





This is to certify that the

dissertation entitled

Oxygen Consumption, Heart Rate and Blood Lactate Levels as Affected by Exercise Intensity, Meals and Diets in Humans During Rest, Exercise and Recovery

presented by

Kristian Lindsted

has been accepted towards fulfillment of the requirements for

Ph.D. _____degree in ____Human Nutrition

Jackel A. Chemmel Major professor

Date April 28, 1982

ì

MSU is an Affirmative Action/Equal Opportunity Institution

0-12771

MSU LIBRARIES	ok drop to checkout from . <u>FINES</u> will if book is ter the date
DEC 2 0 1993	

Oxygen Consumption, Heart Rate and Blood Lactate Levels as Affected by Exercise Intensity, Meals and Diets in Humans During Rest, Exercise and Recovery

By

Kristian Lindsted

A DISSERTATION

Submitted to

Michigan State University

in partial fulfillment of the requirements

for the degree of

Doctor of Philosophy

Department of Food Science and Human Nutrition

ABSTRACT

Oxygen Consumption, Heart Rate and Blood Lactate Levels as Affected by Exercise Intensity, Meals and Diets in Humans During Rest, Exercise and Recovery

By

Kristian Lindsted

One hundred linear regression equations (HROXCR) between HR (HR) and oxygen consumption (OXC) were developed based on 10 incremental exercise tests in each of 10 subjects. Both slopes and y-intercepts were found to be significantly inhomogenous within as well as among subjects. The inhomogeneity of the HROXCR was increased rather than decreased by using incremental HR. The HROXCR was singificantly affected by previous exercise but not by diet. The repeatability (R = 0.7) for the slopes and y-intercepts of the HROXCR indicates that the variation among subjects was larger than the variation within the subjects.

The HROXCR observed during exercise was not followed during recovery. OXC returned to resting values faster than HR during early recovery (3-4 min). Duration rather than intensity was related to the HR recovery lag at greater than 65% max OXC. After 40 min of recovery the OXC had returned to the preexercise level, but the HR was still elevated 20 beats/min.

The effects of 700 kcal meals from either fat, protein or carbohydrate on OXC during rest was investigated in five male subjects. The postabsorptive state served as control for each subject. The

Kristian Lindsted

mean OXC for control, fat, protein and carbohydrate meal respectively was 3.6, 3.8, 4.3 and 4.1 ml/minute/kg. The thermogenic responses were as follows: fat - borderline significance; protein - significant > 6 hrs; carbohydrate - significant > 3 hrs. Only following the carbohydrate meal was there a significant HROXC correlation (r = 0.5). It may be that the regulatory thermogenic response is related to carbohydrate.

Only the carbohydrate meal affected the RQ during rest (\overline{X} = 0.84 compared to 0.75). There were no significant treatment effects upon exercise RQs. There was no effect of meals or diets on the substrate utilization during exercise at 80% maxOXC. During rest, the carbohydrate meal caused a 3 to 4 fold increase in the blood lactate concentration. During 40 min of recovery from exercise at 80% maxOXC after the control, protein and fat meals, the x for lactate decreased from 3 to 1 mM. However, following the carbohydrate meal, the x for lactate remained about 2 mM.

ACKNOWLEDGMENTS

The author wishes to express his appreciation to his major professor, Dr. R. Schemmel and to Dr. W. VanHuss, for their guidance, suggestions, and support throughout the research program and for their assistance in the preparation of the dissertation.

Appreciation is expressed to Dr. W. Chenoweth, Dr. A. Sparrow, and Dr. W.G. Bergen for serving on the guidance committee. Special thanks are expressed to M.B. Savinski and L. Coleman for assistance with the research and to B. Andrea, D. Ankrap, B. Foran, C. Hoover, B. Hughes, G. Hunter, R. Kimbell, D.G. Kirsch, M. Todd, T. Riebow, T. Talsma, P. Thiry, C. Vossekuil, J. Snow, E.L. Andavan and K. Luxton for serving as subjects.

The author is especially grateful to his wife, Cheri, for her understanding and assistance with typing.

ii

TABLE OF CONTENTS

	Page
INTRODUCTION	1
CHAPTER 1 - LITERATURE REVIEW	5
Introduction	6 7 10 13 13 14 16 17 18 18 18 19 22 28 28 31
CHAPTER 2 - THE VARIABILITY OF THE HEART RATE-OXYGEN CONSUMP- TION RELATIONSHIP WITH TIME AND COMPARISON OF INDIVIDUAL REGRESSION LINES VS A COMMON REGRES-	
SION LINE FOR ALL SUBJECTS	33 34 35 37 37 37 37 39
Test protocol	39 40 40 41 49 51

Page

CHAPTER 3 - EFFECT OF DIET, PREVIOUS EXERCISE, AND RECOVERY	
UPON THE LINEAR REGRESSION BETWEEN HEART AND	
OXYGEN CONSUMPTION	58
Synopsis	59
	60
Methods	62
	62
Informed consent.	62
	62
	66
Fredsurencents	66
Exercise tests	68
Statistical analysis	69 71
Results	71
Effect of diet on the HROXCR during exercise	71
Effect of previous exercise on HROXCR	71
Effect of recovery on HROXCR	74
Discussion.	104
Effect of diet and previous exercise	104
Recovery from exercise and HROXCR	105
CURDIED A _ DIETROV INDUCED JUEDMOGENEERIE DOUDING DEET AND	
CHAPTER 4 - DIETARY INDUCED THERMOGENESIS DURING REST AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION	
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN	110
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE	
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE	111
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE	111 112
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE	111 112 115
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis. . Introduction. . Methods . Subjects. .	111 112 115 115
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE	111 112 115 115 115
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis. . Introduction. . Methods . Subjects. . Informed consent. . Diets .	111 112 115 115 115 115 115
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 115 119
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 115 119 120
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 119 120 121
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 119 120 121 124
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 119 120 121 124 125
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 119 120 121 124 125 125
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 119 120 121 124 125 125 132
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 115 119 120 121 124 125 125 132 136
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 119 120 121 124 125 125 125 132 136 136
EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE. Synopsis.	111 112 115 115 115 115 115 119 120 121 124 125 125 132 136

Page

CHAF	PTER	5 -	EF	FE	CT C)F	ME	AL:	S	AN	ID	DI	ET	F ()N	PC)S1	E)	(EF	RCI	SE	E	BLC	00)		
			LA	ACT/	ATE	LE	VE	LS	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	143
	Sync	opsi	s.					•	•	•	•	•	•	•	•	•	•	•		•	•	•	•		•		144
	Inti	rodu	cti	ion.		•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•		145
	Metl	nods	•	• •		•	•	•	•		•	•	•	•	•	•	•	•		•	•		•	•	•	•	147
		Sub	jec	ts.		•	•	•	•	•		•	•		•	•	•	•		•		•	•	•	•	•	147
					cor																						147
																											147
					ents																						151
					tes																						152
					ntal																						153
					cal																						156
	Resu																										157
	Disc																										166
	0130	,uss	101		••	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	100
CHAF	PTER	6 -	CC)NCI	USI	ON	S	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	171
	BIBI	IOG	RAF	γHY.	• •	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	175
	APPI	ENDI	CES	5		•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•	•		•	•	197

LIST OF TABLES

Page

72

.

CHAPTER 2 -	THE VARIABILITY OF THE HEART RATE-OXYGEN CONSUMP- TION RELATIONSHIP WITH TIME AND COMPARISON OF INDIVIDUAL REGRESSION LINES VS A COMMON REGRES- SION LINE FOR ALL SUBJECTS.	33
1	Age, height, weight, VO2 max for the ten subjects. The slopes B_1 and y-intercepts B_0 and the corresponding variances based on all ten trials in each subject is given	38
2	Calculated F-ratio for test of homogenity within the ten trials in each subject with observed HR (F-1) and with corrected HR (F-2)	46
3	Repeatability R, calculated and critical F-ratio $(\alpha=0.05)$ for the slope B], the y-intercept B0, the y-intercept corrected for resting heart rate B0 Cor, and B1-1 and B0-1 Cor when the subject with a large deviation in the max VO2 is disregarded	47
CHAPTER 3 -	EFFECT OF DIET, PREVIOUS EXERCISE, AND RECOVERY UPON THE LINEAR REGRESSION BETWEEN HEART RATE AND OXYGEN CONSUMPTION	58
1	Physical characteristics of the two groups of five adult male subjects	63
2	Relative intensity of exercise for various tread- mill settings used in incremental exercise tests.	63
3	Composition of meals and diets. The energy con- tent of the diets varied between 2,100 and 2,400 kcal for the different subjects	64
4	Test of significant difference between the regression equations between HR and OXC for the five treatments and the regression equation for	

. . . .

control

Page

5	Test of significance between the regression equations for two subjects exercised twice, one hour apart. Results are included with the regression lines corrected for the resting heart rate (cor)	73
6	Comparison of mean recovery values and their respective standard deviation (SD) for eight trials in each of five subjects	79
7	Mean recovery increamental heart rates after exercise on a treadmill at 4 different speeds, each speed maintained for 10 and 30 minutes, respectively. Mean of four subjects	89
8	Mean recovery oxygen consumption in four sub- jects after exercise on a treadmill at four different speeds, two runs at each speed for 10 and 30 minutes, respectively	90
9	Mean predicted oxygen consumption during recovery following exercise on a treadmill at four differ- ent speeds in four subjects. Each speed is repeated twice for 10 and 30 minutes, respec- tively. Time is given in minutes. Standard deviation (SD) is also given	91
10	Mean difference (4 subjects) between the pre- dicted oxygen consumption and the actual oxygen consumption during recovery after running on a treadmill at four different speeds. The subjects ran at each speed twice for 10 and 30 minutes, respectively	92
11	Mean values of 95% lower confidence limit (LCL) for predicted oxygen consumption (POXC), recovery oxygen consumption (ROXC), and the difference (DLCL) between LCL and ROXC in ml/kg/min follow- ing exercise on a treadmill at four different speeds (S) for 10 and 30 minutes (D), respec- tively. Mean of four subjects	101
12	Test of significance for IRHR, ROXC and DOXC	103

Page

CHAPTER 4 -	DIETARY INDUCED THERMOGENESIS DURING REST AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE	110
1	Physical characteristics of the two groups of five adult male subjects	116
2	Composition of meals and diets. The energy con- tent of the diets varied between 2,100 and 2,400 kcal for the different subjects	117
3	Mean oxygen consumption following the ingestion of various meals for 5 male subjects in the resting state. Control was the postabsorptive state	126
4	Mean respiratory quotient following the ingestion following ingestion of various meals for 5 male subjects in the resting state. Control was the postabsorptive state	127
5	Mean correlation coefficient (4) between HR and OXC during control, following a fat meal, pro- tein meal, and a carbohydrate meal. Number of subjects equal to 6 for control, 7 for fat meal, and 8 for carbohydrate meal. n=30 in each treat- ment for each subject. r=mean value for the subjects completing the treatment	135
CHAPTER 5 -	EFFECT OF MEALS AND DIET ON POSTEXERCISE BLOOD LACTATE LEVELS	143
١	Physical characteristics of the two groups of five adult male subjects	148
2	Composition of meals and diets. The energy con- tent of the diets varied between 2,100 and 2,400 kcal for the different subjects	149
3	Blood lactate in arterialized blood for 6 hours following a carbohydrate meal, and in the post- absorptive state following an overnight fast as control. Mean of five subjects	158

Page

4	Mean blood lactate concentration during 40 minutes recovery from 20 minutes of exer- cise at 80% of maximal OXC in 5 subjects	163
5	Bonferroni t for test of significance of post- exercise blood lactate concentration. CH-M is tested for interaction (between 5 and 40 minutes) against C and F-M. Total (40 minute) response to CH-M is tested against total res- ponse to F-M; and the postabsorptive (P) trials (C, F-D, CH-D) are tested against the absorp- tive (A) trials (F-M and P-M) at 5 minutes postexercise. There are four contrasts	
	(n=4)	164

.

LIST OF FIGURES

CHAPTER 2 -	THE VARIABILITY OF THE HEART RATE-OXYGEN CON- SUMPTION RELATIONSHIP WITH TIME AND COMPARI- SON OF INVIDIDUAL REGRESSION LINES VS A COMMON REGRESSION LINE FOR ALL SUBJECTS	33
1	Regression line between the OXC and the HR for a typical subject (#8) based on a single incremental exercise test.	42
2	Regression line between OXC and HR in a typical subject (#8) based on ten incremental exercise tests	44
3	Regression lines between HR and OXC for a hypothetical cases	53
4	Incremental and corrected HR	55
CHAPTER 3 -	EFFECT OF DIET, PREVIOUS EXERCISE, AND RECOVERY UPON THE LINEAR REGRESSION BETWEEN HEART RATE AND OXYGEN CONSUMPTION	58
1	Two regression lines based on exercise trials one hour apart in one subject	75
2	Predicted OXC (POXC), lower confidence limits (LCL) and recovery OXC (ROXC) as a function of recovery HR	77
3	HR during 40-minute recovery. Mean of five subjects with eight exercise tests in each	80
4	Predicted OXC (POXC), upper curve. Lower 95% confidence level for OXC (LCL), middle curve. Recovery OXC (ROXC), lower curve, as a function of time	82
5	Difference between predicted OXC and recovery OXC (DOXC) as a function of time during 40 min- ute recovery	84

Page

6	DOXC as % of POXC vs recovery time (40- minute recovery)	87
7	Mean incremental HR (IHR) for four subjects during 40-minute recovery after exercise for 10 minutes at speed 4 (8.8 km/hr), lower curve; and speed 6 (12.5 km/hr), upper curve	93
8	Incremental HR during 40-minute recovery (RIHR) after exercise at speed 5 (10.5 km/hr)	95
9	Recovery OXC (ROXC) as a function of time	97
10	Mean DOXC (difference between predicted and recovery OXC for four subjects	9 9
CHAPTER 4 -	DIETARY INDUCED THERMOGENESIS DURING REST AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE	110
1	OXC as a function of time	128
2	Respiratory Quotient (R.Q.) as a function of time for CH-M and Control (C)	130
3	Blood lactate as a function of time after a CH-M and C	133
CHAPTER 5 -	EFFECT OF MEALS AND DIET ON POSTEXERCISE BLOOD LACTATE LEVELS	143
1	Blood lactate concentration (mean of five sub- jects) in the postabsorptive state (following an overnight fast) as control (lower line) and following a 2.94 MJ carbohydrate meal (bananas) .	159
2	Blood lactate concentration during recovery from exercise at 80% of maximal oxygen consumption for 20 minutes (mean of five subjects)	161

INTRODUCTION

In order to evaluate the energy balance, it is necessary to have an accurate method for measuring both the energy intake and energy expenditure in free living individuals; but at present there is no generally accepted method for measuring the energy expenditure (Acheson et al. 1980). The oxygen consumption (OXC) is an indirect method for measuring the energy expenditure. The heart rate (HR) has been used as a predictor of OXC in many studies (Bradfield, 1971) because it is simple to use. As pointed out by Durnin (1977), the correlation between the HR and OXChas not been thoroughly evaluated. The possible variation between tests of the linear regression between the HR and OXC has generally not been considered since the regression coefficient between the HR and OXC in a given experiment is very high. This was also true in our experiments where a continuous test with stepwise increased work rate was evaluated r = 0.99. However, this does not mean that one will obtain the same correlation line if the test is repeated. Furthermore, it does not quarantee that HR and OXC will be correlated if only small changes in workload are considered, such as that observed in the resting state. Since many people spend most of their time at relatively low levels of activity it is important to know whether the HR can predict the OXC at rest and during low work loads.

Another question that has not been answered is whether the regression equation for HR and OXC obtained during exercise is also valid during the recovery period following exercise. This is important,

since most subjects probably spend more time recovering from physical activity than actually engaged in it. If the regression equation obtained during exercise is still valid during recovery, this implies that HR and OXC return to the resting state at the same speed. While the OXC in low intensity exercise has been shown to return to resting values within 1-3 minutes, the HR recovery was slower (Linnarsson, 1974). Thus, it is important to evaluate the time relationship between HR and OXC during recovery and the effects of intensity and duration of the exercise.

Diet is often mentioned as a factor that may alter the HROXCR (Durnin, 1978). Physical activity, however, rather than diet, was found by Lundgren (1946) to be the factor responsible. The effect of exercise and dietary modifications was therefore included in the study.

The term "specific dynamic effect" goes back to Rubner (1902). Recently, however, Garrow and Hawes (1972) have forwarded the view that all the energy sources (fat, carbohydrates and proteins) have an equal thermogenic effect. Based primarily on overfeeding experiments in pigs, Gurr et al. (1980) have concluded that it is carbohydrate rather than protein that exerts the primary thermogenic effect. There is presently much research activity (James and Trayhurn, 1981; Rothwell and Stock, 1979) in dietary induced thermogenesis (DIT). This is because variations in DIT are thought to be an explanation for obesity in many people. However, a criticism of many of these studies is that physical activity is not well controlled. When a thermogenic response is measured as an increase in OXC or heat production, it takes very little activity to exceed the thermogenic effect of a meal. The

thermogenic response to a fat, protein and carbohydrate meal was therefore determined in human subjects by measuring the increase in OXC in the resting state.

Many investigators believe that exercise will augment the thermogenic response to meals and/or overeating (Bray et al, 1974); however, others (Norgan and Durwin, 1980; Glick et al, 1977) have found no such effect. It is possible, as pointed out by Bray, et al (1974) that there is a critical meal size below which no effect is seen. An increased thermogenic response to a normal sized meal could have important implications for weight control and was therefore investigated in the present study.

It has been shown that a carbohydrate-free diet increases fat utilization during exercise compared to a carbohydrate-rich diet (Bergstrom et al, 1967). It is therefore generally assumed that diet can modify substrate utilization; but the experimental evidence is conflicting. Hurni et al (1982) found an effect at rest but not during exercise. Ahlborg and Felig (1977) found an effect during low intensity exercise. Costill et al (1977) by elevating plasma FFA with fat meal and heparin found an effect during high intensity exercise; whereas Bergstrom et al (1969) found an effect during low but not high intensity exercise. Finally, Brooke (1981) found no effect regardless of the exercise intensity. However, it has been shown that when glycogen is depleted there is an effect of carbohydrate upon the carbohydrate utilization during exercise (Maughan et al, 1978; B^Onen et al, 1981). It was therefore decided to study the effects of fat, carbohydrates and protein meals upon the substrate utilization without

affecting the glycogen stores during exercise at 80% of the maximal OXC.

The concept of a percentage of maximal oxygen consumption as an equalizer of the exercise load is almost universally used. It is, however, often criticized because people with the same maximal oxygen consumption may be in different degrees of endurance training. For this reason, many investigators have considered the use of blood lactate as an equalizer of the work load (Katz et al, 1978). If the exercise intensity is increased gradually, the blood lactate will increase slowly at first until it reaches about 2 mM/1 (40-60% of maximal oxygen consumption). It then increases somewhat faster until it reaches about 4 mM/l (65-90% of maximal oxygen consumption), at which time it begins to increase very rapidly. Two and 4 mM/llactate is arbitrarily defined as the aerobic and anaerobic thresholds, respectively (Skinner and McLellan, 1980). These levels of blood lactate, however, are of the same order as one could expect following a carbohydrate load (Owen et al, 1980); and diet, therefore, presumably could affect these thresholds without having any effect upon the aerobic/anaerobic metabolism. For this reason, blood lactate was investigated during recovery from exercise at 80% of maximal oxygen consumption following different dietary treatments.

LITERATURE REVIEW

. CHAPTER 1

LITERATURE REVIEW

INTRODUCTION

The measurement of the work output component by the heart rate (HR) method and some aspects of dietary induced thermogenesis will be the subject of this review. It will also include the effect of diet upon substrate utilization during rest and exercise, and dietary effects upon whole blood lactate production during rest and exercise.

THE ENERGY EQUATION

A simplified energy balance can be written as follows (Brobeck, 1981):

Food Intake = Work Output + Heat Loss + Change in Energy Stores As Brody (1945) pointed out, provided an animal stays alive, body weight will eventually stabilize at some level. At this level, food intake has equilibrated with the other three components of the equation. The conventional concept that the body energy stores are controlled by a single set point, such as the size of the fat stores, has recently been challenged (Brobeck, 1981). It is argued that any set point depends upon a number of factors: (1) dietary composition, (2) work output, and (3) environmental temperature. Regardless of the set point, the above equation remains true at all times, provided all the terms are determined at the same point in time.

It should be mentioned that the change in energy stores are not necessarily equivalent to a change in body mass, because different energy stores have different energy density (Garrow, 1974). The term "heat loss" is conveniently divided into several distinct components:

Heat Loss = RMR + CIT + DIT + TISE + LUXC

where:

RMR = Resting metabolic rate

CIT = Cold induced thermogenesis

DIT = Dietary induced thermogenesis

TISE = Energy required to build new tissue and store energy

LUXC = Luxus consumption

Most of these terms are well known, but a few require further comment. The basal metabolic rate (BMR) is well known, but because of difficulties in measuring it, RMR is often used instead (Dauncey, 1979). One cannot expect deposition of energy and synthesis of new tissue to be 100 percent effective, and consequently, Norgan and Durnin (1980) have proposed the TISE to account for the cost of change in tissue and energy stores. The term LUXC was proposed to account for the fact that overfeeding did not result in the expected weight gain (Apfelbaum, et al, 1971). The term, however, is very controversial (Norgan and Durnin, 1980).

HEART RATE AS A MEASURE OF ENERGY EXPENDITURE

It is well known that the HR is linearly related to the oxygen consumption (OXC) (Astrand and Rodahl, 1977). This should not be

interpreted as a causative relationship, since other factors affect the delivery of oxygen to the peripheral tissue. These factors include arterial-venous oxygen difference (AVOD), stroke volume (SV), and hemoglobin concentration (HEMC).

$$OXC = HR \times SV \times AVOD$$

Astrand, et al (1964) found that the AVOD increased gradually from about 7 ml $0_2/100$ ml blood at rest to 17 ml $0_2/100$ ml blood during maximal exercise, and that the SV increased from 63% of maximal volume at rest to 100% at 40% of maximal OXC, and then remained constant as the exercise load was increased further.

It has been shown that the body position affects the relation between HR and OXC (HROXCR) (Andrews, 1969; Sato and Tanaka, 1973; Vokac, et al, 1975). The primary factor here appears to be whether or not the legs are involved in the exercise, because of the effect venous pooling in the legs has on the venous return, and thereby, presumably, on the SV.

Another factor that affects the HROXCR is environmental temperature (Williams, et al, 1962; Rowell, et al, 1966; Myhre, et al, 1979; and Studier, et al, 1975). The HR increases at both lower and higher temperatures with a minimum HR at about 20° C. While the cardiac output (CO) is unchanged at elevated temperatures, the SV is decreased with a corresponding increase in the HR.

Training also appears to affect the HROXCR (Saltin, et al, 1968; Pannier, et al, 1980; Taylor, et al, 1963). Training generally decreases the resting HR and increases the blood volume, and thereby

presumably the SV. Its effect upon the HROXCR is to increase the slope and decrease the OXC at a given HR.

Sex and age are two factors that affect the HROXCR (Dill and Consolazio, 1962; Montoye, et al, 1968; and Sheffield, et al, 1978). HR generally decreases with age in both sexes and it is higher in females than in males, which may partly be a training effect. As a result, women generally have less slope on the HROXCR than men. The effect of age is unknown because both HR and OXC decrease with age.

Stress and added weight are also known to have an effect on the HROXCR (Taylor, et al, 1963; Saltin, et al, 1968; and Borghols, et al, 1978). As expected, the effect of added weight would cause a linear increase in both the HR and OXC, and presumably there will be no change in the regression line if the extra weight is taken into account. The effect of stress, in general, is not well studied, but presumably it will have an effect upon both HR and OXC. However, it may still change the regression line, and in certain situations, it has been shown to have an effect upon the HROXCR.

It is still controversial whether it is better to use an individual calibration line for the HROXCR for each subject or to use a common calibration line for all subjects (Bradfield, et al, 1970; Andrews, 1971; Bradfield, 1971; Payne, et al, 1971; and Astrand and Rodahl, 1977). Andrews (1971) reported that using incremental HR (Actual HR - Resting HR) removed the differences among the subjects.

The effects of meals and exercise on HROXCR are little studied, but it has been reported that previous exercise affects the HROXCR, presumably by decreasing the SV; however, the duration of this effect is not

known. Conflicting results have been reported for the effect of meals on HROXCR; thus, Lundgren (1946) found it to be due to physical activity, but Schutz, et al (1981) have recently reported an effect of meals.

RELATION BETWEEN HEART RATE, STROKE VOLUME, AND CIRCULATION

There are many factors controlling the HR, but the central nervous system (CNS) plays a dominant role (Nyberg, 1981). Thus, during supine rest, the sympathetic nervous system contributes about 25% to the control of the HR, while the parasympathetic system dominates; but at maximal exercise, the sympathetic nervous system's contribution is virtually 100%. The parasympathetic contribution decreases simultaneously to almost zero at maximal exercise. During acute circulatory stress, such as exercise, the arterial pressure is controlled almost entirely by nervous reflex mechanisms (Guyton, 1981). However, it has been shown (Billman, et al, 1981) that the baroreflex control of the heart rate is reduced by central blood volume shifts (i.e., a smaller decrement of HR is obtained by a given systolic blood pressure increase, when central blood volume is increased).

It is well known that HR decreases with training (Lewis, et al, 1980). These authors found that the bradycardia of training was not due to an autonomic component, but that the decrease in HR paralleled a decrease in OXC during constant workload; and they concluded that it might be due to the cardiac enlargement. However, it has also been shown by some (Ekblom, et al, 1972; Gulbring, et al, 1960; and Pace, et al, 1947) that an increase in blood volume (as also occurs during exercise training) increases the oxygen delivering capacity, which

could operate through an effect on the SV. Apparently, however, this is not the case (Ekblom, et al, 1976); and the necessary adjustments are made in the oxygen extraction.

It has been observed that dehydration, whether caused by heat exposure or exercise, causes a decrease in plasma volume (Saltin, 1964a; Saltin, 1964b; and Costill and Fink, 1974). In submaximal exercise following the dehydration, SV was decreased and the HR increased, but at maximal exercise, there was no difference. Damoto, et al (1966) found that a change in body position from supine to standing caused a decrease in SV and an increase in HR, with only a slight decrease in cardiac output.

Roberts and Wenger (1979, 1980) have recently obtained similar results for the SV and HR. Additionally, they found that at high environmental temperatures, the skin blood flow increases, compared to cooler temperatures. This leads to an increased cutaneous blood flow in order to divert the metabolic heat. However, at high levels of skin blood flow, peripheral vascular pooling and fluid losses by filtration leads to reduced central venous pressure. This, in turn, lowers SV and increases HR. Reflexes which arise from receptors in working muscles produce vasoconstriction in a number of central and peripheral vascular beds. In the long term, physical conditioning and heat acclimation lead to increases in sweat output during thermal stress, which decreases the loss of vascular volume due to exercise and heat stress (because of more effective cooling and less cutaneous pooling of blood).

Senay (1970) and Gaebelein and Senay (1980) have likewise found that endurance training results in modification of the vascular dynamics

during exercise; that is, vascular volume becomes stabilized. In the untrained individual, heat exposure exaggerates body fluid shifts during exercise. With training, stability of the vascular volume is attained during heat exposure, but maximum protective responses towards exercise in heat are only gained upon heat acclimation.

It has been shown (Nadel, 1980; and Nadel, et al, 1980) that plasma volume is lost continuously throughout exercise, particularly at higher intensities, but the loss is most drastic during the first six minutes (at 70% of maximal OXC). This may be the reason why the HR continues to increase throughout exercise (80% maximal OXC), but most drastically at the beginning (Tanaka, et al, 1979) and does not return to preexercise levels until after 45 minutes of recovery. There are mechanisms to compensate for loss of vascular volume during exercise. Thus, vasoconstriction in the splanchnic (Rowell, et al, 1968) and renal (Grimby, 1965) vascular beds has been observed during exercise. Further reduction of blood flow to the non-exercising muscle also occurs (Johnson and Rowell, 1975). Finally, it has been demonstrated that during heavy exercise, as it becomes increasingly difficult to compensate for loss of vascular volume by increased HR, a cutaneous vasoconstriction superimposes itself upon the vasodilator drive at a certain point as skin blood flow increases (Brengelmann, et al, 1977; Johnson, et al, 1974; and Nadel, et al, 1979).

At a given intensity of exercise, it has been demonstrated (Nielsen, et al, 1971; Greenleaf and Castle, 1971; and Nadel, et al, 1980) that the internal body temperature is higher in dehydrated subjects compared to normally hydrated subjects at a given work

intensity. It has also been demonstrated that dehydration causes a decrease in SV during exercise (Saltin, 1964b; and Nielsen, et al, 1971). Thus, the circulatory instability during exercise depends upon the initial state of hydration (Nadel, et al, 1980). Therefore, the variation in total body water with muscle glycogen changes may become important (Olsson and Saltin, 1970).

THERMOGENESIS

Thermogenesis has recently received considerable interest because of its possible role in the regulation of body weight or body energy stores. It is believed that the overweight of genetically obese mice (ob/ob) is primarily due to decreased energy expenditure (Trayhurn, et al, 1979); and the ability of young lean rats to prevent obesity is probably due to increased dietary induced thermogenesis (DIT) (Rothwell and Stock, 1979).

Non-shivering Thermogenesis

Shivering involves the contraction of the muscles in order to produce heat. Non-shivering thermogenesis (NST), on the other hand, takes place without muscular contraction. Jansky (1973) has divided NST into basal NST and regulatory NST. The basal NST constitutes the heat production involved in BMR, while the regulatory NST is produced in response to specific thermoregulatory requirements. It has not been determined if DIT is regulatory or basal (Dauncey, 1979).

Cold-induced Thermogenesis

It is sometimes assumed that cold induced thermogenesis (CIT) need not be considered in man, since he does not normally expose himself to

cold severe enough to induce a metabolic response (Garrow, 1978). However, Dauncy (1979) found a significant thermogenic response in man due to slight (6^oC) changes in ambient temperature, which indicates that man at least has the capacity for CIT (NST). Obese people have more thermal insulation than lean counterparts, which results in a reduced heat loss. And obese individuals appear to have a lower metabolic rate in the cold, compared to normal individuals (Buskirk, et al, 1963; Jequier, et al, 1974; and Keatinge, 1960). By analogy to obese mice (ob/ob), this, however, may be due to a decreased CIT (Trayhurn, et al, 1979). The increased metabolic rate of Eskimos in the cold may be a result of their high protein diet (Heinbecker, 1931). Dietary-induced Thermogenesis (DIT)

A number of terms have been used for DIT: post-prandial thermogenesis, specific dynamic action, and luxus-consumption. However, each covers more or less well-defined aspects of DIT. Thus, postprandial thermogenesis refers to the increased metabolic rate which follows a meal; specific dynamic action is the response in metabolic rate to a protein meal; and luxus consumption relates to the presumed increase in heat production following overeating. DIT covers both the immediate and long-term increase in heat production directly related to feeding, and this term is commonly used today. The basal DIT consists of the inevitable energy cost of digesting, absorbing, and processing or storing substrates, such as conversion of glucose to glycogen or fat and its storage in tissue, which may need to be synthesized. LUXC, on the other hand, is purely regulatory, if it exists at all (Trayhurn and James, 1981; James and Trayhurn, 1981).

The most convincing evidence in support of LUXC comes from a study in pigs (Miller and Payne, 1962) fed a low protein diet. On the low protein diets, the piglets ate five times as much energy as the ones on a high protein diet, without gaining any more weight. These studies have recently been repeated by Gurr, et al (1980). Similar experiments have also been carried out in rats fed a "cafeteria diet" (Rothwell and Stock, 1979). These authors also found that insulin plays a role in the DIT (Rothwell and Stock, 1981). This phenomenon has also been found in adult human subjects (Miller and Mumford, 1967). In some of the animal studies, there is reason to seriously question if the activity of the animals was controlled. Thus, Gurr, et al (1980) states that activity could not be involved, since the pigs were chained. However, it is easy to conceive that the chaining could cause the animals to have even more muscular activity; and there is evidence that diet may affect activity (Hart, 1978; Schemmel, 1967). The study in piglets (Gurr, et al, 1980) was also carried out in older pigs, and there, the excess energy intake on a low carbohydrate diet was stored as fat. In the cafeteria-fed rats (Rothwell and Stock, 1979), an increased metabolic response to norepinephrine is also seen in cold acclimatized rats; and warm adapted rats cease shivering much sooner when exposed to cold after being fed a "cafeteria diet" compared to a chow diet. This has led to the view that CIT and DIT are based on a similar mechanism (Trayhurn and James, 1981).

Control Mechanisms

Brown Adipose Tissue

According to recent studies by Foster and Frydman (1978a, 1978b, and 1979), brown adipose tissue (BAT) is the major site of NST, while skeletal muscle plays little or no role. According to the works of Nicholls (1979), thermogenesis is initiated by the release of norepinephrine, which binds to a receptor on the plasma membrane of the brown adipocyte. Adenyl cyclase is then activated and C-AMP is produced. This in turn activates the triacylglycerol lipase which causes release of FFA and glycerol. When the FFA's are oxidized in the mitochondria, protons (H+) are produced which pass through the mitochondrial membrane. Normally the passage of H+ back through the membrane is linked to the synthesis of ATP, but in BAT, the H+ gradient can be dissipated by the movement of H+ through a proton conductance pathway, without ATP synthesis in association with a specific protein (uncoupling protein) unique to BAT. Joy (1963) has demonstrated a metabolic response of cold acclimatized subjects to NE.

Hormones

Kuroshima and Yahata (1979) have shown that glucagon has twice the thermogenic response of norepinephrine, and that unlike the response to NE, the glucagon response is not affected by cold acclimatation (although reduced by heat acclimatation).

Danforth and Burger (1981) state that the two hormones involved in regulation of thermogenesis are thyroid hormone (T_3) and centrally released catecholamines (NE). They seem to work in combination to regulate cellular thermogenesis. T₃ appears to be responsible for

slow adjustments, while NE with its fast disappearance, appears to regulate the fast adjustment in thermogenesis. Thyroid hormones appear to regulate the sensitivity of BAT to NE. Shetty, et al (1979) found that during energy restriction, when the calorigenic hormones (T_3 and NE) decrease, there was a decrease in thermogenesis. But when levadopa (a precursor of NE) was given, there was no decrease in BMR, despite a fall in T_3 . This indicates that NE is the primary short-term regulator of thermogenesis.

Thermogenesis and Exercise

Miller, et al (1967) found an almost 60% increase in energy expenditure during exercise following a 4.75 MJ meal. The subjects participated in a mild stepping exercise (mounting an 11-inch step 12 times per minute for 30 minutes). Apfelbaum, et al (1971) likewise found an increase in energy expenditure (20-30%) during exercise following a 6.2 MJ supplement per day for 15 days. Bray, et al (1974) reported that breakfasts of 4.2 or 12.6 MJ increased the RMR by 10% and the metabolic rate following exercise by 20%. Neither response was affected by the size of the meal. Bray, et al (1974) found no thermic effect during exercise after overeating (16 MJ/day) for 30 days. In contrast, Swindells (1972), found no effect of exercise following meals of 2.5-3.8 MJ. Bray, et al (1974) has concluded that this may be due to the smaller size of their meals. Norgan and Durnin (1980), during a six-week over-feeding study, found no thermic effect during exercise, when the weight gain was taken into consideration.

