THE EFFECTS OF EXERCISE INTENSITY AND DURATION ON EXCESS POST-EXERCISE OXYGEN CONSUMPTION

By

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A Thesis
Submitted to the School of Graduate Studies
in Partial Fulfillment of the Requirements
for the Degree
Master of Science

McMaster University

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July, 1990

MASTER OF SCIENCE (1990) McMASTER UNIVERSITY

(Human Biodynamics) Hamilton, Ont

TITLE: The Effects of Exercise Intensity and Duration on Excess Post-Exercise Oxygen Consumption

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NUMBER OF PAGES: xiii and 140

ABSTRACT

The effects of exercise intensity and duration on excess post-exercise oxygen consumption (EPOC) were examined. Eight males exercised in a thermoneutral environment at 60% of maximal aerobic power (VO_{2max}) for 30 min and performed the same total work at 80% and 40% VO_{2max} by varying exercise durations. In addition, 2 work bouts were performed at 60% VO_{2max} for 90 and 60 min. A standardized meal was consumed 2 h post-exercise. Oxygen consumption (VO_2), respiratory exchange ratio (RER), rectal temperature (Tc) and heart rate (HR) were monitored on a control day and before, during and for 3 h following exercise. VO_2 was equivalent to control day values within 30 min post-exercise on each of the 5 d and no consistent relation between exercise condition and duration of EPOC was observed. When total post-exercise VO_2 was expressed relative to control values, differences were greatest during the first 30 min post-exercise. Total net caloric expenditure was small (32.5-57.9 kcal) in all cases. RER tended to remain equivalent to control levels post-exercise, with intermittent elevations. Significant elevations in Tc were obtained until 60 to 150 min post-exercise. No significant exercise-related effects were noted for dietary induced thermogenesis or the

cumulative effect of the 5 exercise bouts on resting metabolic rate.

Although exercise over a wide range of intensities and durations resulted in a significant EPOC, in all cases it was of short duration and the total 3 h energy expenditure was small. Neither duration nor magnitude of EPOC was associated with post-exercise RER or Tc.

ACKNOWLEDGEMENTS

I would first like to thank Dr. Digby Sale for agreeing to supervise my research, and for the many hours of invaluable guidance and help that he gave me. I am indebted to him for his kindness and his optimism, which enabled me to believe that completing this thesis was within the realm of human possibility. I would also like to express my appreciation to Dr. MacDougall for being instrumental in the development of the research topic, and for his involvement, advice and encouragement. Dr. Audrey Hicks merits special mention; her concise editorial comments led to the most major revisions in the thesis.

I would like to thank Jennifer Dent for being a constant source of support and inspiration. And Joan Martin has always been more than willing to share the benefits of her experiences with me, and was instrumental in my weathering out a few storms.

The data collection phase of this thesis had several highlights. First, it gave me the opportunity to discover that there are students on this campus who will endure the most unpleasant procedures, and will still come out smiling. I am grateful to them for their participation. Second, I had

the unexpected pleasure to work with Tali Bar-Or, who brought an overwhelming energy and vitality to even the most monotonous tasks.

Finally, I am deeply indebted to John Moroz, who spent almost as much time in the physiology lab during the summer of 1989 as I did. This study could never have been completed without his technical expertise, his patience and many, many hours of his time.

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LIST OF ABBREVIATIONS

CP	creatine phosphate
EOC	exercise oxygen consumption (total volume of oxygen consumed during exercise)
EPOC	excess post-exercise oxygen consumption
h	hour(s)
HR	heart rate (beats/min^{-1})
L	litre(s)
min	minute(s)
NS	nonsignificant
RER	respiratory exchange ratio
RMR	resting metabolic rate
Tc	core temperature (refers to rectal temperature in the present study)
TEF	thermal effect of feeding
μL	microlitre(s) (litre x 10^{-6})
VE	expiratory gas volume/min^{-1} (expressed in litres gas/min^{-1})
$\dot{V}O_2$	volume of oxygen consumed/min^{-1}*
VO_{2max}	maximal aerobic capacity
y	year(s)

*Dot has not been placed above VO_2 in text; all references to VO_2 imply uptake of oxygen 'per minute'.

1 INTRODUCTION

Exercise-induced energy expenditure affects total caloric balance and is of potential benefit in the prevention and treatment of obesity (ACSM, 1986). In recent years, there has been extensive documentation that elevations in oxygen consumption (VO_2) persist for a period of time following exercise, but a controversy exists in the literature with respect to the significance of the contribution of excess post-exercise oxygen consumption (EPOC) toward total exercise-induced energy expenditure. There are reports that EPOC lasts 4.5 to 12 h, and is of large total magnitude (approximately 150 kcal) (Bahr et al., 1987; Bielinski et al., 1985; Chad & Wenger, 1988; Maehlum et al., 1986). Investigators of these studies have contended that EPOC is an important component of the total caloric expenditure caused by exercise.

In contrast, the literature also includes a number of studies which have found EPOC to be of brief duration and/or small total magnitude (Brehm & Gutin, 1986; Deuster et al., 1989; Elliot et al., 1988; Freedman-Akabas, et al., 1984; Kaminsky et al., 1987; Knuttgen, 1970; Pacy et al., 1985; Sedlock et al., 1989; Shah et al., 1988). Although a significant EPOC has been documented in all cases,

investigators included in the latter group of studies have concluded that the contribution of EPOC towards the total energy cost of the exercise is not sufficient to promote weight loss.

Subject populations, exercise conditions and designs are similar among the studies cited, and do not appear to account for the differences among findings. Findings of a large EPOC may be attributable to the use of pre-determined recovery periods (Bahr et al., 1987; Bielinski et al., 1985; Maehlum et al., 1986), or may be the result of diurnal increases in VO_2 which were not accounted for by the inclusion of a control day (Chad & Wenger, 1988).

Differences among findings of the duration and magnitude of EPOC are clearly not merely a function of the caloric expenditure during the work-bout, since the relation of the oxygen consumed during exercise (EOC) to the magnitude of EPOC is rarely comparable among reports. When EPOC is expressed as a percentage of EOC, values range from 2% (Sedlock et al., 1989) to 28% (Chad & Wenger, 1988). The duration of EPOC following exercise of similar durations and intensities has also varied considerably, lasting between 10 min (Kaminsky et al., 1987) and 12 h (Bahr et al., 1987; Maehlum et al., 1986), while net magnitudes of EPOC reported range from 3 kcal (Frechette et al., 1987) to 180 kcal (Chad & Wenger, 1988).

The influence of specific exercise conditions on the duration and magnitude of EPOC is not known. The magnitude and duration of post-exercise VO_2 has increased with elevations in exercise intensity (Brehm & Gutin, 1986; Gore & Withers, 1990; Sedlock et al., 1989), increased with exercise duration when exercise intensity was reduced (Chad & Wenger, 1985), and the duration of EPOC has been equivalent (magnitudes not reported) following high and low intensity exercise when total work was kept constant (Kaminsky et al., 1987). The relation between the magnitude of EPOC and exercise duration has also not been established. It may be necessary to attain a threshold exercise intensity before duration affects total EPOC (Gore & Withers, 1990).

The extent to which EPOC is altered by changes in exercise intensity and duration may vary with the length and size of the total EPOC reported. Chad and Wenger (1985), who reported a large total EPOC, documented a 60% increase in magnitude of EPOC when exercise intensity was reduced from 70% to 50% of maximal aerobic capacity (VO_{2max}). Conversely, investigators who reported an EPOC of brief duration and small total magnitude found that increases in exercise intensity had no effect on EPOC (Kaminsky et al., 1987), or enhanced total EPOC only slightly (1.03 L O_2) (Brehm & Gutin, 1986); a change that was statistically significant but would not be of practical benefit in a weight loss program. Similarly, increases in exercise duration at a given exercise intensity

have been associated with marked increases in total EPOC in reports in which EPOC has lasted for several hours (Bahr et al., 1987; Chad & Wenger, 1988), while others have documented no change in EPOC (Sedlock et al., 1989).

The mechanisms underlying EPOC are still being investigated. Exercise-induced increases in core temperature (Tc) have been partially correlated with elevations in metabolism post-exercise (Brehm & Gutin, 1986; Chad & Wenger, 1988; Hagburg et al., 1980), although others have disputed these findings (Bahr et al., 1987; Maehlum et al., 1986). Elevations in plasma concentrations of catecholamines may increase post-exercise VO_2 by stimulating energy-requiring processes in the cell (for example, Na+-K+ pump activity) (Gaesser & Brooks, 1984; Horwitz, 1979), and by promoting the utilization of fatty acids, which generate less ATP per unit of O_2 consumed than do proteins or carbohydrates (Kalis et al., 1988). Elevations in circulating levels of catecholamines have been reported 10 to 20 min post-exercise (Deuster et al., 1989; Tarnopolsky et al., 1990), so that they may exert an influence on post-exercise VO_2. Thyroxine may also participate in EPOC by acting directly on the mitochondria (Gaesser & Brooks, 1984), or by facilitating the adrenergically mediated activities (Goodman et al., 1988).

The following study was undertaken in order to resolve some of the inconsistencies present in the recent literature examining the relation between exercise and post-exercise VO_2.

The effects of both intensity and duration were examined by having subjects perform exercise of varied intensity with total work equated, and exercise of varying durations at a constant intensity. The total exercise performed at different intensities was standardized to a work bout (30 min at 60% VO_{2max}) which represented a midpoint between the work output in which intensity (Brehm & Gutin, 1986; Sedlock, 1989) versus duration (Chad & Wenger, 1985) had predominated as the main factor determining EPOC. This work output was selected in order to determine whether there was a threshold work output at which specific exercise characteristics had a greater influence on EPOC.

In order to determine whether discrepancies among reports of the effects of exercise duration on EPOC were related to differences in the intensity at which exercise was performed (50% versus 70% VO_{2max}), subjects in the present study cycled at an intermediate level (60% VO_{2max}), and for the same durations as those previously reported (30 min and 60 min). Moreover, in the event that findings of the present study replicated those of Chad and Wenger (1988), and a non-linear increase in EPOC with increasing exercise time was observed, a 90 min exercise bout was also included.

Possible mechanisms related to EPOC were explored through the monitoring of subjects' Tc and RER. The data were also analyzed for the cumulative effect of exercise on

subjects' resting metabolic rate (RMR) and the effect of food consumption on EPOC.

In the following chapter (Chapter 2), a review of the literature relevant to the study of EPOC in humans will be presented. This section will include a brief overview of the development of the original 'O_2 debt' hypothesis and the failure of subsequent research to verify the inital formulation. The possible mechanisms underlying EPOC will also be discussed. The experimental study which was performed in order to examine the possible influences of exercise intensity and duration on EPOC will be presented in Chapter 3.

2 LITERATURE REVIEW

2.1 INTRODUCTION

Elevations in VO_2 following muscular work have been documented since the early 1900's. Investigations conducted on humans revealed that VO_2 returned to the pre-exercise baseline in a curvilinear fashion; a single exponential component defined the curve following moderate intensity exercise while 2 components could be discerned followed prolonged or severe work (Hill & Lupton, 1923). Margaria and colleagues (1933) described the 3 components of the decline in VO_2 which follows exercise performed at an intensity $\geq$ 66% VO2max: 1) an inital rapid phase (1/2 time of approximately 30 s) 2) a slow component (approximately 15 min) and 3) protracted elevations in baseline VO_2 lasting for up to several hours.

Although some of the first studies in this area monitored VO_2 in humans for several hours post-exercise (Benedict & Carpenter, 1910; Edwards et al., 1935), the majority of early investigations (e.g. Hill & Lupton, 1923; Meyerhof, 1920) focused on the early recovery phases in an attempt to explain the contribution of lactic acid metabolism

and glycogen resynthesis to the elevations in VO_2. Findings obtained from excised frog muscles and in vivo studies of humans led to the formulation of the 'O_2 debt' concept (Hill & Lupton, 1923). It was postulated that VO_2 during muscular contractions was insufficient to meet total energy requirements, so that energy was derived from the anaerobic catabolism of intramuscular glycogen stores. During recovery, a portion of the lactate produced was oxidized to provide energy for the resynthesis of the remaining lactate to glycogen. The amount of O_2 taken up following contractions in excess of an estimated baseline level constituted the O_2 debt, and was considered to represent the magnitude of anaerobic energy production which had occurred during the contractions.

A multitude of subsequent investigations into the O_2 debt concept have challenged many of its essential tenets and the primary factors which mediate the post-exercise elevations in VO_2 continue to be explored. As a result, many investigators choose to use the term 'excess post-exercise consumption' (EPOC) (Gaesser & Brooks, 1984) rather than O_2 debt, which implies a knowledge of the underlying mechanisms. The increasing recognition that there is no universally accepted theory which adequately explains the EPOC phenomenon has also discouraged investigators from distinguishing between the various phases of EPOC when they are reporting results, since it is possible that the same factors are operative during the various phases and only change in terms of their

relative contributions. As a result, much of the literature which has accumulated during the past decade measures post-exercise VO_2 until baseline levels have been restored, rather than focusing on the early post-exercise period. Despite this change in approach, differences persist among investigators' criteria for determining baseline VO_2 and recovery times post-exercise. As a result, a controversy continues to exist with regards to the magnitude and duration of EPOC. While some reports document elevations in post-exercise metabolism corresponding to considerable increases in caloric expenditure (e.g. 100 to 150 kcal) and lasting several hours, other findings indicate that EPOC makes a very small contribution to total exercise-induced energy expenditure.

It is my contention that the variations among determinations of resting VO_2 and the duration of the post-exercise period are partially responsible for the apparent discrepancies among reports. This issue will be addressed in the following review.

The review is divided into 3 parts. Part I traces the development of the O_2 debt concept and provides a brief critique of the original formulation. In Part II, a synopsis of the literature available on the duration and magnitude of EPOC in humans is presented, together with possible reasons for the discrepancies among reports. Part III summarizes the various mechanisms which are thought to contribute to EPOC.

2.2. THE O_2 DEBT CONCEPT

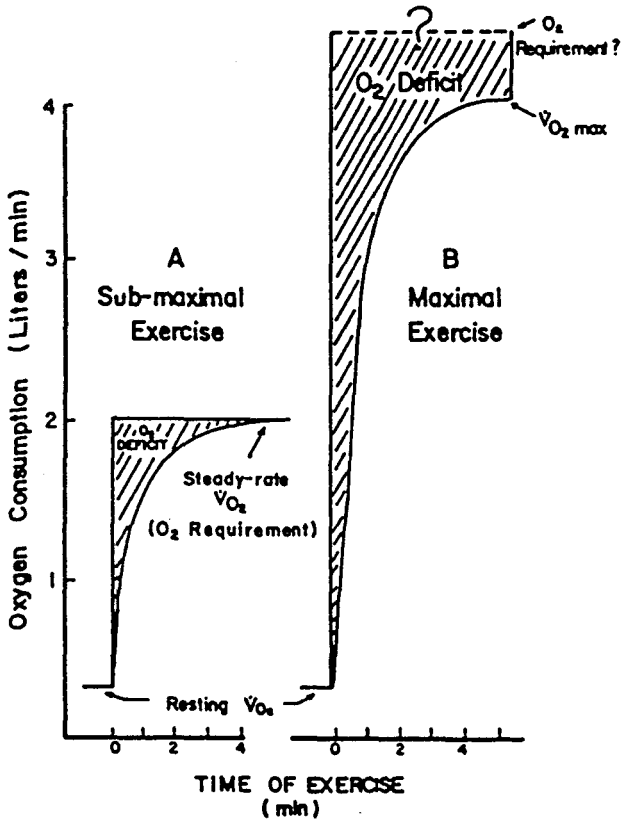
2.2.1 The Initial Formulation of 'O_2 Debt'

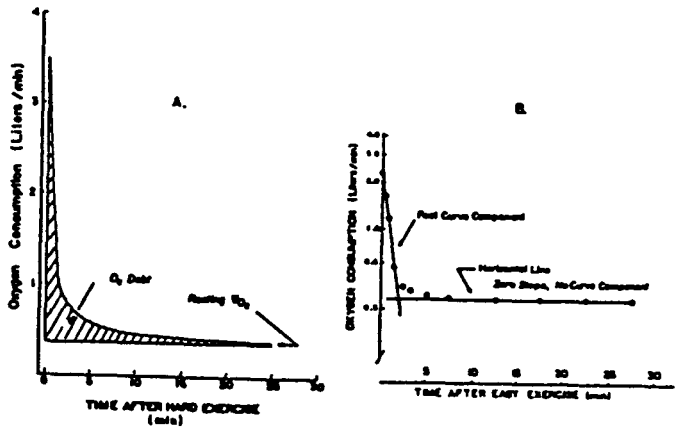
The concept of 'O_2 debt' was presented by Hill and Lupton in 1923. They applied findings obtained from isolated amphibian muscle to exercising humans in order to formulate their theory. It was hypothesized that an 'O_2 deficit' was accrued during the initial phase of exercise, before VO_2 had reached the O_2 requirements of the workload. During this time, energy was provided from the anaerobic metabolism of intramuscular glycogen stores. Hence, exercise was characterized by an initial 'lag' phase, which corresponded to the difference between the actual total energy production and that which could have been attributed to oxidative phorphorylation had VO_2 increased instantaneously to steady-state level. The elevation in VO_2 which persisted for a period of time after exercise was considered to comprise the O_2 debt, and was equivalent to the initial deficit in O_2 intake. During severe exercise, VO_2 never caught up with the O_2 requirements, so that a larger O_2 deficit was accrued which had to be paid off during recovery (Figure 1).

Figure 1 (a). Oxygen deficit acquired during steady-rate submaximal (A) and maximal (B) exercise. The O_2 deficit is equivalent to the difference between the amount of O_2 consumed during exercise and the amount of O_2 required.

Figure 1(b). VO_2 following vigorous (A) and mild (B) intensity steady-rate exercise. The O_2 debt is the extra O_2 consumed above the rsting baseline (the area under the VO_2 curve). Note the rapid and slow components of the decline in VO_2 toward baseline levels.

