RELIABILITY AND CIRCADIAN RHYTHMICITY OF BLOOD LACTATE

By

Paula McFadyen
B.Sc. (Human Performance) University of Victoria

A Thesis Submitted in Partial Fulfilment of
the Requirements for the Degree of

MASTER OF SCIENCE

in the School of Physical Education

ACCEPTED
[F]ULTY OF GRADUATE STUDIES
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[DA]TE 92/04/28

We accept this thesis as conforming
to the required standard

Dr. H.A. Wenger, Supervisor

Dr. D. Docherty, Departmental Committee Member

Dr. J.E. Petersen, External Committee Member

Dr. R. Backus, Additional Committee Member

Dr. J.G. Wilkinson, External Examiner

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University of Victoria

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Supervisor: Dr. H. A. Wenger

ABSTRACT

The reliability in measurement of, the circadian variation in, and the effects of catecholamine concentration on venous and arterialized lactate during rest and exercise were examined in twelve trained males (26.4 ± 5.4 yrs, 76.7 ± 8.5 kg, 180.5 ± 8.5 cm, and 58.1 ± 4.6 $ml{\cdot}kg^{-1}{\cdot}min^{-1}$). The subjects completed four cycle tests each spaced three days apart. Two tests were performed between 0700-0830 h, one at 1200-1330 h and one at 1700-1830 h. To determine blood lactate reliability, an additional 11 male subjects were tested twice between 0700-0830 h. Baseline measures of heart rate (HR), core temperature (T_c), ventilation ($\dot{V}_E$), and oxygen consumption ($\dot{V}O_2$) were recorded during minutes 25-30 of rest. Venous blood was drawn from the antecubital vein and was subsequently analyzed for plasma catecholamine and blood lactate concentration. During exercise, power output increased 20-45 W every 3 minutes until ventilatory threshold (VT) and every minute until the criterion endpoint was reached (CEP). Criterion endpoint was determined to be 20-45 W below the power output at which peak $\dot{V}O_2$ was reached. All of the cardiorespiratory variables and T_c were measured every minute during exercise and venous and arterialized blood samples were taken at VT and 3 minutes post-CEP. A circadian rhythm was demonstrated in T_c, HR, $\dot{V}O_2$, and $\dot{V}_E$ at rest with the peak measurement occurring in the evening but only T_c demonstrated a discernable rhythm at VT and CEP. Blood lactate did not demonstrate a circadian rhythm under any of the three measurement conditions. Reliability coefficient for both venous and arterialized lactate were R=0.75 and R=0.81 at VT and were R=0.90 and R=0.90 at CEP respectively. The standard error of measurement (SEM) for venous lactate at the same time of day during exercise ranged between 0.3-0.4 $mmol{\cdot}L^{-1}$ and the SEM increased when blood lactate concentration was compared at different times of the day. When blood lactate was sampled from different sites, venous lactate concentration was approximately 1 $mmol{\cdot}L^{-1}$ lower than arterialized

lactate concentration at VT and CEP. Changes in venous lactate concentration from rest to VT were correlated with changes in morning plasma epinephrine (r=0.81). The findings of this study suggest that blood lactate measurement is reliable during exercise when sampled during the morning; that differences across sampling sites and concentration of plasma epinephrine affect blood lactate concentration; and during exercise, no discernable circadian changes in cardiorespiratory variables, catecholamines, and blood lactate were apparent at three different sampling times. Eventhough circadian rhythmicity was not a confounding factor in blood lactate concentration during exercise, the low correlations and the large SEM across times suggest that sampling time should be standardized.

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ACKNOWLEDGEMENTS

I wish to thank Dr. Howard Wenger for his inspiration, encouragement and guidance throughout my degree. His humour, open door policy, and friendship were greatly appreciated. Many thanks to my advisory committee members, Dr. D. Docherty, Dr. R. Backus, and Dr. J. Petersen, for their interest and input into my study.

I would like to acknowledge the participation and dedication of all of the subjects in my study as well as the assistance of Deborah Bachand, Wendy Pethick, P.J. Naylor, Rachel Parfitt, and Steve MacKenzie in data collection. Their time commitment during the early morning hours and late evening was appreciated. A special thanks to Roger McLeod, from Shaughnessy Hospital, for his assistance in analyzing the catecholamines.

Many thanks to Lynneth Wolski, my thesis partner in crime; to my other fellow graduate students for their humour and friendship; and to Gladys Whittal for her administration wizardry and friendly reminders of important deadlines. Special acknowledgement goes to my husband, Ross, and my family for their faith, encouragement, and support these past two years. I know they will be glad to finally see the finished product.

Finally, I would like to acknowledge that this study was supported by a grant from the University of Victoria.

DEDICATION

To my husband, Ross, for keeping the waters calm in times of panic and to my late Grandfather, Emilio Maralia, for his words of wisdom "if you are going to do something, do it right or don't do it at all".

CHAPTER 1: INTRODUCTION

Circadian rhythms, which are controlled by endogenous pacemakers and exogenous zeitgebers such as light and darkness, oscillate and reach minimal and maximal levels within a 24 h period (Mills, 1966; Moore-Ede, 1983; Shephard, 1984). Human circadian rhythms have been demonstrated in several physiological and behavioral variables at rest and during exercise. When milliseconds or fractions of a centimetre separate finalists, circadian variation of cardiorespiratory, neuromuscular, metabolic, and psychomotor functions may affect performance (Eichner, 1988).

Performance in track, rowing, and swimming events have demonstrated circadian rhythmicity with peak performance occurring in the evening (Baxter & Reilly, 1983; Conroy & O'Brien, 1974; Reilly & Brooks, 1986). Similarly, circadian rhythmicity of other variables such as heart rate (HR) (Crockford & Davies, 1969; Reilly & Brooks, 1982), core temperature (T_c)(Cable & Reilly, 1987; Cohen & Muehl, 1977), oxygen consumption ($\dot{V}O_2$) (Cable & Reilly, 1987; Reilly & Brooks, 1982), ventilation ($\dot{V}_E$)(Cable & Reilly, 1987; Reilly, 1982), and rate of perceived exertion (Faria & Drummond, 1982; Ilmarinen, Ilmarinen, Korhonen, & Nurminen, 1980) reach their crest in the evening. However, discernable circadian rhythms are not always apparent for HR, $\dot{V}O_2$, and $\dot{V}_E$ near maximal levels of exercise (Faria & Drummond, 1982; Ilmarinen et al., 1980).

Another variable measured during exercise to evaluate or to predict athletic performance is blood lactate. Reviews on circadian rhythmicity (Winget, DeRoshia, & Holley, 1985; Reilly, 1985) do not report data on the circadian variation of blood lactate but one study found that blood lactate increased significantly throughout the day (Reilly and Baxter, 1983). However, the 21% increase in blood lactate in the evening could not be interpreted as a circadian rhythm due to the associated 41% increase in power output. Further research is required to determine if blood lactate does demonstrate circadian rhythmicity during exercise and the implications that circadian oscillations may have on sampling time and data interpretation.

To interpret daily variation or training adaptations across trials at lactate threshold (LT) and at a fixed blood lactate concentration, high reliability of blood lactate is critical. A previous study determined the reliability of venous lactate at anaerobic threshold to be r=0.93 (Davis, Vodak, Wilmore, Vodak, & Kurtz, 1976). Most studies have used ventilatory gas exchange variables to determine threshold reliability, but the consistency of venous, capillary and arterialized blood across days has not been established.

Blood lactate concentration is determined by the differences between production and release from muscle and the uptake and removal by various tissues (Brooks, 1991; Stainsby & Brooks, 1990). This process is very complex and as a result, there are differences in blood lactate concentration depending on the sampling site and fraction of blood analyzed. Foxdal et al. (1990) reported arterial lactate concentration to be 8% higher in comparison to venous lactate concentration. For ease of sampling and safety during exercise, blood is drawn from either venous, capillary, or arterialized sites in the arm, hand, or earlobe. The differences in blood lactate concentration between these sites has not been extensively studied.

Blood lactate concentration is dependent, in part, on lactate production, and since glycolytic flux is directly altered by catecholamine concentration, it has been suggested that blood lactate concentration will increase in proportion with elevated catecholamine levels (Stainsby & Brooks, 1990). Increases in plasma epinephrine concentration during exercise have been correlated to blood lactate appearance (r=0.97) during submaximal exercise (Gregg, Mazzeo, Budinger, & Brooks, 1989). Research by others have produced similar results (Mazzeo and Marshall, 1989; Stainsby, Sumners, Eitzman, 1985; Stainsby, Sumners, & Andrews, 1984). However, catecholamine infusion and blockade studies indicate that changes in norepinephrine were not correlated to changes in blood lactate but were indicative of sympathetic neural activity (Chasiotis, Sahlin, & Hultman, 1983; Stainsby & Brooks, 1990). Catecholamines demonstrate a crest in circadian rhythms in the early afternoon and perhaps oscillations in plasma epinephrine may be mirrored by similar changes in blood lactate.

RESEARCH QUESTIONS

Therefore, this study examined the changes in blood lactate, cardiorespiratory variables, core temperature, and catecholamines at rest, at ventilatory threshold (VT), and at maximal power outputs (CEP) throughout a day, to determine:

1) whether core temperature, cardiorespiratory variables, blood lactate, and plasma catecholamines demonstrate circadian rhythmicity?
2) the reliability of venous and arterialized lactate measurements.
3) whether there are differences in blood lactate concentration across different sampling sites?
4) whether changes in blood lactate are correlated to changes in plasma catecholamines?

Definitions of Terms:

Circadian: comes from the latin word 'circa dies' which means about a day. It represents a cycle or rhythm with maximum and minimum fluctuations during a 24 ± 4 h period that governs the physiological and psychological systems of the body (Winget et al., 1985). In the present study, a 13 h data collection period was chosen to represent the time of day in which competitions and training would take place. The term 'circadian' was used because the 13 h recording period represented the maximum and minimum oscillatory times for core temperature.

Trained: for the purposes of the present study, this term describes males who regularly participate in exercise four or more times per week, are accustomed to vigorous exercise, and have a peak $\dot{V}O_2$ higher than 50 $ml{\cdot}kg^{-1}{\cdot}min^{-1}$.