SUBSTRATE UTILIZATION

It is well established both from the respiratory quotient (RQ) (Christensen and Hansen, 1939) and from 14 C turnover studies (Ahlborg, et al, 1974) that in the resting postabsorptive state FFA is the major energy substrate. During rest as well as exercise, it is, however, necessary to consider the availability of substrate. During exercise it also becomes necessary to account for both the type of exercise, and its duration and intensity. Furthermore, the two major substrates (fat and carbohydrate) can be supplied intramuscularly or through the blood.

Glycogen

Above 90% of maximal oxygen consumption, glycogen is the most important substrate, but it is far from depleted at exhaustion due to the relatively short intensity which exercise of this duration can be sustained (Hultman & Bergstrom, 1973). At 70-80% of maximal oxygen consumption, glycogen concentration decreases in a curvilinear fashion and the time of exhaustion often coincides with glycogen depletion (Hultman and Bergstrom, 1973). Below 60% of maximal oxygen consumption, muscle glycogen, however, is not depleted at exhaustion; but liver glycogen may become a limiting factor because blood glucose is a very important substrate (Hultman and Nilsson, 1973).

Intramuscular Lipids

Below 60% of maximal oxygen consumption, FFA is the most important substrate; however, blood born FFA only accounts for about 50% of the total lipid oxidation, and the rest is derived from local lipid stores (intra- and intermuscular lipid stores). It has also been

shown that utilization of blood born FFA is proportional to the arterial concentration (Hagenfeldt and Wahren, 1972; Essen, 1977). Intramuscular lipid stores appear to supply FFA whenever blood born substrates are not available in sufficient quantity.

Different modes of exercise involve different muscles to different degrees. The higher the work intensity, the more the type II fibers are recruited, and it has been shown that type II fibers are more likely to use glycogen as substrate than type I fibers. Furthermore, type I fibers can continue to use intramuscular fat as substrate even if the glycogen stores are depleted (Essen, 1977).

Control of Substrate Utilization

Glucose

Several reactions in glycolysis are nonequilibrium: glucose transport, hexokinase, phosphofructokinase, and pyruvate kinase; and none of these pathways appear to be substrate saturated. Thus, blood glucose appears to be the flux generating step; the blood glucose, in turn, provides a feedback link between the glucose utilization and the rate of glycogenolysis in the liver (Newsholme and Crabtree, 1979).

The muscle hexokinase shows product inhibition (G-6-P), whereas inorganic phosphate (Pi) is an activator. Thus, when G-6-P builds up in the cell, hexokinase is inhibited and glucose concentration will increase until it reaches equilibrium with the plasma level. The phosphofructokinase (PFK) is activated by ADP, AMP, Pi and creatine-P and inhibited by the ATP/F-6-P ratio and creatine-P. Thus, glucose transport, hexokinase and PFK act in concert to control the flux of

glucose (Newsholme and Start, 1973; Newsholme, 1977; Newsholme and Crabtree, 1979).

Glycogen

Glycogen utilization is regulated by the enzyme phosphorylase, which exists in two forms (a and b). Phosphorylase b is under metabolite control: ATP and G-G-P inhibit, whereas AMP and Pi activate. Thus, its activity is increased in concert with PFK to insure adequate substrate supply if the demand is higher than can be supplied by blood glucose and FFA. Under the influence of epinephrine (E) and nervous impulse, phosphorylase b is converted to phosphorylase a, which always has maximal activity; the result being that during exercise of high intensity (with release of NE from CNS and E from the adrenal medulla); particularly at the onset of exercise, glycogenolysis is fully activated (Foulkes and Cohen, 1979; Galbo, et al, 1977; Essen, 1978; Newsholme, 1977). The glycogenolysis during heavy exercise is of sufficient magnitude to prevent utilization of blood glucose and can actually lead to a net release of glucose from the working muscle (Wahren, 1970; Ahlborg, et al, 1974; Essen, 1978).

FFA

Citrate generated by the metabolism of FFA will inhibit PFK, and, therefore, glucose utilization as long as sufficient substrate (FFA) to cover the demand is present. As the intensity of exercise increases, sufficient oxidation of FFA is no longer possible and AMP, NH_4^+ , Pi, and FDP increase and release PFK from the citrate inhibition, thereby facilitating the increase in glucose utilization (Newsholme, 1977).

Hormonal Control

It has been shown that E increases C-AMP which activates protein kinase that phosphorylates a phosphorylase kinase, which in turn converts the phosphorylase a to phosphorylase b. Recently, it has also been demonstrated that E also causes phosphorylation of a protein (inhibitor-1) which inhibits the phosphorylase phosphatase that dephosphorylate phosphorylase a to phosphorylase b. The above reactions are also stimulated by CNS stimulus (Foulkes and Cohen, 1979).

Insulin decreases during exercise, and the decrease appears to be caused by the increased α -adrenergic activity, which inhibits the insulin secretion. Glucagon, on the other hand, increases during prolonged exercise, and this increase correlates with a decrease in the blood glucose concentration. At the same time, there is an increase in adrenal E, and these hormones also control the output of glucose from the liver. NE and E increase with the intensity of exercise, while glucagon is primarily affected by the duration of exercise when blood glucose decreases. Exercise also causes changes in growth hormone and cortisol (Galbo, et al, 1977).

Other Factors

Caffeine has been shown to affect substrate utilization (by increased lipid oxidation, both during rest (Acheson, et al, 1980) and during exercise (Costill, et al, 1978), presumably by FFA mobilization. Training increases fat utilization (Johnson, et al, 1969). It is interesting to note that during heavy exercise, there is an increase in glucose utilization despite a decrease in the insulin/ glucagon ratio (Wahren, 1979). The fall in insulin during exercise

decreases with training (Wirth, et al, 1979). During exercise, kinin is liberated from kininogen and this has been shown to greatly increase the effect of insulin on glucose uptake by skeletal muscle in man (Dietze, et al, 1980). Nicotinic acid decreases FFA mobilization from adipose tissue, and heparin increases FFA mobilization. Costill, et al (1977) found that elevating plasma FFA with heparin at 70% maximal oxygen consumption decreased the rate of muscle glycogen utilization by 40%. However, experiments where adipose tissue lypolysis was blocked with heparin, showed there was no effect on glycogen utilization at high intensity work (Bergstrom, et al, 1969). It is possible that greatly elevated FFA (by heparin in combination with a fat meal) does increase, even though a decreased blood FFA does not affect fat oxidation at high intensity work.

Effect of Meals and Exercise on Substrate Utilization

Hurni, et al (1982) found that during rest the diet quickly affects the substrate utilization, but during exercise of relatively low intensity, it had no effect upon the substrate used. As will be described in the following, many investigators have found an effect of diet upon substrate utilization during exercise; however, it is necessary to consider whether or not the glycogen stores were affected, as well as the intensity of exercise. Hurni, et al (1982) used a whole body calorimeter for determination of RQ, and since the subjects were fed regularly, there is no reason to expect a significant effect upon the glycogen stores.

Effect of Meals on Blood Substrate Levels

Stock (1980) found that a 1.67 MJ meal after one day of fasting significantly decreased the blood FFA level and increased blood glucose. Owen, et al (1980) similarly found that after a typical American breakfast (3.2 MJ), blood glucose, lactate, and triglycerides increased for 2-3 hours; whereas blood levels of FFA were depressed for about 4 hours. Alanine and total amino acids were also increased, while urea nitrogen was depressed for at least four hours. Crapo, et al (1981) likewise found an increase in blood glucose and insulin, while FFA were depressed following a standard meal.

Low Intensity Exercise Without Any Change in Muscle Glycogen

Luyckx, et al (1978) studied the effect of 100 g of orally ingested glucose upon plasma glucagon, FFA, and insulin. During rest, they found the expected increase in blood glucose and insulin, and a decrease in FFA and glucagon. The glucose was ingested during exercise (50% maximal oxygen consumption) or before the start of exercise. When preexercise glucose was compared to no glucose intake, blood glucose was elevated the first two hours of the exercise period. From 2 to about 3½ hours, the blood glucose was depressed compared to control (no glucose), with a rebound after about 4 hours. As expected, plasma insulin fell in the control trial, but after the preexercise glucose meal, insulin rose in the same manner as in the resting state. After glucose, the plasma FFA was depressed in the same manner as during rest, whereas in the control trial, the FFA continued to rise throughout exercise. Preexercise glucose ingestion also prevented the expected increase in glucagon. When the glucose

was ingested after 15 minutes of exercise, the raise in blood glucose was reduced by about 1/3 compared to the preexercise glucose trial. Plasma insulin neither rose nor fell, and plasma FFA and glucagon rose, although less than during the control experiment.

Using the same experimental protocol with 100 g 13 C-glucose as metabolic tracer, Pirnay, et al (1977) found that ingestion of glucose after 15 minutes of walking on the treadmill did not affect protein utilization (1-2%), but increased the percentage of energy derived from glucose. It significantly reduced the utilization of endogenous glucose.

Ahlborg and Felig (1977) studied the effect of ingestion of 200 g of glucose on substrate utilization during exercise at 30% maximal oxygen consumption for 4 hours. They found that arterial glucose increased 35%, arterial glycerol decreased 65%, and FFA failed to increase for 2 hours of exercise in the glucose-fed group compared to control. Plasma insulin increased two- to three-fold, but glucagon levels decreased 70% compared to control. Glucose uptake in the leg (by catheterization) was increased 55% compared to 35% in controls, and splanchnic glucose output was about 110% increased, whereas splanchnic uptake of gluconeogenic precursors was decreased (80%) compared to control. So at low levels of exercise (<60% of maximal oxygen consumption), preexercise glucose increases glucose utilization and decreases fat oxidation, and decreased hepatic gluconeogenesis. The ingestion of glucose, while increasing total glucose utilization, spares the endogenous glucose. It appears to make a difference if the glucose is ingested before or during exercise, and these changes

are hormonally determined.

Low Intensity Exercise with Glycogen Depletion

It has been observed that a greater proportion of an oral glucose load escapes the hepatic retention in glycogen depleted subjects compared to control (Maughan, et al, 1978). This glucose is taken up preferentially by the depleted muscle. Rennie and Holloszy (1977), however, found that a high plasma FFA level, which occurs after glycogen depletion, inhibits muscular glucose uptake as well as its oxidation. Ravussin, et al (1979) studied the effect of exercise one hour following 100 g orally ingested ¹³C-glucose at 40% of maximal oxygen consumption in normal controls and glycogen depleted subjects. The major difference between the two groups was that the glycogen depleted subjects used primarily fat as substrate (70%), whereas the control subjects used primarily carbohydrate (65%). The exogenous glucose represented 20% and 24%, respectively. In the depleted subjects, plasma FFA remained 2-3 times higher than in control. So despite glycogen depletion, these subjects did not use exogenous carbohydrate to a higher extent.

During exercise following glycogen depletion (by prior exhaustive exercise) and either a normal-, low-, or high-carbohydrate diet, a lower RQ, blood lactate, blood glucose, and blood triglycerides were found compared to elevated plasma FFA and plasma glycerol following a low carbohydrate diet than a high CHO diet. The control (normal) was very similar to the high carbohydrate diet in all aspects studied (Maughan, et al, 1978).

High Work Intensity and Glycogen Depletion

Bonen, et al (1981) used a regimen consisting of exhaustive exercise followed by about 40 hours of fast to deplete muscle and liver glycogen. The authors then studied the response to ingestion of carbohydrate (1.5 g/kg) 15 minutes prior to exercise or 3-5 minutes after the start of exercise. An exercise group with no glucose intake, and a rest group with glucose were used as control. As one could expect, the exercising control had no increase in blood glucose, and the increase during exercise was less than during rest, both in regard to blood glucose and insulin. During these workloads (80% maximal oxygen consumption), the preexercise glucose group had an initially sharp increase in both glucose and insulin (prior to exercise) followed by a sharp decrease during exercise (25 minutes) and a moderate increase during recovery. The during exercise glucose group of course did not have the preexercise increase, but both blood glucose and insulin was very similar to the pre-exercise glucose group, during exercise, with a more sharp increase during recovery.

Maughan and Poole (1981) compared subjects with depleted, normal, and supercompensated glycogen stores at 105% of maximal oxygen consumption. Because of the rapid production of lactate at this high workload, it was not possible to determine substrate utilization (invalid RQ), but depleted subjects had lower blood glucose than normal and supercompensated subjects.

Martin, et al (1978) studied the effect of exhaustive exercise following a normal-, high carbohydrate-, and high-fat-diet for 3 days in

humans. The high fat diet provided 90% of energy from fat and the high carbohydrate diet, 75% of energy from carbohydrate. The effect of these dietary regimens on muscle glycogen are uncertain, but they may very well have had a significant effect, particularly since the exercise was exhausting. However, the high fat-protein diet had significantly lower RQ than either the mixed or high carbohydrate diet, and the blood concentration of FFA was elevated.

High Work Intensity Without Glycogen Depletion

Jones, et al (1980) compared heavy exercise (70% maximal oxygen consumption) with light exercise (40% maximal oxygen consumption) during 40 minutes. As expected, relatively less fat was used during the heavy work. During light work, palmitate turnover rate (14 C) was unchanged from rest, but a 40% decrease was observed during heavy work, as well as a fall in plasma FFA. However, during heavy work, plasma glycerol was increased (5-fold) compared to light work. This was interpreted to mean that heavy work caused a shift from adipose tissue lipolysis to muscle lipolysis.

Foster, et al (1979) compared the effect of 75 g of glucose taken before exercise with a standard liquid meal (composition similar to a normal American diet)(water was used as control) during exercise to exhaustion at 80 and 100% of maximal oxygen consumption. The authors concluded that FFA mobilization was impeded by glucose ingestion. They also found a significant decrease in serum glucose following the glucose meal after 30 minutes of exercise compared to control, despite a higher initial level. They further reported an increase in the RQ following the oral glucose load compared to control. This difference, however, was not

significant. Mainly on the strength of the decreased serum FFA, the authors concluded that a glucose meal increased utilization of glucose as substrate; it is, however, necessary to also consider intramuscular FFA mobilization, and the plasma level of substrates is no certain indication of substrate utilization during heavy exercise.

Conclusions

During rest, it is well established that the diet affects the substrate utilization, and this probably extends to exercise of low intensities (<60% of maximal oxygen consumption). However, during heavy exercise, there is little support for this hypothesis. During rest and low intensity exercise, blood borne substrates (FFA and glucose) are of major importance in the supply of energy, but during heavy exercise, intramuscular substrates assume an increasing role and this change is mediated through hormones (insulin, glucagon, and E) and CNS. As a consequence, one would not expect diet to have a major effect upon the substrate utilization unless intramuscular substrate is depleted; but the experimental evidence is inconclusive at present.

POSTEXERCISE LACTATE

Effect of Exercise

At the start of low level exercise, there is a rapid release of lactate from the exercising muscle (100-150 mmol/min/100 ml), which gradually decreases toward resting level during l hour exercise. At the same time, there is net release of glucose (the first 2-3 minutes), indicating rapid glyconeogenesis. Soon, however, net uptake of glucose from the working muscle is observed.

As the work intensity increases, so does the lactate production. However, when a given work intensity is maintained for a sufficiently long time, the lactate concentration is generally considered to remain fairly constant once equilibrium has been reached (Hermansen, 1971). Freund and Gendry (1978) have fitted mathematic functions to the lactate response during recovery at rest. The blood lactate during recovery continues to increase during the first 2-6 minutes (depending on work intensity) of recovery, while there is a rapid decrease in the intramuscular lactate. After 10 minutes, there is very little difference between muscle and blood lactate (Freund and Gendry, 1978).

Skinner & McLellan (1980) have divided exercise into three phases (1-III). During phase I, the RQ is between 0.7 and 0.8 and little or no lactate is formed during this low intensity steady state exercise. Between 40 and 60% of maximal oxygen consumption, phase II is reached and blood lactate level is about doubled. When the work intensity is increased further (65-90% maximal oxygen consumption), the blood lactate will rise above 4 mM, and then begin to increase rapidly as the subject approaches his maximal oxygen consumption. Above a subject's maximal oxygen consumption, blood lactate continues to increase with time and no steady state is reached. (Therefore, RQ is no longer a valid measurement of the substrate utilization.) The sharp rise in blood lactate with the increase in work intensity after a blood level of about 4 mM is reached (MacDougall, 1978; Green, et al, 1979), corresponds to the anaerobic threshold.

The early research by Hill, et al (1924) concluded that lactate was produced when there was an insufficient oxygen supply. It is well known

that training decreases lactate production during exercise at the same absolute work load (Hermansen, 1971; Johnson, et al, 1969). The argument is used that since the total oxygen consumption is not increased following training or breathing a high oxygen gas (60-100% oxygen) (Holloszy, 1976; Welch, et al, 1977; Skinner & McLellan, 1980; Graham, 1978), hypoxia cannot be the cause of lactate production. However, this is probably incorrect, since under conditions where lactate is produced, it is reconverted to glucose in the liver or oxidized in other muscle fiber that is not hypoxic and therefore probably uses the oxygen saved during anaerobic glycolysis (for example, to reconvert lactate to pyruvate). Bylund-Fellenius et al. (1981) recently presented strong evidence that the oxygen partial pressure in the exercising muscle indeed determines the lactate production.

It has been shown that the muscle respiratory capacity is of primary importance in determining the work rate at which blood lactate accumulation begins (Ivy, et al, 1980), and it is well known that type I fibers have higher respiratory capacity and capillary density than type II B fibers (Essen, 1978). It is well known that type I fibers are preferentially recruited at low work intensity, and both the recruitment pattern, respiratory capacity, and fiber composition may be affected by training (Saltin, et al, 1977; Baldwin and Winder, 1977; Booth, 1977). The mode of exercise (intermittent vs continuous) also affects the lactate production (Essen, 1978). Intermittent exercise of twice the exercise intensity produced the same blood lactate level as continuous exercise. This difference is best explained by a greater oxygen availability during intermittent exercise due to reloading of the muscle

myoglobin stores and to the decreased utilization of muscle glycogen during the intermittent exercise. Furthermore, it is well demonstrated that during low intensity exercise, lactate is a substrate for the exercising muscle (McGrail, et al, 1978; Stamford, et al, 1981; Poortmans, et al, 1978).

Effect of Diet

It has been shown that following a meal containing carbohydrates, blood glucose and lactate becomes elevated, while FFA are depressed (Owen, et al, 1980; Capro, et al, 1981). On this basis, one would expect an interaction between carbohydrate intake and exercise following a carbohydrate meal. However, it has also been shown that the prime precursor of lactate is muscle glycogen (Wahren, et al, 1971); furthermore, that the muscle glycogen concentration affects the lactate production (Jacobs, 1981). Lactate production was significantly reduced when glycogen levels fell below about 40 mmol glycosyl units/kg. When considering dietary effects upon lactate production, it is therefore important to also consider the dietary effect upon the glycogen stores.

During low intensity work after depletion of muscle glycogen stores, a high carbohydrate diet increased blood lactate compared to a low carbohydrate diet, both during exercise (50% maximal oxygen consumption) and recovery. As expected at these workloads, the lactate production is very low (about 1 mM), and it may very well be a dietary effect unrelated to exercise.

In high intensity work (105% maximal oxygen consumption) following glycogen depletion and either a carbohydrate-free or carbohydrate-rich diet, Maughan and Poole (1981) found that carbohydrate loading resulted

in highly significant increased blood lactate following exercise compared to the carbohydrate-free regimen. Bonen, et al (1981) exercised their subjects at 80% of maximal oxygen consumption following glycogen depletion and either during control (no glucose) or 1.5 g/kg glucose before (15 minutes) or during (3-5 minutes) exercise. They found that preexercise glucose gave the highest blood lactate during exercise and recovery, while the lowest values were observed during the control treatment. Apparently, the effect of diet when muscle glycogen is unaffected has not been studied.

CHAPTER 2

THE VARIABILITY OF THE HEART RATE-OXYGEN CONSUMPTION RELATIONSHIP WITH TIME AND COMPARISON OF INDIVIDUAL REGRESSION LINES VS A COMMON REGRESSION LINE FOR ALL SUBJECTS

.

.

.

Synopsis

The variation with time of the heart rate oxygen consumption relationship (HROXCR) was studied in ten subjects with 10 trials in each subject and 40-minute recovery. A significant inhomogeneity was found in all but one subject. The significance was not decreased by using corrected heart rate (corrected HR = Actual HR + 60 - Resting HR). The slope differed little in subjects with similar maximal oxygen consumption, although significant differences existed. The y-intercepts were also significantly different, and the significance did not disappear when using corrected heart rate, unless only subjects with similar maximal oxygen consumptions were considered.

It is concluded that it is best to use individual regression lines, but because of the variability within subjects, it should be based on more than one trial. Even the relatively small variations in the oxygen consumption from day to day is positively correlated with the HR.

Introduction

The heart rate (HR) is often used as a predictor of the oxygen consumption (OXC) and/or degree of physical activity (Andrews, 1971; Payne et al, 1971; Bradfield, 1971; Bradfield et al, 1971; Astrand, 1971; Dauncy & James, 1979). One reason for this is the good linear correlation between HR and OXC in any given experiment (Londeree & Ames, 1976). This implies that the HR is a major determinant of the oxygen supply. However, besides the HR, the stroke volume (SV) and the arterial-venous oxygen difference (AVD) are other factors used to determine oxygen supply, and all three are known to vary with the intensity of exercise (Astrand et al, 1964; Damato et al, 1966; Williams et al, 1962).

The relationship between the HR and the OXC (HROXCR) has in fact, been shown to depend upon physical training (Saltin, et al, 1968; Pannier, et al, 1980), age (Sheffield et al, 1978; Montoye, et al, 1968; Dill & Consolazio, 1962), body position (Andrews, 1971; Vokzc, et al, 1975; Sato & Tanaka, 1973), and environmental temperature (Dill & Consolazio, 1962; Rowel et al, 1966; Taylor et al, 1963; Myhre et al, 1979). While sex, age and training are easily controlled, the effects of body position and environmental temperature cannot always be easily controlled. Since the effects of temperature and position on the HROXCR in general are not very large, they are probably safely ignored for many applications (Andrews, 1971).

It has not been determined if the HROXCR remains constant for a reasonable time period, although this assumption forms the basis for the use of HR to predict the OXC. Furthermore, it is controversial whether a common regression equation can be used for all subjects, or groups of subjects of similar age, sex, and physical condition (Andrews, 1971), or an individual equation should be used for each subject (Bradfield et al., 1970).

The purpose of this study was to determine if the linear relationship between the HR and OXC is constant with time when no training effect is introduced. Andrews (1971) has suggested that the use of incremental HR will remove any significant differences in the regression equation between subjects, and this was also tested in this study. Furthermore, this study addressed the question of whether individual or common regression lines best predict OXC. Durnin (1978) has argued that since most activity occurs at low level, the normal variability could easily obscure true differences. It is therefore necessary to test whether the variability in OXC at resting level of activity is correlated with the HR.

Methods

Subjects

Ten male students, 20-30 years of age, who were moderately fit and accustomed to exercise on a treadmill participated in this study. The subjects did not participate in regular physical training or athletic competition. None of the subjects were obese, that is, they were within ± 10% of desirable weight (Metropolitan Life Insurance Company, 1959), or received any medication during, and at least a month prior to, the trials. Anthropometric data and maximal oxygen consumption for the subjects are given in Table 1. The subjects gave informed consent, and the protocol used was approved by the University Committee on Research Involving Human Subjects (UCRIHS) at Michigan State University.

Measurement of Oxygen Consumption (OXC) and Heart Rate (HR)

The OXC was determined through a modified Douglas method (Consolazio et al, 1963). The expired air was collected through a low resistance (Daniels) valve and collected in light-weight (neoprene) bags. The composition of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers. The air volume was determined by metering through (using constant flow) a Singer dry gas meter. The HR was determined from a 3-lead electrocardiogram, and recorded continuously on a Sargent

Age, height, weight, and VO _{2 max} for the ten subjects. The slopes \overline{B}_1 and y-intercepts	subject is given.
e slopes	in each
ts. The	trials
i subject	all ten
e ten	uo p
for th	es base
VO2 max	variance
weight, and	rresponding
Age, height,	3n and the co
Table 1. 1	

Subject Nr.	-	2	æ	4	ъ	9	7	8	6	10	Mean ³
Age (years)	20	26	25	23	30	30	29	20	29	26	26.8
Height (cm)	198	168	156	183	187	194	192	165	167	180	179
Weight (kg) ¹	84.9	80.8	70.8	93.3	82.0	90.0	100.1	63.2	57.2	78.0	80.0
Max V ₀₂ ² (ml/kg/min)	50.1	49.0	45.7	47.0	51.8	52.1	53.3	54.6	66.0	53.9	52.4
B ₁ a	.344	.352	.345	.344	.326	.364	.359	.375	484	.368	.366
Var _l b	.0017	.0004	.0008	.0005	.0003	.0008	.0013	6000.	.0014	.0004	.0017
<u>В</u> о с	-19.5	-15.8	-16.1	-13.8	-11.5	-16.6	-8.6	-21.5	-27.6	-17.4	-16.8
Var ₀ d	21.8	2.8	13.9	5.1	2.3	10.2	2.3	11.4	18.8	7.4	25.2
Bo cor ^e	-17.1	-17.1	-16.9	-17.1	-16.7	-18.8	-18.8	-17.5	-27.8	-18.2	-18.6
Var ₀ d	7.4	2.4	3.0	1.4	2.3	2.8	5.4	2.4	5.6	1.4	9.9

¹Mean of ten determinations, one prior to each exercise test.

²Mean of two determinations (in week one and ten respectively).

 $^3\mathrm{The}$ means of B_1 and B_0 are obtained by calculating the regression equation based on all trials in all subjects, and the corresponding variances are the between subjects variances.

 $^{d}\overline{B}_{l}$ is the mean slope of the regression equation for the ten trials in each subject.

^bVarן is the variance for B_l.

 $^{c}f B_0$ is the mean y-intercept of the regression equation for the ten trials in each subject.

^dVar₀ is the variance for \overline{B}_0 .

 $^{e}\bar{B}_{0}$ Cor is the y-intercept after correction for the resting heart rate.

recorder. The mean HR for the time corresponding to each bag of expired air was determined. In an attempt to reduce error due to variation in resting HR, a corrected HR was calculated by adding or subtracting the difference between 60 and the resting HR from each HR measurement. 60 being the mean HR is arbitrarily chosen as standard.

Test Protocol

The ten subjects completed an incremental exercise test once a week for ten consecutive weeks. Each subject was assigned a specific day and time, on which he would arrive in the laboratory at least 5 hours postabsorptive (usually after an overnight fast). If a subject could not meet his appointment for a particular week, he would be rescheduled for another day the same week. On the test day, the subjects were instructed not to consume coffee or tea and not to engage in heavy exercise.

The imcremental exercise test was performed on a treadmill. The steps consisted of resting and the following speeds (S-2 to S-7): 4.8, 6.8, 8.7, 9.7, 12.5, and 14.3 (miles/hour). At rest (S = 0) the subjects were sitting relaxed in an armchair on the treadmill, but HR and expired air was collected as during exercise. During rest, three 5-minute bags were collected; for S-2 and S-3, three 2-minute bags; and for the remaining speeds (S-4 to S-7), three 1-minute bags were collected. Not all subjects completed all the steps, but the trial was terminated when the subject approached his maximal oxygen consumption as determined in two separate tests.

Measurements of Resting HR and OXC

Five different subjects (mean age \pm SD = 26.6 years \pm 1.7, mean weight \pm SD = 78.2 kg \pm 11.3 and mean height \pm SD = 182 cm \pm 6.1) were used to determine OXC and HR once an hour for 9 hours. The subjects were seated in an armchair while 3 consecutive 5 minute bags of expired air were collected and the mean HR corresponding to each bag determined from the HR-recordings. The correlation coefficient between HR and OXC was calculated for a 9 hour period in each subject.

The resting values for HR and OXC for 9 of the 10 subjects used for the determinations of the regression equations as described above, were used for calculation of the correlation coefficients. These correlation coefficients are therefore based on simultaneous measurements of OXC and HR on ten different days one week apart.

Treatment of Data and Statistical Procedures

A computer program was used to calculate OXC from the composition of the expired air on a standard temperature, pressure, dry basis. The OXC for each collection period and the corresponding mean HR were used to calculate a regression equation for each trial in each subject. A regression equation for each subject, based on all ten trials, was calculated, as was a common regression equation based on all 100 trials in the ten subjects. The slopes B_1 and the y-intercepts (B_0) for the individual trials were used as independent variables in a one-way analysis of variance (random model). The repeatability

R =
$$\frac{\hat{\sigma}_{R}^{2}}{\hat{\sigma}_{B}^{2} + \hat{\sigma}_{\omega}^{2}}$$
 is calculated to evaluate the relative contributions of

within and between subjects variance: $\hat{\sigma}_{\omega}^2 = mS_E$ and $\hat{\sigma}_{\beta}^2 = \frac{mS_S - mS_E}{10}$ where mS_E is the mean square error term, and mS_S is the mean square treatment term from the analysis of variance (Gill, 1978). The sum of squares (SS_{Ei}) for the individual regression equation and the total sum of squares for the summation regression equation in each subject (SS_{ET}) are used to test for homogeneity of regression within a subject and between subjects (Gill, 1978). The critical value is t_{α} , 2(t-1) (r-2), where t= trials and r=number of observations in a trial. Correlation coefficients were determined in the usual manner and mean and confidence intervals are calculated by transformation to z=0.5 ln $\frac{1+r}{1-4}$ (Gill, 1978).

<u>Results</u>

The regression line for a single incremental exercise test is shown in Figure 1 for a representative subject. The correlation coefficient between HR and OXC is very high (>0.99), which is partly due to the use of mean (integrated) HR. The regression line based on all ten incremental exercise tests in a typical subject is plotted in Figure 2.

The calculated F-statistics for test of homogeneity of regression between independent samples are given in Table 2. The critical F value is 1.94 (α =0.05) and in all but one subject, there is significant inhomogeneity. Also shown are the calculated F-statistics after correcting for resting HR by adding 60-resting HR to all HR measurements. The critical F-value is again 1.94 and it is seen that rather Figure 1. Regression line between the OXC and the HR for a typical subject (#8) based on a single incremental exercise test. The slope $B_1 - 0.377$, the y-intercept $B_0 = 22.5$, and the standard error of estimate SE = 0.86 ml $0_2/min/kg$. S2-7 is the different speeds in the incremental exercise tests.

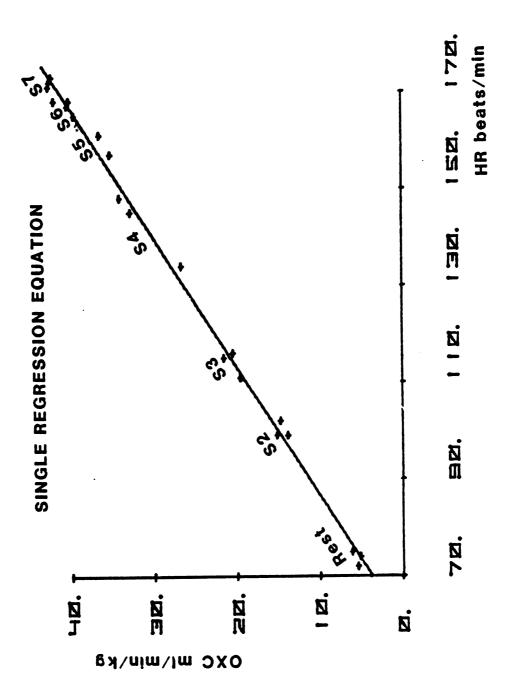


Figure 2. Regression line between OXC and HR in a typical subject (#8) based on ten incremental exercist tests. The slope $B_1 = 0.372$, the y-intercept $B_0 = 21.1$, and SE = 2.03 ml $0_2/min/kg$.

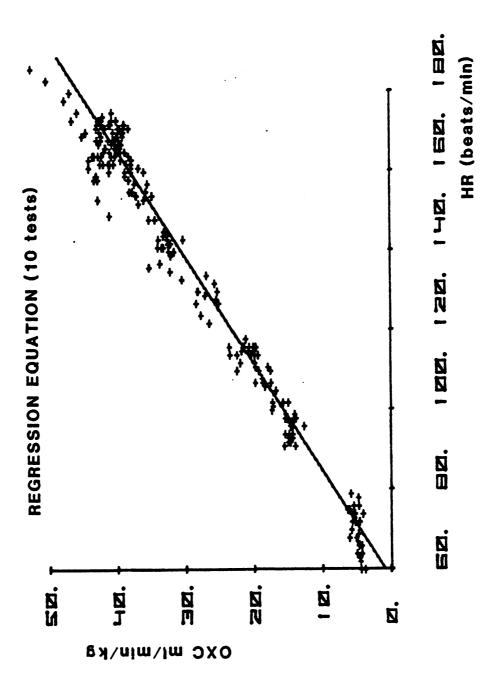


FIGURE 2

Table 2.	Calculated F-ratio for
	test of homogenity with-
in the ter	n trials in each subject
with obser	rved HR (F-1) and with
corrected	HR (F-2). ^a

$\begin{array}{c ccccccccccccccccccccccccccccccccccc$				
2 12.50 6.36 3 9.95 22.30 4 2.49 14.60 5 1.58 3.51 6 13.70 12.74 7 11.65 13.04 8 11.16 27.49 9 3.17 9.55 10 24.78 25.71	Subject No.	F-1	F-2	
3 9.95 22.30 4 2.49 14.60 5 1.58 3.51 6 13.70 12.74 7 11.65 13.04 8 11.16 27.49 9 3.17 9.55 10 24.78 25.71	1	20.01	28.37	
4 2.49 14.60 5 1.58 3.51 6 13.70 12.74 7 11.65 13.04 8 11.16 27.49 9 3.17 9.55 10 24.78 25.71	2	12.50	6.36	
5 1.58 3.51 6 13.70 12.74 7 11.65 13.04 8 11.16 27.49 9 3.17 9.55 10 24.78 25.71	3	9.95	22.30	
6 13.70 12.74 7 11.65 13.04 8 11.16 27.49 9 3.17 9.55 10 24.78 25.71	4	2.49	14.60	
7 11.65 13.04 8 11.16 27.49 9 3.17 9.55 10 24.78 25.71	5	1.58	3.51	
8 11.16 27.49 9 3.17 9.55 10 24.78 25.71	6	13.70	12.74	
9 3.17 9.55 10 24.78 25.71	7	11.65	13.04	
10 24.78 25.71	8	11.16	27.49	
	9	3.17	9.55	
h	10	24.78	25.71	
otal ^b 245.53 107.00	Total ^b	245.53	107.00	

^aThe critical F-ratio is 1.96 (α =0.05).

^bTotal is the F-ratio for inhomogenity between subjects.

Repeatability R, calculated and critical F-ratio (α =0.05) for the slope B₁, the y-intercept B₀, the y-intercept corrected for resting heart rate B₀ Cor, and B₁-1 and B₀⁻¹Cor when the subject with a large deviation in the max V₀₂ is disregarded.^a Table 3.

	BJ	B1-1	BO	80-1	B _O Cor	B0 ⁻¹ Cor
Repeatibility R	0.696	0.158	0.741	0.632	0.755	0.092
Calculated F	22.66	2.91	25.5	18.21	31.85	2.02
Criticial F	1.99	2.06	1.99	2.06	1.99	2.06

 $^{\rm a}_{\rm \ HR}$ is corrected by adding 60-resting HR to each observed HR.

