NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Reproduction in full or in part of this microform is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30, and subsequent amendments.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

La reproduction, même partielle, de cette microforme est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30, et ses amendements subséquents.

The Effects of Leg Cycling Training on Lactate Threshold and Maximal Oxygen Consumption Measured During Leg Cycling and Arm Cranking Exercise

by

Nancy E. Saumure

University of Ottawa

A thesis
presented to the University of Ottawa
in fulfillment of the
thesis requirement for the degree of
Master of Science
in
Kinanthropology

ISBN 0-315-68078-4

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

DEDICATION

I dedicate this thesis to my husband Denis, whose encouragement, patience and love have enabled me to complete this work, and to my mother for her constant love and support..

ACKNOWLEDGEMENTS

In completing this thesis, I find that there are a number of people to whom I owe a great deal of thanks.

I must first express my extreme gratitude to Dr. J. Thoden for his guidance, experienced opinion, and support throughout the course of my studies.

Sincere thanks to Dr. A. Reed and Dr. F. Reardon for their helpful suggestions in the development and presentation of this research.

A special thank you is extended to my experimental and pilot subjects for their considerable time, effort and perseverance. Without their dedication this study would not have been possible.

Many thanks also to those who have given generously of their time during the testing sessions, in particular Lynn Belisle, Rob Seguin, Glen Kenny and Suzanne Cormier.

Finally, I must express my utmost thanks to all of my family (too numerous to list) and special friends like Brenda Elliott, Sheila Windle, Meg Lunney, Rita Petrocco and especially Denis, whose love & friendship have brought me this far.

ABSTRACT

The purpose of this study was to determine if the training effects on lactate threshold (LT) and maximal oxygen consumption (VO_2max) are specific to musculature involved in training or if there is evidence of a general training effect, such that adaptations are also found during exercise with untrained muscle groups. Seven moderately active male students participated in an eight week progressive endurance training program that involved leg cycling at specific intensities above and below the pre-training LT to give a total of 30 minutes of training above LT, three times per week. All subjects were tested before and after training for LT and VO_2max while performing leg cycling and arm cranking exercises. VO_2max showed a significant increase during both leg cycling and arm cranking exercise following training. Conversely, increases in both absolute and relative LT were confined to leg cycling exercise only. It is suggested that peripheral adaptive responses of oxidative capacity within the trained muscles are primarily responsible for the specificity of the LT response, while cardiovascular adaptations were beneficial to VO_2max of both of the muscle groups tested. Furthermore, the significant improvement in relative LT during leg cycling and of VO_2max to both arm and leg exercise suggests that adaptive responses of LT and VO_2max to training are not governed by the same physiological processes. Therefore, it was concluded that, for the conditions of this experiment, the concept of specificity of training applies to LT but not to VO_2max when comparing exercise modalities which involve separate musculature.

CONTENTS

FIGURES

TABLES

THE PROBLEM

INTRODUCTION AND RATIONALE

Training and athletic performance have been key research issues for many investigators. In the past, maximum aerobic capacity or maximal oxygen consumption (VO_2max) was thought to be the primary limiting factor in endurance performance (Saltin, Hartley, Kilborn, & Astrand, 1969) and was, therefore, emphasized in evaluating athletes. VO_2max is sensitive to training with both central cardiovascular transport and local active tissue adaptations responsible for its improvement. However, local muscular adaptations are more directly linked to the adaptations which are specific to a particular mode of exercise and VO_2max values are, therefore, highly dependent upon the mode of exercise chosen for testing. Also, the VO_2max response to training demonstrates some degree of specificity, similar to testing results, since changes in VO_2max are greatest for exercise involving the trained musculature (Clausen, Klausen, Rasmussen, & Trap-Jensen, 1973; Henriksson, 1977; Ridge, Pyke, & Roberts, 1976; Saltin, Nazar, Costill, Stein, Jansson, Essen et al., 1976). However, VO_2max has also been shown to improve during exercise involving untrained musculature (Clausen et al., 1973; Lewis, Thompson, Areskog, Vodak, Marconyak, DeBusk et al., 1980; Loftin, Boileau, Massey, & Lohman, 1988; Rosler, Hoppeler, Conley, Claassen, Gehr, & Howald, 1985; Saltin et al., 1976). Therefore, these studies indicate a general training effect on VO_2max but with greater adaptations in the training mode.

Lactate threshold (LT), also commonly referred to as anaerobic threshold, is another variable which has been identified as a limiting factor in endurance performance and should be addressed in consideration of specificity of testing and training. Numerous studies have suggested that the additional consideration of LT is at least as, if not more important, than VO_2max in monitoring endurance performance and the prediction thereof (Coyle, Coggan, Hopper, & Walter, 1988; Farrell, Wilmore, Coyle, Billing, & Costill, 1979; Jacobs, Schele, & Sjodin, 1985; Sjodin & Jacobs, 1981; Sjodin, Jacobs, & Svendenhag, 1982). LT is also thought to be useful in identifying an appropriate and optimal training intensity for athletes, for the purpose of placing a high degree of stimulation on oxidative metabolism (Kinderman, Simon, & Keul, 1979). Therefore, the effect of training on LT is a pertinent issue for investigators, coaches and athletes, particularly those who are involved in several competitive endurance sports, such as triathalons.

LT represents the upper limit of continuous exercise and is important to aerobic training and performance in events such as cycling, cross country skiing, rowing and running (Droghetti, Borsetto, Casoni, Cellini, Ferrari, Paolini et al., 1985), all of which depend upon some portion of the performance being conducted at the highest possible rate of work for extended periods. Kinderman et al. (1979) defined the exercise intensity during incremental exercise which stimulates rapid lactate accumulation, at a rate which departs from a curvilinear increase to become exponential (approximately 4 mmol/l lactate), as anaerobic threshold (AT). The exercise intensity, during incremental exercise, after which BLa concentration increases by 1 mmol/l may also be used to identify this point (Hagberg & Coyle, 1983; Simon, Berg, Dickhugh, Simon-Alt, & Keul, 1981). In this paper, the AT intensity will be referred to as LT, in order to avoid confusion with anaerobic threshold as defined by respiratory parameters (Wasserman, Whipp, Koyal, & Beaver, 1973). It is this

intensity of work, or physiological landmark, with which athletes are primarily concerned, since continuous exercise at a higher intensity will result in a loss of equilibrium between muscle lactate production and its elimination and in a continuous increase of BLa (Stegmann & Kindermann, 1982). This will eventually force a reduction in work rate. LT, the exercise intensity above which lactate production will exceed lactate removal, is commonly described in terms of the VO_2 which exists at the time this occurs and is referred to as absolute lactate threshold (ALT). It may also be expressed as occurring at a percent of VO_2max and referred to as relative lactate threshold (RLT). The term LT is also used by Ivy, Withers, Van Handel, Elger, and Costill (1980) to indicate the work rate at which the first significant rise in BLa occurs. However, this latter definition is referred to as the aerobic threshold by Kinderman et al. (1979).

High concentrations of BLa are associated with many types of exercise induced fatigue. They are thought to have a negative influence on endurance performance by lowering pH, thus inhibiting fatty acid mobilization or having adverse effects on the contractile properties of the muscle, the myofibrils, and enzymes of the glycolytic and oxidative pathways (Gollnick, Bayly, & Hodgson, 1986). Therefore, it is advantageous in endurance competitions to have a higher LT in order to delay the onset of significant pH reduction.

The rate of BLa accumulation may be reduced following endurance training due to diminished lactate production and/or enhanced lactate removal (Donovan & Brooks, 1983; Walsh & Bannister, 1988). A number of training studies have found changes specific to the trained muscle or specific to exercise responses with trained musculature which affect submaximal exercise BLa concentration. These include reduced VO_2on response time (Cerretelli, Pendergast, Paganelli, & Rennie, 1979), increased hepatic-

splanchnic blood flow (Clausen et al., 1973), increased SDH activity (Henriksson, 1977; Saltin et al., 1976), increased subsarcolemal mitochondria (Hoppeler, Howald, Conley, Lindstedt, Claassen, Vock et al. 1985), increased capillary per fibre ratio and mitochondrial volume density (Rosler et al., 1985), and decreased glycogen depletion (Saltin et al., 1976). These findings suggest that the effect of endurance training on LT will be specific to trained musculature, but there is little direct data to support this hypothesis.

Although a great deal of research has focused on the factors influencing lactate production, the identification of threshold variables, and the effect of training on BLa concentrations, the specific effect of training LT has received relatively less attention compared to the specific effect of training VO_2max. Therefore, this research was designed to examine if the effects of training LT are specific to the muscles which are involved in the training. Exercise responses to training were compared between the trained mode of exercise, leg cycling, and an untrained mode of exercise, arm cranking.

STATEMENT OF THE PROBLEM

The primary objectives of this study were:

1. to determine if changes in lactate threshold measured during exercise with trained musculature (legs) would also be evident during exercise with untrained musculature (arms).

2. to determine if changes in maximal oxygen consumption measured during exercise with trained musculature (legs) would also be evident during exercise with untrained musculature (arms).

Other subobjectives of this study were:

3. to investigate the effect of training on heart rate measured during submaximal exercise with trained musculature (legs) and untrained musculature (arms).

4. to investigate the effect of training on heart rate, ventilation and oxygen pulse associated with LT for arm and for leg exercise

EXPERIMENTAL HYPOTHESIS

1. Due to the findings of previous studies showing that adaptations of factors which could affect LT are specific to the trained musculature, it was expected that training induced changes in LT would be evident only during exercise with trained musculature (legs). No changes were expected in LT for arm cranking exercise.

2. Due to previous findings which showed that VO_2max may exhibit a general training effect, it was expected that VO_2max would exhibit an increase following training during exercise with both trained and untrained musculature.

OPERATIONAL DEFINITIONS

Lactate threshold (LT): the exercise intensity which identifies the upper limit on constant workload endurance exercise beyond which blood lactate concentrations continue to increase. This value, represented in oxygen consumption (VO_2) units, is initially determined at the exercise intensity during continuous progressive, aerobic exercise above which blood lactate concentration increases by a rate greater than or equal to 1 mmol/l per 2 min stage that persists in subsequent exercise increments, or

the point of exponential rise on a lactate versus resistance graph. This value is then verified by constant workload tests.

Absolute lactate threshold (ALT): LT expressed in VO_2 units - l/min.

Relative lactate threshold (RLT): LT with respect to VO_2max. It is expressed as a percentage of maximum oxygen consumption (%VO_2max).

Maximum oxygen consumption (VO_2max): the highest rate of oxygen consumption which is measured during a progressive, aerobic, exercise test with a minimum duration of six minutes. This criterion is evidenced by a plateau in VO_2, an increase no greater than 2 ml/kg/min in VO_2, or a fall in VO_2 compared to the immediately previous increment of exercise.

Absolute maximal oxygen consumption (AVO_2max): is VO_2max expressed in l/min.

Relative maximal oxygen consumption (RVO_2max): is VO_2max expressed in ml/kg/min.

LIMITATIONS OF THE STUDY

Due to the relatively small number of subjects (n=7) the results decrease in statistical power.

Training effects found to be specific to the trained musculature may not be found in all cases, especially when the activities chosen involve some of the same muscle groups. The results of this study may, however, suggest the degree to which there is a specific effect of training, related to such variables as LT, when completely separate muscle groups are involved. Also, any conclusions drawn from this study may only be

projected with confidence to a group of similarly trained subjects of the same age, performing similar exercise activities and following a related intermittent training regime.

The subjects were requested to refrain from regular endurance activity, i.e. 2 - 3 times per week for 15 or more minutes, particularly activity with heavy arm involvement. They were asked to monitor their activity with log books. However, while the subjects were not engaged in regular training for endurance or competitive activity, maintenance of normal lifestyle which includes spontaneous activity was accepted.

ABBREVIATIONS

ADP - Adenosine diphosphate

AMP - Adenosine monophosphate

ALT - Absolute lactate threshold

AT - Anaerobic threshold

ATP - Adenosine triphosphate

AVO_2max -Absolute maximum oxygen consumption

BLa - Blood lactate

Ca^{2+}- Calcium ion

CO_2 - Carbon dioxide

CO - Cytochrome oxidase

CP - Creatine phosphate

FT - Fast twitch muscle fibres

GADPH - glyceraldehydephosphate dehydrogenase

G-6-P - Glucose-6-phosphate

GP shuttle - α-glycerophosphate shuttle

H^+ - Hydrogen ion

HAD - 3-hydroxyacyl-COA dehydrogenase

H-LDH - Heart specific LDH isozyme

H_2O - Water

IM - Myofibrillar ATPase intermediate fibres

IMP - Inosine monophosphate

K^+ - Potassium ion

LDH - Lactate dehydrogenase

LT - Lactate threshold

LTHR - Lactate threshold heart rate

LTO_2P - Lactate threshold oxygen pulse

LTVe - Lactate threshold ventilation

LTWL - Lactate threshold workload

MA shuttle - Malate-aspartate shuttle

MAP - Maximal aerobic power

MaxHR - Maximal heart rate

MaxVe - Maximal ventilation

M-LDH - Muscle specific LDH isozyme

NADH - Reduced nicotinamide adenine dinucleotide

NAD^+ - Oxidized nicotinamide adenine dinucleotide

NH_4^+ - Ammonium ion

O_2 - Oxygen

OBLA - Onset of blood lactate accumulation

OPLA - Onset of plasma lactate accumulation

PCO_2 - Partial pressure of CO_2

PDH - Pyruvate dehydrogenase

PFK - Phosphofructokinase

pH_i - Intracellular pH

pH_o - Extracellular pH

Pi - Inorganic phosphate

PO_2 - Partial pressure of oxygen

RLT - Relative lactate threshold

rpm - Revolutions per minute

RVO_2max -Relative maximum oxygen consumption

ST - Slow twitch muscle fibres

SubHR - Heart rate at 75% AVO_2max

$t_{1/2}$ VO_2on - Half time of the oxygen uptake on-response

Ve - Minute ventilation

VM - Marathon velocity

VO_2 - Oxygen consumption per minute

VO_2max - Maximal oxygen consumption

VOBLA - Treadmill velocity at OBLA

VT - Ventilatory threshold

REVIEW OF LITERATURE

INTRODUCTION

This study was conducted to determine if training adaptations, in LT and VO_2max in particular, are specific to trained musculature or if there is a general training effect, such that adaptations are also observed during exercise with untrained musculature. Certain areas of the literature must be reviewed in order to properly investigate this problem.

Since it is important to understand the metabolic origins of lactic acid, the first section will address the mechanisms of lactate production through the metabolic pathways, its function, and its role in the development of fatigue.

The next section will introduce the factors influencing blood lactate concentrations. These will be subdivided into the production and removal of lactic acid and the variables which affect these processes.

The third section will introduce the anaerobic and lactate threshold phenomenons. The identification of LT and its importance will be discussed.

The fourth section will examine the effect of training on lactate kinetics and the metabolic adaptations which could account for any changes. Research which supports the specificity of training LT will also be reviewed.

Finally, some considerations for the training and testing protocols will be examined.

METABOLIC PATHWAYS AND LACTIC ACID

ATP Generation for Muscular Contraction

Physical movement may occur as a consequence of skeletal muscle contraction. This process is dependent upon the availability of adenosine triphosphate (ATP). It is the breaking of high energy bonds within ATP which supplies the energy for cross bridge movement between the actin and myosin filaments of the myofibril, thereby producing movement. Without ATP regeneration at the contractile site, the muscle is only capable of a few contractions. ATP is also required for relaxation from muscular contractions. The cross bridges are broken when the a new ATP molecule attaches to the myosin head (Astrand & Rodahl, 1986). There are several different metabolic pathways available for the repletion of ATP in man.

Intramuscularly stored creatine phosphate (CP) is required for anaerobic alactic metabolism in which a phosphate group is transferred to an ADP molecule, a by-product of ATP splitting, in order to resynthesize ATP in one step. In the presence of the enzyme creatine kinase, CP and ADP will react to produce ATP and creatine. However, CP stores are limited in a muscle fibre and can only cover the energy costs in maximal effort for less than 10 seconds (Astrand & Rodahl,1986). Therefore, the rate of ATP resynthesis via anaerobic alactic metabolism declines rapidly (McGrail, Bonen, & Belcastro, 1977), and the net ATP contribution for this means of energy production is small during continuous physical activity.

The two primary metabolic processes involved in continuous work are anaerobic and aerobic metabolism. The catabolism of glucose or glycogen to form pyruvate are common pathways for both types of metabolism. However, it is the metabolic pathway which pyruvate subsequently follows that determines if ATP is generated anaerobically or aerobically. The relative contributions of these two systems to a given activity depend upon factors such as its duration, intensity, availability and type of stored fuels, fibre type composition of the muscles involved, and the trained state of the active muscles.

Glycolysis

Glycolysis is the 10 step sequence of glucose catabolism to pyruvate with the concomitant release of a net two ATP from specific reactions (see Figure 1). Glucose is the substrate required and converted into glucose-6-phosphate (G-6-P) in the first reaction. Alternatively, G-6-P may be formed from the catabolism of glycogen stored in the muscle. This is known as glycogenolysis.

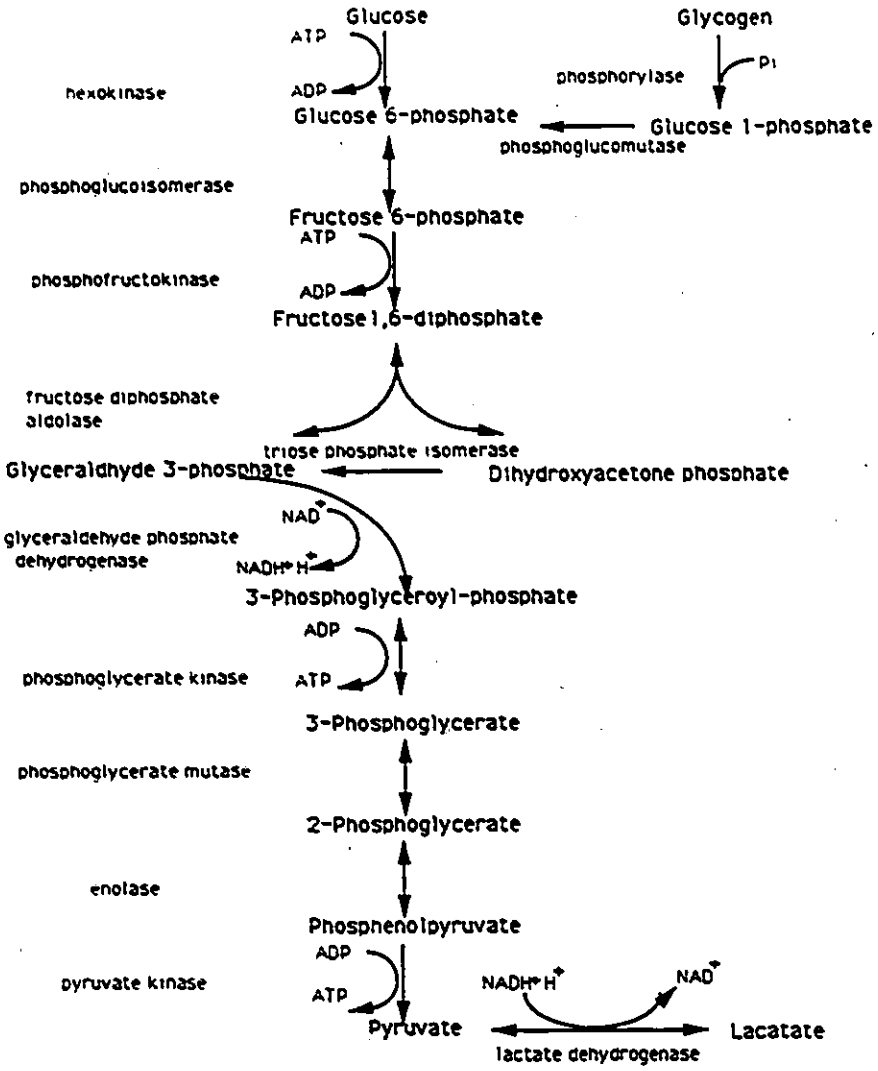

Figure 1. The glycolytic pathway. Adapted from Lehninger (1982)

Pyruvate

The formation of pyruvate in the cytoplasm is the branch point for anaerobic lactic and aerobic metabolism. In the presence of oxygen, aerobic metabolism, pyruvate may be oxidized, with the loss of its carboxyl group as carbon dioxide (CO_2), to form acetyl-coenzyme A. This reaction is catalyzed by the enzyme pyruvate dehydrogenase (PDH) (Lehninger, 1982). This acetyl group is then completely oxidized to CO_2 and water (H_2O), and 34 ATP are produced via Krebs Cycle and the Electron Transport Chain. Fatty acids and amino acids may also enter Krebs Cycle by being converted to acetyl-coenzyme A (Lehninger, 1982), and therefore, they represent another potential source of energy for muscular contraction.

In what is termed anaerobic lactic metabolism (O_2 not required), pyruvate does not enter Krebs Cycle but is reduced to lactate with the catalytic action of lactate dehydrogenase (LDH) (see Figure 2). Therefore, only the original net two or three ATP generated from the catabolism of glucose or glycogen, respectively, are produced.

Another metabolic fate of pyruvate is its transamination to form alanine. It is also a precursor in the synthesis of fatty acids and a product of fat metabolism (Wasserman, Beaver, Davis, Pu, Herber, & Whipp, 1985).

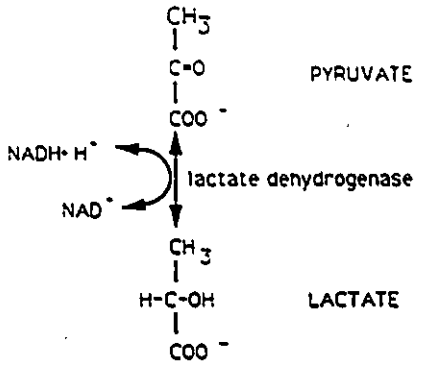

Figure 2. Reversible transformation of pyruvate to lactate. Adapted from Lehninger (1982)

Lactate Dehydrogenase (LDH)

Lactate dehydrogenase (LDH), E 1.1.1.27, catalyzes the following reversible reaction: Lactate + NAD^+ — Pyruvate + NADH + H^+

The LDH molecule is a tetramer with different combinations of M and H polypeptide chains yielding 5 isozyme forms: H_4, H_3M, H_2M_2, HM_3, and M_4. The LDH isozymes H_4 and H_3M (H-LDH) are predominant in the heart and favor the rapid oxidation of lactate to pyruvate. In contrast, the LDH isozymes M_4 and HM_3 (M-LDH) predominate in skeletal muscle and favor rapid reduction of pyruvate to lactate. The other isozymes have intermediate kinetic properties. Other tissues contain a mixture of the five possible forms (Lehninger, 1982; Sjodin, Thorstensson, & Karlsson, 1976).

Since M-LDH has the greatest catalytic activity (VMax) of any glycolytic enzyme, lactic acid production is possible with low intensity exercise. In fact, it exceeds the activity of enzymes providing alternate pathways for pyruvate metabolism (Brooks, 1985).

LDH isozymes within a muscle fibre are not necessarily mainly the M-LDH type. The types and relative distribution of the LDH isozymes in a muscle are dependent upon the fibre type(s), and this may be influenced by training (Skinner & McLellan, 1980). These topics will be discussed later with regard to influences on lactate production as well as training adaptations.

Lactic Acid/Lactate and its Role in Metabolism

Lactate is a small and easily diffusible molecule capable of moving rapidly from the cells in which it is produced to all water compartments (Gollnick & Hermansen, 1973). Lactate is simply the dissociated form of lactic acid (see Figure 3), and the two terms are often used interchangeably. Since lactic acid is a strong acid with a low dissociation constant (pk) of 3.86 (Lehninger, 1982), this molecule is found in the dissociated form, lactate, 99% of the time at normal pH (Mainwood & Renaud, 1985).

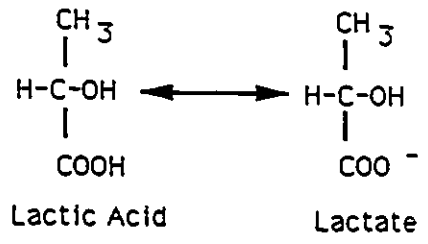

Figure 3. Lactic acid and its dissociated form - lactate

The role of lactate production in metabolism has not always been clear. In fact, in the early 1900's Hill and Myerhof proposed that it was the immediate energy donor for muscular contraction (Hollman, 1985). However, work by Lundsgaard in the 1930's

suggested that muscular contractions could occur without lactate production (Gollnick, Bayly, & Hodgson, 1986); therefore lactate was obviously not an energy donor. Hill, Long, and Lupton (1924) showed that large quantities of lactic acid are produced in muscles lacking oxygen, from which the oxygen insufficiency theory of lactate production has developed.

Today it is recognized that lactate production is a consequence of glycolysis and can occur in the presence as well as the absence of oxygen (Gollnick et al., 1986). It is an ongoing process within the resting and active individual (Brooks, 1986), forming when pyruvate and nicotinamide-adenine dinucleotide (NADH) are available to LDH regardless of how much O_2 is present (Holloszy & Coyle, 1984).

With the formation of lactate from pyruvate, the lactate molecule still contains approximately 93% of the energy available in a glucose molecule (Lehninger, 1982). Therefore, lactate production could do little to supplement aerobic ATP production, a suggested role of lactate production by those who believe a muscle fibre becomes anoxic and then mitochondrial respiration is limited. Lactate production must serve several metabolic function(s) in order for the cell to carry out processes yielding only 2 ATP anaerobically instead of a potential 38 ATP from oxidative phosphorylation of glucose (see Figure 4).

