DIFFERENCES IN THE RATE OF LACTATE REMOVAL FROM SKELETAL MUSCLE FOLLOWING INTENSE EXERCISE IN GROUPS WITH DIFFERENT AEROBIC POWER

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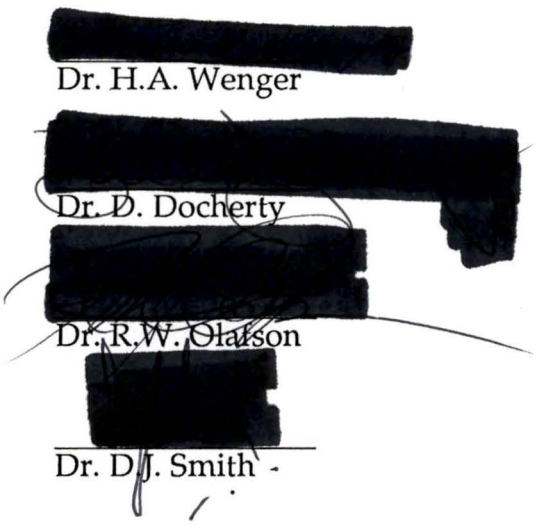
ABSTRACT

To determine the effect of different levels of aerobic power ($\dot{V}O_2max$) on the rate of lactate removal from skeletal muscle following intense exercise, 17 subjects, blocked into high (n=7) and low (n=10) $\dot{V}O_2max$ groups, completed four 30 second intervals at maximal power on a cycle ergometer. Needle biopsies from the vastus lateralis and venous blood samples were taken immediately after the first interval (T1A), four minutes post the first interval (T1B) and again immediately (T4A) and four minutes post (T4B) the fourth interval. Muscle and blood lactates were determined enzymatically, using fluorometric and spectrophotometric methods, respectively.

Muscle lactate concentration immediately after the first (T1) and fourth (T4) intervals was 12.3 and 19.4 mmol kg^{-1} for the high $\dot{V}O_2max$ group and 14.6 and 20.2 mmol kg^{-1} for the low $\dot{V}O_2max$ group. Lactate removal rates at T1 and T4 were calculated to be 25.9 and 37.5 umol kg^{-1} s^{-1} for the high group and 33.6 and 40.0 umol kg^{-1} s^{-1} for the low group. Muscle lactate concentration and lactate removal rates were not significantly different between the high and low groups. The same was found for blood lactate concentration and changes in blood lactate levels. Muscle lactate concentration correlated significantly to lactate removal rates in T1 and T4. These data suggest that the rate of removal from skeletal muscle following intense exercise is not affected by different levels of aerobic power. However, they do suggest the possible existence of a relationship

between the concentration of lactate present in the muscle and the ability of the muscle to remove it.

Examiners:



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DEDICATION

This thesis is dedicated to the memory of my father.

DIFFERENCES IN THE RATE OF LACTATE REMOVAL FROM SKELETAL MUSCLE FOLLOWING INTENSE EXERCISE IN GROUPS WITH DIFFERENT AEROBIC POWER

INTRODUCTION

When the rate of energy expenditure by muscle is too high to be met by the aerobic combustion of fuels, substantial amounts of energy will be supplied through the incomplete breakdown of glucose with the resultant production of lactate (Hultman and Sahlin, 1980). Lactate production may occur at the onset of exercise, in the transition from one workload to a higher load or during heavy exercise (Jones, 1980).

When lactate production exceeds removal, it accumulates in muscle and blood (Sahlin et al., 1976). Karlsson and Saltin (1970) proposed that lactate accumulation in muscle, with its concurrent decrease in pH, was the reason for exhaustion at heavy and medium workloads. It has also been shown that muscle pH will decrease after dynamic exercise (Hermansen and Osnes, 1972) and isometric exercise (Sahlin et al., 1975) and it is this acidification which has been postulated to be a limiting factor for physical performance of high intensity short duration (Jones et al., 1977; Sahlin et al., 1976; Steinhagen et al., 1976). Several authors have attempted to explain the mechanisms responsible for the detrimental effect of pH on performance. It has been suggested that increases in H^+ concentration can effect energy metabolism by inhibition of glycolysis (Kowalchuk et al., 1984; Sutton et al., 1981) and/or of the contraction process (Duchateau et al., 1987; Mainwood & Renaud, 1985; Tesch, 1980).

The extent to which pH decreases, depends upon the rate of lactic acid production, the buffering capacity of muscle and the amount of H^+ released from muscle (Hultman and Sahlin, 1980). But, Jones et al. (1980) and Piiper (1972) suggest that, although increases in H^+ concentration are reduced by the buffering capacity of muscle, the metabolic flux is so great in heavy exercise that even large buffering capacities may be unable to control pH within tolerable limits. Thus any processes able to remove H^+ from the muscles will enhance the ability of muscle to continue work.

It is well established that some of the effects of aerobic power (endurance) training include increased skeletal muscle capillarization (Hickson et al., 1981; Anderson and Henriksson, 1977; Brodal et al., 1977), increased cardiac output (Eckblom & Hermansen, 1968; Saltin et al., 1968) and increased stroke volume (Frick et al., 1963). It has been postulated that these factors, influenced by aerobic power training, can enhance both fatigue resistant aerobic metabolism and lactic acid removal (Wenger, 1981). Therefore, this study will consider the following questions:

1. Is there a difference in lactate concentration in skeletal muscle after high intensity effort in groups with different aerobic power?
2. Is there a difference in the rate of lactate removal from skeletal muscle after high intensity effort in groups with different aerobic power?
3. Is there a difference in blood lactate concentration or changes in blood lactate concentration after high intensity effort in groups with different aerobic power?

