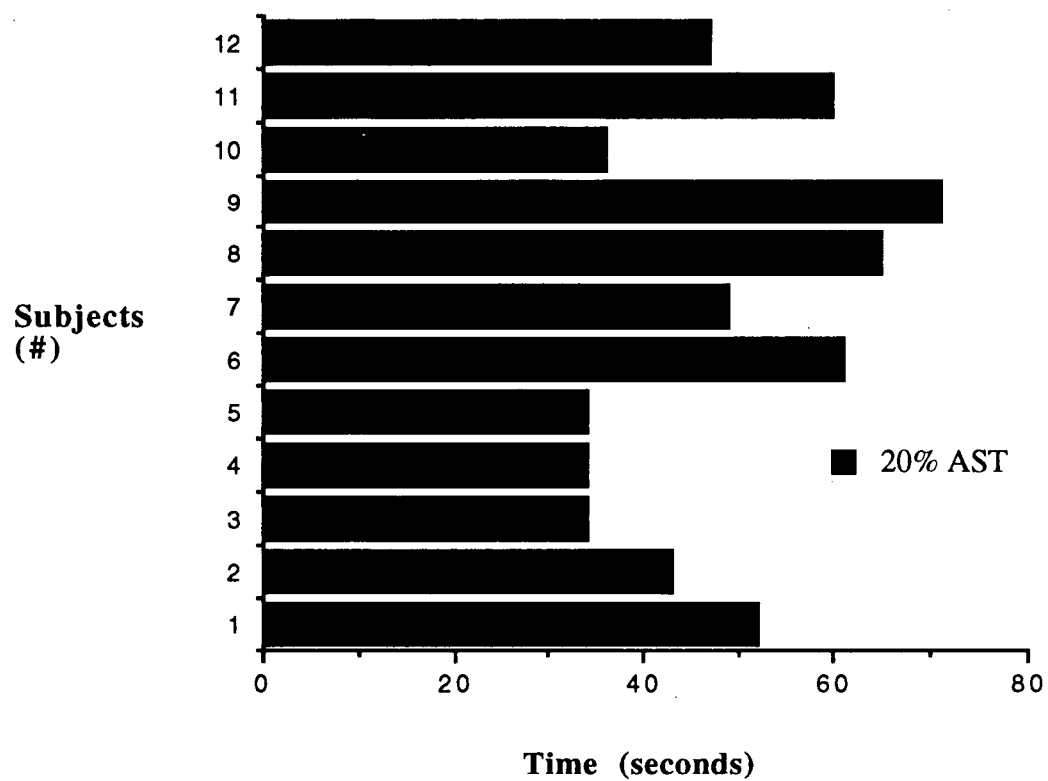


Figure 10: Anaerobic capacity as indicated by AST Performance

	VO2max	AST perf.	Peak BLA	EPOC 20%	EPOC 15%-1m.	EPOC 2 min	T1 20%	T1 15%-1m.	T1 2 min	T2 20%	T2 15%-1m.	T2 2 min
VO2max		.24 p=.45	.60 [^] p=.04	.08 p=.80	.19 p=.56	.04 p=.90	.06 p=.86	.21 p=.52	-.002 p=.99	.22 p=.48	.59 [^] p=.04	.11 p=.73
AST perf.			-.64 [^] p=.03	.74 [^] p=.01	.29 p=.37	.62 [^] p=.03	.28 p=.38	-.14 p=.66	-.18 p=.58	-.05 p=.87	.38 p=.23	.32 p=.31
Peak BLA				.53 p=.08	-.13 p=.70	.47 p=.12	.29 p=.35	-.05 p=.89	-.02 p=.95	.21 p=.51	-.44 p=.15	.22 p=.50
EPOC 20%							.25 p=.44			-.07 p=.82		
EPOC 15%-1m.								.28 p=.37			.07 p=.84	
EPOC 2 min									-.15 p=.64			.22 p=.50

Table 8: Correlation Matrix

Note: Shaded areas indicate significant correlation
^ 15%- 1 min AST

	EPOCfst 20%	EPOCfst 15%-1m	EPOCfst 2 min	EPOCslw 20%	EPOCslw 15%-1m	EPOCslw 2 min
VO2max	.49 p=.10	.63 [^] p=.03	.46 p=.13	-.01 p=.97	.13 p=.69	.07 p=.83
AST perf.	.69 [^] p=.01	.66 [^] p=.02	.46 p=.14	.65 [^] p=.02	.01 p=.97	.67 [^] p=.02
Peak BLA	.02 p=.94	-.36 p=.26	.27 p=.39	.50 p=.10	-.04 p=.91	.29 p=.36
EPOC 20%	.75 [^] p=.01			.82 [^] p=.001		
EPOC 15%-1m.		.73 [^] p=.01			.73 [^] p=.01	
EPOC 2 min			.62 [^] p=.03			.89 [^] p=.000

4.4 Results of Hypotheses

- | | | |
|----|------------------------------------------------------------------------------------------------------|--------|
| 1) | significant negative correlation between VO_2max and
EPOC 15%- 1 min | reject |
| 2) | no significant correlation between VO_2max and EPOC
20% and 2 min | accept |
| 3) | EPOC 2 min > EPOC 15%- 1 min | accept |
| | EPOC 2 min > EPOC 20% | reject |
| | EPOC 20% > EPOC 15%- 1 min | reject |
| 4) | significant negative correlation between rate (τ_1 , τ_2)
and VO_2max | reject |
| 5) | no significant difference in τ_1 across exercise conditions | accept |
| 6) | no significant difference in τ_2 across exercise conditions | accept |
| 7) | significant positive correlation between blood lactate and
EPOC | reject |
| 8) | lactate 2 min > lactate 20% | reject |
| | lactate 2 min > lactate 15%- 1 min | accept |
| | lactate 20% > lactate 15%- 1 min | accept |

DISCUSSION

In the past, researchers have investigated the effect of athletic conditioning on O₂ debt, now termed EPOC (Henry, 1950; Henry and Berg, 1950; Girandola and Katch, 1973; Hagberg et al., 1980). These studies were able to document significant physiological changes resulting from an improved aerobic fitness, including a smaller EPOC and a more rapid rate of recovery for the same absolute workload. It has also been shown that recovery rates improve for the same relative workload after training (Hagberg et al., 1980). However, more recent research involving cross-sectional samples rather than training studies, have produced inconclusive results. The majority of these studies have been unable to demonstrate a training effect on EPOC (Graham and Andrew, 1973; Freedman-Akabas et al., 1985; Brehm and Gutin, 1986; Kaminsky et al., 1987; Chad and Quigley, 1991), whereas others have found supporting evidence (Elliot et al., 1988; Frey et al., 1993). The reasons for these discrepancies remain unknown, but the exercise intensities and durations used may be partly responsible (Frey et al., 1993). The protocols used in this study are unique to the literature thus, making comparisons of previous data with those presented in this paper, sometimes difficult.