(Adapted from Brooks & Fahey, 1984, p. 193)


O_2 Requirement?
O_2 Deficit
$\dot{V}O_2$ max
A
Sub-maximal
Exercise
B
Maximal
Exercise
O_2 deficit
Steady-rate $\dot{V}O_2$
(O_2 Requirement)
Resting $\dot{V}O_2$
Oxygen Consumption (Liters / min)
TIME OF EXERCISE
(min)


A.
B.
Oxygen Consumption (Liters /min)
TIME AFTER HARD EXERCISE
TIME AFTER EASY EXERCISE

2.2.2 Evolution of the O_2 Debt Concept

The O_2 debt concept developed out of studies into the conditions necessary for lactate formation in muscle. Fletcher and Hopkins (1907) had shown lactate production in excised frog muscles at rest and following evoked contractions in the absence of O_2. They also found that when the fatigued muscle was placed in pure O_2 for 2 h, the lactate concentration was reduced by approximately 30% and another 20% reduction was observed after several more hours. In 1913 Hill reported that a latent burst of heat was generated following a muscle contraction if O_2 was present. The 'recovery heat-production' was approximately 1/8 of that which would have been produced by the oxidation of the quantity of lactate observed. These findings, in conjunction with those of Fletcher and Hopkins, led Hill to suggest that the majority of the lactate was "...replaced in its previous position, under the influence and with the energy of the oxidation, either (a) of a small part of the lactic acid itself, or (b) of some other body" (Hill, 1914, p xi).

The O_2 debt concept was formulated by Hill and Lupton after Meyerhof identified the lactate precursor as muscle glycogen (1920). In experiments on isolated frog muscle stimulated to contract, Meyerhof found that glycogen disappeared and lactate appeared in a corresponding amount. Following muscle contraction, events reversed themselves; the glycogen reappeared, less 25%, which Meyerhof speculated had

been oxidized and provided energy for the reconstitution of the remaining lactate to muscle glycogen. In 1922, Hartree and Hill showed that, at a constant O_2 tension, the rate of recovery heat-production (i.e., calories per gram muscle per second over approximately 6 min following a tetanic stimulus) increased in proportion to the length of the stimulus and the corresponding increase in heat produced during muscle contraction. They concluded that the rate and magnitude of the recovery heat was dependent on the extent of the preceding muscle 'effort'. It appeared that the essential mechanisms underlying the O_2 debt had been identified, and a comprehensive theory based on the findings up to that point emerged and was translated to the in vivo situation (Hill & Lupton, 1923):

The oxidation of a portion of the lactate produced during exercise provided energy utilized in the resynthesis of the remaining lactate to muscle glycogen. During supramaximal exercise in man, the rate of lactate production exceeded the muscles' capacity to oxidize it, so that oxidation of lactate only occured following exercise. During moderate intensity exercise, O_2 delivery was sufficient for oxidation of the muscle lactate produced during exercise. Hill described a 'steady state' in which lactate production was balanced by the rate of its oxidative removal, so that after an initial period of exercise, during which VO_2 increased in proportion to lactate production, both lactic acid concentration and VO_2

remained constant at a given workload. Elevations in post-exercise VO_2 were thought to represent post-exercise lactate oxidation which was utilized in the resynthesis of the remaining lactate to glycogen. Since, at a given O_2 tension, oxidation became more rapid with increases in lactate availability, the magnitude of muscle exertion governed recovery rate.

Hill and Lupton applied their theory to experimental findings they had obtained from a single subject's VO_2 during and following exercise performed at different intensities. In all cases, they found that VO_2 rose rapidly during the early exercise phase and attained a constant level within approximately 2 1/2 min. The investigators concluded that during bouts of moderate intensity running, lactate which accumulated within the muscle was continuously oxidized: "...the body has reached a state of equilibrium, in which break-down is balanced by recovery" (Hill & Lupton, 1923, p. 151). When the subject ran at an intensity which he could only maintain for a few minutes, the fact that VO_2 stabilized within 2 1/2 min indicated only that its maximal value had been attained; a constant VO_2 at a severe exercise intensity did not represent a balance between lactate accumulation and oxidation.

The authors did not verify their conclusions with measures of muscle lactate. However, they did show that the magnitude and duration of post-exercise VO_2 during 15 min

measurement periods were greater following vigorous as opposed to moderate exercise. They contended that VO_2 after completion of severe exercise represented the oxidation of 1) the lactate produced during the initial phase of exercise before a maximal level of VO_2 had been attained (the 'lag volume') and 2) the lactate produced continuously during the exercise bout. Following moderate exercise, the only O_2 debt was that produced at the beginning of exercise. Krogh and Lindhard's report (1920) of exercise-induced changes in VO_2 in man lent support to the O_2 debt theory. They found that the lag volume, or O_2 deficit, was approximately equal to the excess amount of oxygen taken in after exercise. The equating of the O_2 debt with the O_2 deficit lent support to the contentions of Hill and associates that O_2 utilized in the aerobic synthesis of glycogen was equivalent to the amount substituted by the anaerobic breakdown of muscle and liver glycogen stores during exercise.

2.2.3 Short Versus Long Term Elevation of Post-Exercise O_2 Consumption

A distinction must be made between the O_2 debt and the observations of a sustained increase in basal metabolic rate (BMR) occurring after exercise. The O_2 debt was characterized by specific biochemical processes which provided for the repletion of the anaerobically catabolized substrates. These oxidative processes were responsible for the early elevations

in VO_2 observed post-exercise and were not related to any long-term elevations in post-exercise BMR. However, persistent increases in VO_2 lasting for at least 24 h after exercise had been reported as early as 1910 by Benedict and Carpenter. Hill and colleagues attributed the long-term effects to a "general circulatory and metabolic disturbance produced by exercise" (Hill, Long & Lupton, 1924). Margaria and associates (1933) reinforced the distinction made between the short and long term effect of exercise on VO_2. They described a post-exercise VO_2 curve consisting of three phases; the first two phases were both defined by velocity constants and were completed within approximately 15 min, while the third phase could not be defined mathematically and represented "an increase in resting metabolism caused by the exercise" (Margaria, Edwards & Dill, 1933). Newsholme referred to the long term elevations in VO_2 as the 'ultraslow' phase (Newsholme, 1978). Hence, the long-term effect was reputed to be the result of different physiological mechanisms. Although this view was criticized by some (see, for example, Harris, 1969), it dominated the literature until the 1980's. Findings of long-term changes in metabolism will be discussed in Part II.

2.2.4 Alactacid Component of the O_2 Debt

According to Hill and associates, glycogen was the sole source of anaerobically supplied energy during muscle contraction. An important modification was made to the O_2 debt concept early on, following the discovery of creatine phosphate (CP) (Eggleton & Eggleton, 1927) and adenosine triphosphate (ATP) (Lohmann, 1931) in muscle. In 1930, Lundsgaard elicited contractions in muscle poisoned with iodoacetate, which prevented lactate formation, and showed a simultaneous decline in muscle phosphate compounds. In so doing, he demonstrated that CP provided an energy source for muscle contraction. It was he who coined the terms 'lactacid and alactacid anaerobic energy output' (1932), in order to distinguish between energy derived from the conversion of glycogen to lactate and that produced by the breakdown of CP and ATP stored in the muscle.

These terms were subsequently adopted by Margaria, Edwards and Dill (1933) in order to differentiate between different components of the O_2 debt. The latter group of investigators focused on the hypothesized relationship between the production and removal of lactate and the payment of the O_2 debt. Data were obtained from a single subject following treadmill exercise performed at a range of intensities. No increase in plasma lactate was detected following exercise performed at intensities less than 2/3 of the subject's maximal aerobic capacity. They concluded that lactate was not

in fact produced during moderate exercise, and that another 'alactic' mechanism was responsible for the O_2 debts ($\leq$ 3 L) recorded after these exercise bouts.

It also appeared that an alactic mechanism was operative during vigorous exercise. A rapid fall in post-exercise VO_2 was observed after running bouts of 3 to 10 min, prior to any declines occurring in plasma lactate levels (Margaria, Edwards & Dill, 1933). Moreover, the decline in plasma lactate levels followed a single logarithmic curve, whereas post-exercise reductions in VO_2 were characterized by 2 distinct phases - rapid and slow - each of which was exponential (these phases did not include the ultraslow component). Hill, Long and Lupton (1924) had made similar observations of the O_2 debt curves. They had attributed the initial, rapid phase of recovery to the oxidative removal of lactate from the exercised muscles, while the slow phase represented the uptake of lactate which had diffused from the muscles into the blood. Margaria and colleagues concluded that the rapid phase represented an 'alactic' payment of the O_2 debt, during which muscle phosphate stores were being replaced. The oxidation of lactate occurred subsequent to this, and was reflected by the slower declines in post-exercise VO_2.

2.3 CRITIQUE OF THE O_2 DEBT HYPOTHESIS

Numerous studies conducted on animals and humans during the past 60 years have failed to substantiate several basic tenets of the O_2 concept. The original assumptions and the results of subsequent investigations will be discussed in the following section.

2.3.1 Conditions for Lactic Acid Production

According to Hill and Lupton (1923), lactate production occurred under conditions of muscle hypoxia, when fuel had to be supplied anaerobically. It is now known that under normoxic conditions, lactate can be produced at all levels of work, except perhaps very light exercise (Jones, 1980). As early as 1925, Collazo and Lewicki showed that the ingestion of 100 g of sucrose by human volunteers led to elevations in plasma lactate levels. Similar results were obtained in rats (Cori & Cori, 1929). It was also suggested that a critical determinant of adequate O_2 supply to the cell might be the diffusion rate of O_2 from blood to muscle (Krogh, 1929). Subsequent investigators showed that the minimum capillary PO_2 necessary to ensure sufficient diffusion rates was as low as 10 mmHg (Saltin et al., 1968; Stainsby & Otis, 1964); lactate could also appear under these conditions. The recent introduction of quick freezing microspectrophotometric techniques has made it possible to measure PO_2 directly, and

it has since been shown that the PO_2 required for maximal ATP production is always present in the cell (Walsh & Banister, 1988). Muscle hypoxia, therefore, is not a necessary precondition for lactate production.

2.3.2 Dissociation Between Post-Exercise VO_2 and Lactate Levels

An essential tenet of the O_2 debt concept originally described by Hill and associates was that lactate metabolism was inherently linked to the magnitude and duration of post-exercise elevations in VO_2. Although Margaria and colleagues later postulated that the initial rapid decline in post-exercise VO_2 represented the resynthesis of creatine phosphate, their observations of post-exercise declines in plasma lactate levels and VO_2 led them to conclude that at high work intensities, the slow component of the O_2 debt represented the oxidation of lactic acid. However, the similarities which they observed between the time courses of VO_2 and lactate declines post-exercise were subsequently attributed to their selection of 8 to 10 min exercise bouts. Bang (1936) showed that plasma lactate levels peaked within 5 to 10 min after exercise onset, regardless of exercise intensity or duration. Harris, in his critique of the O_2 debt concept (1969), points out that the recovery VO_2 curve was divided into two components without any evidence that they coincided with the physiological variables they were supposed

to represent. Indeed, until the utilization of isotopic lactate tracers, it was not possible to determine what proportion of lactate was oxidized or resynthesized into glycogen; or whether lactate was in fact used as a substrate in other metabolic reactions. Harris writes that "... by manipulating a ruler a little bit one way or another, many...complex curves can be resolved graphically into two main exponential components and the temptation to ascribe these components to physiological realities has so often proved irresistible" (Harris, 1969, p 385).

The evidence to date has failed to corroborate an association between the time course of declines in post-exercise lactate and VO_2 in animals or man. Marked, post-contractile elevations in the plasma lactate levels of amphibian and reptile skeletal muscle were shown to persist for several hours after exercise, long after VO_2 had normalized (Bennet et al., 1973; Bennet et al., 1978; Gleeson et al., 1980). Gaesser and Brooks (1980) found the reverse situation in rats following prolonged exercise to exhaustion. Lactate concentrations in liver and muscle were equivalent to pre-exercise levels within 15 minutes, whereas VO_2 was only re-established after 2 h.

Dissociations between declines in plasma lactate levels and VO_2 following exercise have also been documented in humans. When the effects of exercise performed at 30% and 55% of VO_{2max} on the O_2 debt were compared, O_2 debt was found to

increase with exercise intensity while no changes were observed in lactate concentrations (Schneider et al., 1968). When exercise duration was extended, increases in the slow phase of post-exercise VO_2 were documented simultaneously with declines in plasma lactate levels (Knuttgen, 1970). Studies of glycogen-depleted subjects during and following exercise have shown significant reductions in plasma lactate levels, while total EPOC has been equivalent to glycogen-sufficient controls (Segal & Brooks, 1979). Roth and associates (1988) recently investigated the effects of experimentally induced lactacidemia on EPOC by occluding leg blood flow of male subjects between the 6th and 8th minute of exercise during 12 min of cycle ergometry. Blood lactate remained significantly higher than control levels 18 min into recovery but total EPOC was equivalent between the two conditions (13.71 $\pm$ .45 L O_2 versus 13.44 $\pm$ .61 L O_2). The hypothesized connection between lactate oxidation and the slow phase of post-exercise declines in VO_2 has not been substantiated by measures of the time courses of the two variables. Brooks and Gaesser (1984) write that "Any apparent relationship between the time course of blood lactate decline after exercise and the slow phase of the excess post-exercise VO_2 is probably coincidental, and most likely the result of the selection of exercise conditions" (p 34).

2.3.3 Fate of Excess Lactate

A third premise of the O_2 debt theory was that in the whole body, declines in plasma lactate represented the substrate's uptake and resynthesis to glycogen. This was considered to be the major fate of lactate with a smaller fraction (16% to 25%) being oxidized to provide energy for the process. Meyerhof's (1920) original report that glyconeogenesis occurred in vitro amphibian muscle led Hill and Lupton (1923) to conclude that the exercised muscles were the sites for glycogen resynthesis. This theory was challenged early on, following unsuccessful attempts by Cori (see 1931 review) and others (Eggleton & Evans, 1930; Sacks & Sacks, 1935) to replicate Meyerhof's findings in vivo animals. Krebs and associates were unable to demonstrate activity of the key glyconeogenic enzymes (PEP carboxykinase and F-1,6, bis-phosphatase) in mammalian skeletal muscle and concluded that skeletal muscle lacked the capacity to synthesize glycogen from lactate (Krebs & Woodford, 1965; Ross et al., 1967). The Cori cycle was proposed to explain the role of lactate in glycogen synthesis following exercise. Lactate was transported from the exercised muscles to the liver for conversion to glucose and/or glycogen. Hepatic glucose was subsequently released into the blood and taken up by the muscles where it was synthesized to glycogen (Cori, 1931).

Brooks and Gaesser (1980), who used isotopic tracers in order to determine the post-exercise fate of lactate, were instrumental in demonstrating that a primary route of lactate

elimination is the oxidation of lactate to CO_2 and H_2O. When [1-^{14}C] lactate was injected into rats after completion of prolonged exhaustive exercise, they found that most of the lactate was oxidized (55% to 70%) within 4 h, while less than 20% was converted to muscle and liver glycogen. Major sites for lactate oxidation appear to be the heart muscle, the kidney cortex (Newsholme and Leech, 1983) and the skeletal muscles. It is known that Type I muscle fibres, which have a high oxidative capacity, lower lactate concentrations and a lower NADH/NAD ratio than the Type II fast-twitch fibres, will take up lactate and convert it to pyruvate for oxidative phosphorylation via the following reaction:

$$\text{lactate} + 3\ O_2 \rightarrow CO_2 + HCO_3^- + 3\ H_2O$$

This metabolic reaction allows muscle to use lactate as fuel during exercise (Jones, 1980). It is likely that lactate oxidation occurs to a greater extent in the exercised than in the inactive muscles (Belcastro & Bonen, 1975; Bonen et al., 1989) although there is also evidence of lactate uptake by the inactive muscles during the first 10 minutes following 30 sec of maximal exercise (Kowalchuk et al., 1988). Duration and intensity of exercise and rate of lactate accumulation may influence the role played by inactive muscles in the oxidation of lactate.

A second major site of lactate removal is the liver, where lactate is converted to glucose or glycogen and subsequently utilized in the repletion of hepatic glycogen

stores or released by the liver as glucose for muscle glycogen resynthesis (Brooks, 1986). Gluconeogenesis is less efficient than oxidation because it is energy consuming; it also reduces liver pH. The conversion of lactate to glucose results in the following net energy exchange:

$$2\ \text{lactate}^- + 4\ ATP^{4-} + 2\ GTP^{4-} + 6\ H2O \rightarrow$$

$$\text{glucose} + 4\ ADP^{3-} + 2GDP^{3-} + 6P^{2-} + 4H^+$$

Thus, glucose production may lower energy stores and increase hepatic hydrogen ion levels (Jones, 1980).

Internal metabolic conditions at the cessation of exercise likely partially determine lactate's pathway of elimination. Lactate may be preferentially utilized as a precursor for gluconeogenesis in an attempt to maintain blood glucose levels following prolonged, exhaustive exercise, when glycogen stores and blood glucose concentrations are low (Brooks & Fahey, 1984). The hyperglycemia observed following intermittent, high intensity exercise may increase lactate oxidation. When the splanchnic uptake of lactate during recovery from brief exhaustive exercise was measured, it accounted for less than 10% of the total lactate produced (Astrand et al., 1986).

The route utilized for the elimination of lactate may also be affected by concentrations of circulating lactate. There appears to be a maximal rate for hepatic uptake of lactate; beyond this, gluconeogenesis is prohibited by declines in hepatic ATP levels and pH. It has been suggested

that glucose production from lactate is maximal when plasma lactate levels reach 5 mmol.L^{-1} (Jones, 1980).

Traditional arguments against glyconeogenesis occurring directly within the muscle have recently been challenged by findings of post-exercise muscle glycogen repletion which cannot be explained by rates of hepatic uptake of lactate or release of glucose (Astrand et al., 1986; for review, see Bonen et al., 1989). However, there is no direct evidence to date of lactate conversion to glycogen in human skeletal muscle.

In addition to the two primary routes of lactate elimination - lactate oxidation and gluconeogenesis - lactate also appears to serve as a carbon source for transamination reactions post-exercise. [U-^{14}C]lactate injected into rats at exercise cessation appeared in the protein constituents (e.g. glutamate and alanine) of various tissues (Brooks and Gaesser, 1980).

In summary, the original O_2 debt theory rested on several assumptions which have not been validated by subsequent research. This is primarily due to the fact that the initial formulation was derived from studies of isolated frog muscle and did not take into account the multiplicity of factors operating in the whole body. The authors did not consider that lactate might be utilized in a number of different metabolic pathways both during and following exercise. The cumulative evidence to date shows that: 1)

post-exercise plasma lactate levels do not provide an accurate index of exercise-induced anaerobic glycogenolysis, 2) there is not a relation between the time course of declines in VO_2 and lactate following exercise and 3) the primary fate of lactate is not glyconeogenesis. Gaesser and Brooks (1984) suggested replacing the term 'O_2 debt' with the descriptive title of 'EPOC' since the possible mediators of changes in VO_2 are still unknown. The latter term will be used in the following 2 sections of this review.