METHODS

SUBJECTS

Seventeen volunteers, seven female and ten male from the University of Victoria signed informed consent and acted as subjects. Physical characteristics are shown in Table I. All subjects were familiarized with testing procedures prior to commencement of the study.

Subjects were blocked into high and low aerobic power ($\dot{V}O_2max$) groups based on the following criteria: high $\dot{V}O_2max$ group, n=7, $\dot{V}O_2max > 54.6$ ml kg^{-1} min^{-1} and low $\dot{V}O_2max$ group, n=10, $\dot{V}O_2max < 50.9$ ml kg^{-1} min^{-1}. Mean $\dot{V}O_2max$ values for the high and low groups were 57.8 ml kg^{-1} min^{-1} and 47.9 ml kg^{-1} min^{-1}, respectively.

TESTING PROCEDURE

The testing schedule is illustrated in Figure 1. All subjects were asked to refrain from any physical activity one day prior to testing. Anthropometric measures included height, weight and sum of six skin folds (bicep, tricep, suprailiac, subscapular, front thigh and medial calf). Two - legged $\dot{V}O_2max$ and heart rate (HR) were determined on an incremental cycle ergometer test to fatigue (Thoden et al., 1982). Respiratory gases were collected every 30 seconds on a Beckman Metabolic Measurement Cart. Heart rates were recorded every minute during the test and for two minutes during recovery on a Cambridge VF4 electrocardiograph.

On a subsequent day (7 - 47 days post), subjects performed four repeats of a 30 second maximal anaerobic power test on a cycle ergometer with a work to rest ratio of approximately one to fifteen. To achieve elevated lactate levels in all four trials, resistance was set at 0.075 kg kg^{-1} body weight with the exception of the more accomplished cyclists where resistance was set at 0.09 kg kg^{-1} body weight. Average power output (PO) for each 30 second bout was determined from pedal revolutions and resistance and expressed as watts kg^{-1} body weight.

Muscle samples were obtained by needle biopsy (Bergstrom, 1962) adapted for suction (Evans et al., 1982) from the medial portion of the vastus lateralis muscle. Biopsies 1 (T1A) and 2 (T1B) were taken immediately and four minutes post trial 1 on one leg, chosen randomly, while biopsies 3 (T4A) and 4 (T4B) were taken immediately and four minutes post trial 4 on the other leg. Antecubital venous blood samples were drawn to coincide with the muscle biopsies as well as after trials 2 and 3.

BIOCHEMICAL ANALYSES

Muscle tissue samples 20 to 50 mg in size were quickly extracted and frozen in liquid nitrogen in a mean time of 32 seconds. Samples were wrapped in labelled tinfoil and stored at - 80 ^{o} C. Tissue samples were prepared for analysis according to Costill et al. (1982) and analyzed fluorometrically for lactate as described by Lowry and Passonneau (1972). Lactate removal rates in umol kg^{-1} min^{-1} wet weight were calculated from the difference in muscle lactate concentration appearing immediately post and four minutes post trials 1 and 4 divided by the exact time interval between the two biopsies.

Venous blood samples were deproteinized in 4% perchloric acid, centrifuged and analyzed spectrophotometrically for blood lactate according to Sigma method 826-UV (Sigma Chemical Company, 1981). Changes in blood lactate concentration in mmol L^{-1} were calculated from the difference in blood lactate concentration appearing immediately post and four minutes post each of the trials.

Paired Student t-tests were used to determine statistical significance between high and low $\dot{V}O_2max$ groups while Pearson product-moment correlations were used to determine relationships between variables and groups. Significance was accepted at $p < 0.05$.

TABLE 1

Physical characteristics of the subjects.

Subject	Sex (m/f)	Age (yr)	Height (cm)	Weight (kg)	Sum of skinfolds* (mm)	$\dot{V}O_2max$ (ml kg^{-1} min^{-1})
JT	m	22	180.6	68.0	42.9	60.3
GS	m	29	175.9	68.4	35.2	60.2
DB	m	20	188.5	84.5	41.9	59.2
SC	m	22	180.6	77.0	30.7	57.7
JM	f	27	171.0	61.5	42.9	56.3
PW	m	28	181.8	75.2	32.9	56.2
CD	f	21	170.5	66.2	74.1	54.6
PN	f	26	160.4	60.3	88.2	50.9
AS	f	27	176.0	72.6	59.6	50.3
TL	m	20	186.6	95.6	62.5	50.3
SP	m	39	176.6	85.7	46.2	49.9
CG	m	22	186.3	79.8	41.8	49.6
BR	m	24	199.3	93.5	39.5	49.4
PF	m	32	183.8	88.1	50.1	48.5
AT	f	27	173.4	76.6	81.7	45.6
SH	f	23	172.6	75.2	104.6	43.5
JS	f	25	169.0	55.8	66.1	40.7
$\bar{x}$		25.5	178.4	75.5	55.3	52.0
SD		4.8	9.1	11.2	21.3	5.8

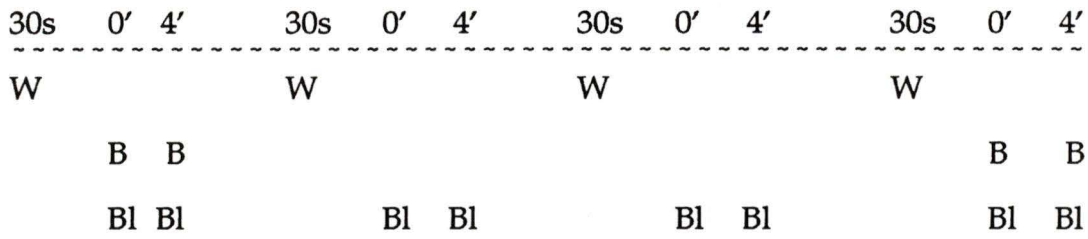
* sum of skinfolds = tricep + bicep + subscaspular + suprailiac + front thigh + medial calf.