Anaerobic Catabolism of Glucose:

$$\text{Glucose} + 2\text{ATP} + 4\text{ADP} + 2\text{Pi} + 2\text{coenzyme} \longrightarrow 2\text{Lactic Acid} + 2\text{ATP} + 2H_2O + 2\text{coenzyme}$$

Aerobic Catabolism of Glucose:

$$\text{Glucose} + 38\text{ADP} + 38\text{Pi} + 6O_2 \longrightarrow 6CO_2 + 6H_2O + 38\text{ATP}$$

Figure 4. Anaerobic and aerobic catabolism of glucose. Adapted from Vander et al. (1980)

The rate of energy production is much higher when lactate is produced compared to the rate when fats and carbohydrates are completely oxidized to CO_2 and H_2O (Jacobs, 1986; Walsh & Banister, 1988). With an increased glycolytic rate, pyruvate and cytosolic NADH formation are enhanced. Lactate production makes possible the regeneration of cytoplasmic NAD^+ (see Figure 2) which helps to maintain the $NADH/NAD^+$ ratio (Katz & Sahlin, 1987). This allows the continued conversion of glyceraldehyde 3-phosphate (G-3-P) to 1,3-diphosphoglycerate (1,3-DPG) in glycolysis and continued anaerobic ATP production, as well as aerobic ATP production when pyruvate is oxidized. The Electron Transport Chain regenerates mitochondrial NAD^+ through the oxidation of NADH, and in conjunction with the NADH shuttles, this enables the regeneration of cytoplasmic NAD^+. However, during strenuous exercise, the rate of NAD^+ requirement for glycolysis may surpass the amount regenerated by the mitochondria and/or transported by the shuttles back into the cytoplasm. Thus, an increase in the cytoplasmic $NADH/NAD^+$ will be reflected by an increased conversion of pyruvate to lactate (Katz & Sahlin, 1987; 1988), thereby performing a regulatory function.

Lactate production also helps to maintain blood glucose levels via gluconeogenesis. This is the conversion of lactate to glucose which occurs mainly within the liver (Brooks, 1986).

Lactate Accumulation and Fatigue

ACIDOSIS

Lactate accumulation has been linked to the development of fatigue; however, the mechanisms by which this may occur are not entirely clear. Increased blood lactate levels are associated with a decrease in intracellular pH (pH_i) and extracellular pH

(pH_i) which are thought to be a contributing factors to fatigue. A decreased pH (acidosis) is caused by an increased concentration of H^+ ions within the working muscle cells. Lactic acid is primarily in the dissociated form at the normal cellular pH of 7.0. Therefore, it contributes more than 85% of the liberated H^+ ions, with other sources being G-1-P, glycerol-1-P, pyruvate, citrate, and malate (Sahlin, 1983). Most of the accumulated H^+ ions are released to the blood and are buffered by the bicarbonate and protein buffer systems to minimize the effect of the proton load.

Although acidosis is associated with fatigue, it is not necessarily the cause, as many biochemical and biophysical changes occur at the same time fatigue is developing (Mainwood & Renaud, 1984). Yet acidosis does play a direct role in contractile suppression and influences metabolic reactions, since enzymes have an optimal pH.

THE CONTRACTILE PROCESS AND THE EFFECTS OF ACIDOSIS

Researchers have suggested several possible mechanisms of action whereby a decrease pH_i could interfere with the muscular contraction process. Therefore, a brief review of the contractile process is required.

Calcium (Ca^{2+}) is released from the sarcoplasmic reticulum of a muscle cell when an action potential depolarizes the muscle cell membrane, the sarcolemma. The T-tubules transmit the signal to the sarcoplasmic reticulum. In the absence of Ca^{2+}, tropomyosin blocks the cross-bridge binding sites on actin. However, upon release, Ca^{2+} binds to troponin C causing tropomyosin to move, thereby exposing the cross-bridge binding sites. Energized myosin molecules bind to the exposed actin sites resulting in a discharge of the stored energy and production of an angular movement of each cross bridge, synonymous with contraction (Vander, Sherman, & Luciano, 1985).

The contractile process could be influenced by acidosis from the very point of initial depolarization. An increased $[K^+]$ in the extracellular space, which accompanies acidosis, would decrease a muscle cell's resting membrane potential and make it more difficult to recruit the muscle fibre (Sahlin, 1983).

Calcium storage and release could also be affected. The amount of Ca^{2+} available to activate contraction could be reduced by decreased Ca^{2+} binding by cytosolic protein (Mainwood & Renaud, 1985). Alternatively, the sarcoplasmic reticulum could reduce its Ca^{2+} release when PH decreases, since in the opposite situation, small increases in pH_o increase Ca^{2+} release (Mainwood & Renaud, 1985). Accessibility of Ca^{2+} binding sites on troponin C may be altered (Mainwood & Renaud, 1985), or there could be an increased requirement for Ca^{2+} to trigger contraction (Sahlin, 1983).

Finally, decreases in pH_i could influence cross bridge formation to reduce force development (Mainwood & Renaud, 1985) by decreasing myosin-ATP-ase activity, which catalyzes the reaction to energize the myosin molecule (Sahlin, 1983).

Lactic acidosis in muscle could also be a performance limiting factor through indirect contractile suppression. Lactate and H^+ efflux from the muscle cell are related to pH_o. Mainwood and Worsley-Brown (1975) demonstrated that increasing pH_o during recovery allows greater lactate and H^+ efflux from the muscle and subsequently redevelopment of muscular tension capabilities. If there is poor muscle perfusion during exercise then the buffering capacity of H^+ is limited and pH_o would decline rapidly, thus limiting both lactate and H^+ efflux. In this case, pH_i would increase causing possible damage within the muscle. Hence, acidosis could serve as a protective feedback mechanism to prevent overload resulting from mismatching of muscle perfusion with its work load (Mainwood & Renaud, 1985).

Similarly, fatigue accompanied by acidosis could be viewed as a safety mechanism to protect the muscle cell from ATP depletion (Sahlin, 1983). During acidosis, the protonated form of ATP is a potent inhibitor of PFK, the main regulatory enzyme in glycolysis. Therefore, glycolysis and glycogenolysis are inhibited (Davis, 1985b; Sahlin, 1983). Inhibition of lipolysis is also evident during exercise above LT (Ribeiro, Hughes, Fielding, Holden, Evans, & Knuttgen, 1985). With a reduction in the capacities of ATP generation from these energy systems and accelerated CP catabolism (Hultman & Sahlin, 1980), both ATP and CP stores fall. This could trigger a safety mechanism to prevent their concentrations from falling too low.

BLOOD LACTATE CONCENTRATIONS

BLa concentrations depend upon the balance between production, diffusion of lactate from the muscle to blood, and its subsequent uptake by numerous tissues (Farrell et al., 1979; Gollnick & Hermansen, 1973; Wasserman, 1987). This section will include a review of the factors influencing lactate production during exercise, followed by a discussion on the mechanisms of lactate removal during exercise.

Factors Affecting the Lactate Production During Exercise

CELLULAR REGULATION OF LACTATE PRODUCTION

As previously mentioned, it is generally understood that lactate is produced at rest. Another well accepted phenomenon is that cellular production of lactate rises with increasing metabolic demands, i.e. exercise intensity. However, the precise regulating factors of lactate production within muscle cells are still debated.

The rate at which glycolysis proceeds is primarily under the control of regulatory enzymes. Hexokinase and glycogen phosphorylase control the rate of glucose entry into the glycolytic pathway. Hexokinase catalyzes the phosphorylation of glucose to glucose-6-phosphate (G-6-P) and is allosterically inhibited by the product of this reaction in skeletal muscle tissue (Lehninger, 1982). Glycogen phosphorylase will catalyze the breaking off of a glucose residue from a molecule of glycogen to produce G-1-P. This enzyme must first be activated via adrenergic hormones or by a contractile factor (Gollnick & Hermansen, 1973), such as CA^{2+}.

Once glucose has entered the glycolytic sequence, the two major regulatory steps are catalyzed by phosphofructokinase (PFK) and pyruvate kinase. PFK has both active and inactive forms and is allosterically inhibited by ATP, CP and citrate, while on the other hand, it is activated by AMP and inorganic phosphate (Pi) (Gollnick & Hermansen, 1973). Pyruvate kinase, another allosteric enzyme, is inhibited when ATP levels in the cell are high and by long-chain fatty acids, which would provide ample fuel for mitochondrial respiration (Lehninger, 1982). Hence, with increasing exercise intensity, a decrease in the [ATP]/[ADP]x[Pi] ratio favors an acceleration in the catalytic activity of the enzymes regulating glycolysis.

With accelerated metabolism and, therefore, an increase in the glycolytic rate, pyruvate could begin to accumulate within the cell more rapidly than it could be utilized by the mitochondria. If this were the case then lactate production would increase due to mass action. By studying the lactate/pyruvate ratio during incremental exercise, Wasserman et al. (1985) have dismissed this mechanism since the lactate/pyruvate ratio did not change during low intensity exercise and at a threshold work rate only lactate increased abruptly. Also, since pyruvate concentrations and LDH activity increase to a minor extent during exercise (Gollnick & Hermansen,

1973), there must exist some other mechanism within the cell for regulation of lactate production.

Classically, lactate production has been attributed to an oxygen deficiency in contracting muscle. It is believed that this limits ATP production via oxidative phosphorylation; hence, resulting in an increased glycolytic rate and production of lactate to supplement ATP production. Support for this theory comes from studies which induced respiratory hypoxia and found increased BLa concentrations, which was thought to imply increased lactate production (Hughes, Clode, Edwards, Goodwin, & Jones, 1968; Linnarsson, Karlsson, Fagraeus, & Saltin, 1974). Respiratory hypoxia is thought to reduce PO_2 at the mitchondrial level and limit mitochondrial ATP production. ATP must then be produced anaerobically, resulting in increased lactate production. However, in normal physiological testing conditions, the oxygen content of inspired air is normoxic (21% O_2) and constant; therefore, these studies simply demonstrate that atmospheric oxygen content may be an influencing factor in lactate production.

Supporters of the O_2 deficiency theory also point to research demonstrating a decreased lactate level with an increased O_2 content of inspired air. They suggest that the results are due to a removal of local hypoxia within the working muscle. The relationship may not be this simple however, since hyperoxia is toxic to glycolytic enzymes, especially glyceraldehyde 3-phosphate dehydrogenase. Hence, inhibition of glycolysis may be responsible for decreased lactate production (Walsh & Banister, 1988).

This much debated theory, therefore, suggests that the regulatory factor in lactate production is the PO_2 at the level of the mitochondria in the contracting fibres. At this time, measuring mitochondrial PO_2 is not within technological limits; however,

research carried out studying myoglobin saturation of oxygen has estimated that the critical PO_2 required for maximal aerobic production of ATP is between 0.1 and 0.5 Torr (Walsh & Banister, 1988). Connett, Gayeski, and Honig (1984) have estimated a higher mitochondrial PO_2, between 1.0 and 2.0 Torr, with supramaximal stimulation of the dog gracilis muscle in situ. This would represent approximately 50% VO_2max with in vivo muscle stimulation, a point at which BLa concentration is increased above resting levels and the PO_2 has not yet reached a critical level. Therefore, this research suggests that increased lactate production does not seem to be caused by local muscle hypoxia.

Regulation of lactate production is believed to be related to changes in the NADH + H^+/NAD^+ ratio, with lactate production increasing as a consequence of elevated cytoplasmic NADH + H^+ concentrations (Gollnick, 1973; Katz & Sahlin, 1988; Wasserman et al., 1985). With increasing exercise intensity, glycolysis is accelerated due to allosteric regulation, as previously discussed, and the recruitment of typeII muscle fibres (FT), which have higher levels of glycolytic enzymes and lower mitochondrial volume density, is increased (Schantz, 1986). This generates greater amounts of cytosolic NADH + H^+ to be transported to the mitochondria for rapid reoxidation via the malate-aspartate shuttle (MA shuttle) (see Figure 5) or the o-glycerophosphate shuttle (GP shuttle), since the NAD^+ content in the muscle is limited (Katz & Sahlin, 1988; Schantz, Sjoberg, & Svendenhag, 1986). The enzymes regulating the control of the GP shuttle are higher in typeII fibres while typeI fibres have higher enzyme levels for those regulating the MA shuttle. However, it is suggested that the MA shuttle is the more important of the two shuttle systems in human skeletal muscle, since its lowest enzyme activity is about 30 times higher than the lowest activity in the GP shuttle (Katz & Sahlin, 1988; Lehninger, 1982; Schantz,1986).

With an increase in glycolytic rate cytosolic NADH production increases. If there is an imbalance in the rate of cytosolic NADH production and transport of the reducing equivalents across the mitochondrial membrane by the shuttle systems then cytosolic NADH/NAD^+ will increase (Schantz, 1986; Wasserman et al., 1985). Since the rate of lactate formation from pyruvate is limited by the production of NADH, then it follows that an increase in lactate/pyruvate ratio reflects an increase NADH/NAD^+ (Connett et al., 1984; Katz & Sahlin, 1987). Connett et al. (1984) and Schantz (1986) proposed that the capacities of the MA shuttle are not exceeded even at 100% VO_2max, but an increased NADH/NAD^+ ratio or reduced redox state is necessary to create a driving force for the NADH shuttles, resulting in increased lactate production.

The activity of the shuttle systems is influenced by the difference in NADH concentration between the mitochondria and the cytosol; therefore, cytosolic NADH may also increase if there is an increase in mitochondrial NADH (Katz & Sahlin, 1987). Katz and Sahlin (1987) suggested that mitochondrial NADH, reflected by muscle NADH, increases due to muscle hypoxia not because the enzymatic capacity of the mitochondria to handle reducing equivalents is exceeded. Hence, the literature indicates that the redox state of the muscle cell has a regulatory effect on lactate production, but more research is needed to determine if mitochondrial NADH increases during normoxic submaximal exercise are due to decreased PO_2. Future research should also address the issue of possible differentiation in the changes of NADH during exercise in ST and FT muscle fibres.

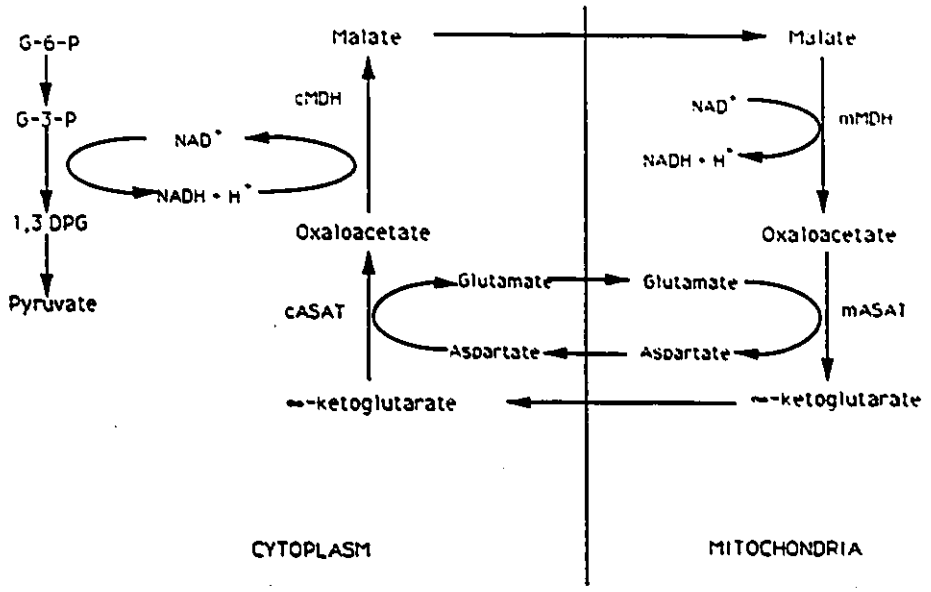

Figure 5. The malate-aspartate shuttle. Adapted from Lehninger (1982)

MUSCLE FIBRE COMPOSITION

Human muscle fibres are composed of different fibre types, and their distribution varies between individuals. The two most distinct fibre types identified are the type II or fast twitch (FT) fibres, which are glycolytic in nature and therefore better suited for anaerobic work, and the type I or slow twitch (ST) fibres, which are higher in oxidative capacity and better suited for aerobic work (Essen, Jansson, Henriksson, Taylor, & Saltin, 1975; McGrail, Bonen & Belcastro, 1977). These fibres have very different contractile and metabolic properties which have led to their characterization. However, based on myofibrillar ATPase staining, after preincubation at different pH values, it was discovered in the 1970's that type II fibres may be subdivided in IIa, IIb and IIc (Schantz, 1986). Also, Essen et al. (1975) identified type Ib which has a slightly different staining pattern than that of type I. Of the type II fibres, the type IIb

are the most common. The type IIa fibres or fast oxidative glycolytic (FOG) fibres have greater oxidative capacities than the type IIb and are therefore intermediate to FT and ST (Edgerton, Smith, & Simpson, 1975). These subpopulations exhibit ranges of contractile, histochemical and biochemical properties. Yet in most literature, fibre type discussion or analysis is limited to ST (type I), FT (type IIb) and FOG (type IIa) fibres.

The five isozyme forms of LDH, which were previously introduced, represent one distinguishing characteristic of FT and ST muscle fibres. M-LDH, which favors lactate formation from pyruvate, is relatively more abundant in FT fibres. H-LDH, on the other hand, is more predominant in ST fibres and favors the reformation of pyruvate from lactate (Gollnick & Hermansen, 1973). Hence, the FT:ST ratio in exercising muscle groups may influence the rate of lactate production and removal and subsequently affect LT. In fact, Aunola and Rusko (1986) and Ivy et al. (1980) found significant relationships between the percentage of ST fibres in exercising muscles and LT. This is understandable, since increased ST recruitment simultaneously reduces lactate production and increases its removal. Unrecruited ST fibres may also contribute to lactate removal.

As work intensity increases in an incremental exercise test, it has been demonstrated that initially ST fibres are recruited and then progressively more FT fibres are recruited, which are capable of greater contractile tensions and faster rates of ATP production (Gollnick, Piehl, & Sahlin, 1974). The LT phenomenon is likely related to this progressive recruitment of larger motor units. This is known as the recruitment theory to explain lactate accumulation. Increased lactate production accompanies increased FT recruitment due to the biochemical characteristics of FT fibres such as greater percentage of M-LDH, greater glycolytic capacity and smaller

oxidative capacity, smaller capillary/fibre ratio, and reduced mitochondrial density (Walsh & Banister, 1988). Furthermore, NH_4^+ may accumulate with FT recruitment. This occurs due to conversion of two moles of ADP to ATP and AMP. AMP is subsequently catabolized to IMP and NH_3 with the catalytic action of adenylate deaminase (Lehninger, 1982). NH_4^+ accumulation stimulates the production of pyruvate but inhibits its oxidation, thereby further contributing to increased lactate production (Walsh & Banister, 1988).

CATECHOLAMINES

Catecholamine concentration in the blood can influence the rate of lactate production within the muscle fibre through stimulation of glycogenolysis and therefore augment lactate production (Brooks, 1986). Catecholamines act on the cell membrane to activate the enzyme adenyl cyclase, and through a cascade effect, glycogen phosphorylase b is eventually converted to glycogen phosphorylase a. This allows increased glycogen utilization via glycolysis (Lehninger, 1982).

It is believed that increased catecholamine release, due to increased sympathetic activity during exercise, may be a possible mechanism for lactate accumulation and hence LT (Walsh & Banister, 1988). Studies have shown significant relationships between LT and the catecholamine threshold during incremental exercise (Galbo, Holst, & Christensen, 1975; Van Harn & Brooks, 1985). In fact, a LT is not possible without catecholaminergic stimulation. This was demonstrated by Chirtel, Barbee, and Stainsby (1984) who found that lactate and VO_2 merely demonstrated a linear relationship during progressive, in situ, electrical stimulation of the dog gastrocnemius-plantaris muscle. The lack of an identifiable threshold may be attributed to the absence of increased sympathoadrenal activity with electrical stimulation, which

normally occurs during exercise. Therefore, while the LT phenomenon may be closely related to catecholamine concentration, it cannot account for all lactate production that does occur with incremental muscular activation.

Interestingly, catecholamines may also influence lactate production in non-contracting tissue, since catecholamine infusion at rest into whole organisms or isolated muscle preparations has been shown to augment lactate production (Walsh & Banister, 1988). Stainsby, Sumners, and Eitzman (1985) concluded that a major portion of elevated blood lactate concentration comes from non-active muscle, since only 4-8% of the increase in blood lactate could be attributed to the contracting muscles following norepinephrine infusion.

SUBSTRATE AVAILABILITY

The type of substrate that is available to the muscle cell pre-exercise will have a significant influence on the rate of lactate production. Elevation of blood, free fatty acid levels delays the onset of lactate accumulation due to an inhibition of carbohydrate metabolism (Skinner & McLellan, 1980). Exercise with muscles low in glycogen will result in lower lactate values, and there will be a greater reliance on the oxidation of fat (Gollnick et al., 1986; Hughes, Turner, & Brooks, 1982). Conversely, muscles high in stored glycogen will stimulate glycolysis to a greater extent.

ENDURANCE TRAINING

It has long been recognized that endurance training leads to adaptations which result in lower lactate levels in trained compared to untrained subjects at identical workloads (Crescitelli & Taylor, 1944; Holmgren & Strom, 1959; Robinson & Harmon, 1941). Some believe that the inability to convert FT to ST muscle fibres, to some

degree, limits the trainability of LT (Ivy et al., 1980). However, muscle fibres may increase their oxidative capacities (Henriksson, 1977). This permits increased fat and pyruvate oxidation and results in reduced lactate production at a given exercise intensity. The convertibility of FT to ST fibres will be addressed in the section on training adaptations.

Recent research has uncovered some of the adaptive responses to endurance training which are responsible for the changes in LT. To date, they are numerous; therefore, they will be discussed in detail in a separate section.

Lactate Removal During Exercise

Lactate accumulation is also related to the rate at which lactate can be removed from the circulation. If the rate of removal falls below the rate of production with increased work load, and cannot catch up, then lactate will continue to accumulate in the blood during continuous or incremental activity. Lactate can diffuse from the muscle cell into the interstitial space and then into the capillaries. Once in the blood stream, it may be redistributed and removed by oxidation in either cardiac tissue or skeletal muscle fibres containing the H-LDH isozyme. This is possible since the conversion of pyruvate to lactate by M-LDH is reversible by H-LDH. Pyruvate may then be metabolized in the normal sequence in Krebs Cycle. The alternative to oxidation is gluconeogenesis in the liver and kidneys, which involves the reversal of certain glycolytic reactions. The liver and kidneys have the key enzymes to perform the reversible reactions, unlike skeletal muscle tissue which lack the required enzymes. These enzymes are phosphoenol pyruvate carboxylase, pyruvate carboxylase, fructose 1,6-diphosphatase and glucose 6-phosphatase (Lehninger, 1982). However, during exercise, splanchnic blood flow is significantly reduced limiting the potential for gluconeogenesis.

Using liver blood flow values reported by Rowell, Kraning, Evens, Kennedy, Blackman and Kasumi (1966), Gollnick and Hermansen (1973) calculated that about 4% of lactate is removed by the liver during steady state exercise. This is small compared to the amount oxidized by skeletal muscle during exercise, to which blood flow is increased. The contribution of cardiac tissue to lactate removal is less than 10% of the total removed (Newman, Dill, Edwards, & Webster, 1973). The fraction of lactate turnover that is oxidized increases greatly, from 42 to 72%, during the transition from rest to exercise in rats (Donovan & Brooks, 1983). During rest in humans approximately 40-50% of lactate is oxidized and at 50% VO_2max this increases to 90% of the lactate removed. Hence, skeletal muscle is regarded as the main site of lactate removal during exercise (Gollnick & Hermansen, 1973).

It seems contradictory that skeletal muscles are at the same time both responsible for the production of lactate and primarily responsible for its removal during exercise. This is possible since it occurs between different fibre types. Owles was the first to suggest such a possibility in 1930 (Gollnick et al., 1986). Lactate released from FT fibres, which have low oxidative capacities and the M-LDH isozyme, may be oxidized in ST and FOG fibres, which have higher oxidative capacities and the H-LDH isozyme (Brooks, 1986; McGrail et al., 1977).

Lactate oxidation is not, however, limited to the ST and FOG fibres in exercising muscles. Nonexercising muscles have been shown to take up lactate produced by the exercising muscles. Ahlborg, Hagenfeldt, and Wahren (1975) found that the femoral, arterial-venous lactate difference in a resting leg changed from a negative to a positive difference when either the other leg or arms were exercised. This indicated uptake of lactate by the resting leg. In fact, 70% of the resting leg's VO_2 could be accounted for by lactate and glucose catabolism during one-leg exercise, and this increased to 90%

during arm exercise. In another study, incremental bicycle exercise tests were performed by eight males while both arterial and venous lactate levels were measured in the forearm (Yoshida, Takeuchi, & Suda, 1982). Before exercise, there was no significant difference between arterial and venous lactate levels; however, venous lactate values were lower during exercise. They found that the first rises in lactate above resting levels (AT) occurred at 37% VO_2max in arterial blood but at 55% VO_2max in venous blood. Therefore, the inactive forearm was responsible for blood lactate removal. It is partly due to the removal of lactate from the blood by tissue other than active muscle that there is a significant difference between muscle and BLa concentrations. Hence, BLa level is not merely a reflection of lactate production in the active muscle (Walsh & Banister, 1988).

Donovan and Brooks (1983) proposed that the rapid accumulation of lactate in the blood could be explained by a decreased rate of removal during incremental exercise. Hermansen and Stensvold (1972) found that approximately 60-70% VO_2max is a critical level for not only lactate production but also for removal. Exercise above and below this intensity demonstrated a decreased rate of lactate removal during recovery from previous high intensity exercise. Therefore, exercise intensity during incremental exercise could affect the rate of removal, thus contributing the LT phenomenon. This could occur due to a decreased blood flow to inactive muscles and the liver which actively remove lactate (Donovan & Brooks, 1983). It could be partially attributed to a saturation of lactate uptake mechanisms, if they exist (Walsh & Banister, 1988).