÷

than decreasing the inhomogeneity, it generally increases, resulting in significant inhomogeneity in all subjects. The critical F-value for test of inhomogeneity between subjects 1.88 ($\alpha = 0.05$) and the calculated F is highly significant; but in this case, the inhomogeneity is drastically reduced by correcting for resting HR, although it is still highly significant (the F-ratio decrease from 246 to 107).

From Table 1 it is seen that one subject (#9) has significantly higher maximal oxygen consumption than the other subjects (66 vs a mean of 52 ± 2.8). Since the regression equation can be expected to vary with maximal oxygen consumption, the analysis of variance is done with and without this subject. Using corrected HR does not affect the slope (B_1) , but the y-intercept (B_0) . The repeatability, calculated F, and critical F are given in Table 3. R can vary between 0 and 1: R = 0 means that all the variability lies within the subjects, and R = 1 means that all the variability lies between the subjects. From Table 3, it is seen that correction for differences in resting HR has no effect on R for the y-intercept (0.741 vs 0.755), and removing the inhomogenous subject (#9) has no effect in itself (0.741 vs 0.632); but using corrected HR and removing subject #9 causes practically all the variation to be within subjects (0.755 vs 0.092). As seen from Table 2, using corrected HR drastically reduces the variation within subjects (from a mean of 25.2 to 9.9); but since it has no effect on R and B_{Ω} Corr is highly significant (F = 31.9) as seen in Table 3, it means that it reduces the variability equally within and among the subjects. When removing subject #9 from the analysis and

using corrected HR, there is no longer evidence of inhomogeneity (F = 2.02). R for the slopes is likewise reduced by removing subject #9 (0.696 vs 0.158); but in this case, the slopes are still significantly different between subjects (F = 2.91).

In conclusion, it can be said that each subject has a characteristic B_1 and B_0 around which the individual incremental exercise test values vary. When subjects are homogenous with respect to maximal oxygen consumption, the individual variation is far more important than the variation among subjects for the slope, and also for B_0 if corrected HR is used.

Correlation Between HR and OXC at Constant Workload

The resting HR within a subject generally varies by ± 10 beats/ minute from minute to minute. But, during exercise, the short-term variation is decreased to ± 2 to 3 beats/minute at high workloads. The mean HR corresponding to each bag of expired air has a short-term (15 min) variation of ± 1 beat/minute or less; however, over extended test periods of up to 10 hours, there is variation in the mean HR of ± 5 beats/minute. There is also a variation in the resting HR from day `to day of about 15-20 beats/minute.

The mean correlation between HR and OXC taken at rest at 9 different times throughout a day for the 5 subjects was -0.032 and the confidence interval -0.199 to 0.135. The mean standard deviations for HR and OXC were 2.91 and 0.19 respectively. This means at a given workload (rest), HR and OXC vary independently of each other in the postabsorptive state. In 9 different subjects the correlation between

49

HR and OXC was determined from measurements taken in the resting state on ten different days. In this case the mean SD for HR and OXC were 4.55 and 0.47 respectively, that is approximately doubled due to day-to-day variation. The mean correlation coefficient \bar{r} was 0.478 with the confidence interval from 0.375 to 0.570. This means that the relatively small variation in OXC observed from day to day in the resting state is significantly correlated with the HR.

Discussion

The HR is frequently used as a predictor of OXC (Bradfield, 1971). The excellent regression between HR and OXC found in this study (r>0.99) when several different workloads are considered, gives support for the use of HR as a predictor of OXC. The mean standard error of estimate (SE) for the 100 regression equations calculated in this study was 1.40 ml oxygen/min/kg which corresponds to 0.49 kcal/ min. This compares favorably with the value of 0.37 reported by Andrews (1971), since we generally used a wider range of workloads; and the SE tends to increase with increasing workload.

As discussed by Dauncy and James (1979), in most applications the HR varies within a relative narrow range (60-100 beat/min or less). It is therefore important to see how well HR and OXC is correlated within a narrow workload. The correlation (r) was calculated between HR and OXC in five subjects from periodic measurements throughout a day with no physical activity. Under these conditions there was no correlation between HR and OXC; and the standard deviations for HR and OXC were 2.9 and 0.19 respectively. When the correlation was based on measurements of HR and OXC on 10 different days (subjects again in the resting state) the correlation (r=0.478) was statistically different from 0 (α =0.05) and could explain about 25% of the variation, the remaining variation being due to random variation in HR and OXC. In the measurements from day to day, the SD for HR and OXC were 4.6 and 0.47 respectively. Thus even relatively small changes in

HR are positively correlated with the OXC.

Andrews (1971) reported that substituting HR for incremental HR (IHR) (actual HR - resting HR) removes the differences in y-intercept for the regression lines between HR and OXC. Using IHR is equivalent to having all the individual regression lines going through a common point (IHR = 0 and OXC = Resting OXC), since the resting OXC generally varies very little compared to the SE of the regression equation (SE ~ 0.3 and 1.4 respectively). When the IHR is used the y-intercept corresponds to the resting OXC; since this, in our experience, invariably lies on the regression line; and an analysis of variance of the y-intercept therefore becomes meaningless. The corrected HR (CHR=actual HR + 60 -Resting HR) was therefore used in the present study. The effects of using corrected and incremental HR is illustrated in Figure 3 and Figure 4 for a hypothetical case. Like the IHR, the CHR adjust all the regression lines to a common resting HR; but instead of this being 0 it is 60, and the variability of the y-intercept due to variation in slope is thus retained. Using CHR has no effect upon the slope; and only where very homogenous subjects (with respect to maximal OXC) were considered did it decrease the variability in the y-intercept between subjects. However, as seen from Table 2, using CHR does not decrease the variability in the y-intercept within a subject. It is therefore concluded that there is no advantage in using CHR (or IHR).

Whether to use a common regression equation between HR and OXC for all the subjects as recommended by Andrews (1971) or an individual determined regression equation as recommended by Bradfield <u>et al</u>. (1970) and Astrand and Rodahl (1977) is still controversial. Since

Figure 3. Regression lines between HR and OXC for a hypothetical case: Training decrease HR and increase slope. Line a and b illustrate variation in the HROXCR such as caused by exercise.

•

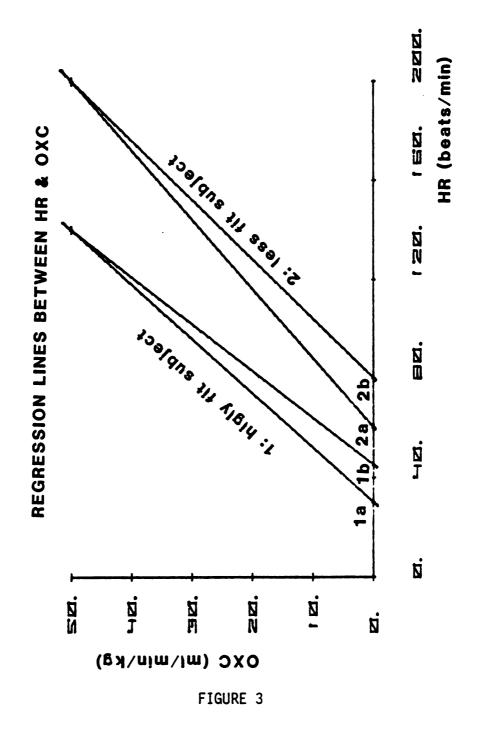
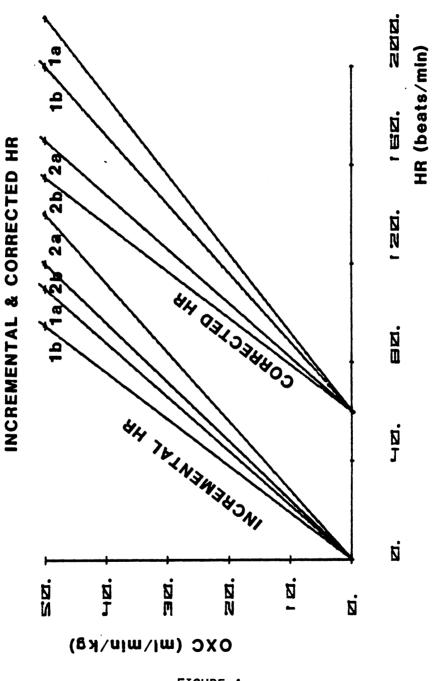


Figure 4. Incremental and Corrected HR. IHR = Actual HR - Resting HR CHR = Actual HR + 60 - Resting HR. Both adjustments reduce the variability of the y-intercept; but not necessarily the difference between the regression equations.





both the slope and the y-intercept are significantly different even within homogenous subjects, it is always better to use individually determined regression lines. This can also be seen from the repeatability R (R=0.70 for the slope and 0.74 for the y-intercept), which means that most of the variability is between subjects even when only homogenous subjects are considered (R=0.63). The finding of significant inhomogeneity among the regression lines makes it necessary to determine the regression based on data from at least 3 exercise test spaced at least 2 days apart, in order to ascertain that the regression line is typical for the subject.

Payne <u>et al</u>. (1971) reported that HR and OXC obtained during sitting and standing rest did not fall on the regression equation obtained during exercise. They did find, however, that HR and OXC obtained during supine rest fell on the regression line. In the present study 15 minutes of sitting rest was used as the first step in the incremental exercise test, and the HR and OXC obtained during rest almost invariably lay on the regression line. The reason for this discrepancy may lie in how the resting measures are conducted. As described by Dauncey and James (1979) it is important that the subjects moves the legs periodically in order to avoid venous pooling.

As reported by Hirsch and Bishop (1981) the breathing pattern affects the HR and causes a cyclic variation. In the present study this effect was clearly seen at rest (±10 beats/min), but it disappeared at higher workloads. Since the HR was recorded continuously, and the mean HR corresponding to each bag of expired air was calculated, this variation had no effects on the results.

CHAPTER 3

.

EFFECT OF DIET, PREVIOUS EXERCISE, AND RECOVERY UPON THE LINEAR REGRESSION BETWEEN HEART RATE AND OXYGEN CONSUMPTION

Synopsis

We have studied the effect of diet composition upon the regression between heart rate (HR) and oxygen consumption (OXC) during an incremental exercise test. Our results do not give any evidence for a dietary effect. Repeated exercise one hour apart did, however, have a significant effect upon both the slope and y-intercept of the regression equation between HR and OXC. During recovery, the HR did not predict the OXC based on the regression equation obtained during exercise. Both the exercise intensity (S) and duration (D) had a significant effect upon the recovery HR (RHR), whereas only S had a significant effect upon recovery OXC (ROXC). Consequently, RHR lagged behind the ROXC the first three minutes of recovery, but then returned toward resting values at the same rate as the OXC. The difference between ROXC and predicted OXC (POXC) is primarily determined by D.

Introduction

The heart rate (HR) is often used as a predictor of the oxygen consumption (OXC) and/or degree of physical activity (Andrews, 1971; Payne, et al, 1971; Bradfield, 1971; Bradfield, 1971; Astrand, 1971; Dauncy & James, 1979). One reason for this is the high linear correlation between HR and OXC in any given experiment (Londeree and Ames, 1976). This implies that the HR is a major determinant of the oxygen supply. However, besides the HR, the stroke volume (SV) and the arterial-venous oxygen difference are other factors that determine the oxygen supply; and all three are known to vary with the intensity of exercise (Astrand et al, 1964; Damato et al, 1966; Williams et al, 1962).

The relationship between the HR and OXC (HROXCR) has been shown to vary with physical training (Saltin et al, 1968; Pannier et al, 1980), age (Sheffield, et al, 1978; Montoye et al, 1968; Dill & Consolzio, 1962), body position (Andrews, 1971; Vokzc et al, 1975; Sato & Tanaka, 1973), and environmental temperature (Dill & Consolazio, 1962; Rowell et al, 1966; Taylor et al, 1963; Myhre et al, 1979). While it is relatively easy to control for sex, age, and physical training, in most applications, it is difficult to control for body position and environmental temperature. However, in most experiments, it may be safe to assume that body position and environmental temperature have an equal effect upon control and experimental groups.

The effects of diet, previous exercise or stress on HROXCR are not well studied (21). However, stress has been shown to increase the HR at constant OXC (Taylor et al, 1963; Sonne & Galbo, 1980) and previous physical activity does affect the regression equation (Lundgren, 1946), whereas conflicting results are reported for dietary effects (Lundgren, 1946; Schutz et al, 1981). Since the average subject will probably spend more time recovering from physical activity than he/she will spend exercising, it is also important to know if the HROXCR obtained during exercise is followed during recovery.

The objectives of the present study were (1) to determine the effects of dietary composition upon the regression equation between HR and OXC, (2) to determine if the HROXCR obtained during an incremental exercise test also was valid during recovery, and (3) to determine what effect previous exercise might have upon the HROXCR.

Methods

The study was divided into two parts. The first part examined the relationship between HR and OXC during recovery following 20 min of exercise at 80% of maximal OXC. In the second part the effects of duration(D) and intensity (S) of the exercise period upon the relationship between the HR and OXC during recovery was studied.

Subjects

Two groups of five healthy male college students volunteered for the study. The physical characteristics of the subjects are given in Table 1. None of the subjects took any medication; and on the day of the experiment, they were requested not to drink any caffeine-containing beverages. They also did not participate in any strenuous physical activity on the day before the experiments were conducted.

Informed Consent

Each subject signed an informed consent form after the details of the experiments were described to him. The form stated the experimental procedures, identified possible risks, and noted that a subject could terminate his participation at any time.

Diets

The composition of the diets and meals is given in Table 3. Each meal contained 700 kcal. The fat meal consisted of 288 ml whipping cream, which the subjects drank in less than 10 minutes. The protein

Physical	<u>Group</u>	<u>I (n=5)</u>	<u>Group II (n=4)</u>
<u>Characteristics</u>	Mean		<u>Mean</u> <u>SD</u>
Ages (years) Body Weight (kg) Height (cm) VO ₂ -max (ml/min/kg) ^a Mean running speed ^D %VO ₂ -max (mean) ^b	24.8 74.5 173 54.1 10.9 78	3.1 11.9 7.4 6.6 0.8 0.6	28.8 2.2 81.9 7.0 184 16.5 49.9 2.6

Table 1. Physical characteristics of the two groups of five adult male subjects. (SD = Standard Deviation.)

^a Maximal	010	b _{final}	treadmill	settina
max IIIIa I		1 1 1 1 1 1 1 1	creaumini	Security

Table 2.	Relative intensity of exercise for various treadmill settings
	used in incremental exercise tests.

Setting	Speed (km/hr)	% VO ₂ -max ^a	Duration (D) of Exercise (min)
0	0 (rest) 4.8	10	15
2	4.8	38	6
3	6.8	52	6
4	8.8	64	3
5	10.5	74	3
6	12.5	84	3
7	14.3	93	3

^aMaximal OXC: mean of four subjects.

^bThe time given is for an intermediate step. When 4-7 was the final speed D was either 10 or 30 minutes.

Table 3.	Compositio kcal for t	Composition of meals and diets. kcal for the different subjects.	diets. The energy content of the diets varied between 2,100 and 2,400 bjects.	t of the	diets vari	ed betweer	, 2,100 and	2,400
			Total	8	% of total energy	ergy	% by weight	ight
Diets or Meals	Meals	Method	Energy (Kcal)	Fat	Protein	Carbo- hydrate	Water	Ash
Fat Meal		Food Tables ^a	700	92	ę	5		
Protein Meal	lea l	Food Tables	700	22	76	2		
Carbohydrate Mea	ate Meal	Food Tables	700	2	5	93		
Fat Diet		Food Tables	2,150	65	15	20		
Carbohydrate Diet	ate Diet	Food Tables	2,150	20	15	65		
Fat Diet		Chemical Analysis	2,150	66	16	19	68.0	4.0
Carbohydrate Diet	ate Diet	Chemical Analysis	2,150	22	15	63	72.8	3.4
^a Watt and	i Merrill (1	^a Watt and Merrill (1963); Church & Church	Church (1975).					

meal consisted of 94 g creamed cottage cheese and 545 g (raw weight) skinned chicken breast, with all visible fat removed. The subjects ate the protein meal in 30 minutes. The carbohydrate meal consisted of 824 g ripe bananas (peeled), which were eaten in less than 10 minutes. The subjects drank 340 cc of ginger ale (1 kcal) with the meals (Vernors, Detroit, MI), and were allowed salt, pepper, and water ad lib.

During the experimental periods the subjects ate 2,100 to 2,400 kcal per day either as a high fat or as a high carbohydrate diet. The composition is given in Table 3. The subjects were fed one of the two experimental diets in random order for three days prior to the exercise test, which was performed in the postabsorptive state the fourth day. The diet and test meals were prepared and eaten in a kitchen adjacent to the exercise laboratory. Two menus were used for each diet: one for breakfast and one for dinner and supper. The menus were repeated for all three days. The subjects were supervised during the meals and ate all the allotted food. Duplicate portions were used for chemical analysis. Fat was analyzed by ether extraction, protein by micro-kjeldahl, minerals as ash $(600^{\circ}C)$ and carbohydrate by difference.

For both diets regular food items were used. Sources of fat were: margarine, mayonnaise, cheese, eggs, bacon and olives; while sources of carbohydrate were: bananas, bread, potatoes, sweet potatoes, beans, carrots and peas. These foods contain some protein. Additional Protein came from soy protein. For the fat diet imitation chicken, and for the carbohydrate diet imitation beef (Worthington Foods, Worthington, OH) were used.

Measurements

Heart rate (HR) was obtained from an electrocardiogram (lead 2 with the positive lead in V5 position). The EKG signal was converted to heart rate through a cardiotachometer (built in our lab) and continuously recorded by a calibrated Sargent Recorder, Model DTM-115-4 (Sargent and Company, Chicago, IL). The mean HR in beats/minute corresponding to each collection of expired air was calculated from the recording.

The oxygen consumption (OXC) was determined by a modified Douglas method (Consolazio, et al, 1963). The expired air was collected through a low resistance valve (Otis-McKerrow, Warren Collins, Inc., Braintree, MA) in light-weight neoprene bags. The composition (O_2 and CO_2 contents) of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers, respectively (Beckman Instruments, Schiller Park, IL). The air volume was determined by metering through (using a constant flow of 50 1/min) a Singer dry gas meter (American Meter Company, Philadelphia, PA).

The mean OXC for the control experiment was compared to the baseline (mean resting OXC prior to the ingestion of the meal). To correct for the day to day variation in the resting OXC in each subject the difference between the control and baseline values were added or subtracted from the OXC for that day's treatment.

Exercise Tests

The maximal OXC was determined in a continuous incremental test with the subjects exercising on the treadmill until exhaustion. The speed was increased stewpise at 8.8, 10.5, 12.5 and 14.3 km/hour. When

the subjects reached the highest speed they could comfortably run (12.5 or 14.3 km/hr) further increases in the workload were accomplished by increasing the treadmill inclination in steps of 2%. The subjects ran 3 minutes at each step, the HR was recorded continuously, three one-minute bags of expired air were collected at each step, and the oxygen consumption calculated for each minute.

The OXC following the different treatments was determined through an incremental exercise test with the final workload corresponding to 80% of the subjects' maximal OXC. Three collections of expired air were made at each step and the mean HR corresponding to each air collection was recorded. The test started at rest with the subject seated in an armchair, while 3 consecutive 5-minute collections of expired air were made. During the following steps the subjects would walk or run on the treadmill. At the 4.8 km/hr and 6.8 km/hr levels the subjects exercised for 6 minutes each, and at the other levels they exercised for 3 minutes each; except when they reached the final speed (the one corresponding closest to 80% of their maximal OXC), they continued the exercise for 20 minutes. During the final stage, one-minute bags of expired air were collected, every second minute starting with the second minute.

Following the exercise, the HR and OXC were determined during 40 minutes of recovery. Two one-minute bags, one three-minute and one five-minute bag of expired air were collected during the first 10 minutes of recovery. During the next 30 minutes, 3 five-minute bags of expired air were collected every second 5-minute period. During the alternate 5-minute periods, when no collections were made, the subject

remained seated in the armchair, but was relieved of the face mask with air-collection valve and the noseclip. Arterialized blood samples were collected from one of the fingers at 5, 15, 25 and 40 minutes of recovery for determination of the blood lactate concentration.

In the second group (group II) of subjects the final setting (S) of the treadmill was varied between 4 and 7 in order to investigate the effect of exercise intensity and the duration (D) of each speed was either 10 or 30 min. The various treatment combinations were administered to each subject in random order. The speeds, duration and mean % of maximal OXC are given in Table 2. The collection of expired air during recovery was slightly modified in this group. Instead of collecting 2 one min bags and one 3 min during the first 5 min of recovery, one 1 min bag and 2 two min bags were collected.

Experimental Protocol

In the second part of the experiment four subjects (group II) participated. The final setting of the treadmill was varied between 4 and 7. The corresponding speeds and % maximal OXC (mean) are given in Table 2. The duration at this setting was either 10 or 30 min. From these data the effect of speed and intensity on HR and OXC during recovery was evaluated. Not all subjects completed the higher exercise intensities; but statistical analysis is based only on the settings completed by all subjects.

Exercise

The exercise experiments were carried out over a 10-week period with each test given one week apart. During the first and last tests the maximal OXC were determined. The second test was always a

control (postabsorptive state) prior to which the subjects ate their normal diet. Prior to the third test, the subjects consumed one of the experimental diets for three days (two started with the high fat diet and three with the high carbohydrate diet). Then followed another control test before the opposite experimental diet was introduced, three days prior to the fifth test. Following both dietary treatments, the test was carried out in the postabsorptive state. The three meals and a final control were introduced in random order so that all the subjects would not consume the meals in the same order. The exercise tests began 30 minutes after termination of the carbohydrate meals and 2-3 hours following the fat and protein meals, which we had found to correspond to the maximal thermogenic response.

Statistical Analysis

The incremental recovery HR(IRHR), recovery OXC (ROXC) and the difference between the predicted OXC (POXC) based on the regression equation obtained during the preceding exercise in each subject (DOXC) were analyzed by a split block (block = subjects) repeated measurements design (each subject serves as his own control).

In the first part of the experiment the HROXCR during recovery was compared to the linear regression line during exercise by calculating the 95% confidence limits. In the second part of the experiment the treatment means for ROXC, IRHR and DOXC were compared by designed contrasts (Bonferroni t statistics; Miller, 1966).

The regression lines between HR and OXC during exercise were calculated for the 5 subjects in part I of the study. A common regression line was calculated for the 3 control experiments. Dietary

effects on the HROXCR were evaluated by testing for differences in slopes and Y-intercepts between the regression lines; but because the regression equation can vary with time random significant results can be expected in some cases. Incremental HR as used by Andrews (1971) is the actual HR-resting HR in the individual subjects. By using incremental HR the Y-intercept of the regression line corresponds to the resting OXC; and it therefore does not have much meaning to test for differences in the Y-intercept. A "corrected" HR is therefore used in this study instead. The corrected HR is obtained by adding 60-resting HR to the measured HR.

Results

Effect of diet on the HROXCR during exercise

There was no evidence that any of the dietary treatments had an effect upon the linear regression between HR and OXC during exercise. The calculated slopes and y intercepts are given in Table 4 for the 5 dietary treatments and control. The control line was calculated from HR and OXC for 3 separate experiments in order to compensate for possible variation in the regression equation with time. All the treatments are compared to the control line for each subject separately. In 5 cases, the regression line for a treatment differed from that of control, but in no case did it occur twice in the same subject. It is therefore not a treatment effect, but a change occurrence due to the variation of the regression line.

Effect of previous exercise on HROXCR

Exercise changes both the slope and y-intercept of the regression equation between HR and OXC. Two of the subjects in part I were exercised as usual at 80% of their maximal OXC, and after one hour rest the same exercise protocol was repeated. One hour after the first exercise test, the OXC had returned to preexercise levels, but the HR was still elevated (20 beats/min). Table 5 gives the values for the slopes (B_1), y-intercepts (B_0) and standard error of estimate (SE) for the two regression lines. The calculated t-statistics for the test of significance is also given. Both the slope and y-intercept are

		- •	Die	ts ^b		Meals ^b	
Subject	Treatment	Control ^a	Fat D	CHO D	Fat M	Pro M	CHO M
1	Slopes Y-Intercept S.E. ^C T _{1d} T ₀	0.372 -20.6 1.59	0.337 -23.4 2.71 0.36 1.41	0.347 -17.2 1.34 2.21* 2.38*	0.385 -23.2 1.25 1.02 1.59	0.372 -22.0 0.84 0.01 0.90	0.371 -21.3 1.49 0.07 0.43
2	Slopes Y-Intercept S.E. T ₁ T ₀	0.364 -17.1 2.03	0.365 -17.4 0.74 0.07 0.21	0.374 -18.7 1.13 0.73 0.92	0.367 -13.8 1.10 0.52 1.98	0.369 -15.6 1.07 0.35 0.86	0.405 -22.9 0.91 2.72* 3.28*
3	Slopes Y-Intercept S.E. T ₁ T ₀	0.474 -26.1 4.03	0.461 -23.5 2.86 0.44 1.37	0.473 -23.5 3.11 0.05 1.32	0.436 -22.6 2.72 1.23 1.83	0.495 -31.6 3.10 0.59 2.34*	0.498 -29.1 2.65 0.68 1.43
4	Slopes Y-Intercept S.E. T ₁ T ₀	0.366 -16.6 1.99	0.331 -12.8 1.30 2.80* 2.53*	0.372 -18.1 1.38 0.42 0.98	0.386 -18.7 1.55 1.49 1.32	0.345 -15.3 1.57 1.67 0.81	0.387 -18.6 1.46 1.64 1.27
5	Slopes Y-Intercept S.E. T ₁ T ₀	0.321 -12.6 1.74	0.307 -12.0 0.95 1.34 0.49	0.305 -12.5 0.96 1.53 0.05	0.330 -14.2 1.44 0.79 1.10	0.290 -11.0 1.13 2.92* 1.20	0.348 -15.7 2.13 1.98 1.90

Test of significant difference between the regression equations between HR and OXC for the five treatments and the regression equation for control. Critical T = 1.99. Table 4.

^an = 54 ^bn = 18 ^cStandard error of means in ml oxygen/kg/min. ^dT₁ and T₀ are the calculated t-values for test of difference between the particular regression line and the control line. *Indicates significance ($\alpha = 0.05$).

Table 5. Test of significance between the regression equations for two subjects exercised twice, one hour apart. Results are included with the regression lines corrected for the resting heart rate (cor).

Subject	Trial	Slope	Y-Intercept	SE ^a	τ ₁ ^b	т _о ь
	1	0.293	-13.4	1.27	A 24	
•	2	0.347	-23.6	1.31	4.34	6.21
1	1 cor .	0.293	-13.4	1.27		0.00
	2 cor	0.348	-18.2	1.31		2.92
2	1	0.359	-16.1	1.39	2 17	4.88
	2	0.404	-25.5	2.14	3.17	4.88
	1 cor	0.358	-18.1	1.39		1 10
	2 cor	0.405	-20.7	2.14		1.19

^aSE = standard error of estimate.

 ${}^{b}T_{1}$ and T_{0} are the calculated t-statistics for slope and Y-Intercept, respectively. The critical t-value for both T_{1} and T_{0} are 2.04 for subject 1 and 2.02 for subject 2. (n = 18 and 22 respectively.) significantly different between the two regression equations in each subject. Using corrected HR decreases of the y-intercepts, but in the one subject the difference is still significant. The regression lines are shown in Figure 1.

Effect of recovery on HROXCR

The linear regression equation between HR and OXC obtained during exercise is not followed during the 40 min of recovery. The ROXC is lower than the lower confidence limit (LCL) during the entire 40 min recovery period; and the predicted OXC (POXC) can be as much as 300% above the recovery OXC (ROXC). These results are based on 8 trials in each of 5 subjects, and are shown in Figure 2 and Table 6. After 40 min of recovery the RHR was still elevated 20 beats/min, and is only decreasing 1 beat/10 minutes. At the end of recovery the OXChas returned to the resting level (Figure 4). Two subjects were followed to complete recovery of the HR, which took at least 3 hours; and appeared to depend on the training of the subject. The RHR is shown in Figure 3 and POXC, LCL and ROXC in Figure 4. Since POXC and LCL are both derived from the RHR and the linear regression equation between HR and OXC obtained during exercise, it is not surprising that RHR, POXC and LCL have a very similar time course, although the values for LCL of course are somewhat lower. As seen from Figure 4 and Table 6, the ROXC falls much faster toward the resting preexercise OXC (the x-axis in Figure 4). The difference between POXC and ROXC (DOXC) quickly reaches a maximum at about 3 min of recovery and then decreases very slowly (Figure 5). Expressed as percentage of POXC, DOXC, however, reaches a maximum of 62% at 3 min, and then remains constant for the

Figure 1. Two regression lines based on exercise trials one hour apart in one subject. The second line begins at a higher HR. Both slopes and y-intercepts are significantly different. The regression lines for the first and second test is marked 1 and 2 respectively.

•

r

.

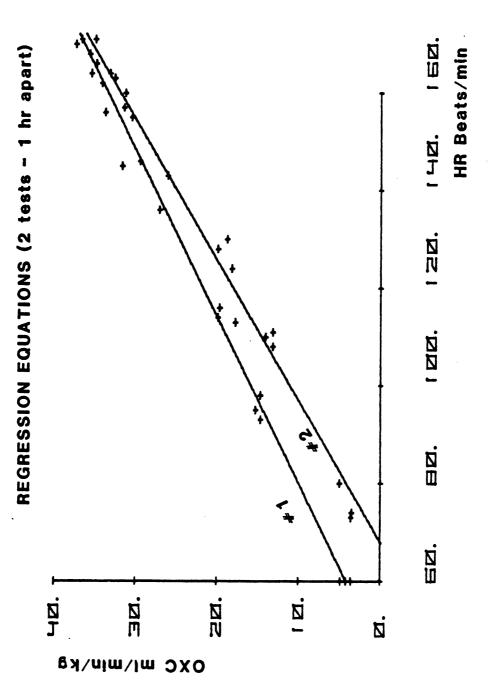
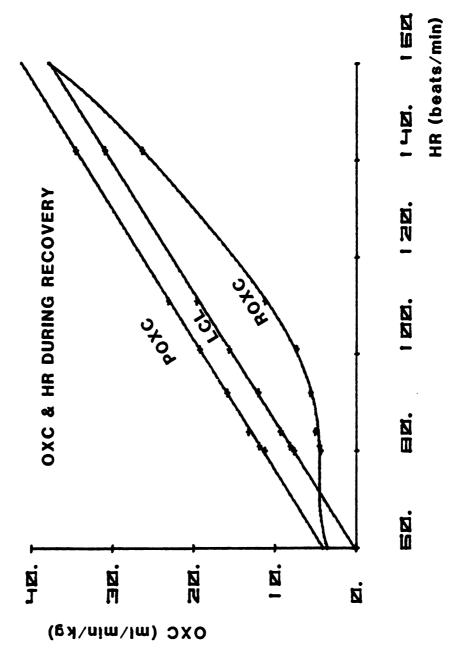


FIGURE 1

Figure 2. Predicted OXC (POXC), lower confidence limits (LCL) and recovery OXC (ROXC) as a function of recovery HR. Mean of five subjects with eight exercise tests in each.





Time ^a (min)	0.5	1.5	3.5	7.5	17.5	27.5	37.5
RHRb	142	111	101	92	84	81	80
SD	12.4	7.8	7.0	8.9	5.6	4.6	4.4
ROXC ^C	26.4	11.3	7.4	· 5.7	5.1	4.6	4.4
SD	2.5	0.85	1.4	0.54	0.59	0.57	0.59
LCL ^d	31.0	19.7	15.7	12.1	9.4	8.2	7.7
SD	3.3	2.1	2.0	2.4	2.2	2.1	2.2
POXC ^e	34.6	23.2	19.3	15.9	13.3	12.0	11.3
SD	3.0	2.0	2.2	1.8	1.2	0.7	0.8
DLCL ^f	4.6	8.4	8.3	6.4	4.3	3.6	3.3
DOXC9	8.2	12.1	11.9	10.2	8.2	7.4	6.9
%DOXC ^h	24	52	62	64	62	62	61

Table 6. Comparison of mean recovery values and their respective standard deviation (SD) for eight trials in each of five subjects.

^aTime refers to midpoint for collection of expired air.

^bRecovery heart rate in beats/min. Resting HR = 60 ± 6 .

^CRecovery oxygen consumption (ml/min/kg). Resting OXC = 4.2 ± 0.4 . d95% lower confidence limit for predicted oxygen consumption.

ePredicted oxygen consumption during recovery, based on the RHR and the prediction equation for previous exercise.

^fDifference between LCL and ROXC (DLCL).

^gDifference between POXC and ROXC (DOXC).

^hDOXC as % of POXC.

Figure 3. HR during 40-minute recovery. Mean of five subjects with eight exercise tests in each. The x-axis intersects the y-axis at the preexercise HR.

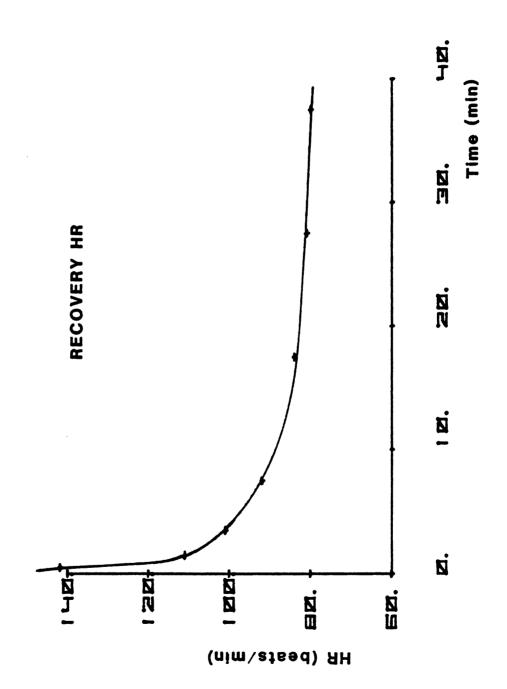




Figure 4. Predicted OXC (POXC), upper curve. Lower 95% confidence level for OXC (LCL), middle curve. Recovery OXC (ROXC), lower curve, as a function of time. The x-axis represents resting value. Mean of five subjects with eight exercise tests in each.