FIGURE 1

The testing schedule.

Day 1: Body weight, height, skinfolds, $\dot{V}O_2max$ (two -legged) and heart rate.

Day 2: Muscle biopsies and blood samples post exercise and after four minutes of recovery.



W = work
B = biopsy
Bl = blood

RESULTS

Relative $\dot{V}O_2max$ (ml kg^{-1} min^{-1}) was significantly different ($p < 0.01$) between the high and low groups. No statistical significance between the high and low $\dot{V}O_2max$ groups was shown for muscle lactate concentration (Table 2) or rates of lactate removal (Table 3) from skeletal muscle following repeated high power anaerobic work. There was also no significant high versus low difference for blood lactate concentration (Table 4) or changes in blood lactate concentration (Table 5) following four minutes of recovery in the two groups. Muscle and blood lactate concentrations are presented graphically in Figure 2.

Muscle lactate concentration was not related to power output in either trial 1 or 4. However, muscle lactate concentration in trial 1 was highly correlated to lactate removal in trial 1 for both the high and low $\dot{V}O_2max$ groups. A significant relationship also existed between lactate concentration and rate of removal in trial 4 for the high $\dot{V}O_2max$ group but not the low $\dot{V}O_2max$ group (Table 6). Significant correlations were also evident in both trials for the total group.

Average 30 second power output was significantly different between the high and low $\dot{V}O_2max$ groups on the fourth trial, decreasing to a greater extent in the low $\dot{V}O_2max$ group (Table 7).

TABLE 2

Mean values (±SE) of muscle lactate concentration(mmol kg^{-1}) in high and low fitness groups following the first and fourth sprint intervals.

	T1A	T4A
High $\dot{V}O_2max$ (n=7) ($\bar{x}$ = 57.8 ml kg^{-1} min^{-1})	12.3 (1.5)	19.4 (1.7)
Low $\dot{V}O_2max$ (n=10) ($\bar{x}$ = 47.9 ml kg^{-1} min^{-1})	14.6 (1.2)	20.2 (1.1)

TABLE 3

Mean values (±SE) of lactate removal rates (umol kg^{-1} s^{-1}) in high and low fitness groups following the first and fourth sprint intervals.

	T1	T4
High $\dot{V}O_2max$ (n=7) ($\bar{x}$ = 57.8 ml kg^{-1} min^{-1})	25.9 (5.5)	37.5 (7.7)
Low $\dot{V}O_2max$ (n=10) ($\bar{x}$ = 47.9 ml kg^{-1} min^{-1})	33.6 (4.7)	40.0 (4.6)

TABLE 4

Mean values (±SE) of blood lactate concentration (mmol L^{-1}) in high and low fitness groups following the first and fourth sprint intervals.

	T1A	T4A
High $\dot{V}O_2$max (n=7) ($\bar{x}$ = 57.8 ml kg^{-1} min^{-1})	5.4 (0.5)	13.7 (1.5)
Low $\dot{V}O_2$max (n=10) ($\bar{x}$ = 47.9 ml kg^{-1} min^{-1})	5.5 (0.7)	12.4 (1.0)

TABLE 5

Mean values (±SE) of changes in blood lactate concentration (mmol L^{-1}) in high and low fitness groups following the first and fourth sprint intervals.

	T1	T4
High $\dot{V}O_2$max (n=7) ($\bar{x}$ = 57.8 ml kg^{-1} min^{-1})	2.6 (0.5)	-1.1 (1.0)
Low $\dot{V}O_2$max (n=10) ($\bar{x}$ = 47.9 ml kg^{-1} min^{-1})	2.3 (0.6)	0.1 (0.6)

FIGURE 2

Muscle and blood lactate concentrations following four 30 second sprint intervals.