5.1 Subjects

The athletes used in this study appear to be descriptively typical of other trained subjects used for researching EPOC. The fasting RMR ($.27 \pm .03$ l·min⁻¹) is within the range of other reported findings (Bahr et al., 1987; 1991a) and the descriptive characteristics of height, weight, age and VO₂max are very similar to those reported by Bahr et al., 1987, 1991a, and 1992, the last of which involved supramaximal exercise bouts.

5.2 Metabolic basis of EPOC

While considerable debate surrounds the metabolic basis of EPOC, there is little dispute over the existence of EPOC (Bahr et al., 1992; Gore and Withers, 1990b; Maehlum et al., 1986; Stainsby and Barclay, 1970) and its validity as a measure of homeostatic disturbance (Sedlock et al., 1989). Until the processes involved in producing EPOC are determined, EPOC can only be used as a measurement tool of overall post-exercise recovery metabolism (Gaesser and Brooks, 1984).

The mechanisms behind EPOC remain elusive. Several hypotheses have been postulated, including replenishment of O₂ stores in blood and muscle, resynthesis of ATP and creatine phosphate, increased core temperature, conversion of lactate to glucose or glycogen, increased levels of catecholamines, increased rate of triglyceride-fatty acid (TG-FA) cycling, and altered levels of intra- and extra-cellular ion concentrations (Gaesser and Brooks, 1984; Gore and Withers, 1990; Bahr et al., 1992; Frey et al., 1993), which all represent a disturbance to resting metabolism as a result of an exercise bout (Gore and Withers, 1990). The replenishment of O₂ stores and resynthesis of ATP and CP believed to be associated with the rapid or "alactacid" component are probably completed within a few minutes after exercise, and the contribution to EPOC is small (Gaesser and Brooks, 1984; Bangsbo et al., 1990). Although complete recovery of ATP required 1.0 - 1.5 hours, restoration of CP required only 5 minutes following exhausting exercise in juvenile rainbow trout (Scarabello et al., 1991). Lactate removal, on the other hand, has been thought to account for a major portion of the recovery period. Based on the original O₂ debt hypothesis, one would predict that 75-90% of the lactate produced during exercise is converted to muscle glycogen in the post-exercise period. The energy required for this process was presumed to come in part from the oxidation of the remaining 10-25% of the

lactic acid (Gaesser and Brooks, 1984). Numerous lactate-tracer studies (see Gaesser and Brooks, 1984 pg.36 for references), have demonstrated that oxidation accounts for as much as 55-70% of the lactate present at the end of exercise. Interestingly, Astrand et al. (1986), found that in strenuous exercise, in which large amounts of lactate are produced in exercising muscles, approximately 50% of the lactate formed is transformed to glycogen. They calculated that approximately 40% of the lactate is oxidized with the remaining amounts being taken up by the liver and retained in body water, and suggested the existence of a biochemical apparatus for glyconeogenesis, at least in fast-twitch muscle. It has also been suggested that, because the mitochondrion is the locus of O_2 consumption in the cell, the explanation for the elevated post-exercise VO_2 may be confined to this organelle (Gaesser and Brooks, 1984). Support for this hypothesis involves the indirect control catecholamines, thyroxine, and glucocorticoids may have over mitochondrial respiration.

EPOC has been shown to be influenced by training status (Hagberg et al., 1980, exercise intensity (Hagberg et al., 1980; Bahr et al., 1987; 1991; 1992; Sedlock et al., 1989; Gore and Withers, 1990; Smith and McNaughton, 1993; Frey et al., 1993), exercise duration (Knuttgen, 1970; Chad and Wenger, 1985; 1988; Sedlock, 1991), as well as the thermic effect of food (Maehlum et al., 1986). However, it remains to be fully determined what these differential effects have on the exercise threshold for eliciting a significant EPOC (Quinn et al., 1994).

Unfortunately, while many of the above mentioned factors are routinely observed in accordance with an elevated post-exercise VO_2 , they are not always present. If present, these factors are not always evident or related to the same degree, thus, making

identification of any consistent contributions to EPOC very difficult. The relationship of blood lactate to EPOC is a good example of this and will be discussed later. Therefore, EPOC is technologically limited to estimating the magnitude of homeostatic disturbance (Sedlock et al., 1989).

5.3 Comparison of EPOC

Although EPOC reflects more than anaerobic catabolism (Stainsby and Barclay, 1970; Gaesser and Brooks, 1984; Medbo et al., 1988; Bangsbo et al., 1989), it is a sensitive measure of performance improvements and of the capacity to perform exhaustive exercise of short duration (Hermansen, 1969). Despite the ability of EPOC to metabolically compare non-steady state exercises, previous studies investigating EPOC have focused predominately on prolonged submaximal exercise. Very few have measured post-exercise oxygen consumption following short bouts of supramaximal exercise involving a significant anaerobic contribution and high levels of blood lactate (Hermansen et al., 1969; Bahr et al., 1992; Roberts and Rhodes, 1993). These studies looked at EPOC across varying degrees of intensity and duration within the scope of supramaximal exercise. Bahr et al. (1992), compared EPOC from three separate intermittent 2 min exercise bouts on a cycle ergometer at 108% $\text{VO}_{2\text{max}}$. They reported 1 hour EPOCs of 7.8 ± 0.7 l (3 x 2 min), 6.7 ± 0.4 l (2 x 2 min), and 5.6 ± 0.4 l (1 x 2 min). EPOCs in this study were much larger despite only a 30 minute post-exercise recovery period (9.29 ± 2.22 l, 9.04 ± 2.03 l, and 7.56 ± 1.13 l for 20%, 15%- 1 min, and 2 min ASTs, respectively). The higher EPOCs in this study are most likely due to the running protocol used, as opposed to the cycling protocol, in which a larger muscle mass was involved. Roberts and Rhodes (1993), found higher EPOCs from four separate sprint conditions involving either an isokinetic device

(APM) or free sprinting. They reported 30 minute EPOCs of 15.16 ± 2.59 l, 11.38 ± 2.72 l, 9.88 ± 2.80 l, and 9.09 ± 2.51 l for a 2 min AST, 5 APM, 10 free, and 5 free, respectively. While the modalities used in their research differed from the present study, the 2 min ASTs used in both studies were executed identically. The much larger 2 min EPOC of their study can be attributed to the subject pool which was comprised of eight highly trained sprinters. They would most likely have a larger muscle mass and a greater lactate tolerance, in comparison to the training status of this research's subject pool. 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 minute of exhausting exercise (Medbo, 1985). Graham and Andrew (1973), collected what they called 'maximum oxygen debt' for thirty minutes following a VO_{2max} test. Oxygen consumption in the recovery varied from 7.4 l or 103.6 ml·kg (track team) to 8.4 l or 115.3 ml·kg (cross-country skiers). The lower values of these results in comparison to those previously mentioned, suggest that this procedure does not elicit a maximum EPOC.