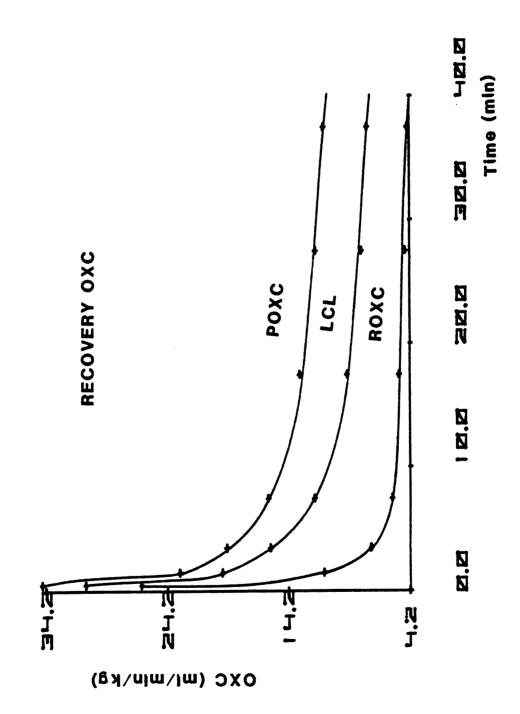
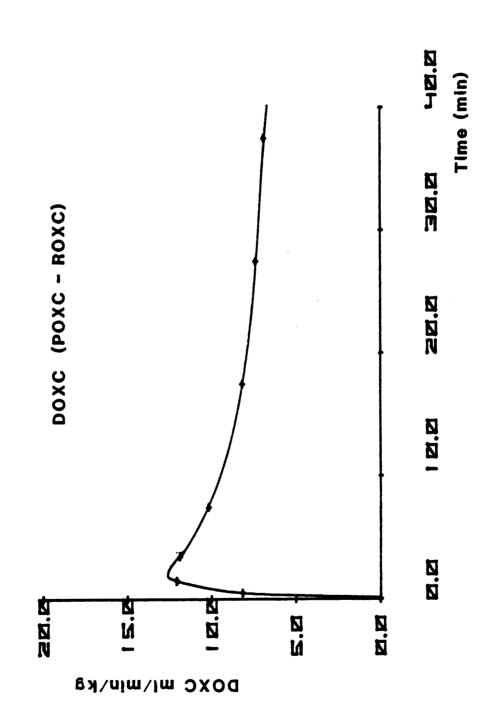




Figure 5. Difference between predicted OXC and recovery OXC (DOXC) as a function of time during 40 minute recovery. Mean of eight trials in each of five subjects.



remainder of the 40 min recovery (Figure 6). This means that during the first 3 minutes of recovery, the HR progressively lags behind the ROXC, but thereafter, returns toward the resting levels at the same speed until ROXC reaches the resting level or slightly below. The RHR does not return to resting level until three or more hours later.

The effect of exercise intensity (S) and duration (D) on this lag in RHR was investigated in four subjects. Because not all subjects completed all the S-levels, only the first five treatments are included in the statistical analysis, while the data are reported for any treatment completed by at least one subject. In order to reduce intrasubject variability, the IHR(HR-RHR) is used. The means for IHR are shown in Table 7, the mean ROXC in Table 8 mean POXC in Table 9, and mean DOXC in Table 10. IHR is plotted in Figure 7 for 2 exercise intensities and in Figure 8 for 10 and 30 min of exercise, while Figure 9 shows ROXC at 2 exercise intensities and Figure 10, DOXC as a function of time. LCL, ROXC and DOXC are shown for all the treatments in Table 11. Except for speed 4 at 10 minutes, ROXC is always less than LCL and the relationship is similar to that already discussed for recovery after exercise at constant speed (Figure 2). The effect of duration of exercise appears stronger than the effect of intensity of exercise on DOXC. Thus, the mean (over the 40 min recovery) DOXC for 30 minutes is 10 ml/min/kg and for 10 minutes is 6.6 ml/min/kg.

The results of the analysis of variance show that both treatment and time have a highly significant effect upon IRHR, ROXC, and DPOXC.

Figure 6. DOXC as % of POXC vs recovery time (40-minute recovery). Mean of eight trials in five subjects.

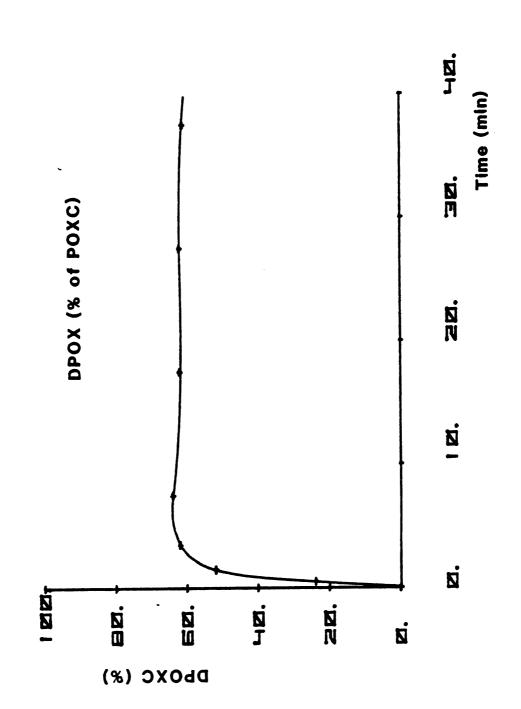




Table 7. Mean recovery incremental^a heart rates after exercise on a treadmill at 4 different speeds, each speed maintained for 10 and 30 minutes, respectively. Mean of four subjects.

	•			Recov	very time ⁰	:	
Setting	Time ^b (min)	0.5	3.0	7.5	17.5	27.5	37.4
4	10	57	25	17	14	11	10
4	30	73	38	28	24	20	14
5	10	71	32	26	22	18	16
5	30	84	47	36	31	27	24
6	10	90	45	32	30	26	22
6d	30	100	56	40	32	29	21
7 ^d	10	103	51	39	34	31	22
7e	30	112	63	54	44	41	37

^aIncremental = measured HR - resting HR in each subject.

^bTime refers to duration of exercise at the final setting.

^CRecovery time refers to the midpoint for collection of expired air.

^dThree subjects only.

^eOne subject, only.

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error for difference between 2 treatments at one time is 4.74 (beats/min).

Standard error of difference between 2 times for one treatment is 5.58 (beats/min).

	-	Recovery time (min) ^b							
Setting	Time ^a (min)	0.5	3.0	7.5	17.5	27.5	37.5		
4	10	19.6	6.1	4.2	4.0	3.9	3.8		
4	30	19.0	6.2	4.6	4.7	4.0	4.0		
5	10	22.9	6.9	4.8	4.5	4.2	4.3		
5	30	24.3	7.4	5.0	4.3	4.3	3.8		
6	10	26.9	8.3	5.4	4.7	4.1	3.8		
6 ^C	30	29.3	10.2	6.5	5.5	5.2	4.9		
7 ^C	10	30.9	8.9	6.6	4.9	4.7	4.3		
7d	30	31.2	8.9	5.8	5.2	4.4	3.5		

Table 8. Mean recovery oxygen consumption in four subjects after exercise on a treadmill at four different speeds, two runs at each speed for 10 and 30 minutes, respectively.

^aDuration D of final treadmill setting.

^bRefers to midpoint of collection of expired air.

^CThree subjects only.

^dOne subject only.

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error for difference between 2 treatments at one time is 0.698 (ml/min/kg).

Standard error for difference between 2 times for one treatment is 2.13 (ml/min/kg).

Resting OXC = 3.6 ml/min/kg.

Table 9. Mean predicted oxygen consumption during recovery following exercise on a treadmill at four different speeds in four subjects. Each speed is repeated twice for 10 and 30 minutes, respectively. Time is given in minutes. Standard deviation (SD) is also given.

				Recovery time (min) ^b						
Settin	g	Time ^a (min)	0.5	3.0	7.5	17.5	27.5	37.5		
		()			POXC in	ml/min/kg	•			
4		10	23.9	13.1	9.7	9.0	7.5	7.1		
	SD		5.5	4.4	3.5	3.7	2.6	2.0		
4		30	27.3	17.2	12.9	11.4	9.9	8.3		
	SD		5.1	3.8	3.2	3.5	2.1	2.4		
5		10	29.8	15.6	12.2	10.9	9.3	8.6		
	SD		6.8	3.8	3.7	. 3.2	1.8	2.1		
5		30	32.6	19.5	15.6	14.0	12.5	11.5		
	SD		4.3	4.7	3.5	4.1	4.4	4.1		
6		10	33.4	19.1	14.0	13.2	12.0	10.7		
	SD		5.8	3.6	2.5	3.4	3.1	1.9		
6 ^C		30	38.8	23.4	18.0	15.2	14.2	11.5		
	SD		4.2	3.4	3.5	2.2	2.4	3.2		
7 ^C		10	38.1	20.4	17.7	15.0	13.6	10.7		
	SD		2.2	2.4	2.6	2.2	2.2	2.3		
7 ^d		30	40.0	23.8	20.9	17.6	16.6	15.3		

^aDuration (D) of final exercise step.

^bRefers to midpoint for collection of expired air.

^cThree subjects only. ^dOne subject only.

							_
				Recovery	time (mi	n) ^b	
Setting	Time ^a (min)	0.5	3.0	7.5	17.5	27.5	37.5
4	10	4.3	6.7	5.5	4.6	3.6	3.3
4	30	8.8	11.4	9.4	7.3	6.9	5.4
5	10	6.9	8.5	7.5	6.3	5.1	4.4
5	30	. 8.4	11.9	10.6	9.7	8.2	7.6
6	10	6.1	9.7	8.7	8.5	7.1	6.8
6 ^C	30	9.5	13.3	11.6	9.8	9.0	6.5
7 ^c	10	8.2	11.8	10.1	9.1	9.4	6.6
7 ^d	30	8.8	15.0	15.1	12.4	12.2	11.8
Mear	1	7.6	11.1	9.8	8.4	7.8	6.6
D10 D30	e f	6.4 8.9	9.2 12.9	8.0 11.7	7.1 9.8	6.3 9.1	5.3 7.8

Table 10. Mean difference (4 subjects) between the predicted oxygen consumption and the actual oxygen consumption during recovery after running on a treadmill at four different speeds. The subjects ran at each speed twice for 10 and 30 minutes, respectively.

^aDuration (D) of final exercise step.

^bRefers to midpoint for collection of expired air.

^cThree subjects only. ^dOne subject only.

^eMean for 10 min. ^fMean for 30 min.

DOXC in ml/min/kg.

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error for difference between 2 treatments at one time is 1.8 (ml/min/kg).

Standard error for difference between 2 times for one treatment is 0.97 (ml/min/kg).

. . .

Figure 7. Mean incremental HR (IHR) for four subjects during 40-minute recovery after exercise for 10 minutes at speed 4 (8.8 km/hr), lower curve; and speed 6 (12.5 km/hr), upper curve. Significant difference at 0.5, 3.0, 7.5, 17.5, and 27.5 minutes. Mean values for four subjects. Standard error for difference of 2 treatments at one time is 4.74 (beats/ min). Standard error for difference of 2 times for one treatment is 5.58 (beats/min).

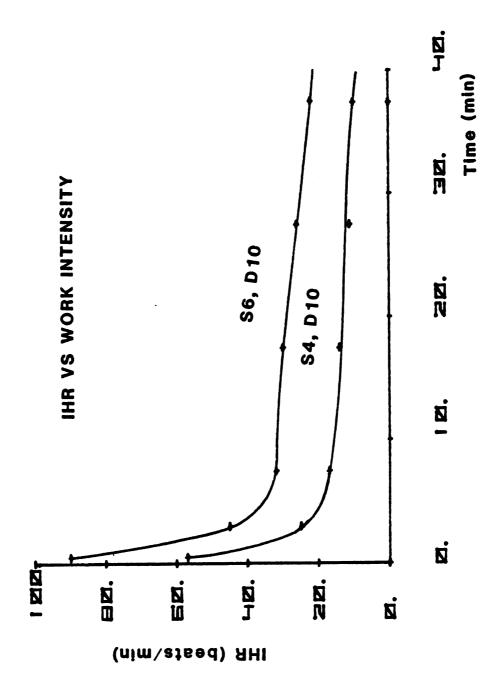


FIGURE 7

Figure 8. Incremental HR during 40-minute recovery (RIHR) after exercise at speed 5 (10.5 km/hr). Upper curve for D = 30 minutes, and lower curve for D = 10 minutes.

.

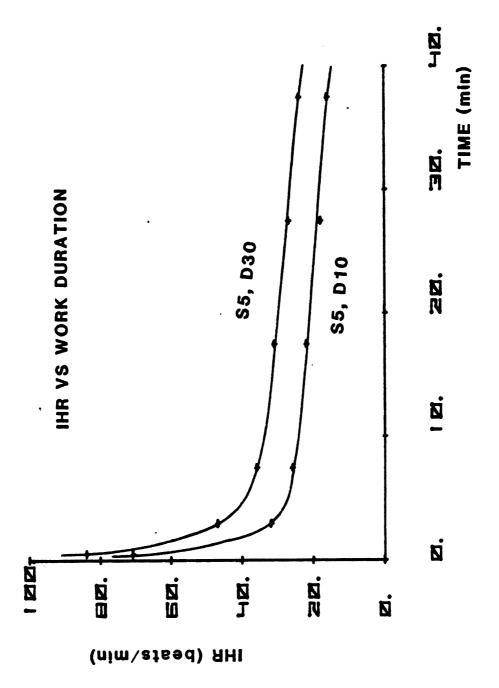




Figure 9. Recovery OXC (ROXC) as a function of time. As there is no difference in ROXC due to the duration of exercise, 10- and 30-minute periods are combined. Upper curve is for speed 4 (8.8 km/hr) and lower curve for speed 6 (12.5 km/hr). Significant differences for the first minute of recovery only. Mean values for four subjects. Standard error for difference of two treatments at one time is 0.698 (ml/min/kg). Standard error for difference of two times at one treatment is 2.13 (ml/min/kg).

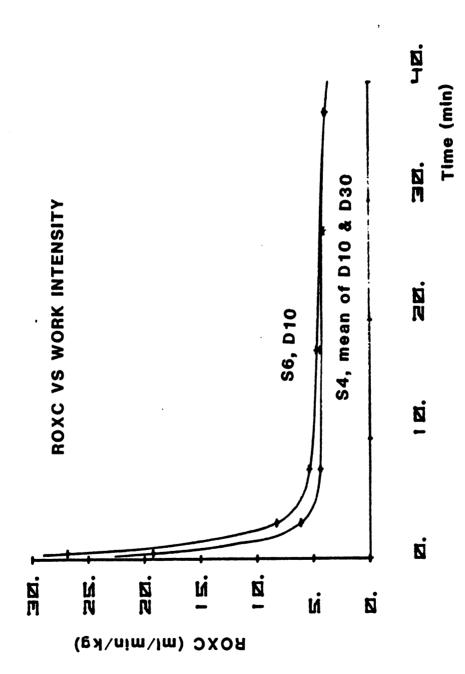


FIGURE 9

Figure 10. Mean DOXC (difference between predicted and recovery OXC) for four subjects. Significant differences at 3.0 and 7.5 minutes. Since there was no significant speed effects, the mean for 30 minutes for Speed 4 - Speed 7 (upper curve) is compared to the mean for 10 minutes of exercise for Speed 4 - Speed 7 (middle curve). The lower curve is for the lowest speed (8.8 km/hr) which is significantly different from S6 when all time points are considered.

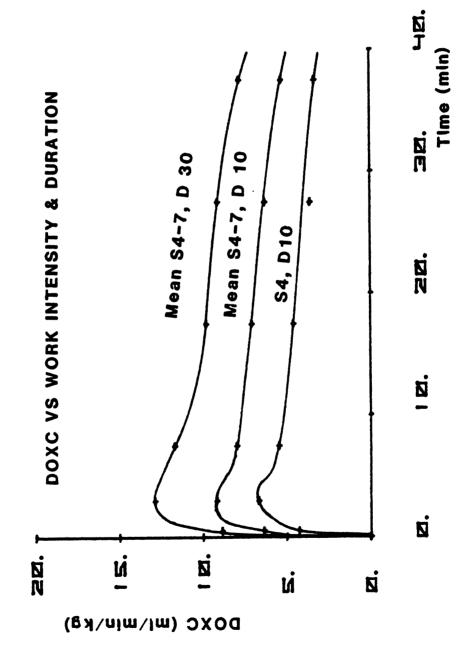




Table 11. Mean values of 95% lower confidence limit (LCL) for predicted oxygen consumption (POXC), recovery oxygen consumption (ROXC), and the difference (DLCL) between LCL and ROXC in ml/kg/min following exercise on a treadmill at four different speeds (S) for 10 and 30 minutes (D), respectively. Mean of four subjects.

					Recovery	time (mir		
	Sa	Dp	0.5	3.0	7.5	17.5	27.5	37.5
LCL ROXC DLCL	4	10	20.5 19.6 0.9	9.6 6.1 3.5	6.2 4.2 2.0	4.9 4.0 0.9	3.8 3.9 -0.1	3.4 3.8 -0.4
LCL ROXC DLCL	4	30	25.2 19.0 6.2	15.2 6.2 9.0	10.8 4.6 6.2	8.8 4.7 4.1	7.8 4.0 3.8	6.2 4.0 2.2
LCL ROXC DLCL	5	10	27.4 22.9 4.5	13.2 6.9 6.3	9.8 ⁻ 4.8 5.0	8.5 4.5 4.0	6.9 4.2 2.7	6.2 4.3 1.9
LCL ROXC DLCL	5	30	29.8 24.3 5.5	16.6 7.4 9.2	12.8 5.0 7.8	11.2 4.3 6.9	9.6 4.3 5.3	8.6 3.8 4.8
LCL ROXC DLCL	6	10	30.6 26.9 3.7	16.5 8.3 8.2	11.7 5.4 6.3	10.4 4.7 5.7	9.0 4.1 4.9	8.3 3.8 4.5
LCL ROXC DLCL	6 ^d	30	35.7 29.3 6.4	20.5 10.2 10.3	15.2 6.5 8.7	12.3 5.5 6.8	11.3 5.2 6.1	8.5 4.9 3.6
LCL ROXC DLCL	7 ^d	10	34.8 30.9 3.9	17.2 8.9 8.3	13.3 6.6 6.7	11.5 4.9 6.6	10.4 4.7 5.7	7.5 4.3 3.2
LCL ROXC DLCL	7 ^e	30	36.3 31.2 5.1	20.4 8.9 11.5	17.4 5.8 9.6	14.1 5.2 8.9	13.1 4.4 8.7	11.8 3.5 8.3

^aTreadmill setting.

^bDuration of final setting.

^CRefers to midpoint for collection of expired air.

^dThree subjects only.

^eOne subject only

Whereas the time response for IRHR and ROXC depends on the treatment, this is not the case for DPOXC. The results of designed contrast using Bonferroni t (Miller, 1966) are shown in Table 12. There is no interaction between speed and duration of exercise for either IRHR, ROXC, or DOXC, although there is a strong trend for DOXC at all six recovery times. IRHR is significantly affected by both speed and duration of exercise, whereas ROXC is only affected by speeds and only at 0.5 minutes. There is a strong trend for ROXC during the first 10 minutes of recovery. DOXC is significantly affected by duration of exercise only although there is a strong trend for exercise intensity (S), which becomes significant when the total response during recovery (over all time points) is considered.

In conclusion, it can be said that the HROXCR is different during exercise and recovery, and the reason is that the HR lags behind ROXC during the first 3 minutes of recovery. This lag in RHR is primarily effected by duration of exercise, and above 75% of maximal oxygen consumption there is little if any effect of exercise intensity.

DOXC.
and
ROXC
IRHR,
for
significance ^a
of
Test
12.
Table

						•		
Var.	Test	Contrast	0.5	3.0	Recovery time 7.5	me (min) 17.5	27.5	37.5
IRHR	Inter. Time Speed Speed	1,4VS2,3 1,3VS2,4 1,2VS3,4 1VS5	-0.40 3.87b 3.34b 6.23b	0.27 3.74b 2.14 3.77b	-0.13 2.80b 2.27 2.83b	-0.13 2.54 3.02b	0.00 2.40 1.87 2.83 ^c	0.53 1.60 2.14 2.26
ROXC	Inter. Time Speed Speed	1,4VS2,3 1,3VS2,4 1,2VS3.4 1VS5	2.05 0.81 8.71b 7.40b	0.41 -0.61 2.03 2.23	-0.20 -0.61 1.01 1.22	$\begin{array}{c} -0.91 \\ 0.51 \\ 0.10 \\ 0.71 \end{array}$	0.00 0.20 0.61 0.20	-0.71 -0.30 0.30 0.00
рохс ^d	Inter. Time Speed Speed	1,4VS2,3 1,3VS2,4 1,2VS3,4 1VS5	-1.17 -2.34 -0.86 	-0.51 -3.15b 0.89 	-0.31 -2.72 ^c 1.25 ^{be}	0.27 -2.38 1.60	-0.08 -2.50 1.09	0.43 -2.07 1.28
^a Desig t = <u>+</u> 2 the 90 ^e Contr	^a Designed cont t = <u>+</u> 2.56; (α the 90% level. ^e Contrast base	<pre>rast (Bor = 0.1). d(m = 5) d on all</pre>	<pre>(Bonferroni t; M 1). ^bSignificant = 5), t = <u>+</u> 3.06 all six recovery</pre>	Miller,1966) nt at the 95% 06 (α = 0.05 ry times.		d	(α = nifican .68 (α	0.05). t at =0.1).

Discussion

Effect of Diet and Previous Exercise

There is no evicence of any effect of the different dietary treatments upon either the slope or y-intercept of the regression equation between the HR and OXC. The HROXCR varies with time around one for the subject's characteristic value. One can therefore expect to find occasional significant values. As can be seen from Table 4, the significance does not occur at a particular treatment in more than one subject, but occurs at random.

As shown in Table 5, where the regression equation between HR and OXC is determined twice one hour apart in the same subject, the regression lines are significantly different, both in regard to slope and y-intercept. After one hour of rest, the OXC has returned to baseline values, but the HR is still elevated by about 20 beats/minute. Using IHR will correct for the differences in resting HR, and as shown in Table 5, it does decrease the significance of the differences in the y-intercepts. However, since the slopes are different, this does not necessarily mean that the regression lines are more homogenous.

Schutz, et al (1981) have presented evidence that meals change the HROXCR. In their protocol, they first determined the regression equation, then fed the meals before re-establishing the regression line. Thus, the effect they found may equally well be due to an effect of the previous exercise. The older subjects presumably were not very fit; so even though they used low intensity exercise, an effect of previous exercise cannot be excluded. The effect of previous exercise, in our experience, is dependent upon the training of the subjects.

Lundgren (1946) in his study on lumberjacks, also found an effect of breakfast on the HROXCR, but upon further study concluded that it was due to increased activity unrelated to the meal. He found a similar effect after a day of lumbering. Others have found that prolonged, heavy exercise causes an increase in HR (Saltin, 1964; Rowell, 1974).

Recovery from Exercise and the HROXCR

The HROXCR during exercise and recovery are statistically significantly different throughout exercise at all work intensities, since the recovery OXC (ROXC) is less than the lower 95% confidence limit (LCL) for the predicted OXC (POXC) based on the RHR and the prediction equation obtained during exercise. It is remarkable that DOXC increases rapidly during the first 3-4 min of exercise, and thereafter decreasing in parallel with the RHR. This means that the RHR falls behind the ROXC during the first few minutes of recovery, and then returns toward the resting value at the expected rate based on ROXC. During exercise there is a redistribution of the blood volume, with less blood going to the viscera and unused muscles (Saltin, 1964b; Saltin et al, 1968). During recovery the perfusion to these organs must increase which can further decrease an already compromised vascular volume and cause a decrease in SV and a compensatory increase in HR (Tanaka et al, 1979; Nadel et al, 1980). In Figure 3, the RHR is plotted as a function of time. The HR stabilizes at a level of about 20 beats/minute above the resting level (x-axis). Figure 7 demonstrates the effect on the IHR of increasing the workload, and the differences are significant except at the last time interval (Table 12). Nandi and Spodick (1977) have obtained similar results, although they only followed the recovery for 5 minutes. However, as shown in Figure 8 for IRHR, there is also a significant effect of duration of exercise. The difference due to D slowly decreases with time, and it is significant for the first 10 minutes only. When we look at the ROXC as shown in Figure 4 (lower curve), we see that the OXC decreases rapidly at first, and then slowly approaches the baseline level (the x-axis = preexercise OXC). As shown in two subjects followed for 5 hours post-exercise, the ROXC ends approaching the POXC (Fig. 2), and both RHR and ROXC are lower than the preexercise resting level.

For the ROXC there are significant differences for the first minute only. There is no effect of D upon the ROXC. This condition is clearly different from what was seen for the HR, where both speed and duration of exercise have significant effect. These results are in agreement with the data of Hagberg, et al (1980), who found that the OXC response was two-phasic during recovery. A rapid phase of approximately 2 minutes duration was significantly affected by the work intensity, but not by duration. Neither speed nor duration had any effect on the slow component, except for work intensities of higher than 65% of maximal oxygen consumption. In the present study, we found highly significant effects of speed for the first minute only (fast component),

but no effect of duration. Although not significant, we found a strong positive effect of speed upon the slow component, even at 60% of the maximal oxygen consumption.

In Figure 4, the two upper lines show that POXC and 95% lower confidence limit (LCL), and since both are based on the actual RHR, it is not surprising that they lag behind ROXC (lower curve). In Figure 2, POXC, LCL and ROXC are plotted as a function of RHR to illustrate the statistical significance. Only duration of exercise has a statistically significant effect upon DOXC, which is illustrated in Figure 10 by the two upper curves. However, as seen from Table 12, the effect of speed is significant when the lowest and highest speeds are contrasted for all times simultaneously.

It is concluded that the RHR does not predict the ROXC based on the regression equation during the preceding exercise period, because the RHR lags behind the ROXC during the first three minutes of recovery. This lag is affected by duration of exercise only above 65% of maximal oxygen consumption. At lower work intensities, there is an effect of intensity (S) also; and this lag in RHR is not observed below 50% of maximal oxygen consumption in moderately fit subjects.

Analysis of the regression equations for HR and OXC during exercise following different dietary treatments provided no evidence for a dietary effect upon the HROXCR; but exercise clearly changes both the slope and y-intercept. It is the duration of activity which is primarily responsible for the lag in RHR, although at low work intensities (around 50% of maximal oxygen consumption, depending on the physical training of the subjects), it is not observed.

Despite the intra-subject variability, and the deviation of ROXC from POXC, the HROGSR still forms the best basis for prediction of activity and HR and OXC are still correlated when only the day-to-day variation in resting OXC is considered. The deviation during recovery will make the observed differences appear greater than they really are, which may be acceptable for many applications. Mode of work (Andrews, 1971) and environmental temperature (Dill & Consolazio, 1962) also affect the HROXCR. It is recommended that at least three determinations of the individual regression line be made; but if that is not possible, it may be better to use a common slope.

During steady state exercise, the HR slowly increases. That is why duration of exercise has a significant effect upon RHR, as has also been found by others (Tanaka et al, 1979). This increase in HR appears to be correlated with an increase in body temperature (Tanaka et al., 1979). As the HR increases, there is a simultaneous decrease in stroke volume (Roberts & Wenger, 1980; Nadel et al, 1980). SV, in addition to exercise, is also affected by the state of hydration (Nadel et al, 1980); and in fact, the effect of exercise is probably related to the decrease in vascular volume (Saltin, 1964b). Both thermal and metabolic dehydration cause a decrease in stroke volume and an increase in HR to compensate for the decrease in stroke volume at submaximal workload, while there is no difference in HR and OXC at maximal workload (Saltin, 1964a). Costill and Fink (1974) reported that dehydration due to heat was similar to that caused by exercise in the effect upon HR and stroke volume, provided the same weight loss was obtained. Senay (1979) reported that training decreases the plasma

volume loss due to exercise, and in this study the best-trained subjects (as evidenced by lactate accumulation) had a less pronounced increase in HR during exercise and a faster recovery than less welltrained subjects.

CHAPTER 4

DIETARY INDUCED THERMOGENESIS DURING REST AND EXERCISE: EFFECT OF MEALS AND DIET ON OXYGEN CONSUMPTION AND ENERGY SUBSTRATE UTILIZATION DURING REST AND EXERCISE

Synopsis

In this study the oxygen consumption was followed for 8 hours after consumption of a fat, carbohydrate, or protein meal respectively with measurements in the post-absorptive state (no food) as control. We found that each meal had a (statistically significant) different effect on the oxygen consumption during rest, whereas neither the meals nor a high fat or high carbohydrate diet (for 3 days prior to exercise) had any effect during exercise. Substrate utilization during rest (as evaluated by the RQ) was affected only by the carbohydrate meal, whereas none of the treatments had any effect during exercise. Exercise following the meals compared to control (postabsorptive state) did not show any thermogenic effect.

Introduction

Overfeeding is generally thought to cause an increase in thermogenesis. It has been shown that subjects generally gain less weight than the excess energy intakes suggest (Dauncy, 1979). Conflicting evidence comes from Glick, et al (1977) who reported that there was no effect of overfeeding on thermogenesis. One explanation for the controversy may be that energy cost of physical activity is increased due to overfeeding, but Norgan and Durnin (1980) found no such effect. Norgan and Durnin also argue against what is termed "luxus-consumption" (Apfelbaum, et al, 1971). It has also been shown that underfeeding results in a decreased resting metabolic rate (RMR) (Apfelbaum, et al, 1971; Dauncy, 1979).

Rubner (1902) and Lusk (1928) found that protein had a specific thermogenic response; but more recently, Garrow and Hawes (1972) found a similar thermogenic effect of a test meal regardless of its nutrient content. Shetty, et al (1980) found similar results, and Owen, et al (1980) found a thermogenic response to breakfast as evaluated by the oral temperature. However, in an animal (pigs) study, Gurr, et al (1980) found a major response to a low protein diet, but none to a high protein diet. Dauncy (1979) reported a significant increase in 24-hour energy expenditure in man, when the energy intake was changed from glucose to protein.

Cold induced thermogenesis (CIT) due to epinepherine acting on brown adipose tissue and muscle is readily demonstrated in laboratory animals (Heldmeir, 1971) and also in man (Joy, 1963). This response can be reduced by β -adrenergic-blockade with propranolol (Dauncy and Ingram, 1979). In rats, DIT as well as CIT involves changes in the activity of the sympathetic nervous system (Rothwell and Stock, 1979; Glick, et al, 1981).

Capro, et al (1981) demonstrated that the ingestion of a standard meal caused an increase in blood insulin and glucose, but a decrease in plasma free fatty acids in man. In the present study, both protein and carbohydrate meals had a thermogenic effect (maximal response was 38 and 27% respectively). The effect of a carbohydrate meal lasted less than 6 hrs compared to more than 8 hrs for a protein meal.

The effect of diet on muscle glycogen content and therefore exercise endurance is well demonstrated (Karlson and Saltin, 1971). Under conditions where the glycogen stores are not expected to change, it has been shown that substrate concentration in the blood and the RQ are affected, whereas the OXC is not (Foster, et al, 1979) at exercise intensities of 80-100% of maximal oxygen consumption. Others have found an effect of diet upon energy substrate utilization at rest, but not during exercise (Hurni, et al, 1980). It is surprising that pre-exercise glucose feeding should have an effect on the substrate utilization during exercise, unless it affects the glycogen stores, since it has been shown that even at much lower work intensities, the major substrate is intramuscular (Essen, 1978). In the present study, there was no effect of diet or meals upon energy substrate utilization

during exercise (80% of maximal oxygen consumption), but carbohydrate meals increased utilization of carbohydrates during rest.

Apfelbaum, et al (1971) and Miller, et al (1967) found an increase in OXC during exercise following overeating. Bray, et al (1974) found no effect of overfeeding on OXC, but reported that a 4.2 and 12.6 MJ meal increased the OXC during exercise. However, in the present study, we found no thermogenic effect on exercise following a meal.

Methods

The study was divided into two parts. The first part examined the effect of meals on OXC and the relative contribution of fat and carbohydrate to the energy utilization during rest. In the second part the effect of meals and diet composition upon the same parameters was investigated during exercise.

Subjects

Two groups of five healthy male college students volunteered for the study. The physical characteristics of the subjects are given in Table 1. None of the subjects took any medication; and on the day of the experiment, they were requested not to drink any caffeine-containing beverages. They also did not participate in any strenuous physical activity on the day before the experiments were conducted.

Informed Consent

Each subject signed an informed consent form after the details of the experiments were described to him. The form stated the experimental procedures, identified possible risks, and noted that a subject could terminate his participation at any time.

Diets

The composition of the diets and meals is given in Table 2. Each meal contained 700 kcal. The fat meal consisted of 288 ml whipping cream, which the subjects drank in less than 10 minutes. The protein

Physical	Parts					
Characteristics -	R	est	Exer	cise		
	Mean	<u>SD</u>	Mean	<u>SD</u>		
Age (years)	26.6	1.7	24.8	3.1		
Body Weight (kg)	78.2	11.3	74.5	11.9		
Height (cm)	182	6.1	173	7.4		
V _{O2} -max ^a (ml O ₂ /min/kg)			54.1	6.6		

.

Table 1. Physical characteristics of the two groups of five adult male subjects. (SD = Standard Deviation).

^aMaximal OXC.

Table 2.	Compositio kcal for t	Composition of meals and diets. kcal for the different subjects.	The energy content of the diets varied between 2,100 and 2,400	t of the	diets vari	ed between	2,100 and	2,400
			Total	8	% of total energy	ergy	% by weight	ight
Diets or Meals	lea 1 s	Method	Energy (Kcal)	Fat	Protein	Carbo- hydrate	Water	Ash
Fat Meal		Food Tables ^a	700	92	m	ى ا		
Protein Meal	a l	Food Tables	700	22	76	2		
Carbohydrate Meal	te Meal	Food Tables	700	2	2	63		
Fat Diet		Food Tables	2,150	65	15	20		
Carbohydrate Diet	te Diet	Food Tables	2,150	20	15	65		
Fat Diet		Chemical Analysis	2,150	99	16	61	68.0	4.0
Carbohydrate Diet	te Diet	Chemical Analysis	2,150	22	15	63	72.8	3.4
^a Watt and	Merrill (1	^a Watt and Merrill (1963); Church & Church (1975).	(1975).					

•
(1975)
Church
õ
Church
••
(1963);
Merrill
and
la t t

meal consisted of 94 g creamed cottage cheese and 545 g (raw weight) skinned chicken breast, with all visible fat removed. The subjects ate the protein meal in 30 minutes. The carbohydrate meal consisted of 824 g ripe bananas (peeled), which were eaten in less than 10 minutes. The subjects drank 340 cc of ginger ale with the meals containing 1 kcal (Vernors, Detroit, MI), and were allowed salt, pepper, and water ad lib.

During the experimental periods the subjects ate 2,100 to 2,400 kcal per day either as a high fat or as a high carbohydrate diet. The composition is given in Table 2. The subjects were fed one of the two experimental diets in random order for three days prior to the exercise test, which was performed in the postabsorptive state the fourth day. The diet and meals were prepared and eaten in a kitchen adjacent to the exercise laboratory. Two menus were used for each diet: one for breakfast and one for dinner and supper. The menus were repeated for all three days. The subjects were supervised during the meals and ate all the allotted food. Duplicate portions were used for chemical analysis. Fat was analyzed by ether extraction, protein by micro-kjeldahl, minerals as ash $(600^{\circ}C)$ and carbohydrate by difference.

For both diets regular food items were used. Sources of fat were: margarine, mayonnaise, cheese, eggs, bacon and olives; while sources of carbohydrate were: bananas, bread, potatoes, sweet potatoes, beans, carrots and peas. These foods contain some protein. Additional protein came from soy protein. For the fat diet it was imitation chicken, and for the carbohydrate diet it was imitation beef (Worthington Foods, Worthington, OH).

Measurements

Heart rate (HR) was obtained from an electrocardiogram (lead 2 with the positive lead in V5 position). The EKG signal was converted to heart rate through a cardiotachometer (built in our lab) and continuously recorded by a calibrated Sargent Recorder, Model DTM-115-4 (Sargent and Company, Chicago, IL). The mean HR in beats/minute corresponding to each collection of expired air was calculated from the recording.

The oxygen consumption (OXC) was determined by a modified Douglas method (Consolazio, et al, 1963). The expired air was collected through a low resistance valve (Otis-McKerrow, Warren Collins, Inc., Braintree, MA) in light-weight neoprene bags. The composition (O_2 and CO_2 contents) of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers, respectively (Beckman Instruments, Schiller Park, IL). The air volume was determined by metering through (using a constant flow of 50 1/min) a Singer dry gas meter (American Meter Company, Philadelphia, PA).