Factors other than exercise intensity have been proposed to influence blood lactate removal. Aunola and Rusko (1986) proposed that lactate clearance is dependent upon the muscles' oxidative capacity, which is influenced by the fibre type distribution. Glycogen concentration of working muscles is a factor in lactate removal, just as it was

in lactate production. Muscles low in glycogen will take up increasing amounts of lactate for oxidation (Gollnick et al., 1986; McGrail et al., 1977).

THE ANAEROBIC AND LACTATE THRESHOLDS

The Anaerobic Threshold

Research related to BLa concentration and accumulation has been popular due to its proposed relationship with increased ventilation, pH changes and subsequently fatigue (Wasserman, Van Kessel, & Burton, 1967). Many researchers have tried noninvasively, via gas exchange measurements, to identify the point of metabolic or lactic acidosis, reflective of a rise in lactate above resting levels (Davis, Frank, Whipp, & Wasserman, 1979; Wasserman et al., 1967; Wasserman et al., 1973).

Increases in lactic acid concentration have an influence on arterial PCO_2 and H^+. And since ventilatory control mechanisms try to maintain homeostasis of arterial PCO_2 and H^+ concentrations, it is believed that lactate threshold may be determined from respiratory measures (Davis, 1985a). Lactic acid has a pK of approximately 3.9 and is consequently almost totally dissociated in the physiologic range of pH; therefore, the excess H^+ ions accompanying increased lactic acid production must be buffered. H^+ immediately reacts with bicarbonate [HCO^-3] within the cell, leading to an increased CO_2 production via the reaction catalyzed by carbonic anhydrase (Davis, 1985):

$H^+ + HCO_3^- \rightarrow H_2CO_3 \rightarrow CO_2 + H_2O$.

The additional CO_2 produced from the buffering of lactic acid places an additional load on the respiratory system. Ventilation is increased in relation to VO_2 to prevent PCO_2 from rising. This is known as isocapnic buffering where Ve/VO_2 is rising while Ve/VCO_2 remains constant. However, with further increases in lactate production,

ventilation is stimulated to an ever greater extent such that Ve/VCO_2 increases reflecting respiratory compensation from the metabolic acidosis of exercise (Wasserman, Hansen, Sue, & Whipp, 1987). Thus, there are really two ventilatory thresholds and investigators define one or the other to represent the anaerobic threshold (AT). The break in ventilation where Ve/VCO_2 remains constant is the preferential definition of AT, but the second ventilatory threshold is satisfactory for incremental exercise with stages of four minutes in length (Wasserman, 1987).

The controversy exists, however, as to whether AT determined by one of these methods is in fact associated with changes in BLa concentration, known as LT. After reviewing the literature, Walsh and Banister (1988) concluded that any association between LT and AT is coincidental since the two may be dissociated by factors such as training and various testing protocols. Also, ventilation may be controlled by factors other than a lactate mediated decrease in pH. Therefore, the focus of this research is in regard to changes in lactate metabolism measured directly through blood sampling and not by noninvasive techniques.

Investigators often use the term AT to identify the first rise in BLa concentration above resting levels either invasively or non-invasively, as previously discussed. Exercise above this intensity may, however, be endured in continuous exercise due to an eventual stabilization in BLa concentration, since the rate of catabolism can catch up to the rate of production (Kinderman et al., 1979; Scheen, Juches, & Cession-Fossion, 1981; Simon, Young, Gutin, Blood, & Case, 1983; Wasserman, 1987). Hence, it is not this exercise intensity with which endurance athletes are concerned; rather, one seeks to identify the exercise intensity (VO_2) beyond which lactate levels do not exhibit an eventual plateau during continuous exercise. Endurance exercise above this intensity will lead to fatigue and the inability to continue for extended

periods. This physiological landmark will be defined in this paper as LT, which corresponds to the VO_2 during incremental exercise beyond which the BLa concentrations increase abruptly or exponentially (Brooks, 1985; Kinderman et al., 1979; Stegmann & Kinderman, 1982).

Lactate Threshold.

DEFINITION

LT is a critical exercise intensity since individuals may exercise up to this intensity with little or no lactate accumulation; however, once it is surpassed lactate begins to accumulate in the blood exponentially (Campbell, Hughson, & Green, 1989; Gollnick et al., 1986; Hughson, Weisiger, & Swanson, 1987). This point may correspond to BLa levels above or below four mmol/l - the fixed AT (Stegmann & Kindermann, 1982; Stegmann, Kindermann, & Schnabel, 1981). Below LT in steady state exercise, lactate concentrations may remain unchanged or increase temporarily and then decline or plateau. This is due to the balancing of the rate of appearance with the rate of disappearance of lactate in the blood (Brooks, 1985). LT represents the highest exercise intensity that can be maintained without a progressive increase in lactate during 20 to 40 minutes of exercise (Ribeiro et al., 1986). Above this intensity, there is an accumulation of lactate since the rate of appearance is greater than the rate of its removal from the blood (Brooks, 1985).

This critical intensity has been expressed according to workload, power output, velocity and VO_2. However, it is best expressed as the VO_2 in incremental exercise, corresponding to the lactate breakpoint, since thresholds have been shown to occur at different power outputs depending on the protocol (Ribeiro et al., 1986). LT may be expressed as an absolute (l/min or ml/kg/min) or relative value (% VO_2max).

Researchers have given many names to exercise intensities corresponding to a given level of lactate, its increase above resting values, or the point of exponential increase. These include aerobic threshold, anerobic threshold, onset of blood lactate accumulation (OBLA), onset of plasma lactate accumulation (OPLA), lactate threshold, lactate turning point, maximal steady state, the individual anaerobic threshold, excess lactate, and aerobic capacity (Jacobs, 1986). The exercise intensity reflected by these terms may be identical or vary due to the method of identification. Therefore, the field of lactate kinetics is often confusing and controversial making comparison of studies difficult.

Nevertheless, the development of micro blood sampling in the 1970's has made threshold determination quite popular, and routine and the literature continues to grow. The identification of LT is now considered quite important due to the association of lactate accumulation with metabolic changes and fatigue. BLa parameters have demonstrated their usefulness in the assessment of physical fitness by contributing to coaching and training effectiveness.

LACTATE THRESHOLD AND ENDURANCE PERFORMANCE

VO_2max has been recognized as an important determinant of endurance performance (Astrand & Rodahl, 1986). However, many studies indicate that it is more accurate to express an individual's metabolic capability for endurance exercise by reporting VO_2 at LT, or a similar physiologic landmark, than it is to report VO_2max. After a few years of intense endurance training, athletes are likely to achieve high aerobic capacities, but their performance continues to improve over the following years. Therefore, within such a small range of VO_2max, this variable is not a suitable parameter with which to predict performance. Studies have suggested that lactate

related variables may be more sensitive indicators of training adaptations than VO_2max (Jacobs, 1986). Coyle et al. (1988) found that competitive cyclists who had trained for 3-12 years had similarly high values for VO_2max values but differed considerably in the duration for which they could cycle at 88% VO_2max. The results of this investigation revealed that for these athletes the single best predictor of performance time to fatigue was the %VO_2max at LT when cycling.

The exercise intensity associated with the rapid exponential rise in BLa has been applied in research to predict endurance running performance (Farrell et al., 1979; Jacobs et al., 1985; Sjodin & Jacobs, 1981). The critical intensity investigated by Farrell et al. (1979) was defined as the onset of plasma lactate accumulation (OPLA). Subjects performed runs of various distances 3.2, 9.7, 15, and 19.3 kilometers. It was found that the correlation coefficients relating both VO_2 at OPLA and treadmill velocity at OPLA to running performance were greater than or equal to 0.91. This indicates that endurance runners tend to self regulate their running intensity close to LT.

In studies by Jacobs et al. (1985) and by Sjodin and Jacobs (1981), the physiological landmark investigated was defined as OBLA - a blood lactate concentration of 4 mmol/l. Jacobs et al. (1985) designed their study to compare the predictive power of a lactate related index (WOBLA) during submaximal exercise to that of an exhaustive cycle ergometer test for evaluating the endurance capacity of soldiers. The subjects, 48 male soldiers, performed a continuous exercise test to voluntary exhaustion on a cycle ergometer. The subjects cycled at 60 rpm with a 50 W power output increase every fourth minute. The power output associated with OBLA (WOBLA) was determined through plots of lactate concentrations vs cycle power outputs. From the results of the exhaustive test, the maximum power output that could

be maintained for 6 minutes (Wmax) was calculated using a mathematical formula. The subjects also completed a 3000 m cross country run within two weeks of the cycle test, upon which they were timed. The same tests were completed five months later, but the 3000 m run was referred to as a "loaded run" as the soldiers were in full uniform carrying weapons and equipment. The time for the 3000 m run for each test was significantly correlated with both Wmax and WOBLA. When Wmax and WOBLA were expressed relative to body weight the relationships were stronger. The two variables had similar predictive power. Sixty-nine and 62% of the variation in running performance on the first and second tests, respectively, could be accounted for by the variation in the combination of WOBLA and body weight. Based on this data, the researchers suggested that a submaximal test based on BLa could be substituted for an exhaustive exercise test without a significant loss of predictive ability of running performance in the field.

Sjodin and Jacobs (1981) recognized the importance of evaluating endurance exercise performance and designed an experiment to study marathon performance as it related to OBLA, training volume, and muscle fibre characteristics. Eighteen, male volunteers, were trained by submaximal, long distance running in this study. Two to three weeks prior to their participation in the marathon, the subjects completed a continuous stepwise, progressive protocol in order to determine the treadmill velocity which corresponded to a BLa accumulation of 4 mmol/l (VOBLA). Immediately after the marathon, muscle biopsies were taken from the vastus lateralis in order to determine muscle fibre type distribution as well as capillary density. Marathon velocity (VM), the means to evaluate exercise performance, was the dependent variable. It was found that VOBLA was the independent variable with which VM correlated the best, and 92% of the VM variation was accounted for by this variable. Four percent more of the variance in VM was accounted for by adding training kilometrage to VM.

All performance variables were positively correlated to percentage of ST muscle fibre distribution and capillary density. It was concluded that endurance exercise performance, in this case marathon performance, is closely related to VOBLA and to the ability to run at a pace close to that velocity during the race. Percentage of ST muscle fibres, capillary density, and training volume prior to the event were also related to VM. Sjodin and Jacobs (1981), unlike Jacobs et al. (1985), used the same activity mode for LT determination and the performance to be measured. Perhaps, this may explain why they achieved better correlations between the treadmill velocity at which BLa accumulation of 4 mmol/l occurred (VOBLA) and marathon running performance, measured as velocity (VM). VOBLA accounted for 92% of the variation in VM.

In summary, these studies support the current preference for measuring LT to predict endurance performance. The latter two studies suggest that OBLA or LT is best used for application research when it is determined in the specific activity mode to which it is to be applied. This implies that LT or the point of rapid lactate accumulation is sport specific.

ENDURANCE TRAINING AND LACTATE THRESHOLD

Training Adaptations and Lower Blood Lactate Concentrations

There are central and peripheral adaptations which occur as a result of endurance training. Central adaptations generally represent improvements in the cardiovascular system, allowing enhanced oxygen delivery. These changes are reflective of a more efficient heart and include reduced heart rates at rest and during submaximal work, decreased systolic blood pressure during rest and work, prolonged diastole, and

reductions in contractility of the heart. Other central adaptations include a reduced peripheral resistance and decreased release of catecholamines at given work loads (Hollmann, Rost, Liessen, Dufaux, Heck, & Mader, 1981). Peripheral adaptations to endurance training that have been cited indicate increased size and number of mitochondria as well as increased mitochondrial enzyme activity. Shifts in substrate utilization result in increased free fatty acid metabolism during submaximal exercise, and increases in muscle glycogen stores allow for improvements in endurance. Increased oxygen extraction from the blood is also possible due to rises in myoglobin content, increased capillarization, and the development of collaterals (Hollmann et al., 1981).

Lower BLa levels are demonstrated at the same absolute and relative work loads following training (Denis, Dormois, Castells, Bonnefay, Padilla, Geyssant et al., 1988; Karlsson, Nordesjo, Jorfeldt, & Saltin, 1972). This could be explained by one or more of the above mentioned adaptations. Holloszy and Coyle (1984) suggested that biochemical adaptations within the trained musculature account for the lower lactate levels, not central cardiovascular adaptations that allow improved oxygen delivery. They argued that if O_2 delivery was responsible for lactate formation then a higher oxygen consumption, at a given absolute workload, should accompany lower lactate levels after training, and this is not seen. If peripheral adaptations within the trained musculature are indeed responsible for the changes observed in lactate metabolism following training then the specificity of LT to training should be predictable and understandable. Therefore, it is pertinent to review some of the physiological adaptations to which could influence lactate metabolism and LT.

Endurance training constitutes a powerful stimulus for capillary proliferation in human skeletal muscle. Andersen and Henriksson (1977a) as well as Klausen, Andersen, and Pelle (1981) found a 20% increase in capillary density in the vastus lateralis following eight weeks of bicycle ergometer training. Hoppeler et al. (1985) reported a similar 29% enhancement of capillary density along with a 26% increase in capillary to fibre ratio following a six week training regime on a bicycle ergometer. In a cross-sectional study of six untrained and six endurance-trained runners, capillary density and capillaries per fibre of the quadriceps femoris were 36% and 53% higher, respectively, in the trained muscles. Following low intensity, long duration, cross country ski training the number of capillaries per fibre and the capillary density increased 40-50% in the triceps brachi (Schantz, 1986).

The ability of individuals with cardiovascular disease, such as decreased blood flow, to increase their exercise tolerance after training is attributed in part to increased capillarization. This will increase capillary transit time of blood and allow greater oxygen extraction for cellular respiration (Terjung, Mathien, Erney, & Ogilvie, 1988). This implies a reduction in glycolysis and lactate production for people receiving inadequate oxygen supply. However, with the normal person, where this topic is highly debatable, an alternative explanation for the effect of increased capillarity on LT is that it may allow for increased lactate removal from within exercising muscle fibres (Tesch & Wright, 1983).

In support of the specificity of training LT, Terjung et al. (1988) suggested that enhanced capillary density is not dependent on muscle fibre type, but rather, it seems to be associated with the fibres recruited during exercise. Even FT fibres may then show improved capillarity. Rosler et al. (1985) demonstrated that capillary proliferation is specific to trained limbs only.

BLOOD FLOW

Increased blood flow or better distribution through active and nonactive muscles tissue would enhance lactate removal. Capillary proliferation has already been discussed and would represent one mechanism by which blood flow in active muscle could be improved following training. Decreased peripheral resistance, a central adaptation, (Hollmann et al., 1981; Klausen, Secher, Clausen, Hartling, & Trap-Jensen, 1982) could augment blood flow through active and nonactive tissue to increase lactate removal. Better blood redistribution to active motor units within the muscle is a possible peripheral adaptation (Terjung et al., 1988).

Maintenance of blood flow to the liver during exercise could influence lactate removal. Clausen et al. (1973) found the reduction to hepatic-splanchnic blood flow during exercise to be less marked after endurance training in man. Donovan and Brooks (1983) suggested that the increased metabolic clearance rate in trained compared to untrained rats during exercise could be attributed to the maintenance of splanchnic blood flow in the trained animals. This explanation also accounts for the higher blood glucose levels and greater conversion of lactate into glucose during hard exercise in the trained compared to untrained rats.

CATECHOLAMINES

Endurance training seems to reduce the sympathoadrenal response to submaximal exercise. There are smaller increases in norepinephrine and epinephrine at the same absolute and relative workloads after endurance training compared to before training (Gollnick et al., 1986). Lehman and Keul (1986) found that trained cyclists demonstrated lower catecholamine responses and BLa levels compared to untrained controls in exhaustive, incremental exercise.

As previously discussed, blood catecholamine concentrations are linked to BLa accumulation via the activation of glycolysis in both active and nonactive muscle tissue. A reduction in sympathetic activation and subsequently lower catecholamine levels in response to training is categorized as a central adaptation (Hollmann et al., 1981), which may result in reduced BLa concentrations.

MUSCLE FIBRE COMPOSITION

Muscle fibre types have classically been viewed as being inconvertible from FT to ST (Baldwin, Klinkerfuss, Terjung, Mole, & Holloszy, 1972). It was believed that FT fibres simply increase their oxidative capacity through endurance training and subsequently produce less lactic acid (Gollnick, Armstrong, Saubert, Piehl, & Saltin, 1972; Henriksson, 1977; Holloszy & Coyle, 1984). This results in a transformation of typeIIb (FT) fibres into type IIa (FOG) fibres (Andersen & Henriksson, 1977b; Ingjer, 1979). However, new technological developments have allowed a better understanding of muscle fiber diversity and fibre type transitions. As previously discussed, the muscle fibres currently identifiable may be classified as type I, type Ib, type IIa, type IIb and type IIc. Type IIc and type Ib may be collectively referred to as myofibrillar ATPase intermediate (IM) fibres (Schantz, 1986). After cross country ski training for six and eight week periods, it was found that the proportion of IM fibres increased (Schantz, 1986). Since the IM fibres had almost as high an oxidative capacity and a slightly higher glycolytic capacity compared to type I fibres, this is viewed as a sign of ongoing type II to type I transformation. In addition, the IM fibre areas and mitochondrial volume density were intermediate between those of type I and type IIa, and capillary supply was comparable to type I. Hence, IM fibres have properties intermediate between type I and II with both slow and fast isoforms of contractile and regulatory proteins.

Animal studies also support the assumption that extensive physical training and other forms of increased load (chronic nerve stimulation) may produce alterations in fibre type composition (Pette, 1984). These transformations may be due to increased activity of the motor units rather than nerve discharge frequencies, which was previously believed and originated from the cross innervation studies of Buller et al. in 1960 (Pette, 1984; Schantz, 1986). However, in order to induce these changes, endurance exercise should demand intensities which exceed the tension generating capabilities of type I fibres. This will ensure the recruitment of a greater proportion of motor units innervating FT fibres and provide the stimulus of increased activity to these fibres (Schantz, 1986).

The conversion of recruited muscle fibre types or simply their increased oxidative capacities following training results in lower lactate concentrations due mainly to the enzymatic profile changes, which are characteristic to fibre types. Adaptations in glycolytic and mitochondrial enzymes in trained muscle tissue will be discussed in the following sections.

GLYCOLYTIC ENZYMES

Glycolytic enzyme levels and activity ratios of PFK, GADPH and LDH do not respond to endurance training, or they exhibit a decreased level of activity (Klausen et al., 1981; Schantz, 1986; Simoneau, 1983). However, an adjustment in the relative contributions of glycolysis and oxidative phosphorylation to metabolism after training may be reflected by enzyme ratios. Sjodin et al. (1982) found that the ratio of PFK to CS decreased significantly after training, due to a combination of nonsignificant increases in CS and significant decreases in PFK. Furthermore, the ratios of LDH/CS and PFK/CS have been shown to be directly related to the exercise intensity at OBLA (Jacobs & Sjodin, 1983; 1985; Sjodin & Jacobs, 1981)

Lactate production and/or lactate removal may be influenced by the shifts in LDH isozymes associated with endurance training, even if total LDH activity decreases (Holloszy & Coyle, 1984; Sahlin & Henriksson, 1984; Schantz, 1986). Sjodin et al. (1982) reported LDH levels were unchanged in trained musculature after training. However, there was an increase in the relative activity of H-LDH due to either an increase in absolute H-LDH activity or a decrease in M-LDH. Similarly, Sjodin et al. (1976) found that long distance running at 70-80% maximum heart rate increases the H-LDH/M-LDH ratio. This could simultaneously increase the rate of lactate oxidation and decrease the rate of production.

MITOCHONDRIAL ENZYMES

Endurance training has the ability to increase the activity levels of certain mitochondrial enzymes. The increased enzymatic activity level along with increases in mitochondrial size and number contribute to the enhanced generation of ATP via oxidative phosphorylation and oxidation of pyruvate following training (Holloszy & Coyle, 1984; Schantz, 1986). Mitchondrial content may increase even in type II fibres by four fold or more with strenuous endurance training (Holloszy & Coyle, 1984). Saltin et al. (1976) and Henriksson (1977) showed adaptations are limited to the exercised leg when only one is trained. Likewise, Rosler et al. (1985) measured a 40% increase in mitochondrial volume density in trained legs but found a 17% decrease in the untrained arms.

Increased levels of mitochondrial enzymes of the pathway for fatty acid oxidation, the citrate cycle, and the respiratory chain have been demonstrated in the muscles of endurance trained people (Holloszy & Coyle, 1984). The activities of SDH and CO, enzymes of the citric acid cycle and electron transport chain, respectively, are 27-92%

greater in trained compared to untrained musculature (Andersen & Henriksson, 1977a; Gollnick et al., 1972, Henriksson, 1977; Klaussen et al., 1981; Sahlin & Henriksson, 1984; Schantz, 1986). The activity of HAD, a key enzyme in fat oxidation, is also enhanced in active tissue by endurance training (Sahlin & Henriksson, 1984; Schantz, 1986; Simoneau et al., 1983). Increases in these oxidative enzymes is evident in both type I and type II fibres (Schantz, 1981)

The higher levels of mitochondrial enzymes will have two main effects according to Michaelis-Menten kinetics. Firstly, the maximal catalytic rate will increase. Secondly, a given substrate concentration will result in a greater catalytic rate, or a lower substrate concentration can achieve the same catalytic rate as before. Since the maximal catalytic activity of the mitochondrial enzymes are not likely exceeded, the second effect is more likely applicable to metabolic adaptations during submaximal exercise. This type of enzymatic adaptation may be used to explain lower BLa levels at a given workload after endurance training. Mitochondrial content and subsequently mitochondrial enzyme levels have been related to the sensitivity of respiratory control, such that muscles high in mitochondrial content require a smaller increase in [ADP] to stimulate a given rate of mitochondrial respiration (Holloszy & Coyle, 1984). This results in less stimulation of PFK and therefore glycolysis due to lower ADP, AMP, and Pi concentrations, as well as inhibition of PFK by higher ATP levels. With less glycolytic stimulation at a given exercise intensity after training, BLa concentrations will be reduced.

NADH SHUTTLE SYSTEM ENZYMES

Endurance training has the ability to locally increase the activity of the MA shuttle enzymes but leaves the GP enzymes unaltered (Schantz, 1986). The activity of the GP

shuttle reflects the ability of the muscles to oxidize α-glycerophosphate. This parallels the muscles' glycolytic capacity and is inversely related to the capacity for mitochondrial respiration (Holloszy & Coyle, 1984). Enhancement of the MA shuttle may influence LT by contributing to both decreased lactate production and increased lactate removal. Increased enzymatic activity of the MA shuttle may mean a less reduced cytoplasmic redox state is necessary to provide the driving force to accelerate the shuttle's activity. This means there will be a decreased NADH concentration in the cytoplasm at a given VO_2, thereby decreasing the availability of NADH to participate in the conversion of pyruvate to lactate (Schantz, 1986). The MA shuttle enzyme levels were found to be higher in type I fibres both in untrained and trained musculature (Schantz, 1986). This could enhance lactate removal by type I fibres after training since cytosolic NADH is kept lower, and low NADH levels facilitate the conversion of lactate to pyruvate for oxidation.

SUBSTRATE UTILIZATION

Endurance training may significantly increase the capacity of the muscle to oxidize fats and reduce glycogen depletion (Karlson, Nordesjo, & Saltin, 1974). This is due to enhancement of the muscle cell's mitochondrial density, β-oxidation, oxidative phosphorylation, and the electron transport chain (Walsh & Banister, 1988). In addition, preferential proliferation of subsarcolemmal mitochondria, which is closer to the capillaries than interfibrillar mitochondria, suggests better use of blood borne free fatty acids after endurance training (Hoppeler et al., 1985). Greater utilization of fats is supported by an increased volume density of intracellular lipids from 0.47 to 0.92% (Hoppeler et al., 1985). A lower respiratory exchange ratio at both the same absolute and relative intensity after training also indicates a greater reliance on fat fuels (Holloszy & Coyle, 1984). Free fatty acid oxidation has an inhibitory effect on glucose

uptake and glycolysis (Hickson, Rennie, Conlee, Winder, & Holloszy, 1977) and is, therefore, likely related to the decrement in glycogen depletion in trained versus untrained legs (Saltin et al., 1976). Since glycogen depletion is related to fatigue and increased oxidative phosphorylation offsets lactate production, shifts in substrate utilization following training contribute to greater endurance.

Test Specificity of Lactate and/or Anaerobic Threshold

Lactate/anaerobic threshold is subject to variation within the same individual depending on the mode of exercise employed. This is to be expected due to differences in muscle fibre composition throughout the body and the trained state of the muscles, due either to intentional training or habitual use. The results of the following studies provide support for the hypothesis that the effect of training on LT is sport or modality specific.

The first study to measure AT exhibited during different modes of exercise was conducted by Davis and Vodak (1976). They tested 30 male subjects during arm cranking activity, leg cycling and treadmill walking. An additional nine subjects were tested to investigate the validity of AT detection using laboratory measures of gas exchange compared to BLa values. The subjects had no training for four months prior to the experiment. The AT was determined by nonlinear increases in Ve and VCO_2 and abrupt increases in FEO_2. AT detection methods by gas exchange corresponded to those employed by Wasserman et al. (1973), which according to Kinderman et al. (1979) would define the Aerobic Threshold. The results indicated no significant difference existed between the two modes of exercise using the legs. However, the relative AT for arm cranking was significantly lower than both leg cycling and treadmill walking. The investigators suggested that this was due to smaller muscle

mass of the arms and possibly due to specific training adaptations induced by daily activity patterns of the subjects, such as cycling. They also concluded that the gas exchange AT was a valid and valuable indirect method for the detection of the development of lactic acidosis during incremental exercise. In essence then, the results of this experiment do not indicate any specificity in the point of rapid lactate accumulation (LT), but they do suggest specificity in the blood lactate parameter of the first rise in BLa, indicated by respiratory patterns. However, the investigators have not controlled for the effect of training.