As evidenced by the research mentioned above, EPOC can be manipulated through changes in the intensity and duration of the exercise bouts. While the literature is inconclusive as to the intensity / duration effects, this study found significant manipulations as a result of varying durations. The 2 min AST was the longest duration incurred by the athletes and also produced the largest volume of EPOC. However, one must be careful in making assumptions based on these results. The grade of the treadmill used for the 2 min AST varied among subjects, as it was based on their performance curve. The grades ranged from 8 - 15% thus, some subjects were working at double the absolute intensity of others. This also meant that the 15%- 1 min AST may have been close to maximum for the less anaerobically trained subjects. Evidence of this can be seen by examining the EPOCs for each individual across the three conditions as shown in Table 7 in the APPENDIX.

Differences between the 2 min and 15%- 1 min EPOCs varied from .68 l to 6.26 l. Since EPOC is being used as an indicator of metabolic disturbance, the similar EPOCs produced by certain subjects suggest that the different exercise conditions may have been too similar in relative terms, to elicit any significant intensity / duration effects for these subjects. Thus, with the intensity factor not controlled, the duration effects are difficult to defend. However, when duration of the 20% AST was correlated with EPOC, a significant positive relationship was found ($r=.74$, $p=.01$). Thus, the longer the subject ran, the larger the magnitude of the EPOC. Here, intensity was held constant (20% grade, 8 mph) however, there was no control over caloric expenditure. Bahr et al. (1987), have suggested that the total work performed during exercise and the subsequent EPOC may be related. These results are in agreement with the findings of Knuttgen (1970), who found total debt became elevated as a result of carrying on the work for longer periods of time. He also found the slow component of the debt to be completely responsible for this increase. When the mean EPOCs for each AST in this study were broken down into fast and slow components, the slow component was visibly the dominating factor to the change in overall magnitude amongst the conditions. Hagberg et al. (1980) found the slow component of recovery was not significantly altered by exercise intensity or duration at 50 and 65% of $VO_2\text{max}$. However, after 20 minutes of exercise at 80% $VO_2\text{max}$, the slow component of recovery was 5 times ($p<.01$) larger than after 5 min of exercise at this intensity. The results of this research clearly show the magnitude of the slow component to be predominately responsible for the change in overall magnitude amongst the conditions.

AST performance was used as a measure of anaerobic capacity. This AST performance was significantly correlated with EPOC ($r=.74$, $p=.01$ and $r=.62$, $p=.03$ for the 20% and 2 min ASTs respectively) thus, lending more support for a duration effect. Sedlock et al. (1989), found that when energy expenditure was held constant, exercise intensity affected

both the magnitude and duration of EPOC. However, when the duration of the moderate intensity exercise was manipulated, the magnitude of EPOC was not affected. Thus, since energy expenditure was not controlled in this study, and if Bahr et al. (1987), are correct in suggesting that total work performed during the exercise and EPOC are related, then the duration effects observed in this study should be interpreted with scrutiny. Interestingly, this may be why past studies investigating duration effects vary in their findings. Quinn et al. (1994), found exercise duration to significantly increase EPOC, in a non-linear fashion. By contrast, Bahr et al. (1987), found a linear relationship between submaximal exercise duration and EPOC, as well as for supramaximal intensities (1992).

In examination of intensity effects, the mean 20% EPOC was significantly greater than the 15%- 1 min EPOC. This clearly shows that intensity of supramaximal exercise can manipulate the resultant EPOC. Since the mean duration of the 20% AST was 48.8 seconds, the durations of both ASTs were quite similar, thus, making it unlikely that any duration effects interacted with the intensity effect. Of course, time to exhaustion declines as intensity is increased, thus, interaction between duration and intensity may be difficult to assess (Bahr and Sejersted, 1991). It is likely that for the 2 min AST, EPOC may be affected by an intensity duration interaction, although this data can not support such a theory. Gore and Withers (1990a), found that intensity explained five times the variance in EPOC volume as opposed to duration or the intensity duration interaction. Thus, it can be suggested that comparisons of the 20% and 15%- 1 min ASTs support an intensity effect, and comparisons within the 20% AST (between subjects) would support a duration effect. These findings would indicate that both intensity and duration have a significant influence on EPOC from supramaximal exercise.

5.4 Blood Lactate

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). Peak lactate concentration after supramaximal exercise of a 1 minute duration is often used as an indicator of anaerobic glycolysis (Hagberg et al., 1980b; Jacob, 1986). It has been determined that the lactate response to supramaximal exercise was a sensitive indicator of adaptation to sprint training and it correlates to supramaximal performance (Hermansen, 1969; Jacobs, 1986; Weyand et al., 1994). This is in agreement with the present results as shown by the significant correlation found between AST performance and peak blood lactate. Sprint-trained subjects have been found to accumulate more lactate in the blood than endurance-trained subjects during 1 minute of exhaustive exercise (Medbo and Sejersted, 1985), and the results of this study display this trend. Hermansen (1969), reported mean peak blood lactate increased from 15.4 mmol·l to 18.0 mmol·l following training. They also found when training stopped, a rapid decline in the ability to produce lactic acid occurred. Contrary to this, Medbo and Burgers (1990), found no significant changes in blood lactate over a 6 week training period despite an improved anaerobic capacity of 10%. However, when a sprint-trained group (individuals involved in anaerobic training for 5 years or more) was compared with the newly trained group (originally comprised of untrained and endurance-trained subjects), the former displayed a 30% larger anaerobic capacity. Only short-term training effects were studied, and it is very possible that years of training may have a greater influence on anaerobic capacity. There is also the possibility that genetic factors, or the combination of training and genetic factors, are responsible for the lower anaerobic capacity and lack of peak blood lactate adaptations. This would explain the variability of the blood lactate values found in this research, as the

subjects used were predominantly from the same soccer team and would presumably have been exposed to similar training regimes.

The mean peak blood lactate value derived from the 2 min AST in this research (15.74 mmol·l⁻¹) is very similar to those reported for the same test in other studies. Values reported in the literature are 13.4 mmol·l⁻¹ (Medbo et al., 1988), 14.9 mmol·l⁻¹ (Medbo and Burgers, 1990) and 14.83 mmol·l⁻¹ (Roberts and Rhodes, 1993). It is interesting to observe however, the mean peak lactate values of this study and that of Roberts and Rhodes (1993). Lactate values were within .91 mmol·l⁻¹ of each other despite a 5.87 l difference in mean EPOCs reported for the same test. It has been suggested that the intra cellular buffer capacity between sprint-trained and endurance trained may vary considerably (Parkhouse et al., 1982). Since fatigue is experienced during short exercise bouts, even though blood and muscle accumulations are far from maximal, factors other than acidosis may inhibit strenuous exercise at this high intensity (Medbo et al., 1988).