2.4. A REVIEW OF THE STUDIES ON EXCESS POST-EXERCISE OXYGEN CONSUMPTION IN HUMANS

2.4.1 Introduction

More than a decade before the 1923 publication of Hill and Lupton's O_2 debt concept, Benedict and Carpenter (1910) provided evidence that exercise might have long-term effects on RMR. They reported that one subject's metabolic rate remained elevated for several hours after he performed severe and very severe work. The total amount of his post-exercise VO_2 was increased by 8% and 25%, respectively, above resting levels. However, their findings were largely ignored after the introduction of the O_2 debt hypothesis in 1923. Most investigators chose to focus on the early phases of EPOC in order to more clearly identify the underlying 'alactic' and 'lactic' processes which comprised the O_2 debt (see Bahr & Maehlum, 1986). Although there are a few early reports of the prolonged effects of exercise on post-exercise metabolism, interest in this area has increased markedly during the past decade. Two factors are responsible for this shift in orientation. First, more attention has been given to the role of exercise in weight control, and it has been suggested that one of the ways in which exercise reduces body fat is by acting as a chronic stimulant of metabolism (Goodman, 1980). Second, since many of the original tenets of the O_2 debt theory have been brought into question, it is no longer

meaningful to emphasize only the early components of EPOC. As a result, a body of literature is becoming available on the duration and magnitude of total EPOC observed in response to a range of intensities and durations of exercise.

In the following section, the accumulated results of the studies of EPOC in humans will be presented. The review will be limited to studies in which post-exercise VO_2 was monitored until it was considered to be restored to pre-exercise levels. Reports which did not incorporate observed long-term elevations in RMR into calculations of total EPOC will not be included. The investigations can be roughly classified into 2 groups, according to the magnitude and duration of EPOC obtained in response to a given bout of exercise. The first group comprises the investigations which have shown EPOC to be of considerable duration and magnitude, and which consequently emphasize its contribution to overall exercise-induced energy expenditure. Group 2 comprises the majority of recent reports, which have documented relatively insignificant post-exercise elevations in metabolism, both in terms of total caloric expenditure and with respect to the duration of the effects.

Many of the discrepancies between the findings of the 2 groups can be accounted for by differences in design and methodology. Results of the early studies (ie. 1910 to about 1970) are confounded by their small sample sizes, their inclusions of many different exercise intensities and

durations, and their lack of control for diet and post-exercise activity levels. The investigations conducted in the last 10 y, however, have generally controlled for these factors. Yet differences persist among the methods utilized to determine baseline levels, and the duration of the post-exercise recovery period. The methodological variations, and their significance to investigators' interpretations of EPOC, will be highlighted throughout the review. The effects of exercise training on resting metabolism and meal-induced thermogenesis will also be presented.

2.4.2 Definition of Resting Metabolic Rate and Thermic Effect of Feeding

The individual's RMR accounts for 60 to 75% of his/her total daily energy expenditure and as such, constitutes the single largest component of total energy cost in sedentary humans (Poehlman, 1989a). It comprises the cumulative costs of maintaining body temperature and physiological homeostasis at rest, including the preservation of electrolyte gradients, respiratory and cardiovascular work and the continued functioning of the central nervous system.

The RMR can be obscured by the 2 other components of energy expenditure: EPOC and the thermic effect of feeding (TEF). The TEF constitutes about 10% of daily energy expenditure, and includes the energy cost of food absorption, metabolism and storage within the body. It also refers to

additional energy which some investigators claim is expended in response to meal ingestion and which is not accounted for by obligatory nutrient disposal and storage (Acheson et al., 1984; Thiebaud et al., 1983). This energy has been termed 'facultative thermogenesis'. Most of the recent studies have controlled for the confounding influences of EPOC and TEF on RMR by keeping diet and activity profile consistent among subjects and exercise conditions. However, these factors have varied among studies, and may account for some of the discrepancies among findings. For example, Chad and Wenger (1985; 1988) did not feed their subjects for several hours after exercise completion, and subjects' post-exercise VO_2 exceeded that in other studies after similar work bouts (Table 1). Measures of RMR are also inconsistent among studies; values have been averaged over a pre-determined period of time (Chad and Wenger, 1985; 1988; Freedman-Akabas, 1985), monitored until a given minimal variance is attained (Brehm and Gutin, 1986), or values obtained at corresponding times on a control day have been used (Bahr et al., 1987; Maehlum et al., 1986). RMR measures may be further confounded by the inclusion of female subjects, since menstrual cycles are associated with corresponding fluctuations in basal metabolic rates (Solomon et al., 1982).

2.4.3 Reports of EPOC of Marked Duration and Magnitude

A small number of investigators followed up on Benedict and Carpenter's original report of sustained elevations in post-exercise RMR. Brief bouts of vigorous exercise (for example, an 800 m race) (Radtke, 1927) were associated with heightened VO_2 lasting 36 to 48 h (Herxheimer et al., 1926) and 24 h (mean increase of 15%) (Radtke, 1927). Two hours of football augmented VO_2 by 25% during a 15 h post-exercise period (Edwards et al., 1935). These studies were valuable because they provided suggestive evidence that exercise induces prolonged physiological changes, which are reflected in persistent changes in RMR. However, the results of these studies could not be considered conclusive due to limitations in experimental design. Diurnal variations in RMR, dietary intake and activity level post-exercise may well have been responsible for the observed elevations in VO_2.

Few subsequent reports have documented such prolonged elevations. This is partially due to the fact that post-exercise VO_2 has generally not been measured for such extended periods. When BMR has been measured 1 d following exercise completion, findings have not been consistent (Bahr et al., 1987; Bielinski et al., 1985; Maehlum et al., 1986; Shah et al., 1988). Differences cannot be accounted for by variations in total work, intensity or duration of exercise, or subject populations. It could be argued that the increases in VO_2

which were observed the morning following the exercise bout (Bielinski et al., 1985; Maehlum et al., 1986) were not a direct result of the exercise itself. In one study (Bielinski et al., 1985), subjects' VO_2 were equivalent on the experimental and control days during the evening and night, so that a significant difference was only obtained after a lapse of many hours. No actual data are provided by Maehlum and associates, and it may be that the elevations fell within the normal range of intra-individual variations in daily BMR (1.8% to 2.2%) (De Boer et al., 1987; Rumpler et al., 1990) Furthermore, VO_2 was not measured during the 12 to 24 h interim post-exercise, so that VO2 may have normalized and increased again just prior to the morning measurement.

Investigators have tended not to monitor VO_2 for as long as 24 h after exercise because resting levels are attained long before. In some cases, however, VO_2 has been measured for extensive periods, and an EPOC of several hours duration (4.5 h to 12 h; Table 1) has been reported (Bahr et al., 1987; Bielinski et al., 1985; Chad & Wenger, 1988; de Vries, 1963; Maehlum et al., 1986; Passmore and Johnson, 1960). The prolonged durations have been observed after a range of exercise intensities (50% to 70%) and durations (20 min to 3 h), and total work performed has varied among the studies. Moreover, exercise bouts similar in intensity and

Table 1. Summary of EPOC Studies.

GROUP I (Reports of EPOC of considerable duration and magnitude)

Study	Subjects		Exercise			EPOC		
	N & Gender	Fitness Level	Duration (min)	Intensity (%VO_{2max})	Total Work	Duration	Magnitude	%EOC
Bahr et al., 1987	6 M	mod-high	20	70%	56.06 L	12 h	11.1 L	19.8%
			40	"	120.49 L	"	14.7 L	12.2%
			80	"	234.56 L	"	31.9 L	13.6%
Bielinski et al., 1985	10 M	high	180	50%	2100 kcal	4.5 h	40 kcal	9%
Chad & Wenger, 1988	2M + 3F	untrained	30	70%	63.13 L	128 min	6.78 L	10.74%
			45		95.24 L	204 min	16 L	16.8%
			60		128.43 L	455 min	35.96 L	28%
De Vries & Gray, 1963	2 M	low-average	NP	NP	NP	6 - 8 h	53 kcal	NP
Maehlum et al., 1986	4M + 4F	moderate	80	71.1%	191.8 L	12 h	26 L	13.6%

GROUP II (Reports which stressed that EPOC is not of any practical significance)

Study	Subjects		Exercise			EPOC		
	N & Gender	Fitness Level	Duration	Intensity (%VO_{2max})	Total Work	Duration	Magnitude	%EOC
Brehm & Gutin, 1986	4M + 6F	high	60	18.2	39.7 L	18.8 min	.825 L	2.08%
			30	32.6	35.5 L	42.3 min	1.95 L	5.5%
			24	50.1	43.7 L	31.1 min	1.89 L	4.3%
			17	68.1	42 L	48 min	2.92 L	7%
Deuster et al., 1989	21 M	7 untr	20	50%	NP	10-20 min	NP	NP
		7 mod	20	70%	"			
		7 high	10	90%	"			
Elliot et al., 1988	3M + 3F	range	10	80% HR_{max}	"	<30 min	11.4kcal over 90 min measurement period	"
			30	"	"			
Freedman-Akabas et al., 1984	10M + 13F	untrained	20	AT	"	<40 min	NP	"
	4M + 3F	high	20	AT+2mph	"	"	"	"
			40	AT(+)1mph	"	"	"	"
Kaminsky et al., 1987	14M(group1)	moderate	60	35%	417 kcal	15 min	"	"
	10M(group2)	"	30	70%	394 kcal	10 min	"	"
Knuttgen, 1970	7M + 5F	untrained	15	45-98%	NP	NP	1.4-1.8 L	"
Pacy et al., 1985	2M + 2F	moderate	20/h x 4h*	35-55%	"	40 min after each session	NP	"
Sedlock et al., 1989	10 M	triathletes	19.9	75%	304 kcal	33.3	29.4 kcal	9.7%
			29.6	50%	307 kcal	19.8	14.3 kcal	4.7%
			59.2	50%	611 kcal	28.4	12.1 kcal	2%
Shah et al., 1988	16F(post-obese)	untr	30 x 4	mild	430 kcal	3-4 h	50 k/24 h	11.6%
	16F(lean)	"	10 x 4	60-80%	510 kcal	(predetermined time)	30 k/24 h	5.9%

Values represent means of data. Durations of exercise and EPOC presented in minutes, unless hours indicated with 'h'. Total work of exercise and magnitudes of EPOC presented in L/O_2 ('L') or kilocalories ('kcal'). '%EOC' represents percentage relation of EPOC to the total O_2 consumed during exercise. 'NP' signifies data not provided; 'AT' signifies anaerobic threshold.

* Subjects exercised for 20 min and rested for 40 min for four consecutive hours. A meal was consumed after the second 20 min exercise bout.

duration to those listed above have been associated with a much briefer EPOC (Table 1). Therefore, the lengthy durations in EPOC reported cannot be attributed to specific exercise characteristics.

The use of different criteria to define baseline and recovery time may explain the variations among the lengths of EPOC reported. In some cases, pre-exercise VO_2 was used as the baseline (Chad & Wenger, 1988; Passmore & Johnson, 1960), and it is possible that post-exercise elevations in VO_2 merely reflected diurnal increases in resting rates (Winget et al., 1985). The durations of EPOC reported by other investigators may be misleading because they calculated the total magnitude of post-exercise VO_2 for a pre-determined period of time. As a result, their measures of EPOC include periods during which VO_2 was not significantly higher than control values. For example, Bielinski and associates (1985) reported a significant 9% increase in total VO_2 for the 4.5 h which followed a 3 h exercise bout and a meal. However, when differences between experimental and control VO_2 are evaluated at 30 min intervals, VO_2 elevations are only significant during the first and third hours of the 4.5 h period (see their Figure 2). If the authors had ceased measuring VO_2 once control values were attained, the duration of EPOC would have been reduced from 4.5 h to 60 min. Similarly, the studies performed in Bahr and Maehlum's laboratory were presented as evidence that exercise increases metabolic rate for at least

12 h. Yet the authors did not identify the specific sampling times at which significant differences occurred between experimental and control data. The use of pre-determined recovery periods may have enabled the afore-cited investigators to incorporate data which were not significantly greater than control values but were suggestive of a trend, so that their inclusion increased measures of total EPOC.

2.4.4 Reports of EPOC of Brief Duration and Small Magnitude

Other investigators have tabulated the duration of EPOC from the data collected when differences between experimental and control data were statistically significant (Brehm & Gutin, 1986; Chad & Wenger, 1988; Kaminsky et al., 1987; Sedlock et al., 1989). With one exception (Chad & Wenger, 1988), a briefer EPOC has been reported when EPOC is only calculated from VO_2 measures which significantly exceeded control values. It can be seen that differences in the duration and magnitude of EPOC were found between the 2 groups of studies, despite the fact that in several cases, subjects performed similar work bouts (60% VO_{2max} for 30 min to 70% VO_{2max} for 20 min). In some cases, total EPOC is negligible (less than 15 kcal) (Brehm and Gutin, 1986; Elliot et al., 1988). Differences among subject populations cannot explain the variations among results.

Reports of excess caloric expenditure post-exercise have ranged from 7 kcal (Knuttgen, 1970) to 174 kcal (Chad & Wenger, 1988), and the variatons among findings have generated a controversy in the literature regarding the significance of the contribution of EPOC to exercise-induced energy expenditure.

2.4.5 The Effect of Exercise Training on Resting Metabolic Rate and the Thermic Response to Feeding

Longitudinal and cross-sectional studies have been conducted to investigate possible changes in RMR with chronic exercise. Several recent investigations have explored the combined effects of dietary restriction and exercise on RMR. Reductions in caloric intake have frequently lowered resting metabolism (Heymsefield et al., 1989; Mole et al., 1989; Phinney et al., 1988), although increases in RMR per kilogram body mass with dieting have also been reported (Lennon et al., 1985). The addition of exercise has been associated with increases in RMR per kilogram body mass (Lennon et al., 1985; Nieman et al., 1988) or has attenuated the observed declines (Mole et al., 1989). In other cases, prolonged bouts of mild to moderate intensity exercise performed over a 4 to 5 week period have resulted in further reductions in RMR compared to controls when expressed per kilogram fat free mass (8%) (Heymsfield et al., 1989) and per kilgram body mass (17%) (Phinney et al., 1988). The observed declines in RMR may

represent compensatory mechanisms which occur in response to the exercise-induced enhancements in energy expenditure (Richard & Rivest, 1989).

The literature contains very few longitudinal reports of the effects of exercise training on RMR when subjects are not placed on low calorie diets. When eight obese women were instructed not to alter their diets during an 11 wk training program, significant increases in RMR per kilogram fat free mass were observed (Tremblay et al., 1986). However, mean resting metabolism did not change following 22 d of prolonged (116 min/d) exercise performed at 58% VO_{2max} (Poehlman et al., 1986) or during 9 wk of exercise training by normal weight men and women (Bingham et al., 1989). Reasons for the differences among findings are not readily apparent but may be related to the effects of exercise on energy balance. For example, mean reductions of 20% (Bingham et al., 1989) and 33% (Poehlman et al., 1986) in subjects' caloric balance were reported and it is possible that subjects' RMR was influenced by this change. Dietary intake was not supervised in Tremblay et al.'s (1986) study, so that potential changes in energy balance are not known. The possible relation between energy blance and RMR is emphasized by findings of increases in resting metabolism in female subjects who also gained weight during an exercise training program (Lawson et al., 1987). The elevations in RMR were attributed to increases in caloric intake and a consequent rise in TEF.

The evidence from cross-sectional studies is also inconclusive, with approximately 1 in 2 investigations showing significant elevations in the RMR of trained individuals. State of training appears to be a crucial variable. Higher metabolic rates have been observed in subjects with a VO_{2max} of 65 to 80 ml/kg/min (Poehlman et al., 1989; Tremblay et al., 1986), as opposed to individuals whose VO_{2max} levels did not exceed 60 ml/kg/min (Davis et al., 1983; LeBlanc et al., 1984; LeBlanc et al., 1984a). The need to distinguish between moderately and well-trained subjects was recently demonstrated by Poehlman and associates (1989), who included very active and moderately active participants in their study. They found that the RMR of highly fit individuals exceeded that of the moderately fit and sedentary subjects, and values were equivalent between the latter two groups.

Other factors may contribute to the apparent relation between level of training and RMR. Poehlman and colleagues (1989a) describe an enhanced 'substrate flux' caused by a high energy intake as well as a high energy expenditure. Since energy consumption is very high in a trained athlete, elevations in RMR may represent an enhanced energy turnover resulting from their diets and exercise regimes. Gender may also reduce the impact of exercise on RMR. When postovulatory women were classified into highly active, moderately active and sedentary, RMR was equivalent among all groups (LeBlanc et al., 1984a). The lack of significant differences may be

related to a lower substrate flux among women, since they may adhere to more rigid diets. There also appears to be a strong genetic component determining individual variation in RMR. When monozygotic twins exercised daily for 22 d at 58% VO_{2max}, no consistent group training effect was observed; RMR had declined in some pairs of twins and increased in others at the completion of the program (Poehlman et al., 1986). However, there was a strong intrapair correlation ($r=.81$, $p<.01$) for absolute changes in RMR. Hence, individuals may be predisposed to respond to exercise training according to their genotype.

The literature contains some evidence that exercise affects post-exercise energy expenditure by altering TEF. Cross-sectional studies have shown an elevated (Davis et al., 1983; Lundholm et al., 1986) and reduced (Tremblay et al., 1983; LeBlanc et al., 1984; Poehlman et al., 1989) TEF in trained individuals. Studies involving female subjects have shown either no difference during a 4 h period post-meal ingestion (11 kcal/kg) (Owen et al., 1986) or TEF has been lower during the first h after a mixed meal (818 kcal) but total 2 h TEF has been equivalent among trained and untrained subjects (LeBlanc et al., 1984a). Poehlman and associates (1989a) have postulated that an inverted-U relationship characterizes the association between fitness level and TEF, which may explain the discrepancies among reports. When individuals of a wide range of activity profiles are compared,

TEF may be highest among the moderately active and decline in individuals at either extreme of the scale. When Poehlman and colleagues (1989) measured the 3 h TEF of the above groups, values (kcal/kg/180 min) were significantly higher in the moderately fit group than in the untrained and highly trained groups (.97±.05 versus .83±.04 and .77±.04 respectively). The trained subjects involved in the studies showing increases in TEF with training (Davis et al., 1983; Lundholm et al., 1986) were actually similar to Poehlman et al.'s (1989) moderately active subjects. Hence, differences among investigators' criteria for defining fitness level may be the primary factor responsible for the apparent divergent findings, with regards to both TEF and RMR.