Withers, Sherman, Miller, and Costill (1981) investigated the specificity of AT in endurance trained cyclists and runners. This experiment was designed to determine the relative and absolute AT of endurance trained cyclists and runners on both their specific and non-specific exercise modalities. The study involved 10 endurance trained cyclists and 10 endurance trained runners. The sex of the subjects is not stated, but the morphological data suggests that they were male. Each subject performed an incremental exercise test to exhaustion on both a bicycle ergometer and treadmill. Their criteria for identifying AT were identical to those recommended by Davis et al. (1979) which were: 1) an increase in Ve/VO_2 without an increase in Ve/VCO_2 and 2) an increase in FEO_2 without a decrease in $FECO_2$. Again, the physiological landmark measured does not reflect an exponential increase in BLa but rather the first significant increase. Interestingly though, they reported no significant differences in AT, expressed as a percentage of VO_2max, between the cyclists and runners on the bicycle ergometer or between the two groups on the treadmill. When AT was expressed in l/min and ml/kg/min there were significant differences. Based on this data, the researchers suggested that the adaptive responses to exercise (VO_2max and AT) are in part a function of the specific movement patterns executed in training. They recommended further laboratory testing to determine if either of these two

variables involve specifically trained musculature. However, these conclusions based on differences in absolute AT may not be of great significance since as Davis et al. (1979) explained, AT itself provides insight into the circulatory and metabolic responses of exercise stress but "...expressing the AT as a percent fraction of maximal aerobic power allows meaningful comparisons between subjects and modes of activity." The results of this study would suggest that relative AT would not be not significantly different between the trained cyclists and runners. Similarly, Davis et al. (1979) found no significant difference in relative AT between measurements recorded cycling and treadmill running. The involvement of similar muscle groups could help to explain these findings.

Conversely, Wiswell, Girandola, and deVries (1979) as well as Jacobs and Sjodin (1983; 1985) did find significant differences in the relative AT and OBLA, respectively, between bicycling and treadmill. Wiswell et al. (1979) engaged 30 male college students in maximal capacity tests, and AT was determined noninvasively. AT of leg cycling and treadmill running were recorded as 69.4% and 74.5% of VO_2max, respectively. It was concluded in this study that AT may be exercise modality dependent. Similarly, Jacobs and Sjodin (1985) found that OBLA occurred at an oxygen consumption 16% higher during treadmill running than leg cycling exercise in 12 subjects. OBLA was at 85% of treadmill VO_2max and 79% of leg cycling VO_2max. The differences in OBLA (16%) are not commensurate with the differences in peak aerobic capacity between the two modes (9%). Also, the VO_2 at OBLA was not significantly correlated to peak VO_2 on either ergometer. This reemphasizes that different mechanisms exist for the control of these two variables and that LT is a more sensitive indicator of aerobic fitness than VO_2max.

The differences in absolute and relative LT between biking and treadmill running were also not related to the differences in VO_2max in an earlier study by Jacobs and Sjodin in 1983. In both studies (Jacobs & Sjodin, 1983; 1985), the ratio of key glycolytic to key oxidative enzymes were related to the differences in relative LT. The lateral head of the gastronemius and vastus lateralis were chosen to be representative of the predominant muscle groups involved in running and cycling, respectively. The two muscles did not differ in absolute activity levels of CS, PFK and LDH in either study. The ratios of CS/LDH and CS/PFK did differ between the two muscles. In fact, the CS/LDH ratio was significantly higher in the gastronemius than the vastus lateralis in both studies, and the correlation coefficient relating this ratio to the difference in treadmill and cycling relative LT was 0.71 (Jacobs & Sjodin, 1983). These findings suggest that peripheral metabolic characteristics are more significant than central cardiovascular capacities in accounting for the differences in LT between cycling and treadmill running.

Training Lactate Threshold and Other Blood Lactate Parameters

The majority of literature suggests that LT and AT, an indication of the first rise in lactate above resting levels, are trainable if the training program is designed to affect these parameters. The purpose of the research by Kindermann et al. (1979) was to show that work load intensities markedly above AT, as identified according to the gas methods of Wasserman et al. (1973), can be maintained with slightly elevated lactate levels for prolonged periods of time. The experimental procedures involved seven cross country skiers who performed an exhaustive exercise test on the treadmill. Treadmill speed was increased by 2 km/h every three minutes while a constant grade of 5% was maintained. Through interpolation, treadmill speed, VO_2, and heart rates

at the BLa concentration of 4 mmol/l were determined. The beginning of the steep rise in lactate level, 4 mmol/l, was reached at above 80% of maximal work capacity in all athletes. This data dictated the intensities of two further exercise bouts of 30 minutes each. During the first of these subsequent exercise tests, the heart rate coinciding with 4 mmol/l lactate was kept constant. Exercise at a constant heart rate resulted in initial lactate rises to 4 mmol/l in the first 10 minutes. After this, they continuously reduced as a result of treadmill speed reduction. The second test involved maintenance of a constant treadmill speed; the speed at which the lactate concentration of 4 mmol/l was expressed. When exercise was performed at a constant running speed, the lactate values rose to approximately 4 mmol/l around the five minute mark and remained relatively stable for the duration of the test. Most of the subjects were able to continue for 10 to 15 minutes beyond the required 30 minutes without a change in lactate concentration. The results showed that endurance training at intensity levels leading to lactate values around 4 mmol/l can be maintained for 45 to 60 min and longer in several cases. Therefore, they suggested that the optimal intensity of endurance training, which is maintainable and places a high stimulation on oxidative metabolism, is near the "aerobic-anaerobic threshold" of 4 mmol/l lactate. They stated, "In contrast, work load intensities in the range of the AT, as defined by Wasserman et al. (1964, 1973, 1978) are markedly lower." Kinderman et al. (1979) suggested a new arrangement of anaerobic and aerobic-anaerobic concepts. Wasserman's AT was redefined as aerobic threshold; the aerobic-anaerobic transition was proposed to represent approximately 2 to 4 mmol/l lactate; and the former aerobic-anaerobic threshold was redefined as AT - when lactate concentrations increase exponentially at approximately 4mmol/l. This AT proposed by Kinderman et al. (1979) is synonymous with LT defined in this paper.

Yoshida et al. (1982) followed the recommendations of Kinderman et al. (1979) and trained seven male subjects at the workload corresponding to 4mmol/l lactate for 15 minutes, three days/week for eight weeks. The exercise intensity was approximately 80-95% VO_2max. The effects of training on the point at which arterial lactate levels rose above the resting level, defined as AT, were investigated. Training resulted in a 14% improvement in VO_2max and a 37% increase in absolute AT, indicating significant adaptations at this training intensity.

Five men and five women trained on a bicycle ergometer at workloads which elicited heart rates after 10 minutes similar to those measured at the 4mmol/l level in an incremental exhaustive test (Hoppeler et al., 1985). They exercised for 30 minutes, five days/week for six weeks. The training program followed a format of progression such that the workload of the following session was increased by 8 or 9 Watts as soon as the heart rate at the 10 minute mark and the end of the training session dropped enough to indicate the subject could perform at the next higher work load. The results indicated no change in heart rate at the intensity corresponding to BLA levels of 4 mmol/l in the post-training, incremental test. There was a 14% increase in VO_2max and a 7% increase in the VO_2 at which BLa concentrations of 4 mmol/l were measured. The investigators concluded that each subject worked at greater than 85% of maximal heart rate for 2/3 of the program with this protocol and that it was very effective.

Changes in the exercise intensity corresponding to OBLA were studied on eight well-trained male middle and long distance runners after a training program at OBLA (Sjodin et al., 1982). These runners added one 20 minute training session on the treadmill to their normal training program, each week for 14 weeks. The treadmill velocity which corresponded with OBLA (VOBLA) was significantly improved after

the 14 weeks. However, the VO_2 defined by OBLA increased insignificantly from 58.6 to 60.6 ml/kg/min as did the relative VO_2 from 85.3% to 86.6% VO_2max. The initial high level of aerobic fitness and the relatively few training sessions at OBLA may have contributed to the minor changes in oxygen cost at OBLA (Bouchard, Boulay, Thibault, Carrier, & Dulac, 1980). However, there was an increase in VOBLA, shifts in LDH isozymes, and a decrease in the PFK/CS ratio, in the active skeletal muscles without a significant change in maximum aerobic power. This may provide support to Kindermann et al. (1979) who suggested that an optimal training intensity was at BLa concentration of around 4 mmol/l. Therefore, these investigators also suggested that "... VOBLA may be a more sensitive parameter for the monitoring adaptations of training than is the determination of VO_2max."

An early study which examined BLa concentrations after training was carried out by Saltin et al. in 1969. This study involved 42 sedentary males with a mean age of 40.5 years. The subjects were pre-tested and post-tested on a Krogh bicycle ergometer during submaximal and maximal exercise. Blood lactate fluctuations were measured in submaximal tests during which the subjects exercised on different occasions for six to seven minutes at 300, 600 and 900 kpm/min. After the training program, a 1200 kpm/min workout was included for some of the subjects. The training program ran for 8-10 weeks for two to three sessions each week. However, it is important to note that the mode of training differed from the mode of exercise tested. The subjects ran about two miles each session. This distance was covered with high intensity intervals two days per week and continuous running one day per week. It may have been that is was this failure to use the same mode of exercise for testing and training which resulted in a lack of relative BLa changes. Other factors may have been the relatively low training frequency and the small number of BLa samples taken.

Henritze, Weltman, Schurrer, and Barlow (1985) trained female subjects at or above LT. Their definition of LT represents the highest work rate and associated VO_2 attained that was not associated with an elevation in lactate above baseline levels. Although this intensity is not the same as LT defined in this thesis study, it deserves attention. The training intensities determined according to LT are relatively low and yet significant training adaptations were observed. Training at LT (mean value 44% VO_2max) did not increase VO_2max, but relative LT was raised by 16%. Training above LT elicited no significant improvement in VO_2max but did increase absolute and relative LT by 48% and 42%, respectively. However, they suggested that their test for LT may not have been sensitive enough, since increases of 25-35 Watts per stage in the incremental tests resulted in rises in VO_2 of 10 to 15% of VO_2max. Error in LT determination may, therefore, account for the large increases in LT with such low intensity training. The results of this study suggest the importance of very gradual increases in workload in an incremental test to accurately identify LT.

A study designed to determine if LT could be increased significantly within the first three weeks of exercise training was conducted by Gaesser and Poole in 1986. Training involved cycle ergometer exercise at 70-80% of pretraining VO_2max for three weeks, six days per week for 30 minutes. LT was determined from BLa vs VO_2 graphs and identified as the VO_2 prior to that point at which an abrupt and nonlinear increase in BLa occurred. There was no significant difference before training in LT and in VT, the VO_2 at which Ve/VO_2 increased without a simultaneous increase in Ve/VCO_2. After the three week period, exercise training produced changes in LT but not VT. Absolute and relative LT increased 29.3% and 14.2%, respectively. VO_2max was augmented by 11.1%. Gaesser and Poole (1986) proposed that the most significant adaptations to LT occurred within the first few weeks of training, when compared to other studies of comparable frequency and duration but differing in number of weeks training.

Poole and Gaesser (1985) chose to investigate the influence of high and low-intensity, continuous training and high-intensity, interval training on LT and ventilatory threshold (VT). Seventeen sedentary young males volunteered to participate in the training which lasted for four weeks with three training sessions per week. The subjects were divided into the three different training groups. All exercise as well as pre and post-testing was done on cycle ergometers. LT was calculated as the VO_2 associated with the work load prior to an increase in venous lactate above resting values. "This corresponded with the onset of an exponential rise in BLa." VT was determined at the VO_2 at which Ve/VO_2 increased without a simultaneous increase in Ve/VCO_2. The results for all three training groups showed increases in VO_2max, maximum work rate, and relative and absolute LT. There were no significant differences between groups with respect to the variables measured. It was concluded that continuous and interval training were equally effective in elevating LT and VO_2max in a four week training program. Further increases above a training intensity of 50% VO_2max (similar to the low intensity, continuous group) did not alter LT to any greater extent. Another important point to be drawn from this study was the observation of a significantly greater increase in LT than VT for the low intensity, continuous group. This suggests that LT and VT are regulated by different mechanisms; therefore, the two parameters should not be used interchangeably to measure the effects of training.

A number of studies have used noninvasive methods to determine the effect of training on the first rise in BLa concentrations above resting levels. Assuming that there is some correlation in ventilatory parameters and BLa concentrations, it may be of interest to review these studies.

Davis et al. (1979) evaluated the relative alterations in AT and VO_2max of nine middle-aged normal sedentary males after nine weeks of endurance training. AT determined by gas analysis actually corresponded to the aerobic threshold identification by Kindermann et al., (1979), thought to indicate the first elevation of BLa above resting levels. The subjects exercised on bicycle ergometers five days per week for 45 minutes at 50% of the way between "AT" and VO_2max for the first four weeks and at 70% of the difference for the last five weeks. Both absolute and relative "AT", as identified during a maximal exercise test, increased significantly after training by 44% and 15%, respectively. VO_2max improved by 25%, and maximum ventilation and maximum work rate also improved. During a constant load cycling test at an intensity just below the pretraining "AT", the subjects were able to perform a greater amount of work without an accumulation of BLa. The investigators concluded that "AT" of previously sedentary, middle-aged males is profoundly influenced by endurance exercise.

Another study, almost identical in protocol, trained both males and females for eight weeks on a bicycle ergometer, five days per week for 45 minutes each session (Casaburi, Storer, Ben Dov, & Wasserman, 1987). The exercise intensity for the first four weeks was 50% of the way between AT and VO_2max, but the intensity was increased to 75% of the difference for the last four weeks. The results were also comparable to those of Davis et al. (1979) with improvements in relative and absolute AT of 38% and 16%, respectively. VO_2max increased by 15% after training.

In summary, there are different training methods which will influence BLa parameters, and there is little good reason to include or exclude a specific model on the basis of these varied results. However, the design and duration chosen for the experiment should be carefully controlled. The studies indicate that after training BLa

concentrations are lower compared to the same absolute and relative exercise intensity before training. Increases in LT may occur with moderate training intensities and with or without concomitant changes in maximal aerobic capacity.

Specificity of Training Adaptations and Blood Lactate Concentrations

Training has demonstrated a transfer effect of increased maximal aerobic capacity in activity with untrained musculature (Clausen et al., 1973; Lewis et al., 1989; Loftin et al., 1988; Rosler et al., 1985; Saltin et al., 1976). However, this does not seem likely to apply to LT. Decreases in muscle and BLa at the same absolute and relative submaximal workloads following an endurance training program have been demonstrated while exercising with the training muscles. However, these changes were absent for exercise with untrained muscles. These findings provide support for the specificity of training LT.

Donovan and Brooks (1983) proposed that LT is improved due to increased lactate removal rather than decreased lactate production. The liver is an important site of lactate removal, but during exercise its influence is decreased due to reduced blood to the organs (Donovan & Brooks, 1983). However, Clausen et al. (1973) gave evidence that hepatic-splanchnic blood flow at a given VO_2 was less reduced during exercise following training. This finding is of further significance since ICG clearance, used to indicate hepatic-splanchnic blood flow, was unchanged during exercise with untrained muscles. This suggests specificity of a training response which could influence BLa concentrations, although they were not measured in the study by Clausen et al. (1973).

Adaptations to one leg training were investigated by Klausen et al. (1982). The training program consisted of one leg cycling with each leg for 30 minutes, three days per week for a duration of eight weeks. Comparisons of results were made between one and two leg exercise before and after training. During one leg exercise the arterial lactate concentrations were about 4 mmol/l higher than during two leg exercise at the same VO_2, as would be expected given the smaller muscle mass for one leg cycling. Arterial BLa concentrations decreased after training during submaximal exercise for both one and two leg exercise. The venoarterial lactate difference was only slightly decreased during two leg exercise, while it was substantially reduced during one leg exercise. This could be attributed to the increase in leg blood flow seen with one leg exercise but not with two legs. This indicates that increased lactate removal is possible in the specific activity pattern for which the individual was trained.

One legged bicycle training was employed with 13 male students by Saltin et al. (1976) to distinguish between the local and general effects of training with different training procedures. Training of one leg was done by either an endurance or sprint procedure, and a third group trained both legs using the two different approaches. When exercising with the trained leg, subjects exhibited an increased VO_2max, lower heart rates, and decreased BLa responses at submaximal work levels compared to measurements obtained from exercise with the untrained leg. It was hypothesized that the lower lactate levels could be explained by increased oxidative capacity, as evidenced by changes in SDH activity found in the trained leg, as well as a significantly lower glycogen depletion in the trained versus untrained leg. It was concluded that the adaptations to training were primarily local in nature, but improved central circulation was evidenced during exercise with untrained muscles as the non-trained leg exhibited a 6% increase in VO_2max.

Henriksson (1977) engaged subjects in one-leg bicycle exercise for two months. SDH activity increased significantly in the trained leg as did fat oxidation. This suggested that metabolism of fat was a function of increased muscle oxidative capacity. From these findings, it would be expected that BLa level would be reduced in trained leg exercise. In fact, the BLa level at 100 W was significantly lower in the trained leg compared to pretraining level. The untrained leg also exhibited a lower BLa level after training, but this change was not significant.

A slow VO_2 on-response appears to be associated with lactate accumulation. Cerretelli et al. (1979) set out to investigate if specific muscle training influences the interrelationships between half time of the VO_2 on-response ($t_{1/2}$ VO_2on) and early BLa increases. In a cross sectional study, it was found that subjects with already trained arms or legs exhibited shorter $t_{1/2}$ VO_2on and lower BLa concentration when exercising with trained limbs. In a longitudinal study, subjects were trained either leg cycling or arm cranking for five weeks, five days per week for a duration of 30 minutes. The results again showed a shorter $t_{1/2}$ VO_2on accompanied by lower lactate levels for the trained limbs. It was previously discussed that trained muscle cells require a smaller disturbance in intracellular homeostasis in order to stimulate a given level of mitochondrial respiration, and this may result in less lactate production. The findings of these two studies suggest that this is true and that these adaptations are specific to the trained limbs.

Rosler et al. (1985) investigated whether the transfer effect in aerobic capacity of trained to untrained muscles could be explained by structural changes in the untrained muscles. They hypothesized that increases in mitochondrial volume density, capillary per fibre ratio, and number of capillaries for muscle fibre area would not be found in the untrained muscle, since these changes are usually evidenced as local training

adaptations believed to be responsible for increased VO_2max. Their experiment was designed to study the effects of eight weeks of bicycle training, performed five days per week at the maximum power output that could be maintained for 30 minutes. The 10 male subjects were pre- and post-tested during arm and leg ergometry and on the ultrastructure of the untrained arm muscle (deltoid) and trained leg muscle (vastus lateralis). The results showed an increased leg VO_2max of 13% accompanied by a 40% increase in mitochondrial volume density and a 15% increase in capillary per fibre ratio. In contrast, the mitochondrial volume density unexpectedly decreased by 17% in the arm, and the capillary per fibre ratio remained unchanged even though arm VO_2max improved by 9%. In search for an answer to this contradiction, the investigators concluded that the improvements in arm VO_2max could not be attributed to an enhanced cardiovascular fitness, since arm exercise before and after the training did not fully tax the system. They hypothesized that during arm exercise the increased capacity after training could be accounted for by a greater uptake and oxidation of lactate by the legs, which accompanies endurance training. Their discussion is well argued; however, it does not take into consideration their BLa results. These showed that 4 mmol/l levels of plasma lactate were reached at the same absolute workload before and after training during arm exercise, although this power output increased by 27% in leg exercise. Since BLa values should represent the difference between output and removal, one would expect the lactate levels to have decreased during arm exercise if in fact the legs were metabolizing a significantly increased amount of lactate, sufficient to increase the arm VO_2max by 9%. Since absolute LT remained stable during arm exercise after training and VO_2max increased, it is likely that had calculations been made on the relative LT, it would have shown a decrease. This has not been supported by other studies. The findings of this study, therefore, seem to support the specificity of training absolute LT, but no data was presented with respect

to relative LT changes. The study being presented in this paper is similar in design to the one undertaken by Rosler et al. (1985); however, changes in relative LT will also be examined.

Kayak and bicycle training were used to compare the circulatory and metabolic responses to both submaximal and maximal kayak ergometer performance in an investigation by Ridge et al. in 1976. Ten healthy males who were untrained for both kayaking and cycling were chosen as subjects and tested during two submaximal load tests and a maximal exercise test on the kayak ergometer, before and after a four week training program either kayaking or cycling. Following training, the kayak group demonstrated significantly reduced VO_2 at both submaximal loads while kayaking, but the bicycle trained group did not. As expected, the VO_2max kayaking showed a significant improvement for the kayak trained group, but the bicycle group did not improve. BLa accumulation was significantly reduced after training in the kayak trained group at the same relative work loads on the kayak ergometer. The bicycle group failed to show a significant improvement after training. The researchers postulated that these training adaptations could be related to increased oxidative capacity of the muscle fibres or improvements in efficiency. They suggested that the major effect of training on the performance of arm work was due to circulatory and/or metabolic adaptations at the local muscle level.

In summary, these studies support the hypothesis that specific muscular adaptations account for changes in BLa concentrations and/or physiological variables which could be related to lactate metabolism.

PROTOCOL

Considerations for the Training Protocol

The two most important variables in a training program are duration and intensity. If training adaptations that will affect LT are to be elicited these must be carefully considered and controlled. Schantz (1986) suggested that if the training stimulus is below a critical point it cannot be compensated for by long duration exercise. Fibre type transformations are especially dependent upon a sufficiently high training intensity (Pette, 1984; Schantz, 1986). The greater the demands placed on the human body, the more intensively it will adapt and become more efficient, within physiologic limits (Hollmann et al., 1981). This suggests that if one wishes to train LT it may be best to exercise at intensities above this critical intensity.

A successful training program will also incorporate a progression of the training intensity. This is necessary since the individuals' capacity will increase during the training period, and stimulus well within the capacity of the untrained individual will not provide a major adaptive stimulus (Holloszy & Coyle, 1984). The most common means of increasing intensity is by raising the workload, thus ensuring that the training intensity remains above LT throughout the training program. By training at intervals above LT and then recovering inbetween, the individuals must cope with a high BLa concentration and subsequently handle the process of lactate removal.

Prescribing training intensity with respect to LT rather than as an arbitrary percentage of maximal heart rate or VO_2max will result in similar training stresses on the individuals (Dwyer & Bybee, 1983; Jacobs, 1986). Although heart rate cannot be used to identify LT, it is a tool with which training intensity may be monitored (Dwyer & Bybee, 1983). However, above LT heart rate continues to drift towards maximum during steady state activity (Ribeiro et al., 1986).

Lactate Threshold Determination

Exercise tests conducted to identify LT are often incorporated into tests designed to measure maximal aerobic capacity. Consideration of stage lengths and workload increments are very important in order to obtain valid and reliable data for both LT and VO_2. Significantly higher VO_2max values were found for a one minute incremental test compared to either a three or five minute incremental test, but OBLA was not significantly different in the three test conditions (McLellan, 1985). This suggests that in order to obtain accurate relative LT values, which are dependent on valid VO_2max measurements, the stage lengths should not be too long, since total test time will then be lengthened. Yet, if the stage increments are too often, LT may be overestimated due to insufficient time to allow the development of even close to steady state conditions at that workload. It has been suggested that three minute stage durations are optimal for LT determination (Yoshida, 1984); however, this may compromise the accuracy of VO_2max by creating too long of a test. Wasserman (1987) suggested that maximal aerobic capacity is highest when the test length is 6-15 minutes in length. On the other hand, total test time is also dependent upon the magnitude of the workload increment. With large increments, which shorten test time, there is a loss in sensitivity of identifying LT. Recall that Henritze et al. (1985) found large improvements in LT after training which may have been erroneous due to the large increments in workload. Preliminary studies have revealed that for the purpose of this experiment two minute stage lengths combined with 0.25 kp increments for arm cranking and 0.25-0.5 kp increments for leg cycling have been reliable in identifying both LT and VO_2max, while generally keeping the total test time between 14 -17 minutes.

Considerations for the Arm Cranking Protocol

Arm cranking is a valid and reproducible mode of testing for measuring the aerobic capacity of an individual for upper body exercise (Bar-Or & Zwiren, 1975; Sawka, 1986). Arm exercise it not limited by central cardiovascular abilities, however, since a smaller muscle mass is recruited compared to leg exercise. On the average, arm cranking VO_2max is 65-70% of leg VO_2max in individuals untrained for upper body exercise (Bar-Or, 1975; Franklin, 1985; Sawka, 1986). Other comparative observations between arm cranking and leg cycling include higher heart rates and BLa concentrations and lower stroke volumes for arm ergometry, at a given submaximal VO_2. Higher lactate levels, heart rates and VO_2 are also seen at a given power output during arm ergometry, since it represents a greater relative intensity than during leg cycling (Franklin, 1985; Sawka, 1986). A review of the literature also revealed that maximal heart rates are generally lower during arm versus leg exercise. The difference ranges from 3 to 23 beats/minute, and there is a mean difference of 11 beats/minute (Franklin, 1985). Although the two exercise modes elicit different physiological responses at a given workload, the same general test principles used for lower body exercise should be employed for upper body testing (Sawka, 1986).