The mean OXC for the control experiment was compared the baseline (mean resting OXC prior to the ingestion of the meal). To correct for the day to day variation in the resting OXC in each subject the difference between the control and baseline values were added or subtracted from the OXC for that day's treatment.

Lactic acid was determined in duplicate arterialized blood samples (Gambino, 1961; Jung et al, 1966) collected from the fingers after the hand was immersed in warm water (45⁰C) for two minutes. The finger was dried, cleaned with alcohol and punctured with a lancet.

The first drop of blood was wiped off with sterile gauze. One hundred µl of blood was collected in a capillary tube centrifuged at 2000 rpm in 8% perchloric acid. The sample was then incubated at room temperature with lactate dehydrogenase for one hour. The nicotine adenine dinucleotide (NADH) generated was measured on a monocromatic spectrophotometer (Gilford Stasar II, Gilford Instruments, Inc., Oberlin, OH) at 340 nm.

Exercise Tests

The maximal OXC was determined in a continuous incremental test with the subjects exercising on the treadmill until exhaustion. The speed was increased stepwise at 8.8, 10.5, 12.5 and 14.3 km/hour. When the subjects reached the highest speed they could comfortably run (12.5 or 14.3 km/hr) further increases in the workload were accomplished by increasing the treadmill inclination in steps of 2%. The subjects ran 3 minutes at each step, the HR was recorded continuously, three one-minute bags of expired air were collected at each step, and the oxygen consumption calculated for each minute.

The OXC following the different treatments was determined through an incremental exercise test with the final workload corresponding to 80% of the subjects' maximal OXC. Three collections of expired air were made at each step and the mean HR corresponding to each air collection was recorded. The test started at rest with the subject seated in an armchair, while 3 consecutive 5-minute collections of expired air were made. During the following steps the subjects would walk or run on the treadmill. At the 4.8 km/hr and 6.8 km/hr levels

the subjects exercised for 6 minutes each, and at the other levels they exercised for 3 minutes each; except when they reached the final speed (the one corresponding closest to 80% of their maximal OXC), they continued the exercise for 20 minutes. During the final stage, one-minute bags of expired air were collected, every second minute starting with the second minute.

Following the exercise the HR and OXC were determined during 40 minutes of recovery. Two one-minute bags, one three-minute and one five-minute bag of expired air were collected during the first 10 minute of recovery. During the next 30 minutes, 3 five-minute bags of expired air were collected every second 5-minute period. During the alternate 5-minute periods, when no collections were made, the subject remained seated in the armchair, but was relieved of the face mask with air-collection valve and the noseclip. Arterialized blood samples were collected from one of the fingers at 5, 15, 25 and 40 minutes of recovery for determination of the blood lactate concentration.

Experimental Protocol

Rest

For measurements taken during rest, the subjects were seated in an armchair. The subjects were asked to restrict their movement, but at the same time, to change the leg position approximately once every minute to avoid venous pooling. The temperature of the laboratory was kept constant $(\pm 1^{\circ}C)$ throughout the experiment, but it did vary somewhat between experimental days depending on the environmental

temperature. The humidity and barometric pressure could not be controlled.

The day of the experiment, the subjects arrived in the laboratory about 8:00 am in the postabsorptive state (after overnight fast), and spent the day in the laboratory resting, reading, or talking. No food or beverages except the experimental meal were consumed, but the subjects drank as much water as they wanted. The three treatments were (1) the fat meal, (2) the protein meal, and (3) the carbohydrate meal described previously, and for the control treatment, no food was given. The four treatments were done in a random order one week apart.

Before the experimental meal was given, six five-minute baseline measurements of OXC and HR were taken. The subject was seated in an armchair for three consecutive five-minute periods while expired air was collected and HR recorded. The subject was then given a fiveminute break in which he walked around in the laboratory before another series of three consecutive five-minute collections of expired air and recordings of HR were carried out. For each five minutes, one bag of expired air was collected for analysis of OXC and RQ. The mean HR was calculated for each bag of expired air. The mean of the six determinations was used as the baseline (time 0) for that particular treatment (meal). Duplicate arterialized blood samples for lactate determinations were collected before the meals were eaten.

After baseline determination, the subjects ate the experimental meal and three consecutive five-minute collections of expired air and recordings of HR were taken at 30 minutes and at one hour following the completion of the meal and thereafter at one-hour intervals for eight

hours following the meal. The mean of the three measurements was used. During the control treatment, when no meal was given, the protocol was the same, except that no "baseline" measurements were taken. Blood was collected for lactate determination at the same times as the other measurements were taken except for the first hour, when blood samples were taken every 15 minutes. Blood collections were discontinued after 6 hours. During rest lactate was measured only during the control treatment and following the carbohydrate meal.

Exercise

The exercise experiments were carried out over a 10-week period with each test given one week apart. During the first and last tests the maximal OXC were determined. The second test was always a control (postabsorptive state) prior to which the subjects ate their normal diet. Prior to the third test, the subjects consumed one of the experimental diets for three days (two started with the high fat diet and three with the high carbohydrate diet). Then followed another control test before the opposite experimental diet was introduced, three days prior to the fifth test. Following both dietary treatments, the test was carried out in the postabsorptive state. The three meals and a final control were introduced in random order so that all the subjects would not consume the meals in the same order. The exercise tests began 30 minutes after termination of the carbohydrate meals and 2-3 hours following the fat and protein meals, which we had found to correspond to the maximal thermogenic response.

Statistical Analysis

OXC, RQ and lactate were analyzed by a split block (block = subjects) repeated measurements design. The differences among treatment means were evaluated by Tukey's test (Tukey, 1953). The correlation coefficient between HR and OXC was calculated for each test at rest in each subject, and the mean and confidence limits for each dietary treatment and control were calculated by conversion to the Z-score (Steel and Torrie, 1960).

Results

Experiments During Rest

Table 3 shows resting OXC for eight hrs following the experimental meals and during control when no food was given. All treatments were compared with control and each other and the Tukey test was used to determine the significance of the treatment differences shown in Table 3. Although the fat meal caused an increased OXC compared to control from 1 to 6 hrs after the meal it was not significant. The protein meal caused a significant increase in OXC compared to control from 1 to 6 hrs after the meal; and the carbohydrate meal caused OXC to be significantly different from control to 3 hrs following the meal. The protein meal caused a significant increase in OXC from 3 to 6 hrs compared to the carbohydrate meal. The mean increases in OXC compared to control following the fat, protein and carbohydrate meals was 7.3%, 21.2% and 12.8% respectively, while the peak increases were 8.9%, 33.0% and 27.4% respectively. The different thermogenic responses for the treatments are shown in Figure 1.

Table 4 shows the effects of the treatments on the respiratory quotient (RQ). Only following the carbohydrate meal is there a significant effect upon the RQ compared to control. Although the RQ is increased for the eight hrs observed, the increase is only significant (p<0.05) for 3 hrs. The maximal increase in RQ compared to control is 0.14 and the mean increase is 0.09. The RQ for the carbohydrate meal and control is plotted in Figure 2.

Time		Me	eals	
(hrs)	Control	Fat	Protein	Carbohydrate
0	3.60	3.56	3.62	3.56
0.5	3.58	3.62	4.04 ^{cf}	4.06 ^b
1.0	3.60	3.82	4.32 ^{af}	4.32 ^a
2.0	3.62	3.90	4.66 ^{ad}	4.56 ^a
3.0	3.58	3.90	4.76 ^{ag}	4.14 ^{bg}
4.0	3.56	3.90 ^C	4.68 ^{adg}	3.84 ^g
5.0	3.48	3.82 ^C	4.54 ^{adg}	3.70 ⁹
6.0	3.60	3.70	4.22 ^{ach}	3.66 ^h
7.0	3.56	3.62	3.96 ^{cf}	3.64
8.0	3.58	3.58	3.92 ^{cf}	3.58

Table 3. Mean oxygen consumption following the ingestion of various meals for 5 male subjects in the resting state. Control was the postabsorptive state.

a/b/cValues for any of the meals are significantly different from control at the same time (a, p<0.01, b p<0.05; c, p<0.10).</p>

- d/e/fValues for the carbohydrate or protein meal are significantly
 different from the fat meal at the same time (d, p<0.01;
 e, p<0.05; f, p<0.10).</pre>
- g/h/iValues for the protein meal are significantly different from the carbohydrate meal at the same time (g, p<0.01; h, p<0.05; i, p<0.10).</pre>

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error of difference of 2 treatments at one time is 0.157.

Standard error of difference of 2 times for one treatment is 0.088.

OXC in ml/min/kg.

	Me	eals	
Control	Fat	Protein	Carbohydrate
0.750	0.762	0.756	0.760
0.750	0.756	0.756	0.856 ^{cfi}
0.746	0.760	0.760	0.880 ^{eh}
0.750	0.746	0.762	0.892 ^{adh}
0.744	0.736	0.736	0.876 bdg
0.746	0.740	0.754	0.842 ^{cf}
0.764	0.732	0.712	0.808 ⁱ
0.750	0.736	0.752	0.780
0.754	0.738	0.740	0.796
0.742	0.748	0.744	0.790
	0.750 0.750 0.746 0.750 0.744 0.746 0.746 0.764 0.750 0.754	ControlFat0.7500.7620.7500.7560.7460.7600.7500.7460.7500.7460.7440.7360.7460.7400.7640.7320.7500.7360.7540.738	0.7500.7620.7560.7500.7560.7560.7460.7600.7600.7500.7460.7620.7440.7360.7360.7460.7400.7540.7640.7320.7120.7500.7360.7520.7540.7380.740

Table 4. Mean respiratory quotient following the ingestion following ingestion of various meals for 5 male subjects in the resting state. Control was the postabsorbtive state.

a/b/cValues for any one of the meals are significantly different from control at the same time (a, p<0.01; b, p<0.05; c, p<0.10).</p>

d/e/fValues for the carbohydrate - or protein meal are significantly
 different from the fat meal at the same time (d, p<0.01; e,
 p<0.05; f, p<0.10).</pre>

g/h/i Values for the protein and carbohydrate meals are significantly different from each other (g, p<0.01; h, p<0.05; i, p<0.10).

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error of difference of 2 treatments at one time is 0.035.

Standard error of difference of 2 times for one treatment is 0.023.

Figure 1. OXC as a function of time. Treatments are: C = control (lower curve), CH-M = carbohydrate meal (middle curve), and P-M = protein meal (upper curve). All treatments are the mean of 5 subjects. The 5% least significant difference (LSD) is 0.47. SE for difference between the treatment means at the same time is 0.111 ml/min/kg, and the SE for the difference between two time means in the same treatment is 0.062 ml/min/kg.

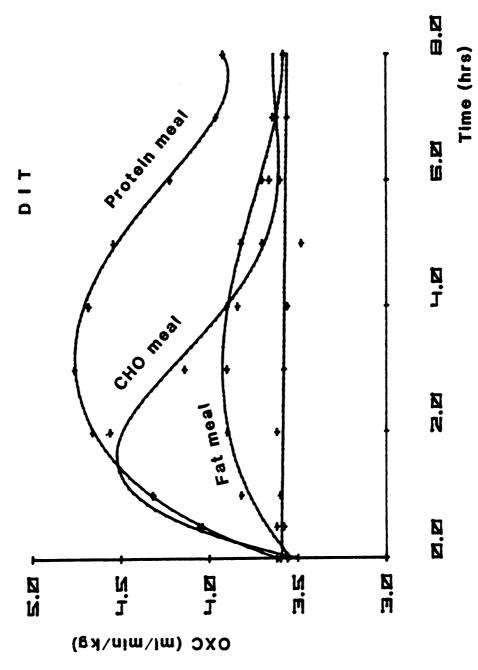


FIGURE 1

Figure 2. Respiratory Quotient (R.Q.) as a function of time for CH-M and Control (C). No other treatment had a significant effect. LSD is (5%) = 0.104. Mean of 5 subjects. (SE for the difference between the treatment means at the same time is 0.025 and the SE for difference between two time means in the same treatment is 0.016.)

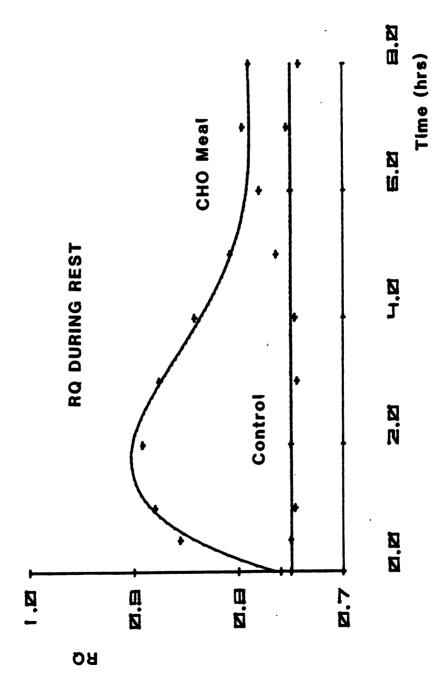


FIGURE 2

As will be reported elsewhere the blood lactate concentration was measured following the carbohydrate meal and during the control. The results are shown in Figure 3. The peak lactate response of about 2.4 mM occurred 1 hr following the meal and the response was significant for 3 hrs.

The mean correlation coefficient (\bar{r}) between the HR and OXC was calculated for each treatment. As seen from Table 5 only for the carbohydrate meal was \bar{r} significantly different from 0 $(\bar{r} = 0.516)$.

Exercise Experiments

The mean OXC for the 20 min during exercise following the fat, protein, carbohydrate meal, the high fat, high carbohydrate diet and control were: 41.7, 41.8, 42.0, 41.5, 41.6 and 42.4 ml/min/kg body weight. The standard error for difference of 2 treatments at one time is 2.58 ml/min/kg. The RQ during exercise following the fat, protein, carbohydrate meals, the high fat, high carbohydrate diets and control were: 0.84, 0.84, 0.87, 0.86, 0.83, 0.85; and the standard error was 0.069. There were no statistical significant treatment differences for either OXC or RQ.

Figure 3. Blood lactate as a function of time after a CH-M and C (at least 5 hours postabsorptive). Mean of 5 subjects. Five percent LSD = 1.471. (The SE for difference between the two treatment means at the same time is 0.239 mM, and the SE for difference between the two time means in the same treatment is 0.072 mM.)

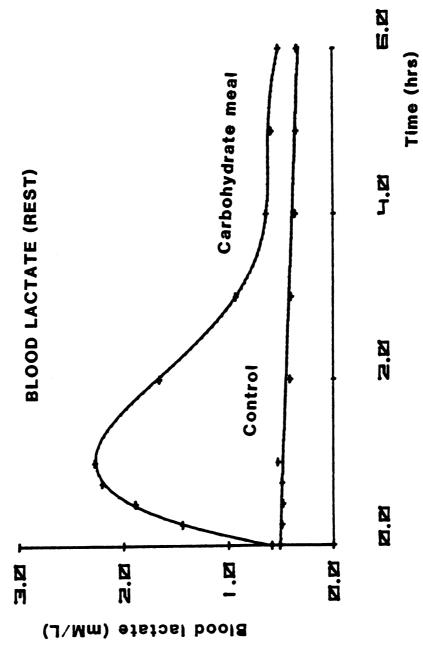


Table 5. Mean correlation coefficient (4) between HR and OXC during control, following a fat meal, protein meal, and a carbohydrate meal. Number of subjects equal to 6 for control, 7 for fat meal, and 8 for carbohydrate meal. n=30 in each treatment for each subject. r=mean value for the subjects completing the treatment.

Treatment	Control	Fat Meal	Protein Meal	Carbohydrate Meal
r	-0.075	0.058	0.041	0.516
r _U a	0.094	0.211	0.195	0.609
r _L b	-2.243	-0.101	-0.117	0.390

 $a_{r_{ij}}$ = Upper 95% confidence limit.

 b_{r_1} = Lower 95% confidence limit.

Discussion

OXC During Rest

CIT is caused by the action of catecholamines upon brown adipose tissue and muscles (Cannon, et al, 1977) and it is generally assumed that DIT operates in a similar manner (Dauncey, 1979; Rothwell and Stock, 1979; Glick, 1981). In the present study there was a specific effect of each energy nutrient on the OXC; however, only the carbohydrate meal caused a simultaneous change in the energy substrate utilization from fat to carbohydrate. This is further substantiated by the increase in RQ. Furthermore, only the change in OXC caused by carbohydrate meal correlates with the HR, as shown in Table 5. As shown by Sonne and Galbo (1980)., catecholamines would be expected to have a simultaneous effect upon HR and OXC. The fact that this is the case for the carbohydrate meal may be related to the effect of carbohydrate upon the central nervous system. Thus, Lansberg and Young (1978) found that overfeeding stimulates and fasting suppresses the sympathetic nervous system in mice, and they state "changes in sympathoadrenal activity may, at least in part, explain the fluctuations in metabolic rate and heat production that occur with changes in diet". Crandall and Fernstrom (1980) found that carbohydrate feeding increased brain indoles in rats, whereas protein meals had no effect.

It is not surprising that fat does not have much effect upon the OXC, since the subjects (after an overnight fast) were already primarily consuming fat, as evidenced by the relatively low RQ's of about

0.76 (Table 4). However, in one subject there was a clear response; and this subject normally did not consume fat; this raises the questions of whether customary food consumption habits affect the DIT. Stock (1980) found no effect of fasting and overeating for one day on the thermogenic response to a test meal, but that does not exclude an effect on the composition of the diet a subject has customarily consumed. One could, for example, anticipate that subjects who normally subsist on low levels of proteins may respond differently to a P-M from subjects who habitually consume large amounts of protein.

Gurr, et al (1980) found that in pigs a high carbohydrate diet caused a larger DIT response than a high protein diet, and similar results have been obtained in rats (Tulp, et al, 1977); but it is important to distinguish between true DIT and effect of increased activity. The feeding pattern and presumably also the composition of the diet can affect the activity (Spangler and Johnson, 1981). The experimental protocol used in the present study was very demanding upon the subjects, and in order to make it more acceptable we tried to let the subjects listen to music. We found, however, that the music caused large variation in the OXC and HR and was therefore not used. In preliminary studies it was also found that the energy content of the meal, and the time the subjects spent eating appeared to effect the thermogenic response. For fat and carbohydrate meals, eating time of about 30 minutes could totally abolish the response; and for protein meal, reducing the energy content to half also reduced the thermogenic response to about half. These factors need to be systematically studied, but they may explain why originally, only

protein was thought to have an effect, and why mixed meals sometimes gave different responses from a meal consisting of a single nutrient (Garrow and Hawes, 1972). In the present study, smoking and caffeinecontaining beverages were excluded, since coffee has been shown to have an effect on OXC (Acheson, et al, 1980).

For the carbohydrate meal the OXC, RQ and blood lactate showed similar time responses, although the lactate response decreased about an hour earlier. The other treatments (fat and protein meals) had no effect on the RQ, and in the case of lactate, were only studied in two subjects (no response). The similarity in the OXC, RQ and lactate response points to the possibility that DIT is a metabolic response unrelated to CIT (and catecholamines). Capro, et al (1981) found that a mixed meal caused a postprandial increase in plasma glucose and insulin that lasted about 4 hours, and a decrease in the free fatty acids for about 5 hours. Similar results were obtained by Owen, et al (1980). Rothwell and Stock (1981) found that insulin was increased during diet-induced thermogenesis in cafeteria-fed rats.

While the data (Table 3) show specific increases in OXC for the protein and carbohydrate meals, the OXC correlates with the HR only for the carbohydrate meal and may indicate a difference in the underlying mechanism for the thermogenic response. It is doubtful that activity played any role in the thermogenic responses found in the present study, but it may have played a major role in many animal studies, as discussed by Scheibel, et al (1979). Thyroid hormones are also involved in thermogenesis (Sestoft, 1980; Danforth, et al, 1979),

1979; Elkin, et al, 1980; Davidson and Chopra, 1979) but experiments of Jung, et al (1979) indicate that thyroid hormones are not involved in DIT.

OXC During Exercise

In contrast to the situation during rest, we find that the dietary treatments have no significant effect on OXC during exercise. The mean OXC during exercise is 42 ml/min/kg and the standard error for difference between treatment at the same time is $2.58 \text{ ml } 0_2/\text{min/kg}$, which gives a coefficient of variation (CV) of about 6%. In a study designed to test the variability in the energy expenditure of standard activity, Durnin and Namyslowski (1958) found a CV of about 10% in ten male subjects, which is the same order of magnitude as in the present study.

Several authors have found an effect of diet upon the OXC during exercise (Miller, et al, 1967; Apfelbaum, et al, 1971; Bray, et al, 1974), either as a result of overfeeding or following a meal. However, Swindells (1972) did not find any effect of meals upon DIT during exercise, and Norgan and Durnin (1980) did not find any effect of overfeeding when correcting for increases in body weight (as in our study). It is possible, as suggested by Bray, et al (1974), that the size of a meal makes a difference, so that an effect is only apparent after intake of at least 4.2 MJ. Few people normally eat a meal consisting of 12.6 MJ as used by Bray, et al (1974), and using single nutrients, as we did, it is impossible for most subjects to eat more than about 3 MJ. Garby and Lambert (1977) found no effect of a 4 MJ meal.

Energy Substrate Utilization During Exercise

There is no effect of any dietary treatment on the type of substrate used during exercise. The mean RQ during exercise was 0.85, the SE is 0.07, and the CV is 7%. In the present study, the subjects exercised at about 80% of their maximal oxygen consumption, and the exercise lasted for only 20 minutes in order to avoid an effect of diminishing muscle glycogen stores. When evaluating the effect of diet upon the substrate utilization during exercise, it is necessary to consider both the relative work load and the duration of exercise. The percent of substrate supplied by glucose increases from negligible at rest to about 35% at moderate work loads for one hour (Wahren, et al, 1971); but during longer exercise times, the glucose utilization increases further and peaks at 90-180 minutes of exercise (Alborg, et al, 1974).

At relatively low levels of activity (30-50% of maximal oxygen consumption), where blood borne substrates play an important role in the energy supply, pre-exercise glucose feeding increases glucose utilization during exercise. The changes appear to be hormonally mediated (insulin and glucagon) (Ahlborg and Felig, 1977; Pirnay, et al, 1977; Luyckx, et al, 1978; Maughan, et al, 1978). Martin, et al (1978) found that a high fat diet for 3 days (90% of energy as fat or protein) at 70% of the maximal oxygen consumption markedly decreased carbohydrate utilization, but such a diet very likely affected the muscle glycogen stores (Astrand, 1967). At 80% and 100% of maximal oxygen consumption, Foster, et al (1979) found that ingestion of glucose compared to water gave a small increase in the

RQ, but the variations in the RQ were not significant at 20 minutes. Hurnie, et al (1980) found that although diet affected the RQ at rest, this was not the case during exercise.

Ravussin, et al (1979) studied the effect of exercise (40% of maximal oxygen consumption) on glucose utilization in glycogen depleted subjects and controls following ingestion of 100 g¹³C-glucose, and found that the use of exogenous glucose was similar, but the decreased glycogen utilization in depleted subjects was replaced by lipid oxidation, not endogenous carbohydrate.

Artificially elevating plasma FFA with heparin caused an increased utilization of fat, whereas 75 g of glucose injested 45 minutes prior to exercise increased glucose utilization (RQ) at 70% of maximal oxygen consumption (Costill, et al, 1977). Bergstrom, et al (1979) used nicotinic acid to block the release of FFA from adipose tissue. Up to about 65% of maximal oxygen consumption, it caused an increase in the RQ. Despite a marked decrease in plasma FFA, it had no effect upon the muscle substrate utilization at higher work loads. Hagenfeldt and Wahren (1972) found that at high work loads, fat oxidation exceeded FFA uptake, suggesting utilization of intramuscular lipid stores. Jones, et al (1980) likewise found that at light exercise, FFA is extracted from the circulation, whereas in heavy exercise, muscle triglycerides play a more important role. Essen (1978) has also shown a significant contribution of intramuscular substrates (glycogen and triglycerides) during exercise, and this contribution increases with the intensity of the exercise.

Bylund-Felenius, et al (1981) have presented evidence that the intramuscular oxygen tension (pO_2) determines the substrate utilization during exercise, but hormones (insulin, glucagon and catecholamines) probably also play a role (Norris, et al, 1978). At rest and during low intensity exercise, it is clear that diet affects both blood nutrient levels and substrate utilization (Ahlborg and Felig, 1977). While at high work intensities, blood levels of nutrients are still affected by diet (Foster, et al, 1979); the present study gives no support for a dietary effect upon substrate utilization at high work loads (>75% of maximal oxygen consumption) provided the glycogen stores are not affected. Similar results were obtained by Bergstrom, et al (1969).

CHAPTER 5

٠

EFFECT OF MEALS AND DIET ON POSTEXERCISE BLOOD LACTATE LEVELS

•

Synopsis

The effects of a high fat diet and a high carbohydrate diet; and fat, protein and carbohydrate meals on postexercise lactate were investigated. Five subjects were exercised for 20 minutes at approximately 80% of maximal oxygen consumption. After each of the diets and control they were exercised in the postabsorptive state, and after the meals in the absorptive state. The lactate production was also measured in the resting state for six hours following a carbohydrate meal, and during control (postabsorptive state). Following a carbohydrate meal, there was a four-fold increase in the whole blood lactate concentration compared to control. After exercise, blood lactate was measured at 5, 15, 25, and 40 minutes. There was a significant time by treatment interaction for carbohydrate meal compared to control and fat meal. The total response was also significantly different for carbohydrate meal compared to fat meal. These results do not support the hypothesis that diet affects the lactate production during exercise, although a carbohydrate meal has a significant effect upon the decrease in blood lactate concentration during recovery.

Introduction

The widely used concept that the percentage of maximal oxygen consumption can be used to equalize the physiological work load has recently been questioned (Katch, et al, 1978). The authors showed that at the same relative percent oxygen uptake, the subjects had different anaerobic thresholds. Kinderman, et al (1979) have showed that the aerobic-anaerobic threshold is a better predictor of the physiological work load.

It is controversial whether decreased muscle oxygen tension, high rate of glycogenolysis, or both are responsible for lactate production (Bylund-Fellenius, 1981; Jacobs, 1981). However, it is clear that a number of factors affect the blood lactate. These include muscle respiratory capacity and fiber composition, mode (rest or exercise) of recovery (McGrail, et al, 1978; Poortmans, et al, 1978; Stanford, et al, 1981), as well as muscle glycogen content (Graham, 1978; Ivy, et al, 1980; Jacobs, 1981).

A number of studies have demonstrated how previous exercise and diet can affect muscle glycogen and lactate production (Jacobs, 1981; Essen, 1978; Bonen et al, 1981; Maughan, et al, 1978; and Maughan and Poole, 1981). Thus, Maughan (1978, 1981) has shown that at both high and low intensity work, an exercise-diet regimen that increases muscle glycogen also increases lactate production during exercise and vice versa. The effect of lactate agrees with the explanation of Newsholme and Crabtree (1979) that lack of substrate (glycogen) will reduce the flow through a metabolic pathway. Robin and Hance (1980) emphasize the importance of enzymes and substrates in regulation of the glycogenolytic rate, and it has been demonstrated that blood FFA's affect the glycolytic rate and lactate production (Bergstrom, et al, 1969; Foster, et al, 1979); although Bergstrom found no effect at high work loads.

In the present study we evaluated the effect of diet on the post-exercise blood lactate under conditions in which one would not expect any effect on muscle glycogen. We found that diet did effect blood lactate recovery rate but not the blood lactate concentration 5 min following exercise.

Methods

The study was divided into two parts. The first part examined the effect of meals on OXC and the relative contribution of fat and carbohydrate to the energy utilization during rest. In the second part the effect of meals and diet composition upon the same parameters was investigated during exercise.

Subjects

Two groups of five healthy male college students volunteered for the study. The physical characteristics of the subjects are given in Table 1. None of the subjects took any medication; and on the day of the experiment, they were requested not to drink any caffeine-containing beverages. They also did not participate in any strenuous physical activity on the day before the experiments were conducted.

Informed Consent

Each subject signed an informed consent form after the details of the experiments were described to him. The form stated the experimental procedures, identified possible risks, and noted that a subject could terminate his participation at any time.

<u>Diets</u>

The composition of the diets and meals is given in Table 2. Each meal contained 700 kcal. The fat meal consisted of 288 ml whipping cream, which the subjects drank in less than 10 minutes. The protein

Physical	Parts					
Characteristics	R	est	Exer	Exercise		
	Mean	<u>SD</u> ^a	Mean	<u>SD</u>		
Age (years)	26.6	1.7	24.8	3.1		
Body Weight (kg)	78.2	11.3	74.5	11.9		
Height (cm)	182	6.1	173	7.4		
MaxOXC ^b (ml/min/kg)			54.1	6.6		

Table 1. Physical characteristics of the two groups of five adult male subjects.

.

^aSD = Standard Deviation.

^bMaximal OXC.

r

Table 2. Compositic kcal for t	Composition of meals and diets. kcal for the different subjects.	The energy content of the diets varied between 2,100 and 2,400	t of the	diets vario	ed between	2,100 and	2,400
and a second		Total	8	% of total energy	ergy	% by weight	ight
Diets or Meals	Method	Energy (Kcal)	Fat	Protein	Carbo- hydrate	Water	Ash
Fat Meal	Food Tables ^a	700	92	ო	2		
Protein Meal	Food Tables	700	22	76	2		
Carbohydrate Meal	Food Tables	700	2	5	63		
Fat Diet	Food Tables	2,150	65	15	20		
Carbohydrate Diet	Food Tables	2,150	20	15	65		
Fat Diet	Chemical Analysis	2,150	99	16	19	68.0	4.0
Carbohydrate Diet	Chemical Analysis	2,150	22	15	63	72.8	3.4
a.							

,

^aWatt and Merrill (1963); Church & Church (1975).

meal consisted of 94 g creamed cottage cheese and 545 g (raw weight) skinned chicken breast, with all visible fat removed. The subjects ate the protein meal in 30 minutes. The carbohydrate meal consisted of 824 g ripe bananas (peeled), which were eaten in less than 10 minutes. The subjects drank 340 cc of ginger ale with the meals containing 1 kcal (Vernors, Detroit, MI), and were allowed salt, pepper, and water ad lib.

During the experimental periods the subjects ate 2,100 to 2,400 kcal per day either as a high fat or as a high carbohydrate diet. The composition is given in Table 2. The subjects were fed one of the two experimental diets in random order for three days prior to the exercise test, which was performed in the postabsorptive state the fourth day. The diet and meals were prepared and eaten in a kitchen adjacent to the exercise laboratory. Two menus were used for each diet: one for breakfast and one for dinner and supper. The menus were repeated for all three days. The subjects were supervised during the meals and ate all the allotted food. Duplicate portions were used for chemical analysis. Fat was analyzed by ether extraction, protein by micro-kjeldahl, minerals as ash $(600^{\circ}C)$ and carbohydrate by difference.

For both diets regular food items were used. Sources of fat were: margarine, mayonnaise, cheese, eggs, bacon and olives; while sources of carbohydrate were: bananas, bread, potatoes, sweet potatoes, beans, carrots and peas. These foods contain some protein. Additional protein came from soy protein. For the fat diet it was imitation chicken, and for the carbohydrate diet it was imitation beef (Worthington Foods, Worthington, OH).

Measurements

Heart rate (HR) was obtained from an electrocardiogram (lead 2 with the positive lead in V5 position). The EKG signal was converted to heart rate through a cardiotachometer (built in our lab) and continuously recorded by a calibrated Sargent Recorder, Model DTM-115-4 (Sargent and Company, Chicago, IL). The mean HR in beats/minute corresponding to each collection of expired air was calculated from the recording.

The oxygen consumption (OXC) was determined by a modified Douglas method (Consolazio, et al, 1963). The expired air was collected through a low resistance valve (Otis-McKerrow, Warren Collins, Inc., Braintree, MA) in light-weight neoprene bags. The composition (O_2 and CO_2 contents) of the collected air was immediately determined using the Beckman LB-2 carbon dioxide and Beckman OM-11 oxygen analyzers, respectively (Beckman Instruments, Schiller Park, IL). The air volume was determined by metering through (using a constant flow of 50 1/min) a Singer dry gas meter (American Meter Company, Philadelphia, PA).

The mean OXC for the control experiment was compared the baseline (mean resting OXC prior to the ingestion of the meal). To correct for the day to day variation in the resting OXC in each subject the difference between the control and baseline values were added or subtracted from the OXC for that day's treatment.

Lactic acid was determined in duplicate arterialized blood samples (Gambino, 1961; Jung et al, 1966) collected from the fingers after the hand was immersed in warm water (45⁰C) for two minutes. The finger was dried, cleaned with alcohol and punctured with a lancet.

The first drop of blood was wiped off with sterile gauze. One hundred µl of blood was collected in a capillary tube centrifuged at 2000 rpm in 8% perchloric acid. The sample was then incubated at room temperature with lactate dehydrogenase for one hour. The nicotine adenine dinucleotide (NADH) generated was measured on a monocromatic spectrophotometer (Gilford Stasar II, Gilford Instruments, Inc., Oberlin, OH) at 340 nm.

Exercise Tests

The maximal OXC was determined in a continuous incremental test with the subjects exercising on the treadmill until exhaustion. The speed was increased stepwise at 8.8, 10.5, 12.5 and 14.3 km/hour. When the subjects reached the highest speed they could comfortably run (12.5 or 14.3 km/hr) further increases in the workload were accomplished by increasing the treadmill inclination in steps of 2%. The subjects ran 3 minutes at each step, the HR was recorded continuously, three one-minute bags of expired air were collected at each step, and the oxygen consumption calculated for each minute.

The OXC following the different treatments was determined through an incremental exercise test with the final workload corresponding to 80% of the subjects' maximal OXC. Three collections of expired air were made at each step and the mean HR corresponding to each air collection was recorded. The test started at rest with the subject seated in an armchair, while 3 consecutive 5-minute collections of expired air were made. During the following steps the subjects would walk or run on the treadmill. At the 4.8 km/hr and 6.8 km/hr levels

the subjects exercised for 6 minutes each, and at the other levels they exercised for 3 minutes each; except when they reached the final speed (the one corresponding closest to 80% of their maximal OXC), they continued the exercise for 20 minutes. During the final stage, one-minute bags of expired air were collected, every second minute starting with the second minute.

Following the exercise the HR and OXC were determined during 40 minutes of recovery. Two one-minute bags, one three-minute and one five-minute bag of expired air were collected during the first 10 minute of recovery. During the next 30 minutes, 3 five-minute bags of expired air were collected every second 5-minute period. During the alternate 5-minute periods, when no collections were made, the subject remained seated in the armchair, but was relieved of the face mask with air-collection valve and the noseclip. Arterialized blood samples were collected from one of the fingers at 5, 15, 25 and 40 minutes of recovery for determination of the blood lactate concentration.

Experimental Protocol

Rest

For measurements taken during rest, the subjects were seated in an armchair. The subjects were asked to restrict their movement, but at the same time, to change the leg position approximately once every minute to avoid venous pooling. The temperature of the laboratory was kept constant $(\pm 1^{\circ}C)$ throughout the experiment, but it did vary somewhat between experimental days depending on the environmental

temperature. The humidity and barometric pressure could not be controlled.