Criterion Endpoint: 20-45 W below the power output at which peak $\dot{V}O_2$ was reached.

CHAPTER 2: RESEARCH METHODS

2.1 Subjects

Twelve healthy trained males, 26.4 ± 5.4 years, 76.7 ± 8.5 kg, 180.1 ± 8.5 cm, and peak $\dot{V}O_2$ of 58.1 ± 4.6 $ml \cdot kg^{-1} \cdot min^{-1}$ (Mean ± SD) volunteered to participate in this study. All subjects were pre-screened based on medical history and training background. Prior to testing, each subject was informed of the procedures, equipment, and nature of the study before giving written consent (Appendix A). To determine the reliability of blood lactate, an additional eleven males were tested. Their physical characteristics were not significantly different from the group above. When referring to the reliability of lactate measurements, an n=23 was used compared to an n=12 for the other sections of this study.

2.2 Experimental Design

The subjects were randomly divided into two groups: Group A was tested on Monday and Thursday and Group B was tested on Tuesday and Friday. Each subject completed four cycle tests in two weeks: two in the morning (0700-0830 h), and one in the afternoon (1200-1330 h) and evening (1700-1830 h) (Appendix B). Each testing session lasted approximately one hour for each subject. Twenty four hours before each test, no exercise was performed and a carbohydrate (CHO) rich diet (5 g CHO/kg) was prescribed based on individual body weight (Appendix C). All food intake was recorded and points were totalled to ensure that CHO intake was standardized across trials. A snack consisting of 20g of CHO was consumed 2 h prior to each test. These procedures were employed to ensure that muscle glycogen stores were replenished for each testing session.

2.3 Resting State

The subjects arrived at the laboratory 30 minutes prior to starting their cycle test. They were weighed on a calibrated scale, an indwelling catheter (Insight 20" x 1.25g) was inserted into a vein in the antecubital space by an IV therapist, a disposable rectal probe was inserted (10cm), and a Sport Tester HR monitor (PE 3000) was attached across the mid-sternum region. The subjects sat quietly for 25 minutes to establish baseline values and recordings were made from minutes 25-30. Resting values for HR, T_c, and expired gases were recorded every minute. A resting venous blood sample was drawn during the last 15s of the rest period.

2.4 Cycle Protocol

Prior to starting the study, peak $\dot{V}O_2$ was determined using a step-wise incremental protocol on a friction braked Monark cycle ergometer (Model 818E). Based on each peak $\dot{V}O_2$ test, VT and criterion endpoint (CEP) were determined. VT was identified by a significant change in $\dot{V}_E/\dot{V}O_2$ with no change in $\dot{V}_E/\dot{V}CO_2$, non-linear increase in $\dot{V}_E$, and an RER > 1.0 (Wasserman, Whipp, Koyal, & Beaver, 1973). To ensure each subject could complete all four tests, the CEP was set 20-45 W below the power output at which peak $\dot{V}O_2$ was attained. In each test, toeclips were secured and each subject cycled at a specific number of revolutions per minute (75-90 rpm) depending on their cycling ability. Power output was initially set at 150-175 W and increased 20-45 W every 3 minutes until VT was reached. Then, power output was increased 20-45 W every minute until peak $\dot{V}O_2$ or CEP was attained. The peak $\dot{V}O_2$ test was terminated when two of the following criteria were attained: O_2 plateaued with an increase in power output, subjects stopped due to volitional exhaustion, or RER exceeded 1.15 (Thoden, 1991).

Expired gases were analyzed by a Beckman Metabolic Measurement cart (MMC) every 30 s. Prior to and after each test, the MMC was calibrated against primary gas standards. Temperature, volume, and barometric pressure were calibrated before each testing session. Throughout the test, HR and T_c were recorded every minute.

2.5 Blood Analysis

Both venous and arterialized samples were simultaneously drawn during the last 15s of the 3 minute workload at VT and 3 minutes post-CEP. Arterialized blood samples (100uL) were taken from the fingertip of a warmed hand at VT and 3 minutes post-CEP and immediately analyzed in duplicate for blood lactate concentration by a Yellow Springs Instrument lactate analyzer (model #23L). One ml of venous blood was drawn into a syringe and a 100uL was stored in heparinized capillary tubes prior to lactate analysis.

Catecholamine sampling occurred during the second morning and the afternoon testing sessions. Seven mls of venous blood was collected into a syringe and placed immediately into a chilled vacutainer containing EDTA. The blood was centrifuged for 15 minutes at 3000 rpm and the plasma was pipetted and frozen at -70°C. Then, the plasma was stored in liquid nitrogen and analyzed in duplicate in a hospital laboratory within four weeks of collection. High performance liquid chromatography with electrochemical detection procedures were used to determine plasma catecholamine concentration (Bouloux, Perrett & Besser, 1985).

2.6 Statistical Analysis

All data were statistically analyzed using SPSS-PC program (version 4.01). Circadian rhythms of $\dot{V}O_2$, T_c, HR, and $\dot{V}_E$, were analyzed using a one-way analysis of variance (ANOVA) with repeated measures. Circadian rhythmicity of blood lactate (0700, 1200, and 1700 h) and differences between sampling sites were analyzed using a 3x2 factorial ANOVA with repeated measures on both factors. When significant F ratios occurred throughout a day, Neuman-Keuls post-hoc test was employed. Significance levels were set at $p<0.05$.

Pearson Product Moment correlations were used to determine the relationship of blood lactate changes throughout a day and across sampling sites. Similarly, the change scores from rest to VT for venous lactate were correlated to the change scores of epinephrine and norepinephrine. Significance levels were set at $p<0.01$. Intraclass correlation coefficients were calculated to

determine the reliability of venous and arterialized lactate across morning testing sessions under all three measurement conditions. No sample distribution tables are available to determine the level of significance for intraclass correlations. The standard error of measurement (SEM) was calculated across days at the same time and throughout a day to quantify the error expected in any score.

CHAPTER 3: RESULTS

Do core temperature, heart rate, oxygen consumption, and ventilation demonstrate a circadian rhythm at rest, at VT, or at CEP?

Circadian rhythmicity was demonstrated in T_c at rest, at VT, and at CEP. Under all conditions, T_c was lowest in the morning (36.8°C) and increased significantly 0.4-0.5°C in the afternoon (37.3°C). From the afternoon to evening session, there was no significant increase in T_c (37.4°C) (Table 1).

HR, $\dot{V}O_2$, and $\dot{V}_E$, demonstrated circadian rhythmicity at rest with a difference of 6.6 $b \cdot min^{-1}$, 0.8 $ml \cdot kg^{-1} \cdot min^{-1}$, and 1.8 $l \cdot min^{-1}$ respectively between 0700 and 1700 h, but no significant circadian differences occurred at VT or at CEP (Table 1). The comparison of correlations and standard error of measurement across sampling times for T_c and cardiorespiratory variables are reported in Appendix D.

What is the reliability of venous and arterialized lactate concentration at rest and during exercise?

Mean blood lactate concentration measured on two different days at rest, VT, and CEP were not significantly different for either venous or arterialized samples (Table 2). During exercise, Pearson product moment correlations were significant and intraclass correlations ranged between 0.75-0.90. The SEM at VT and CEP for venous concentration were 0.3 $mmol \cdot L^{-1}$ and 0.4 $mmol \cdot L^{-1}$ and 0.7 $mmol \cdot L^{-1}$ and 0.4 $mmol \cdot L^{-1}$ for arterialized samples respectively. Venous lactate correlations at rest were non-significant and SEM was 0.2 $mmol \cdot L^{-1}$.

Does venous or arterialized blood lactate demonstrate circadian rhythmicity at rest, at VT, or at CEP?

Neither venous nor arterialized blood lactate demonstrated circadian rhythmicity at rest, at VT, or at CEP (Table 3). All means were similar throughout the day under each measurement condition and no significant correlations occurred throughout the day except at VT between 1200 and 1700 h for arterialized samples (Table 4). The SEM of blood lactate concentration was higher when compared across times in comparison to samples taken at the same time of day.

Are there differences between venous and arterialized lactate concentration at VT and CEP?

Arterialized and venous samples were significantly different when measured at the same time of day and under standardized conditions. At VT and CEP, venous lactate was approximately 1 $mmol{\cdot}L^{-1}$ lower than arterialized lactate concentration. Throughout a day (0700, 1200, & 1700 h), the percentage differences between venous and arterialized blood lactate were 35 ± 8%, 25 ± 9%, and 42 ± 17% at VT and 13 ± 6%, 14 ± 7%, and 14 ± 7% at CEP respectively. The venous and arterialized samples were not significantly correlated except in the afternoon at VT (r=0.96) and evening at CEP (r=0.72) (Table 5).

Does plasma epinephrine and plasma norepinephrine demonstrate a circadian rhythm at rest and at VT?

During both testing sessions, plasma epinephrine and plasma norepinephrine increased significantly from rest to VT but no circadian rhythmicity was demonstrated (Table 6). Across morning and afternoon testing sessions, resting plasma epinephrine correlations were r=0.53 and r=0.38 at VT and plasma norepinephrine correlations were r=-0.12 and r=-0.39 respectively.

What is the correlation between changes in venous lactate and changes in plasma epinephrine and plasma norepinephrine from rest to VT?

The change score for plasma epinephrine from rest to VT was significantly correlated to the change score of venous lactate in the morning (r=0.83) but the same relationship was not demonstrated in the afternoon (r=0.51). Under either sampling condition, norepinephrine change scores were not significantly correlated with venous lactate (r=0.29 and r=0.06 respectively)

Table 1

Mean (SD) core temperature, heart rate, oxygen consumption and ventilation measured throughout a day at rest and at two different exercise intensities (n=12).