There is little evidence of an acute interaction effect between exercise and meal ingestion. Pacy and associates (1985) found no change in post-exercise VO_2 following the ingesting of an 800 kcal mixed meal. Bahr and associates (1987) reported that 64% of total EPOC was measured during the two 5 h periods which followed each meal consumed after exercise completion. However, these elevations may have occurred solely as a result of the exercise bouts. Subjects would have had to perform an additional bout of exercise under fasting conditions in order to evaluate the contribution of TEF.

2.5.1 POSSIBLE MECHANISMS UNDERLYING EXCESS POST-EXERCISE OXYGEN CONSUMPTION

It is likely that a number of different factors contribute to EPOC. These factors can be classified into 2 groups. The first group includes the variables which restore the body to its resting homeostasis: 1) the direct repletion of fluid and tissue O_2 stores, 2) ion redistribution and tissue repair and 3) the resynthesis of substrates utilized during exercise. In the second category are those mechanisms elicited in response to exercise which continue to have residual effects after exercise completion: 4) cardiorespiratory work, 5) increased lipid oxidation; 6) temperature effects and 7) hormonal effects. It is also likely that each of the various factors contributes primarily to a particular phase of EPOC. The mechanisms will be discussed with reference to the potential magnitude and time course of their effect on EPOC.

2.5.2 Fluid and Tissue O_2 Repletion

Approximations of the amount of O_2 required for repletion of blood O_2 levels have indicated that a maximum of 9.5 ml O_2 may be consumed post-exercise for the purposes of blood and tissue O_2 repletion (Stainsby & Barclay, 1970). In a 70 kg individual, this would be equivalent to a caloric expenditure of approximately 3 kcal, which represents a negligible proportion of EPOC.

2.5.3 Ion Redistribution and Tissue Repair

The amount of O_2 which is utilized in the repair of tissue and ion redistribution post-exercise has not been established. It is likely that the amount of tissue damage which occurs is highly variable and dependent on the individual's training regime, fitness level, the type of exercise performed and its intensity and duration. Ion redistribution would include transport of Na+, K+ and Ca++ across cell membranes, and Ca++ sequestration by the sarcoplasmic reticulum. Ca++ may also disrupt the process of oxidative phosphorylation, which would elevate the VO_2 (Gaesser & Brooks, 1984). Mitochondrial sequestration of Ca++ released during exercise results in increases in VO_2 without corresponding elevations in ATP production (Carofole & Lehninger, 1971).

2.5.4 Substrate Resynthesis

CP and glycogen depletion at exercise completion varies with the intensity and duration of the exercise performed. CP is resynthesized during prolonged, moderate intensity exercise. When the quadriceps femoris of male subjects was electrically stimulated (tetanic trains at 20 Hz lasting 1.6 s) for 45 min, CP levels declined to 26% of rest values within the first 80 s of exercise, and increased to 73% of rest values following 45 min of contractions (Hultman & Spriet, 1986). The restoration of CP to pre-exercise levels would have only required the resynthesis of a further 21.6 mmol CP/kg^{-1} dry muscle, or approximately 7 ml O_2/kg^{-1} dry muscle, if the ATP utilized for the CP repletion was generated via oxidative phosphorylation. The time course of CP repletion following short-term, exhaustive exercise appears to follow a biphasic curve, with the majority of the substrate restored within 10 min post-exercise. For example, CP levels were reduced by 60% to 85% following one-legged, dynamic knee-extensor exercise performed to exhaustion, and had increased to 72% to 77% of their pre-exercise concentrations within 4 to 10 min post-exercise (Harris et al., 1976 & Bangsbo et al., 1990, respectively). Brooks and associates (1971), who assumed complete hydrolysis of muscle CP during exercise and no disruption of mitochondrial respiration, calculated the maximal amount of O_2 required for rephosphorylation of creatine and ADP in a 70 kg person to be 1.5 L. Therefore,

most of the CP utilized during exercise is resynthesized during the early post-exercise phase and it is likely that the metabolic process only makes a minor contribution to total EPOC.

Studies which have utilized the muscle biopsy technique in order to measure muscle glycogen utilization during exercise and the subsequent time course of its resynthesis have shown that a substantial portion of intramuscular glycogen stores are depleted following brief, maximal exercise and prolonged moderate to high (55% to 70%) intensity exercise. Exercise performed at 70% VO_{2max} for 97.5 $\pm$ 6.6 min (Maehlum et al., 1977) and 89 $\pm$ 5 min (Maehlum & Hermansen, 1978) have resulted in 80% and 95% mean reduction in muscle glycogen stores. Lower intensity exercise (55%) performed for 60 min showed a 64% depletion (Essen et al., 1976) and stores were 44% lower following approximately 5 min of exhaustive exercise (Astrand et al., 1986). Percentage reductions in glycogen post-exercise will be influenced by resting muscle and liver glycogen concentrations, as well as the intensity and duration of the exercise. Resting levels can be altered by exercise training and diet, so that it is important to assess these factors when estimating exercise-induced glycogen depletion (Blom et al., 1986). The energy required to replenish glycogen stores following exercise is clearly dependent on the extent to which glycogen was utilized during the exercise bout.

Glycogen repletion follows a curvilinear time course. There is evidence that resynthesis starts almost immediately (Adofsson, 1972; Kowalchuk et al., 1988) and a 35% increase has been documented 1 h post-exercise (Astrand et al., 1986). The largest component of glycogen resynthesis occurs within the first 4 to 5 h (MacDougall et al., 1977; Maehlum & Hermansen, 1978; Maehlum et al., 1977; Piehl, 1974), although resynthesis continues for as long as 46 h following prolonged exercise (2 h) (Piehl, 1974) and 24 h after brief, exhaustive exercise (MacDougall et al., 1977). Rates of resynthesis vary with post-exercise diet and activity. However, significant elevations in glycogen stores were shown in fasting subjects 4 h after prolonged exercise (Maehlum and Hermansen, 1978), which indicates that even under unusual conditions, the first few hours post-exercise are a crucial period for repletion of muscle carbohydrate stores. If 1 L O_2 is consumed in the resynthesis of 6.8 mmol glycosyl units (Bahr et al., 1987), the O_2 cost of glycogen repletion during the first 4 to 5 h following prolonged exercise may be 4 to 6 L (Maehlum et al., 1977; Piehl, 1974). The relative significance of such an O_2 cost varies with the reported magnitudes of EPOC. However, the prolonged time frame of glycogen resynthesis suggests that it augments VO_2 for a lengthy period, so that its contribution to EPOC only attains signficance when the total magnitude of post-exercise VO_2 is obtained over a period of several hours.

2.5.5 Cardiorespiratory Work

Persistent elevations in ventilation (Katch et al., 1971; Welch et al., 1971) and heart rate (Bielinski et al., 1985; Maehlum et al., 1986) have been documented following exercise. It appears that ventilation is only elevated for several minutes post-exercise, whereas heart rate may remain elevated for several hours. When the energy costs of ventilation were estimated using Cournand's regression line of VO_2 per amount of ventilation (Shephard, 1966), the mean V_E cost after completion of 10 min of moderately heavy cycle ergometry (n=33) was found to be .53 L/min, or 11% of total VO_2 during a 15 min recovery period (Katch et al., 1972). It is likely that hyperventilation contributes to the early EPOC, but is not involved in prolonged elevations in baseline VO_2.

Myocardial O_2 costs do not appear to play a major role in EPOC. Recovery heart rates are highest during the first few minutes following exercise, yet the contribution of myocardial VO_2 to EPOC when VO_2 was measured for 15 min post-exercise was minimal (<3%) (Katch et al., 1971). Since myocardial O_2 demand only increases slightly with elevations in heart rate, even persistent elevations in post-exercise heart rate do not have a major impact on VO_2. Visual analysis of figures depicting post-exercise heart rate shows average long term increases of approximately 8 bpm (Bielinski et al., 1985; Mahlum et al., 1986). Since the myocardial VO_2 is only

increased by 2 ml O_2/min per 100 g left ventricle during light exercise (Messer & Neill, 1962), long term increases of a few beats per minute are not likely have a significant impact on EPOC.

2.5.6 Increased Lipid Oxidation

Several recent EPOC studies have documented declines in the respiratory exchange ratio (RER) and changes in plasma levels of glucose, insulin and free fatty acids indicative of an increased reliance on fats as opposed to carbohydrates lasting for several hours after exercise (Bahr et al., 1987; Bielinski et al., 1985; Chad & Quigley, 1989; Chad & Wenger, 1988; Maehlum et al., 1986). Less ATP is generated per litre of O_2 consumed from the oxidation of lipids, and it has been suggested that elevations in lipid utilization post-exercise may partially account for EPOC (Bahr et al., 1987; Chad & Wenger, 1988; Chad & Wenger, 1989). Recent evidence in support of this hypothesis has been provided by Chad and Wenger (1989). They showed that caffeine ingestion, which has been found to increase fat mobilization (Essig et al., 1980; Tarnopolsky et al., 1989), was associated with significant reductions in the RER and elevations in VO_2 for 60 min following exercise.

It is not possible to ascertain the precise magnitude of the declines in RER which have been reported as actual values are not provided (Bahr et al., 1987; Bielinski et al.,

1985; Chad & Wenger, 1988; Chad & Wenger, 1989; Maehlum et al., 1986). Reductions in RER appear to average approximately .05 for the various durations of EPOC. While VO_2 may be slightly increased in order to fulfill ATP requirements, it is unlikely that these elevations would account for more than a small fraction of the total EPOC. Maehlum and associates (1986) reported a total VO_2 of 185 $\pm$ 13 L over a 12 h control period. If post-exercise RER decreased by .05, for example, from .85 to .80, an additional 2.4 L O_2 would have had to be consumed over a 12 h period. Maehlum reported a 12 h EPOC of 26 L, so that the increased lipid oxidation may have accounted for about 1% to 2% of the total increase in oxygen consumption.

2.5.7 Temperature Effects

Body temperature has been cited as being a primary mediator of EPOC (Gaesser & Brooks, 1984; Hagberg et al., 1980). Elevations in temperature may contribute to EPOC by increasing energy requirements during exercise, or post-exercise VO_2 may be augmented directly by one or more temperature-related mechanisms. Temperature associated increases in VO_2 during prolonged, constant workload exercise have been found at normal (MacDougall et al., 1974; Saltin & Stenberg, 1964) and elevated (MacDougall et al., 1974) temperatures. When subjects performed the same exercise under different thermic conditions, the magnitude and rate of rise

in VO_2 was significantly greater in the hyperthermal condition. However, the attenuation of rectal temperature rise observed at four different work rates following eight weeks of endurance training did not correlate with reductions which occurred in the slow rise in exercise VO_2 (VO_2 drift) (r=.15) (Casaburi et al., 1987). Furthermore, a recent report found no differences in VO_2 during exercise in which body temperature was varied using water-perfused suits (Savard et al., 1988). In the latter study, the work-outs were performed in direct succession, so that results may have been confounded by the residual effects of the previous exercise condition.

The effect of exercise-induced hyperthermia on EPOC has not yet been established. There is no apparent relation between the duration of the post-exercise elevations of body temperature and VO_2 post-exercise. In some cases, body temperature has reached control levels several hours prior to VO_2 (Bahr et al., 1987; Chad & Wenger, 1988; Maehlum et al., 1986), while others have found that VO_2 normalized more rapidly (Brehm & Gutin, 1986). The reasons for the discrepancies among the time course of declines in temperatures is unknown; it may be related to differences among investigators' methods for determining baseline values or the temperature and/or relative humidity of the laboratories. Patterns of change also seem to differ between the two variables. Temperature has been shown to decline linearly following exercise; declines in VO_2 frequently follow

a curvilinear time course, characterized by an initial rapid phase and a subsequent slow phase (Brehm & Gutin, 1986; Chad & Wenger, 1988).

Significant correlations between tympanic or rectal temperature and post-exercise VO_2 have been reported. However, correlation coefficients have shown considerable inter-individual variation (.27-.78) (Brehm & Gutin, 1986), or have been markedly reduced by the manipulation of a second variable (.64-.75 was reduced to .04-.19 when the RER was held constant) (Chad & Wenger, 1988). Hagberg, whose 1980 study provided the original evidence in support of a temperature effect on EPOC in humans only examined the relation of temperature to the initial fast and slow phases of post-exercise declines in VO_2. Recovery VO_2 was plotted against a 'working' baseline, during which subjects exercised for 15 min at 150 $kpm \cdot min^{-1}$, and possible long-term elevations in BMR were not monitored.

No studies to date have attempted to establish the presence of a cause-effect relationship between body temperature and EPOC by measuring EPOC under different thermic conditions. In one case, it has been possible to compare the effects of small changes in body temperature following exercise performed at different intensities (approximately .2°C to .6°C difference between conditions) (Brehm & Gutin, 1986). Exercise intensity had a significant effect on both VO_2 and body temperature post-exercise, although the responses

of the two variables to changes in exercise intensity differed in magnitude and duration. It is likely that more dramatic differences in body temperature must be elicited before a potential effect of temperature on VO_2 post-exercise can be accurately assessed.

Several mechanisms have been postulated to account for observations of temperature-mediated increases in VO_2. In vivo studies have focused on the influences of hyperthermia on exercise VO_2. The extent to which these mechanisms are operant during the post-exercise phase is not known, but presumably this may be gauged by the longevity of the post-exercise hyperthermia. Elevations in temperature may increase post-exercise VO_2 in 3 ways. First, increases in peripheral flow and sweat gland activity occur at the commencement of exercise (Gisolfi & Bruce, 1984; MacDougall et al., 1974) and consequently, more energy is expended during the work bout. Post-exercise VO_2 may be enhanced by the energy cost of these heat-dissipating mechanisms, which remain operant until resting temperatures are restored.

Elevations in temperature may also impair the mechanical efficiency of skeletal muscle (Rowell et al., 1969), and augment exercise-induced elevations in minute ventilatory volumes (MacDougall et al., 1974).

There is evidence that hyperthermia increases post-exercise VO_2 by altering mitochondrial energetics. When mitochondria isolated from rat skeletal muscle were incubated

at temperatures ranging from 25°C to 45°C, respiration rates and ATPase activity increased significantly when temperatures exceeded 37°C (Brooks et al., 1971). The authors postulated that the enhanced ATPase activity was responsible for the elevations in respiration. Heightened ATPase activity, also known as the Q_{10} effect (Weibel, 1984), increases O_2 requirements because ATP is resynthesized at a more rapid rate secondary to accelerations in ATP hydrolysis and increased ADP concentrations. However, since Q_{10} values have not been firmly established in humans, it is not possible to calculate the VO_2 cost of a given increase in rectal temperature (Casaburi et al., 1987). Increases in ATP synthesis also augment total substrate depletion, so that more O_2 is required for the purposes of oxidative resynthesis.

An additional mechanism has been postulated by Brooks and associates (1971) who found that elevations in temperature reduced phosphorylative efficiency. Their conclusions were based on the increases in mitochondrial respiration rates (200%) measured under hyperthermic conditions; these rates were too high to accommodate equivalent increases in ATP resynthesis. The ADP:O_2 ratios were also significantly reduced when mitochondria were incubated above 40°C, indicating that greater amounts of O_2 were being consumed for the synthesis of a given amount of ATP.

2.5.8 Hormonal Effects

Exercise-induced increases in levels of circulating hormones may also augment post-exercise VO_2. Norepinephrine and epinephrine remained elevated for 10 to 20 min after short bouts of exercise (10 to 20 min) performed at three different intensities on separate occasions (50%, 70% and 90% VO_{2max}) (Deuster et al., 1989). The time to return to baseline corresponded with the re-establishment of baseline values for VO_2 and heart rates. Similarly, urine concentrations of norepinephrine were elevated 4 to 5 h post-exercise, which corresponded to the duration of the observed EPOC (Bielinski et al., 1985). However, interpretations of hormonal activity may be erroneous if they are based solely on plasma or urine levels. Changes in catecholamine concentrations must be considered in light of a number of factors, including rate of delivery to target cell, clearance rates, diurnal variations and density and activity of receptor sites (Frey et al., 1989; Sutton & Farrell, 1988).

The calorigenic effects of catecholamines have been demonstrated in animals (Chapler et al., 1980; Cain & Chapler, 1981) and humans (Fellows et al., 1985; Sjostrom et al., 1983). Skeletal muscle may become more sensitive to norepinephrine during the post-contractile phase. When norepinephrine was infused into canine gastrocnemius-plantaris muscle groups at rest, during contraction (1.0 Hz for 10 min) and during recovery, the most pronounced increase in VO_2 when compared to control conditions occurred during a 10 min

recovery phase (46.7±3.7 μL $O_2 \cdot g^{-1}$ vs 33.4±5.2 μL $O_2 g^{-1}$; p=.07) (Gladden et al., 1980). This was entirely the result of an increased O_2 extraction, since blood flow was reduced relative to the control period. The authors were unable to explain why VO_2 was particularly sensitive to the norepinephrine infusions during recovery from the contractile phase. They postulated a direct effect of norepinephrine on metabolic rate, or an elevated VO_2 secondary to norepinephrine-induced hypoxia during the preceding contractions. Results of infusion studies must be interpreted with caution, since infused and endogenous norepinephrine may have different physiological effects. During endogenous sympathetic stimulation, when norepinephrine acts primarily as a neurotransmitter, a steep gradient exists between the synaptic cleft and blood. This gradient is not present during infusion (Casaburi et al., 1987).

Epinephrine facilitates glycogenolysis in exercising muscles (Issekutz, 1985) and recent evidence obtained from studies of exercising rats shows that it stimulates glycogen breakdown in the inactive muscles as well (McDermott et al., 1987). Catecholamines may increase VO_2 by stimulating energy requiring processes in the cell. Norepinephrine increases cell permeability to Na+ and K+, which likely augments Na+-K+ pump activity and ATP demand (Horwitz, 1979). There is also evidence that plasma catecholamines increase VO_2 by promoting lipolysis, which is a less efficient energy source (ie. more

O_2 consumed per kilocalorie energy released) (Kalis et al., 1988).