The day of the experiment, the subjects arrived in the laboratory about 8:00 am in the postabsorptive state (after overnight fast), and spent the day in the laboratory resting, reading, or talking. No food or beverages except the experimental meal were consumed, but the subjects drank as much water as they wanted. The three treatments were (1) the fat meal, (2) the protein meal, and (3) the carbohydrate meal described previously, and for the control treatment, no food was given. The four treatments were done in a random order one week apart.

Before the experimental meal was given, six five-minute baseline measurements of OXC and HR were taken. The subject was seated in an armchair for three consecutive five-minute periods while expired air was collected and HR recorded. The subject was then given a fiveminute break in which he walked around in the laboratory before another series of three consecutive five-minute collections of expired air and recordings of HR were carried out. For each five minutes, one bag of expired air was collected for analysis of OXC and RQ. The mean HR was calculated for each bag of expired air. The mean of the six determinations was used as the baseline (time 0) for that particular treatment (meal). Duplicate arterialized blood samples for lactate determinations were collected before the meals were eaten.

After baseline determination, the subjects ate the experimental meal and three consecutive five-minute collections of expired air and recordings of HR were taken at 30 minutes and at one hour following the completion of the meal and thereafter at one-hour intervals for eight

hours following the meal. The mean of the three measurements was used. During the control treatment, when no meal was given, the protocol was the same, except that no "baseline" measurements were taken. Blood was collected for lactate determination at the same times as the other measurements were taken except for the first hour, when blood samples were taken every 15 minutes. Blood collections were discontinued after 6 hours. During rest lactate was measured only during the control treatment and following the carbohydrate meal.

Exercise

The exercise experiments were carried out over a 10-week period with each test given one week apart. During the first and last tests the maximal OXC were determined. The second test was always a control (postabsorptive state) prior to which the subjects ate their normal diet. Prior to the third test, the subjects consumed one of the experimental diets for three days (two started with the high fat diet and three with the high carbohydrate diet). Then followed another control test before the opposite experimental diet was introduced, three days prior to the fifth test. Following both dietary treatments, the test was carried out in the postabsorptive state. The three meals and a final control were introduced in random order so that all the subjects would not consume the meals in the same order. The exercise tests began 30 minutes after termination of the carbohydrate meals and 2-3 hours following the fat and protein meals, which we had found to correspond to the maximal thermogenic response.

Statistical Analysis

Blood lactate during rest and recovery from exercise following the various treatments was analyzed by a split block (block = subject) repeat measurements design (each subject served as his own control). The differences between treatment means were evaluated by designed contrast (Bonferroni t statistics; Miller, 1966).

Results

The resting blood lactate in the postabsorptive (after an overnight fast) resting state was about 0.5 mM and decreased slightly with time. Following the carbohydrate meal there was a rapid increase in the blood lactate concentration to a mean value of 2.3 mM, which was significantly different from control for the first 3 hrs. The results are given in Table 3 and plotted in Figure 1.

Regardless of time the postexercise blood lactate level are similar in the postabsorptive state for controls and when the subjects were fed a high fat or a high carbohydrate diet for 3 days. The mean for the 3 conditions was calculated and presented as "D" in Table 4 and plotted in Figure 2. The fat and protein meals give similar values and the mean is presented as "M" in Table 4 and plotted in Figure 2. For D the blood lactate decreases from 3.0 at 5 min of recovery to 0.7 at 40 min of recovery. The values for M - although lower - are not statistically different as shown in Table 5. Following the carbohydrate meal the postexercise blood lactate decreases from 2.6 mM at 5 min to 1.8 mM at 40 min of recovery and is plotted separately in Figure 2. The total blood lactate response following the carbohydrate meal is significantly different from that following the fat meal as shown in Table 5. There is also a significant interaction between the post-exercise blood lactate following the carbohydrate meal and both the control and fat meal at 5 and 40 min of recovery. Thus

Time (hrs)	Control	Carbohydrate Meal	Dunnett's Test* ^t D
0.00	0.51	0.59	0.37
0.25	0.49	1.44	4.43 ^a
0.50	0.48	1.89	6.58 ^a
0.75	0.49	2.21	8.03 ^a
1.00	0.53	2.28	8.17 ^a
2.00	0.41	1.66	5.84 ^a
3.00	0.40	0.93	2.47 ^b
4.00	0.36	0.64	1.31
5.00	0.35	0.59	1.12
6.00	0.34	0.52	0.84

Table 3. Blood lactate in arterialized blood for 6 hours following a carbohydrate meal, and in the postabsorptive state following an overnight fast as control. Mean of five subjects.

*Significant difference between treatment means of the same time was determined by Dunnett's test (Dunnett, 1955, 1964).

^aSignificant at 1% level (t = 3.36).

^bSignificant at 5% level (t = 2.31).

Standard errors of individual treatment means at one time are irrelevant for comparisons or reliability.

Standard error of difference of two treatments at one time is 0.214 (mM).

Standard error of difference of two times for one treatment is 0.102 (mM).

Figure 1. Blood lactate concentration (mean of five subjects) in the postabsorptive state (following an overnight fast) as control (lower line) and following a 2.94 MJ carbohydrate meal (bananas). The S.E. for difference between the treatment means at the same line is 0.339 (mM); and the S.E. for difference between the means at two times is 0.102 (mM).

x

.

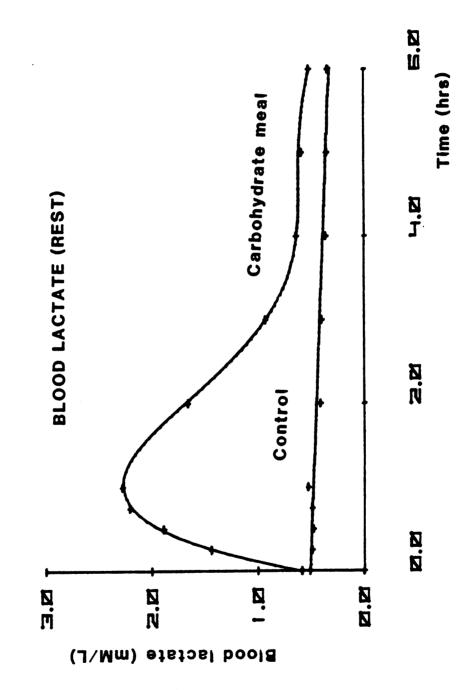


Figure 1

Figure 2. Blood lactate concentration during recovery from exercise at 80% of maximal oxygen consumption for 20 minutes (mean of five subjects). Upper curve following CH-M; middle curve is the postabsorptive state; and lower curve is mean of F-M and P-M. The S.E. for differences between the treatment means at the same time is 0.483 (mM); the S.E. for differences between two times is 1.13 (mM), and the S.E. for comparing interaction between two treatments at two different times is 0.282 (mM).

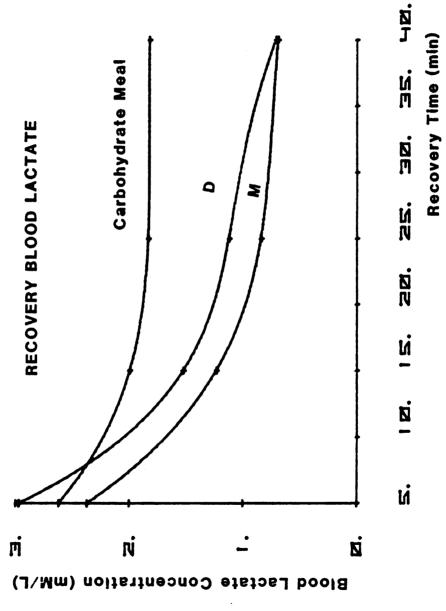


FIGURE 2

	Blood Lactate (mM)				
Treatments	Pre- exercise	5 min	15 min	25 min	40 min
Control	0.782	2.94	1.76	1.10	0.68
Fa t Diet	0.517	2.99	1.48	1.22	0.75
Carbohydrate Diet	0.782	2.99	1.33	1.03	0.69
Fat Meal	0.543	2.21	1.03	0.65	0.45
Pro te in Meal	0.772	2.53	1.43	1.02	0.93
Carbohydrate Meal	0.698	2.62	1.99	1.83	1.82
Mean D ^a		2.97	1.52	1.12	0.71
Mearn M ^b		2.37	1.23	0.84	0.69
		2.31	1.23	0.04	0.0

Table 4. Mean blood lactate concentration during 40 minutes recovery from 20 minutes of exercise at 80% of maximal 0 x C in 5 subjects.

^aMean of Control, Fat and Carbohydrate diets.

^bMean of Fat and Protein meals.

Standard errors of individual treatment means at one time are relevant for comparisons or reliability.

Standard error of difference of two treatments at one time is 520 (mM).

Standard error of difference of two times for one treatment $\mathbf{i} \le 0.200 \pmod{\mathbf{m}}$.

Table 5. Bonferroni t for test of significance of postexercise blood lactate concentration. CH-M is tested for interaction (between 5 and 40 minutes) against C and F-M. Total (40 minute) response to CH-M is tested against total response to F-M; and the postabsorptive (P) trials (C, F-D, CH-D) are tested against the absorptive (A) trials (F-M and P-M) at 5 minutes postexercise. There are four contrasts (m = 4).

Test	Contrasts	т _в
Interaction	CH-M vs C at 5 & 40 min	^a 5.17 ¹
Interaction	CH-M vs F-M at 5 & 40 min	^a 3.40 ¹
Total CH vs Total F	CH-M vs F-M at all times	^a 3.70 ²
M vs D	F-M, P-M vs C, F-D, CH-D at 5 min	1.77 ²

^aSignificant at 5% level

¹Tabular $T_B = 2.66$ at 5% level ²Tabular $T_B = 2.74$ at 5% level the rate of return for the post-exercise blood lactate differ from the other treatments.

Discussion

Shortly after ingestion of the carbohydrate meal there is a significant increase in the blood lactate during rest compared to control. Although blood glucose was not measured in the present study, Capro, et al (1981) found that plasma glucose and insulin were elevated for approximately four hours following standard meal, and that plasma FFA were depressed for approximately 5 hours. Similar responses to ingestion of 100 g glucose were found for blood glucose, insulin, and FFA by Ravussin, et al (1979) and Luyckx, et al (1978). Owen, et al (1980) found that a typical American breakfast elevated blood glucose, lactate, and pyruvate for about 2 hours, while triglycerides were still elevated at 3 hours. They also found that FFA, glycerol and ketone bodies were depressed for about 3 hours following the breakfast.

The blood lactate following the ingestion of the fat and protein meal was not studied except in a preliminary study in one subject, where no effect was found compared to control. However, one would expect a decrease in blood glucose and lactate and an increase in FFA (Seyffert, et al, 1967) if any change occurs, since fat is already the major energy substrate used in the postabsorptive state during rest.

It is controversial whether the oxygen tension in muscle tissue is the primary determinant of lactate production during exercise (Graham, 1978); but Bylund-Fellenius, et al (1981) have recently presented evidence in support of this concept. However, the individual variation in lactate production is also determined by muscle respiratory capacity and fiber composition (Ivy, et al, 1980), which in turn is modified by training (Johnson, 1969).

Jacobs (1981) has shown that the level of muscle glycogen affects lactate production during exercise. Thus, at low levels of muscle glycogen, glycogenolysis decreased drastically, but above about 40 mM/kg there is no effect. It has been shown in rats (Stankiewicz-Choroszucha and Gorski, 1978) that when muscle glycogen is not available, muscle triglycerides become the major source of energy. They also reported that the level of blood FFA did not affect intramuscular utilization of triglycerides, but blood FFA level did affect the rate of glycogen utilization. In agreement with these findings, Maughan, et al (1978, 1981) found that a high carbohydrate diet (after glycogen depleting exercise) elevated plasma lactate during and following exercise compared to control, whereas low carbohydrate diet increased plasma FFA under these conditions. These dietary effects were seen both after low and high intensity work. Bonen, et al (1981) also found that a high carbohydrate diet increased blood lactate during high intensity work. Both these authors used a protocol designed to deplete muscle glycogen, and under such conditions, it is clear that diet has an effect upon blood lactate and substrate utilization.

Costill, et al (1977) found that artificially elevating FFA (with heparin) following a fat meal increased utilization of fat as a substrate during high intensity work (70% of maximally oxygen consumption) compared to preexercise ingestion of 75 g glucose. Consequently,

the utilization of muscle glycogen was decreased 40% compared to control. They also found that ingestion of glucose increased glycogen utilization 17% (partly at the expense of blood carbohydrate). Bergstrom, et al (1969), however, found that decreasing arterial FFA by nicotinic acid did affect substrate utilization, only at low intensity exercise (below about 65% of maximal oxygen consumption).

In the present study, our diets and exercise were designed not to have a significant effect upon muscle glycogen stores, because there was sufficient carbohydrate present in the high fat diet and the exercise was of relatively short duration. In the case of the carbohydrate meal, there clearly was a significant interaction (Table 5), which means that the carbohydrate meal interferes with the normal post-exercise recovery of blood lactate. This is not surprising, in view of the highly significant response in blood lactate to the carbohydrate meal at rest. There are great intra-individual differences in blood lactate: one subject (more fit, as indicated by his maximal oxygen consumption of 66 ml/min/kg compared to about 50 for the other subjects) had very little lactate response to exercise. Following the carbohydrate meal lactate response was decreased at five minutes following exercise, but then the blood lactate actually increased to that expected following a carbohydrate meal at rest (1.5 mM). Apparently, either the liver does not produce lactate during exercise, or it is metabolized by the exercising muscle (Poortmans, 1978). These results do not support the hypothesis that diet affects the blood lactate concentration during high intensity exercise, although a carbohydrate meal clearly affects the rate of decrease in blood

lactate during the recovery period.

CHAPTER 6

CONCLUSIONS

CONCLUSIONS

We developed one hundred regression equations between HR and OXC from 10 exercise test in each of 10 subjects. Several aspects of the regression equation between HR and OXC were investigated based on these equations. Inhomogeneity of the regression lines was found both within and between the subjects. Most (about 70%) of the variability is between subject variability as evaluated by the repeatability R₁ and R is only slightly decreased by using more homogenous subjects.

It is not valid to use the regression equation between HR and OXC measured during exercise to predict recovery OXC. This can lead to as much as 300% overestimation of OXC. The reason for this was found to be a lag in the HR recovery relative to the OXC recovery, which occurred during the first 3 min of the recovery period after exercise at 80% of the maximal OXC. This lag in the HR recovery is presumably caused by a loss of vascular volume, which causes a decrease in stroke volume and a compensatory increase in HR. Duration of exercise rather than the intensity of exercise was found to be primarily responsible for this lag in recovery HR. Repeated exercise was found to be one factor that could cause the regression line to change with respect to slope and y-intercept.

Meal feeding compared to the postabsorptive state and a high fat compared to a high carbohydrate diet did not alter the linear regression equation between the HR and OXC.

There is a need to confirm and expand the present finding in groups of different physical training, which will also allow for comparing the effect of physical training on the linear regression equation between HR and OXC. Furthermore the effects of coffee, smoking and mental stress (such as anxiety) on the regression equation between HR and OXC need to be studied.

The effects of a high fat, a high protein and a high carbohydrate meal (700 kcal) on dietary induced thermogenesis were studied at rest. Each meal differed in its thermogenic response as measured by OXC, both with respect to the maximal response and its duration. The maximal increases in OXC was 8.9%, 33.0% and 27.4% for the fat, protein and carbohydrate meals respectively. There was also an increase in blood lactate (about 3 mM) and respiratory quotient following the carbohydrate meal, which indicates a shift in the metabolism. Only during the carbohydrate meal was the increase in OXC positively correlated with the HR, giving indirect evidence that the regulatory mechanism of DIT for the 3 meals may be different. There was no thermogenic response during exercise under our experimental procedures.

There is a need for a study of the regulatory mechanisms behind the thermogenic response to the different meals, as the results indicate that they are different. If the thermogenic response to a carbohydrate is regulated differently, it can resolve the controversy about the thermogenic response to the energy substrates and may help explain why some people develop obesity. There is also a need to study the effects of meal size, eating time and composition on habitual food intake on the thermogenic response to fat, protein and carbohydrate

meals. Furthermore, it may be of interest to study the effect of smoking on DIT since smoking cessation can lead to increased body weight. A carbohydrate meal was shown to significantly increase blood lactate concentration compared to control; and when the meal preceeded exercise it resulted in elevated blood lactate levels during recovery (2mM). However, there was no effect of either meals or diets high in fat or carbohydrate on the substrate utilization or blood lactate concentration during exercise at 80% of maximal OXC. BIBLIOGRAPHY

References to Chapter I

- Acheson, KJ, Zahorska-Markiewicz B, Anantharaman K, et al. (1980) Caffeine and coffee: Their influence on metabolic rate and substrate utilization in normal weight and obese individuals. Am J Clin Nutr 33:989-997.
- Ahlborg G & Felig P (1977) Substrate utilization during prolonged exercise preceded by ingestion of glucose. Am J Physiol 233: E188-E194.
- Ahlborg G, Felig P, Hagenfeldt L, et al (1974) Substrate turnover during prolonged exercise in man: Splanchnic and leg metabolism of glucose, free fatty acids and amino acids. J Clin Invest 53: 1080-1090.
- Andrews RB (1969) The relationship between measures of heart rate and rate of energy expenditure. AIIE Transactions 1:2-10.
- Andrews RB (1971) Net heart rate as a substitute for respiratory calorimetry. Am J Clin Nutr 24:1139-1147.
- Apfelbaum M, Bostsarron J & Lacatis D (1971) Effect of caloric restriction and excessive caloric intake on energy expenditure. Am J Clin Nutr 24:1405-1409.
- Astrand P, Guddy TE, Saltin B, et al (1964) Cardiac output during submaximal and maximal work. J Appl. Physiol 19:268-274.
- Astrand P & Rodahl K (1977) Textbook of Work Physiology: Physiological Bases of Exercise. McGraw-Hill, New York, pp. 455-459.
- Baldwin KM and Winder WW (1977) Adaptive responses in different types of muscle fibers to endurance exercise. Ann New York Acad Sci 301:411-423.
- Bergstrom J, Hultman E, Jorfeldt L, et al (1969) Effect of nicotinic acid on physical working capacity and on metabolism of muscle glycogen in man. J Appl Physiol 26:170-176.
- Billman GE, Dickey DT, Teoh KK, et al (1981) Effect of central venous blood volume shifts on arterial baroreflex control of heart rate. Am J. Physiol 241:H571-H575.

- Bonen A, Malcolm SA, Kilgour RD, et al (1981) Glucose ingestion before and during intense exercise. J Appl. Physiol 50:766-771.
- Booth F (1977) Effects of endurance exercise on cytochrome C turnover in skeletal muscle. Ann New York Acad Sci 301:431-439.
- Borghols EAM, Dresen MHW & Hollander AP (1978) Influence of heavy weight carrying on the cardiorespiratory system during exercise. Eur J Appl Physiol 38:161-169.
- Bradfield RB (1971) A technique for determination of usual daily energy expenditure in the field. Am J Clin Nutr 24:1148-1154.
- Bradfield RB, Huntzicker PB & Fruehan GJ (1970) Errors of group regressions for prediction of individual energy expenditure. Am J Clin Nutr 23:1015-1016.
- Bray GA, Whipp BJ & Koyal SN (1974) The acute effects of food intake on energy expenditure during cycle ergometry. Am J Clin Nutr 27: 254-259.
- Brengelmann GL, Johnson JM, Hermansen, L, et al (1977) Altered control of skin blood flow during exercise at high internal temperatures. J Appl Physiol 43:790-794.
- Brobeck JR (1981) Models for analysing energy balance in body weight regulation. In: Cioffi LA, James WPT, Van Itallie TB (eds) The Body Weight Regulatory System: Normal and Disturbed Mechanisms. Raven Press, New York, pp 1-7.

Brody S (1945) Bioenergetics and Growth. Reinhold, New York.

- Buskirk, ER, Thompson RH & Whedon GD (1963) Metabolic response to cold air in men and women in relation to total body fat content. J Appl Physiol 18:603-612.
- Bylund-Fellenius A-C, Walker PM, Elander A, et al (1981) Energy metabolism in relation to oxygen partial pressure in human skeletal muscle during exercise. Biochem J 200:245-255.
- Carlson LA and Pernow B (1961) Studies on the peripheral circulation and metabolism in man. I. Oxygen utilization and lactate-pyruvate formation in the legs at rest and during exercise in healthy subjects. Acta Physiol Scand 52:328-342.
- Christensen EH & Hansen O (1939) Abreitsf igkeit und Ehrnarung. Skandinavishes Archiv fur Physiologie 81:160-171.
- Costill DL, Coyle E, Dalsky G, et al (1977) Effects of elevated plasma FFA and insulin on muscle glycogen usage during exercise. J Appl Physiol 43:695-699.

- Costill DL, Dalsky TP & Fink WJ (1978) Effects of caffein ingestion on metabolism and exercise performance. Med Sci Sports 10:155-158.
- Costill DL & Fink WJ (1974) Plasma volume changes following exercise and thermal dehydration. J Appl Physiol 37:521-525.
- Crapo PA, Reaven GM & Olefsky JM (1981) Hormonal and substrate responses to a standard meal in normal and hypertriglyceridemic subjects. Metabolism 30:331-334.
- Damato AN, Galante JG & Smith WM (1966) Hemodynamic response to treadmill exercise in normal subjects. J Appl Physiol 21:959-966.
- Danforth E, Jr & Burger AG (1981) Hormonal control of thermogenesis. In: Cioffi LA, James WPT, Van Itallie TB (eds) The Body Weight Regulatory System: Normal and Disturbed Mechanisms. Raven Press, New York, pp 107-114.
- Dauncey MJ (1979) Energy metabolism in man and the influence of diet and temperature: A review. J Human Nutr 33:259-269.
- Dietze G, Wicklmayr M, Bottger I, et al (1980) The kallikrein-kinin system and muscle metabolism: Biochemical aspects. Agents and Actions 10:335-338.
- Dill DB & Consolazio CF (1962) Responses to exercise as related to age and environmental temperature. J Appl. Physiol 17:645-648.
- Ekblom B, Goldbarg A & Gullbring B (1972) Response to exercise after blood loss and reinfusion. J Appl Physiol 33:175-180.
- Ekblom B, Wilson G & Astrand PO (1976) Central circulation during exercise after venesection and reinfusion of red blood cells. J Appl Physiol 40:379-383.
- Essen B (1977) Intramuscular substrate utilization during prolonged exercise. Ann New York Acad Sci 301:30-44.
- Essen B (1978) Studies on the regulation of metabolism in human skeletal muscle using intermittent exercise as an experimental model. Acta Physiol Scand (Supple 454), Stockholm.
- Foster C, Costill DL & Fink WJ (1979) Effects of preexercise feedings on endurance performance. Med Sci Sports 11:1-5.
- Foster DO & Frydman ML (1978a) Comparison of microspheres and ⁸⁶Rb⁺ as tracers of the distribution of cardiac output in rats indicates invalidity of ⁸⁶Rb⁺-based measurements. Can J Physiol Pharmacol 56:97-109.

- Foster DO & Frydman ML (1978b) Nonshivering thermogenesis in the rat. II. Measurement of blood flow with microspheres point to brown adipose tissue as the dominant site of the calorigenesis induced by noradrenaline. Can J Physiol Pharmacol 56:110-122.
- Foster DO & Frydman ML (1979) Tissue distribution of cold-induced thermogenesis in conscious warm- or cold-acclimated rate reevaluated from changes in tissue blood flow: The dominant role of brown adipose tissue in the replacement of shivering by nonshivering thermogenesis. Can J Physiol Pharmacol 57:257-270.
- Foulkes JG & Cohen P (1979) The hormonal control of glycogen metabolism: Phosphorylation of protein phosphatase inhibitor-1 in vivo in response to adrenaline. Eur J Biochem 97:251-256.
- Freund H & Gendry P (1978) Lactate kinetics after short strenuous exercise in man. Eur J Appl Physiol 39:123-135.
- Gaebelein CJ & Senay LC, Jr (1980) Influence of exercise type, hydration, and heat on palsma volume shifts in men. J Appl Phsyiol 49:119-123.
- Galbo EA, Hilsted RJ, Holst JJ, et al (1977) Hormonal regulation during prolonged exercise. Ann New York Acad Sči 301:72-80.
- Garrow JS (1978) Energy Balance and Obesity in Man (2nd ed). American Elsevier Publishing Company, New York.
- Garrow JS (1974) Energy Balance and Obesity in Man. American Elseview Publishing Company, Inc., New York, pp 39-45.
- Graham TE (1978) Oxygen delivery and blood and muscle lactate changes during muscular activity. Canadian J Appl Sport Sci 3:153-159.
- Green J, Daub G, Painter D, et al (1979) Anaerobic threshold and muscle fiber type, area and oxidative enzyme activity during graded cycling. Med Sci Sports 11:113-114.
- Greenleaf JE & Castle BL (1971) Exercise temperature regulation in man during hypohydration and hyperhydration. J Appl Physiol 30:847-753.
- Grimby G (1965) Renal clearances during prolonged supine exercise at different loads. J Appl Physiol 20:1294-1298.
- Gullbring A, Holmgren A, Sjostrand T, et al (1960) The effect of blood volume variations on the pulse ratio in supine and upright positions and during exercise. Acta Physiol Scand 50:62-71.

- Gurr MI, Mawson R, Rothwell NJ, et al (1980) Effects of manipulating dietary protein and energy intake on energy balance and thermogenesis in the pig. J of Nutr 110:532-542.
- Guyton AC (1981) The relationship of cardiac output and arterial pressure control. Circulation 64:1079-1088.
- Hagenfeldt L & Wahren J (1972) Human forearm muscle metabolism during exercise. VII. FFA uptake and oxidation at different work intensities. Scand J Clin Lab Invest 30:429-436.
- Hart WD (1978) Effects of dietary fats and carbohydrates on the volitional activity in three strains of rats, the mongolian gerbil and the domestic pig. Ph.D. Thesis, Michigan State University.
- Heinbecker P (1931) Further studies on the metabolism of Eskimos. J Biol Chem 93:327-336.
- Hermansen L (1971) Lactate production during exercise. In: Pernow B and Waltin B (eds) Muscle Metabolism During Exercise. Plenum Press, New York, pp 401-407.
- Hill A, Long C & Lupton H (1924) Muscular exercise, lactic acid and supply and utilization of oxygen. Proc Royal Society B 97:84.
- Holloszy J (1976) Adaptations of muscular tissue to training. Progress in Cardiovas Diseases 18:445-458.
- Hultman E & Bergstrom J (1973) Local energy-supplying substrates as limiting factors in different types of leg muscle work in normal man. In: Keul J (ed) Limiting Factors of Physical Performance, Part III. Georgn Thiene, Stuttgart, pp 113-125.
- Hultman E & Nilsson L (1973) Liver glycogen as a glucose-supplying source during exercise. In: Keul J (ed) Limiting Factors of Physical Performance, Part III. Georgn Thiene, Stuttgart, pp 179-187.
- Hurni M, Burnand B, Pittet pH, et al (1982) Metabolic effects of a mixed and a high-carbohydrate low-fat diet in man, measured over 24 h in a respiration chamber. Br J Nutr 47:33-43.
- Ivy JL, Withers T, Van Handel PJ, et al (1980) Muscle respiratory capacity and fiber type as determinants of the lactate threshold. J Appl Physiol 48:523-527.
- Jacobs I (1981) Lactate, muscle glycogen and exercise performance in man. Acta Physiol Scand, Suppl 495:1-35.
- James WPT & Trayhurn P (1981) Thermogenesis and obesity. Br Med Bull 37:43-48.

- Jansky L (1973) Non-shivering thermogenesis and its thermoregulatory significance. Biol Rev 48:85-132.
- Jequier E, Gygax P, Pittet P, et al (1974) Increased thermal body insulation: relationship to the development of obesity. J Appl Physiol 36:674-678.
- Johnson JM & Rowell LB (1975) Forearm skin and muscle vascular responses to prolonged leg exercise in man.
- Johnson JM, Rowell LB & Brengelmann GL (1974) Modification of the skin blood flow-body temperature relationship by upright exercise. J Appl Physiol 37:880-886.
- Johnson RH, Walton JL, Krebs HA, et al (1969) Metabolic fuels during and after severe exercise in athletes and non-athletes. Lancet August 30:452-455.
- Jones NL, Heigenhauser GJF, Kuksis A, et al (1980) Fat metabolism in heavy exercise. Clin Sci 59:469-478.
- Joy RJT (1963) Responses of cold-acclimatized mean to infused norepinephrine. J Appl Physiol 18:1209-1212.
- Keatinge WR (1960) The effects of subcutaneous fat and of previous exposure to cold on the body temperature, peripheral blood flow and metabolic rate of men in cold water. J Physiol 153:166-178.
- Keul J, Doll E & Keppler D (1967) The substrate supply of the human skeletal muscle at rest, during and after work. Experientia 23: 1-6.
- Kindermann W, Simon G & Keul J (1979) The significance of aerobicanaerobic transition for the determination of work load intensities during endurance training. Eur J Appl Physiol 42:25-34.
- Kuroshima A & Yahata T (1979) Thermogenic responses of brown adipocytes to noradrenaline and glucagon in heat-acclimated and cold-acclimated rats. Jap J Physiol 29:683-690.
- Lewis SF, Nylander E&Gad P, et al (1980) Non-autonomic component in bradycardia of endurance trained men at rest and during exercise. Acta Physiol Scand 109:297-305.
- Lundgren NPV (1946) The physiological effects of time schedule work on lumber-workers. Acta Physiol Scand 13:Suppl 41, 30-131.
- Luyckx AS, Pirnay F & Lefebvre PJ (1978) Effect of glucose on plasma glucagon and free fatty acids during prolonged exercise. Eur J Appl Physiol 39:53-61.

- MacDougall J (1978) The anaerobic threshold: Its significance for the endurance athlete. Canadian J Appl Sport Sci 2:137-140.
- Martin B, Robinson S & Robertshaw D (1978) Influence of diet on leg uptake of glucose during heavy exercise. Am J Clin Nutr 31:62-67.
- Maughan RJ & Poole DC (1981) The effects of a glycogen-loading regimen on the capacity to perform anaerobic exercise. Eur J Appl Physiol 46:211-219.
- Maughan RJ, Williams C & Campbell DM, et al (1978) Fat and carbohydrate metabolism during low intensity exercise: Effects of the availability of muscle glycogen. Eur J Appl Physiol 39: 7-16.
- McGrail JC, Bonen A & Belcastro AN (1978) Dependence of lactate removal on muscle metabolism in man. Eur J Appl Physiol 39: 89-97.
- Miller DS & Mumford P (1967) Gluttony. 1. An experimental study of overeating on high and low-protein diets. Am J Clin Nutr 20:1212-1222.
- Miller DS, Mumford P & Stock MJ (1967) Gluttony. 2. Thermogenesis in over-eating man. Am J Clin Nutr 20:1223-1229.
- Miller DS & Payne RP (1962) Weight maintenance and food intake. J Nutr 78:255-262.
- Montoye JH, Willis PW & Cunningham D (1968) Heart rate response to submaximal exercise: Relation to age and sex. J Gerontol 23: 127-133.
- Myhre LG, Oddershede IB & Dill DB, et al (1979) Cardiac output during rest and exercise in desert meat. Med Sci Sports 11: 234-238.
- Nadel ER (1980) Circulatory and thermal regulations during exercise. Federation Proc 39:1491-1497.
- Nadel ER, Cafarelli E & Roberts MJ, et al (1979) Circulatory regulation during exercise in different ambient temperatures. J Appl Physiol 46:430-437.
- Nadel ER, Fortney SM & Wenger CB (1980) Effect of hydration state on circulatory and thermal regulations. J Appl Physiol 49:715-721.
- Newsholme EA (1977) The regulation of intracellular and extracellular fuel supply during sustained exercise. Ann New York Acad Sci 301: 82-91.

-

- Newsholme EA & Crabtree B (1979) Theoretical principles in the approaches to control of metabolic pathways and their application to glycolysis in muscle. J Molecular & Cellular Cardiol 11: 839-856.
- Newsholme EA & Start C (1973) Regulation in Metabolism. John Wiley & Sons, New York, pp 147-185.
- Nicholls DG (1979) Brown adipose tissue mitochondria. Biochim Biophys Acta 549:1-29.
- Nielsen B, Hansen G, Jorgensen SO, et al (1971) Thermoregulation in exercising man during dehydration and hyperhydration with water and saline. Int J Biometerorol 15:195-200.
- Norgan NG & Durnin JVGA (1980) The effect of 6 weeks of overfeeding on the body weight, body composition, and energy metabolism of young men. Am J Clin Nutr 33:978-988.
- Nyberg G (1981) Vagal and sympathetic contributions to the heart rate at rest and during isometric and dynamic exercise in young healthy men. J Cardiovas Pharmacol 3:1243-1250.
- Olsson K-E & Saltin B (1970) Variation in total body water with muscle glycogen changes in man. Acta Physiol Scand 80:11-18.
- Owen OE, Mozzoli MA & Boden G, et al (1980) Substrate, hormone, and temperature responses in males and females to a common breakfast. Metabolism 29:511-523.
- Pace N, Lozner E & Consolazio W, et al (1947) The increase in hypoxia tolerance of normal men accompanying the polycythemia induced by transfusion of erythrocytes. Amer J Physiol 148:152-163.
- Pannier JL, Vrijens J & Van Cauter C (1980) Cardiorespiratory response to treadmill and bicycle exercise in runners. Eur J Appl Physiol 43:243-251.
- Payne PR, Wheeler EF & Salvosa CB (1971) Prediction of daily energy expenditure from average pulse rate. Am J Clin Nutr 24:1164-1170.
- Pirnay F, Lacroix M & Mosora F, et al (1977) Effect of glucose ingestion on energy substrate utilization during prolonged muscular exercise. Eur J Appl Physiol 36:247-254.
- Poortmans JR, Bossche J & Leclercq R (1978) Lactate uptake by inactive forearm during progressive leg exercise. J Appl Physiol 45:835-839.

- Ravussin E, Pahud P & Dorner A, et al (1979) Substrate utilization during prolonged exercise preceded by ingestion of 13C-glucose in glycogen depleted and control subjects. Pflugers Arch 382:197-202.
- Rennie MJ & Holloszy JO (1977) Inhibition of glucose uptake and glycogenolysis by availability of oleate in well-oxygenated perfused skeletal muscle. Biochem J 168:161-170.
- Roberts AD & Morton AR (1978) Total and alactic oxygen debts after submaximal work. Eur J Appl Physiol 38:281-289.
- Roberts MF & Wenger CB (1979) Control of skin circulation during exercise and heat stress. Med Sci Sports 11:36-41.
- `Roberts MF & Wenger CB (1980) Control of skin blood flow during exercise by thermal reflexes and baroreflexes. J Appl Physiol 48:717-723.
- Rothwell NJ & Stock MJ (1979) A role for brown adipose tissue in diet-induced thermogenesis. Nature (Lond) 281:31-35.
- Rothwell NJ & Stock MJ (1981) A role for insulin in the dietinduced thermogenesis of cafeteria-fed rats. Metabolism 30: 673-678.
- Rowell LB, Brengelmann GL & Blackmon JR, et al (1968) Splanchnic blood flow and metabolism in heat-stressed man. J Appl Physiol 24:475-484.
- Saltin B (1964a) Aerobic and anaerobic work capacity after dehydration. J Appl Physiol 19:1114-1118.
- Saltin B (1964b) Circulatory response to submaximal and maximal exercise after thermal dehydration. J Appl Physiol 19:1125-1132.
- Saltin B, Blomqvist G, Mitchell JH, et al (1968) Response to exercise after bed rest and after training: A longitudinal study of adaptive changes in oxygen transport and body composition. Circulation 38:(Suppl 7) 1-55.
- Saltin B, Henriksson J, Nygaard E, et al (1977) Fiber types and metabolic potentials of skeletal muscles in sedentary man and endurance runners. Ann New York Acad Sci 301:3-29.
- Sato M & Tanaka S (1973) Postural effect on the relation between oxygen consumption and heart rate. J Human Ergol 2:21-30.
- Schemmel R (1967) The effect of a high fat ration on body weight, body composition, and adipose tissue weights of rats as influenced by age, strain and weight reduction of obese rats. Ph.D. Thesis, Michigan State University.