	Morning 0700-0830 h	Afternoon 1200-1330 h	Evening 1700-1830 h
Core Temperature (°C)			
Rest	36.6 (0.2)[ab]	37.0 (0.2)	37.2 (0.2)
VT	36.8 (0.3)[ab]	37.2 (0.2)	37.3 (0.2)
CEP	37.0 (0.2)[ab]	37.5 (0.2)	37.6 (0.2)
Heart Rate ($b \cdot min^{-1}$)			
Rest	54.7 (4.6)[ab]	59.4 (7.4)	61.3 (7.9)
VT	155.1 (9.0)	156.1 (10.3)	157.1 (11.1)
CEP	182.0 (5.8)	184.5 (5.6)	186.0 (8.9)
Oxygen Consumption ($ml \cdot kg^{-1} \cdot min^{-1}$)			
Rest	4.0 (0.2)[ab]	4.7 (0.6)	4.8 (0.6)
VT	39.3 (4.2)	39.7 (5.4)	40.9 (5.1)
CEP	54.0 (6.0)	54.8 (6.1)	57.1 (5.8)
Ventilation ($l \cdot min^{-1}$)			
Rest	9.9 (1.5)[ab]	11.7 (1.8)	11.8 (1.8)
VT	76.8 (8.7)	78.5 (6.9)	80.5 (9.9)
CEP	143.7 (18.1)	142.7 (16.3)	149.8 (19.8)

Rest = resting values were recorded after sitting quietly for 30 minutes.
VT = ventilatory threshold
CEP = criterion endpoint (20-45 W below the power output at which peak $\dot{V}O_2$ was reached).
a = morning is significantly different ($p<0.05$) than afternoon
b = morning is significantly different ($p<0.05$) than evening

Table 2

Means (SD), correlations, and standard error of measurement for blood lactate concentration measured across days under standardized conditions (n=23).

	Venous Blood Lactate		Arterialized Blood Lactate	
	Day 1	Day 2	Day 1	Day 2
Rest				
Mean (mmol·L^{-1})	0.6 (0.2)	0.6 (0.3)		
r	0.29			
R	0.49			
SEM (mmol·L^{-1})	0.2			
VT				
Mean (mmol·L^{-1})	1.8 (0.7)	1.7 (0.6)	3.0 (1.2)	2.7 (1.7)
r	0.59*		0.70*	
R	0.75		0.81	
SEM (mmol·L^{-1})	0.3		0.7	
CEP				
Mean (mmol·L^{-1})	7.2 (1.0)	7.4 (1.6)	8.0 (0.9)	8.0 (1.5)
r	0.82*		0.83*	
R	0.90		0.90	
SEM (mmol·L^{-1})	0.4		0.4	

Rest = resting values were recorded after sitting quietly for 30 minutes.
VT = ventilatory threshold
CEP = criterion endpoint (20-45 W below the power output at which peak $\dot{V}O_2$ was reached).
r = Pearson's Product Moment Correlation coefficient
R = Intraclass reliability coefficient
SEM = standard error of measurement
* = significance at $p<0.01$

Table 3

Mean (SD) venous and arterialized blood lactate concentration measured throughout a day under three different measurement conditions (n=12).

	Morning 0700-0830 h	Afternoon 1200-1330 h	Evening 1700-1830 h
Venous lactate ($mmol \cdot L^{-1}$)			
Rest	0.6 (0.2)	0.6 (0.3)	0.5 (0.2)
VT	1.8 (0.7)	1.9 (0.5)	1.7 (0.8)
CEP	7.2 (1.0)	6.8 (0.7)	7.2 (1.3)
Arterialized lactate ($mmol \cdot L^{-1}$)			
VT	3.0 (1.2)	2.6 (0.8)	2.9 (0.7)
CEP	8.0 (0.9)	8.0 (0.7)	8.0 (0.8)

Rest = resting values were recorded after sitting quietly for 30 minutes.
VT = ventilatory threshold
CEP = criterion endpoint (20-45 W below the power output at which peak $\dot{V}O_2$ was reached).

Table 4

Venous and arterialized lactate correlations and standard error of the measurement across sampling times.

	r	SEM	r	SEM
Rest (n=12)				
	0700-V		1200-V	
0700-V				
1200-V	-0.28	0.2		
1700-V	-0.23	0.2	0.64	0.2
VT (n=23)				
	0700-V 0700-A		1200-V 1200-A	
0700-V 0700-A				
1200-V 1200-A	0.51 0.18	0.5 0.7		
1700-V 1700-A	0.52 0.20	0.5 0.7	0.70 0.84*	0.3 0.3
CEP (n=23)				
	0700-V 0700-A		1200-V 1200-A	
0700-V 0700-A				
1200-V 1200-A	-0.07 0.32	0.9 0.7		
1700-V 1700-A	0.16 0.52	1.1 0.6	-0.15 0.49	1.1 0.8

V = venous lactate concentration
A = arterialized lactate concentration
SEM = standard error of the measurement is expressed in $mmol \cdot L^{-1}$
Rest=resting values were recorded after 30 minutes of sitting quietly
VT = ventilatory threshold
CEP= criterion endpoint (20-45 W below the power output at which peak $\dot{V}O_2$ was reached).
* = significance at $p<0.01$

Table 5

Venous and arterialized lactate correlations across sampling sites (n=12).

	0700V-A	1200V-A	1700V-A
VT	0.56	0.96*	0.62
CEP	0.53	0.61	0.72*

V = venous lactate concentration
A = arterialized lactate concentration
VT= ventilatory threshold
CEP= criterion endpoint (20-45 W below the power output reached at peak $\dot{V}O_2$).
* = significance at $p<0.01$

Table 6

Mean (SD) of plasma epinephrine and plasma norepinephrine measured in the morning and afternoon at rest and VT (n=10).

	Morning 0700-0830 h	Afternoon 1200-1330 h
Epinephrine ($nmol \cdot L^{-1}$)		
Rest	0.34 (0.18)	0.45 (0.20)
VT	1.02 (0.58)	1.13 (0.53)
Norepinephrine ($nmol \cdot L^{-1}$)		
Rest	3.08 (2.18)	2.65 (2.45)
VT	5.81 (1.54)	6.63 (2.12)

Rest= resting values were recorded after sitting quietly for 30 minutes
VT = ventilatory threshold

CHAPTER 4: DISCUSSION

During rest and exercise, the present study investigated: a) the circadian rhythmicity of cardiorespiratory variables, blood lactate, and catecholamines, b) the reliability of blood lactate measurements, c) differences between arterialized and venous lactate concentration, and d) the relationship between changes in blood lactate and plasma catecholamines.

4.1 Circadian Rhythmicity of Control Variables

Resting circadian oscillations have been demonstrated in many physiological variables that may influence athletic performance such as T_c, HR, $\dot{V}_E$, and $\dot{V}O_2$, but during both submaximal and maximal exercise, conflicting results have been reported. T_c has demonstrated a discernable circadian rhythm throughout a day reaching the lowest level at approximately 0600 h and maximum at approximately 1700 h (Cable & Reilly, 1987; Faria & Drummond, 1982; Winget, DeRoshia, Vernikos-Danellis, Rosenblatt, & Hetherington, 1977). In the present study, T_c demonstrated a significant circadian rhythm under all three measurement conditions. Minimum values were reached between 0700-0830 h and maximum values between 1700-1830 h. No significant difference occurred between afternoon and evening values.

Moore-Ede (1983) hypothesized that the control centre for T_c is the X pacemaker which is located in the ventromedian nucleus or lateral hypothalamic area. This pacemaker regulates T_c as well as rapid eye movement, urinary potassium secretion, and plasma cortisol. The other pacemaker (Y) controls the rest-activity cycle and is located in the suprachiasmatic nuclei. It regulates skin temperature, plasma growth hormone, urinary calcium secretion, and circadian timing of slow-wave sleep (Moore-Ede, 1983). Both pacemakers differ in their oscillatory strength with the X pacemaker being approximately 12 fold stronger than the Y pacemaker. The Y pacemaker is affected more by exogenous control of zeitgebers (Aschoff & Wever, 1976).

T_c is more stable during the early morning hours and least stable during 1300-2100 h due to

a progressive decline in strength of the light zeitgeber (Winget et al., 1977). Over a 12 h recording period, the average change of T_c at rest and during exercise is 0.5°C (Moore-Ede, Czeisler, & Richardson, 1983) which corresponded with the findings of this study (Table 1).

Other physiological variables such as $\dot{V}_E$, $\dot{V}O_2$, and HR demonstrated circadian rhythmicity at rest with the crest occurring in the evening but during exercise, no discernable rhythm was detected. The means for all three variables increased throughout the day but were not significantly different at VT or CEP. Perhaps the minimum values were not attained within the time restrictions of the study and therefore the difference between 0700-1830 h means was not large enough to demonstrate a statistically discernable rhythm. Many studies have examined the circadian rhythms of these three variables at rest and during exercise, but conflicting results have occurred because of low subject number (Faria & Drummond, 1982; Reilly, 1982), different modes of exercise (Faria & Drummond, 1982; Reilly & Brooks, 1982), varied exercise intensity, and frequency and timing of measurement (Cable & Reilly, 1987; Reilly, Robinson, & Minors, 1984). Large individual variance within a group may also mask the between hour variance (Conroy & Mills, 1970). In the present study, the large individual variance of HR, $\dot{V}O_2$, and $\dot{V}_E$ may have dampened the circadian differences of these variables. Similarly, output from the medulla oblongata, which governs cardiovascular responses to exercise, may override the circadian rhythms of these three variables (Moore-Ede, 1983). Based on the above findings, when testing under standardized conditions, the time of day is not a critical factor to consider when measuring HR, $\dot{V}O_2$, and $\dot{V}_E$.

HR cyclic variation has been shown to precede changes in T_c with the trough occurring between 0200-0500 h and the crest occurring at approximately 1600 h (Davies & Sargeant, 1975; Yamaji, Sakamoto, Nakaguchi, Kitamura, & Shephard, 1981). Fluctuations in sympathetic drive to the heart associated with T_c and a biological clock localized in the heart have been suggested as control regulators (Malpas & Purdie, 1990; Reilly et al., 1984).