However, the single best test methodology for upper body exercise has not been determined, and a review of the literature reveals that little standardization in the protocols has been employed to date. Investigators seem evenly split on the utilization of continuous or intermittent protocols to measure arm VO_2max, with rest periods during discontinuous exercise ranging from 1 to 20 minutes (Sawka, 1986). It is suggested that intermittent protocols may enable improved maximal performance by minimizing the effects of localized fatigue (Sawka, 1986). It also allows for fingertip blood sampling during the intervals. However, for the purposes of standardization

between arm and leg ergometry and the identification of LT, the continuous protocol is more suitable for this study.

There is inconsistency in the continuous protocols with respect to both stage lengths and workload increments. Stage lengths vary from one to six minutes, with the majority of studies using two or three minutes. The increments in workload vary from 5 to 25 Watts, while increments around 16 Watts appear to be the most common (Franklin, 1985; Sawka, 1986).

There is variation in the relative position of the crank arm, such that the subject has been positioned with the center of the ergometer shaft level with the shoulder (Bar-Or & Zwiren, 1975; Ceretelli et al., 1979; Lewis et al., 1980; Pendergast, Ceretelli, & Rennie, 1979; Tesch & Lindeberg, 1984), heart level (Loftin et al., 1988), glenohumeral joint (Reybrouck, Heigenhauser, & Faulkner, 1975) as well as at midsternum and table top levels in other studies (Sawka, 1986). The influence of the crank axis position on physiological responses is conflicting and requires further research (Sawka, 1986). Blood samples have been obtained from the earlobe (Rosler et al. 1985), finger tip during a discontinuous protocol (Steinacker, Marx, Marx, & Lormes, 1986; Tesch & Lindeberg, 1984), and antecubical vein (Pendergast et al., 1979). The rate of arm cranking is also not standardized with all investigations. A range of 30 to 90 RPM has been used, with most studies averaging around 50 to 60 RPM (Franklin, 1985; Sawka, 1986).

In summary, the wide variety of testing protocols suggests that the investigator should experiment before hand to develop a protocol that is suitable to both the subjects and purpose of the study.

METHODOLOGY

INTRODUCTION

This study was conducted to determine if the effects of leg cycling training were specific to this mode of exercise or if there was also evidence of training adaptations for arm cranking exercise. This chapter describes and outlines the subject population, the testing and training procedures followed, the means of data collection, the analysis of blood samples employed in order to investigate the problem, and the statistical design used to analyze the data.

SUBJECTS

Seven healthy male volunteers participated in this study. All were moderately active individuals who were not engaged in any regular endurance or weight training regime at the onset of the study nor in the recent past.

The subjects were fully informed of the experimental procedures, as well as the risks and discomforts associated with the study, before they consented to participate. Prior to any testing, each subject signed a consent form (see Appendix C) which verified that they had understood the verbal explanation of the treatments and tests. Before any testing sessions, the subjects were brought to the lab for familiarization with the environment and the equipment involved in the testing and training. Subjects were

instructed to refrain from large meals and caffeine for three hours preceding each test. They were also urged to avoid intense exercise on the day before and day of testing.

EXPERIMENTAL DESIGN

Three healthy male subjects were tested several months before the experiment began. Their lactate threshold and maximal oxygen consumption were measured during both arm cranking and leg cycling exercise before and after an eight week period with no endurance training. The data collected from these subjects indicated that neither lactate threshold nor maximal oxygen consumption were significantly altered following this eight week period of normal activity. On the basis of these data, it was decided not to include a control group in the experimental design of this study. A fourth subject was also tested at the same time as the experimental subjects, and measurements of lactate threshold and maximal oxygen consumption were not different between the two tests. The results for these four subjects were combined for statistical analysis and are presented in Table 1. These pilot subjects had a mean age, weight and height of 22 years, 78.4 kg, and 179.5 cm, respectively, which was similar to the experimental group's mean age, weight and height of 21 years, 77.9 kg, 179.1 cm, respectively. No significant differences were found between pre- and post-testing values for absolute and relative VO_2max nor for absolute and relative LT.

All subjects underwent maximal aerobic power (MAP) and LT exercise testing before and after an eight week endurance training program. The exercise testing, both pre and post training, included one progressive, incremental, MAP-LT test while arm cranking and one while leg cycling. Each complete MAP-LT Test involved a phase of

Table 1

Summary of Results for Non-experimental Subjects

		Absolute VO_2max (l/min)	Relative VO_2max (ml/kg/min)	Absolute LT (l/min)	Relative LT (ml/kg/min)
Leg Cycling	Pre	3.51 ± 0.35	45.1 ±4.2	2.42 ±0.44	68.5 ±7.0
	Post	3.53 ±0.41	45.1 ±3.4	2.51 ±0.47	70.6 ±6.9
Arm Cranking	Pre	2.29 ± 0.19	29.5 ±2.9	1.35 ±0.13	59.0 ±2.2
	Post	2.28 ±0.05	29.6 ±3.9	1.38 ±0.19	60.1 ±5.3

N = 4; Values are means ± SD
VO_2max, maximal oxygen consumption
LT, lactate threshold

continuous, progressive exercise, following the format of Table 2, until the subject was unable to maintain the workload. If a plateau in VO_2 was not attained in this test then a phase of continuous exercise at the maximal intensity identified during the progressive phase was administered, following recovery to a heart rate of less than 100 bpm (approximately 10-15 minutes). In addition, each subject performed a LT verification test for each exercise mode (see Figure 6). All tests were conducted on separate days within one week, such that four test days were required per subject before and after training. The order of the maximal tests was randomized for the pre-training test period and the order was maintained for the post-testing sessions. The purposes of these tests were:

1) to establish the absolute and relative VO_2max for each exercise mode

2) to identify the absolute and relative LT for each exercise mode

3) to determine the training intensities for the intervals above and below LT when leg cycling

The training program involved leg cycling exercise which was performed at intensities which alternated from above to below LT, with a minimum of one day between training sessions. It consisted of eight weeks of training three times per week. All post tests were conducted within one week of the last training session.

MAP-LT TEST

Subjects performed two step-wise, incremental MAP-LT tests within one week according to the protocol in Table 2. The workload associated with LT was used as the first steady state load in the LT Verification Test. One MAP-LT test involved leg cycling on a Monark bicycle ergometer, and the other test involved arm cranking on the same Monark bicycle ergometer with modified hand grips substituted for the pedals. For arm cranking, the subjects were seated with the bicycle stabilized on a table. Half of the subjects were randomly assigned to begin testing with the leg cycling MAP-LT Test, and the other half began testing with the arm cranking MAP-LT Test. The same order was maintained before and after the 8 week interval.

Special care was taken to ensure that the subjects were positioned correctly before and during the test. For leg cycling, the bicycle seat was adjusted to a height that was both comfortable and allowed a slight bend in the knee when the leg was fully extended during pedaling. This height was recorded and used for all subsequent tests. Similarly, the stool on which the subjects sat for arm cranking was adjusted so that the crank axis was approximately at the height of the glenohumeral joint, and this height was recorded and used for all subsequent arm cranking tests. The subject sat on the

stool some distance from the table on which the ergometer was placed, such that the elbows never fully extended at any time when cranking. Considerable care was taken to center the stool and the subject on the stool so that the work would be divided as equally as possible between each arm.

MAP-LT Test Phase I

The subjects' height and weight were recorded before they began their five minute warm-up at a resistance lower than the first test intensity. Subjects were encouraged to stretch well after the warm-up. The tests began when the subjects indicated readiness. A cadence of 60 rpm was maintained throughout both arm and leg cycling tests. The initial resistance on the ergometer for the pre-testing ranged from 0.5-1.0 kp and 1.5-2.0 kp for the arm cranking and leg cycling tests, respectively, in order to elicit heart rates of approximately 100-120 bpm. The work load was subsequently increased every two minutes until completion of the test. During arm cranking, the workload was increased by a resistance of 0.25 kp each stage (14.7 Watts). During leg cycling, the workload was increased by 0.5 kp (29.4 Watts) after each two minute stage until the subject's heart rate was approximately 140 bpm. After this point, the resistance was increased by only 0.25 kp for each new two minute stage. The resistance for the first exercise stage of the leg cycling post-training test ranged from 2.0-3.0 kp in order to attain the same heart rates that had been seen in the first stage of the pre-training test.

Subjects were verbally encouraged to continue until they reached their maximal aerobic power (VO_2max). This normally coincides with a perceived, complete exhaustion of the ability to perform at the prescribed intensity[1] or the inability to maintain the proper cadence. The criterion for VO_2max was:

[1] Exhaustion is relative to a specific intensity performance and does not relate to a breakdown of cardiopulmonary, CNS, metabolic or postural functions.

1) a levelling of VO_2 between the last stage two or more stages, i.e. a change in VO_2 of less than 2 ml/kg/min. with a heart rate greater than 180 bpm or equal to the known maximum heart rate

2) a decrease in VO_2 from a previous peak, with a heart rate greater than 180 bpm or equal to the known maximum heart rate

Table 2

Sample Protocol for a Leg Cycling MAP-LT Test

Time (min)	Stage	Resistance (kp)	Time of Blood Sample (min)
0 to 2	1	1.50	2:00 to 2:15
2 to 4	2	2.00	4:00 to 4:15
4 to 6	3	2.50	6:00 to 6:15
6 to 8	4	2.75	8:00 to 8:15
8 to 10	5	3.00	10:00 to 10:15
10 to 12	5	3.25	12:00 to 12:15
Pattern continues until test termination			

Note.
Initial resitance and the stage at which the resistance begins to increase by only 0.25 kp varies between subjects.

MAP-LT Test Phase II

If neither of the outlined conditions for a obtaining a true VO_2max were met at the end of the MAP-LT Test then the subject continued with Phase II. A 10-15 minute recovery period was allowed until the heart rate was below 100 bpm. The subject then

resumed exercising at the workload of the last completed stage of phase I and continued exercising until unable to continue, usually at least two to three minutes later. The highest VO_2 of the two tests was taken as the VO_2max.

Blood Lactate Measurement

Capillary blood samples of approximately 30 uL were taken from a fingertip in the leg cycling tests and from a pre-warmed toe in the arm cycling tests, in order to allow continuous exercise. Warming of the toes with an electric heating pad, set at medium, was necessary in order to obtain adequate blood flow. All blood sampling was done within 15 seconds following each increment in workload. Exactly 20 uL of the blood in the heparinized capillary was then transferred into a vial containing 380 uL of diluting solution and thereby haemolysed. Blood samples were measured within five hours[2] using a Kontron Model 640 Medical Lactate Analyzer[3] to determine their lactate concentration.

Oxygen Consumption Measurement

During each test, VO_2 was measured via the open circuit method with the subjects breathing through a two way, non-rebreathing, T-shaped valve (Hans Rudolph - Large 2 Way, No. 2700). Expired air was passed through a Roxon Metabolic Cart (Ametek) which calculated the VO_2 at 30 second intervals using inspiratory volume information measured by a Morgan Ventilometer Mark 2 turbine attached to the mouth piece.

---

2 Samples are stable for at least 48 hours (Kontron Medical, 1981).

3 Lactate concentration is determined by enzymatic-electochemical processes. The oxidation of lactate to pyruvate by the enzyme cytochrome b2 results in the production of an electrochemically active substance which produces a current in proportion to the sample's lactate concentration (Kontron Medical, 1981).

These VO_2 values were used to obtain immediate feedback on the progression of the test. Calibration of the Roxon Metabolic Cart's gas analyzers and of the ventilometer was performed prior to each testing session. The VO_2 values which were used for the data comparisons were determined using expired volume measurements obtained by sampling with a 120 l tissot tank during the last 30 seconds of every two minute stage and during the final 30 seconds of the test.

Heart Rate Measurement

Heart rate was monitored continuously with a Sport Tester PE3000 (Polar Electro Ky) which the subjects wore around their chests throughout the tests. The recording sport tester watch was set on memory to store heart rates at five second intervals throughout exercise and recovery. These data were filed using interfacing with an IBM computer and PE3000 software.

LT DETERMINATION

LT was initially identified from a graph of BLa concentration (mmol/l) versus resistance (kp) using the data obtained in the MAP-LT tests. LT was represented as the VO_2 associated with the workload just before BLa concentration exhibited an abrupt rise (approximately equal to 1 mmol/l during one test stage) and continued to rise sharply with subsequent increases in workload. LT identified by this means was subsequently verified with the LT Verification Test protocol.

LT VERIFICATION TEST

LT in a progressive exercise test, with two to three minutes at each work increment, should coincide with the highest exercise intensity at which BLa concentrations can still exhibit a plateau. Therefore, the LT Verification tests were performed in order to substantiate that exercise at the ALT VO_2, as initially identified by the MAP-LT tests, represented an intensity for which BLa concentrations did not rise continuously. The protocol for the LT Verification Test (see Figure 6) was based on previous, unpublished research in our laboratory (Sylvia Weihrer, 1989) which demonstrated that BLa concentrations have stabilized between the 8 and 12 minute mark during a 20 minute, constant workload test performed at LT intensity. This observation allowed for a shorter test time to determine if a given exercise intensity was above or below LT.

On a day following the initial determination of LT from each MAP-LT Test, each subject performed a LT Verification Test at the same 60 rpm cadence used for MAP-LT tests. A test involved two 12 minute exercises separated by a five to eight minute active recovery period rest. The first 12 minute workload (Load 1) was that which was associated with LT in the MAP-LT tests. The second 12 minute workload was 0.25 kp (14.7 Watts) above the first workload (Load 2A) or 0.25 kp below the first workload (Load 2B), depending on the BLa response to the first 12 minute period. Subjects were given a five minute warm-up at 30% VO_2max, after which the ergometer resistance was increased to the first workload. The subjects exercised at this load for 12 minutes during which time heart rate and VO_2 were monitored. Capillary blood samples were taken at the 8th and 12th minute. Each sample was analyzed immediately after it was taken in order to determine if lactate levels were rising, stable or decreasing. If the BLa concentrations had increased by a rate greater than 0.05

mmol/l/min for the workload period between samples then the subject was judged to be working above LT, and the workload for the second 12 minute stage was decreased by 0.25 kp (14.7 Watts). On the other hand, if the BLa concentrations between 8 and 12 minutes exhibited a decrease or failed to increase by more than 0.05 mmol/l/min then the subject was judged to be at or below LT, and the workload was increased by 0.25 kp (14.7 Watts) for the second 12 minute test. Before the second phase, subjects were allowed to recover for a period of four minutes at 50% VO_2max. If the heart rate had not recovered to approximately 120 bpm, the recovery workload was further lowered to the warm-up workload (30% VO_2max) for one to three more minutes, thus allowing approximately five to eight minutes of recovery, at approximately 30-50% VO_2max, between steady state loads. Blood samples were taken again at the 8 and 12 minute mark of the second constant workload.

If the subject failed to exhibit a rise in BLa concentrations in both of the verification workloads then further testing was required to find the exercise intensity corresponding to LT above which lactate levels continue to rise in the test. On a separate day, the subject performed at the final exercise intensity of the first LT Verification Test followed by active recovery and then another 12 minutes at a workload higher by 0.25 kp (14.7 Watts).

LT was defined as the highest mean VO_2 and the constant workload during which BLa concentrations stabilized or decreased.

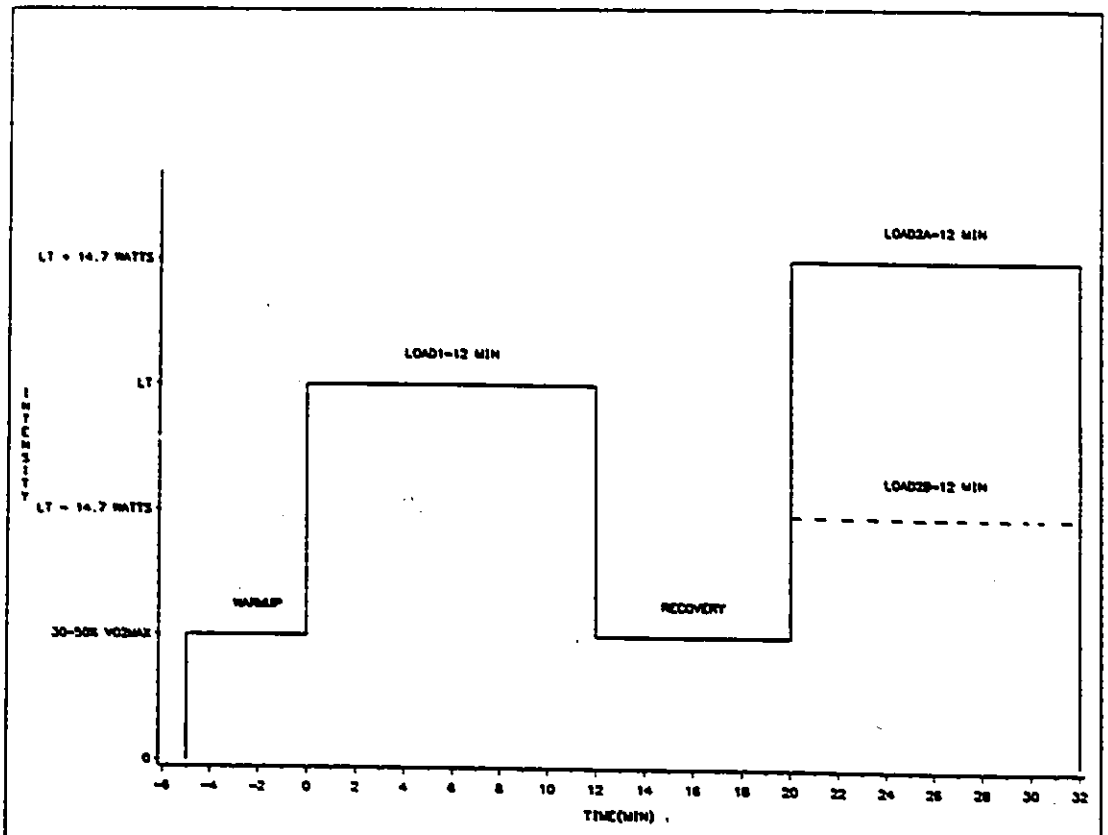

Figure 6. Diagramatic representation of the LT Verification Test protocol. The exercise intensity for the second 12 minute period of exercise, Load2A or Load2b, depends upon the blood lactate response to the first 12 minute period of exercise, Load1.

EXAMPLE OF LT DETERMINATION

Sample data from a leg cycling MAP-LT Test is presented in Table 3. These data have been graphed with BLa concentration (mmol/l) as a function of pedaling resistance (kp) for each two minute stage (see Figure 7). These data show that after two minutes of exercise at each stage up to 2.5 kp, the subject's BLa concentration had risen by less than 1.00 mmol/l between samples. However, after exercise at 2.75 kp,

the subject's BLa concentration had risen by 1.26 mmol/l to 4.06 mmol/l, and this represented the first stage of exercise with an increase in BLa concentration of greater than or equal to 1 mmol/l. After this workload, the values continued to rise sharply. Therefore, the 2.5 kp stage was judged to be the LT workload and was chosen as the first workload for the LT Verification Test.

Table 3

Sample Data From a Leg Cycling MAP-LT Test

Resistance (kp)	Time (min)	VO_2 (ml/kg/min)	Heart Rate (bpm)	Lactate (mmo/l)
1.5	2:00	19.2	127	1.84
2.0	4:00	24.0	135	1.94
2.5	6:00	27.7	154	2.80
2.75	8:00	32.2	164	4.06
3.0	10:00	34.8	177	5.34
3.25	12:00	37.7	187	7.00
3.5	14:00	44.5	198	8.00
3.75	15:00	43.5	201	10.70

VO_2, oxygen consumption
HR, heart rate

The results for the LT Verification Test for this subject are presented in Table 4. The first 12 minutes of constant workload exercise were performed at a resistance of 2.5 kp, as determined from the MAP-LT Test. Blood samples were taken at the 8th

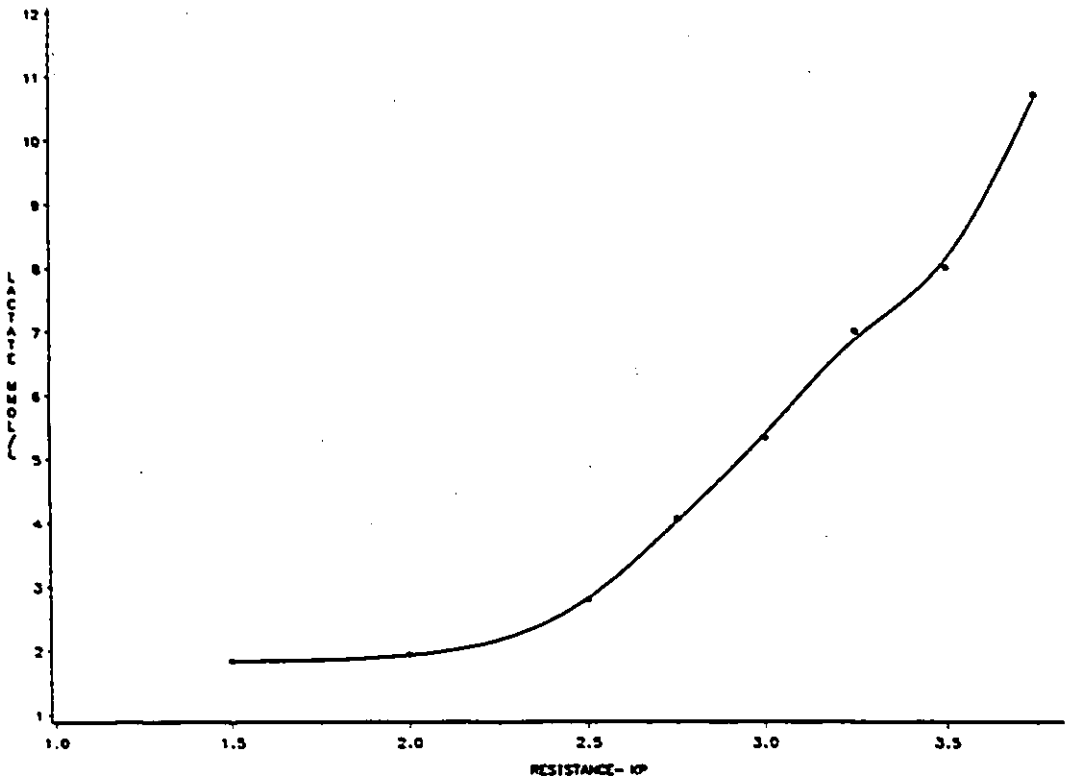

Figure 7. Blood lactate concentration (mmol/l) versus resistance (kp) graph of sample MAP-LT data in order to initially determine lactate threshold and the associated workload.

and 12th minute of this test, and it was found that the lactate concentration had decreased from the 8 minute to the 12 minute sample. The rate of change averaged

-0.05 mmol/l/min. Therefore, this exercise intensity could be maintained for this subject without a continual rise in BLa concentration. In order to confirm that this was the highest exercise intensity at which maintenance of BLa concentration was possible, the subject to exercised for another 12 minutes at a resistance of 2.75 kp. During this phase of the test, the subject's BLa concentration increased by a rate of 0.25 mmol/l/min from the 8 to 12 minute mark. This rate of change was greater than the criterion of 0.05 mmol/l/min that represented the maximum rate of increase at which BLa concentrations would be considered stable. Therefore, this test confirmed that LT could be identified during constant workload exercise at a resistance of 2.5 kp. The oxygen consumption for the 12 minutes at this workload remained relatively stable. Therefore, the VO_2 measurements, calculated using tissot sampling at the end of each 2 minutes, were averaged to give a LT value of 29.2 ml/kg/min.

Table 4

Sample Data From a Leg Cycling LT Verification Test

Resistance (kp)	Time (min)	VO_2 (ml/kg/min)	Heart Rate (bpm)	Lactate (mmo/l)
2.5	2:00	29.2	154	
	4:00	28.0	156	
	6:00	29.3	158	
	8:00	28.9	154	5.06
	10:00	29.1	158	
	12:00	30.9	158	4.84
Rate of change (mmol/l/min)				-0.05
LT = Mean VO_2 (ml/kg/min)				29.2

Resistance (kp)	Time (min)	VO_2 (ml/kg/min)	Heart Rate (bpm)	Lactate (mmo/l)
2.5	2:00	33.1	162	
	4:00	28.9	164	
	6:00	34.0	168	
	8:00	31.9	173	6.08
	10:00	32.0	170	
	12:00	35.2	172	7.08
Rate of change (mmol/l/min)				+0.25

Note. The above data is from two 12 minute periods of exercise; the first at 2.5 kp and the second at 2.75 kp.
VO_2, oxygen consumption
HR, heart rate

ENDURANCE TRAINING PROGRAM

The subjects began their training program within a week of their first pre-training MAP-LT test. The training program was conducted with leg cycling only on a Monark bicycle ergometer for eight weeks of three sessions per week. Each training session followed a standard format (see Figure 8).