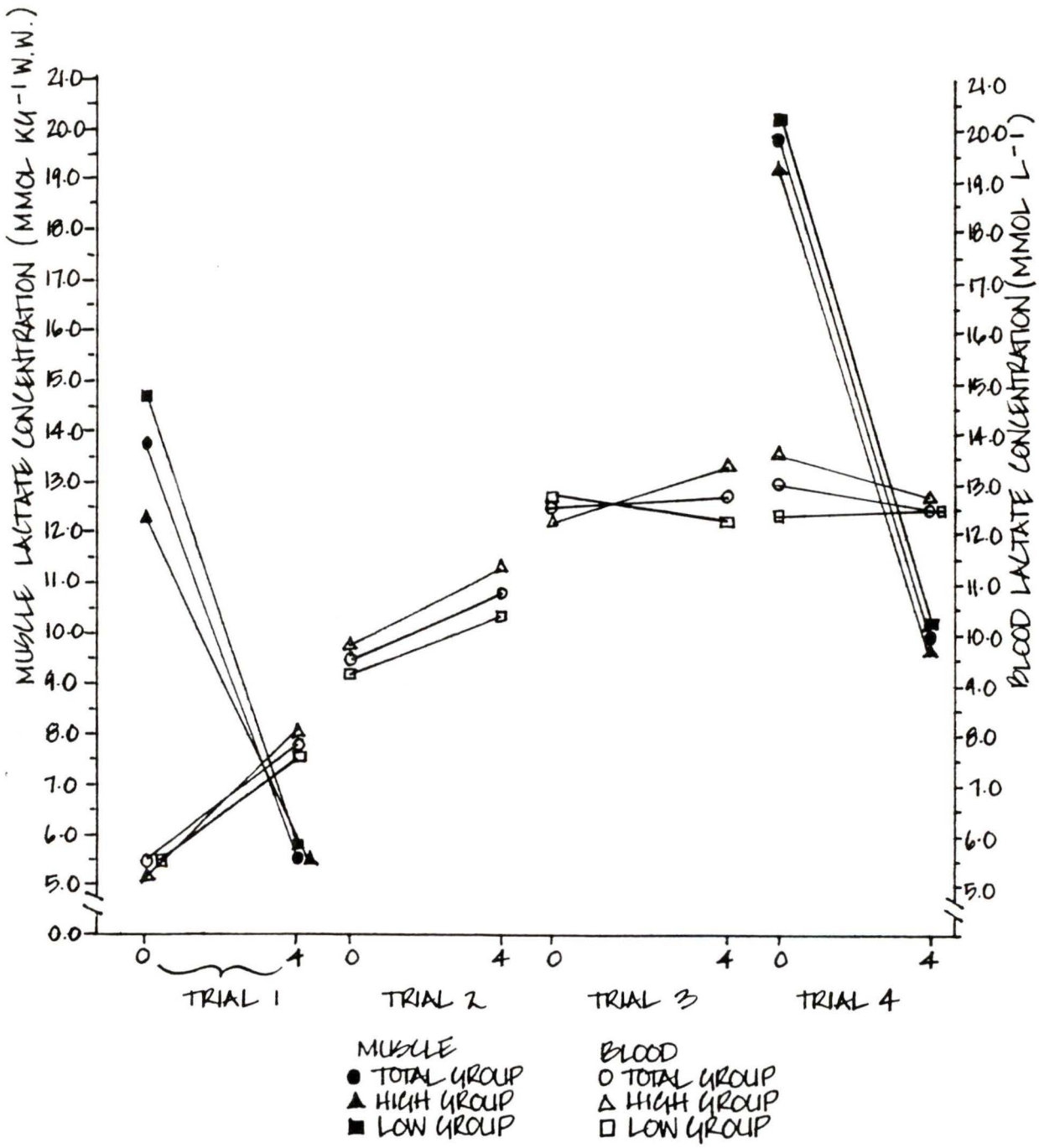


TABLE 6

Correlation (r) between muscle lactate concentration and lactate removal rates in the whole group and high and low fitness groups following the first and fourth sprint intervals.

	T1	T4
Total group (n=17)	0.82 *	0.58 *
High $\dot{V}O_2$max (n=7) ($\bar{x}$ = 57.8 ml kg^{-1} min^{-1})	0.87 *	0.71 *
Low $\dot{V}O_2$max (n=10) ($\bar{x}$ = 47.9 ml kg^{-1} min^{-1})	0.76 *	0.43

* significant, $p < 0.05$

TABLE 7

Mean values (±SE) of 30 second power outputs (watts kg^{-1}) in high and low fitness groups.

	T1	T2	T3	T4
High $\dot{V}O_2$max (n=7) ($\bar{x}$ = 57.8 ml kg^{-1} min^{-1})	9.0 (0.4)	8.9 (0.4)	8.7 (0.3)	8.5 (0.3) *
Low $\dot{V}O_2$max (n=10) ($\bar{x}$ = 47.9 ml kg^{-1} min^{-1})	8.3 (0.3)	7.9 (0.3)	7.6 (0.2)	7.6 (0.2)

* significant high versus low difference, $p < 0.05$.

DISCUSSION

The needle biopsy procedure described by Bergstrom (1962) is recognized as an important technique in the study of human skeletal muscle. Since the energy demands during maximal effort result in the rapid diffusion of hydrogen ion and lactic acid from the muscle, it has been assumed that needle biopsy samples must be taken immediately after exercise in order to accurately reflect muscle status during activity. Costill et al. (1982) have shown that a delay of 1.0 - 1.5 minutes after the cessation of exhaustive exercise before taking the biopsy does not appear to alter muscle lactate values. The time interval between cessation of exercise and taking of the biopsy in this study averaged 64.0 $\pm$14.7 seconds. Costill et al. (1982) also showed that although it is crucial to freeze the biopsy tissue in less that 2 seconds after extraction for the measurement of muscular pH, muscle lactate values remained unchanged after a 30 second time delay in freezing. Samples in this study were extracted and frozen in liquid nitrogen in a mean time of 31.9 $\pm$11.5 seconds.

One important adaptation to the muscle biopsy procedure has been the use of suction applied to the needle inserted into the muscle allowing a larger amount of tissue to be pulled into the central bore of the needle (Evans et al., 1982). This larger sample size, ranging between 50 - 100 mg, increases the variety of analytical procedures that could be done without causing undue discomfort to the subject (Evans et al., 1982). The sample sizes in this study ranged from 20 - 50 mg in size and were adequate for fluorometric lactate analysis (Lowry and Passonneau, 1972) using the whole muscle technique described by Costill et al. (1982).

Muscle and blood lactate responses reported in this study compare to other reports of maximal effort of short duration. Hirvonen et al. (1987) measured blood lactate at 8.3 mmol L^{-1} following a 100 meter sprint and Tesch (1978) measured muscle lactate at 20.8 mmol kg^{-1} wet weight after 60 seconds of maximal effort. Both Hermansen and Vaage (1977) and Karlsson (1971) have shown muscle and blood lactate values of 26.4 mmol kg^{-1} wet weight and 20.9 mol L^{-1} after three 60 second bouts of maximal cycling and 15.6 mmol kg^{-1} wet weight and 9.3 mmol L^{-1} following brief maximal running, respectively. Gass et al.(1981), Karlsson et al. (1972) and Karlsson and Saltin (1970) also observed similar values after exercise causing exhaustion in two to eight minutes. Muscle lactate values in the present study are however, lower than those reported by McCartney et al. (1986) who showed values reaching 35.1 mmol kg^{-1} wet weight using an isokinetic cycle ergometer and similar test lengths but shorter time intervals between trials. The power output generated by their subjects was also greater than that demonstrated by subjects of the present study. Although in the present study muscle lactate concentration was not correlated with power output it has been suggested that higher muscle lactate values may be a function of greater power output. Jacobs et al. (1983) showed greater lactate concentrations in subjects with greater power output following supramaximal execise of 10 and 30 seconds in duration.