$\dot{V}_E$ and $\dot{V}O_2$ have demonstrated circadian rhythms at rest and during submaximal exercise with the trough occurring between 0200-0600 h and the crest peaking at 1400-1700 h (Cable & Reilly,

1987; Davies & Sargeant, 1975; Reilly, 1982). However, as exercise intensity increases, $\dot{V}O_2$ continues to remain stable throughout a day and does not demonstrate circadian oscillations at $\dot{V}O_2max$ (Davies & Sargeant, 1975; Faria & Drummond, 1982) as demonstrated in the present study. Reilly (1982) hypothesized that the periodicity of $\dot{V}_E$ may be partially independent of the oscillator linking T_c and metabolic rate. Further investigation is required to determine the conditions under which conditions, if any, $\dot{V}O_2$ and $\dot{V}_E$ demonstrate circadian rhythmicity and which pacemaker governs their rhythmical control.

4.2 Reliability of Blood Lactate

When assessing or predicting athletic performance, high reliability of blood lactates across repeated trials is critical. However the reliability of LT or fixed blood lactate concentration during exercise has not been researched extensively. Weltman et al. (1990) found Pearson Product Moment correlation coefficients to be greater than 0.85 for HR and $\dot{V}O_2$ at LT, 2, 2.5, 4 mmol·L^{-1}, and at peak lactate. Similarly, Davis et al. (1976) found the reliability of the anaerobic threshold (AnT), as determined by the lactate break point, to be 0.93. However, these two studies used Pearson Product Moment correlation which is a bivariate statistic that does not reflect changes in the means and variance. To determine the reliability of any variable, univariate statistics such as intraclass correlations should be used (Kroll, 1962).

In the present study, both venous and arterialized lactate measurements were reliable at VT and CEP when measured under standardized dietary and exercise conditions across days. Reliability coefficients were high and SEM was low (Table 2). Therefore if an exercise test was repeated in the morning across days, a change of 0.3-0.4 mmol·L^{-1} would be within the range of expected error 68% of the time for venous samples. Further investigation is required to quantify the metabolic changes that occur after training in order to differentiate between training adaptations and day to day variation. Most training studies have quantified changes in $\dot{V}O_2$, HR, and power output at fixed lactate concentrations (Hurley et al., 1984) but the absolute change in lactate

concentration after a training regimen has not been quantified.

Even though carbohydrate consumption, meal times, and exercise 24 h prior to each test were standardized, the reliability of resting venous lactate was lower than during exercise (R=0.49). The low resting correlation could have been influenced by a smaller n, the small range of resting blood lactate values (0.2-1.2 mmol·L^{-1}) (Table 2), and changes in circadian rhythmicity of metabolites, hormones, and cardiorespiratory variables. It has also been suggested that at low lactate concentrations, low signal to noise ratios and low peroxide production in the YSI analyzers may contribute to the low reliability (Bishop, Smith, Kime, Mayo, & Tin, 1992).

4.3 Circadian Rhythmicity of Blood Lactate

When investigating the metabolic effects of different training regimens or predicting endurance performance, blood lactate is often used as a dependent variable (Jacobs, 1986). Therefore, other factors which affect blood lactate concentration must be both understood and controlled. Factors such as diet, acid-base balance, and hypoxia and their effect on blood lactate concentration have been studied extensively (Gollnick, Bayly, & Hodgson, 1986). However, the effect that circadian rhythmicity may have on blood lactate concentration has not been examined and was a focus of this study.

The results demonstrated that no discernable rhythm was detected in either venous or arterialized blood from morning, to afternoon, to evening. Mean blood lactates at rest, at VT, and at CEP were similar throughout the day (Table 3) and all correlations were low, except at VT for arterialized blood across 1200 and 1700 h (Table 4). These results suggest that blood lactate does not demonstrate circadian rhythmicity in physically fit males. However, when lactate concentration was compared at different times of the day, the low correlations and higher SEM suggest that sampling time should be standardized at the same time of day to minimize biological variation and other factors not controlled or measured in this study.

To date, only one other study has examined the circadian rhythmicity of blood lactate. Reilly

and Baxter (1983) measured changes in blood lactate from 0630-2200 h during exercise. They found a 21% increase in blood lactate from morning to evening samples but the significant increase in blood lactate, in association with a 41% increase in power output, could not be interpreted as a circadian rhythm .

Lactate appearance in the blood is both a function of production in skeletal muscle, liver, skin, and intestine and of removal in active and inactive skeletal muscle, heart, liver, and kidneys (Stainsby & Brooks, 1990). The appearance of blood lactate is also regulated by a variety of other factors. These include the hormonal action of catecholamines which stimulate muscle glycogenolysis and glycolysis (Mazzeo & Marshall, 1989; Stainsby et al., 1985), sarcolemmal transporters (Roth, 1991), as well as substrate availability of glucose and glycogen (Gollnick, Pernow, Essen, Jansson, & Saltin, 1981). Lactate transporters have been established in erythrocytes, intestinal brush border cells (Hildeman, Storelli, Haase, Barac-Nieto & Murer, 1980), and skeletal muscle (Brooks & Roth, 1989; Roth & Brooks, 1990). The sarcolemmal transporters in skeletal muscle are affected by lactate concentration and proton gradients in muscle tissue.

The formation of lactate in muscle, tissue exchange, consumption, and utilization is a complex process. The rate of production may be influenced by the circadian rhythmicity of regulatory hormones and enzymes, but the removal process, via passive and facilitated diffusion, may mask any circadian rhythms. The regulatory control that each factor exerts on lactate formation is not known. Consequently, blood lactate accumulation may be affected by such a wide variety of factors that it may be difficult to demonstrate a discernable circadian rhythm during exercise. If circadian rhythmicity of blood lactate does exist, then it is hypothesized that it would coincide with the peak and trough of T_c, catecholamines, metabolic rate, cardiorespiratory variables and athletic performance.

Blood lactate was only sampled at three times throughout the day. Further research is required to determine if circadian rhythmicity of blood lactate exists outside these given time limits. The times chosen in this study represented training and competition hours during which blood lactate

would likely be sampled and the results indicate that circadian rhythmicity does not confound blood lactate concentration. However, measurements should be taken at the same time of day to minimize biological variation.

4.4 Venous and Arterialized Lactate Concentration Differences

Various sampling sites and methods of analysis are used to determine the inflection point of blood lactate (OBLA, OPLA, 4 mmol·L^{-1}, IAT, and LT). The findings of this study indicated that arterialized lactate concentration is significantly higher ($p<0.01$) than venous lactate concentration at VT and CEP when measured in the same limb. This finding is supported by Foxdal, Sjodin, Ostman & Sjodin (1991), Foxdal et al. (1990), Robergs et al. (1990), and Yoshida, Takeuchi, and Suda (1982).

At rest, venous and arterialized samples are not significantly different but as exercise intensity increases, arterial lactate concentration rises significantly higher than venous lactate levels (Yoshida et al., 1982). The percentage differences may result from an increase in lactate uptake by both inactive and active skeletal muscles, heart, and liver, as well as a redistribution of lactate in the arm and between intravascular and extravascular compartments (Foxdal et al., 1990; Robergs et al., 1990).

The onset of arterial lactate accumulation occurs earlier than venous lactate accumulation and these changes coincide with a fall in pH and bicarbonate in the arterial system. Similar changes were not evident in the venous system (Yoshida, et al., 1982). Robergs et al. (1990) examined blood lactate concentration at VT, 350 W, OBLA, and $\dot{V}O_2max$ and found arterialized lactate, from a hyperemized earlobe, to be significantly higher than venous lactate, from the antecubital vein. There was a difference of 31% at VT, 30% at 4 mmol·L^{-1}, and 26% at $\dot{V}O_2max$. Similarly, Foxdal et al. (1991) examined capillary, venous, and arterial blood to determine exercise intensity at 4 mmol·L^{-1}. They found exercise intensity to be 12% less when comparing capillary to venous and 17% lower when comparing plasma to whole blood samples. In the present study, the mean

difference at VT and CEP across sites was 30% and 15% respectively.

Changes in the redistribution of lactate, blood flow, and metabolic removal alter lactate accumulation at different sampling sites. The differences in lactate concentration can alter the interpretation of LT and the prescribed exercise intensity. When comparing blood lactate concentration across testing sessions or studies, it is important to compare only those samples taken from the same site and analyzed from the same fraction of blood.

4.5 Circadian Rhythmicity of Catecholamines

Circadian rhythmicity has been demonstrated in plasma and urinary catecholamines at rest (Akerstedt & Gillberg, 1983; Froberg, Karlsson, Levi, & Lidberg, 1975; Linsell, Mullen, Brown, & Causon, 1985). Both norepinephrine and epinephrine levels peak early in the afternoon and reach a minimum level during the night with the crest and trough of norepinephrine preceding epinephrine by 1-2 hours. Of the two catecholamines, norepinephrine is more unstable throughout a day and peak periodicity is more difficult to detect because norepinephrine secretion is affected by changes in posture and sleep (Akerstedt & Gillberg, 1983; Froberg et al., 1975).

Across morning and afternoon testing sessions, no circadian rhythmicity was demonstrated at rest or at VT. All values increased in the afternoon except norepinephrine resting values which decreased 0.43 nmol·L^{-1}. The normal range for resting values for epinephrine is 0.05-1.07nmol·L^{-1} and 0.46-3.08 nmol·L^{-1} for norepinephrine (Ratage, Baumgardt, Knoll, and Wisser, 1983). Both resting values were within the normal range but the standard deviations were very high. Resting correlations for both catecholamines were low, which may also be a reflection of the high interindividual variance. Peronnet et al. (1981) examined the reliability of catecholamines across days and found that group, but not individual results, were replicable. The standard error ranged from 14-50% for norepinephrine and 14-37% for epinephrine. They concluded that cyclic variations may influence the lack of reliability and that a single determination should not be used as a basis for drawing conclusions regarding the activity of the sympathoadrenal system.

4.6 Relationship between Catecholamines and Blood Lactate

Previous studies have demonstrated a strong correlation between plasma epinephrine concentration and blood lactate at rest and during exercise. These results are consistent across dogs, and humans (Mazzeo & Marshall, 1989; Stainsby et al., 1984). In the present study, plasma epinephrine was significantly correlated to venous lactate in the morning but not in the afternoon. This discrepancy could be attributed to the large variability of epinephrine in resting and exercise measures. Deuster et al. (1989) found the variability of epinephrine across three trials to be 8.5-111.3% and attributed the large variance to measurement error and effect of the emotional state and level of anticipation of the subject on the sympathoadrenal activity. Perhaps 30 minutes of sitting quietly was not sufficient to establish resting catecholamine levels.