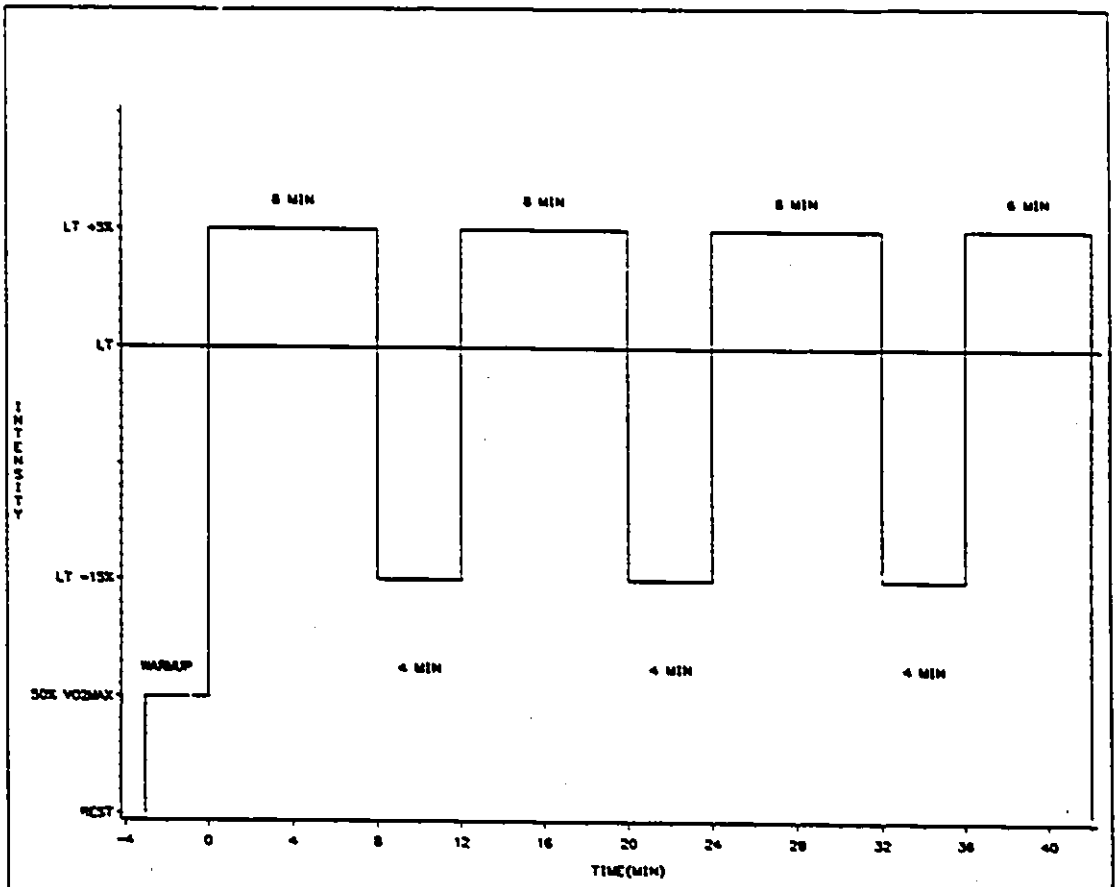

Figure 8. Diagramatic representation of the format for a training session. Subjects begin with a 3 minute warm-up and then alternate between exercise intervals above and below lactate threshold.

The subjects began each training session with a three minute warm-up at 50% VO_2max. This represented an exercise intensity below LT for all subjects and was reasonable in order to provide an adequate but not a difficult warm-up. The workload was then increased to a resistance that was 0.25 kp greater than the resistance at LT, as determined in the leg cycling LT Verification Test. The initial training intensity, therefore, corresponded to approximately LT plus 5% VO_2max and was intended to

stimulate a continual rise in BLa concentrations while not being so anaerobic or difficult as to force termination of exercise. In order to assure that training exercise was done at the appropriate intensity relative to LT, the heart rate was continually observed on PE3000 monitors worn during all training sessions, and the pedaling resistance was adjusted to stimulate average heart rates that compared with those recorded during suprathreshold intensities seen during the MAP-LT Verification Tests. As OBLA and heart rate have been observed to follow a reasonably constant relationship throughout training (Hoppeler et al., 1985; Rosler et al., 1985), it was expected that use of heart rate monitoring would achieve a progression in training intensity that matched any training adaptations. Subjects were required to maintain the same pedaling frequency of 60 rpm as employed in the pre-testing sessions. The subjects exercised at this workload for eight minutes. This was followed by a four minute recovery at the workload corresponding to approximately LT minus 15% of pre-training VO_2max, in order to facilitate both recovery and lactate removal. This range of exercise intensity was from 53-66% leg VO_2max, and falls within the range of optimal exercise intensities for BLa recovery which have been previously reported (Hermansen & Stensvold, 1972; Belcastro & Bonen, 1975). The resistance was then increased again to the training load for another eight minutes of exercise above LT. The training and recovery resistances and intensities for each subject are presented in Table 5.

A total volume of 30 minutes of training above threshold was achieved by performing four intervals of eight minutes and one interval of six minutes, each separated by four minute recovery periods at an intensity below LT. As the training program progressed, the workload was increased by a small amount, approximately 7 Watts, when the subject's heart rate in the previous session failed to reach the target heart rate prescribed for the first training session. All training sessions were done in the laboratory and supervised by the investigator.

Within a week of terminating the training program, all subjects were tested arm cranking and leg cycling using the same pre-testing protocols.

Table 5

Training and Recovery Resistances and Intensities

Subject	Initial Training Resistance (kp)	Initial Training Intensity (%VO_2max)	Final Training Resistance (kp)	Recovery Resistance (kp)	Recovery Intensity (%VO_2max)
MB	3.75	80	4.5	2.75	59
DC	3.25	73	4.0	2.0	53
DI	3.75	87	4.5	3.0	66
DM	3.5	77	4.25	2.5	56
PP	3.75	78	4.63	2.75	58
DS	3.5	77	4.13	2.5	57
CV	3.0	77	3.75	2.0	53

%VO_2max, percentage of maximal oxygen consumption

STATISTICAL ANALYSIS

A Doubly Multivariate (DM) MANOVA design was used for statistical analysis of the data at the 0.05 level of significance. When a significant omnibus test was found, univariate analyses were used to determine for which variables there were significant time and/or limb main effects and interactions. If there were significant main effects, DM MANOVA analyses were performed for that factor level (limb or time) to

determine which contrasts were significant. The 0.05 level of significance was adjusted to 0.025 in this follow-up procedure, using the Bonferonni technique in order to prevent inflation of the Type I error rate which occurs because each factor has two cells.

A dependent t-test was performed in order to analyse the difference in mean weight pre- and post-training.

RESULTS

INTRODUCTION

The main objectives of this study were to determine if the training effects from an eight week, leg cycling training program on both LT and VO_2max were specific to the training mode or if there was a general training effect. The mean data and statistical analysis are presented in the following section, and the raw data is presented in Appendix A. These observations are based on 100% attendance by all subjects at all testing and training sessions.

SUBJECTS

Seven moderately active male students volunteered to participate in this study. Mean, descriptive data of the subjects is presented in Table 6. They ranged in age from 19 to 24 years with a mean $\pm$ SE of 20.9 $\pm$ 0.07 and had a mean height of 179.1 $\pm$ 1.9 cm. All of the subjects showed a significant ($p<0.01$) pre-post training decrease in body weight from 77.9 $\pm$ 2.2 kg to 76.4 $\pm$ 2.0 kg.

Table 6

Subjects' Descriptive Data

Age (years)	Height (cm)	Weight (kg)	Leg Cycling VO_2max (l/min)	Arm Cranking Lactate Threshold (l/min)
20.9 ±0.7	179.1 ±1.9	77.9 ±2.2	3.89 ±0.11	2.82 ±0.09

N = 7; Values are means ± SE
VO_2max, maximal oxygen consumption

SUMMARY OF DESCRIPTIVE ANALYSIS

The dependent variables compared were measured during MAP-LT Tests and LT Verification Tests. The variables obtained and compared during maximal exercise in the MAP-LT tests were: absolute maximal oxygen consumption expressed in l/min (AVO_2max), relative maximal oxygen consumption expressed in ml/kg/min (RVO_2max), maximal heart rate (MaxHR), and maximal ventilation measured in l/min (MaxVE) as presented in Table 7. Information obtained during submaximal exercise from the LT Verification Test included absolute lactate threshold expressed as a rate of oxygen consumption in l/min (ALT), relative lactate threshold taken as a percentage of AVO_2max (RLT), and lactate threshold workload measured in kpm/min (LTWL) (see Table 8). Also recorded at the lactate threshold intensity were heart rate (LTHR), ventilation in l/min (LTVE), and oxygen pulse in l/beat (LTO_2P). SubHR was determined from the MAP-LT Test data but was measured at a submaximal exercise intensity to represent the submaximal heart rate at the workload associated with approximately 75% of leg AVO_2max and 75% of arm AVO_2max. Following training, SubHR was determined at the same absolute workload as had been used to determine pre-testing SubHR (see Table 10).

All of the dependent variables recorded were significantly lower ($p<0.01$) when measured during arm cranking as compared to leg cycling, both pre- and post-training, except SubHR for which there was not a significant difference between the arm and leg exercise values.

Maximal Exercise Results.

Post training adaptations of VO_2max for both modes of exercise are presented in Figures 9 and 10. Following leg cycling training, AVO_2max values increased significantly ($p<0.01$) during both leg cycling (9%) and arm cranking (5%). The significant changes ($p<0.01$) in RVO_2max values for legs and arms, (11% and 7%, respectively) were greater. Positive interactions for both variables indicates that the magnitude of increase was significantly greater ($p<0.05$) for leg-AVO_2max and RVO_2max than for arm-AVO_2max and RVO_2max. This is supported by the change in arm-AVO_2max/leg-AVO_2max ratio from 0.694 to 0.669.

There were no significant post-training changes in either mode of exercise for MaxHR and MaxVE, and this suggests that there was no learning effect and that the subjects attained true maximal effort in both testing sessions.

Table 7

Effect of Training on Maximal Exercise Results

		Absolute VO_2max (l/min)	Relative VO_2max (ml/kg/min)	Maximal Heart Rate (bpm)	Maximal Ventilation (l/min)
Leg Cycling	Pre	3.89 ± 0.11	50.1 ±1.6	195 ±2.6	135.1 ±6.9
	Post	4.23 ** ±0.10	55.4 ** ±1.2	192 ±2.4	142.4 ±7.2
Arm Cranking	Pre	2.70 ± 0.06	34.9 ±1.0	187 ±2.3	106.2 ±4.3
	Post	2.83 ** ±0.05	37.3 ** ±1.1	185 ±2.5	114.7 ±6.9

N = 7; Values are means ± SE
VO_2max, maximal oxygen consumption
Significantly different from pre-training, * $p<0.05$, ** $p<0.01$

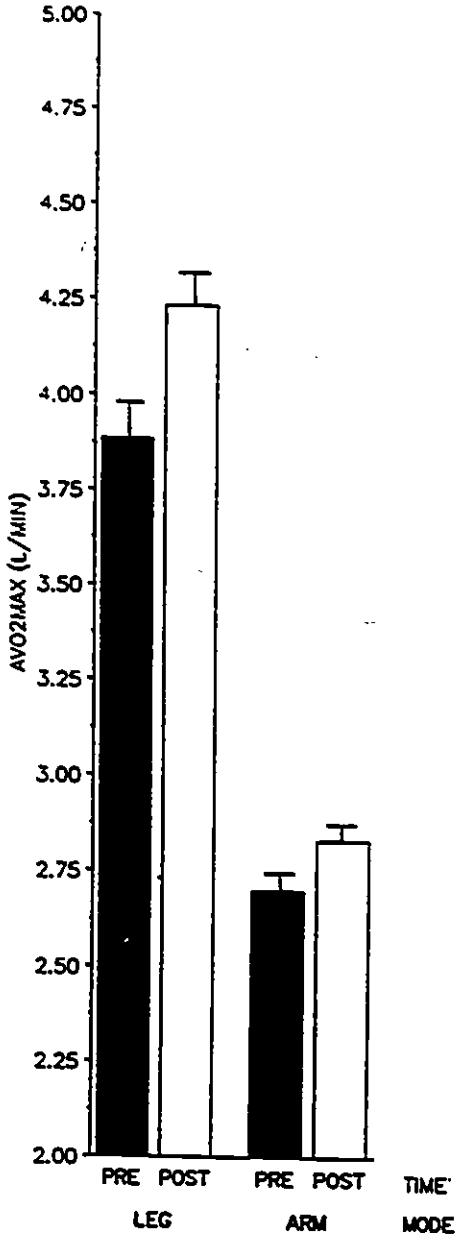

Figure 9. Comparison of the mean values and SE for absolute maximal oxygen consumption (AVO_2max) measured during leg and arm exercise pre- and post-training on 7 subjects. AVO_2max increased significantly by 11% and 7% for leg cycling and arm cranking, respectively.

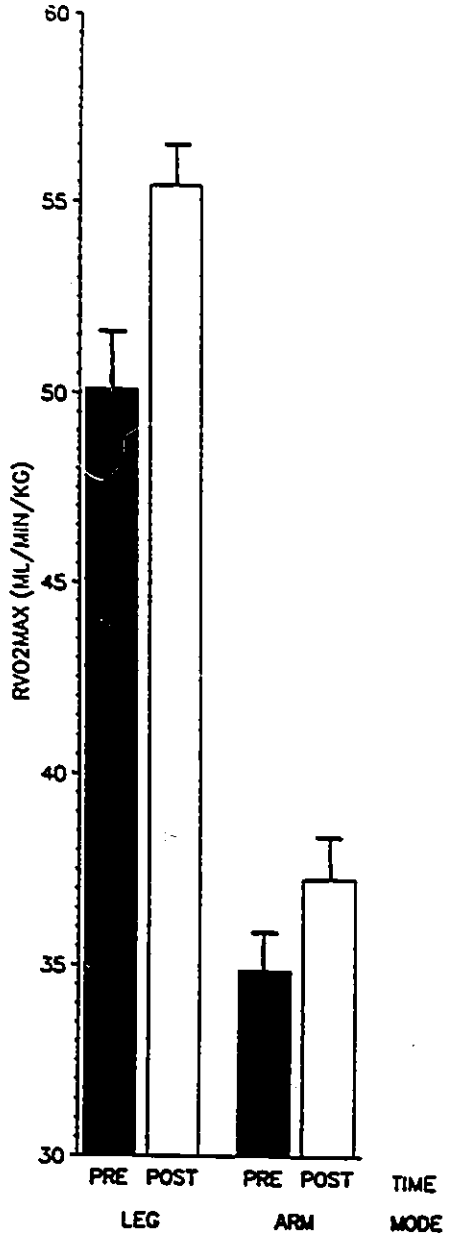

Figure 10. Comparison of the mean values and SE for relative maximal oxygen consumption (RVO_2max) measured during leg and arm exercise pre- and post-training on 7 subjects. RVO_2max increased significantly by 9% and 5% for leg cycling and arm cranking, respectively.

Submaximal Exercise Results

Post training values showed significant increases in ALT (18%, $p<0.01$) and RLT (8%, $p<0.05$) measured during leg cycling, but there was no significant difference observed during arm cranking (see Figures 11 and 12). ALT values were expressed in l/min rather than ml/kg/min in order to eliminate the effect of weight loss. Similar results were found for the workload at which the lactate threshold was measured. Leg LTWL increased by 18% following training ($p<0.01$), but arm LTWL remained unchanged. However, neither LTHR nor LTVE showed significant changes following an eight week training program for either mode of exercise.

The heart rate response during the MAP-LT exercise test was compared for a given mode of exercise at the same submaximal workload pre- and post-training. This workload represented an intensity of approximately 75% of VO_2max for the respective type of exercise. Therefore, the effect of training on heart rate was evaluated at a similar relative intensity for both arm cranking and leg cycling. This is confirmed by the lack of a significant mode main effect. There was a significant ($p<0.01$) reduction in leg SubHR by 25 bpm following endurance training and 16 bpm for arm SubHR. Although the univariate test of significance for arm SubHR was significant ($p=0.02$), the difference between pre and post arm SubHR must be considered non-significant since the multivariate analysis for arm SubHR and LTO_2P combined showed an insignificant time main effect ($p=0.066$). The lack of significance shown in multivariate analysis of the time main effect precludes consideration of the univariate results.

Table 8

Effect of Training on Lactate Threshold

		Absolute LT (l/min)	Reltative LT (%AVO_2max)	LT Workload (kpm/min)
Leg Cycling	Pre	2.82 ± 0.09	72.6 ±1.6	1182.8 ±45.8
	Post	3.34 ** ±0.05	79.2** ±1.6	1401.4 ** ±33.2
Arm Cranking	Pre	1.63 ± 0.12	60.2 ±3.8	501.4 ±33.2
	Post	1.75 ±0.06	62.1 ±2.3	514.3 ±16.6

N = 7; Values are means ± SE
LT, lactate threshold.
% $AV0_2max$, percentage of absolute maximal oxygen consumption
Significantly different from pre-training, * $p<0.05$, ** $p<0.01$

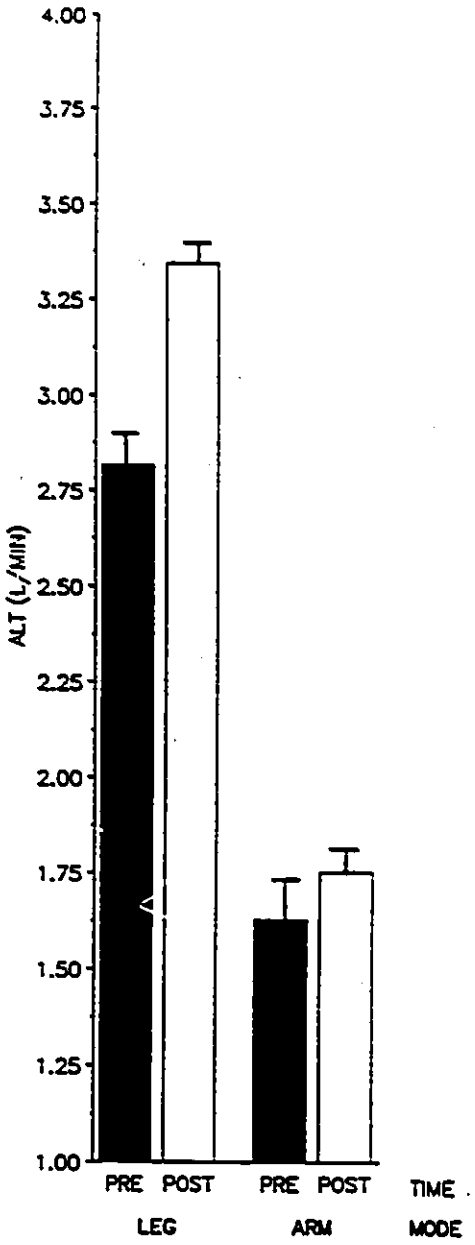

Figure 11. Comparison of the mean values and SE for absolute lactate threshold consumption (ALT) measured during leg and arm exercise pre- and post-training on 7 subjects. ALT increased significantly for leg cycling only by 18%.

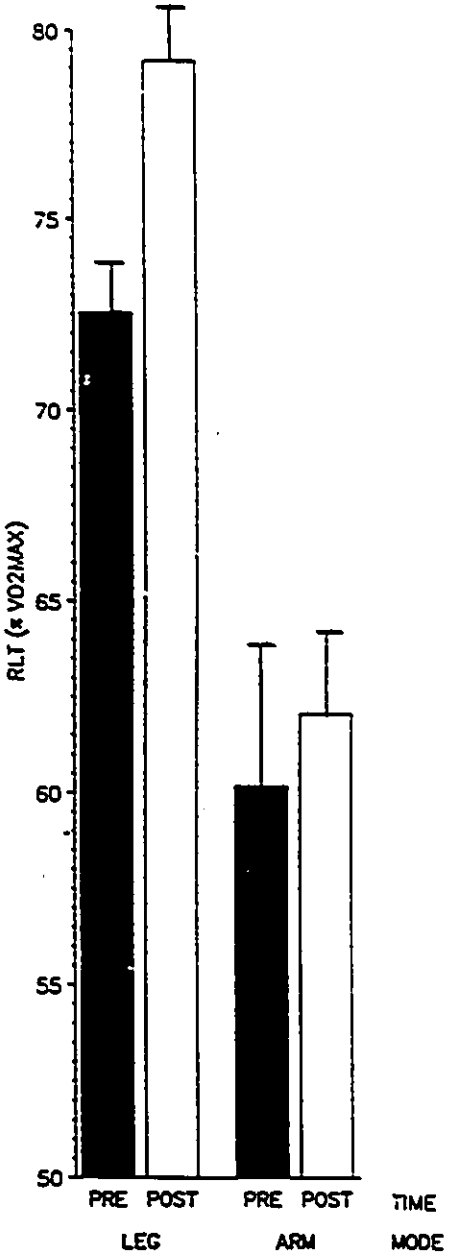

Figure 12. Comparison of the mean values and SE for relative lactate threshold consumption (RLT) measured during leg and arm exercise pre- and post-training on 7 subjects. RLT increased significantly for leg cycling only by 9%.

Table 9

Effect of Training on Variables Measured at Lactate Threshold

		LT Heart Rate (bpm)	LT Ventilation (l/min)	LT Oxygen Pulse (l/beat)
Leg Cycling	Pre	167 ±1.8	72.7 ±2.4	0.017 ±0.0006
	Post	170 ±1.8	84.5 ±2.2	0.020** ±0.0005
Arm Cranking	Pre	142 ±4.5	48.9 ±4.7	0.011 ±0.0006
	Post	146 ±5.2	52.6 ±1.2	0.012 ±0.0003

N = 7; Values are means ± SE
LT, lactate threshold
Significantly different from pre-training, * p<0.05, ** p<0.01

Table 10

Effect of Training on Submaximal Heart Rate

		SubHR (bpm)
Leg Cycling	Pre	171 ±3.2
	Post	146 ±4.4
Arm Cranking	Pre	165 ±5.9
	Post	149 ±4.9

N = 7; Values are means ± SE
SubHR, Submaximal heart rate at approximately 75% maximal oxygen consumption
Significantly different from pre-training, * p<0.05, ** p<0.01

DISCUSSION

In order to determine if training adaptations are specific to trained musculature, the present study compared the subjects' physiological responses to arm exercise and leg exercise before and after leg cycling training. These two modes of exercise were chosen to represent activity involving primarily separate muscle groups. Although it is possible that trunk muscle fibres may have been recruited during both activities, the subjects were constantly observed and instructed to minimize trunk activity during arm cranking. Muscular overlap was not considered as a problematic factor in the study involving arm and leg exercise by Lewis et al. (1980). Since Ahlborg et al. (1975) found no EMG activity of the arms during leg cycling, Lewis et al. (1980) felt that the contribution of muscles involved in arm cranking would "...represent, at most, moderate static work for stabilization.", during leg cycling. Therefore, unintended training of the musculature involved in arm cranking during the leg cycling training program is unlikely. Furthermore, Bar-Or and Zwiren (1975) proposed that any trunk muscle contribution would be the same in two testing sessions, and the contribution of trunk muscles to arm VO_2max would, therefore, be similar before and after training.

LT measured during leg cycling was responsive to the eight week, endurance training program. ALT and RLT increased 18% and 9%, respectively, and LTWI also improved 18%. This study was similar in design to that of Rosler et al. (1985) except that their training intensity was the maximum power maintainable for 30 minutes, five times per week. They found a 27% increase in the workload at OBLA, but they did not report on any changes in OBLA relative to maximal oxygen consumption. Their training program resulted in a 13% improvement in leg

$\dot{V}O_2$max, which is greater than this study's increase of 9%. However, the mean initial aerobic fitness level ($\dot{V}O_2$max = 3.66 l/min) was lower for their subjects which, along with the effectiveness of their training protocol, may have contributed to the greater magnitude of adaptations that they observed.

The present investigation verified the findings of Rosler et al. (1985) that training adaptations in BLa related variables such as OBLA or LT are specific to the training mode of exercise. They found no significant difference in the workload at OBLA for arm cranking. Similarly, this study failed to produce any significant alteration in arm ALT as a result of leg cycling training. On the other hand, arm and leg $\dot{V}O_2$max both increased following the leg cycling training program. The specificity of LT adaptations and the generality of adaptations in $\dot{V}O_2$max support the concept that these variables are to some extent dependent upon different physiological processes (Ready & Quinney, 1982). In addition, the greater enhancement in ALT compared to $\dot{V}O_2$max and the associated increase in RLT, suggest that peripheral rather than central factors have a greater potential for improvement. Andersen and Henriksson (1977a) have demonstrated this in a training study which found a 20% and 40% increase in capillary density and oxidative enzyme activity, respectively, while $\dot{V}O_2$max increased by a smaller margin of 16%.

Many peripheral adaptations which may allow increased lactate removal and/or decreased lactate production to result in an enhanced LT have demonstrated specificity to the training mode or muscles. These include decreased glycogen depletion and greater fat oxidation, enhanced enzymatic oxidation potential (Henriksson, 1977; Saltin et al., 1976), decreased delay in $\dot{V}O_2$-on response (Cerretelli et al., 1979), and increased volume density of mitochondria (Rosler et al., 1985). Klausen et al. (1974) observed a reduction in veno-arterial lactate difference for

trained musculature only. It is speculated that alterations in these factors contribute to the specificity to training that LT has demonstrated in this study. A diminished catecholamine release during submaximal exercise is found after training and could also influence BLa concentrations or LT due to a decreased stimulation of glycogenolysis (Richter, Ruderman, Gravas, & Galbo, 1982).

The increase in LT may be attributed to either decreased lactate production by the exercising muscle fibres or to increased removal by exercising and non-exercising musculature, or possibly some combination of these two effects. Since BLa concentrations are only a gross indicator of metabolic activity and a product of both lactate production and removal, it is not possible to determine the cause for an increased LT in this study. Donovan and Brooks (1983) and Favier, Constable, Chen, and Holloszy (1986) both conducted research with rats in an attempt to solve this problem. Favier et al. (1986) concluded that endurance training induced adaptations resulting in reduced lactate production by contracting muscle. Conversely, Donovan and Brooks (1983) suggested that training results in a higher metabolic clearance rate of lactate. The higher metabolic clearance rate is thought to be related to increased removal by untrained as well as trained muscle fibres (Donovan & Brooks, 1983). Attributing increased lactate removal to non-active musculature implies that, in this study, the untrained musculature used for arm cranking would have become more capable of oxidizing lactate following training. If this were true then one would also have expected to observe an increase in arm LT. However, as arm ALT did not show any significant improvement, it is not probable that an increased removal by untrained or non-exercising musculature contributed to the enhancement in leg ALT following training.