This inconsistency with lactate and EPOC is not new to the literature. Many investigators have found a significant relationship between peak blood lactate at the end of exercise and the resultant EPOC: $r=.79$, $p<.05$ (Graham and Andrew, 1973); $r=.86$, $p<.0005$ (Bahr et al., 1992); $r=.90$, $p<.05$ (Frey et al., 1993); and $r=.87$, $p<.05$ (Roberts and Rhodes, 1993). Despite these significant correlations, most of these investigators conclude that a large part of total EPOC cannot be accounted for by lactate removal (Bahr et al., 1992; Frey et al., 1993), but rather, they are spuriously related by the effect of intensity (Roberts and Rhodes, 1993). No significant relationships between lactate and EPOC were found in this study. Knuttgen (1970) and Bahr and Sejersted (1991) were also unable to find any significant relationships between lactate and EPOC. Providing support for these results are the earlier findings of Roth et al. (1988), in which it was revealed that manipulation of

exercise blood lactate level was found to have no significant effect on EPOC. Also, Knuttgen (1970), found substantial magnitudes for the slow component of EPOC even at work loads producing little or no blood lactate changes thus, providing support for the concept of the exercise causing a general disturbance to the body, independent of muscle and blood lactate accumulation. 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 (Bangsbo et al., 1990).

5.5 Rate of Recovery

The $\tau_{1/2}$ of the recovery VO_2 in this study varied from 1.65 to 3.51 minutes for the fast component, and from 32.9 to 109.4 minutes for the slow component. By contrast, Hagberg et al. (1980) analyzing the recovery curves as a single exponential, reported $\tau_{1/2}$ s of 23 to 35 seconds for work rates of 50% and 70% $\text{VO}_{2\text{max}}$ before training and 58% (previously 70%) and 70% after training. It was expected that the results of Hagberg should differ from the present study's results due to the exercise protocols used. As a result of the supramaximal intensities used in this study, a greater recovery time was required to bring VO_2 to baseline following such a large metabolic disturbance. The magnitudes reported in Hagberg's study were .76 l (50% of new $\text{VO}_{2\text{max}}$) and 1.47 l (70% of new $\text{VO}_{2\text{max}}$) for a ten minute recovery period following 10 minutes of submaximal exercise. Thus the values produced in this study between 7.56 and 9.24 l would correspond with much larger $\tau_{1/2}$ s. Unfortunately, research regarding the rate of recovery in terms of τ , is very limited and further comparisons are not possible. However, inspection of τ patterns within this research are possible. The τ_{1s} and τ_{2s} were not significantly different across exercise conditions. This would suggest that for a given individual, the rate of recovery is the same regardless of varying supramaximal conditions.

thus, despite changes in EPOC across exercise conditions, the rate to baseline remains constant. Henry and Berg (1950) and Hagberg et al. (1980), found recovery rate to change (increase) following 9 weeks of training. Even for the same relative work load, recovery rate was shown to improve despite much higher VO_2 s (Hagberg et al., 1980). It was also observed that at work rates requiring 70% of VO_2max , the return of VO_2 to base line prior to training had both a fast and a slow component. Training resulted in almost complete elimination of the slow component of the decline in VO_2 during the recovery (Hagberg et al., 1980). Henry and Berg (1950) noted that conditioning produced more significant change in the amount of debt than in the rate of payoff. The results of the present research however, are based on repeated tests within a time frame particularly designed to avoid training effects. The between subjects variability in recovery rate was quite large. Unfortunately, these previous researchers did not compare the recovery rates across conditions or between subjects, although the literature has often mentioned inter-subject variability in EPOC. Thus, this investigation of recovery rate is unique to the literature. It would be interesting to compare recovery rate patterns to those of other athletes, particularly for exercise of a supramaximal nature, as recovery plays a very important role in the performance of intermittent sports.

5.6 The Recovery-Training Status Relationship

Surprisingly, none of the recovery variables studied correlated with VO_2max in this study. Thus, an individual's rate of recovery or degree of homeostatic disturbance, cannot be predicted by their level of aerobic fitness. The training studies in which an improved rate of recovery was found as a result of an improved aerobic fitness (Henry and Berg, 1950; Girandola and Katch, 1973; Hagberg et al., 1980), only investigated intra subject effects of

the training and made no reports on the between subject differences ie., whether or not VO_2max correlated with recovery. Various cross-sectional studies have investigated the magnitude of EPOC and time to RMR from submaximal and maximal exercise, and its relationship to training status (Graham and Andrew, 1973; Freedman-Akabas et al., 1985; Brehm and Gutin, 1986; Kaminsky et al., 1987; Chad and Quigley, 1991; Frey et al., 1993). When cross-country skiers and track athletes were compared with non-athletic subjects, no differences in 'max O_2 debt' could be found despite significantly higher aerobic capacities ($p < .05$) for the athletes (Graham and Andrew, 1973). The researchers concluded that the wide range in max O_2 debt values found between individuals could not be attributed to different levels of fitness of the subjects. Likewise, Freedman-Akabas et al. (1985), and Brehm and Gutin (1986), were unable to find any differences in post-exercise VO_2 or the time to baseline recovery between fit and unfit. Kaminsky et al. (1987), also concluded that the magnitude of EPOC was not statistically related to maximal aerobic power, and in fact, total EPOC was similar between subjects. They suggested that a higher metabolic heat load of aerobically fitter subjects who worked a greater absolute, albeit equal relative workload, may have been responsible for these similar magnitudes. They further propose it may be possible that subjects with greater maximal aerobic power have a more rapid time course of early recovery but similar total EPOC reflecting the influence of increased metabolic heat load. In support of this theory, Chad and Quigley (1991), found that trained individuals had significantly greater extended EPOCs than untrained individuals for 30 minutes of exercise at 50% VO_2max . This theory was further substantiated when Frey et al. (1993), found trained subjects produced greater magnitudes for the fast component of EPOC when compared with their untrained counterparts. They were exercising at 65% and 80% until 300 kcal was expended. As a result of the higher VO_2max for trained subjects, exercise at similar relative intensities required them to work at higher absolute VO_2 s during the exercise conditions compared with untrained subjects.