Epinephrine concentration affects cardiovascular and metabolic responses during exercise. Increases in blood lactate, both at rest and during exercise, have been attributed to the strong beta-adrenergic stimulation of epinephrine (Brooks, 1991). Elevated cAMP levels induce a transformation of phosphorylase b to phosphorylase a in excess of what is required for oxidative purposes. An imbalance in production and oxidation of pyruvate occurs which results in an increase in muscle and blood lactate (Stainsby et al., 1985). Epinephrine may also decrease lactate uptake in inactive skeletal muscle and splanchnic areas and increase net lactate release from other tissues (Ahborg & Felig, 1982).

Norepinephrine, which reflects the activity of the sympathetic nervous system, did not correlate highly with venous lactate concentration. Even though both epinephrine and norepinephrine increase non-linearly like blood lactate during graded exercise, epinephrine plays a greater role in the release of lactate from muscle (Stainsby et al., 1984; Stainsby et al., 1985). During tetanic contractions, Stainsby et al. (1985) found that infusions of epinephrine and norepinephrine versus epinephrine, resulted in similar increases in net lactate output. Blockade studies of epinephrine

with dogs have shown that blood lactate appearance during treadmill running decreases, whereas blood lactate concentration remains unchanged following norepinephrine blockades (Issekutz, 1984).

A threshold for the activity of the sympathoadrenal system has been hypothesized to explain the effects of catecholamines on cardiorespiratory and metabolic control. During moderate to heavy exercise, a 4-5 fold increase of epinephrine above basal levels has a direct effect on blood lactate and glucose (Clutter, Bier, Shah, & Cryer, 1980). In the present study, epinephrine increased 0.68 $nmol \cdot L^{-1}$, a 2-3 fold increase from rest to VT in the morning and the afternoon. . This is consistent with the 2-13 fold increase found during moderate to intense exercise (Galbo, Holst, Christensen, & Hilsted, 1975). The increase of epinephrine in the afternoon may not have been strong enough to elicit an effect on glycogenolysis and thus the correlation was lower. Norepinephrine production must exceed epinephrine productions by 10% in order to exert any effect on hemodynamic and metabolic actions (Clutter, et al., 1980). Therefore, epinephrine is considered to be a stronger activator of the above systems.

In summary, the present study demonstrated that blood lactate concentration is reliable across trials measured at the same time of day but concentration varied when measured throughout a day, at different sampling sites, and in relation to plasma epinephrine concentration. Under standardized exercise and dietary conditions, no discernable circadian rhythm was detected at rest or during exercise in venous or arterialized lactate but the time of sampling and conditions should be standardized to minimize biological variation.

4.7 Conclusions

1. Core temperature and cardiorespiratory variables, such as HR, $\dot{V}O_2$, and $\dot{V}_E$, demonstrated circadian rhythms at rest but during exercise a circadian rhythm was only apparent in T_c.

2. Blood lactate measurement is reliable across trials when samples are taken at the same time of day. Even though blood lactate does not demonstrate a circadian rhythm at rest or during exercise, to produce the most reliable results, blood lactate should be measured at the same time of day to minimize biological variation of related variables.

3. Blood lactate concentration varies in relation to the sampling site. When samples of are drawn from the arm, arterialized lactate concentration exceeds venous concentration. Therefore, the sampling site is an important factor to standardize when comparing blood lactate values across studies or individuals.

4. Within the time restrictions of this study, plasma catecholamines did not demonstrate a discernable rhythm. Plasma epinephrine was correlated to changes in blood lactate and may play a regulatory role in lactate appearance in the blood. No relationship was established between blood lactate and plasma norepinephrine.

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Appendix A: Informed Consent

BLOOD LACTATE STUDY - INFORMED CONSENT

The purpose of this study is to determine if blood lactate demonstrates circadian rhythms at rest and during progressive exercise to $\dot{V}O_2max$ and to measure the variability of blood lactate across days and the correlation with plasma epinephrine. The findings of this study will help coaches and sport scientists to accurately interpret blood lactate changes over time.

I, ________________________, do hereby acknowledge:

* I consent to perform four $\dot{V}O_2max$ cycle ergometer tests, two tests each week three days apart during specified times and under controlled conditions:
  a) no exercise 24h prior to test
  b) fitness levels be maintained (training 2-3x per week only)
  c) consume a mixed diet 24h before each test
  d) adhere to set meal and snack times
  e) eight hours of sleep the night before each test
  f) no caffeine 12h prior to test
  g) no alcohol 12h prior to test

* I understand that 3-10 mls of blood will be drawn by an IV therapist during each test at rest, ventilatory threshold and three minutes post-exercise. The blood will be subsequently analyzed for lactate and epinephrine concentrations.

* I understand that a heart rate monitor will be attached across my mid-sternum region to record my heart rate every 30 s during exercise; core temperature will be recorded every three minutes by the insertion of a disposable rectal probe; a mouthpiece will be worn and expired gases will be analyzed by a Beckman metabolic measurement cart every 30s for respiratory and metabolic factors.

* Even though, I will be undergoing exercise to the point of temporary exhaustion, I understand there is very little risk involved if I am a healthy,active individual and that emergency equipment and trained personnel are available to deal with unusual situations that may arise.

* I understand that I may temporarily experience local muscle fatigue and discomfort in the legs, nausea, and light headedness when cycling to exhaustion. I understand that I may also experience some discomfort upon insertion of the catheter and small amount of bruising may result at the puncture site.

* I understand I may ask any questions or request further explanations or information about the procedures at any time before, during or after testing.

* I understand that I am in control at all times, that I am able to withdraw from, reduce or modify my involvement in the study at any time and that the test may be terminated by the investigators upon observation of any symptoms of distress or abnormal responses.

* I understand that all my results are strictly confidential, that they will be sited only by an ID label or used to calculate a group mean and that all data will be securely locked in a cabinet at all times.

* that I do hereby release, ____________________________, and its employees
  Name of the institution
  from any liability with respect to any injury or damage that I may suffer during participation in this study except where the damage or injury is caused by negligence of ______________________________.
  Name of the institution

I acknowledge that I have read, understood, and agree to the contents of this informed consent agreement in its entirety.

________________________ ______________
Signature Date

________________________ ______________
Witness Date

Appendix B: Time Guidelines of the Study

	WEEK 1		WEEK 2	
	MONDAY	THURSDAY	MONDAY	THURSDAY
Group A				
S1	0700	0700	1200	1700
S2	0730	0730	1230	1730
S3	0800	0800	1300	1800
Group B				
S4	1200	1700	0700	0700
S5	1230	1730	0730	0730
S6	1300	1800	0800	0800

Note: the same cycle was repeated for subjects 7-12 on Tuesday and Friday.
for reliability purposes, the additional 11 subjects repeated the same cycle during Week 1 as S1-S3.

Figure 1. Circadian rhythmicity timeline for measurement of blood lactate, cardiorespiratory variables, and core temperature.

Appendix C: Dietary Intake

DIETARY INTAKE 24 H PRIOR TO THE TEST

Choose any of the following foods during the 24 hours prior to each test and consume enough food to reach the total number of recommended points ___________. Please chart your carbohydrate intake and points on the attached sheet.

TWO POINTS

Fruit	**Portion**
Strawberries	1 cup
cantaloupe	1/2 medium
plum	1 medium/2 small
watermelon	1 cup

Vegetables	
asparagus	1 cup
beans - green, yellow	1 cup
beets	1 cup
broccoli	1 cup
brussel sprouts	12 medium
cabbage - cooked	1/2 cup
cabbage - raw	1 cup
cauliflower	1 cup
corn	1/2 cup/3" cob
green pepper	1/2 large
mushrooms	20 small
peas	1/2 cup
spinach	1 cup frozen or raw
tomato juice	1 cup
tomato - spaghetti sauce	1/2 cup
zucchini	1 cup

tomato-raw, lettuce, celery receive no points

Snacks	
arrowroot cookies	2
chocolate chip cookies	1
cream filled cookies	1
doughnuts	1 small
fig bars	2 small
ginger snaps	1
oatmeal cookies	1
peanut butter cookies	1
popcorn-plain, flavoured	1 cup
candied popcorn	1/2 cup

Miscellaneous	
cream soup with water	1 cup
ice-cream bars	1
jello	1/2 cup

sugar white or brown	2 teaspoons
teriyaki sauce	1 ounce
white sauce	1/2 cup
Dairy	
milk (skim, 1%, 2%)	1 cup

THREE POINTS

Fruit	**Portion**
nectarine	1
orange	1
prunes	3
raspberries	1 cup
Grains/Starches	
breads-all kinds	1 slice
bagel	1/2 medium
croissant	1 medium
dinner roll	1 small
english muffin	1/2
french toast	1 slice
graham wafers	3
granola bar	1
pancake	1 medium
pasta-cooked	1/2 cup
rice- cooked	1/2 cup
ritz crackers	7 or 8
scone/biscuit	1 medium
soda crackers	7 or 8
taco shell	2
tortilla shell	1
triscuit	5
waffle	1
Potato	1 medium
hashbrowns	3/4 cup
french fries	10 medium
scalloped potatoes	1/2 cup
Legumes	
chick peas	1/2 cup
dried beans	1/2 cup
dried peas	1/2 cup
kidney beans	1/2 cup
lentils	1/2 cup
cream soup with milk	1 cup
stock soup with vegetables	1 cup

Dairy	
chocolate milk	1/2 cup
carnation instant breakfast	1/2 cup
custard	1/2 cup
ice-cream	1/2 cup
pudding	1/2 cup
pudding pop	1
yogurt-flavoured	1/2 cup
yogurt-plain	1 cup
yogurt-frozen	1/2 cup

Miscellaneous	
honey	1 Tablespoon
jam	1 Tablespoon
jelly	1 Tablespoon
marmalade	1 Tablespoon
sweet and sour sauce	1 Tablespoon
syrup	1 Tablespoon