Several studies have shown that lactate production is greater in fast twitch muscle fiber (Tesch, 1978; Harris et al., 1977; Essen & Henriksson, 1974; Baldwin & Tipton, 1972). The rate of lactate production is approximately 0.5 umol g^{-1} s^{-1} for fast twitch fiber (Dudley & Terjung, 1985; Mainwood & Renaud, 1985) and about half that in slow twitch fiber (Meyer & Terjung, 1979). Although it has been demonstrated that in normal man the quadriceps muscle consists of equal

portions of fast and slow twitch fibers (Gollnick et al., 1972), this can neither be supported nor refuted in this study as fiber type distribution was not calculated. Therefore, whether the higher power output demonstrated by the high group by trial 4 was fiber type related, fitness related or due to other factors cannot be determined.

The rate of lactate removal from muscle to blood following maximal exercise is difficult to compare due to differences in exercise mode and length of test, and differences in procedures for calculation of removal rate. Most reports have calculated lactate output or lactate disappearance from muscle as the product of total blood flow through a muscle bed and the venoarterial difference. Chirtel et al., (1984) calculated lactate output values ranging from 0.03 to 0.24 umol g^{-1} min^{-1} following 25 minutes of continuous work and Hermansen & Vaage (1977) calculated the rate of lactate disappearance as 0.74 mmol kg^{-1} min^{-1} wet weight. Calculations in this study were based on measured muscle lactate values averaged over a period of 255 $\pm$8.6 seconds. A higher correlation between removal of lactate and lactate concentration was evident in trial 1 where lactate concentration was lower as compared to trial 4 where the correlation was not as strong and actual lactate values were greater. This supports studies showing a linear relationship between concentration and removal at lower concentrations (Jorfeldt et al., 1978) or work rates (Connett et al., 1986). The lower correlations between concentration and removal at higher concentrations of lactate as seen in trial 4 could suggest that there is a saturation of the translocation process where a further increase in concentration does not increase the rate of efflux as suggested by Connett et al. (1986), Jorfeldt et al.(1978), Karlsson et al. (1972) and Karlsson (1971).

A major finding of this study was that there was no difference in the concentration of muscle or blood lactate accumulated in muscle or the rate of lactate removal from muscle following maximal exercise in groups with different levels of aerobic power. It has been postulated that the benefits of aerobic power such as increased aerobic metabolism of lactate, increased capillarization, increased muscle blood flow and blood volume, increased mitochondrial concentration and size, increases in aerobic enzymes, isozyme changes, cellular membrane changes and increased membrane transport should enhance waste removal following intense exercise (Fox et al., 1988; Hickson et al., 1981; Wenger, 1981; Hermansen & Vaage, 1977; Karlsson, et al., 1975). However, that a relationship was not apparent in this study could have been due to the following four factors. First, there may not have been a sufficient difference in aerobic power among the groups to identify a true difference between the two groups (high group, $\dot{V}O_2max$ ranged from 54.6 to 60.3 ml kg^{-1} min^{-1} and low group, $\dot{V}O_2max$ ranged from 40.7 to 50.9 ml kg^{-1} min^{-1}) and that with a greater difference in $\dot{V}O_2max$ between groups the rate of removal may have increased. Secondly, changes were measured over a four minute period and perhaps changes are greater in a shorter period of time or over longer periods since the half time of lactate removal is approximately 10 minutes with active recovery (Sahlin et al., 1976; Bonen & Belcastro, 1976) and 25 minutes with rest recovery (Karlsson & Saltin, 1971; Hermansen et al., 1975). Thirdly, resting recovery rather than active recovery was used in this study which may not have allowed the aerobic benefit to be expressed and, finally, this study described removal rates in two groups whose aerobic powers were already established. It did not elicit increases in aerobic power to determine whether this increases the rate of lactate removal from muscle.

CONCLUSIONS

1. In groups with different aerobic power, there is no difference in the concentration of lactate accumulated in muscle or the rate of lactate removal from muscle following maximal effort.

2. In groups with different aerobic power, there is no difference in the concentration of blood lactate or changes in blood lactate after four minutes of recovery following maximal effort.

FURTHER RESEARCH SUGGESTIONS

1. The measurement of muscle pH corresponding with muscle and blood lactate levels during exercise is suggested in future research to provide a clearer understanding of the acid-base balance in muscle.

2. Further research in measuring lactate removal rates and how they may be affected by skeletal muscle fibre type would enable investigators to isolate the mechanisms involved.

3. Future research is recommended to study the chronic effects of aerobic power on lactate removal rates by implementation of a training program.

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APPENDIX A

Informed consent for research on the effect of aerobic power on the rate of lactate removal from skeletal muscle following intense exercise.

TESTING PROCEDURE

You will be asked to perform four 30 second sprints on a bicycle ergometer with four to six minutes recovery between sprints. Four small pieces of muscle from the thigh (vastus lateralis) will be taken by needle biopsy during the series of 30 second sprints. Also, small amounts of blood (5 ml) will be taken from a peripheral arm vein.

For the biopsy, a 5 mm incision is made in the skin and muscle fascia under local anaesthetic and a hollow needle inserted approximately 1 cm into your muscle. The needle will extract a small amount of tissue (approximately 60 mg). There will be little or no discomfort associated with the procedure, however, you will experience a sensation of pressure. Rarely you could experience some muscle spasm.

RISK

The needle biopsy will be performed by a skilled physician and the blood sample by a skilled nurse. Both procedures are considered safe with little risk of complication although with any laceration there is some risk of infection. Several hours after sampling you may experience minor bruising at the biopsy site.