Changes in ventilatory measures are used by some investigators as indicators of changes in BLa related parameters. AT and LT are often identified by ventilatory measurements (Davis et al., 1979; Kinderman et al., 1979; Wasserman et al., 1967). There was an insignificant trend to increased LTVE for the legs which may provide some support to the use of non-invasive methods of LT identification. However, since the change in LTVE following training was insignificant, the identification of modifications in LT by comparing changes in the ventilatory response to exercise may not be a sensitive enough approach.

ALT was expressed in this study as a rate of oxygen consumption at which concentrations exhibited a specific pattern during incremental and steady state exercise. The protocol of this investigation allowed for a comparison of VO_2 measurements at the same workload during both types of exercise test. The VO_2 values were not always comparable between the incremental MAP-LT Test and the steady state LT Verification Test. Therefore, workload adjustments downward, and less frequently upward, were sometimes required in the second 12 minute steady state phase of the LT Verification Test in order to attain similar VO_2 values and observe a plateau in BLa concentrations. McLellan and Gass (1989) experienced similar difficulties in identifying "anaerobic threshold".

Ramp tests, characterized by small and short increments of work, and step tests, during which larger workload increases are made and maintained for longer intervals, have been found to differ in both the slope of VO_2 increase relative to workload and the total lag time for VO_2 adjustment (Fernandez, Mo.ler, & Butler, 1974; Swanson & Hughson, 1988). Fernandez et al. (1974) suggested that the discrepancies in steady state and progressive test VO_2 measurements appear to be smaller for better conditioned subjects. This may explain why some of the subjects exhibited comparable VO_2 values between the two types of tests and others did not.

From these observations, it is evident that one must be cautious in applying the commonly used method of identifying LT as a workload from an incremental exercise test, since the metabolic responses during incremental and steady state activity may differ. Finding the constant workload associated with the same oxygen cost as LT, as identified in incremental exercise, may avoid improper exercise prescription.

The mean increase in leg ΔVO_2max of 9% in response to the leg cycling training program is comparable to the improvements of 10% and 8% reported by Saltin et al. (1976) and Henriksson (1977), respectively. Present findings of a 5% significant enhancement in arm ΔVO_2max support the concept of a general training effect due to adaptations elicited by leg cycling training. Lewis et al. (1980) and Rosler et al. (1985) referred to this phenomenon as the transfer effect of endurance training to exercise with untrained limbs. Clausen et al. (1973), Lewis et al. (1980), and Rosler et al. (1985) all observed a transfer effect following various leg cycling training regimes. They found 13-17% increases in leg ΔVO_2max accompanied by 9-10% enhancements of arm ΔVO_2max. Although the results presented here show improvements which are smaller in magnitude, this could be attributed in part to a reduced frequency of three training sessions per week used in this investigation compared to their four to five sessions per week. Similarly, this may be a possible explanation for the lack of a transfer effect from leg cycling training to kayak ergometry in a study conducted by Ridge et al. (1976) in which subjects trained for only four weeks, four times per week. Other factors which may have influenced the results of this research compared to other studies are differences in training intensity and initial fitness level of the subjects. The subjects recruited for this investigation were moderately active, and pre-testing revealed that they had above average leg ΔVO_2max, 3.89 l/min, and RVO_2max, 50.11 ml/kg/min.

The explanation for the general or transfer effect of training is unclear. Cardiac output and sympathetic control influence skeletal muscle blood flow which is a critical factor in the limitation of arm and leg AVO_2max (Reybrouck et al., 1975). Clausen et al. (1973) and Saltin et al. (1976) proposed that the primary factor accounting for an increased VO_2max during exercise with untrained musculature is improved cardiac performance following endurance training. This or decreased total peripheral resistance (Loftin et al., 1988) allows enhanced blood flow to untrained musculature and hence higher peak oxygen consumption rates. Clausen et al. (1973) found that arm AVO_2max increased by 10% which was similar in magnitude to the increase in cardiac output (10-12%) during heavy arm exercise following leg cycling training. The similarity in these adaptations could indicate that the increase in arm AVO_2max could be attributed to the enhanced cardiac output during arm cranking exercise (Clausen et al., 1973).

Hypervolemia, a chronic expansion of blood volume due mainly to increased plasma volume, is an exercise adaptation (Green, Jones, Hughson, Painter, & Farrance, 1987; Green, Thompson, Ball, Hughson, & Sharratt, 1984) which could increase end diastolic volume and therefore also increase cardiac output. Since there is not a concomitant increase in red cell volume, hemoglobin concentration is decreased, and therefore, the blood's oxygen-carrying capacity per unit volume of blood is also reduced (Green et al., 1987). These investigators did not find any increase in VO_2max after short term training even though blood volume increased by 12.3%, suggesting that a reduced volume content of O_2 is affected by increased cardiac output. Therefore, it is unlikely that hypervolemia contributed to the increases in VO_2max or its transfer effect with training.

Since Rosler et al. (1985) found arm AVO_2max/ leg AVO_2max was only 0.74, they argued that the cardiovascular system was not the limiting factor in arm ergometry. Arm cranking, they proposed, did not fully tax the cardiovascular system. Therefore, they suggested that it was unlikely that improved cardiac output would affect arm VO_2max. Alternatively, Rosler et al. (1985) suggested that increased lactate oxidation by the trained leg muscles could be responsible for the increased VO_2max during performance with untrained arm musculature. In this case, one would expect to see lower BLa concentrations at maximal and perhaps submaximal workloads during arm exercise. However, their findings do not support this hypothesis. Hence, central cardiovascular adaptations may have been the primary contributing factor to the general training effect which resulted in improvements in VO_2max during exercise involving untrained musculature.

The results of this study also demonstrate a trend toward reduced SubHR during exercise with trained as well as with untrained muscles. This is supported by investigations reported by Clausen et al. (1973) and Lewis et al. (1980). They demonstrated similar training induced reductions in heart rate at a given workload during both arm and leg exercise. However, there are also instances in which training was not shown to influence submaximal heart rate in the non-trained mode of exercise. Saltin et al. (1976) found that training with one leg resulted in only minor reductions in heart rate when cycling with the other untrained leg. Similarly, Clausen et al. (1973) also demonstrated that subjects trained by arm cranking rather than leg cycling had only small decrements in heart rate measured during leg cycling after training, while they had significant changes when arm cranking.

One possible explanation for these contradictory findings may be related to the size and degree of training of the muscles and the influence this would have on

determining whether central or peripheral adaptations are more predominant (Clausen et al., 1973). Arm cranking or one-leg cycling may not involve a large enough muscle mass to stress the central cardiovascular system such that large and transferable adaptations result. Peripheral adaptations may have a greater influence on the heart rate response to exercise With this type of training. Therefore, the resulting reductions in heart rate are specific to exercise with the trained muscles. The peripheral factors which exhibit influence over cardiac function include changes in ion concentration (Tibes & Groth, 1977). Adaptations influencing ion concentrations may result in a decreased firing rate of muscular afferent nerves which would have an influence on the adrenergic control of heart rate (Winder, Hagber, Hickson, Ehsanis, & McLane, 1978). The central adaptations which may contribute to a transfer effect of heart rate reduction include increased stroke volume, due to changes in the contractility of the myocardium and increased venous return, and decreased sympathetic activation of the heart (Lehmann & Keul, 1986; Winder et al., 1978). The findings of this study seem to reflect that both central and peripheral adaptations have influenced the changes in SubHR. There was a considerable although non-significant, reduction in arm SubHR of 16 bpm suggesting a transfer effect. However, the 25 bpm decrease observed for leg SubHR is larger and supports the specificity of training concept.

The heart rate response to exercise at a given absolute VO_2 is typically higher for arm cranking exercise relative to leg cycling exercise (Vokac et al., 1975). Therefore, it follows that in order to elicit similar heart rates between the two modes during submaximal exercise, arm cranking would need to be performed at a lower rate of oxygen consumption or workload. Given that arm ALT occurred at a lower oxygen consumption than leg ALT before and after training, it might have been expected that the LTHR would be similar for upper and lower body exercise. The results, however, indicated that arm LTHR was significantly lower than leg LTHR before and after the

eight weeks of training. This suggests that there are indeed large differences in the endurance capacity for submaximal activity between the upper and lower body in individuals untrained for either activity.

With the increasing use of heart rate to monitor exercise intensity outside of the laboratory, it is important to recognize the specificity of LTHR to the mode of activity as demonstrated here between arm cranking and leg cycling. However, it is possible that LTHR would not differ greatly when compared between two modes of exercise involving similar musculature and activity patterns. Further studies are needed to clarify this matter. The reduced arm LTHR is also of significance for amputees and paraplegics among whom physical activity has become increasingly prevalent. This population often employs upper body training such as arm cranking, and since the exercise intensity prescribed may exceed arm LT, common exercise prescription formulas based on heart rate may not be practical.

Training did not have a significant influence on LTHR for either exercise mode. Perhaps, this parameter is not sensitive to endurance training, or more likely, it is not responsive to a training period of such a relatively short duration in comparison to the training life of an athlete. Similarly, Hoppeler et al. (1985) and Rosler et al. (1985) found no alteration in the heart rate at OBLA for either upper or lower body exercise following 6-8 weeks of leg cycling training.

Heart rate and VO_2 data collected by Vokac, Bell, Bautz-Holter, and Rodahl (1975) showed that oxygen pulse was lower during submaximal arm cranking exercise than during submaximal leg cycling exercise. This may reflect a reduced cardiac efficiency for arm cranking. In this study, oxygen pulse (LTO_2P) was calculated and compared at the LT intensity for arm cranking and leg cycling. The resulting significant difference between arm and leg LTO_2P may indicate differences in cardiac

efficiency even at the same relative intensity. Since the heart rate at threshold was not significantly altered by training, the higher leg LTO_2P following training may be primarily attributed to the increased VO_2 measured at LT and the associated metabolic adaptations. The lack of a significant difference between pre and post arm LTO_2P supports the specificity of metabolic adaptations measured at LT.

Numerous studies have found differences in the body's physiological responses to arm cranking exercise as compared to leg cycling exercise. These findings include the identification of a lower efficiency for arm cranking compared to leg cycling exercise, and consequently that there is a greater oxygen cost and higher BLa concentration elicited by arm cranking at the same submaximal power output. At maximal exertion, VO_2 and workload are lower for arm cranking and are generally accompanied by a reduced heart rate response and BLa concentration (Franklin, 1985; Sawka, 1986).

The findings of this study support that the metabolic stresses and capacities of upper and lower body exercise are different. All of the submaximal and maximal exercise variables measured, except SubHR, were significantly lower ($p<0.001$) during arm cranking than during leg cycling exercise both pre and post training. SubHR was measured at similar intensities relative to VO_2max for the two exercise modes, and no significant difference was found between the arm and leg measurements. This last observation was not expected and is surprising given that MaxHR was lower for arm cranking than leg cycling.

The training effect is said to be maximal when the subjects are initially untrained (Bouchard et al., 1980). Similarly, in order to study the effect of training on exercise involving untrained musculature it is desirable to recruit subjects who are untrained for both modes of activity to be used in the study. Arm cranking VO_2max is generally 65-75% of leg cycling VO_2max for sedentary individuals (Bar-Or & Zwiren, 1975;

Franklin, 1985; Sawka, 1986). Upper body training can significantly reduce this difference to approximately 90% of peak leg cycling VO_2max (Sawka, 1986). This difference is probably also dependent upon the relative distribution of muscle mass in the upper and lower body. Before cycle ergometer training, Lewis et al. (1980) and Rosler et al. (1985) reported arm AVO_2max/leg AVO_2max ratios of 0.64 and 0.74, respectively. The observed ratio in this study was 0.69 which suggests that the subjects recruited for this study were similarly unconditioned for arm exercise. During maximal exercise testing, the peak heart rate achieved for arm cranking before training was significantly lower than the leg cycling peak heart rate by 8 bpm, and it was 7 bpm lower after the exercise program. Previously reported differences range from 3 to 23 bpm with a mean of 11 bpm (Franklin, 1985). Smaller skeletal muscle mass, muscle fibre composition, differences in muscle fibre recruitment patterns, as well as reduced blood perfusion may place limitations on the maximal effort during upper body exercise (Reybrouck et al., 1975; Sawka, 1986). These factors may partially account for the reduced peak oxygen uptake values and maximal heart rates observed during upper body exercise relative to lower body exercise.

In conclusion, the method of leg cycling training employed in the present study was successful in stimulating significant physiological adaptations which may be attributed to either central or peripheral processes, or both. The major findings are as follows:

1. Leg cycling training resulted in an 18% significant increase in leg LT, expressed as an oxygen consumption (ALT), and a corresponding 18% rise in leg LTWL. The enhancement of leg RLT suggests that lactate related variables may be more sensitive indicators of training adaptations than VO_2max, and this supports the hypothesis that different adaptive processes govern the physiological responses of LT and VO_2max to training. This is also supported by the specific effect training had on LT, while

training resulted in a general adaptation or a transfer effect on VO_2max. Therefore, this study supports the idea that training induced increases in LT are specific to the trained mode of exercise when compared to activity involving separate musculature.

2. As previously reported, it is possible to elicit training induced increases in VO_2max measured in an exercise mode for which the subject is untrained. This transfer effect suggests that the general nature of the adaptations are related to elements which are common to separate muscle groups such as the central portion of the cardiovascular system. However, since both ΔVO_2max and RVO_2max increased by a significantly greater amount for leg cycling than for arm cranking, the concept of specificity of training is still pertinent to some degree. Therefore, peripheral adaptations would also seem to be a determining factor in the effect of training on VO_2max.

3. Heart rate measured at submaximal exercise intensities may be significantly reduced through endurance training during exercise in the trained mode, and a trend exists for a smaller decrement in submaximal heart rate during activity involving untrained musculature.

4. Variables such as heart rate, ventilation, and oxygen pulse measured at the LT intensity differed in their responses to training. The heart rate at LT was specific to the exercise mode, and the lack of a training effect after eight weeks suggests that it is a good variable for monitoring training intensity for short term periods. The effect of long term training on LTHR should be investigated. Since ventilation at LT (LTVE) was not significantly altered by training for either mode of exercise, it was found not to be a sensitive enough indicator by which to measure changes in LT. Oxygen pulse at LT (LTO_2P) exhibited training induced increases only for the trained mode of exercise. This supports the hypothesis that there is an influence of local or peripheral factors on adaptations related to the LT intensity.

REFERENCES

Ahlborg, G., Hagenfeldt, L., & Wahren, J. (1975). Substrate utilization by the inactive leg during one-leg or arm exercise. *Journal of Applied Physiology, 39* (5), 718-723.

Andersen, P., & Henriksson, J. (1977a). Capillary supply of quadriceps femoris muscle of man: Adaptive response to exercise. *Journal of Physiology, 270*, 677-690.

Andersen, P., & Henriksson, J. (1977b). Training induced changes in subgroups of human typeII skeletal muscle fibres. *Acta Physiologica Scandinavica, 99*, 123-125.

Astrand, P. O., & Rodahl, K. (1986). *Textbook of work physiology*. New York: McGraw-Hill.

Aunola, S., & Rusko, H. (1986). Aerobic and anaerobic thresholds determined from venous lactate or from ventilation and gas exchange in relation to muscle fibre composition. *International Journal of Sports Medicine, 7*, 161-166.

Baldwin, K., Klinkerfuss, G., Terjung, R., Mole, P., & Holloszy, J. (1972). Respiratory capacity of white, red and intermediate muscle: Adaptive response to exercise. *American Journal of Physiology, 222*, 373-378.

Bar-Or, O., & Zwiren, D. (1975). Maximal oxygen consumption test during arm exercise - reliability and validity. *Journal of Applied Physiology, 38* (3), 424-426.

Belcastro, A. N., & Bonen, A. (1975). Lactic acid removal rates during controlled and uncontrolled recovery exercise. *Journal of Applied Physiology, 39* (6), 932-936.

Bouchard, C., Boulay, M., Thibault, M. C., Carrier, R., & Dulac, S. (1980). Training of submaximal working capacity: frequency, intensity, duration, and their interactions. *Journal of Sports Medicine, 20,* 29-40.

Brooks, G. A. (1985). Anaerobic threshold: Review of the concept and directions for future research. *Medicine and Science in Sports and Exercise, 17* (1), 22-31.

Brooks, G. A. (1986). The lactate shuttle during exercise and recovery. *Medicine and Science in Sports and Exercise, 18* (3), 360-368.

Campbell, M. E., Hughson, R. L., & Green, H. L. (1989). Continuous increase in blood lactate concentration during different ramp exercise protocols. *Journal of Applied Physiology, 66* (3), 1104-1107.

Casaburi, R., Storer, T. W., Ben Dov, I., & Wasserman, K. (1987). Effect of endurance training on possible determinants of VO_2 during heavy exercise. *Journal of Applied Physiology, 62* (1), 199-207.

Cerretelli, P., Pendergast, D., Paganelli, W. C., & Rennie, D. W. (1979). Effects of specific muscle training on VO_2 on-response and early blood lactate. *Journal of Applied Physiology, 47* (4), 761-769.

Chirtel, S. J., Barbee, R.W., & Stainsby, W. N. (1984). Net O_2, CO_2, lactate and acid exchange by muscle during progressive working contractions. *Journal of Applied Physiology, 56* (1), 161-165.

Clausen, J. P., Klausen, K., Rasmussen, B., & Trap-Jensen, J. (1973). Central and peripheral circulatory changes after training of the arms or legs. *American Journal of Physiology, 225,* 675-682.

Connett, R. J., Gayeski, T. E. J., & Honig, C. R. (1984). Lactate accumulation in fully aerobic, working, dog gracilis muscle. *American Journal of Physiology, 246,* H120-H128.

Coyle, E. F., Coggan, A. R., Hopper, M. K., & Walter, T. J. (1988). Determinants of endurance in well trained cyclists. *Journal of Applied Physiology, 64*, 2622-2630.

Cresciteli, F., & Taylor, C. (1944). The lactate response to exercise and its relationship to physical fitness. *American Journal of Physiology, 141*, 630-640.

Davis, J. A. (1985a). Anaerobic threshold: Review of the concept and directions for future research. *Medicine and Science in Sports and Exercise, 17* (1), 6-18.

Davis, J. A. (1985b). Response to Brooks' manuscript. *Medicine and Science in Sports and Exercise, 17*, 32-34.

Davis, J. A., Frank, M. H., Whipp, B. J., & Wasserman, K. (1979). Anaerobic threshold alterations caused by endurance training in middle - aged men. *Journal of Applied Physiology, 46*, 1039-1046.

Davis, J. A., & Vodak, P. (1976). Anaerobic threshold and maximal aerobic power for three modes of exercise. *Journal of Applied Physiology, 41*, 544-550.

Denis, C., Dormois, D., Castells, J., Bonnefay, R., Padilla, S., Geyssant, A., & Lacour, J. R. (1988) Comparison of incremental and steady state tests of endurance training. *European Journal of Applied Physiology, 57*, 474-81.

Donovan, C. M., & Brooks, G. A. (1983). Endurance training affects lactate clearance not lactate production. *American Journal of Physiology, 244*, E83-E92.

Droghetti, P., Borsetto, C., Casoni, I., Cellini, M., Ferrari, M., Paolini, A. R., Ziglio, P. G., & Conconi, F. (1985). Noninvasive determination of the anaerobic threshold in canoeing, cross-country skiing, cycling, roller and iceskating, rowing and walking. *European Journal of Applied Physiology, 53*, 299-303.

Dwyer, J., & Bybee, R.(1983). Heart rate indices of the anaerobic threshold. *Medicine and Science in Sports and Exercise, 15* (1), 72-76.

Edgerton, V. R., Smith, J. L., & Simpson, D. R. (1975). Muscle fibre type populations of human leg muscles. *Histochemical Journal, 7*, 259-266.

Essen, B., Jansson, E., Henriksson, J., Taylor, A., & Saltin, B. (1975) Metabolic characteristics of fibre types in human skeletal muscle. *Acta Physiologica Scandinavica, 95*, 153-165.

Farrell, P. A., Wilmore, J. H., Coyle, E. F., Billing, J. E., & Costill, D. L. (1979). Plasma lactate accumulation and distance running performance. *Medicine in Science and Sports, 11* (4), 338-344.

Favier, R. J., Constable, S. H., Chen, M., & Holloszy, J. O. (1986). Endurance exercise reduces lactate production. *Journal of Applied Physiology, 61* (3), 885-889.

Fernandez, E. A., Mohler, J. G., & Butler, J. P. (1974). Comparison of oxygen consumption measured at steady state and progressive rates of work. *Journal of Applied Physiology, 37* (6), 982-987.

Franklin, B. A. (1985). Exercise testing, training and arm ergometry. *Sports Medicine, 2*, 100-119.

Gaesser, G. A., & Poole, D. C. (1986). Lactate and ventilatory thresholds: disparity in time course of adaptations to training. *Journal of Applied Physiology, 61* (3), 999-1004.

Galbo, H., Holst, J. J., & Christensen, N. J. (1975). Glucagon and plasma catecholamine responses to graded and prolonged exercise in man. *Journal of Applied Physiology, 38*, 70-76.

Green, H. J., Jones, L. L., Hughson, R. L., Painter, D. C., & Farrance, B. W. (1987). Training-induced hypervolemia: lack of an effect on oxygen utilization during exercise. *Medicine and Science in Sports and Exercise, 19* (3), 202-206.

Green, H. J., Thompson, J. A., Ball, M. E., Hughson, R. L., Hughson, M. E., & Sharratt, M. T. (1984). Alterations in blood volume following short-term supramaximal exercise. *Journal of Applied Physiology, 56* (1), 145-149.

Gollnick, P. D., Armstrong, R. B., Saubert, C. W., Piehl, K., & Saltin, B. (1972). Enzyme activity and fibre composition in skeletal muscle of untrained and trained men. *Journal of Applied Physiology, 33*, 312-319.

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 Sciences Reviews, 1*, 1-43.

Gollnick, P. D., Piehl, K., & Sahlin, B. (1974). Selective glycogen depletion patterns in human muscle fibres after exercise of varying intensity and at varying pedal rates. *Journal of Physiology, 241*, 45-47.

Hagberg, J. M. & Coyle, E. F. (1983). Physiological determinants of endurance performance in competitive racewalkers. *Medicine and Science in Sports and Exercise, 15* (4), 287-289.

Henriksson, J. (1977). Training-induced adaptation of skeletal muscle and metabolism during submaximal exercise. *Journal of Physiology, 270*, 661-675.

Henritze, J., Weltman, A., Schurrer, R. L., & Barlow, K. (1985). Effects of training at and above the lactate threshold on the lactate threshold and maximal oxygen uptake. *European Journal of Applied Physiology, 54*, 84-88.

Hermansen, L., & Stensvold, I. (1972). Production and removal of lactate during exercise in man. *Acta Physiologica Scandinavica, 86*, 191-201.

Hickson, R. C., Rennie, M. J., Conlee, R. K., Winder, W. W., & Holloszy, J. O. (1977). Effect of increased plasma fatty acids on glycogen utilization and endurance. *Journal of Applied Physiology, 43*, 829-833.

Hill, A. V., Long, C. N., & Lupton, H. (1924). Muscular exercise, lactic acid and the supply and utilization of oxygen. *Proceedings of the Royal Society of London, 96, 97, 97,* 438, 84, 155.

Hollmann, W. (1985). Historical remarks on the development of the aerobic-anaerobic transition up to 1966. *International Journal of Sports Medicine, 6,* 109-116.

Hollmann, W., Rost, R., Liessen, H., Dufaux, B., Heck, H., & Mader, A. (1981). Assessment of different forms of physical activity with respect to preventive and rehabilitative cardiology. *International Journal of Sports Medicine, 2,* 67-80.

Holloszy, J. O., & Coyle, E. F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology, 56* (4), 831-838.

Holmgren, A., & Strom, G. (1959). Blood lactate concentration in relation to absolute and relative work load in normal men, and in mitral stenosis, atrialseptal defect and vasoregulatory asthenia. *Acta Med. Scand., 3,* 163.

Hoppeler, H., Howald, H., Conley, K., Lindstedt, S. L., Claassen, H., Vock, P., & Weibel, E. R. (1985). Endurance training in humans: Aerobic capacity and structure of skeletal muscle. *Journal of Applied Physiology, 59* (2), 320-327.

Hughes, R. L., Clode, M., Edwards, R. H. T., Goodwin, T. J., & Jones, N. L. (1968). Effect of inspired O_2 on cardiopulmonary and metabolic responses to exercise. *Journal of Applied Physiology, 24,* 336-347.

Hughes, E. F., Turner, S. C., & Brooks, G. A. (1982). Effects of glycogen depletion and pedaling speed on "anaerobic threshold". *Journal of Applied Physiology, 52* (6), 1598-1607.

Hughson, R. L., Weisiger, K. H., & Swanson, G. D. (1987). Blood lactate concentration increases as a continuous function in progressive exercise. *Journal of Applied Physiology, 62* (5), 1975-1981.

Hultman, E., & Sahlin, K. (1980). Acid-base balance during exercise. *Exercise and Sport Science Reviews, 8,* 41-128.

Ingjer, F. (1979). Effects of endurance training on muscle fibre ATPase activity, capillary supply and mitochondrial content in man. *Journal of Physiology, 294,* 419-432.

Ivy, J. L., Withers, R. T., Van Handel, P. J., Elger, D. H., & Costil, D. L. (1980). Muscle-respiratory capacity and fiber type as determinants of the lactate threshold. *Journal of Applied Physiology, 48,* 523-527.