FOUR POINTS

Fruit	**Portion**
apple	1 medium
apple sauce	1 cup
apricot	6
banana	1 medium
blackberries	1 cup
blueberries	1 cup
cherries	20
dates	3
figs	2
grapes	20
grapefruit	1
honeydew melon	1/2 medium
raisins	2 Tablespoons
strawberries	1 cup
fruit juice	1 cup

Grains/Starches	
muffin	1 medium
hamburger bun	1
hotdog bun	1
kaiser roll	1
cereal hot/cold	3/4 cup
granola	1/4 cup
grapenuts	1/4 cup

Snack Foods

brownies	1 small piece
cake/loaf/pie	1 small piece
cupcake/sweet roll	1 small
lasagna	4 ounces (1/2 cup)
meat pie	1/2 piece
milkshake	1/3 cup
nacho chips	1 ounce
pizza	1 large piece
pork and beans	1/2 cup
pop or fruit drink	1 cup
potato chips	1 ounce
pretzels	1 ounce

Appendix D: Correlations and SEM of core temperature and cardiorespiratory variables

Table 1

Correlations and standard error of the measurement (SEM) of core temperature (T_c), heart rate (HR), oxygen consumption (VO_2), and ventilation (V_E) across sampling times.

	r	SEM	r	SEM
T_c (°C)	0700		1200	
1200-R	0.65	0.2		
1200-VT	0.55	0.1		
1200-CEP	0.23	0.2		
1700-R	0.48	0.3	0.58	0.1
1700-VT	0.39	0.2	0.58	0.1
1700-CEP	0.36	0.2	0.58	0.1
HR ($b \cdot min^{-1}$)				
1200-R	0.71	3.5		
1200-VT	0.86**	4.0		
1200-CEP	0.84**	2.2		
1700-R	0.72*	3.8	0.72	1.4
1700-VT	0.88**	3.3	0.86**	1.7
1700-CEP	0.81**	6.0	0.88**	6.7
VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)				
1200-R	0.11	0.5		
1200-VT	0.87**	1.7		
1200-CEP	0.90**	5.4		
1700-R	0.18	1.4	0.43	1.4
1700-VT	0.92**	2.6	0.96**	1.7
1700-CEP	0.91**	5.6	0.86	6.7
V_E ($l \cdot min^{-1}$)				
1200-R	0.74*	1.0		
1200-VT	0.76*	3.8		
1200-CEP	0.82*	7.2		
1700-R	0.43	1.5	0.80*	0.8
1700-VT	0.80*	4.1	0.82*	3.4
1700-CEP	0.93**	4.9	0.65	9.1

Rest=resting values were recorded after 30 minutes of sitting quietly
VT = ventilatory threshold
CEP= criterion endpoint (20-45 W below the power output at which peak $\dot{V}O_2$ was reached).
* = significance at $p<0.01$
** = significance at $p<0.001$

Appendix E: Literature Review

REVIEW OF LITERATURE

Introduction

Several physiological variables are measured in both a laboratory and field setting to monitor training adaptations, to predict performance, and to establish physiological profiles of athletes. Maximal oxygen consumption ($\dot{V}O_2max$), the criterion test of aerobic power, is frequently measured to assess cardiovascular changes. Another variable measured to reflect metabolic changes during exercise is blood lactate. Blood lactate samples are taken at various points throughout exercise to determine the power output, $\dot{V}O_2$, or heart rate (HR) at which 2 $mmol \cdot L^{-1}$ concentration, 4 $mmol \cdot L^{-1}$ concentration, onset of blood lactate accumulation (OBLA), lactate threshold (LT), individual anaerobic threshold (IAT), or a maximum lactate concentration are reached. This data is used to determine the efficacy of training regimens, to prescribe training HR and velocities, and to predict endurance performance (Jacobs,1986; Monpetit, 1984).

Blood lactate accumulation is affected by several physiological variables and varied experimental procedures. The influence of diet, substrate availability, and training has been studied extensively but the affects of catecholamine concentration, site of sampling, and presence of circadian rhythmicity on blood lactate during rest and exercise requires further study.

Lactic Acid

Lactic acid is a metabolite produced by the breakdown of muscle glycogen in both the presence and absence of oxygen (O_2) during exercise. When the rate of glycolysis exceeds the uptake of pyruvic acid and oxidation of NADH at the mitochondrial membrane, the amount of pyruvic acid reduced to lactic acid increases (Wasserman, Beaver, & Whipp, 1986). The conversion of pyruvic acid to lactic acid produces more NAD^+ which permits the production of ATP to continue at a high rate via substrate-level phosphorylation. Upon formation, lactic acid immediately dissociates (pKa-3.7) to its anion (lactate) and proton (H^+) at physiological pH values.

Both ions are removed by different metabolic pathways. Due to the law of mass action, an increase in lactic acid will occur whenever there is an increase in pyruvic acid, H^+, or cytosolic NADH (Katz & Sahlin, 1990).

The increase in lactic acid during exercise has been associated with the classic theory of oxygen deficiency. In 1923, Hill and Lupton stated that as exercise intensity increases lactate concentration rises slowly, but at higher power outputs, blood lactate concentration increases at a rapid rate. They proposed that at higher power outputs, oxygen delivery is inadequate to meet the metabolic demands of the working muscles. However, recent studies have shown that lactate can be formed in fully aerobic tissues such as the heart at rest during carbohydrate loading and epinephrine stimulation (Stainsby & Brooks, 1990). These findings counter the long held belief that lactate is only formed as a result of oxygen limited metabolism. Blood lactate is presently considered to be a useful metabolic fuel and gluconeogenic precursor rather than just a waste product (Brooks, 1991). The effects of other factors such as the kinetics of glycolysis, epinephrine, blood flow, and substrate availability on blood lactate accumulation requires further research (Brooks, 1991; Stainsby & Brooks, 1990).

The difference between muscle and blood lactate concentrations are attributed to the rate of lactate production in the muscle; rate of release from muscle to the blood; and removal from the blood by the liver, heart, and skeletal muscles (Stainsby & Brooks, 1990). At rest, muscle lactate concentration is similar to levels in the blood. During submaximal exercise, muscle lactate values are two to three times higher than blood and at maximal exercise levels, muscle lactate concentration can exceed blood lactate by a factor of 10 (Chwalbinska-Moneta, Robergs, Costill, & Fink, 1989; Walsh & Banister, 1988). During non-steady state exercise, blood lactate increases at a rate dependent on pool size and the imbalance between rate of appearance and rate of removal. After intense exercise, blood lactate peaks within the first few minutes and rapidly declines during the next 10-20 minutes (Gollnick, Bayly, & Hodgson, 1986).

Lactate appearance in the blood is delayed due to the release from muscle and other tissues,

changes in muscle lactate concentration, blood flow, pH, and resistance of lactate efflux through the sarcolemmal, interstitial fluid, and capillary membranes (Roth, 1991). Changes in membrane electrical potential and the charge effect in carrier mediated transport process are factors that affect the lactate release from tissue and appearance in the blood. At low exercise intensity, lactate release from muscle increases linearly, but at high intensity, saturation may be reached at 4.5 $mmol \cdot L^{-1}$ (Jorfeldt, Juhlin-Dannfelt, & Karlsson, 1978).

Lactate accumulation is an individual response influenced by physiological variables and experimental procedures. Experimentally, blood lactate values can vary depending on the sampling site, sampling time, exercise protocol, exercise mode, and method of analysis (Graham, 1984). Training status, intensity and duration of exercise, glycogen stores, hormonal levels, acid-base balance, and muscle fiber recruitment are several physiological factors that may influence blood lactate concentration during rest and exercise (Gollnick et al.,1986). When interpreting changes in blood lactate over time, the influence of these factors must be considered for individual results and interstudy comparisons.

Similarly, the reliability of blood lactate is another factor to consider when comparing blood lactate values across trials. Few studies have examined the reliability of blood lactate but one study found the reliability of venous lactate to be $r=0.93$ (Davis, Vodak, Wilmore, Vodak, & Kurtz, 1976). Most studies have determined the reliability for ventilatory measures (Caiozzo, Davis, Ellis, Azus, & Nandagriff, 1982; Weltman et al, 1990). More research is required to determine the reliability measurement of blood lactate and to quantify the difference across trials.

Experimental Procedures

Lactate accumulation can be measured in both muscle and blood. Muscle biopsies are assayed for lactate concentration primarily during research studies and are not frequently used to monitor training adaptations. In the last decade, measurement of blood lactate has increased significantly due to the ease of sampling and accuracy of analysis (Graham, 1984). The site of

sampling can influence the apparent time course of lactate accumulation in the blood. Based on the removal process, blood lactate concentration does not accurately represent cellular lactate production as lactate may be rapidly oxidized within the same muscle or at other sites such as the liver, heart, skin, and intestines (Graham, 1984; McGrail et al., 1977; Stainsby & Brooks, 1990).

When measuring blood lactate concentration, most sampling occurs away from the site of active muscle for safety and convenience reasons. During research studies, blood lactate is sampled primarily from an indwelling catheter placed in an artery or vein located in the arm which allows the researcher to sample blood lactate continuously throughout exercise. To monitor blood lactate changes, in both the field and laboratory settings, microsamples of blood are drawn from the finger tip and hyperemized earlobe (Jacobs, 1986). The more distant the site of sampling from the source, the more important it is to maintain a high blood flow rate to the sampling site to help minimize the arterial-venous differences (Graham, 1984).

The distribution of lactate is not homogenous in arterial, arterialized, or venous blood. Up to a two fold difference has been recorded between blood lactate concentration in the femoral artery and antecubital vein (Yoshida, Takeuchi, & Suda, 1982). Similarly, arterialized and capillary lactate concentration have both been found to exceed venous lactate concentration by 8% (Foxdal et al., 1990; Robergs et al., 1990). Different sampling sites are not directly comparable and as a result, detection of the power output or heart rate at lactate threshold or fixed blood lactate concentration will vary according to the sampling site.