CONSENT

I have read the above and agree to participate in this research project at my own risk. I am nineteen years of age or older and regularly take part in physical activity at least as intense as these tests. I realize that I may expect a thorough explanation and/or demonstration of any procedures and that I may terminate participation at any time in any or all procedures of my own volition.

Having voluntarily assumed participation and risks thereof, in the project, I hereby disclaim and release the University of Victoria, its agents, servants or employees, including all personnel involved in the research project, from any and all liability that might otherwise arise as a result of my participation as a research subject in this study.

NAME:_____

DATE:_____

PHONE NO.:_____

SIGNATURE:_____

APPENDIX B

Mean values (±SE) of muscle lactate concentration for the whole group and high and low $\dot{V}O_2max$ groups.

Groups (n)	Muscle lactate concentration (mmol kg^{-1})			
	T1A	T1B	T4A	T4B
Total(n=17)	13.7 (1.0)	5.3 (0.5)	19.9 (0.9)	10.0 (0.9)
High(n=7)	12.3 (1.5)	5.8 (0.8)	19.4 (1.7)	9.9 (1.4)
Low(n=10)	14.6 (1.2)	5.8 (0.8)	20.2 (1.1)	10.1 (1.2)

APPENDIX C

Mean values (±SE) of blood lactate concentration for the whole group and high and low $\dot{V}O_2max$ groups.

Groups (n)	Blood lactate concentration (mmol L^{-1})							
	T1A	T1B	T2A	T2B	T3A	T3B	T4A	T4B
Total (n=17)	5.5 (0.5)	7.9 (0.4)	9.3 (0.5)	10.8 (0.6)	12.4 (0.7)	12.8 (0.7)	12.9 (0.8)	12.6 (0.7)
High (n=7)	5.4 (0.5)	8.0 (0.4)	9.6 (0.5)	11.4 (0.7)	12.2 (1.0)	13.4 (1.2)	13.7 (1.5)	12.6 (0.9)
Low (n=10)	5.5 (0.7)	7.8 (0.7)	9.2 (0.8)	10.4 (0.8)	12.5 (0.9)	12.4 (0.8)	12.4 (1.0)	12.5 (1.0)

APPENDIX D

LITERATURE REVIEW

The contraction of skeletal muscle is powered by chemical energy in the form of adenosine triphosphate (ATP). Since the concentration of ATP in the muscle is low, it must be continually regenerated for muscle contraction to continue (Gollnick & Hermansen, 1973). The chemical processes responsible for the resynthesis of ATP are creatine phosphate (CP) splitting, anaerobic glycolysis and aerobic metabolism (Green, 1982).

The three energy systems, while distinctly different, operate in an integrated manner supplying energy as required by the muscle. The proportion, however, supplied by each pathway will vary according to the intensity and duration of the exercise (Green, 1982).

During short work periods (30 seconds to several minutes) the primary source of ATP regeneration is through anaerobic glycolysis (Fox, 1979) where in the absence of oxygen, pyruvic acid, a product of the breakdown of stored glycogen (or glucose), proceeds an additional step to form lactic acid (Brooks & Fahey, 1984). At physiological pH this acid exists in its ionic or dissociable form such that an equimolar amount of hydrogen ion (H^+) is produced in association with lactate (Sahlin et al., 1976). While it is lactic acid that is produced, it is the dissociating salt, lactate that is measured in skeletal muscle and blood.

LACTIC ACID PRODUCTION

The classical concept of lactate metabolism during exercise first described by Meyerhof (1920) and Hill et al. (1924) suggested that inadequate availability of oxygen in the tissues stimulated glycolysis and hence resulted in the formation of lactate. It is now clear that lactate production is an ongoing process and may occur at rest or during light exercise (Gollnick et al., 1986) as well as in the transition from one work load to a higher work load or during heavy exercise (Jones, 1980). In steady state exercise a constant lactate concentration indicates an equilibrium between its production and consumption (Fox & Mathews, 1981). However, when production exceeds consumption, lactate accumulates in muscle and blood (Hirche et al., 1975; Sahlin et al., 1976). This has been clearly demonstrated in studies involving humans (Karlsson et al., 1972; Jorfeldt, 1970) and animals (Hirche et al., 1975; Hirche et al., 1973). This accumulation can result in the following: a drop in muscle and blood pH (Jones, 1980; Sahlin, 1978), inhibition of glycolytic enzymes such as LDH (Karlsson et al., 1974) and PFK (Edgerton et al., 1973; Danforth, 1965) the rate limiting enzyme in glycolysis (Wenger & Reed, 1976; Gollnick & King, 1969) and immobilization of free fatty acids (Boyd et al., 1974; Issekutz et al., 1966). The degree of accumulation, however, is highly variable and influenced by numerous factors such as exercise intensity, the type of contraction, exercise duration and training state (Gollnick et al., 1986).

Lactate concentration in mammalian skeletal muscle has been measured during and following single and multiple bouts of dynamic (McCartney et al., 1986; Chirtel et al., 1984; Jacobs et al., 1983; Tesch, 1978; Karlsson, 1971) and sustained isometric exercise (Sahlin & Henriksson, 1984; Barbee et al., 1983;