The higher VO_2 during exercise most likely accounts for the greater magnitude of the fast component, and thus would also explain the shorter exercise duration as trained subjects would be able to expend 300 kcal more quickly. However, the magnitude of the slow component was greater in the untrained for the 80% exercise when compared with the trained subjects. There were no trends observed for overall magnitude between the groups. Thus, the larger slow component may indicate a greater disturbance for which the untrained subjects had to recover from however, this was not evident in the overall magnitudes likely due to the higher initial VO_2 s. The training effects suggested by these researchers were made based on the duration of EPOC. The trained group had a shorter recovery time than the untrained group which was presumed to be the result of a faster recovery rate. Unfortunately, none of these studies actually examined recovery rate or performed any correlations, and in fact often used group means for comparison. The present research did not separate subjects into groups but rather, looked for direct correlations. Elliot et al. (1988), was the first study to reveal a direct correlation between $\text{VO}_{2\text{max}}$ and recovery energy expenditure. They reported a negative correlation ($r=-.7$, $p<.1$) between maximum oxygen uptake (expressed in ml.kg) and recovery energy expenditure (expressed in kcal) following thirty minutes of exercise at 80% $\text{VO}_{2\text{max}}$. However, the correlation is based on a 10% confidence interval and was derived from the results of only 6 subjects. Additionally, these subjects were male and female thus, the relationship needs substantiating. Further, EPOC was not used in any correlations thus, once again, this research differs from the present literature.

The general finding regarding the effects of endurance training on EPOC is, for a given absolute workload, temperature regulation is improved, lactate clearance is enhanced, and the hormonal response is decreased (Frey et al., 1993). Further, training has been shown to result in a faster recovery in VO_2 , Ve , and heart rate at the same relative workload.

(Hagberg et al., 1980a). However, research has been unable to show whether these training effects represent a particular fitness level which can be related to corresponding recovery patterns, or whether it merely reflects individual changes as a result of improved mechanical and physiological efficiency. From this and the above mentioned research, it can only be concluded that improved recovery, be it rate and/or magnitude, is a function of individual improvements in fitness. However, it should not be assumed that these observations are true in a cross-sectional sample. The individual with the higher aerobic fitness will not necessarily recover faster than one with a lower aerobic fitness. It has been shown that genetic factors may determine an interindividual responses to an identical training stimulus (Hamel et al., 1986; Bouchard et al., 1988). More specifically, it has been found that some mtDNA sequence variations are associated with individual differences in the VO_2max of sedentary subjects or with the changes induced by endurance training (Dionne et al., 1991). Thus, it is quite possible that these genetic factors carry over into how one recovers from an exercise bout.

Unfortunately, the information obtained so far in regards to the relationship between training status and recovery has dealt with aerobic power and submaximal exercise. The results of this research have shed some light on anaerobic power and its effects on the recovery period. These results indicate that anaerobic power is significantly correlated to the magnitude of EPOC. Since subjects were exercising at supramaximal levels, it is not surprising that EPOC would be representative of the energy systems utilized. However, the processes believed to be responsible for EPOC are mainly oxygen derived, thus, it was assumed that aerobic power would carry over into the recovery regardless of the intensity of the work bout. Anaerobic capacity has often been determined through a standardized anaerobic speed test, 20% grade at 8mph (Cunningham and Faulkner, 1969). Since this AST was one of the exercise conditions in this study, results from this test were used to

indicate anaerobic capacity. The anaerobic capacity is presumably highly dependent on the mass of the exercising muscles (Medbo et al., 1988), whereas the maxO_2 uptake is primarily a function of the capacity of the circulatory system when a large muscle mass is engaged (Hermansen, 1969). Gollnick and Hermansen (1973), estimated that aerobic metabolism supplies nearly 20% of the energy during exhaustive exercise of 10 seconds, and 40% of the energy for exhaustive exercise lasting 60 seconds. In contrast, the contribution of anaerobic metabolism to energy expenditure in a typical long-distance event, such as the 10 km run, is less than 3% (Weyand et al., 1994). Medbo and Tabata (1989), reported that the contribution from the two processes was found to be equal for exhausting exercise lasting 60 seconds. Clearly the aerobic energy system contributes significantly in the execution of high intensity, short duration exercise. Regardless of the contribution of the aerobic system to supramaximal exercise, this research was unable to find a significant relationship with EPOC. However, anaerobic capacity plays a significant role on the recovery period from supramaximal exercise. Rate of recovery was unrelated to either of the energy systems and would seem to be independent of training status.

CONCLUSIONS

These findings suggest that the rate and magnitude of recovery from supramaximal work is independent of VO_2max . Magnitude, on the other hand, has been shown to be dependent on anaerobic capacity with the magnitude of the slow component as the dominating factor for this relationship. The rate of recovery was highly individual but did not correlate to either of the energy processes. Rate of recovery was constant for a given individual over the three exercise conditions, thus indicating a fixed rate of recovery regardless of intensity or duration of the work bout, at least for exercise of a supramaximal nature. It has also been shown that the intensity and duration of the supramaximal exercise bout influences the resultant EPOC. An interaction effect between the two factors was postulated but could not be substantiated. Finally, the absence of a lactate-EPOC relationship does not lend support for lactate as a major contributor to the presence of an elevated oxygen consumption post-exercise.

RECOMMENDATIONS

Investigations of EPOC from supramaximal exercise are limited. The present findings of a relationship between anaerobic capacity and EPOC following supramaximal exercise were surprising and requires further study with a larger sample size. Therefore, in looking for relationships between aerobic power and EPOC, it would seem that submaximal protocols are best suited for this purpose. Although previous studies have done this, the various research designs and protocols have not provided consistency. In terms of recovery rate, it was surprising that neither energy systems seemed responsible. Whether aerobic power or anaerobic power is being investigated with EPOC, a longitudinal design should be implemented so that both intra- and inter-subject comparisons can be made. This should provide a better understanding of whether recovery rate is influenced by training and if so, does this trend exist for all individuals. As well, is there a relationship between recovery rate and the fitness level being examined. Up until this point, a training study has shown an improved recovery as a result of training but did not look at the relationship between the two. (Hagberg et al., 1980). As for the cross-sectional studies published, results are inconclusive. Therefore, a longitudinal study can provide a clearer picture of training influences on recovery over time as well as between individuals at any given time. Finally, the investigation of the exercise-EPOC relationship needs to be addressed using women as the sample. The investigations thus far have predominately involved men, as using women involves controlling for phase of menstrual cycle as this influences temperature. However, such investigations are needed as physiological differences between men and women may elicit differences in recovery patterns.