Blood lactate samples can be analyzed in plasma, precipitated whole blood, and non-haemolized, non-precipitated whole blood. Due to the higher concentrations of lactate in plasma than erythrocytes, analysis based on non-haemolysed blood cannot be compared with haemolysed blood (Foxdal et al., 1990). Different assays are performed on spectrophotometers or automated lactate analyzers to determine blood lactate concentration. To produce reliable blood lactate results, it is critical to standardize the storage of blood and calibration of the analyzers. Similarly, it is important to standardize the exercise protocol. As demonstrated by Jacobs (1986),

if the workloads are rapidly increased in small increments (25 W·min^{-1}), blood lactate accumulation is lower at the same workload than if larger increments are made over a longer time period (50 W·4 min^{-1}). Therefore, when interstudy and interlaboratory comparisons are made the sampling time, intensity increments, sampling site, exercise mode, and analysis method must be considered (McLellan, 1985).

Physiological Factors

Training

Aerobic training induces both hormonal and metabolic changes that enhance fat utilization and spare muscle glycogen during exercise. Several hormones such as catecholamines, cortisol, growth hormone, insulin, glucagon, and adrenocorticotrophic hormone (ACTH) influence carbohydrate metabolism and the release of lactate from muscle (Conroy & Mills, 1970). After several weeks of training, there is an apparent reduction in blood and muscle lactate accumulation due to a decreased secretion of hormones, changes in substrate utilization, and an increased efficiency of the oxidative pathways (Gollnick et al., 1986; Walsh & Banister, 1988). In comparison to untrained individuals, trained athletes accumulate less lactate at the same submaximal power output, they reach their LT at higher workloads, and can exercise at higher percentage of $\dot{V}O_2max$ for extended periods of time before the onset of lactate accumulation (Simon, Young, Gutin, Blood, & Case, 1983). However during maximal efforts, an anaerobically trained individual will reach higher lactate concentration than an untrained or aerobically trained individual based on power output and metabolic differences (Maassen & Busse, 1989).

Intensity and Duration

Intensity and duration of a training session can affect the amount of lactate produced and released from muscle and other tissues. At rest, approximately 1 mmol·L^{-1} is present in both muscle and blood due to low levels of muscle and red blood cell metabolism (Gollnick et al., 1986).

During low intensity aerobic exercise (up to 50-60% $\dot{V}O_2max$), little or no blood lactate accumulates as energy is supplied almost entirely by oxidative processes and removal is in equilibrium with production (Stainsby, 1986). When exercising between 60-80% $\dot{V}O_2max$, blood lactate rises initially (between 0-10 minutes) and then plateaus or decreases once steady state is reached (Stainsby, 1986). As intensity exceeds 60-80% $\dot{V}O_2max$, blood lactate rapidly begins to accumulate. The power output at which blood lactate rises exponentially is called LT (Walsh & Banister, 1988). The HR, percent of $\dot{V}O_2max$, or power output at the break point reflects the intensity that can be sustained during prolonged periods of exercise. Beyond this point, the accumulation of lactate and H^+ can lower muscle pH which reduces the activity of key enzymes and cofactors involved in carbohydrate metabolism (phosphofructokinase, cyclic AMP), reduces cross bridge formation (myofibrillar-ATPase), and decreases the mobilization of fatty acids (Gollnick et al., 1986; McGrail et al., 1977). These changes ultimately impede muscular performance. When competing in a race, it is important for athletes to pace themselves to race at or just below LT to prevent the onset of metabolic acidosis.

Muscle Glycogen

Lactate production is also dependent on muscle glycogen stores . If there are insufficient stores due to low carbohydrate intake and/or exhaustive exercise, muscle and blood lactate concentration will be lower at a given workload (Chwalbinska-Moneta et al., 1989; Jacobs, Sjodin, Kaiser, & Karlsson, 1981; Maassen & Busse, 1989; Walsh & Banister, 1988). Similarly, if a diet is rich in carbohydrate, substrate utilization will change, the rate of glycolysis will increase and blood lactate levels will be elevated compared to mixed diet values (Yoshida, 1984). These changes are apparent in both endurance and supramaximal exercise. When measuring lactate, it is important for athletes to consume a balanced diet and to have rested 24 h prior to testing to ensure that muscle glycogen stores are replenished.

Muscle Fiber Types

Muscle fiber types have distinct metabolic characteristics that influence lactate production and removal. Fast twitch fibers (FT) recruited during heavy workloads produce lactate when stimulated. They are characterized by a high concentration of glycolytic enzymes and muscle lactate dehydrogenase (M-LDH) isozymes (Stanley et al., 1986). Slow oxidative fibers (SO) have a high mitochondrial content and contain a high concentration of heart lactate dehydrogenase (H-LDH) isozymes. These fibers readily oxidize lactate to pyruvate (Hermansen & Stenswold, 1972). The FT:SO ratio varies in different muscle groups and among individuals. These differences may explain some of the variability in production and removal rates between individuals, as a person with a high percentage of SO fibers will produce less lactate and oxidatively remove more lactate than a person with higher number of FT fibers (McGrail et al., 1977). Therefore, it is important to use the same subject as his/her own control for comparisons of blood lactate values across time.

Thresholds

Wasserman, Whipp, Koyal and Beaver (1973) introduced the concept of anaerobic threshold (AnT) as the level of work or oxygen consumption just below that at which metabolic acidosis and the associated shift in gas exchange occurs. A great deal of controversy surrounds the determination of AnT and the values determined by invasive (LT) and non-invasive measures (ventilatory threshold). These two breakpoints were believed to occur at the same point, but the cause and effect theory has been challenged by Brooks (1985), Gaesser and Poole (1986), and Neary, MacDougal, Backus and Wenger (1985). Ventilatory threshold (VT) is determined as the point of non-linear increase in ventilation (V_E), volume of carbon dioxide (VCO_2), and V_E/VO_2 ratio (Walsh & Bannister, 1988). Since the discovery of LT, it has been defined in many ways. LT has been set at absolute blood lactate concentrations such as: 2 $mmol \cdot L^{-1}$ (Hughson & Green, 1982) and 4 $mmol \cdot L^{-1}$ (Kindermann, Simon & Keul, 1979), the initial level of increase in blood lactate concentration above resting levels (Davis et al., 1976; Wasserman et al., 1973), IAT (Stegmann,

Kindermann, & Schnabel, 1981), point of systematic increase in blood lactate concentration (Caiozzo et al., 1982), onset of plasma lactate accumulation (Farrell, Wilmore, Coyle, Billing, & Costill, 1979), and lactate turning point (Davis, Bassett, Hughes & Gass, 1983). Brooks (1985) defined LT as the workload at which there was an abrupt increase, or disproportionate non-linear increase in blood lactate concentration. Davis et al. (1976) defined LT as the workload immediately preceding the progressive increase in blood lactate concentration. All methods have been criticized for having a wide margin of inter-observer error in determining the exact threshold level. To minimize errors in determination of VT and LT, a computer linear regression model should be employed (Beaver, Wasserman, & Whipp, 1985).

Circadian Rhythms

Many physiological functions which affect blood lactate accumulation also demonstrate a marked periodicity. Cardiorespiratory functions (HR, blood pressure, blood flow, and metabolic rate), components of physical fitness (muscular strength and power), urinary secretions, core temperature (T_c), and hormonal output all demonstrate characteristics of regularly repeating cycles known as circadian rhythms (Minors & Waterhouse, 1981; Winget, DeRoshia, & Holley, 1985). Circadian rhythms oscillate within a 24 $\pm$ 4 h time period. When synchronization occurs with the 24 h light and darkness cycle, the terms diurnal and nocturnal are used (Mills, 1966; Shephard, 1984). Investigations of circadian rhythms have been documented since the 1800's on both plants and animals, but the majority of research has occurred within the last 25 years (Aschoff & Wever, 1976). The primary focus with humans has been the alteration of circadian rhythms and performance during shift work, space flight, sleep deprivation, international travel, and to some extent athletic performance. Several strategies have been employed to more rapidly resynchronize circadian rhythms. Other rhythms such as circa-annual (t=1 year), circalunar (t=28 d), and circaseptan (t=7 d) have demonstrated cyclical variation for basal metabolic rate, the menstrual cycle, and PWC_{170} respectively (Shephard, 1984). Many physiological variables that influence

performance have demonstrated circadian rhythms at rest, but the consistency of circadian rhythms during exercise remains equivocal.

Circadian rhythms integrate information from both the physiological and psychological systems and are governed by endogenous and exogenous factors. Endogenously, rhythms fluctuate within a 24 ± 4 h time period but the rhythm can be exogenously controlled to oscillate precisely every 24 h (Aschoff & Wever, 1976). Cortisol and T_c are two examples of truly endogenous rhythms that oscillate approximately every 25 h with the removal of environmental cues. Sleep, wakefulness, meal constituents and timing, altitude, social factors, and psychological stressors are exogenous factors that can synchronize or desynchronize circadian oscillations to 24 h (Minors & Waterhouse, 1981; Winget et al., 1985). Therefore, when comparing blood lactate values or any related physiological variables across time, exogenous factors should be controlled to minimize their interference with data interpretation.

Endogenous circadian timing is governed by two pacemakers, X which drives body temperature and Y which drives the rest-activity cycle (Moore-Ede, 1983). The Y pacemaker, located in the suprachiasmatic nuclei of the hypothalamus, primarily governs skin temperature, plasma growth hormone, urinary calcium, and slow wave sleep. The X pacemaker governs rapid eye movement sleep, plasma cortisol, body temperature, and urinary potassium but the location of X has not be identified (Moore-Ede, 1983). Both pacemakers regulate certain variables but there is integration of information between the two sites of control.

Circadian rhythms demonstrate interindividual differences as reflected by mean levels, amplitude, and stability of different variables (Akerstedt & Froberg, 1976). Circadian rhythms also vary from one variable to another and from one day to the next; however, distinct daily maximum and minimum fluctuations are still apparent at specific times of the day (Aschoff & Wever, 1976). Hill, Cureton, Collins, and Grisham (1988) measured diurnal variation of physiological responses to exercise of morning and evening type individuals. They concluded that diurnal variation to exercise is the same regardless of whether the person demonstrates morning or evening type

habits. More research is required to examine the 'owl and lark' theory in relation to circadian rhythms.