Sahlin et al., 1981; Tesch & Karlsson, 1977). At low exercise intensities, (below 40% $\dot{V}O_2max$) lactate remains at resting levels, approximately 1 mmol kg^{-1} wet muscle or litre of blood. As intensity increases, a point is reached where concentration begins to rise. This point, or level of intensity is highly variable. For example, exercise at 50 to 60% $\dot{V}O_2max$ usually elicits a rise in muscle and blood lactate in only relatively unfit individuals. With increasing intensity, muscle and blood lactate concentrations rise exponentially (Gollnick et al., 1986; Brooks & Fahey, 1984; Hermansen, 1971, Karlsson, 1971). Muscle lactate levels as high as 40 mmol kg^{-1} wet weight have been observed after exercise to exhaustion (Shephard, 1984) while blood lactate levels have been recorded above 30 mmol L^{-1} after multiple bouts of dynamic exercise (Hermansen & Vaage, 1977; Hermansen & Stensvold, 1972). Pronounced lactate accumulation has also been observed after single supramaximal bouts of 10 and 30 seconds demonstrating values of 36 and 61 mmol kg^{-1} dry weight, respectively (Jacobs et al., 1983). The maximal rate of lactate production appears to be approximately 0.5 umol g^{-1} s^{-1} wet weight for muscle composed primarily of type II fibers (Dudley & Terjung, 1985; Mainwood & Renaud, 1985) and about half that for predominantly type I fibers (Meyer & Terjung, 1979). However, a more rapid rate of production, about 0.9 umol g^{-1} s^{-1} wet weight has been observed following isometric contraction at 100 % of the maximal voluntary contractile force (Ahlborg et al., 1972). Sahlin and Henriksson (1984) suggest that muscle lactate concentrations are higher after isometric contraction, due to circulatory restriction, when compared to dynamic contraction.

Exercise duration may affect peak muscle and blood lactate concentrations. After one to seven minutes of work to exhaustion muscle and blood lactate levels have been observed to be high (Tesch, 1978; Karlsson, 1971;

Karlsson & Saltin, 1970). Following the cessation of heavy exercise, however, blood lactate concentration continues to increase reaching a peak in approximately five minutes (Freund & Zouloumian, 1981; Gass et al., 1981; Hermansen & Vaage, 1977).

In contrast, during prolonged exercise (one to two hours) muscle and blood lactate concentrations, which initially increase, tend back to their resting values (Karlsson et al., 1974; Karlsson & Saltin, 1970; Astrand et al., 1963). In a series of four 30 second bouts of maximal isokinetic cycling with four minute recovery intervals McCartney et al. (1986) observed increases in muscle lactate concentration following the first and second work bouts to approximately 31 mmol kg^{-1} after which time it remained constant. Blood lactate increased until three minutes of recovery after the third work bout at which point it also plateaued between 21 and 23 mmol L^{-1} and remained constant for 10 minutes following the final work bout. These observations illustrate the point that lactate in active muscle is not in equilibrium with concentrations in the extracellular space and, consequently, blood.

For a given blood lactate concentration, lactate turnover can be two to four times greater during exercise than during rest (Mazzeo et al., 1986; Donovan & Brooks, 1983; Issekutz et al., 1976). This brings up the question of when to collect muscle and/or blood samples to best describe the process taking place. Mazzeo et al. (1986) states that because the steady-rate lactate levels represent a balance between production (entry into circulation) and removal (exit from circulation), it is apparent that measurement of blood lactate concentration alone is inadequate for the estimation of lactate production.

Training can alter lactate production in muscle. The increase in muscle and blood lactate concentration has been shown to be lower at the same absolute submaximal power production in trained subjects than untrained (Hermansen, 1971; Karlsson, 1971). Karlsson (1971) also observed significantly greater mean muscle lactate concentrations after dynamic exercise to exhaustion in trained as compared to untrained subjects. Sahlin & Henriksson (1984), however, found muscle lactate concentration to be 30% lower after isometric contraction to fatigue in trained as compared to sedentary subjects. They concluded that the lower lactate accumulation was related to the training, but suggested that the tendency towards a lower type I fibre percentage in the trained subjects likely contributed to the observed differences. Tesch (1978) has demonstrated lower lactate concentrations in type I as compared to type II muscle fibres following maximal contraction to exhaustion (18.7 and 25.8 mmol kg^{-1} wet muscle, respectively).

Endurance training reduces the accumulation of lactate in muscle and blood during exercise at a given absolute or relative work rate (Hollosy & Coyle, 1984; Sjodin et al., 1982; Wenger & MacNab, 1975; Karlsson et al., 1972). Karlsson et al. (1972) suggest this is due in part to a lower oxygen deficit and/or faster metabolism of the lactate produced. Brooks and Donovan (1983) showed that the effect of endurance training is on lactate clearance rather than lactate production since production rates were the same for trained or untrained.

LACTATE REMOVAL

During and after muscular exercise lactate is removed from muscle and blood as evidenced by the return to near resting values within 30 to 60 minutes after the cessation of exercise (Sahlin, 1978; Hermansen & Vaage, 1977; Hermansen & Osnes, 1972). The speed of removal can be enhanced by performing an active exercise recovery at low intensity (Dodd et al., 1984; Weltman et al., 1979; Issekutz et al., 1976; Hermansen & Stensvold, 1972). The primary mechanism for the accelerated removal of lactate induced by exercise is probably an increased blood flow through the muscle to facilitate oxidation within the muscle, increased efflux from the muscle, excretion via sweat and urine, transport to other tissues for oxidation or resynthesis to glucose and/or glycogen, or a combination of these factors (Gollnick et al., 1986; Fox & Mathews, 1981).

At present there is considerable debate with respect to the fate of lactate. One position argues that the predominant pathway is oxidation. Brooks (1986) suggests that approximately half of the lactate formed in the working muscle bed is released into the venous circulation while the other half as well as lactate removed from arterial circulation is combusted within the muscle and appears as CO_2 and H_2O. This was supported by Stanley et al. (1986) who concluded that extraction by working muscle during graded exercise is directly related to the arterial lactate concentration and metabolic rate.