APPENDIX

Table 7: Individual and Mean EPOC Data (litres)

Subject	AST 20%	AST 15%- 1 min	AST 2 min
1	6.28	6.88	6.15
2	6.10	5.82	6.15
3	8.98	8.13	8.62
4	8.34	6.66	8.89
5	9.51	8.11	10.53
6	9.54	8.06	13.20
7	9.90	9.75	8.03
8	9.65	9.17	9.21
9	11.83	6.57	12.83
10	6.22	7.39	8.0
11	12.28	7.08	9.50
12	9.79	7.06	10.33
Mean	9.04	7.56	9.29
SD	2.03	1.13	2.22

Table 8: Individual and Mean EPOC Data for the fast and slow components (litres)

Subject	AST 20%		AST 15%- 1 min		AST 2 min	
	fast	slow	fast	slow	fast	slow
1	3.71	2.04	4.29	2.23	4.47	1.42
2	4.77	2.46	4.09	2.82	4.58	2.67
3	5.74	2.86	5.57	1.92	5.55	2.65
4	4.64	3.51	4.57	1.79	5.35	3.44
5	4.57	4.12	4.35	2.69	5.29	4.33
6	5.71	3.64	8.34	3.69	5.94	7.26
7	5.44	4.49	5.73	3.95	4.69	3.19
8	4.27	5.32	4.52	4.61	4.18	4.96
9	5.93	5.8	5.15	1.38	5.68	7.18
10	3.97	2.25	4.33	3.08	4.26	3.91
11	5.74	6.54	5.18	1.92	5.15	4.53
12	5.49	4.30	4.66	2.40	5.11	5.16
Mean	5.00	3.94	5.07	2.71	5.02	4.23
SD	.77	1.44	1.16	.97	.58	1.76

Table 9: Individual and Mean Peak Blood Lactate Data (mmol l)

Subject	AST 20%	AST 15%- 1 min	AST 2 min
1	11.66	10.94	11.92
2	11.32	13.62	11.56
3	8.04	12.78	15.54
4	14.58	13.34	12.56
5	13.02	16.62	15.54
6	14.56	12.92	15.08
7	13.26	14.94	16.00
8	15.30	10.30	16.80
9	15.90	12.30	18.62
10	14.74	15.80	18.72
11	15.20	10.86	18.86
12	15.90	11.72	17.62
Mean	13.62	13.01	15.74
SD	2.33	1.99	2.60

Table 10: Individual and Mean τ_1 Data (minutes)

Subject	AST 20%	AST 15%- 1 min	AST 2 min
1	2.14	2.13	2.45
2	2.28	2.39	2.27
3	2.55	2.5	2.66
4	2.77	2.16	2.25
5	2.46	2.79	2.25
6	2.82	2.49	2.38
7	2.20	2.40	2.05
8	3.51	3.0	2.57
9	2.86	1.65	3.13
10	3.03	2.25	2.40
11	2.41	2.52	2.34
12	2.13	1.95	2.10
Mean	2.60	2.35	2.40
SD	.42	.36	.29

Table 11: Individual and Mean τ_2 Data (minutes)

Subject	AST 20%	AST 15%- 1 min	AST 2 min
1	57.09	52.80	42.83
2	46.74	67.71	44.39
3	38.31	32.91	40.30
4	43.53	36.94	40.30
5	40.55	34.60	40.30
6	49.70	33.74	58.63
7	46.87	35.90	33.01
8	31.86	63.89	36.74
9	41.16	39.51	46.31
10	45.10	38.30	48.97
11	109.43	49.58	49.52
12	42.25	52.48	63.54
Mean	49.38	44.86	45.40
SD	19.90	12.11	8.79

REFERENCES

- Astrand, P.O., Hultman, E., Juhlin-Dannfelt, A., Reynolds, G. (1986). Disposal of lactate during and after strenuous exercise in humans. Journal of Applied Physiology, 61, 338-343.
- Bahr, R., Ingnes, I., Vaage, O., Sejersted, O.M., Newsholme, A. (1987). Effect of duration of exercise on excess postexercise O₂ consumption. Journal of Applied Physiology, 62 (2), 485-490.
- Bahr, R., Sejersted, O.M. (1990). Triglyceride/fatty acid cycling is increased after exercise. Metabolism, 39 (9), 993-999.
- Bahr, R., Opstad, P.K., Medbo, J.I., Sejersted, O.M. (1990). Strenuous prolonged exercise elevates resting metabolic rate and causes reduced mechanical efficiency. Acta Physiologica Scandinavica, 114, 555-563.
- Bahr, R., Sejersted, O.M. (1991). Effect of intensity of exercise on excess postexercise O₂ consumption. Metabolism, 40 (8), 836-841.
- Bahr, R., Sejersted, O.M. (1991). Effect of feeding and fasting on excess postexercise metabolism. Journal of Applied Physiology, 68 (6), 2362-2368.
- Bahr, R., Gronnerod, O., Sejersted, O.M. (1992). Effect of supramaximal exercise on excess postexercise O₂ consumption. Medicine and Science in Sports and Exercise, 16(1), 29-43.
- Baldwin, K.M., Campbell, A.M., Cooke, D.A. (1977). Glycogen, lactate, and alanine changes in muscle fiber types during graded exercise. Journal of Applied Physiology, 43, 288-291.
- Bangsbo, J., Gollnick, P.D., Graham, T.E. (1990). Anaerobic energy production and O₂ deficit-debt relationship during exhaustive exercise in humans. Journal of Physiology, 422, 539-559.
- Belcastro, A.N. Seminar in: The possible mechanisms of fatigue for anaerobic and aerobic exercise. UBC. November 10, 1992.
- Belcastro, A.N., Bonen, A. (1975). Lactic acid removal rates during controlled and uncontrolled recovery exercise. Journal of Applied Physiology, 39 (6): 932-936.

- Berg, K.E. (1991). Comparison of energy expenditure in men and women at rest and during exercise recovery. Journal of Sports Medicine and Physical Fitness, 31, 351-356.
- Boileau, R.A., Misner, J.E., Dykstra, G.L., Spitzer, T.A. (1983). Blood lactic acid removal during treadmill and bicycle exercise at various intensities. The Journal of Sports Medicine, 23, 159-165.
- Bond, V., Adams, R.G., Tearney, R.J., Gresham, K., Ruff, W. (1991). Effects of active and passive recovery on subsequent isokinetic muscle function. The Journal of Sports Medicine and Physical Fitness, 31, (3), 357-361.
- Bonen, A., Belcastro, A.N. (1977). A physiological rationale for active recovery exercise. Canadian Journal of Applied Sport Sciences, 2, 63-65.
- Bonen, A., Belcastro, A.N. (1976). Comparison of self-selected recovery methods on lactic acid removal rates. Medicine and Science in Sports and Exercise, 8, (3), 176-178.
- Brehm, B.A., Gutin, B. (1986). Recovery energy expenditure for steady state exercise in runners and nonexercisers. Medicine and Science in Sports and Exercise, 18 (2), 205-210.
- Brooks, G.A. (1991). Current concepts in lactate exchange. Medicine and Science in Sports and Exercise, 23, (8), 895-906.
- Brooks, G.A. (1986). The lactate shuttle during exercise and recovery. Medicine and Science in Sports and Exercise, 18, (3), 360-368.
- Buckley, J.D., Scroop, G.C., Catchside, P.G. (1993). Lactate disposal in resting trained and untrained forearm skeletal muscle during high intensity leg exercise. European Journal of Applied Physiology, 67, 360-366.
- Cafarelli, E., Sim, J., Carolan, B., Liebesman, J. (1990). Vibratory massage and short-term recovery from muscular fatigue. International Journal of Sports Medicine, 11, (6), 474-478.
- Chad, K.E., Quigley, B.M. (1991). Exercise intensity: effect on post exercise oxygen uptake in trained and untrained women. Journal of Applied Physiology, 70 (4), 1713-1719.