- Schutz, Y, Bray GA & Margen S (1981) Effect of a meal on the oxygen consumption-heart rate relationship. Am J Clin Nutr 34:965-966.
- Senay LC, Jr (1970) Effects of exercise in the heat on body fluid distribution. Med Sci Sports 11:42-48.
- Sheffield LT, Maloof JA, Saywer JA, et al (1978) Maximal heart rate and treadmill performance of healthy women in relation to age. Circulation 57:79-84.
- Shetty PS, Jung RT & James WPT (1979) Effect of catecholamine replacement with levodopa on the metabolism response to semi-starvation. Lancet January 13:77-79.
- Skinner JS & McLellan TH (1980) The transition from aerobic to anaerobic metabolism. Research Quarterly for Exercise and Sport 51:234-248.
- Sorenson SG, Ritchie JL, Caldwell JH, et al (1980) Serial exercise radionuclide angiography: Validation of count-derived changes in cardiac output and quantitation of maximal exercise ventricular volume change after nitroglycerin and propranolol in normal men. Circulation 61:600-609.
- Stamford BA, Weltman A, Moffatt R, et al (1981) Exercise recovery above and below anaerobic threshold following maximal work. J Appl Physiol 51:840-844.
- Stock MJ (1980) Effects of fasting and refeeding on the metabolic response to a standard meal in man. Eur J Appl Physiol 43:35-40.
- Studier EH, Dapson RW, & Bigelow RE (1975) Analysis of polynomial functions for determining maximum or minimum conditions in biological systems. Comp Biochem Physiol 52A:19-20.
- Swindells YE (1972) The influence of activity and size of meals on caloric response in women. Brit J Nutr 27:65-71.
- Tanaka M, Volle MA, Brisson GR, et al (1979) Body temperature and heart rate relationships during submaximal bicycle ergometer exercises. Eur J Appl Physiol 42:263-270.
- Taylor HL, Wang Y, Rowell L, et al (1963) The standardization and interpretation of submaximal and maximal tests of working capacity. Pediatrics October, Part II:703-722.
- Trayhurn P & James WPT (1981) Thermogenesis: Dietary and nonshivering aspects. In: Cioffi LA, James WPT, Van Itallie TB (eds) The Body Weight Regulatory System: Normal and Disturbed Mechanisms. Raven Press, New York pp 97-105.

- Trayhurn P. Thurlby PL, Woodward CJH (1979) Thermoregulation in genetically obese rodents: The relationship to metabolic efficiency. In: Festing MFW (ed) Animal Models of Obesity. Macmillan Press, London, pp 191-203.
- Tulp O, Horton ES, Tyzbir ED, et al (1977) Thyroid function in experimental protein malnutrition. Am J Clin Nutr 30:621-625.
- Vokac Z, Bell H, Bautz-Holter E, et al (1975) Oxygen uptake/heart rate relationship in leg and arm exercise, sitting and standing. J Appl Physiol 39:54-59.
- Wahren J (1970) Human forearm muscle metabolism during exercise. IV. Glucose uptake at different work intensities. Scand J Clin Lab Invest 25:129-135.
- Wahren J (1979) Glucose turnover during exercise in healthy man and in patients with diabetes mellitus. Diabetes 28 (Suppl 1):82-88.
- Wahren J, Ahlborg G, Felig P, et al (1971) Glucose metabolism during exercise in man. In: Pernow B & Saltin B (eds) Muscle Metabolism During Exercise. Plenum Press, New York, pp 189-203.
- Welch H. Bonde-Petersen F, Graham T, et al (1977) Effects of hyperoxia on leg blood flow and metabolism during exercise. J Appl Physiol 42:385-390.
- Williams CG, Bredel GAG, Wyndham CH, et al (1962) Circulatory and metabolic reactions to work in heat. J Appl Physiol 17:625-638.
- Wirth A, Neermann G, Eckert W, et al (1979) Metabolic response to heavy physical exercise before and after a 3-month training period. Eur J Appl Physiol 41:51-59.

References Chapter 2 and Chapter 3

- Andrews R (1971). Net heart rate as a substitute for respiratory calorimetary. Am J Clin Nutr 24:1139-1147.
- Astrand I (1971). Estimating the energy expenditure of housekeeping activities. Am J Clin Nutr 24:1471-1475.
- Astrand P, Guddy T, Saltin B, Stenberg J (1964). Cardiac output during submaximal and maximal work. J Appl Phsyiol 19:268-274.
- Astrand PO, Rodahl K (1977). Textbook of Work Physiology (2nd ed) McGraw-Hill Book Company, New York, pp 455-459.
- Bradfield R (1971). A technique for determination of usual daily energy expenditure in the field. Am J Clin Nutr 24:1148-1154.
- Bradfield R, Chan H, Bradfield N, Payne P (1971). Energy expenditures and heart rates of Cambridge boys at school. Am J Clin Nutr 24: 1461-1466.
- Bradfield R, Huntzicker P, Fruehan G (1970). Errors of group regressions for prediction of individual energy expenditure. Am J Clin Nutr 23:1015-1016.
- Consolazio CF, Johnson RE & Pecora LJ (1963). Physiological Measurements of Metabolic Functions in Man. McGraw-Hill Book Company, New York.
- Costill D, Fink W (1974). Plasma volume changes following exercise and thermal dehydration. J Appl. Physiol 37:521-525.
- Damato A, Galante J, Smith W (1966). Hemodynamic response to treadmill exercise in normal subjects. J Appl Physiol 21:959-966.
- Dauncey M, James W (1979) Assessment of the heart-rate method for determining energy expenditure in man, using a whole-body calorimeter. Br J Nutr 42:1-13.
- Dill D, Consolazio C (1962). Responses to exercise as related to age and environmental temperature. J Appl Physiol 17:645-648.

- Durnin JVGA (1978). Indirect calorimetry in man: A critique of practical problems. Proc Nutr Soc 37:5-12.
- Ekelund L (1967). Circulatory and respiratory adaptation during prolonged exercise of moderate intensity in the sitting position. Acta Physiol Scand 69:327-340.
- Ekelund L, Holmgren A (1964). Circulatory and respiratory adaptation during long-term, non-steady state, exercise in the sitting position. Acta Physiol Scand 62:240-255.
- Gill JL (1978) Design and Analysis of Experiments in the Animal and Medical Sciences, Volume 1. The Iowa State University Press, Ames, Iowa, pp 108 and 193.
- Hagberg J, Mullin J, Nagle F (1980). Effect of work intensity and duration on recovery O₂. J Appl Physiol 48:540-544.
- Hirsch J, Bishop B (1981). Respiratory sinus arrhythmia in humans: how breathing pattern modulates heart rate. Am J Physiol 241: H620-H629.
- Londeree B, Ames S (1976). Trend analysis of the % V_{O2} max-HR regression. Med Sci Sports 8:122-125.
- Lundgren N (1946). The physiological effects of time schedule work on lumber-workers. Acta Physiol Scand 13:(Suppl 41)30-131.
- Miller RB, Jr (1966). Simultaneous Statistical Inference. McGraw-Hill Book Company, New York.
- Montoye H, Willis P, Cunningham D (1968) Heart rate response to submaximal exercise: Relation to age and sex. J Gerontol 23: 127-133.
- Myhre L, Oddershede I, Dill D, Yousef M (1979). Cardiac output during rest and exercise in desert heat. Med Sci Sports 11: 234-238.
- Nadel E, Fortney S, Wenger C (1980). Effect of hydration state on circulatory and thermal regulations. J Appl Physiol 49:715-721.
- Nandi P, Spodick D (1977). Recovery from exercise at varying work loads. Br Heart J 39:958-966.
- New Weight Standards for Men and Women. Statistical Bulletin 40, 1 (Nov.-Dec.) 1959. Metropolitan Life Insurance Company, New York.
- Pannier J, Vrijens J, Van Cauter C (1980). Cardiorespiratory response to treadmill and bicycle exercise in runners. Eur J Appl Physiol 43:243-251.

- Payne P, Wheeler E, Salvosa C (1971). Prediction of daily energy expenditure from average pulse rate. Am J Clin Nutr 24:1164-1170.
- Roberts M, Wenger C (1980). Control of skin blood flow during exercise by thermal reflexes and baroreflexes. J Appl Physiol 48:717-723.
- Rowell L, Marx H, Bruce R, Conn R, Kusumi F (1966). Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. J Clin Invest 45: 1801-1816.
- Saltin B (1964a) Aerobic and anaerobic work capacity after dehydration. J Appl Physiol 19:1114-1118.
- Saltin B (1964b) Circulatory response to submaximal and maximal exercise after thermal dehydration. J Appl Physiol 19:1125-1132.
- Saltin B, Blomqvist G, Mitchell J, Johnson R, Wildenthal K, Chapman C (1968). Response to exercise after bed rest and after training. Circulation 38:(Suppl 7)VII-1-VII-55.
- Saltin B, Stenberg J (1964). Circulatory response to prolonged severe exercise. J Appl Physiol 19:833-838.
- Sato M, Tanaka S (1973) Postural effect on the relation between oxygen consumption and heart rate. J Human Ergol 2:21-30.
- Schutz Y, Bray G, Margen S (1981). Effect of a meal on the oxygen consumption-heart rate relationship. Am J Clin Nutr 34:965-966 (letter).
- Senay L (1979) Effects of exercise in the heat on body fluid distribution. Med Sci Sports 11:42-48.
- Sheffield L, Maloof J, Sawyer J, Roitman D (1978) Maximal heart rate and treadmill performance of healthy women in relation to age. Circulation 57:79-84.
- Sonne B, Galbo H (1980). Simultaneous determinations of metabolic and hormonal responses, heart rate, temperature and oxygen uptake in running rats. Acta Physiol Scand 109:201-209.
- Steel RGD and Torrie JH' (1960). Principles and Procedures of Statistics. McGraw-Hill Book Company, New York, pp 188-193.
- Tanaka M, Volle M, Brisson G, Dion M (1979). Body temperature and heart rate relationships during submaximal bicycle ergometer exercises. Eur J Appl Physiol 42:263-270.
- Taylor H, Wang Y, Rowell L, Blomqvist G (1963). The standardization and interpretation of submaximal and maximal tests of working capacity. Pediatrics 32:(Suppl)703-722.

REFERENCES CHAPTER 4 AND 5

- Acheson, K.J., Zahorska-Markiewicz, B. Anantharaman, K. & Jequier, E. (1980). Caffeine and coffee: their influence on metabolic rate and substrate utilization in normal weight and obese individuals. Am. J. Clin. Nutr. 33:989-997.
- Ahlborg, G. & Felig, P. (1977). Substrate utilization during prolonged exercise preceded by ingestion of glucose. Am. J. Physiol. 233, E188-E194.
- Ahlborg, G., Felig, P., Hagenfeldt, L., Hendler, R. & Wahren, J. (1974). Substrate turnover during prolonged exercise in man: splanchnic and leg metabolism of glucose, free fatty acids and amino acids. J. Clin. Invest. 53, 1080-1090.
- Apfelbaum, M., Bostsarron, J. & Lacatis, D. (1971). Effect of caloric restriction and excessive caloric intake on energy expenditure. Am. J. Clin. Nutr. 24, 1405-1409.
- Astrand, P.O., Rodahl, K. (1977). Textbook of Work Physiology (2nd ed). McGraw-Hill Book Company, New York, pp. 455-459.
- Astrand, P. (1967). Diet and athletic performance. Fed. Proc. 26, 1772-1777.
- Bray, G.A., Whipp, B.J. & Koyal, S.N. The acute effects of food intake on energy expenditure during cycle ergometry. Am. J. Clin. Nutr. 27, 254-259.
- Bergstrom, J. & Hultman, E. (1972). Nutrition for maximal sports performance. JAMA 221, 999-1006.
- Bergstrom, J., Hultman, E.; Jorfeldt, L., Pernow, B. & Wahren, J. (1969). Effect of nicotinic acid on physical working capacity and on metabolism of muscle glycogen in man. J. Appl. Physiol. 26, 170-176.
- Bonen, A., Malcolm, S.A., Kilgour, R.D., MacIntyre, K.P. & Belcastro, A.N. (1981). Glucose ingestion before and during intense exercise. J. Appl. Physiol. 50, 766-771.
- Brooke, J.D. (1981). Lowered blood glucose with human fasting, exercise and carbohydrate diet. Nutr. Rep. Int. 24, 1279-1285.

Bylund-Fellenius, A-C, Walker, P.M., Elander, A., Holm, S., Holm, J. & Schersten, T. (1981). Energy metabolism in relation to oxygen

partial pressure in human skeletal muscle during exercise. Biochem. J. 200, 247-255.

- Cannon, B., Nedergaard, J., Romert, L., Sundin, U. & Svartengren, J. (1978). In Strategies in Cold: Natural Torpidity and Thermogenesis, p. 567 (L.C.H. Wang and J.W. Hudson, editors). New York: Academic Press.
- Cannon, 1978. Book The biochemical mechanism of thermogenesis in brown adipose tissue. 567-594.
- Consolazio, C.F., Johnson, R.E. & Pecora, L.J. (1963). Physiological Measurements of Metabolic Function in Man. McGraw-Hill Book Company, New York.
- Costill, D.L., Coyle, E., Dalsky, G., Evans, W., Fink, W. & Hoopes, D. (1977). Effects of elevated plasma FFA and insulin on muscle glycogen usage during exercise. J. Appl. Physiol. 43, 695-699.
- Crandall, E.A. & Fernstrom, J.D. (1980). Acute changes in brain tryptophan and serotonin after carbohydrate or protein ingestion by diabetic rats. Diabetes 29, 460-466.
- Crapo, P.A., Reaven, G.M. & Olefsky, J.M. (1981). Hormonal and substrate responses to a standard meal in normal and hypertriglyceridemic subjects. Metabolism 30, 331-334.
- Danforth, E., Jr., Horton, E.S., O'Connell, M., Sims, E.A.H., Burger, A.G., Ingbar, S.H., Braverman, L. & Gavenakis, A.G. (1979). Dietary-induced alterations in thyroid hormone metabolism during overnutrition. J. Clin. Invest. 64, 1336-1347.
- Dauncey, M.J. (1979). Energy Metabolism in man and the influence of diet and temperature: a review. J. Human Nutr. 33, 259-269.
- Dauncey, M.J. (1980). Metabolic effects of altering the 24 h energy intake in man, using direct and indirect calorimetry. Br. J. Nutr. 43, 257-269.
- Dauncey, M.J. & Ingram, D.L. (1979). Effect of dietary composition and cold exposure on non-shivering thermogenesis in young pigs and its alteration by the β -blocker propranolol. Br. J. Nutr. 41, 361-367.
- Davidson, M.B. & Chopra, I.J. (1979). Effect of carbohydrate and noncarbohydrate sources of calories on plasma 3,5,3'-Triiodothyronine concentrations in man. J. Clin. Endocr. & Metab. 48, 577-581.

- Dunnett, C.W. (1955). A multiple comparison procedure for comparing several treatments with a control. J. Am. Stat. Assoc. 50:1096-1121.
- Dunnett, C.W. (1964). New tables for multiple comparisons with a control. Biometrics 20, 482-491.
- Durnin, J.V.G.A. (1978). Indirect calorimetry in man: A critique of practical problems. Proc. Nutr. Soc. 37:5-12.
- Durnin, J.V.G.A. & Namyslowski, L. (1958). Individual variations in the energy expenditure of standardized activities. J. Physiol. 143, 573-578.
- Elkin, R.G., Featherston, W.R. & Rogler, J.C. (1980). Effects of dietary phenylalanine and tyrosine on circulating thyroid hormone levels and growth in the chick. J. Nutr. 110, 130-138.
- Essen, B. (1978). Studies on the regulation of metabolism in human skeletal muscle using intermittent exercise as an experimental model. Acta Physiol. Scand., Supplement 454, 1-64.
- Foster, C., Costill, D.L. & Fink. W.J. (1979). Effect of preexercise feedings on endurance performance. Med. Sci. Sports 11, 1-5.
- Garby, L. & Lammert, O. (1977). Effect of the preceding day's energy intake on the total energy cost of light exercise. Acta Physiol. Scand. 101, 411-417.
- Garrow, J.S. & Hawes, S.F. (1972). The role of amino acid oxidation in causing 'specific dynamic action' in man. Br. J. Nutr. 27, 211-219.
- Gambino, S.R. (1961). Collection of capillary blood for simultaneous determinations of arterial pH, CO₂ content, pCO₂, and oxygen saturation. Amer. J. Clin. Path. 35, 175.
- Gill, J.L. (1978). Design and Analysis of Experiments in the Animal and Medical Sciences, Volume 1. The Iowa State University Press, Ames, Iowa, pp. 108 and 193.
- Glick, Z., Teague, R.J. & Bray, G.A. (1981). Brown adipose tissue: Thermic response increased by a single low protein, high carbohydrate meal. Science 231, 1125-1127.
- Glick, Z., Shvartz, E., Magazanik, A. & Modan, M. (1977). Absence of increased thermogenesis during short-term overfeeding in normal and overweight women. Am. J. Clin. Nutr. 30, 1026-1035.
- Graham, T.E. (1978). Oxygen delivery and blood and muscle lactate changes during muscular activity. Canadian J. of Appl. Sport Sciences 3, 153-159.

- Gurr, M.I., Mawson, R., Rothwell, N.J. & Stock, M.J. (1980). Effects of manipulating dietary protein and energy intake on energy balance and thermogenesis in the pig. J. Nutr. 110, 532-542.
- Heldmaier, G. (1971). Relationship between non-shivering thermogenesis and body size. In Nonshivering Thermogenesis, p. 73 (L. Jansky, editor). Amsterdam: Swets & Zeitlinger.
- Hagenfeldt, L. & Wahren, J. (1972). Human forearm muscle metabolism during exercise. VII. FFA uptake and oxidation at different work intensities. Scand. J. Clin. Lab. Invest. 30, 429-436.
- Hurni, M., Burnand, B., Pittet, Ph. (1980). Effects of a mixed and of a very low-fat diet on substrate utilization in man, measured in a calorimetric chamber. Internat. J. Vit. Nutr. & Res. 50, 206-207.
- Ivy, J.L., Withers, R.T., Van Handel, P.J., Elger, D.H. & Costill, D.L. (1980). Muscle respiratory capacity and fiber type as determinants of the lactate threshold. J. Appl. Physiol. 48, 523-527.
- Jacobs, I. (1981). Lactate, muscle glycogen and exercise performance in man. Acta Physiol. Scand, Suppl. 495, 1-35.
- Johnson, R.H., Walton, J.L., Krebs, H.A. & Williamson, D.H. (1969). Metabolic fuels during and after severe exercise in athletes and non-athletes. The Lancet, August 30, 452-455.
- Jones, N.L., Heigenhauser, G.J.F., Kuksis, A., Matsos, C.G., Sutton, J.R. & Toews, C.J. (1980). Fat metabolism in heavy exercise. Clin. Sci. 59, 469-478.
- Joy, R.J.T. (1963). Responses of cold-acclimatized men to infused norepinephrine. J. Appl. Physiol. 18, 1209-1212.
- Jung, R.C., Balchum, O.J. & Massey, F.J. (1966). The accuracy of venous and capillary blood for the prediction of arterial pH, Pco₂ and Po₂ measurement. Amer. J. Clin. Path. 45, 129.
- Jung, R.T., Shetty, P.S. & James, W.P.T. (1979). The effects of beta adrenergic blockade on basal metabolism and peripheral thyroid metabolism.
- Karlsson, J. & Saltin, B. (1971). Diet, muscle glycogen, and endurance performance. J. Appl. Physiol. 31, 203-206.
- Katch, V., Weltman, A., Sady, S. & Freedson, P. (1978). Validity of the relative percent concept for equating training intensity. Eur. J. Appl. Physiol. 39, 219-227.

- Kindermann, W., Simon, G. & Keul, J. (1979). The significance of the aerobic-anaerobic transition for the determination of work load intensities during endurance training. Eur. J. Appl. Physiol. 42, 25-34.
- Landsberg, L. & Young, J.B. (1978). Fasting, feeding and regulation of the sympathetic nervous system. N. Eng. J. Med. 298, 1295-1301.
- Lusk, G. (1928). The Elements of the Science of Nutrition, 4th ed. Philadelphia & London: W.B. Saunders.
- Luyckx, A.S., Pirnay, F. & Lefebvre, P.J. (1978). Effect of glucose on plasma glucagon and free fatty acids during prolonged exercise. Eur. J. Appl. Physiol. 39, 53-61.
- Martin, B., Robinson, S. & Robertshaw, D. (1978). Influence of diet on leg uptake of glucose during heavy exercise. Am. J. Clin. Nutr. 31, 62-67.
- Maughan, R.J. & Poole, D.C. (1981). The effects of a glycogen-loading regimen on the capacity to perform anaerobic exercise. Eur. J. Appl. Physiol. 46, 211-219.
- Maughan, R.J., Williams, C., Campbell, D.M. & Hepburn, D. (1978). Fat and carbohydrate metabolism during low intensity exercise: Effects of the availability of muscle glycogen. Eur. J. Appl. Physiol. 39, 7-16.
- McGrail, J.C., Bonen, A. & Belcastro, A.N. (1978). Dependence of lactate removal on muscle metabolism in man. Eur. J. Appl. Physiol. 39, 89-97.

Miller, R.G., Jr. (1966). Simultaneous Statistical Inference.

- Miller, D.S., Mumford, P. & Stock, M.J. (1967). Gluttony, 2. Thermogenesis in overeating man. Am. J. Clin. Nutr. 20, 1223-1229.
- New Weight Standards for Men and Women. (1959). Statistical Bulletin 40, 1 (Nov.-Dec.). Metropolitan Life Insurance Company, New York.
- Norgan, N.G. & Durnin, J.V.G.A. (1980). The effect of 6 weeks of overfeeding on the body weight, body composition, and energy metabolism of young men. Am. J. Clin. Nutr. 33, 978-988.
- Norris, B., Schade, D.S. & Eaton, R.P. (1978). Effects of altered free fatty acid mobilization on the metabolic response to exercise. J. Clin. Endocr. & Metab. 46, 254.

- Owen, O.E., Mozolli, M.A., Boden, G., Patel, M.S., Reichard, G.A., Jr., Trapp, V., Shuman, C.R. & Felig, P. (1980). Substrate, hormone, and temperature responses in males and females to a common breakfast. Metab. 29, 511-523.
- Pirnay, F., Lacroix, M., Mosora, F., Luyckx, A. & Lefebvre, P. (1977). Effect of glucose ingestion on energy substrate utilization during prolonged muscular exercise. Eur. J. Appl. Physiol. 36, 247-254.
- Pirnay, F., Lacroix, M., Mosora, F., Luyckx, A. & Lefebvre, P. (1977). Glucose oxidation during prolonged exercise evaluated with naturally labeled (¹³C) glucose. J. Appl. Physiol. 43, 258-261.
- Poortmans, J.R., Bossche, J. D-V., & Laclercq, R. (1978). Lactate uptake by inactive forearm during progressive leg exercise. J. Appl. Physiol. 45, 835-839.
- Ravussin, E., Pahud, P., Dorner, A., Arnaud, M.J. & Jequier, E. (1979). Substrate utilization during prolonged exercise preceded by ingestion of ¹³C-Glucose in glycogen depleted and control subjects. Pflugers Arch. 382, 197-202.
- Rothwell, N.J. & Stock, M.J. (1979). A role for brown adipose tissue in diet-induced thermogenesis. Nature 281, 31-35.
- Rothwell, N.J. & Stock, M.J. (1981). A role for insulin in the diet-induced thermogenesis of cafeteria-fed rats. Metab. 30, 673-678.
- Rubner, M. (1902). Die Gesetze des Energieverbrauchs bei der Ernahrung. Leipzig: Deutiche.
- Scheibel, M.S., Coon, C.N. & Kelley, K.W. (1979). The heat increment of feeds used in poultry diets. Nutr. Reports Internat. 20, 871-881.
- Sestoft, L. (1980). Metabolic aspects of the calorigenic effect of thyroid hormone in mammals. Clin. Endocr. 13, 489-506.
- Seyffert, W.A., Jr. & Madison, L.L. (1967). Physiologic effects of metabolic fuels on carbohydrate metabolism. I. Acute effect of elevation of plasma free fatty acids on hepatic glucose output, peripheral glucose utilization, serum insulin, and plasma glucagon levels. Diabetes 16, 765-776.
- Shetty, P.S., Jung, R.T., James, W.P.T., Barrand, M.A. & Callingham, B.A. Postprandial thermogenesis in obesity. Clin. Sci. 60, 519-525.

- Sigma Tech. Bulletin No. 726-UV/826-UV. (1974). Saint Louis, Missouri: Sigma Chemical Company.
- Skinner, J.S. & McLellan, T.H. (1980). The transition from aerobic to anaerobic metabolism. Research Quarterly for Exercise and Sport 51, 234-248.
- Sonne, B. & Galbo, H. (1980). Simultaneous determinations of metabolic and hormonal responses, heart rate, temperature and oxygen uptake in running rats. Acta Physiol. Scand. 109, 201-209.
- Spangler, E. & Johnson, D.E. (1981). Influence of feeding pattern on energy balance and activity in rats. J. Nutr. 111, 1297-1304.
- Stamford, B.A., Weltman, A., Moffatt, R. & Sady, S. (1981). Exercise recovery above and below anaerobic threshold following maximal work. J. Appl. Physiol. 51, 840-844.
- Stankiewicz-Choroszucha, B. & Gorski, J. (1978). Effect of decreased availability of substrates on intramuscular triglyceride utilization during exercise. Eur. J. Appl. Physiol. 40, 27-35.
- Steel, R.G.D. and Torrie, J.H. (1960). Principles and Procedures of Statistics. McGraw-Hill Book Company, New York.
- Stock, M.J. (1980). Effects of fasting and refeeding on the metabolic response to a standard meal in man. Eur. J. Appl. Physiol. 43, 35-40.
- Sutton, J.R., Jones, N.L. & Toews, C.J. (1981). Effect of pH on muscle glycolysis during exercise. Clin. Sci. 61, 331-338.
- Swindells, Y.E. (1972). The influence of activity and size of meals on caloric response in women. Br. J. Nutr. 27, 65-71.
- Tukey, J.W. (1953). "The problem of multiple comparisons". Mimeographed. Princeton University, Math Department, Princeton, NJ.
- Tulp, O., Horton, E.S., Tyzbir, E.E., Danforth, E., Drupp, P.P. & Bollinger, J. (1977). Thyroid function in experimental protein mal-nutrition. Am. J. Clin. Nutr. 30, 621-625.
- Wahren, J. (1979). Glucose turnover during exercise in healthy man and in patients with diabetes mellitus. Diabetes 28, 82-88.
- Wahren, J., Felig, P., Ahlborg, G. & Jorfeldt, L. (1971). Glucose metabolism during leg exercise in man. J. Clin. Invest. 50, 2715-2725.
- Withers, R.T., Sherman, W.M., Miller, J.M. & Costill, D.L. (1981). Specificity of the anaerobic threshold in endurance trained cyclists and runners. Eur. J. Appl. Physiol. 47, 93-104.

Tukey JW (1953). "The problem of multiple comparisons". Mimeographed. Princeton University, Math Department, Princeton, NJ.

- Vokzc Z, Bell H, Bautz-Holter E, Rodahl K (1975). Oxygen uptake/ heart rate relationship in leg and arm exercise, sitting and standing. J Appl Physiol 39:54-59.
- Williams C, Bredell G, Wyndham C, Strydom N, Morrison J, Peter J, Fleming P, Ward J (1962). Circulatory and metabolic reactions to work in heat. J Appl Physiol 17:625-638.

APPENDICES

Appendix A

Time = duration of expired air collection (sec)

R.Q. = Respiratory Quotient

V02 = Oxygen consumption (ml/min/kg)

HR = Heart rate

Resting = Collection of expired air samples in the resting state prior to exercise

Exercise = Collection of expired air during the incremental
exercise test

Recovery = Collection of expired air during recovery following the exercise test

APENDIX A. HEART RATE AND DAYGEN CONSUMPTION FOR REGRESSION ANALYSES

1	I	-1	5 53	***222255555555555555555555555555555555	222538		_	.	822	1828888888688858	
7	5-06-80	늰			200000		0-12-00	N.N.		2822284228888222	
EXP. 5	1	. õ			2.4 4.4 9.8 9.8 9.8	EXP. 10	Ĩ			201280828668866 20128088868866 20128088888886	
_	24 C-29	5	222	*****************	288282		05.8 kg		222	38 %%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%%	
SUBJECT ()			000			UBJECT A	ij		000		
Ś	10191	Ĩ	888	<u>5555555555555555555555555555555555555</u>	328888	THOS	te i gi	-	888	<u>888888</u> 888888888888888888888888888888	
4	8	H.H.	823	****	<u> </u>		15-00	N.N.	888	zzz:2523523222	
EXP. 4 (4-03-80	匆		1-9N9-9999999775N99		1	10-15	~		~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~	
- 52		.¥		200001000000000000000000000000000000000	2 	3	5	.ĝ	•••	******	
5	85.5	8.0	598		900000 886888	5	85.		000 288	00000000000000000000000000000000000000	
SUBJECT	te lah t:	Ĩ	888	<u>8888888888888888888888888888888888888</u>	****	Sueucc	He laht:	j	888	*****	
•	_							- .			,
i	I	-i	~ ~ a al	DOBBOUGHERNANDERNNU			1	-1			
7	27-80	н.я	388	88888 <u>882222222223</u> 888883333	2528888	=	04-90	딬	882	33829 888558533222222222	2288228
E J	12-2	20	229	25522222222222222222222222222222222222	22.5 9.1 4.0	E.P 6	Î	2		22222222222222222222222222222222222222	
·	84.1 kg	ö	222	***********************	x 8 x 2 3 8		9.6 kg	đ	222	「おねのりたいたちなのためののないない」	822222
1 13		æi	600			1 13	ht: 04		•••		
SUBJECT	He I ght.	1	888	<u></u>	<u>8282288</u>	SUBJECT		-	888	£82222222222222222222222	22222222
ł	!	×	z z z	888255555555555555555555555555555555555	~~*****	1	1	æİ	2 22	●ちょうちのお~○~~~	
177	2-19-60	πļ			220000	1 1	17-80	±1		28252525252698	28822
ExP.		. Z	444		22.9 6.9 4.7 3.5	EXP. 1	+	201			90 4 m
÷	84.) kg	8.0	222		2288222		85.4 kg	ä	828	202568238383558388 	25232
UBJECT #	at:		666			SUBJECT #	:	ei	000		
Sub	10 1	II.	888	<u>8885555555555555555555555555555555555</u>	*****	ž	5	H	888	<u>8888888888888888888888888888888888888</u>	383855
4	9-80	H. F.	222	**5%5%5%5%2%2%%5%6%5%			8	4. H	8 8 8	88 88 1112 1112 1112 1112 1112 1112 111	5228888E
	21	~	~~~	***			7-01-80	~			
3	2	201	***	9228858555555555555555		- 66	t a	- õ			*=****
	84.1	R. U.	0.00	00000000000000000000000000000000000000		1 1	85.1		822	00.28 00.28 00.08 00.08 00.08 00.08 00.08 00.08 00.08 00.08 00.08 00.08 00.08 00.07 00	8.00 8.00 8.00 8.00 8.00 8.00 8.00 8.00
SUBJECT / 1	te laht:		888	6 888888888888888888888888888888888888		SUBJECT # 1	He Ight:	Ĩ	888	<u>2888888888888888888888888888888888888</u>	
σi		- :	a a ai			. 31	∄ i	=1	ㅋ 주 주]		~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~
			RESILNG	EAE RCISE	RECOVERY				RESTING	Ext. AC 15E	RECOVERY
			З¥	× u	RE				2	ă	REC

APPENDIX A, continued - H.R. and VOZ for Regression Analyses

:

. -

SUBJECT # 2 - E2P. 5 () Helght: 01.5 10 0-17-00	E E.Q. 102 H	300 0.73 4.3 56 300 0.86 3.2 55 300 0.59 4.9 55		6 0.0 15.2 173 182 0.0 15.4 111 182 0.0 15.4 111 182 0.0 15.4 111 200 0.05 5.5 97 200 0.55 5.7 97 200 0.55 5.7 97 200 0.55 5.7 97 200 0.55 5.7 97	SUBJECT # 2 - 620-10 ()	Maight: 80.5 kg 9-25-80	1 8.9. YO2 H	300 0.72 3.6 67 300 0.72 4.3 64 300 0.70 3.7 63		6 0.9 2.7 1.5 8 0.9 2.7 1.5 8 1.17 2.7 1.5 100 0.5 6.0 5.1 100 0.5 5.1 1.8 100 0.5 5.1 1.8 100 0.5 5.1 5.8 100 0.5 5.1 5.8 100 0.5 5.1 5.8 101 5.1 5.8 5.1 5.8 101 5.1 5.8 5.1 5.8 5.8 101 5.1 5.8 5.1 5.8
2 - EXP. 4 () 80.9 kg 8-)4-80	.X02	*** *** ***		88 88 88 40. 112 86 60 86 60 86 80 88 88 88	2 - 59, 9 ()	00.4 kg 9-10-60	100	8 3.6 3.5 3.5 2.4 2.6 2.4 2.6 2.6 2.6 2.6 2.6 2.6 2.6 2.6 2.6 2.6		86 28.5 23 14.4 23 14.4 26 6.9 26 5.5 5.0 5.0 5.0 5.0 82 5.0 82 5.0 82 5.0 82 5.0 82 82 83 85 85 85 85 85 85 85 85 85 85 85 85 85
SUBJECT &	-		66666666666666666666666666666666666666	999999888 888888888	SUBJECT &	1019hE: 0	-		66666666666666666666666666666666666666	88888888
2 - EXP. 3 () 80.5 kg 7-31-80	<u>102</u>	8 () () () () () () () () () () () () () (2 - [10 -]	60.4 kg 9-01-60	. A		2 2 2 2 2 2 2 2 2 2 2 2 2 2	196 26.1 153 197 10.2 114 188 5.9 198 188 5.3 94 188 4.2 88 173 4.2 88
SUBJECT A	8	888	66666666666666666666666666666666666666	888888888 8888888888888888888888888888	/ Li Brens	Helght:		888	<u>EEEEEEEEEEEEEEEEEEEEEEEEEEEEEEEEEEEEE</u>	\$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$ \$
<u>Eue, 2 ()</u>		4.1 3.4 58 3.5	112 8 112 8	22 25 25 25 25 25 25 25 25 25 25 25 25 2	<u> [] [] []</u>	9-28-60	V02 N.R.		111. 111.	30.2 10.3 5.1 5.1 100 5.1 100 4.2 2.2 2 2 2 2
SUBJECT # 2 -	Time B.Q.	300 0.82 300 0.72 300 0.72		50 0.23 300 0.23 300 0.23 300 0.23 300 0.23 300 0.55 0.55 0.55	SINUECT 0 2 -	Me1ght: 00.3 k	[1me 8.9.	285 0.74 300 0.69 300 0.76		6666666
EXP. 1 () te 7-17-80	. õ	4.3 65 4.1 68 4.2 64	SALTA SALA SALA SALA SALA SALA SALA SALA	13.8 162 9.1 123 9.1 123 9.4 6 99 4.6 89 8.4 88	EXP. 6 ()	9 8-21-80	V02 N.R.	4.0 1.7 2.2 2.2 2.2 2.2 2.2 2.2 2.2 2.2 2.2 2	**************************************	
SUBJECT / 2 -	<u>8.0.</u>	87.0 000 001 001 001		60 0.92 120 1.02 100 0.61 100 0.61 100 0.61 100 0.61	SUBLICT / 2 -	Me1ght: 80.9 k	11m B.Q.	300 0.69 330 0.61 270 0.62		6666666
		RESTING	E RE RC 15E	RE COVERY				RESTING	Exercise	RE COVE NY

APPENDIX As continued - H.R. and VO2 for Regression Analyses

1	8	a a	335	838588888835835835835	<u>7</u> 28822	-	_	И. Я.	282	#6#= <u>52755233</u> 5 <u>8</u> 522222	1
[KP. 5]	ŝ	201	3.0	22.22.22.22.23.23.23.23.23.23.23.23.23.2	200955	E. 19	1-22-01	20	00-	12222222222222222222222222222222222222	
3 . 6	70.2 kg		228	75% #5 # 8 # 6 8 6 6 8 8 8 8 8 8 8 6 8 6 8 8 8 8		-	69.4 hg		285	88055585585585888888 8805558558588888888	
SIBUEL L	HIGHL: 7		888	66666666666666666 <u>88888</u> 88888888888		SUBJECT &	ight:		888	66666666666666666666666666666666666666	
3:	<u>.</u>	21	888		******	1 3	-	21	888		١
٦		И.В.	332	8828991275629255558888 199129129292555588888 1991391291292925555888888	<u>252388</u>		_	H. N.	\$ 35	88888888888888888888888888888888888888	
	5-27-80			**************************************	4.5 4.2 4.2 4.2	1	19-22-1	. 2		1211 1212 1212 1212 1212 1212 1212 121	
9X3 - EXP	24.5 kg		58 2	222-20000000000000000000000000000000000	86222	. 6	69.4 kg		222	?2C2888388852228888888 88 0xxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxx	
SUBJECT / J	Height: 7		660	*****************	555555 5 <u>5</u> 88888	SUBJECT /	Metght: 6	il B	000		
3	3		888	<u>8888888888888888888888888888888888888</u>	******	1 3	봐	Ē	<u>888</u>	<u>8888888888888888888888888888888888888</u>	I
٦		н. н.	333	111122 1112 11112 11112 11112 11112 111111	251 26 8 8 8 8	1 1	1	N.N.	33 5		208682Z
CN C .433	5-06-80	207			20244) 0 'di	9-11-8	8	× 4 9	117192222882 88748222882 1141946612711948 1141946612711948	
3 - 51	2.1 19		292	<u>2</u>	~		-		28 5 62	***************************************	8822835
BUECT 1	ات		000		*****	SUBJECT &	Helght: 69	ei i	000		
85	He i gi		888	<u>8855888888888888888888888888888888888</u>	920002		-		<u>888</u>	<u>ଽଽଽଽଽ</u> ଽଌଌଌଌଌଌୡୡୡୡୡୡୡ	82 <u>28888</u>
	1	-1						.1			,
217	4.73-80	H, R	388	388005585885856985 3888005585885855985	55888C	2	8-06-80	A.H	385	8858051245626265577788 88580512456262655577888	201 202 203 203 203 203 203 203 203 203 203
- [1] - [1]	2	<u>702</u>	0.6 1.6	L.E.E.E.E.E.E.E.E.E.E.E.E.E.E.E.E.E.E.E	2.5	EXP. 7	19 19	. <u>2</u>	3.5		29.5 10.1 1.7 9.4
-	. 13.4	<u> </u>	0.0 0.05 0.72	99999999999999999999999999999999999999	0.00 10.00 10.00 10.00 10.00 10.00 10.00 10.00		. 69.2	<u>, 0</u>	0.65 0.65 0.68		
133rans	10 I OF	Ĩ	888	<u>~~</u> ~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~	\$28888	SUBJECT	19	I	888	<u>8888888888888888888888888888888888888</u>	355 853
7	22-80	Н. Я.	***	88.88 801 801 801 801 801 801 801 801 801 8		7	08-61	A.A.	833	88888888888888888888888888888888888888	2598262 2
EXP. 1	kg 4.	. 20M	3.7 3.5			ExP. 6	kg 6-	. 20			20 20 20 20 20 20 20 20 20 20 20 20 20 2
	1.4		0.9 8.0 M.0				20.1 4	R. Q.	0.05	88888888888888888888888888888888888888	0000000 88828888
SUBJECT A 3	He lant:	-	888	<u> </u>		SubJECT / 3	He laht:	Ĩ	222	<u><u>888888888888888888888888888888888888</u></u>	8 2 8 9 8 8 8
			RFSTING	EXE PLISE	RE (. OVE.RT				RESTING	EXENCISE	RE FOVE RY
			RFS.	E XEI	HE CC				RES1	EXER	RECC

APFROIX A. continued - H.R. and VO2 for Regression Analyses

.