Postprandial thermogenesis may be partially mediated by catecholamine activity; activation of the beta-adrenergic receptors appears to be a prerequisite for observations of an enhanced VO_2 following insulin infusions (DeFronzo et al., 1984; Acheson et al., 1984). It is thought that insulin increases energy expenditure after meals by accelerating rates of glucose disposal (Acheson et al., 1984; Proieto et al., 1983), so that exercise-induced increases in catecholamines may interact with the insulin activity. Poehlman and associates (1989a) have suggested that the declines in TEF observed in very fit individuals are related to a diminished sympathetic nervous system activity in this population. Their hypothesis is supported by findings of a significant increase in plasma norepinephrine in sedentary subjects after meal consumption, while hormonal levels were not altered in the trained men (LeBlanc et al., 1984). On the other hand, a recent report documents that the sympathetic response to exercise is similar among trained and untrained individuals working at equivalent relative exercise intensities (Deuster et al., 1989) and this may also apply to caloric intake; in other words, meals may have to be adjusted to subjects' body weight and composition.

Thyroxine has also been suggested as a possible mediator of EPOC (Gaesser & Brooks, 1984), since it acts as a

chronic stimulant of BMR. Thyroid hormones also enhance adrenergically mediated activities, and so might indirectly influence VO_2 (Goodman et al., 1988). However, few studies of EPOC have included measures of thyroxin. Maehlum and associates (1986) measured plasma levels of free T_4 with isotopic tracers in four subjects for 12 h after exercise completion and at corresponding times on a control day. They found no differences between the two days or between the exercise and post-exercise periods. It is unfortunate that they did not measure T_3 levels, since T_3 rather than T_4 is generally considered to be the most potent thyroid hormone (Goodman, 1988). Nonsignificant declines in T_3 levels were reported in 19 obese women immediately following a 40 min exercise bout and levels remained low until the end of the 90 min post-exercise measurement period (Krotkiewski et al., 1984). Similar reductions are also seen during periods of starvation or carbohydrate restriction, which suggests that exercise has the same effect on thyroid hormones as does a weight-reducing diet, and that chronic exercise may in fact lead to reductions in RMR via its effect on T_3.

Declines in T_3 levels have frequently been reported following exercise training (Mathieson et al., 1986; Phinney et al., 1988; Poehlman et al., 1986), although some have found no change, even when a training effect was demonstrated by significant reductions in subjects' resting HR (Caron et al., 1986). Yet the magnitude of the humoral reductions has not

corresponded to the extent of the declines measured in RMR. For example, Poehlman and associates (1986) reported a significant 33% reduction in T_3 with training whereas mean RMR declined only slightly (5.25±.3 ml/kg/min to 4.84±.5 ml/kg/min). When two groups of subjects adhered to isocaloric (530 kcal) high carbohydrate or low carbohydrate diets and to a controlled exercise program, the RMR of the high carbohydrate group declined relatively more while T_3 levels were more reduced in the low carbohydrate group (Mathieson et al., 1986). Therefore, it appears that T_3 is only one of several factors mediating RMR, and that exercise may in fact reduce T3 concentrations.

In summary, several mechanisms have been identified to account for EPOC. The significance of the contribution of each has not yet been determined and is likely affected by the characteristics of the exercise bout and the subject population.

2.6 CONCLUSION

It appears that exercise does induce sustained changes in several physiological parameters, and that these changes are reflected in an increased metabolic rate for a period of time after exercise. The precise duration and magnitude of the increase has not been established with any certainty. Reported magnitudes have varied between about 11 kcal and 174 kcal, over periods ranging from 10 min to 12 h. While some reports indicate that EPOC increases in magnitude and/or duration when exercise intensity or duration is augmented, findings are not consistent among studies.

It is likely that some of the inconsistencies among findings can be attributed to variations among the exercise performed and the sample population. However, the investigators' definitions of baseline and the length of the post-exercise measurement period may also explain some of the discrepancies. Further research is required in order to clarify the effect exercise characteristics and experimental design have had on measures of EPOC.

3 EXPERIMENTAL STUDY

3.1 HYPOTHESES

The present study was undertaken in order to test four sets of hypotheses. The first set pertains to the effect of exercise on the size and duration of EPOC. It is hypothesized that:

1. Exercise of varying durations and intensities is associated with a significant increase in post-exercise VO_2.
2. The use of a pre-determined recovery period augments the duration of EPOC by allowing for the inclusion of data which are slightly, but not significantly, higher than control data.

The effects of changes in exercise conditions on EPOC will also be examined. It is contended that:

1. Increases in exercise intensity with total work equated are associated with increases in the size and duration of EPOC; however the extent of these increases in EPOC may be small and of limited practical value for weight loss.
2. Increases in exercise duration at a constant, moderate exercise intensity augment the magnitude and duration of EPOC. The EPOC response may be curvilinear, so that a

marked increase occurs when exercise is performed for more than 60 min.

Possible mechanisms underlying the EPOC response will be investigated and the following sub-hypotheses will be examined:

a) Declines in Tc post-exercise are related to post-exercise declines in VO_2.

b) RER declines for a prolonged period following exercise and partially accounts for the post-exercise elevations in VO_2.

The data will also be analyzed in order to determine whether:

a) Five daily bouts of exercise have a cumulative effect on subjects' resting metabolic rate (RMR).

b) There is an interactive effect between food consumption and exercise on EPOC.

3.2 METHODS

Subjects

Ten male university students aged 23.4 ± 2.8 y (mean ± standard deviation) were recruited for the study. The subjects' descriptive data are presented in Table 2. Each student was informed of the procedures and potential risks of the study before consenting to participate. Eight subjects completed all study components; only their results are reported. The students were moderately active, exercising 3 to 4 times per week. They were all non-smokers, and had been weight-stable for at least 6 months prior to the study.

Testing Procedures

A continuous, incremental test on an electrically-braked cycle ergometer was initially administered to each subject to determine his VO_{2max}. The subjects inspired room air via a low-resistance breathing valve. Minute ventilation was recorded by a Hewlett-Packard 4000 VR digital pneumotachometer, and the expired air was measured for fractional O_2 and CO_2 using a Goddard Rapox oxygen analyzer and a Hewlett-Packard Capnometer (Model 47210A), respectively. All data were continuously recorded on a custom made computerized VO_2 monitoring system.

Table 2. Subject Characteristics

Subjects	VO2max (L/min)	VO2max ($ml \cdot kg^{-1} \cdot min^{-1}$)	weight (kg)	age (y)
TP	3.75	54.7	68.6	25
MG	4.78	62	77.2	23
LM	3.19	49	65	23
PM	4.18	43.4	96.2	22
NJ	3.71	51	72.7	20
JS	3.79	48.9	77.4	21
TB	3.05	56.6	53.9	24
CB	3.95	44.28	89.2	29
mean	3.8	51.2	75	23.4
SD	.5	6.3	13.4	2.8

The relationship between mechanical power and the VO_2 corresponding to 40%, 60% and 80% of each subject's VO_{2max} was determined during two to three subsequent testing sessions. The ACSM predictive equation for cycle ergometry was used to provide initial approximations of the power outputs which would elicit the appropriate VO2 for each subject (ACSM, 1986). During separate trials, a 5 min warm-up (10 W less than the power output predicted for 40% VO_{2max}) was completed, followed by a 5 min bout of cycling at the precalculated power output. VO_2 was monitored for 2 min durations at 5 min intervals, until a power output which elicited the precise percentage of the subject's VO_{2max} was determined.

Experimental Protocol

Subjects performed 5 exercise conditions on 5 consecutive days in random order (procedure for randomization performed using Table of Random Digits, Runyon & Haber, 1984, p. 451), and completed a 6th control day within 1 week of beginning or completing the five exercise conditions. No other exercise was performed at least 24 h prior to the 5 experimental days and the control day. The 5 exercise conditions are depicted in Figure 2. They included moderate intensity exercise (60% VO_{2max}) performed for 30, 60 and 90 min, high intensity exercise (80% VO_{2max}) and low intensity exercise (40% VO_{2max}). The durations of the high

Figure 2. The 5 exercise bouts performed by each subject.

Exercise Intensity ($\%\ VO_{2max}$)			Duration (min)	
80%				
60%	x	30 min	60 min	90 min
40%				

and low intensity exercise were calculated so that total energy expenditure was equivalent to the total work performed during the 30 min moderate intensity exercise bout. Each exercise bout also included a 5 min warm-up at 10 W below the power output corresponding to 40% of subjects' VO_{2max}.

Exercise intensities corresponding to 40%, 60% and 80% VO_{2max} were selected for 2 reasons. First, they span the range of intensities which are typically performed by healthy adults who wish to maintain or increase their functional capacity. Exercise intensities corresponding to 80% and 60% VO_{2max} represent the upper and lower ranges of the levels recommended to achieve a training effect (ACSM, 1986), while walking is performed at approximately 40% of an individual's VO2max and is frequently prescribed as an effective means of weight reduction. Second, the 3 exercise intensities were separated by a difference of 20% VO_{2max} in an effort to discriminate between potential differential effects of intensity on the EPOC.

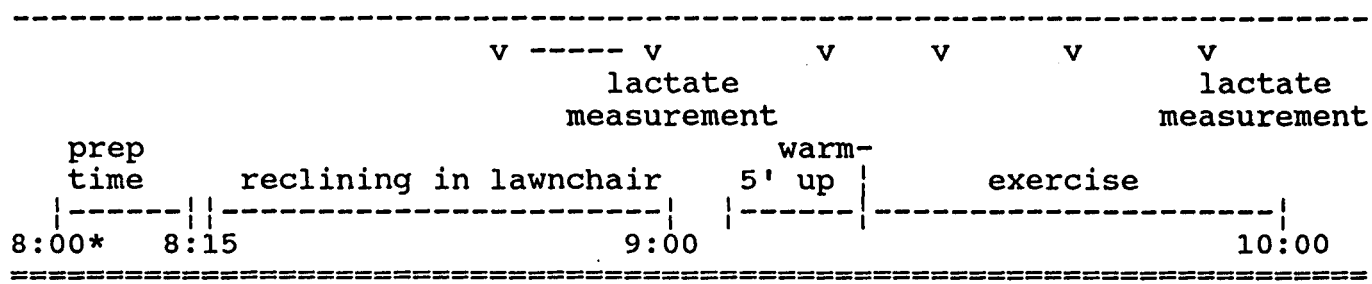
VO_2 Measures

Figure 3 depicts the daily schedule and the sampling times for VO_2 measurement throughout the experiment. Each subject reported to the lab at the same time on each of the 6 study days. Following a 30 min rest in a reclined position, resting VO_2 (pre-exercise) was measured

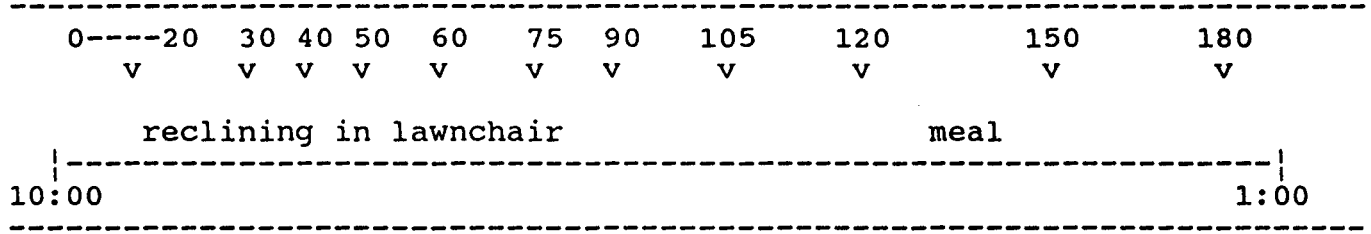
Figure 3. The daily protocol for each exercise condition.

v VO2 sampling periods
* Subjects reported to the lab at the same time on each of the six study days, but starting times differed among the participants. The clock times given represent a typical schedule.

Pre-exercise and Exercise Components



Post-exercise Component



continuously for 15 min. Subsequent to this, 1 of the 5 exercise conditions was performed. Two minute expired gas samples were obtained during the last 2 min of the warm-up phase, and at regular intervals during the exercise period, including 7 to 10 min after onset of exercise and prior to exercise completion, so that the data would reflect any progressive elevations in VO_2 occurring throughout the exercise (Camus et al., 1988).

One and a half minutes before the exercise bout was completed, VO_2 was again continuously monitored until the first 20 min of the recovery phase had elapsed. This period was chosen for continuous sampling since recovery VO_2 declines most rapidly during this time period (Hagburg et al., 1980; Knuttgen, 1970). In order to minimize the effects of movement on recovery VO_2, each participant remained seated on the cycle ergometer throughout the 20 min sampling time.

Two minute samples of expired gas were collected every 10 min for the remainder of the first hour post-exercise, every 15 min during the second hour, and every 30 min during the third hour. Except for the first 20 min of the recovery phase, subjects reclined in a lawn chair and read, wrote, or watched movies during the pre- and post-exercise components of the study days. Heart rates were recorded simultaneously with the oxygen uptake measures.

The magnitude of EPOC was determined by summing VO_2 values obtained during the 3 h post-exercise period of each

experimental day and during the corresponding control period. Oxygen consumption during the non-measurement intervals was interpolated from the measured values which bracketed these intervals. Measures of total EPOC were converted to kilocalories (kcal/L O_2 equivalents for subjects' RER were obtained from McArdle, 1986, Table 8-1) because of the implications of the data for weight control. The results will be expressed in L O_2 or kcal.

Core Temperature Measures

Tc was measured with a rectal thermister, inserted to a depth of 10 cm past the anal sphincter and worn continuously throughout each study day. Temperatures were recorded from a Scanning Tele-thermometer (YSI Model 47) at the end of the 15 min pre-exercise VO_2 sampling period, and each time that exercise and post-exercise VO_2 samples were collected. In order to control for the possible effects of ambient temperature and humidity on recovery VO_2, the entire study was conducted in a Hotpack climate chamber (Model 9153-2, Waterloo, Ontario). Room temperature was held constant at 21° C and relative humidity was 10%.

Lactate Measures and Ratings of Perceived Exertion

In order to determine whether subjects exceeded their lactate threshold during the high intensity exercise bout, capillary blood samples were collected from a fingertip pre- and post-exercise, and analyzed for lactate concentrations. Blood lactate was measured on a YSI 23L Lactate Analyzer. The standard blood lactate concentration of 4 $mmol \cdot L^{-1}$ was used as the index of lactate threshold, according to the method of Karlsson and Jacobs (1982).

Subjects' perceptions of the difficulty of each exercise bout were assessed using the Borg scale, which each subject completed immediately following exercise (Borg, 1982).

Diet

Subjects were instructed to record times and quantities of food and beverage consumption for 12 h preceding the first study day. They were subsequently instructed to adhere to precisely the same diet 12 h prior to all the ensuing study days. Each subject was provided with a meal 2 h following exercise, after a VO_2 measurement, and completed the meal prior to the 150 min post-exercise sampling time. The meal consisted of submarine sandwiches and orange juice; type and quantity was determined by the subject and was held constant throughout the experiment. Meal composition was similar among all subjects; subjects varied with respect to

the total quantity of food ingested rather than the type of food consumed.

In order to ensure that subjects' energy balance at the onset of the recovery period would be equivalent on all 6 d, subjects consumed a nutritional supplement (Ensure, Ross Laboratories, Montreal, Quebec, 15.2% protein, 32% fat, 52.8% carbohydrate) 3 h prior to arriving to the lab. The quantity of the Ensure was adjusted to correspond to the caloric requirements of that day's exercise bout. The timing of the Ensure consumption was based upon an estimated 4 h maximal absorption period (Fink et al., 1983), so that it would provide a partial fuel source during the exercise bout.

Statistical Analysis

The effects of intensity and duration of exercise on recovery VO2 (duration and magnitude), HR, RER and Tc were determined using a two factor (exercise condition by time of sampling) repeated measures analysis of variance for each of the dependent variables. The 5 exercise conditions allowed for the comparison of 3 exercise durations at a constant intensity and 3 different intensities of exercise with total work equated. The effects of exercise intensity and duration were analyzed in separate analyses of variance. The 30 min bout of moderate intensity exercise was included in both analyses, as were the values obtained on the control day, which were used as the baseline measures. Due to the

limitations of the computer program utilized for the statistical tests of significance, only 10 of the 33 sampling times were included in the analyses. The sampling times chosen represent the periods during which a given dependent measure was undergoing the most rapid rate of change. A Tukey post hoc comparisons were made in order to test for differences between exercise and control conditions at a given sampling period. In some cases, it was appropriate to do additional paired t-tests. (See Results Section.) The level of statistical significance was set at $p < .05$.

Values for missing data cells (a total of 19 cells for all subjects and all dependent measures) were calculated using regression equations derived from the data available for individual subjects during the specific exercise condition. Equations were set to include quadratic and linear components, representing the rapid and slow recovery phases.

3.3 RESULTS

Changes in subjects' blood lactate concentrations and their ratings of perceived exertion in response to the 5 exercise conditions are shown in Tables 3 and 4,

Table 3. Blood Lactate Concentrations ($mmol.L^{-1}$) Before and After Exercise[a]

	80% VO2max x 21.2±1.3 min		60% VO2max x 90 min		x 60 min		x 30 min		40% VO2max x 56.3±11.6 min	
	pre	post	pre	post	pre	post	pre	post	pre	post
TP	.9	4.3	2.4	3.2	.8	3.4	1.9	3.1	*	*
MG	.5	5.9	1.3	1.3	1.3	2	1.1	1.3	1.6	1.1
LM	*	*	1.3	2.7	1.1	.9	1.2	1.8	.9	.9
PM	1.1	4.2	1.6	1.2	*	*	1.2	2	1.1	1.1
NJ	1.8	4.3	*	*	1.9	3.8	2.8	3.4	*	*
JS	1.9	4.6	*	*	1.7	1.9	1.1	1.2	1.6	1.5
TB	2.4	5	1.9	1.5	2.6	1.9	2	2.5	1.9	1.4
CB	2.7	7.2	2.7	3.2	1.8	1.4	1.7	2.9	2.5	1.9
mean	1.6	5.1	1.9	2.2	1.6	2.2	1.6	2.3	1.6	1.3
SD	.8	1.1	.6	.9	.6	1	.6	.8	.6	.4

* Lactate measures not obtained.

[a] Blood lactate threshold = 4 $mmol.L^{-1}$ (See Karlson J and Jacobs I. Onset of blood lactate accumulation during exercise as a threshold concept: Theoretical considerations. Int. J. Sports Med. 3:190-210, 1982.)