- Chad, K.E., Wenger, H.A. (1985). The effects of duration and intensity on the exercise and post exercise metabolic rate. The Australian Journal of Science and Medicine in Sport, 17 (4), 14-18.
- Chad, K.E., Wenger, H.A. (1988). The effect of exercise duration on the exercise and post-exercise oxygen consumption. Canadian Journal of Sport Sciences, 13 (4), 204-207.
- Challiss, R.A., Arch, J.R., Newsholme, E.A. (1984). The rate of substrate cycling between fructose 6-phosphate and fructose 1.6-biphosphate in skeletal muscle. Biochemistry Journal, 221, 153-161.
- Cortes, C.W., Kreider, R.B., Al-Mandalawi, S., Lamberth, J., Johnson, K.D., Thompson, W.R., Anderson, H.N. (1988). Lactate clearance during active and passive recovery. Annals of Sports Medicine, 4, (1), 26-28.
- Cotes, J.E., Meade, F. (1959). Physical training in relation to the energy expenditure of walking and factors controlling respiration during exercise. Ergonomics, 2, 195-205.
- Crescitelli, F., Taylor, C. (1944). The lactate response to exercise and its relationship to physical fitness. American Journal of Physiology, 141, 630-639.
- Cunningham, D.A., Faulkner, J.A. (1969). The effect of training on aerobic and anaerobic metabolism during a short exhaustive run. Medicine and Science in Sports and Exercise, 1, 65-69.
- Douglas, F.G., Becklake, M.R. (1968). Effect of seasonal training on maximal cardiac output. Journal of Applied Physiology, 25, 600-605.
- Eklblom, B., Astrand, P.O., Saltin, B., Stenberg, J., Wallstrom, B. (1968). Effect of training on circulatory response to exercise. Journal of Applied Physiology, 24, 518-528.
- Elliot, D.L., Goldberg, L., Kuehl, K.S. (1988). Does aerobic conditioning cause a sustained increase in the metabolic rate? American Journal of Medicine and Science, 296 (4), 249-251.
- Evans, B.W., Cureton, K.J. (1983) Effect of physical conditioning on blood lactate disappearance after supramaximal exercise. British Journal of Sports Medicine, 17, (1), 40-45.

- Freedman-Akabas, S., Colt, E., Kissileff, H.R., Pi-Sunyer, X.F. (1985). Lack of sustained increase in VO₂ following exercise in fit and unfit subjects. American Journal of Clinical Nutrition, 41, 545-549.
- Frey, G.C., Byrnes, W.C., Mazzeo, R.S. (1993). Factors influencing excess post-exercise oxygen consumption in trained and untrained women. Metabolism, 42 (7), 822-828.
- Gaesser, G.A., Brooks, G.A. (1984). Metabolic bases of excess post-exercise oxygen consumption: a review. Medicine and Science in Sports and Exercise, 16, (1), 29-43.
- Girandola, R.N., Katch, F.I. (1973). Effects of physical conditioning on changes in exercise recovery O₂ uptake and efficiency during constant-load ergometer exercise. Medicine and Science in Sports and Exercise, 5 (4), 242-247.
- Gisolfi, C., Robinson, S., Turrell, E.S. (1966). Effects of aerobic work performed during recovery from exhausting work. Journal of Applied Physiology, 21, (6), 1767-1772.
- Gollnick, P.D., Bayly, W.M., Hodgson, D.R. (1986). Exercise intensity, training, diet and lactate concentration in muscle and blood. Medicine and Science in Sports and Exercise, 18, 334-340.
- Gollnick, P.D., Hermansen, L. (1973). Biochemical adaptations to exercise: anaerobic metabolism. Exercise and Sport Science Review, 1, 1-43.
- Gore, C.J., Withers, R.T. (1990). Effect of exercise intensity and duration on postexercise metabolism. Journal of Applied Physiology, 68 (6), 2362-2368.
- Gore, C.J., Withers, R.T. (1990). The effect of exercise intensity and duration on the oxygen deficit and excess post-exercise oxygen consumption. European Journal of Applied Physiology, 60, 169-174.
- Hagberg, J.M., Hickson, R.C., Ehsani, A.A., Holloszy, J.O. (1980). Faster adjustment to and recovery from submaximal exercise in the trained state. Journal of Applied Physiology, 48 (2), 218-224.
- Hagberg, J.M., Mullin, J.P., Nagle, F.J. (1980). Effect of work intensity and duration on recovery O₂. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 48 (3), 540-544.

- Henry, F.M., Berg, W.E. (1950). Physiological and performance changes in athletic conditioning. Journal of Applied Physiology, 3, 103-111.
- Hermansen, L. (1969). Anaerobic Energy Release. Medicine and Science in Sports and Exercise, 1 (1), 32-38.
- Hill, A.V., Lupton, H. (1923). Muscular exercise, lactic acid and the supply and utilization of oxygen. Q.J. Med., 16, 135-171.
- Jacobs, I. (1986). Blood lactate implications for training and sports performance. Sports Medicine, 3, 10-25.
- Jorfeldt, L. (1970). Metabolism of L-(+)-lactate in human skeletal muscle during exercise. Acta Physiologica Scandinavica, Suppl. 338.
- Kaminsky, L.A., Padjen, S., Lattam-Saeger, J. (1990). Effect of split exercise sessions on excess post-exercise oxygen consumption. British Journal of Sports Medicine, 24 (2), 95-98.
- Karlsson, J., Bonde-Peterson, F., Henricksson, J., Knuttgen, H.G. (1975). Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. Journal of Applied Physiology, 38, 763-767.
- Kaminsky, L.A., Kanter, M.M., Lesmes, G.R., Laham-Saeger, J. (1987). Excess oxygen consumption following exercise of different intensity and duration. Canadian Journal of Sport Sciences, 12 (4), 237-239.
- Katch, V.L., Gilliam, T., Weltman, A. (1978). Active versus passive recovery from short-term supramaximal exercise. The Research Quarterly, 49 (2), 153-161.
- Knehr, C.A., Dill, B., Neufeld, W. (1942). Training and its effects on man at rest and at work. American Journal of Physiology, 136, 148-156.
- Knuttgen, H.G. (1970). Oxygen debt after submaximal physical exercise. Journal of Applied Physiology, 29(5), 651-657.
- Maehlum, S., Grandmontagne, M., Newsholme, E.A., Sejersted, O.M. (1986). Magnitude and duration of exercise postexercise oxygen consumption in healthy young subjects. Metabolism, 35 (5), 425-429.