1	I	انے	اموم	~~~~~~~~~~~~~~~~	مععمومما	I	I	_ i	maal		
4	1-22-00	2. 2	***	***555525553555555555555555555555555555	10889882 10	1.	08-52	Н. И.	***	282662217775222222222222222222222222222222	28882223
EXP. 5 (-	20		51125252525555555555555555555555555555	2	EXP. 10		ğ		28.22.2 28.22.2 28.22.2 28.23.2 28.23.2 28.23.2 28.23.2 28.23.2 28.23.2 28.23.2 28.23.2 28.23.2 28.23.2 28.23.2 29.23.2 20.23.	22.8 2.9 2.9 2.9 2.9 2.9 2.9 2.9 2.9 2.9 2.9
	8.6		2.22	00.00 00	88778878 88778878	• • •	99	8.Q.	8.9 8.9 9.6	60000000000000000000000000000000000000	0.50 0.50 0.50 0.50 0.50 0.50
SUBJECT	He I GH C:		888	<u>86555555588888888888888888888888888888</u>	28888888	SUBJECT	He Ight:	Ĩ	¥200	<u>8888888888888888888888888888888888888</u>	82282828
Ч	8	1919	83 X	888088888899 8 800888	58×2×88	4	19-60	И. В.	***	888939922225 445 36338822	22888825
(xP. 4	1-15-80	8		22288888888888888888 8-9-1111-99911998	500000	EXP. 9	4	. <u>8</u>			81189 S S 4
•	<u>82.5 hg</u>	.	RZE	0000208202020202	*******	•	23.5 44	<u></u>	***	£xxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxxx	80,083,83
UBJECT /	ight:	-	888	88222233333333333333333333333333333333	82288888 82288888	SUBJECT A	in a the		9000	66666666666666666666666666666666666666	\$228588
34	31	=	***		-226555	3	3	=I	***	22222	
I	I	 1	333		~*****	1	1	ei	822	8888882 <u>8878444335</u> 8999885	\$6 888233
	8-99-	4.1		###588525533228888859533	200000	1	8-12-80	A.H		668660210144555533355	-
Ω₽, 1	1	.¥		22222222222222222222222222222222222222	28.2 8.6 8.4 8.4 8.4 8.4 8.4 8.4 8.4 8.4 8.4 8.4	E. 9.	kg 8.	204		1995 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5	2.01
	8.3	R.Q.	000 222		2800000 28222886		6.9	R.Q.	0.02	66000000000000000000000000000000000000	8282583 8282583
SUBJECT	Helght:		888	<u>8688888888888888888888888888888888888</u>	8 <u>888888</u>	SUBJECT	te ight:		888	<u><u><u>8</u>22222</u>33333333333333333333333333333</u>	2000 000 000
I	1	=	338	***************************************	8888 58 2 2 2 8 8 8 8	1	1	æj	225	2222222222222222222222	85.055.88%
7	-05-90	=				1	09-90-0	si			00~~~~~
	t a	. 707		222228888668689585888	82	EXP.	5	202			00.444mm
	9.8	R.Q.	0.82 0.69 0.70		8.0 8.0 8.0 8.0 8.0 8.0 8.0 8.0 8.0 8.0		92.1	B.Q.	8.00 8.99 99	6	880000000
SUBJECT	He lot		888	<u><u><u>8</u>888888888888888888888888888888888</u></u>	33888888	SUBJECT	1614		888	<u>8888888888888888888888888888888888888</u>	50 300 300 300 300 300 300 300 300 300 3
	æ	Н. Я.	888	##28882225 <u>5</u> 8432588888222	21288×62		8	R.H.	382	22222222222222222222222222222222222222	82 88 88 88 88 88 88 88 88 88 88 88 88 8
619.1	6-24-	200	5.9 3.8 3.9	¥¥¥\$\$\$\$\$\$\$\$\$ 6.644.449.03333335 6.644.449.03333335 6.644.449.03333 6.644.449.0333 6.644.449.034 6.644.449.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.034 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.0440 6.644.449.04400 6.644.440000000000000000000000000	5.5.6.9.8.4 9.5.6.9.9.4 9.5.7.5.9	EXP. 6	1-29-	. 20V	4.2 4.2 4.2	1911 1911 1911 1911 1911 1911 1911 191	8 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9
	95.4 14	<u>R.Q.</u>	74 69	\$G~\$C%C%CC#8&88888	5288833	•	92.4 kg	허	71 20 68	25222222222222222 25222222222222222222	*******
SUBJECT 0			600			SUBJECT / 4	::	er i	000		
3	1013	1	888	<u> </u>	8 3 8 8 8 8 8 8 9 8 9 8 9 8 9 8 9 8 9 9 9 9	ŝ	le t gh		888	<u>8888888888888888888888888888888888888</u>	888 <u>866996</u>
			RE STING	CKE ACLISE	RE CUVE RY				RESTING	EVERCISE	RELUVERY
			2 3 4	323	R.C.				MES	30	ä

APPLHOLX A, continued - H.R. and VO2 for Regression Analyses

	1 -1	_ 1			, ,	,	.,			1	
() ;	N H	222	282928555555555555555555555555555555555			6-13-80	Н. Я.	225	222 86868 686888888888888888888888888888	<u>22</u> 2032:	822
2	. <u>S</u>	222		23.7 2.5 5.1 3.4	EXP. 10	Ξ	Ň	22.2			
	h .	0.70 18.0 0.80	89.00 27.00 27.00 28.00 29.00 29.00 20.000	*85%?		00.7 kg	N. 0.	222	57777888557785887858 8778788855558585858		
SIGUECT					SUBJECT #	te faht:		666			
리네	1 21	888	<u></u>	85888	3	뵑	Ē	888 8	<u>2222228</u> 2333333333333333333333333333333	83 8 <u>288</u> 3	122
1 1		# \$\$	RRR 888675755555555555555555555555555555555	5228 22	2	98-90-	N. N	222	22.22.888.22.23.2388.2.23 12.22.888.22.23 12.22.888.82.23 12.22.888.82.23 12.22.888.82.23 12.22.888.82.23 12.22.888.82.23 12.22.888.82.23 12.22.888.82.23 12.23.83.83 12.23.83.83 12.23.83.83.23 12.23.83.83.23 12.23.83.83.23 12.23.83.23.23.23 12.23.23.23.23.23.23.23.23.23.23.23.23.23	32 3223	322
1.4	. 2	352		8.99.99- 8.99.99-	1.9	× 6-0	201	223	111.0 11.0 11.0	32.7 28.7 29.9	:::
		252		2882582		81.0 kg	R. Q.	888			
SUBJECT /					SUBLECT /	Helght: 1		000			
8	1 2	888	855888888888888888888888888888888888888	3 <u>88888</u>	1 3	1	-	888	<u>RR22222</u> 8888888888888888888888888888888	88 8 <u>228</u> 8	288
		223	REC 88888227779996	2222	2	8	N.A.	223	80 80 10 12 12 12 12 12 12 12 12 12 12 12 12 12	5857 <u>5</u>	33
5.93				33.86	EXP. 0	5-30-80	8	3.5	1121 1212 1212 1212 1212 1212 1212 121	8.0.4	
			~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~	***	- u	. ) kg		~ 0 @		EREE	
ECT / 5		000		0000	CT 15	10 : 1	R.Q.	0.0	8.000000000000000000000000000000000000	0000	
SUBJECT		888	855333333333333333	<u>8888</u>	SUBJECT	He lat		888	<u> 222222</u> 33333333333333	\$ <u>\$ 8 8 8 8</u>	38
		222	22.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2	20384			Н. Р.	330	88888889888998888888888888888888888888	55 <b>7</b> 885	= sal
( ) - 2 - 1 - 1 M-11-M					1,	-23-60	ΞÌ			22 2003	
2	. ĝ				Exe.	Ś	. <u>2</u>			44 88 89 89 89 89 89 89 89 89 89 89 89 89	
5	t .	600 888	0.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.0000 8.00000 8.0000 8.0000 8.0000 8.0000 8.00000 8.0000 8.0000 8.00000 8.00000 8.00000 8.00000 8.00000 8.00000 8.00000000			61.2	<u>8. 0.</u>	0.2	00000000000000000000000000000000000000	88 8873	0.63
SUBJECT		888	<u>8888888888888888888888888888888888888</u>	88888	SUBJECT	1991	1	888	<u> 888888888888888888888888888888888888</u>		
		~~~~			' <b>3</b> 1	51	FI.	***			5 X XI
;	ائہ ا	اممد				,	4			1	
	a a	423	84228868 <u>82289</u> 82998888		2	9-90	2, H	\$ \$	28288825555744623888	<u>72</u> 58221	53
1.1	. X		E 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2		EXP. 6	5	V05		12222222222222222222222222222222222222	22.2	
- 51		228	2000-000-000-000-000-000-000-000-000-00		· ·	<u>81.9 kg</u>		22	8.8.8.8.8.8.8.8.8.8.8.8.8.8.8.8.8.8.8.	38 89×21	
SUBJECT / 5	i				SUBJECT & S	Velght: 01.		0 0			
3	II 21	885	<u>8888888888888888888888888888888888888</u>		1 30	-	-	응의	<u>~~~</u> ~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~	88 85883	28 28
		RESTING	Life AC I SE	RE COVERY				RESTING	EXERCISE	RE COVE RY	
		RES	ž	2				RES	EXEL	HE CL	

APPENDIX A, continued - H.R. and VO2 for Regression Analyses

		N. N.	382	888***		۲		<u>к, к</u>	***	BBBBBBBBBBBBBBBBBBBBBBBBBBBBBBBBBBBBBB	<u>7</u>
(N. S. ()	7-21-80		0~0	~~~~~~	-	1 01	0-22-00	. 1	~~~		000
- (%)	H	<u> </u>		**********	****	. EU.	3	. 02		ヹヹヹ ヹ ゟヹゟ゙゙゙゙゙゙ゟゟ゚゚ゟ゚ゟ゚゚ゟ゚ゟ゚ゟ゚ゟ゚゚゚゚゚゚゚゚゚゚゚゚゚	215~ 4 vine
*	: 90.7	R. Q.	8.			971	: 00.6	9.0 1	222		29756
SUBJECT	1614		888	<u>8222822222222222222222222222222222222</u>	\$3 <u>88888</u> 8	SUBJECT	te lott	11 mil	888	<u>8888888888888888888888888888888888888</u>	<u>8888888</u> 8
4	8	N. N.	332	**=***	20×882%	4	8	<u>H.R.</u>	52.S	RZCXXX88822833833833	3008888
EXP. 4	7-15-80	2		2222562222255222552 2222552225522552 2225522522		• •	8-18-	ğ		52245522222222555555 575455522225555555555	2.6 8.2 8.5 8.5 8.5 8.5 8.5 8.5 8.5 8.5 8.5 8.5
-	89.0 kg		F\$2	32822222222888828282828282	282232	•	.2 49	ہم. ا	238		8857338
UBUECT &		đ	600			SUBJECT &	PL: 90	-		•••••••	
5	5	1	888	<u>8888888888888888888888888888888888888</u>	82288888	SU	5	-	888	<u>8888888888888888888888888888888888888</u>	<u>82288888</u>
,	,	•1			. 1			.4			1
4	7-08-60	<u>е</u> , н	222	888282555555558885	<u></u>	4	11-80	H .		88885555555555 5 555555353	2229888
E.YP. 3	-7 - P	.X	1		2.4444 2.4444	EXP. B		. Q			
	90.04	8.0	0.22	00000000000000000000000000000000000000	0.00 2.20 2.20 2.20 2.20 2.20	• • • •	90.6	8 .0	555 555		0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.0
SUBJECT	te laht:	I	888	<u> </u>	\$ <u>22222</u>	SUBJECT	telght:		888	<u></u>	\$ <u>8888888</u>
ı	1	e il	- nel		1282232	I	I	e.	835	888866192222566795688882222	22288688
E.P. 2 . 9.1	6-30-80	zi.	222		-	11	8-04-90	I.			
	9 . 9	<u>705</u>	9.5 1.0 1.0	0-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1	6.9 5.2 6.4 6.4 6 7.4 6	EXP.	1	20	904 6		2000444
. 91	69.0	8.0 .	69.0 69.0	8888888777 88888888777	5000000 2888888		1.16		828		88728999 88728
SUBJECT	ite laht.	1	888	<u>8888888888888888888888888888888888888</u>	****	SUBJECT	He I ght	Ĩ	888	822222223333322333333333333333333333333	: 22222228
4	09- Ľ		333		2 2888888		8	N H	***	88888 88885555555555555555555555555555	5 528822
	2	20	~ 00		2.5 5.5 1.5 1.5 1.5 1.5 1.5 1.5 1.5 1.5 1	EXP. 6 _	1-28-	204	222		8~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~
- 50	89.7 kg		222	82%228868888888888888888888888888888888	8528323	•	9 kg		222		2828228
SUBJECT / 6	t	# .				SUBJECT 0 6	Metght: 89.	ei l	000		
7805	He lant:	뢾	888	<u>8888888888888888888888888888888888888</u>	88 <u>88888</u>	SUBL	릚	Ĩ	888	<u>8555555555555555555555555555555555555</u>	288 <u>88888</u>
			RESTING	16HCISE	RECOVERY				RESTING	EZENCISE	RE COVE RV
			Я.	11	2				3	a a a a a a a a a a a a a a a a a a a	Ħ

APPENDIX A, continued - N.R. and VUZ for Regression Analyses

SUBARCT # 7 - EXP. 5 () Maight: 95.6 kg 7-24-80		000 0.% 3.6 3.7 20 000 0.69 3.7 3.6 01 0.69 3.6	88888888888888888888888888888888888888	60 0.74 36.1 130 180 0.280 38.1 130 180 0.280 130 100 200 0.27 5.3 10 200 0.27 5.3 50 200 0.77 4.5 56 200 0.77 4.5 56 200 0.77 4.5 56 200 0.77 4.5 56 200 0.77 4.5 56 200 0.77 4.5 56 200 0.77 4.5 56	SUBJECT / 7 - EXP. 10 ()	He (ght: 100.2 kg 8-27-80	Time E.O. 1982 315 0.00 106 118 200 0.00 118 22 200 0.00 118 23 218 25 218 br>218 218 218 218 218 218 218 218 218	22222222222222222222222222222222222222	60 0.87 24.9 128 178 0.85 5.5 72 200 0.75 4.7 65 200 0.76 4.2 50 200 0.64 4.2 50 200 0.64 4.8 65
<u>910-15 (7 - 510, 4 ()</u> 1010-00-00-00-00-00-00-00-00-00-00-00-00	5.9. YO2	100 0.71 2.9 13 100 0.69 1.4 12 100 0.66 1.1 13	88888888888888888888888888888888888888	88888888888888888888888888888888888888	suarci () - (20- 9 ()	Meight: 99.8 kg 8-20-80	Time B.Q. W2 M.B. 300 0.73 3.5 3.5 30 300 0.73 3.5 3.5 30	20000000000000000000000000000000000000	
SUBJECT & 7 - EXP. 3 () Metamic: 100.8 kg 7-09-80	9:9. YOZ H	000 0.79 2.9 28 000 0.74 1.1 2.9 28 01 0.71 1.5		6 0.3 %.9 128 0.27 %.9 128 0.27 %.9 128 0.27 %.9 168 5.1 168 5	suaject (? - Exp. 8 ()	Helght: 29.2 kg 8-14-80	Time 8.9. 192		St 0.0 0.0 2.1 <th2.1< th=""> <th2.1< th=""> <th2.1< th=""></th2.1<></th2.1<></th2.1<>
sueuscr # 7 - Ez#- 2 () Melent: 101.4 kg 7-02-00		00 0.6 0.6 0.00 00 0.1 0.0 000 00 0.1 0.0 000		60 0.73 15.4 104 128 0.74 6.0 52 200 0.75 2.9 60 200 0.76 2.9 60 200 0.74 3.14 5.0 52 200 0.74 3.14 55	SUBJECT (? - EXP. ? ()	Meight: 101.8 kg 8-06-80	1444 14.0. 192 14.1. 200 0.69 3.0 20 200 0.74 3.0 20		60 0.73 22.5 118 120 0.43 6.5 79 120 0.43 6.5 79 200 0.36 4.5 73 200 0.36 4.5 71 200 0.31 1.6 41 200 0.73 3.6 41 200 0.73 3.6 41
<u>546.45C1 4 7 - E29. 1 ()</u> Margant: 101.3 kg 6-26-80		10 C.E 77.0 00C 2011230 21 C.E 200 0.0C 2011230 21 C.E 200 00C	A A A A A A A A A A A A A A	60 0.91 22.9 134 0.0 1.00 1.12 73 0.0 0.38 6.7 72 0.0 0.38 6.7 72 0.0 0.38 6.7 72 0.0 0.73 4.1 60 0.0 0.74 3.6 60 0.0 0.74 3.6 60 0.0 0.74 3.6 60	Subject of 7 - Exp. 6 ()	Meight: 101.2 kg 7-31-80	Line A.Q. YO2 M.A. RESTING 300 0.68 3.0 27 RESTING 300 0.71 2.9 27		0.78 24.4 0.65 7.9 0.76 5.0 0.78 5.0 0.78 1.6 0.72 1.6

204

APPENDIX A, continued - N.R. and VOZ for Regression Analyses

4	09-52-00	н. п.	283	zzasti 10222033393625	<u> 722</u> 5288	4	08-60-	<u>H.R.</u>	222	8621111222888855388	2 2282888
EXP. 5	kg 10-2	. 2 2			2000.044 V02004/	61. IO	12-0	. 20	252	22.1.1.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.	28-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1-1
CT 1 0 -	1: 63.2	R.Q.	000 222	00000000000000000000000000000000000000	000000000 28882 28882 290000000000000000		C: 6.9 1	R.Q.	000 2×2	86000000000000000000000000000000000000	
SUBJECT	191		888 8	<u>&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&&</u>	33 23228	SUBJECT	No 1 oh	Ĩ	888	<u>8888888888888888888888888888888888888</u>	33 38 <u>8</u> 888888
4	8	Н. И.	688	***555555555555555555555555555555555555	3 282288	٦	8	<u>H. R.</u>	***	5868512777753886889222	2 222288
EXP. 4	10-24-80	. 2	<u>-</u> :::	11111111111111111111111111111111111111	2000 9000 9000 9000 9000 9000 9000 9000	EXP. 9 (09-52-11	<u>. 102</u>	32.3	11111111111111111111111111111111111111	
	63.7 kg	<u>9.9</u>		**************************************	0.0988 0.7388 0.7388 0.7380 0.7380 0.7380 0.7380 0.7380 0.7380 0.7380 0.73800 0.73800 0.73800000000000000000000000000000000000	. 81	63.7 kg	8.9 .	9. 74 0.81 0.62		
SUBJECT	He I gh C		88 8	<u>8888888888888888888888888888888888888</u>	23 <u>88888</u>	SUBJECT	Ne ight:		888	<u> 888888888888888888888888888888888888</u>	3335555
!	9	<u>H. R.</u>	222	***======================	3828858	1	_	н.я.	222	20 & & E SI 20 20 20 20 20 20 20 20 20 20 20 20 20	5 5555588
() (.43	10-21-80	20 20		2.151 2.1512	55	.	11-20-80		222		-
	62.3 kg	N.9.	222		866666666 86622288	1 8 - EXP.	64.3 kg		22.2		88.85.288
SIBLECT	te lah C:		888	<u>2222222</u> 229999999999999999999999999999	****	SUBJECT	Ne ight:	1	888		
1	10-11-00	N.R.	***	***************************************	2823588 5	117	-11-90	н. Н	222	8889111119158888	22 10 12 28 2 20 12 12 28 2
- Exe. 2	kg 10	. 2	323	7278588667996665599 8499668866666666999 84996670000799999	80.000 80.0000 80.0000 80.0000 80.0000 80.0000 80.0000 80.0000 80.00000 80.00000 80.00000 80.0000 80.00000 80.00000 80.00000 80.00000000	Exe	11 61	105	222		
ECT / 8	ht: 63.1		522 000			. 1	t: 62.0		9.00 9.02		
SUBJECT	int july	Ĩ	888	<u>8888888888888888888888888888888888888</u>	22 <u>2222</u> 2222	SUBJECT		8	888	<u>8888888888888888888888888888888888888</u>	33 38888888888888888888888888888888888
4	80	н. н.	z R 8	99822288828888888888888888888888888888	<u>8552888</u> 9	-	8	R.H.	~38	% 88 11 11 11 11 11 11 11 11 11 11 11 11 	88 8 8 8 1 1 1 1 1 1 1 1 1 1 1 1 1 1 1
ExP. 1 (9-28-		9 9 9 9 9 9 9 9 9	1111 1111 1111 1111 1111 1111 1111 1111 1111	10.2 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5 5	EXP. 6 (11-00			4441.000001.001.10000000000000000000000	
	: 62.9 kg		389		8788685	•	63.0 kg		8.8.8		16 0 16 0 16 0 16 0 16 0 16 0 16 0 16 0
SUBJECT	He Laht:	-	888	<u>8888888888888888888888888888888888888</u>	23 <u>55555</u>	SUBJECT / B			888	<u>222222</u> 33338833883388	8 8855338
			REST ING	L XC RC 15E	RE COVE RY				RESTING	CXENCISE	RECUVERY

APPENDIX A, continued - H.R. and W2 for Regression Analyses

ł	_	.	ଌଌୠ	***	******	1	1	_ i	امعما		
1	10-21-50	a. H		***::::::::::::::::::::::::::::::::::::	¥238888	1	12-02-50	N.R	823	**83355595555552553222	5552888
E. 5	10	20X	:::	702288888888888888888888888888888888888	28. 6.7 7.9 7.9 7.9 7.9 7 8 7 8 7 8 7 8 7 8 7 8 7 8 7 8 7 8 7	EXP. 10	21	07	9 8 9 9 8 9 9 9	2221 2222 2222 2222 2222 2222 2222 222	2 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
	55.9		9 7 C		×6888222		50.1 1	R. Q.	2.0 22 22 22 22	999999222228282828282828282828282828282	0.88 0.88 0.73 86.99 0.73 86.99 86.90 86.0
SUBJECT	10100	-	888 8	<u></u>	33 22222	SUBJECT	He loht:	a l	888	<u>ୡୖଽଽଽ</u> ଽଽଽଌଌୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡ	88888888 88888888888888888888888888888
4	8	i.i.	*22	****	<u></u>	Ч	8	<u>N. N.</u>	***	***222222222222222222222222222222222222	538882 55
EXP. 4 ()	10-14-00	200		22179099-19979819999 22222222222222222222222222222222	8.0 2.5 9.8 9.8 9.8 9.8 9.8 9.8 9.8 9.8 9.8 9.8	6.4	11-24-00	201	:::	8888558865586558658 89322288658655865 89585586586558655858 8958558655865585858 8958558655865585858 8957585858655865585858 8957585858655858585858 8957585858585858585858585858585858585858	2.5 5.6 5.5 5.6 5.5 5.5 5.5 5.5 5.5 5.5 5
	56.6 19		85.5	******************	88885555		57.3 kg	.	225	\$2255222222222222888	8888882
SUBJECT	le laht:	~	888	~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~		UBJECT /	te let t		888	66666666666666666666666666666666666666	
স	31	=	***			3	#	티	888	<u></u>	22222222
1	1	-1	1			1		-1		,	1
4	10-12-60	<u>я.н</u>	882	***************************************	<u>25</u> 58858	4	00-11-11	N. N.	323	882021128098882388099 <u>8</u> 8	5588822
E.P. 1 ()	0	201			90055555	EXP. 8		. ğ		22228888668868446589 90949-40764868446589	4 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9 9
- 11	58.1 k		388		8288888 8288888		57.0 k	8.0.	000 888	00000000000000000000000000000000000000	87888558
SUBJECT	He laht:		888	<u>8555555</u> 3833333333333333333333333333333	\$\$ 8888888	SUBJECT	Heleht:	I	888	<u>ଽଽଽଽଽଽ</u> ଽଽଽଽଽଽଽଽ	88 <u>88888</u>
1	ł	انہ	مسما					-1			
4	10-0/-00	н.	282	***:==::::====:::::::::::::::::::::::::	2222882	5	8-8-	H.H	583	5422225266999999999999999999999999999999	22888822
EXP. 2 []	0	ğ	:::	19222222222222222222222222222222222222	*21 	EXP. 7	-11 -	. 201	4 4 4 0 1 1 6	8511 8512 8512 8512 8512 8512 8512 8512	82.5 8.5 9.5 9.5 9.5 9.5 9.5 9.5 9.5 9.5 9.5 9
	56.7		\$.00	89999999999999999999999999999999999999	00000000000000000000000000000000000000	5	57.1 k	1.	232	00000000000000000000000000000000000000	5.00 1.00 1.00 1.00 1.00 1.00 1.00 1.00
SUBJECT	He loht		8 8 8	ଌୢଌୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡୡ	x \$ 800000	SUBJECT	Melght:	1	8 88	<u>ଽଽଽଽଽଽ</u> ଽଽୢଌଌୡୡୡୡୡୡୡୡୡୡ	838 88888
4	8	H.R.	232	32.2.2.2.8.5.5.8.5.5.8.5.8.5.5.5.5.5.5.5.	897516 5	4	23-60	н. к.	823	8288558888994 9 5588888	5588822
EXP. 1	-06-6	202	8 N -	822388255588828588888888888888888888888	8.13. 9.7. 5.8 7.0 7.0 7.0	EXP. 6 (10-23	M02	044	885-982 888-99 1995	22.8 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5 2.5
!	56.6 kg	8 O.	282	*****	2222223	•	5.2 kg		69 65		66688933 6668893
SUBJECT / 9	Height 56	e]	666	6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6 6		SUBJECT 0 9	de lant: 55.	æ		00000000000000000000000000000000000000	600000 0
3		-	888	<u>8888888888888888888888888888888888888</u>	****	SUBJ	1	1	888	<u>222222</u> 3333333333333333333333333333333	833 88883
			RE ST ING	EXENCISE	RE COVE RY				UNG	EXERCISE	RE COVERY
			RES	E XET					RE STING	EXE	NE CU

APPCNUIX A, continued - N.R. and VO2 for Regression Analyses

SUBJECT # 10 - EXP. 5 ()	Meight: 76.7 kg 10-30-90	P. 0. 102 H	300 0.76 3.7 60 300 0.70 3.8 56 300 0.69 3.8 56		6 0.1 15. 111 118 0.7 15. 111 100 0.7 6.1 10 100 0.7 6.1 10 100 0.6 111 119 8 119 8	suesect # 10 - Exe. 10 ()	Meight: 78.1 kg 11-25-80	E 1.0. VQ	000 0.66 3.7 58 000 0.66 4.0 56 0.09 3.8 59		- 1 - 02 - 201 - 1 - 10 - 10 - 10 - 10 - 10 - 10 - 1
SUBUECT # 10 - EXP. 4 ()	te (ght: 77.2 ts 10-23-80	E <u>8.0.</u> 102 H	000 0.72 0.9 56 000 0.71 0.7 56 000 0.71 0.6 57	885722222222222222222222222222222222222	60 0.20 21.8 127 60 0.20 21.4 127 60 0.26 11.4 56 000 0.37 4.2 56 000 0.37 4.2 56 000 0.37 4.2 78 000 0.37 4.2 78 000 0.37 3.2 78 000 0.37 3.2 78	sueject # 10 - Exp. 9 ()	Meight: 78.8 kg 11-21-80	E E.Q. VQ2 H	300 0.76 4.5 70 300 0.77 4.0 66 300 0.76 3.9 63	XXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXXX	0.088 333.3 0.099 533 34 0.078 4.5 0.776 4.6 0.776 4.0 0.76 4.0
suevect / 10 - EXP. 3 ()	Height: 76.2 kg 10-16-00	E.Q. VO2 H	000 0.66 1.8 59 000 0.71 1.7 59 000 0.71 3.3 57	88888888888888888888888888888888888888	66 0.66 23.3 188 0.08 10.6 100 0.76 4.7 100 0.66 4.7 100 0.66 4.7 100 0.66 4.1 111 100 100 0.66 4.1 101 4.1 63 102 4.1 63 103 0.75 4.1 104 4.1 63 107 4.1 63 108 4.1 63 109 0.75 4.1 100 0.66 4.1 101 4.1 63 101 4.1 63 102 4.1 63 103 4.1 63 104 4.1 63 105 4.1 63 105 4.1 63 104 4.1 63	sueusci / 10 - Exp. 6 ()	Meight: 77.9 kg 11-20-80	t 1.9. <u>102</u> H	300 0.69 3.8 65 300 0.71 4.9 65 300 0.69 4.1 65		0.3 0.3 0.3 0.3 0.3 0.3 0.3 0.3
SUBJECT # 10 - EXP. 2 ()	Meight: 77.7 kg 10-09-80	e <u>B.Q.</u> YO2 H	300 0.72 4.0 54 300 0.73 3.5 54 300 0.77 3.7 56		60 0.69 34.1 137 86 0.73 9.2 107 80 0.73 9.2 107 800 0.73 9.2 107 800 0.66 4.4 86 800 0.66 4.1 76 800 0.66 4.1 76 800 0.61 1.3 76	SUBJECT # 10 - EXP. 7 ()	Height: 77.5 11-14-60	E R.Q. YOZ H	000 0.72 4.9 56 000 0.77 4.2 56 000 0.70 4.1 54		0.08 0.28 0.57 0.57 0.56 5.2 0.66 5.2 0.66 5.2 5.2 5.2 5.2 5.2 5.2 5.2 5.2
EXP. 1	Meight: 78.0 kg 10-02-80	E R.Q. W2	000 0.73 3.4 57 315 0.62 3.8 55 285 0.74 3.2 58			suguect # 10 - Exp. 6 ()	Weight: 78.2 kg 11-06-80	R.Q. W02 H	300 0.65 4.1 50 300 0.65 4.4 51	88603982261999663333 -9	60 0.83 40.3 60 0.78 40.3 60 0.77 6.0 199 1.15 4.4 100 0.77 6.0 101 1.15 4.4 100 0.77 6.0 101 0.77 6.0 101 1.15 4.4 1000 0.17 4.4
			RE STING	EKERCISE	RECOVERY				RESING	EXERCISE	ALE LOVE RY

207