Table 4. Ratings of Perceived Exertion Following Exercise

	80% VO2max x 21.2±1.3 min	60% VO2max x 90 min	x 60 min	x 30 min	40% VO2max x 46.3±11.6 min
TP	18	16	14	13	11
MG	17	13	12	12	10
LM	18	11	11	11	11
PM	20	18	16	15	9
NJ	16	16	15	12	13
JS	17	19	15	12	8
TB	18	16	12	13	10
CB	17	18	14	13	12
mean	17.6	15.9	13.6	12.6	10.5
SD	1.2	2.7	1.8	1.2	1.6

respectively. Blood lactate measures obtained for all subjects only exceeded the threshold value (4 $mmol.l^{-1}$) following the high intensity exercise condition, and were not increased in response to changes in duration of moderate intensity exercise. Mean Borg ratings were different following each exercise condition and showed that the perceived difficulty of the exercise bouts increased with exercise intensity, and with exercise duration at a given intensity. For example, mean Borg ratings were higher following 30 min of 60% VO_{2max} exercise than after 56.3 min of 40% VO_{2max} exercise, while maximal ratings were obtained after only 21.2 min of exercise performed at 80% VO_{2max}.

A summary of the significance level of the main effects and interactions of all the variables tested is given in Table 5. Unless otherwise indicated, reference will only be made to statistically significant findings.

Resting Metabolic Rate

Pre-exercise measures of VO_2, RER and Tc were not different among the 6 study days.

Duration of EPOC

Effect of exercise intensity. The decline in VO2 during the first 30 min which followed the 3 work bouts of varying intensity (when rate of change was most rapid), as well as over the entire 3 h post-exercise measurement period

Table 5. Statistical Significance of Differences Obtained Between Experimental and Control Measures For All the Dependent Variables Analyzed (F ratio & p value):

Dependent Measure	Exercise Characteristic	Main Effects		Interaction Effects
		Type of Exercise	Time	Ex x Time
VO_2	Intensity[1]	F=224.85 p<.001	F=258.96 p<.001	F=147.76 p<.001
	Duration[2]	F=76.94 p<.001	F=240.71 p<.001	F=76.88 p<.001
VO_2 Cumulative	Intensity	F=13.34 p<.001	F=383.98 p<.001	F=2.15 p=.002
	Duration	F=12.09 p<.001	F=350.4 p<.001	F=2.86 p<.001
RER	Intensity	F=2.63 p=.076(NS)	F=3.39 p=.002	F=1.83 p=.01
	Duration	F=1.04 p=.396(NS)	F=6.91 p<.001	F=1.1 p=.344(NS)
Body Temperature	Intensity	F=36.32 p<.001	F=60.3 p<.001	F=20.65 p<.001
	Duration	F=34.01 p<.001	F=46.68 p<.001	F=18.22 p<.001
Heart Rate	Intensity	F=134.82 p<.001	F=221.2 p<.001	F=76.81 p<.001
	Duration	F=88.81 p<.001	F=167.14 p<.001	F=58.22 p<.001
TEF*:Effect on VO_2	Intensity	F=.55 p=NS	F=12.82 p<.001	F=1.21 p=.318(NS)
	Duration	F=.5 p=NS	F=11.42 p=.001	F=.93 p=NS
TEF:Effect on Temp	Intensity	F=3.08 p=.049	F=7.78 p=.005	F=1.71 p=.141(NS)
	Duration	F=2.7 p=.07(NS)	F=7.84 p=.005	F=1.71 p=NS

*TEF - thermal effect of feeding

[1] Refers to analysis of the three exercise bouts performed in which intensity was varied and total work kept equivalent.

[2] Refers to analysis of the three exercise bouts performed in which duration was varied and exercise intensity kept constant.

are depicted in Figure 4 (a) and (b), respectively. VO2 levels among the 3 intensities were significantly different from each other immediately post-exercise. Recovery VO2 returned to baseline between 10 and 16 min following high intensity exercise, 16 to 20 min after moderate intensity exercise, and within the first 6 min after low intensity exercise.

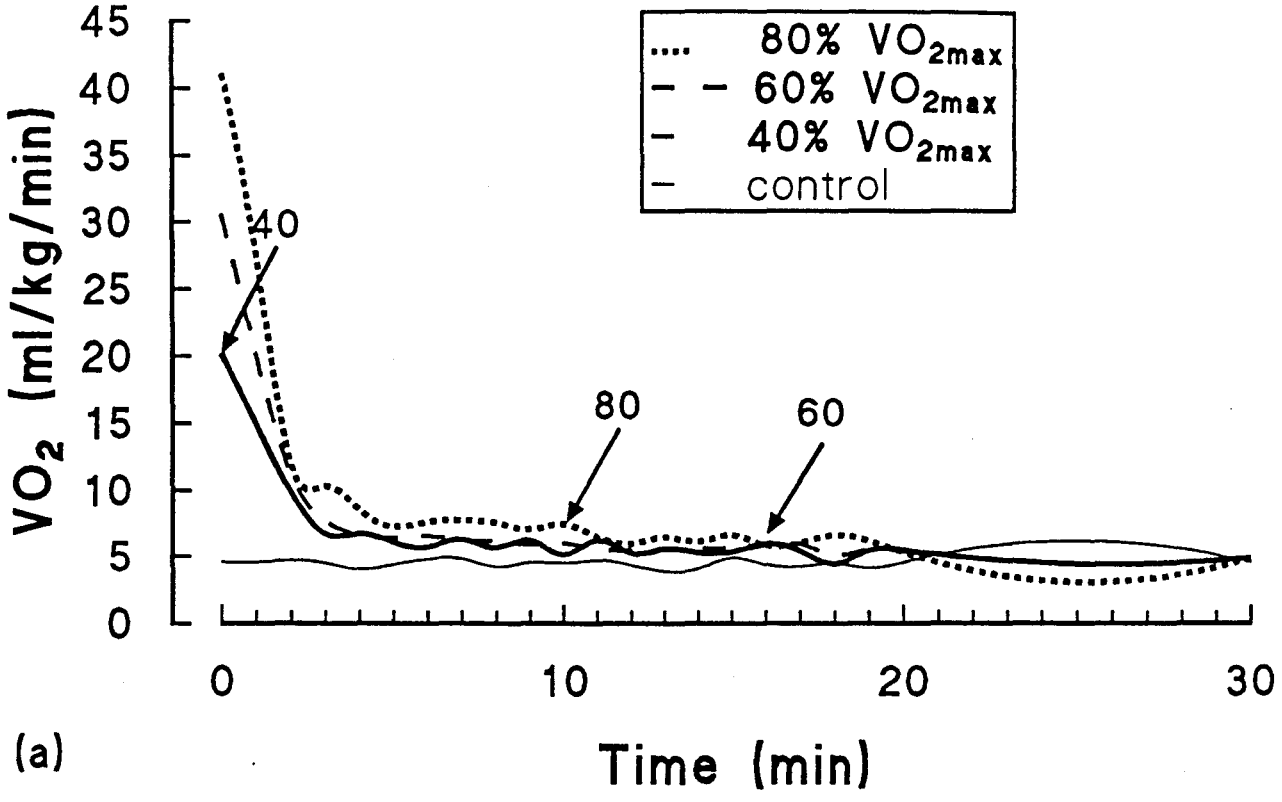
Although the analysis of variance indicated that VO_2 measures obtained between 30 and 180 min post-exercise were equivalent to control values, visual analysis of the raw data showed slight elevations in VO_2 which persisted throughtout the 3 h measurement period. When the data obtained 30 to 180 min following exercise were collapsed across exercise conditions, mean post-exercise VO2 exceeded control measures (4.87 $\pm$ .7 ml/kg/min vs 4.57 $\pm$ .9 ml/kg/min, respectively). The mean VO_2 was further increased when separate calculations were made for data obtained following high intensity exercise (5.01 $\pm$ .7 ml/kg/min). Differences were also observed when the mean values recorded 180 min after exercise completion were collapsed across the 3 exercise conditions and compared to the corresponding control values (5.48 $\pm$ 1.0 ml/kg/min vs 4.86 $\pm$ .8 ml/kg/min, respectively). In order to confirm that these elevations in post-exercise VO_2 were not statistically significant, paired t-tests were performed in addition to the analysis of variance. The results of the

Figure 4 (a). Changes in mean VO_2 for 30 min following exercise performed at varying intensities with total work equated and on the control day (n=8). VO_2 attains baseline levels between 10 and 16 min after high intensity exercise, 16 to 20 min after moderate intensity exercise, and within the first 6 min after low intensity exercise.

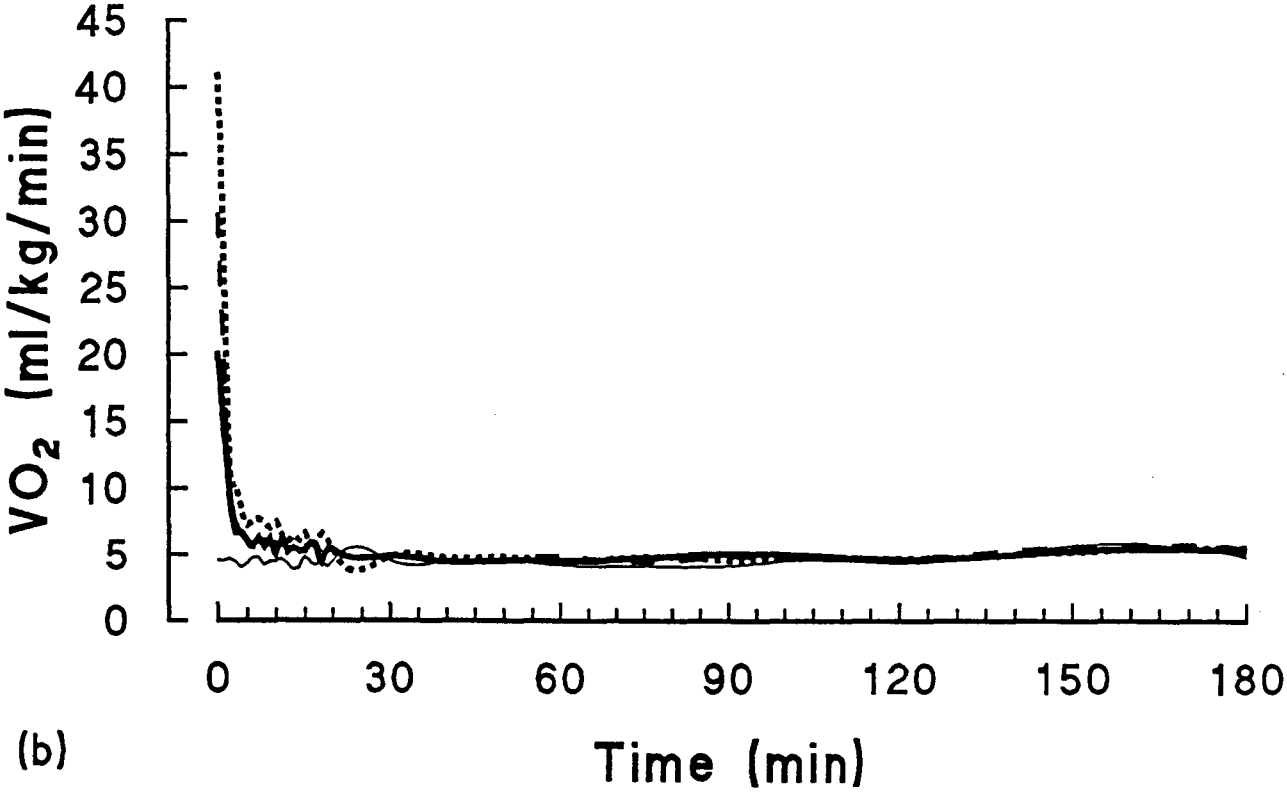
↓ denote the last VO_2 sampling time in which significant differences were found between control and exercise measures, and are labelled with nos. corresponding to the exercise intensity they represent.

Figure 4 (b). Depicts the same exercise conditions as those shown in Figure 4 (a). Changes in mean VO_2 for the entire 180 min post-exercise measurement period are presented.

The following VO_2 measurement periods were included in the statistical analysis examining effects of exercise intensity and exercise duration (see Figure 5) on post-exercise VO_2: pre-exercise period, and 0, 6, 10, 16, 20, 30, 60, 120 and 180 min post-exercise.


80% VO2max
60% VO2max
40% VO2max
control
40
80
60
VO2 (ml/kg/min)
0
5
10
15
20
25
30
35
40
45
0
10
20
30
Time (min)

(a)


VO2 (ml/kg/min)
0
5
10
15
20
25
30
35
40
45
0
30
60
90
120
150
180
Time (min)

(b)

paired t-tests supported those obtained from the analysis of variance; in neither set of comparisons were differences found to be significant ($p > .05$).

Effect of exercise duration. Figure 5 (a) and (b) depict changes in VO_2 following the 3 experimental days in which moderate intensity exercise was performed for 3 different durations. VO2 returned to baseline 6 to 10 min following the 30 min bout and 20 to 30 min following the 60 min exercise bout. VO_2 levels normalized between 10 and 16 min after the 90 min exercise, and were again significantly elevated between 20 and 30 min post-exercise. Oxygen uptake did not vary among the 3 exercise durations at exercise completion.

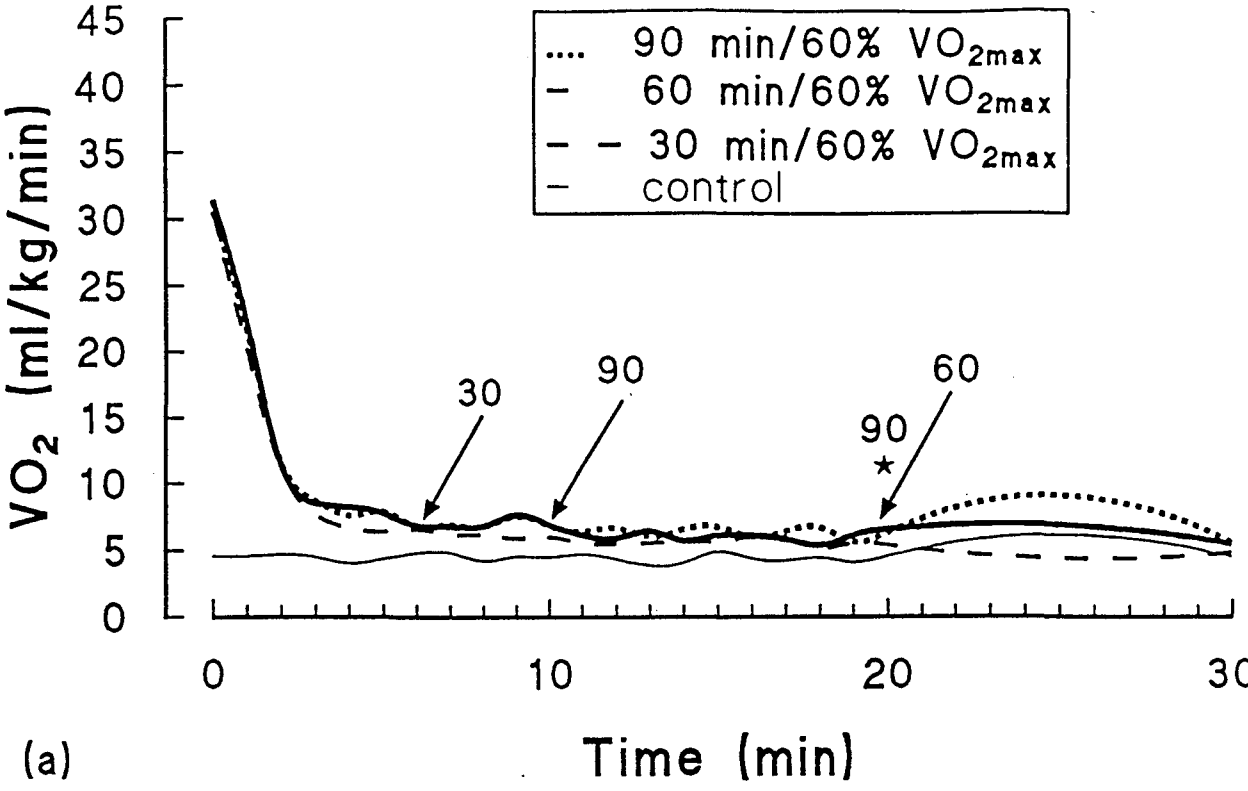
Slight elevations in VO_2 were also noted during the 30 to 180 min following the exercise of varied duration. When the data obtained from 30 to 180 min post-exercise for each subject were collapsed across exercise conditions, the mean value was greater than the control value (4.99 $\pm$ .5 ml/kg/min vs 4.56 $\pm$ .9, respectively). The difference between values was increased when the data compared were confined to the 180 min post-exercise time period (5.39 $\pm$ .7 ml/min/kg vs 4.86 $\pm$ .8 ml/min/kg, respectively). In order to ensure that these differences were not statistically significant, paired t-tests were performed. Results paralleled those obtained following exercise of varied

Figure 5 (a). Changes in mean VO_2 for 30 min after moderate intensity exercise of varying durations and on the control day (n=8). VO_2 reaches control levels 10 to 16 min after the 90 min exercise bout and is elevated again between 20 and 30 min post-exercise. Control values are attained 20 to 30 min after the 60 min exercise and 6 to 10 min after the 30 min exercise bout.