Jacobs, I. (1986). Blood lactate implications for training and sport performances. *Sports Medicine, 3,* 10-25.

Jacobs, I., Schele, R., & Sjodin, B. (1985). Blood lactate vs. exhaustive exercise to evaluate aerobic fitness. *Journal of Applied Physiology, 54,* 151-155.

Jacobs, I. & Sjodin, B. (1983). Muscle enzymes and VO_2Max on blood lactate accumulation during treadmill and cycle exercise. *Canadian Journal of Applied Sport Sciences, 8,* 218.

Jacobs, I., &Sjodin, B. (1985). Relationship of ergometer specific VO_2Max and muscle enzymes to blood lactate during submaximal exercise. *British Journal of Sports Medicine, 19* 77-80.

Karlsson, J., Nordesjo, L. O., Jorfeldt, L., & Saltin, B. (1972). Muscle lactate, ATP, and CP levels during exercise after physical training in man. *Journal of Applied Physiology, 33* (2), 199-203.

Karlsson, J., Nordesjo, L. O., & Saltin, B. (1974). Muscle glycogen utilization during exercise after physical training. *Acta Physiologica Scandinavica, 90,* 210-217.

Katz, A., & Sahlin, K. (1987). Effect of decreased oxygen availability on NADH and lactate content in human skeletal muscle during exercise. *Acta Physiologica Scandinavica, 131,* 119-127.

Katz, A., & Sahlin, K. (1988). Regulation of lactic acid production during exercise. *Journal of Applied Physiology, 65* (2), 509-518.

Kinderman, W., Simon, G., & Keul, J. (1979). The significance of the aerobic-anaerobic transition for the determination of work load intensities during exercise training. *European Journal of Applied Physiology, 42*, 25-34.

Klausen, K., Andersen, L. B., & Pelle, I. (1981). Adaptive changes in work capacity, skeletal muscle capillarization and enzyme levels during training and detraining. *Acta Physiologica Scandinavica, 113*, 9-16.

Klausen, K., Rasmussen, B., Clausen, J. P., & Trap-Jensen J. (1974). Blood lactate from exercising extremities before and after arm or leg training. *American Journal of Physiology, 227* (1), 67-72.

Klausen, K., Secher, N. H., Clausen, J. P., Hartling, O., & Trap-Jensen J. (1982). Central and regional circulatory adaptations to one-leg training. *Journal of Applied Physiology, 52* (41), 976-983.

Kontron Medical. (1981). *Operating Manual for Lactate Analyzer 640* Switzerland: Kontron Medical.

Lehman, M., & Keul, J. (1986). Free plasma catecholamines, heart rates, lactate levels and oxygen uptake in competition weight lifters, cyclists and untrained control subjects. *International Journal of Sports Medicine, 7*, 18-21.

Lehninger, A. L. (1982). *Principles of biochemistry*. New York: Worth Publishers.

Lewis, S., Thompson, P., Areskog, N. H., Vodak, P., Marconyak, M., DeBusk, R., Mellen, S., & Haskell, W. (1980). Transfer effects of endurance training to exercise with untrained limbs. *European Journal of Applied Physiology, 44*, 25-34.

Linnarsson, D., Karlsson, J., Fagraeus, L. & Saltin, B. (1974). Muscle metabolites and oxygen deficit with exercise in hypoxia and hyperoxia. *Journal of Applied Physiology, 36*, 339-402.

Loftin, M., Boileau, R. A., Massey, B. H., & Lohman, T. G. (1988). Effect of arm training on central and peripheral circulatory function. *Medicine and Science in Sports and Exercise, 20* (2), 136-141.

Mainwood, G. W., & Renaud, J. M. (1985). The effect of acid-base balance on fatigue of skeletal muscle. *Canadian Journal of Physiology and Pharmacology, 63*, 403-416.

Mainwood, G. W., & Worsley-Brown, P. (1975). The effects of extracellular pH and buffer concentration on the efflux of lactate from frog sartorius muscle. *Journal of Physiology, 250*, 1-22.

McLellan, T. M. & Gass, G. C. (1989). Metabolic and cardiorespiratory responses relative to the anaerobic threshold. *Medicine and Science in Sports and Exercise, 21* (2), 191-198.

McGrail, J. C., Bonen, A. & Belcastro, A. N. (1977). Lactic acid metabolism during exercise. *Research Papers in Physical Education, 3* (3), 4-9.

McLellan, T. M. (1985). Ventilatory and plasma lactate response with different exercise protocols: a comparison of methods. *International Journal of Sports Medicine, 6*, 30-35.

Newman, F. V., Dill, D. B., Edwards, H. T., & Webster, F. A. (1937). The rate of lactic acid removal in exercise. *American Journal of Physiology, 118*, 457-462.

Pendergast, D., Ceretelli, P., & Rennie, W. (1979). Aerobic and anaerobic metabolism in arm exercise. *Journal of Applied Physiology, 47* (4), 754-760.

Pette, D. (1984). Activity-induced fast to slow transitions in mammalian muscle. *Medicine and Science in Sports and Exercise, 6* (6), 517-528.

Poole, D. C., & Gaesser, G. A. (1985). Response of ventilatory and lactate thresholds to continuous and interval training. *Journal of Applied Physiology, 58*, 1115-1121.

Ready, A. E., & Quinney, H. A. (1982). Alterations in anaerobic threshold as the result of endurance training and detraining. *Medicine and Science in Sports and Exercise, 14* (4), 292-296.

Reybrouck, T., Heigenhauser G. F., & Faulkner, J. A. (1975). Limitations to maximum oxygen uptake in arm, leg, and combined arm-leg ergometry. *Journal of Applied Physiology, 38*, 774-779.

Ribeiro, J. P., Hughes, V. H., Fielding, R. A., Holden, W., Evans, W., & Knuttgen, H. G. (1986). Metabolic and ventilatory responses to steady state exercise relative to lactate thresholds. *European Journal of Applied Physiology, 55*, 215-221.

Richter, E., Ruderman, H., Gravas, H., & Galbo, H. (1982). Muscle glycogenolysis during exercise and control by epinephrine and contractions. *American Journal of Physiology, 5*, E25-E32.

Ridge, B. R., Pyke, F. S., & Roberts, A. D. (1976). Responses to kayak ergometer performance after kayak and bicycle ergometer training. *Medicine and Science in Sports, 8*, 18-22.

Robinson, S., & Harmon, P. M. (1941). The lactic acid mechanism and certain properties of the blood in relation to training. *American Journal of Physiology, 132*, 757.

Rosler, K., Hoppeler, H., Conley, K. E., Claassen, H., Gehr, P., & Howald, H. (1985). Transfer effects in endurance exercise. *European Journal of Applied Physiology, 54*, 355-362.

Rowell, L. B., Kraning, K. K., Evens, T. O., Kennedy, J. W., Blackman, J. F., & Kasumi, F. (1966). Splanchnic removal of lactate and pyruvate during prolonged exercise in man. *Journal of Applied Physiology, 21*, 1773-1783.

Sahlin, K. (1983). Effect of acidosis on energy metabolism and force generation in skeletal muscle. In H. G. Knuttgen,G. A. Vogel, & G. Poortmans (Eds.), *Biochemistry of Exercise* (pp. 151-160). Illinois: Human Kinetic Publishers.

Sahlin, K., & Henriksson, J. (1984). Buffer capacity and lactate accumulation in skeletal muscle of trained and untrained men. *Acta Physiologica Scandinavica, 122*, 331-339.

Saltin, B., Hartley, H. I., Kilbom, A., & Astrand, I. (1969). Physical training in sedentary middle-aged and older men. Oxygen uptake, heart rate, and blood lactate concentrations at submaximal and maximal exercise. *Scandinavian Journal of Clinical Laboratory Investigations, 24*, 323-334.

Saltin, B., Nazar, K., Costill, D. L., Stein, E., Jansson, E., Essen, B., & Gollnick, P. D. (1976). The nature of the training response: Peripheral and central adaptations to one-legged exercise. *Acta Physiologica Scandinavica, 96*, 289-305.

Sawka, M. N. (1986). Physiology of upper body exercise. *Exercise and Sports Sciences Reviews, 14*, 175-210.

Schantz, P. G. (1986). Plasticity of human skeletal muscle with special reference to effects of physical training on enzyme levels of the NADH shuttles and phenotypic expression of slow and fast myofibrillar proteins. *Acta Physiologica Scandinavica*, (Suppl. 558), 1-62.

Schantz, P. G., Sjoberg, B., & Svendenhag, J. (1986). Malate-aspartate and alpha-glycerophosphate shuttle enzyme levels in human skeletal muscle: methodological considerations and effect of endurance training. *Acta Physiologica Scandinavica, 128*, 397-407.

Scheen, A., Juchnes, J., & Cession-Fossion, A. (1981). Critical analysis of the "anaerobic threshold" during exercise at constant workloads. *European Journal of Applied Physiology, 46*, 367-377.

Simon, G., Berg, A., Dickhugh, H. H., Simon-Alt, A., & Keul, J. (1981). Bestimmung der anaeroben Schwelle in Abhaengigkeit vom Alter Und von der Leistungsfaihigkeit. *Dtsch. Z. fuer Sportmedizin, 1*, 7-14.

Simon, J., Young, J. L., Gutin, B., Blood, D. K., & Case, R. B. (1983). Lactate accumulation relative to the anaerobic and respiratory compensation threshold. *Journal of Applied Physiology, 54*, 13-17.

Simoneau, J. A., Lortie, G., Boulay, M .R., Hamel, P., Lesage, R., Theriault, G., & Bouchard, C. (1983). Human muscle enzyme alterations after aerobic and anaerobic training. *Canadian Journal of Applied Sport Sciences, 8*, 218.

Sjodin, B., & Jacobs, I. (1981). Onset of blood lactate accumulation and marathon running performance. *International Journal of Sports Medicine, 2*, 23-26.

Sjodin, B., Jacobs, I., & Svedenhag, J. (1982). Changes in onset of blood lactate accumulation (OBLA) and muscle enzymes after training at OBLA. *European Journal of Applied Physiology, 43*, 45-47.

Sjodin, B., Thorstensson, A., & Karlsson, J. (1976). Effect of physical training on LDH activity and LDH isozyme pattern in human skeletal muscle. *Acta Physiologica Scandinavica, 97*, 150-157.

Skinner, J. S., & McLellan, T. H. (1980). The transition from aerobic to anaerobic metabolism. *Research Quarterly for Exercise and Sport, 51* (1), 234-248.

Stainsby, W. N., Sumners, C., & Eitzman, P. D. (1985). Effects of catecholamines on lactic acid output during progressive working contractions. *Journal of Applied Physiology, 59*, 1809-1814.

Stegmann, H., & Kindermann, W. (1982). Comparison of prolonged exercise tests at the Individual Anaerobic Threshold and the Fixed Anaerobic Threshold of 4 mmol/l lactate. *International Journal of Sports Medicine, 3*, 105-110.

Stegmann, H., & Kindermann, W., & Schnabel, A. (1981). Lactate kinetics and individual anaerobic threshold. *International Journal of Sports Medicine, 2* (3), 160-165.

Steinacker, J. M., Marx, T. R., Marx, U., & Lormes, W. (1986). Oxygen consumption and metabolic strain in rowing ergometry exercise. *European Journal of Applied Physiology, 55*, 240-247.

Stromme, S. B., Inger, F., & Meen, H. D. (1977). Assessment of maximal aerobic power in specifically trained athletes. *Journal of Applied Physiology, 42*, 833-837.

Swanson, G. D., & Richard, L. H. (1988). On the modeling and interpretation of oxygen uptake kinetics from ramp work rate tests. *Journal of Applied Physiology, 65* (6), 2453-2458.

Terjung, R. L., Mathien, G. M., Erney, T. P., & Ogilvie, R. W. (1988). Peripheral adaptations to low blood flow in muscle during exercise. *American Journal of Cardiology, 62*, 15E-19E.

Tesch, P. A., & Lindeberg, S. (1984). Blood lactate accumulation during arm exercise in world class kayak paddlers and strength trained athletes. *European Journal of Applied Physiology, 52*, 441-445.

Tesch, P. A., & Wright, J. E. (1983). Recovery from short term exercise: Its relationship to capillary supply and blood lactate concentration. *European Journal of Applied Physiology,52*, 98-103.

Tibes, U., & Groth, H. H. (1977). Interaction of K^+, osmolality (OSM), orthophosphate (Pi), lactic acid (Lac), and adrenaline at c-fibre receptors in skeletal muscle. *Plugers Arch., 368* (Suppl. R40).

Vander, A. J., Sherman, J. H., & Luciano, D. S. (1985). *Human Physiology. The Mechanisms of Body Function.* New york: McGraw-Hill.

Van Harn, L., & Brooks, G.A. (1985). Plasma catecholamines during progressive exercise in man. *International Journal of Sports Medicine, 6*, 238.

Vokac, Z., Bell, H., Bautz-Holter, E., & Rodahl, K. (1975). Oxygen uptake/heart rate relationship in leg and arm exercise sitting and standing. *Journal of Applied Physiology, 39* (1), 54-59.

Walsh, M. L., & Banister, E. W. (1988). Possible mechanisms of the anaerobic threshold a review. *Sports Medicine, 5*, 269-302.

Wasserman, K. (1987). Determinants and detection of anaerobic threshold and consequences of exercise above It. *Circulation, 76* (Suppl. VI), VI29-VI39.

Wasserman, K., Beaver, W. L., Davis, J. A., Pu, J. Z., Herber, D., & Whipp, B. J. (1985). Lactate, pyruvate, and lactate-to-pyruvate ratio during exercise and recovery. *Journal of Applied Physiology, 59* (3), 935-940.

Wasserman, K., Hansen, J. E., Sue, D. Y., & Whipp, B. J. (1987). *Principles of exercise testing and interpretation.* Philadelphia: Lea & Febiger.

Wasserman, K., Van Kessel, A. L., & Burton, G. G. (1967). Interaction of physiological mechanisms during exercise. *Journal of Applied Physiology, 22*, 71-85.

Wasserman, K., Whipp, B., Koyal, S., & Beaver, W. (1973). Anaerobic threshold and respiratory gas exchange during exercise. *Journal of Applied Physiology, 35*, 236-243.

Winder, W. W., Hagberg, J. M., Hickson, R. C., Ehsanis, A. Ad, & McLane, J. A. (1978). Time course of sympatho-adrenal adaptation to endurance exercise in training in man. *Journal of Applied Physiology, 45* (3), 370-374.

Wiswell, R. A., Girandola, R. N., & deVries, H. A. (1979). Comparison of anaerobic threshold on bicycle and treadmill. *Medicine and Science in Sports, 11*, 88.

Withers, R. T., Sherman, W. M., Miller, J. M, & Costill, D. L. (1981). Specificity of the anaerobic threshold in endurance trained cyclists and runners. *European Journal of Applied Physiology, 47*, 93-104.

Yoshida, T. (1984). Effect of exercise duration during incremental exercise on the determination of anaerobic threshold and the onset of blood lactate accumulation. *European Journal of Applied Physiology, 53*, 196-199.

Yoshida, T., Takeichi, N., & Suda, Y. (1982). Arterial versus venous blood lactate increase in the forearm during incremental bicycle exercise. *European Journal of Applied Physiology, 50,* 87-93.

Appendix A

RAW DATA

Table 11

Anthropometric Raw Data

Subject	Age (yr)	Height (cm)	Pre Weight (kg)	Post Weight (kg)
MB	20	187.3	74.0	73.5
DC	19	179.5	72.4	71.7
DI	22	177.2	77.8	75.7
DM	21	178.3	73.3	72.3
PP	24	181.7	76.9	74.4
DS	21	170.2	88.3	86.9
CV	19	179.2	82.4	80.2

Note. Pre and post weight values are means of 4 pre and 4 post tests.

Table 12

Maximal Exercise Raw Data

Subject	Pre Leg AVO_2max (l/min)	Post Leg AVO_2max	Pre Arm AVO_2max	Post Arm AVO_2max
MB	4.11	4.35	2.78	2.91
DC	3.75	3.94	2.63	2.73
DI	3.50	3.91	2.46	2.61
DM	3.89	4.10	2.86	3.02
PP	3.96	4.56	2.56	2.78
DS	4.36	4.49	2.91	2.88
CV	3.63	4.27	2.71	2.88

Subject	Pre Leg RVO_2max (ml/kg/min)	Post Leg RVO_2max	Pre Arm RVO_2max	Post Arm RVO_2max
MB	55.27	59.88	37.62	40.07
DC	52.02	55.06	36.09	37.70
DI	45.00	52.06	31.62	34.27
DM	52.94	57.53	39.01	41.62
PP	51.81	58.48	33.49	37.68
DS	49.56	51.70	33.53	33.44
CV	44.15	53.32	32.78	36.17

	Pre Leg MaxVE (l/min)	Post Leg MaxVE	Pre Arm MaxVE	Post Arm MaxVE	Pre Leg MaxHR (bpm)	Post Leg MaxHR	Pre Arm MaxHR	Post Arm MaxHR
MB	113.07	162.22	124.94	143.00	199	194	191	188
DC	145.39	152.90	101.51	102.56	201	192	190	186
DI	105.34	106.79	88.58	90.94	184	180	182	175
DM	146.02	139.89	110.67	130.40	201	201	190	194
PP	144.94	160.77	102.54	107.95	192	191	184	181
DS	153.06	140.11	112.26	104.24	189	190	177	179
CV	138.00	134.49	102.83	124.00	201	194	194	189

AVO_2max, absolute maximal oxygen consumption. RVO_2max, relative maximal oxygen consumption
MaxVe, maximal ventilation. MaxHR, maximal heart rate

Table 13

Submaximal Exercise Raw Data

Subject	Pre Leg ALT (l/min)	Post Leg ALT	Pre Arm ALT	Post Arm ALT	Pre Leg RLT (%AVO_2max)	Post Leg RLT	Pre Arm RLT	Post Arm RLT
MB	3.09	3.37	1.19	1.52	75.2	77.5	42.8	52.2
DC	2.56	3.21	1.42	1.62	68.3	81.5	54.0	59.3
DI	2.82	3.38	1.60	1.84	80.6	86.4	65.0	70.5
DM	2.75	3.21	1.95	1.88	70.7	78.3	68.2	62.3
PP	2.88	3.29	1.63	1.78	72.7	72.1	63.7	64.0
DS	3.13	3.59	2.10	1.94	71.8	80.0	72.2	67.4
CV	2.49	3.35	1.50	1.69	68.6	78.5	55.4	58.7

Subject	Pre Leg LTWL (kpm/min)	Post Leg LTWL	Pre Arm LTWL	Post Arm LTWL	Pre Leg LTVE (l/min)	Post Leg LTVE	Pre Arm LTVE	Post Arm LTVE
MB	1260	1440	450	540	77.75	91.40	34.54	49.71
DC	1080	1350	450	450	66.34	84.75	41.03	49.13
DI	1260	1440	450	450	70.26	81.75	41.71	51.84
DM	1170	1530	630	540	76.48	78.51	61.87	52.53
PP	1350	1440	450	450	74.07	77.80	52.93	50.78
DS	1170	1350	630	540	80.72	84.10	68.49	57.63
CV	990	1260	450	540	63.23	93.12	42.06	56.32

Subject	Pre Leg LTHR (bpm)	Post Leg LTHR	Pre Arm LTHR	Post Arm LTHR	Pre Leg SubHR (bpm)	Post Leg SubHR	Pre Arm SubHR	Post Arm SubHR
MB	171	169	129	120	179	142	180	158
DC	168	173	134	143	169	130	174	162
DI	164	170	127	139	159	136	154	140
DM	171	176	148	152	169	157	141	140
PP	158	163	154	147	164	145	160	132
DS	169	166	157	160	174	163	161	161
CV	165	175	143	159	184	152	186	149

Subject	Pre Leg LTO_2P (l/beat)	Post Leg LTO_2P	Pre Arm LTO_2P	Post Arm LTO_2P
MB	0.018	0.020	0.009	0.013
DC	0.015	0.019	0.011	0.011
DI	0.017	0.020	0.013	0.013
DM	0.016	0.018	0.013	0.012
PP	0.018	0.020	0.011	0.012
DS	0.019	0.022	0.013	0.012
CV	0.015	0.019	0.010	0.011

ALT, absolute lactate threshold. RLT, relative lactate threshold. LTWL, lactate threshold workload. LTVE, lactate threshold ventilation. LTHR, lactate threshold heart rate. SubHR, submaximal heart rate. LTO_2P, lactate threshold oxygen pulse

Appendix B

STATISTICAL ANALYSIS

Table 14

Summary of DM MANOVAs for Factors of Mode and Time

Multivariate Tests Sig. of F				Univariate Tests Sig. of F			
Variables	Mode	Time	Interaction	Variable	Mode	Time	Interaction
AVO_2max & RVO_2max	0.000	0.006	0.017	AVO_2max	0.000	0.003	0.011
				RVO_2max	0.000	0.001	0.003
MaxHR &MaxVE	0.002	0.187*	0.601	MaxHR	0.000	0.071	0.289
				MaxVE	0.004	0.165	0.878
ALT, RLT & LTWL	0.000	0.016	0.001	ALT	0.000	0.001	0.005
				RLT	0.000	0.018	0.151
				LTWL	0.000	0.001	0.007
LTHR &LTVE	0.000	0.234*	0.047	LTHR	0.003	0.162	0.921
				LTVE	0.000	0.088	0.016
SubHR &LTO_2P	0.000	0.013	0.085	SubHR	0.730	0.003	0.116
				LTO_2P	0.000	0.002	0.030

Note. * Not significant at $p < 0.05$, therefore will not be valid to analyse these varibles for arm or leg changes over the factor of time.

Table 15

Summary of DM MANOVAs for Factor of Time

Multivariate Tests Sig of F for Time Effect			Univariate Tests Sig of F for Time Effect		
Variables	Arm	Leg	Variable	Arm	Leg
AVO_2max &RVO_2max	0.018	0.007	AVO_2max RVO_2max	0.005 0.003	0.004 0.001
ALT RLT &LTWL	0.081	0.006	ALT RLT LTWL	0.107 0.414 0.689	0.000 0.009 0.009
SubHR &LTO_2P	0.066	0.001	SubHR LTO_2P	0.020 0.413	0.001 0.000

Table 16

Summary of Multivariate MANOVAs for Factor of Mode

Multivariate Tests Sig of F for Mode Effect			Univariate Tests Sig of F for Mode Effect		
Variables	Pre	Post	Variable	Pre	Post
AVO_2max &RVO_2max	0.000	0.000	AVO_2max RVO_2max	0.000 0.000	0.000 0.000
MaxHR &MaxVE	0.005	0.002	MaxHR MaxVE	0.001 0.009	0.000 0.007
ALT RLT &LTWL	0.001	0.000	ALT RLT LTWL	0.000 0.022 0.000	0.000 0.000 0.000
LTHR &LTVE	0.007	0.000	LTHR LTVE	0.003 0.001	0.003 0.000
SubHR &LTO_2P	0.002	0.000	SubHR LTO_2P	0.211 0.000	0.718 0.000

Appendix C

CONSENT FORM

Studies involving human subjects require written consent of the participants. I, _____, authorize Nancy Saumure (231-6747 or 564-9136) of the School of Human Kinetics, University of Ottawa, to administer and conduct exercise tests designed to measure my maximal aerobic power and lactate threshold and supervise all endurance training sessions. This study will be conducted under the supervision of Dr. J. S. Thoden, Department of Human Kinetics (564-5912).

I understand that all testing sessions will be conducted in the presence of two individuals who possess valid CPR certificates and who have access to a telephone in the unlikely event of an emergency to contact an ambulance. I understand that although no medical doctor will be present, there are doctors on campus at the Student Health Services during operating hours.

I understand that I will perform two leg cycling tests and two arm cranking tests on Monark exercise ergometers, both before and after an eight week endurance training program. The first complete test in each exercise mode is performed to measure my maximal aerobic power and lactate threshold and will involve progressively increased workloads every two minutes for a minimum of six minutes and a maximum of 20 minutes. The test will be terminated when I cannot maintain the required cadence due to fatigue or if I become distressed in any way or develop any abnormal response. This test may also require performance of a phase of continuous exercise, after a 10 to

15 minute rest, at the maximal intensity identified during the progressive phase. The second test in each exercise mode, performed to verify the lactate threshold, will involve two 12 minute bouts of continuous, submaximal exercise separated by a 6 to 10 minute active rest period.

During the performance of the pre and post training tests, I will breathe through a mouthpiece and my heart rate will be monitored by a Sport Tester strap attached around my chest. Capillary blood samples will be drawn by micropuncture from a finger while leg cycling and from a prewarmed toe while arm cranking. Blood is taken at the end of each 2 minute stage during a maximal aerobic test and four times during steady state, lactate threshold verification tests and usually requires less than 10 samples for any given test. I will experience a slight pricking sensation when blood samples are collected. I understand that I may experience some local muscular fatigue, dryness of the mouth and throat, and an increased resistance to breathing while exercising; although every effort will be made to conduct the test in such a way as to minimize discomfort and risk. However, I understand that there are potential risks to some individuals while performing an exercise test, these include episodes of transient lightheadedness, fainting, chest discomfort, leg cramps and very rarely, heart attacks. I recognize that a physical examination prior to performing exercise testing is necessary and I will be required to submit a doctor's certificate.

I understand that between pre and post testing sessions, I will participate in a leg cycling, endurance training program consisting of three training session per week for eight consecutive weeks. During each supervised training period, my heart rate will be monitored during submaximal exercise.

I understand all information collected will be kept confidential and presented in an anonymous form in the final report. I understand that I will be informed of my

maximal aerobic power and lactate threshold as well as the results of the study, upon its completion.

I understand that I have the right to withdraw from this study at any time.

DATE: _____ SUBJECT: _____

WITNESS: _____