Astrand et al. (1986), on the other hand, argue that approximately 50% of the lactate formed during heavy exercise is transformed into glycogen via

gluconeogenesis in active and perhaps inactive tissue during recovery. This position supports the earlier work of Hermansen and Vaage (1977) who suggest that only 10% of the generated lactate is released into the circulation. They conclude that the major fraction (75%) is converted to glycogen and less than 15% of the lactate disappearing in muscle during recovery seems to be oxidized to CO_2 and H_2O.

LACTATE EFFLUX

As previously mentioned, moderate to intense muscular exercise results in the production of lactate which accumulates in muscle and blood. At physiological pH an equimolar amount of hydrogen ion (H^+) is produced in association with lactate (Sahlin et al., 1976). During heavy exercise, marked differences between muscle and blood lactate levels have been observed demonstrating a concentration gradient from muscle to blood (Bergstrom et al., 1971; Karlsson, 1971).

Hermansen & Vaage (1977) have shown that disappearance of lactate from muscle after maximal exercise is rapid (mean = 0.74 mmol kg^{-1} min^{-1} wet weight corrected for change in muscle water content). Jorfeldt et al. (1978) observed a linear relationship between lactate release from muscle to blood of approximately 4 to 5 mmol min^{-1} leveling off after the fourth minute of recovery. It was felt that failure of release to keep up with production was not due to inadequate blood flow but rather a translocation hindrance.

These observations are consistent with the existence of a carrier transport system as the mechanism for movement of lactate across cell membranes (Sahlin et al., 1976; Bergstrom et al., 1971; Karlsson, 1971; Karpatkin et al., 1964). Furthermore, Jorfeldt et al. (1978) and Karlsson et al. (1972) have demonstrated a saturation of the translocation process where a further increase in muscle lactate did not increase the rate of lactate efflux.

Several investigators have reported that the rate of lactate efflux from muscle is influenced by the pH and bicarbonate concentration of the extracellular fluid or perfusing medium. Katz et al. (1984) and McCartney et al. (1983) showed markedly slowed rates of efflux with falls in arterial pH. Connett et al. (1986) point to the carrier as the pH sensitive component while Seo (1984) suggests that membrane permeability to lactate is what is affected by pH.

Mainwood et al. (1972) found that lactate efflux from frog sartorius muscles stimulated to fatigue was impaired at low external bicarbonate concentration (1 mmol L^{-1}). Similar results were obtained by Hirche et al. (1975) during stimulation of perfused isolated dog gastronemius muscle, when lactate efflux increased threefold during alkalosis compared to acidosis. In human subjects, decreasing the bicarbonate level will result in a lower plasma lactate concentration for a given level of exercise and a relatively higher muscle lactate concentration (Sutton et al., 1981) indicating a reduction in lactate efflux.

The kinetics of lactate transport from muscle to blood are at the moment poorly understood. As lactic acid at physiological pH is a strong acid (pKa = 3.73) it has been suggested that the dissociated molecule is the permeating

species. Jones (1980) stated that the two ions probably enter blood from muscle at different rates. This view is supported by the earlier work of Osnes & Hermansen (1972), Heisler (1973) and Sahlin et al. (1976). Benade & Heisler (1978), more specifically, demonstrated that the rate of H^+ efflux exceeded that of lactate in rat diaphragm and frog sartorius muscle by factors of 14 and 50, respectively.

In contrast to this view, other investigations indicate that the undissociated molecule might be the permeating species. Hirche et al. (1975), Mainwood & Worsley-Brown (1975), Roos (1975) and Mainwood et al. (1972) describe the same time course for lactate and H^+ efflux, suggesting that lactic acid dissociates only after it has reached the higher pH environment of the perfusing blood. This theory has the attraction of not requiring energy or ionic exchanges, placing the control of lactate distribution between the muscle cell and the extracellular space on the transmembrane pH gradient (Roos, 1975).

MUSCULAR FATIGUE

The ability to perform power activity depends largely on the capacity to tolerate, buffer and/or quickly remove high concentrations of H^+ produced within the working muscle (Sahlin & Henriksson,1984). The results of increased muscle and blood H^+ concentration are inhibition of phosphofructokinase (PFK); decreased membrane permeability; and competition of H^+ with calcium (Ca^{++}) for binding sites on actomyosin (Wenger & Reed, 1976). Collectively these factors inhibit glycolytic energy production (Kowalchuk et al., 1984; Sutton et al., 1981) and consequently force generation in the muscle (Tesch, 1980).

With respect to intramuscular processes, there is no general concensus to support the hypothesis that a depletion of energy stores causes a reduction in the force generated by muscle. However, there is experimental evidence which suggests that a decrease in intracellular pH, related to increased H^+ concentration associated with lactate production, can explain the observed force reduction during fatigue (Duchateau et al., 1987; Mainwood & Renaud, 1985). Furthermore, Duchateau et al. (1987) concluded that in both sustained and intermittent contractions the mechanical failure observed during fatigue was related to lactate production and the resulting intracellular pH drop. They suggested that mechanical failure controlled by the decrease in pH caused a change in the intracellular processes associated with tension development and subsequent relaxation. While Mainwood & Renaud (1985) support the hypothesis that intracellular acidosis accounts for part of the suppression of contractile force, they also suggest that external pH may play a role in the development of fatigue.

SUMMARY

The energy requirements during anaerobic exercise are supplied primarily by anaerobic glycolysis. Anaerobic glycolysis results in the production of lactic acid and a subsequent release of H^+ and decrease in pH. Any process able to remove H^+ from the working muscles will enhance the ability of that muscle to continue work. Sahlin & Henriksson (1984) and Katz et al. (1983) have shown that endurance trained athletes demonstrate lower muscle lactate and higher pH values compared to non-endurance trained controls following maximal exercise to exhaustion. However, while a relationship between some of the effects of endurance training and enhanced removal of metabolites has been inferred (Sahlin & Henriksson, 1984; Katz et al., 1983; Wenger, 1981) a direct link has not been demonstrated.

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