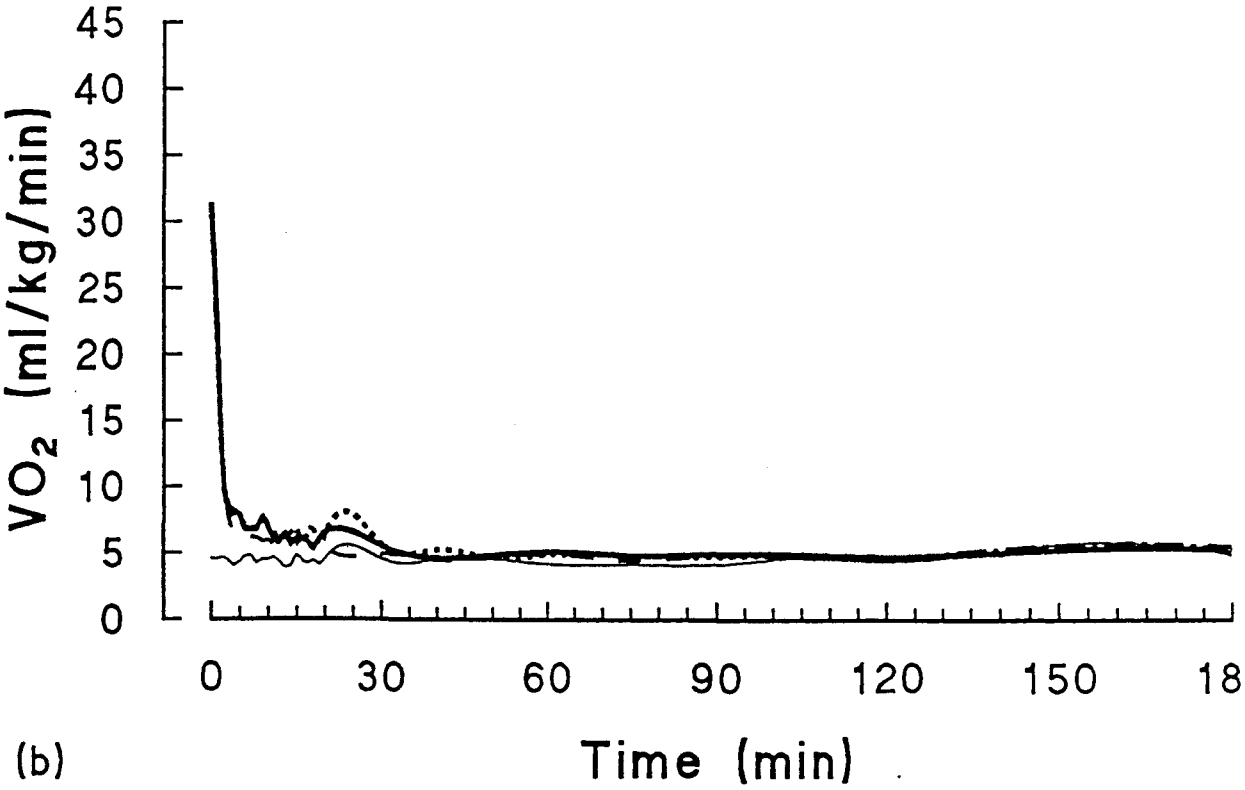
↓ indicate last VO_2 sampling time in which significant differences were found between experimental and control values.
★ denotes last sampling time when a significant difference was obtained for the 90 min exercise after values had returned to normal for a period of time.
Symbols are labelled with nos. corresponding to the exercise duration they represent.

Figure 5 (b). Depicts the same exercise conditions as those shown in Figure 5 (a). Changes in mean VO_2 for the entire 180 min post-exercise measurement period are presented.

See Figure 4 for list of VO_2 measurement periods included in statistical analysis performed.


.... 90 min/60% VO2max
– 60 min/60% VO2max
– – 30 min/60% VO2max
– control
VO2 (ml/kg/min)
45
40
35
30
25
20
15
10
5
0
30
90
90
60
0
10
20
30
Time (min)

(a)


VO2 (ml/kg/min)
45
40
35
30
25
20
15
10
5
0
0
30
60
90
120
150
Time (min)

(b)

intensity. The analysis of variance was confirmed by the t-tests; the slight elevations in VO_2 observed did not attain a statistical significance ($p > .05$).

Magnitude of EPOC

Effect of exercise intensity. The magnitude of VO_2 (cumulative VO_2, expressed in L) recorded during the 3 h period following the 3 exercise bouts of varying intensity exceeded control values (Table 6). Total energy expenditure after high intensity exercise was significantly greater than that obtained after moderate or low intensity exercise. This was reflected in the values obtained when the mean total EPOC for the eight subjects was calculated as a percentage of EOC. Values were highest for the high intensity exercise (17.8%) and similar for the moderate and low intensity conditions (10.9% and 9.3% respectively). However, exercise was associated with little or no increase in net caloric expenditure in several subjects, as indicated by the large standard deviations presented in Table 6.

In Figure 6 (a) the magnitude of VO2 following exercise of varying exercise intensity is expressed as a percentage of the magnitude of the control VO_2, in order to show the rate of decline of total post-exercise VO_2. Immediately post-exercise, VO_2 magnitudes were 273%, 218% and 186% of control values for high, moderate and low intensity exercise respectively. These values had declined

Table 6. Caloric Expenditure and Oxygen Consumption During and Following Exercise

Intensity of Exercise (% VO_{2max})	Duration of Exercise (min)	Energy Expended During Exercise+ (kcal)	Net Energy Expended During 3 h Following Exercise (kcal)	Oxygen Consumed During Exercise (L O_2)	Net Oxygen Consumed During 3 h Following Exercise (L O_2)
80%	21.2 ± 1.3	325.1 ± 39.2	57.9 ± 72.20	64 ± 4.9	11.7 ± 14.6[a] [b]
60%	90	1067.2 ± 146.1	56 ± 57.3	210.2 ± 4.3	11.3 ± 11.6[a] [c]
60%	60	685.6 ± 87.9	51.3 ± 62.3	136 ± 3.9	10.4 ± 12.6[a]
60%	30	343.1 ± 53.4	37.4 ± 53.7	68 ± 4.7	7.51 ± 10.8[a]
40%	46.3 ± 11.6	351.3 ± 54.7	32.5 ± 42.3	70.1 ± 3.2	6.6 ± 8.6[a]

Values represent means ± standard deviations of 8 subjects.

\+ Caloric expenditure was determined by multiplying the VO_2 L/ the kcal/L equivalent associated with the RER (McArdle et al., 1986).

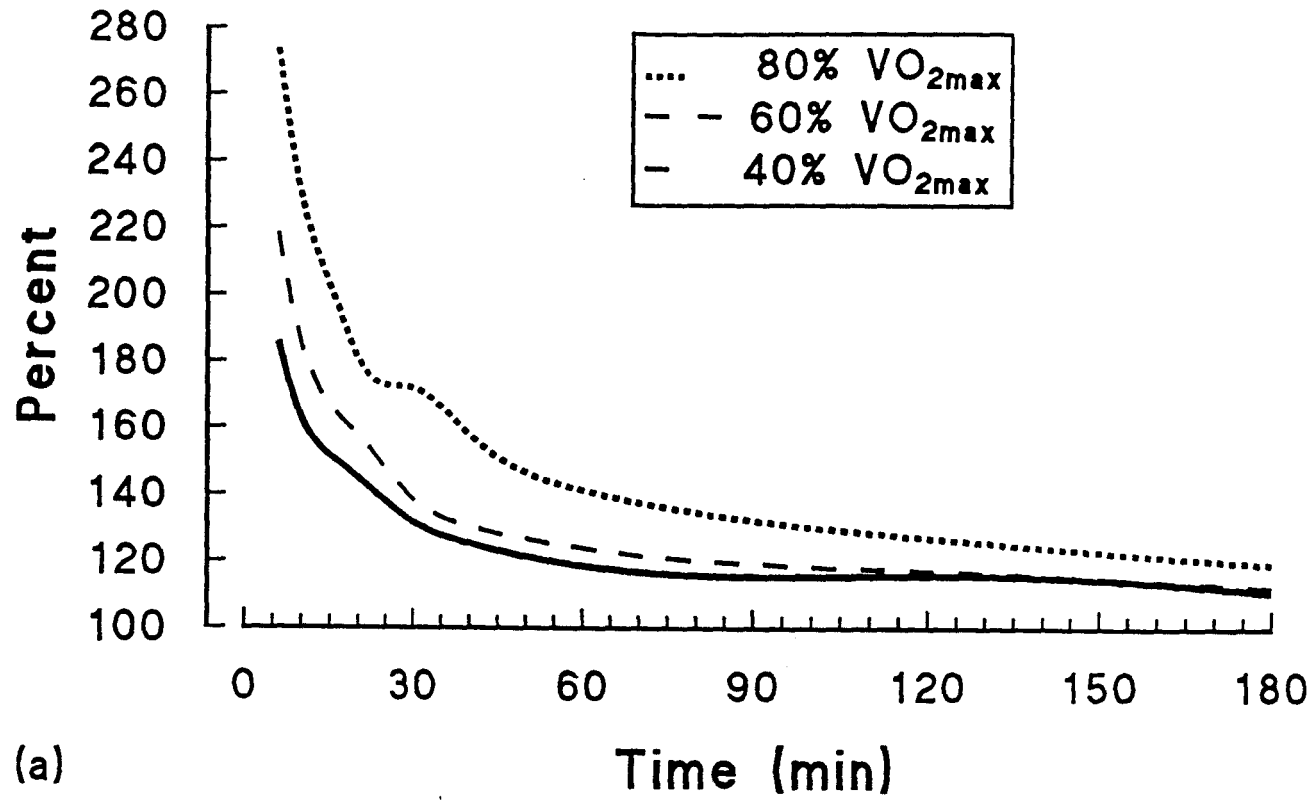
[a] Significantly greater than control value ($p < .05$).

[b] Significantly greater than 40% VO_{2max} and 60% VO_{2max}/30 min conditions ($p < .05$).

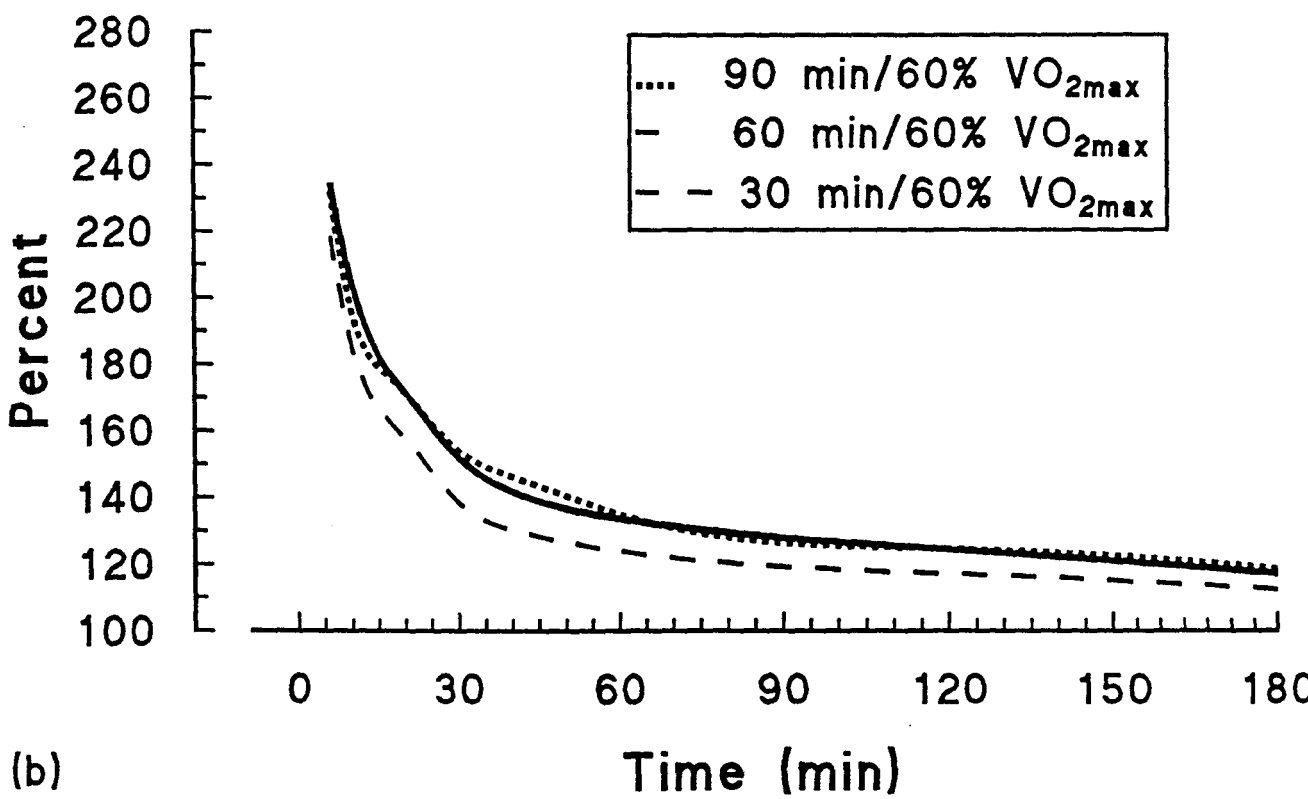
[c] Significantly greater than 60% VO_{2max}/30 min condition ($p < .05$).

Figure 6 (a). Relation of magnitude of VO_2 following exercise with intensity varied and total work equated to magnitude of control VO_2, expressed as a percentage. Values represent means of eight subjects. Values are reduced by 101%, 80% and 54% 30 min post-exercise for the high, moderate and low intensity exercise bouts respectively.

Figure 6 (b). Relation of magnitude of VO_2 following moderate intensity exercise performed for 3 different durations. Values represent means of eight subjects. Thirty minutes after exercise completion, values are 78%, 83% and 80% lower for the 90 min, 60 min and 30 min work bouts respectively.


280
260
240
220
200
180
160
140
120
100
Percent
.... 80% VO2max
– – 60% VO2max
– 40% VO2max
0
30
60
90
120
150
180
Time (min)

(a)


280
260
240
220
200
180
160
140
120
100
Percent
.... 90 min/60% VO2max
– 60 min/60% VO2max
– – 30 min/60% VO2max
0
30
60
90
120
150
180
Time (min)

(b)

by 101%, 80% and 54% 30 min post-exercise and continued to decline at a slower rate throughout the measurement period. This indicates that the greatest contribution of EPOC to the total caloric expenditure during the 3 h post-exercise period occurred within the first 1/2 h.

Figure 6 (a) shows that the total magnitude of post-exercise VO_2 was greater than control VO_2 throughout the 3 h recovery period.

Effect of exercise duration. The magnitude of the EPOC measured following 90 and 60 min of exercise at 60% VO_{2max} was significantly greater than that obtained after the 30 min exercise condition 3 h post-exercise, although marked variations in response were again noted among subjects (Table 6). However the percentage relation of mean EPOC to EOC was negatively related to exercise duration: 5.3% for the 90 min exercise, 7.5% for the 60 min exercise and 10.9% for the 30 min bout.

Figure 6 (b) shows the percent decline in the magnitude of post-exercise VO2 relative to control values. A pattern similar to that obtained following exercise of varied intensities was seen; the sharpest decline in values (78%, 83% and 80% for 90 min, 60 min and 30 min exercise respectively) occurred during the first 1/2 h.

Respiratory Exchange Ratio

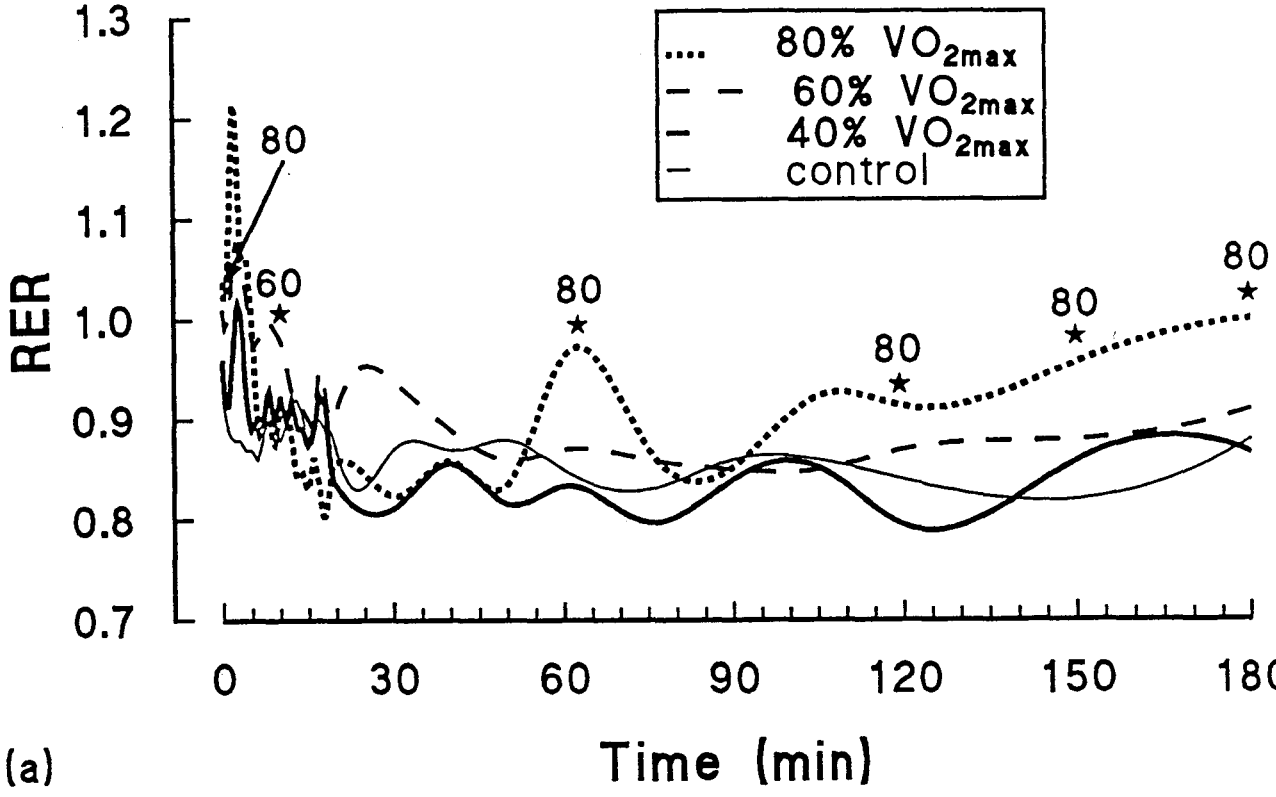
Effect of exercise intensity. Figure 7 (a) depicts the values obtained for RER following exercise of varying intensities with total work equated. A significant effect was obtained for time ($p<.002$) and for the time by exercise condition interaction ($p<.01$), but no significant effect for condition was obtained (Table 3.4). The high intensity exercise was associated with intermittent elevations in RER during the 3 h recovery period (immediately, 60 min, 150 min and 180 min post-exercise), and values were significantly higher than those obtained following low intensity exercise at all times sampled during the last hour of recovery. RER exceeded control values 10 min following the moderate intensity exercise, but was equivalent to control levels immedately post-exercise and during every other measurement period. RER following low intensity exercise did not differ from baseline values at any point during the 3 h measurement period. Values did not fall below baseline after any exercise condition.

Figure 7 (a). Mean RER (n=8) after exercise of varying intensity with total work equated. Significant increases in RER were found immediately, 60, 120, 150 and 180 min following high intensity exercise and 10 min after moderate intensity exercise.