- Maresh, C.M., Abraham, A., DeSouza, M.J. et al. (1992). Oxygen consumption following exercise of moderate intensity and duration. European Journal of Applied Physiology, 65, 421-426.
- Margaria, R., Edwards, H.T., Dill, D.B. (1933). The possible mechanisms of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction. American Journal of Physiology, 106, 689-715.
- Martin, A.D., Spenst, L.F., Drinkwater, D.T., Clarys, J.P. (1990). Anthropometric estimation of muscle mass in men. Medicine and Science and Sports and Exercise, 22(5), 729-733.
- Mazzeo, R.S., Brooks, G.A., Schoeller, D.A., Budinger, T.F. (1986). Disposal of blood lactate in humans during rest and exercise. Journal of Applied Physiology, 1, 232-241.
- McLellan, T.M., Skinner, J.S. (1982). Blood lactate removal during active recovery related to the anaerobic threshold. International Journal of Sports Medicine, 3, (4), 224-229.
- McLoughlin, P., McCaffery, N., Moynihan, J.B. (1991). Gentle exercise with a previously inactive muscle group hastens the decline of blood lactate concentration after strenuous exercise. European Journal of Applied Physiology, 62, 274-278.
- Medbo, J.I., Mohn, A.C., Tabata, I., Bahr, R., Vaage, O., Sejersted, O.M. (1988). Anaerobic capacity determined by maximal accumulated O₂ deficit. Journal of Applied Physiology, 64 (1), 50-60.
- Medbo, J.I., Sejersted, O.M. (1985). Acid base and electrolyte balance after exhausting exercise in endurance-trained and sprint-trained subjects. Acta Physiologica Scandinavica, 125, 97-109.
- Medbo, J.I., Tabata, I. (1989). Relative importance of aerobic and anaerobic energy release during short lasting, exhausting bicycle exercise. Journal of Applied Physiology, 67, 1881-1886.
- Milesis, C.A., Fougeron, M.G., Graham, H. (1982). Effect of active versus passive recovery on cardiac time components. Journal of Sports Medicine, 22, 147-151.

- Oyono-Enguelle, S., Marbach, J., Heitz, A., Oh, C., Gartner, M., Pape, A., Vollmer, J.C., Freund, H. (1990). Lactate removal ability and graded exercise in humans. Journal of Applied Physiology, 68, (3), 905-911.
- Parkhouse, W.S., McKenzie, D.C., Hochachka, P.W., Mommsen, T.P., Ovalle, W.K., Shinn, S.L., Rhodes, E.C. (1982). Muscle buffering capacity, fiber composition and anaerobic capacity of elite athletes. (Abstract). Medicine and Science in Sports and Exercise, 14, 132.
- Poortmans, J.R., Bossche, J.D.V., LeClerc, R. (1978). Lactate uptake by the inactive forearm during prolonged leg exercise. Journal of Applied Physiology, 45, 835-839.
- Roberts, G.A. (1994). A metabolic comparison of isokinetic and free sprinting. Unpublished Masters Thesis. The University of British Columbia.
- Robinson, S., Harmon, P.M. (1941). The effects of training and of geletin upon certain factors which limit muscular work. American Journal of Physiology, 133, 161-169.
- Sahlin, K., Alvestrand, A., Brandt, R., Hultman, E. (1978). Intracellular pH and bicarbonate concentration in human muscle during recovery from exercise. Journal of Applied Physiology, 45, 474-480.
- Sedlock, D.A. (1991). Effect of exercise intensity on postexercise energy expenditure in women. British Journal of Sports Medicine, 25 (1), 38-40.
- Sedlock, D.A. (1992). Post-exercise energy expenditure after cycle ergometer and treadmill exercise. Journal of Applied Sport Science Research, 6, 19-23.
- Sedlock, D.A., Fissinger, J.A., Melby, C.L. (1989). Effect of exercise intensity and duration of post-exercise energy expenditure. Medicine and Science in Sports and Exercise, 21, 662-666.
- Seibers, L.S., McMurray, R.G. (1981). Effects of swimming and walking on exercise recovery and subsequent swim performance. Research Quarterly, 52, 68-75.
- Shephard, R.J. Biochemistry of Physical Activity Charles C. Thomas Publisher, Illinois, 1984.

- Smith, J., McNaughton, L. (1993). The effects of intensity of exercise on excess post-exercise oxygen consumption and energy expenditure in moderately trained men and women. European Journal of Applied Physiology, 67, 420-425.
- Stainsby, W.N., Brooks, G.A. (1990). Control of lactic acid metabolism in contracting muscles during exercise. Exercise and Sport Sciences Reviews, 18, 29-63.
- Stanley, W.C. (1991). Myocardial lactate metabolism during exercise. Medicine and Science in Sports and Exercise, 23, (8), 920-924.
- Stanley, W.C., Gertz, E.W., Wisneski, J.A., Neese, R.A., Brooks, G.A. (1984). Glucose and lactate turnover in man during rest and exercise studied with simultaneous infusion of ^{14}C -glucose and ^{13}C -lactate. Medicine and Science in Sports and Exercise, 16, 136.
- Taylor, H.L., Buskirk, E., Henschel, A. (1953). Maximal oxygen intake as an objective measure of cardiorespiratory performance. Journal of Applied Physiology, 8, 73-80.
- Tesch, P.A., Wright, J.E. (1983). Recovery from short-term intense exercise its relation to capillary supply and blood lactate concentration. European Journal of Applied Physiology, 52, 98-103.
- Watson, R.C., Hanley, R.D. (1986). Application of active recovery techniques for a simulated ice-hockey task. Canadian Journal of Applied Sport Sciences, 11, (2), 82-87.
- Weltman, A., Stamford, B.A., Moffat, R.J., Katch, V.L. (1982). Exercise recovery, lactate removal, and subsequent high intensity exercise performance. Research Quarterly, 48, 786-796.
- Weyand, P.G., Cureton, K.J., Conley, D.S., Sloniger, M.A., Liu, Y.L. (1984). Peak oxygen deficit predicts sprint and middle-distance track performance. Medicine and Science in Sports and Exercise, 26 (9), 1174-1180.
- Whipp, B.J., Wasserman, K. (1972). Oxygen uptake kinetics for various intensities of constant-load work. Journal of Applied Physiology, (3), 351-356.

Wolfe, R.R., Klein, S., Carraro, F., Weber, J.M. (1990). Role of triglyceride-fatty acid cycle in controlling fat metabolism in humans during and after exercise. American Journal of Applied Physiology, 258, E382-E389.

Ziegenfuss, T.N., Sedlock, D.A. (1992). Post-exercise energy expenditure following intermittent exercise. (Abstract) Medicine and Science in Sports and Exercise, 24 (5), S97.