Within day (24 h) and daily (day to day) variations may affect metabolic processes and neuromuscular activity that can directly alter blood lactate response. Any circadian variation may have a direct effect on the interpretation of fitness test results, exercise prescription, and performance (Reilly & Brooks, 1982). Reviews on circadian rhythms and athletic performance by Reilly (1985) and Winget et al. (1985) indicate that only one study to date has examined the influence of time of day on blood lactate response during exercise in healthy adults. Other research on circadian rhythms and blood lactate has focused on clinical implications for cardiac patients and drug administration (Moore-Ede, Czeiler, & Richardson, 1983).

Core Temperature

T_c demonstrates a strong circadian rhythm at rest and throughout exercise and is thought to be reflected in pulse rate and metabolism (Cable & Reilly, 1987; Faria & Drummond, 1982). T_c varies from approximately 36°C at 0600 h and peaks at 37.8°C at 1800 h (Minors & Waterhouse, 1981; Winget et al., 1985). Changes in T_c can affect thermoregulation, metabolic rate, nerve conduction velocity, metabolic enzyme reaction rates, and psychomotor performance (Conroy & Mills, 1970). Peak T_c coincides with peak responses during 1500-2100 h in $V0_2$, V_E, and metabolic rate (Table 1).

Cardiorespiratory Variables

Circadian variation in cardiorespiratory functions influence the delivery rate of O_2, glucose, and hormones to organs and muscles, the removal of metabolites, and distribution of metabolic heat from core to periphery, all of which have an impact on performance (Winget et al., 1985). HR, $\dot{V}O_2$, and $\dot{V}_E$ have consistently demonstrated maximum and minimum fluctuations at rest (Conroy & Mills, 1970; Faria & Drummond, 1982; Reilly & Brooks, 1982; Reilly, Robinson, &

Table 1. Peak circadian oscillations of physiological variables that may affect lactate accumulation.

Physiological Variables	Peak Time of Circadian Rhythms		
	Rest	Submaximal Exercise	Maximal Exercise
Core Temperature (°C)	1600-1800 h	1600-1800 h	1600-1800 h
Minute Ventilation ($l{\cdot}min^{-1}$)	1300-2000 h	1500-1800 h	1500-1800 h
Oxygen Consumption ($ml{\cdot}kg^{-1}{\cdot}min^{-1}$)	1500-1800 h	0800-1400 h	1600-2000 h
Metabolic Rate ($W{\cdot}m^{2}$)	2045 h		
Heart Rate ($b{\cdot}min^{-1}$)	1700 h	1500-1800 h	1200-2000h
ACTH ($ug{\cdot}100ml^{-1}$)	0600 h		
Cortisol ($ug{\cdot}100ml^{-1}$)	0600 h		
Glucagon ($pg{\cdot}min^{-1}$)	1800 h		
Insulin ($uU{\cdot}ml^{-1}$)	1300-1400 h		
Epinephrine ($nmol{\cdot}l^{-1}$)	1300-1500 h		
Norepinephrine ($nmol{\cdot}l^{-1}$)	1200-1400 h		
Performance		2200 h	2200 h

Note: summary information was obtained from Conroy & Mills (1970); Minors & Waterhouse (1981); Winget et al. (1986).

Minors, 1984) but conflicting results have been found during exercise. At submaximal exercise, $\dot{V}_E$ peaks between 1300-2000 h (Crockford & Davies, 1969; Reilly, 1982; Reilly & Brooks, 1982), HR peaks at 1700 h (Crockford & Davies, 1969; Davies & Sargeant, 1975; Minors & Waterhouse, 1981), and $\dot{V}O_2$ peaks at 0800-1400 h (Reilly & Brooks, 1982); however, one investigation found no diurnal variation occurred during exercise (Faria & Drummond, 1982). At maximal exercise, conflicting results have been found for all three variables (Faria & Drummond, 1982; Ilmarinen, Ilmarinen, Korhonen, & Nurminen, 1980). In general, circadian rhythms for T_c and HR persist at rest, and up to maximal levels, but as exercise intensity increases, the amplitude of HR response is reduced. Similarly, the amplitude of circadian rhythms for $\dot{V}_E$, $\dot{V}O_2$, and $\dot{V}CO_2$ declines as exercise intensity increases and oscillations are not clearly detectable at $\dot{V}O_2$max. Discrepancies in the findings may be attributed to experimental differences: large range of sample sizes (n=1-31), measurement of two to eight points throughout a day, ineffective control of exogenous factors, mode of exercise, varied exercise protocols, sleep loss, and physical exhaustion. All of these factors may alter the amplitude of circadian rhythms.

Anaerobic Power and Capacity

Recently, Hill and Smith (1991) examined the circadian rhythmicity of anaerobic power and capacity. When competing, disturbances in physiological parameters may be important when milliseconds separate winning times. Hill and Smith measured power output, oral temperature, and HR during a 30 s modified Wingate test and they found that all three variables peaked at 2100 h. Vanderwalle, Peres, and Monod (1987) determined the reliability of anaerobic power and capacity for day to day variability to be less than 5%. Hill and Smith stated that the 6-9% variation was attributed to circadian rhythmicity. Further research is required to determine the pacemaker and oscillatory site.

Athletic Performance

Circadian rhythms have also been demonstrated in peak athletic performance in several different events. In a 24 h investigation, a circadian rhythm was demonstrated by swimmers with the fastest times for 100m and 400m events occurring at 2200h (Baxter & Reilly, 1983). Similarly, Conroy and O'Brien (1974) found performance peaked in the evening with runners, shot putters, and a rowing crew. Factors contributing to performance advantage during the daytime include a faster reaction time, greater flexibility associated with higher core temperature, and lower rate of perceived exertion (Hill and Smith, 1991). Concomitantly, Hill, Cureton, and Collins (1989) tried to determine when the greatest training adaptations occurred throughout a day. After six weeks of training, they concluded that there was not circadian specificity in adaptation of responses to submaximal or maximal responses of HR or perceived exertion during exercise in relation to performance time.

Hormones

Levels of cortisol, catecholamines, growth hormone, insulin, glucagon, lactate dehydrogenase (LDH), and muscle glycogen may all influence net lactate output under different conditions. Cortisol, which stimulates liver gluconeogenesis and increases fatty acid mobilization, is controlled by secretions of ACTH. ACTH and cortisol peak at 0600 h and reach a minimum value at 1800 h (Minors & Waterhouse, 1981). Catecholamines, epinephrine which stimulate glycogenolysis and norepinephrine that represents sympathoadrenal activity, peak between 1000-1400 h (Akerstedt & Gillberg, 1983). Growth hormone, which elevates metabolic rate and integrates other hormones that regulate macronutrient metabolism and electrolyte production, peaks between 1830-0300h (Conroy & Mills, 1970). Insulin and glucagon regulate blood glucose levels and both demonstrate circadian rhythms under fasting conditions. Maximum secretion occurred at 0700-0800h and 1800h for insulin and glucagon respectively (Conroy & Mills, 1970). No discernable cycles have been demonstrated for blood glucose as blood glucose levels are directly

effected by meal times and meal constituents (Stephenson, et al., 1989). LDH, which regulates the conversion of pyruvate to lactate, peaks between 1600-2000h (Conroy & Mills, 1970). Metabolic changes in growth hormone, LDH, insulin, glucagon, and cortisol demonstrate circadian rhythms at rest but very little research has examined the influence of exercise on circadian rhythmicity.

Catecholamines and Blood Lactate

Evidence of circadian covariation between body temperature, subjective alertness, and performance is substantial; however, few attempts have been made to establish the association of catecholamine excretion with other variables in a circadian setting (Akerstedt & Froberg, 1976). At the onset of exercise, afferent impulses from the working muscles and integration of information at the higher centers follow a set pattern of sympathoadrenal activity according to the relative workload. The release of catecholamines has a significant effect on the cardiovascular, hormonal, metabolic, thermoregulatory, water, and electrolyte homeostasis (Fry, 1988).

Epinephrine accelerates glycogenolysis and glycolysis and increases the rate of release of lactate from active skeletal muscle (Katz and Sahlin, 1990). An exponential rise in muscle lactate production with an increase in power output corresponds to a similar profile in plasma epinephrine. Gregg, Mazzeo, Budinger, and Brooks (1989) demonstrated that a strong correlation exists between lactate appearance in the blood and epinephrine over a wide range of exercise intensities ($r=0.97$). As well as elevating lactate release from muscle and other tissues, epinephrine also reduces blood flow to the splanchnic area, decreasing lactate clearance. Based on catecholamine infusion and blockade studies, increases in norepinephrine concentration do not correlate with changes in blood lactate appearance (Issekutz, 1984; Stainsby & Brooks, 1990). Increases in norepinephrine have been associated with an increase in sympathetic activity.

Circadian Rhythms and Blood Lactate

Several studies have examined the circadian variation of variables that influence blood lactate, but only one study to date has examined daily variation of blood lactate at rest and during exercise. Reilly and Baxter (1983) measured blood lactate response during cycling at 95% $\dot{V}O_2max$ and found a 41% and 21% increase in total work output and blood lactate respectively from 0630h compared to 2200h. The significant change in blood lactate could not be attributed to circadian variation due to the significant increase in total work performed. Coaches and sport scientists will be able to understand and interpret blood lactate changes more effectively if the amount of fluctuation expected based on circadian variation and daily fluctuations are known both at rest and during exercise. They will be able to determine the degree to which the change of blood lactate represents a circadian rhythm, day to day variation, or a training adaptation. Further research is also required to determine the relationship of epinephrine concentration and blood lactate appearance and clearance rates and the difference in blood lactate concentration across different sampling sites.

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VITA

Surname: **McFADYEN** Given names: **PAULA**

Place of Birth: Kamloops, B.C. Date of birth: December 3, 1964

Educational Institutions Attended:

University of Victoria	1982 to 1987
University of Victoria	1990 to 1992

Degrees Awarded:

B.Sc. (Human Performance) - University of Victoria	1987

Honors and Awards:

University of Victoria Fellowship	1990 to 1991
Canadian Association Sport Science Award	1987

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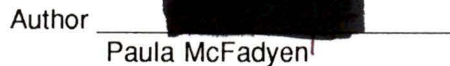
Author 
Paula McFadyen

Date April 27, 1992