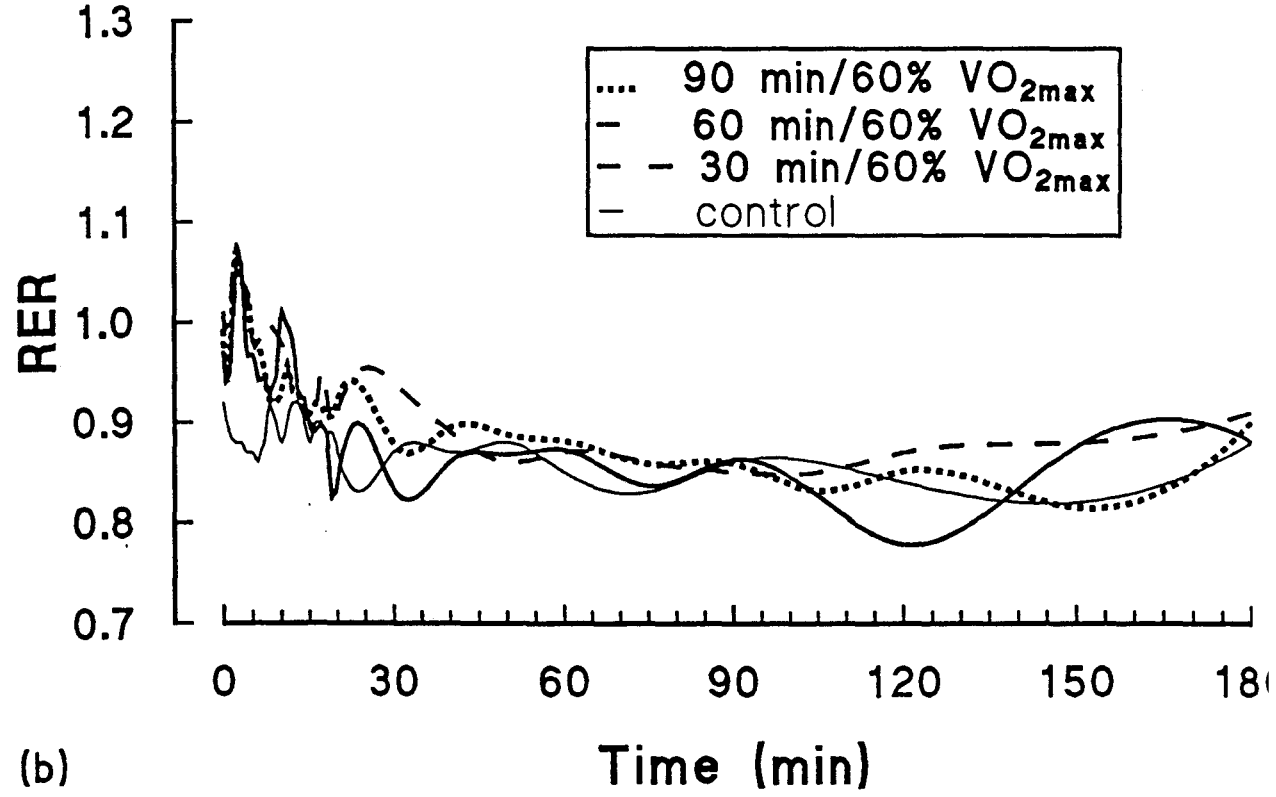
↓ denotes the significant elevation found immediately following high intensity exercise.
★ indicates the sampling times when significant differences were obtained after values had temporarily normalized.
Symbols are labelled with nos. corresponding to the exercise intensity they represent.

Figure 7 (b). Mean RER (n=8) after moderate intensity exercise of varying durations. Values are equivalent to baseline within approximately 20 min.

The following RER measurement periods were included in the statistical analysis of the effect of exercise on post-exercise RER: pre-exercise measures, and 0, 10, 20, 30, 40, 60, 120, 150 and 180 min post-exercise.


80% VO_{2max}
60% VO_{2max}
40% VO_{2max}
control
RER
1.3
1.2
1.1
1.0
0.9
0.8
0.7
80
60
80
80
80
80
0
30
60
90
120
150
Time (min)

(a)


90 min/60% VO_{2max}
60 min/60% VO_{2max}
30 min/60% VO_{2max}
control
RER
1.3
1.2
1.1
1.0
0.9
0.8
0.7
0
30
60
90
120
150
Time (min)

(b)

Effect of exercise duration. Figure 7 (b) illustrates the values obtained for the RER following exercise of varying durations at the same intensity. Elevations in RER were recorded immediately after exercise, but were equivalent to baseline levels within approximately 20 min.

Body Temperature

Effect of exercise intensity. Figure 8 (a) shows the post-exercise decline in Tc towards control values in response to differing exercise intensities. Values obtained following moderate intensity exercise exceeded control levels for 120 to 150 min, while Tc after completion of high intensity exercise remained elevated for 60 min to 120 min. Measures of Tc following low intensity exercise remained elevated until 30 to 40 min post-exercise. Tc was significantly lower following low intensity exercise than following the other 2 experimental conditions at all measurement periods during the first hour and 3 h post-exercise, but was not significantly lower during the intervening sampling periods.

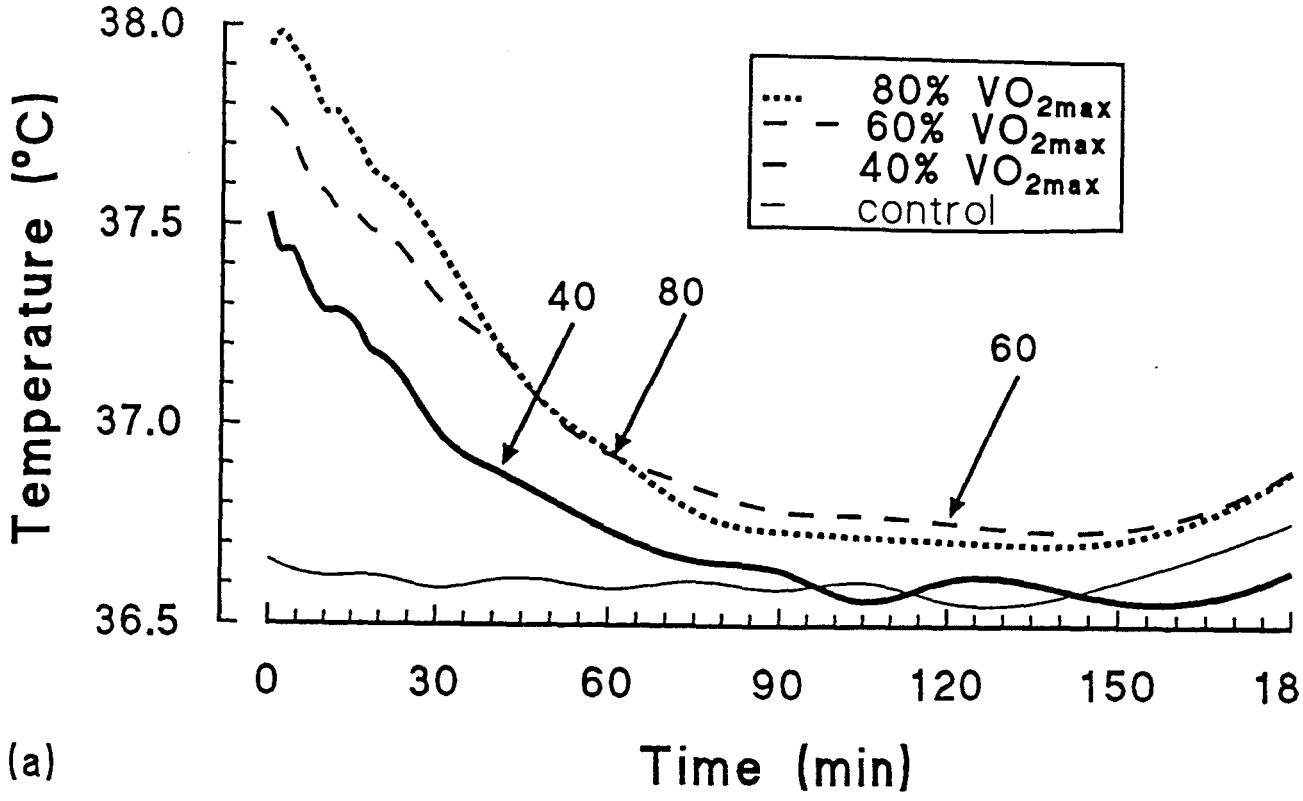
Effect of exercise duration. Body temperatures recorded following moderate intensity exercise are shown in Figure 8 (b). Temperatures were significantly higher than control values 2 h after cessation of exercise, and had

Figure 8 (a). Mean Tc (n=8) following high, moderate and mild intensity exercise and on control day. Tc reaches control levels between 60 and 120 min after high intensity exercise, 120 to 150 min after moderate intensity exercise and 40 to 60 min after mild intensity exercise.

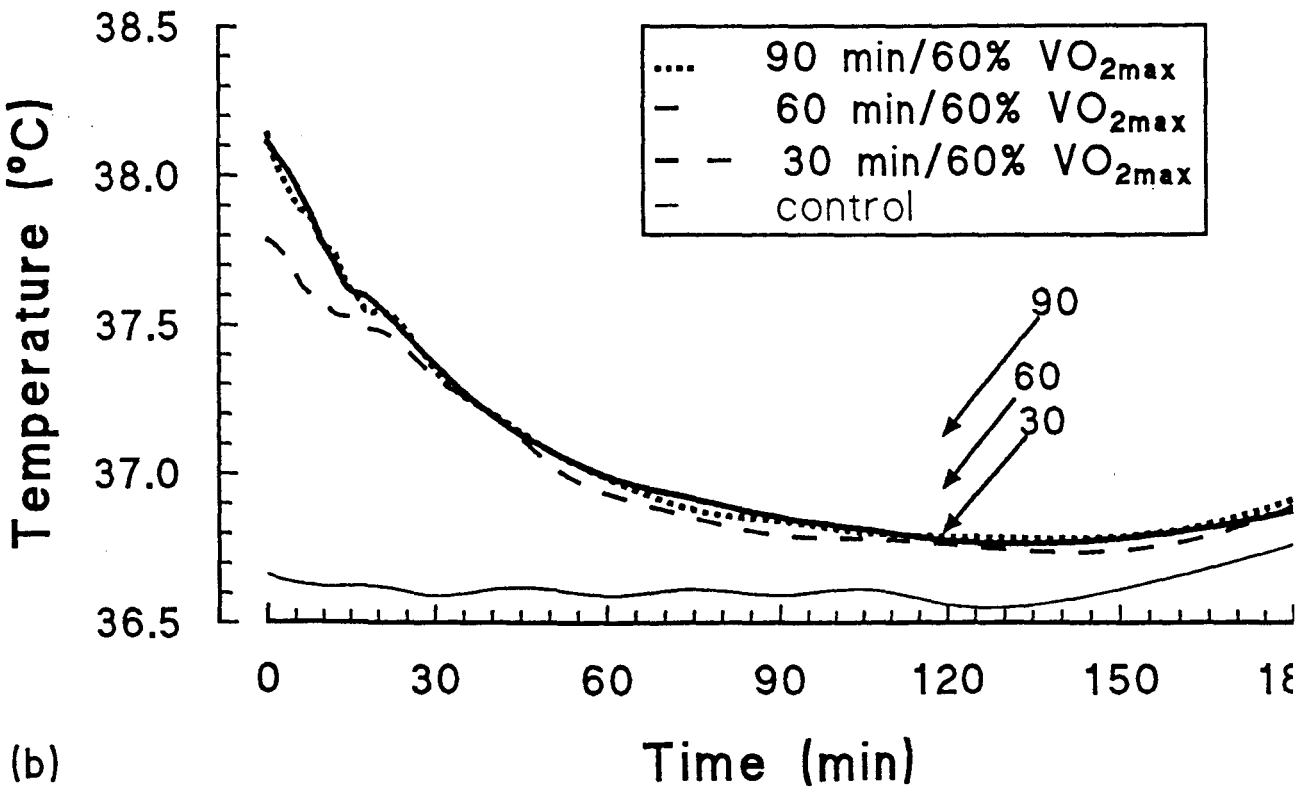
Figure 8 (b). Mean Tc (n=8) following 90 min, 60 min and 30 min moderate intensity exercise. Tc is significantly higher than control values 2 h after cessation of all three exercise bouts and has returned to baseline levels by 150 min post-exercise.

↓ denotes the last sampling period in which significant differences were found between control and experimental values, and are labelled with nos. corresponding to the exercise intensity or duration they represent.

The following Tc measurement periods were included in the statistical analysis of the effect of exercise on post-exercise Tc: pre-exercise period, and 0, 10, 20, 30, 40, 60, 120, 150 and 180 min post-exercise.


38.0
37.5
37.0
36.5
Temperature (°C)
80% VO2max
60% VO2max
40% VO2max
control
40
80
60
0
30
60
90
120
150
18
Time (min)

(a)


38.5
38.0
37.5
37.0
36.5
Temperature (°C)
90 min/60% VO2max
60 min/60% VO2max
30 min/60% VO2max
control
90
60
30
0
30
60
90
120
150
18
Time (min)

(b)

returned to baseline 2 1/2 h post-exercise. Significant differences among the 3 exercise conditions were only obtained immediately post-exercise (90 & 60 min condition > 30 min conditions) and 10 min post-exercise (90 and 60 min conditions > 30 min condition).

Heart Rate

Effect of exercise intensity. Pre-exercise values were equivalent on the three days in which exercise intensity was compared and on the control day. Figure 9 (a) shows the change in HR following exercise of varying intensity. HR had returned to baseline 2 1/2 h after high and moderate intensity exercise. HR exceeded baseline levels 20 to 30 min after low intensity exercise and significant elevations were also obtained 120 and 180 min post-exercise, although values were equivalent to control levels 150 min post-exercise.

Effect of exercise duration. Resting values obtained prior to the 30 min exercise bout and on the control day (64 $\pm$ 7 and 63 $\pm$ 9 beats per minute) differed from the resting measures obtained on the 60 and 90 min exercise days (70 $\pm$ 12 and 69 $\pm$ 11 beats per minute). Following all three bouts of exercise, HR exceeded baseline at all sampling times for 2 h. HR began to increase 150 min following the two longer bouts of exercise, and was

significantly higher than control levels 180 min post-exercise (See Figure 9 (b).) At all time periods sampled post-exercise, the differences between experimental and control values were greater than the differences obtained at rest, which suggests that the post-exercise elevations were a function of the exercise performed.

Cumulative Effect of Exercise on RMR

Figure 10 illustrates the means and standard errors of subjects' RMR values on exercise days 1 to 5, consecutively. Differences among values were not found to be significant when the data were analyzed using a one-way analysis of variance ($F = 2.31$, $p = .082$).

Thermal Effect of Feeding

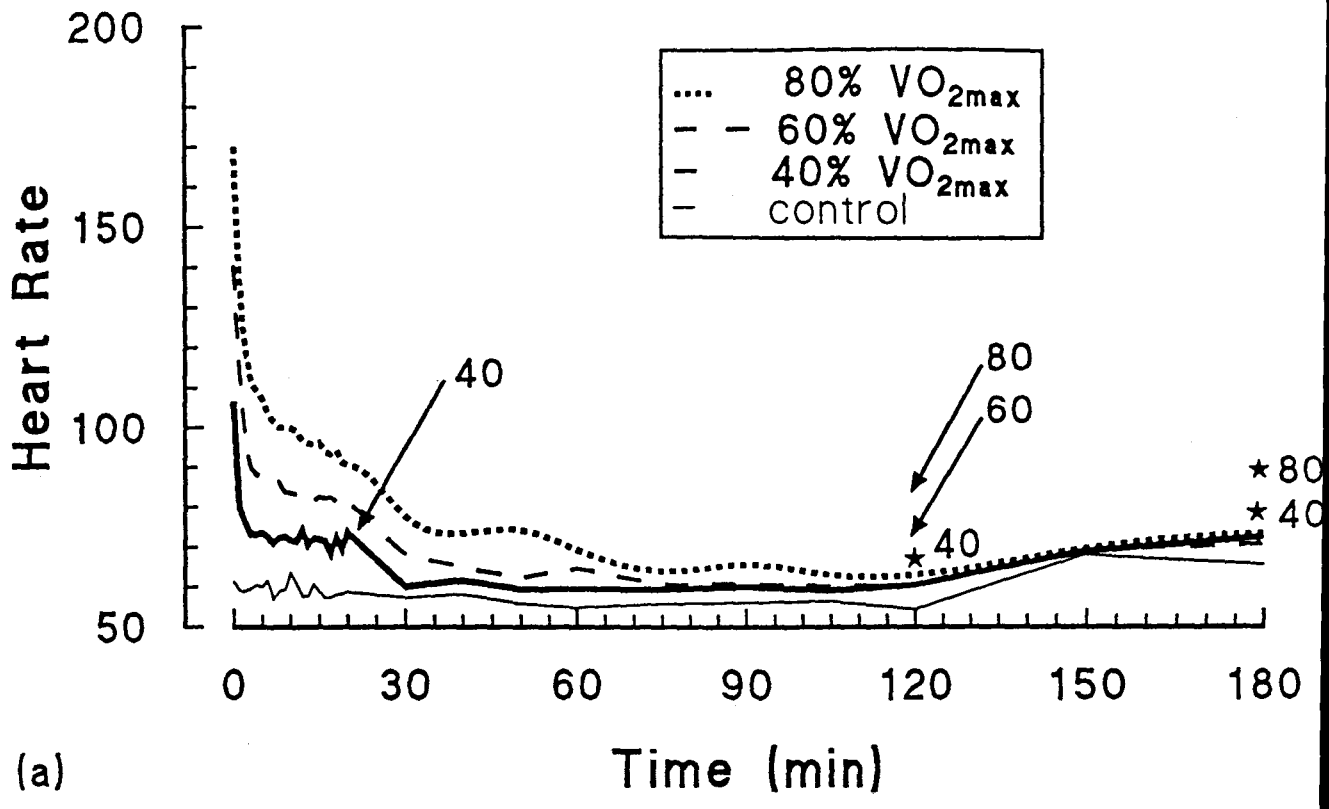
Subjects ate a meal immediately following the 120 min post-exercise sampling period. They were given until the 150 min post-exercise sampling period to complete the meal. The VO_2 and Tc measures obtained 120 min, 150 min and 180 min post-exercise and the corresponding control data were analyzed for a possible interaction between meal consumption and exercise. VO_2 values immediately post-meal ingestion were consistently higher than those obtained just prior to meal ingestion (120 min post-exercise) (see Figures 4 and 5), whereas increases in Tc were only observed approximately 30 min after completion of the meal

Figure 9 (a). Mean HR (n=8) following exercise of varying intensity with total work equated and on control day. HR returns to baseline between 120 and 150 min following high and moderate intensity exercise. Levels begin to climb again 150 min after high intensity exercise and are significantly elevated by 180 min post-exercise. HR attains control levels between 20 and 30 min after low intensity exercise and values are also signficantly higher than control levels 120 and 180 min post-exercise.

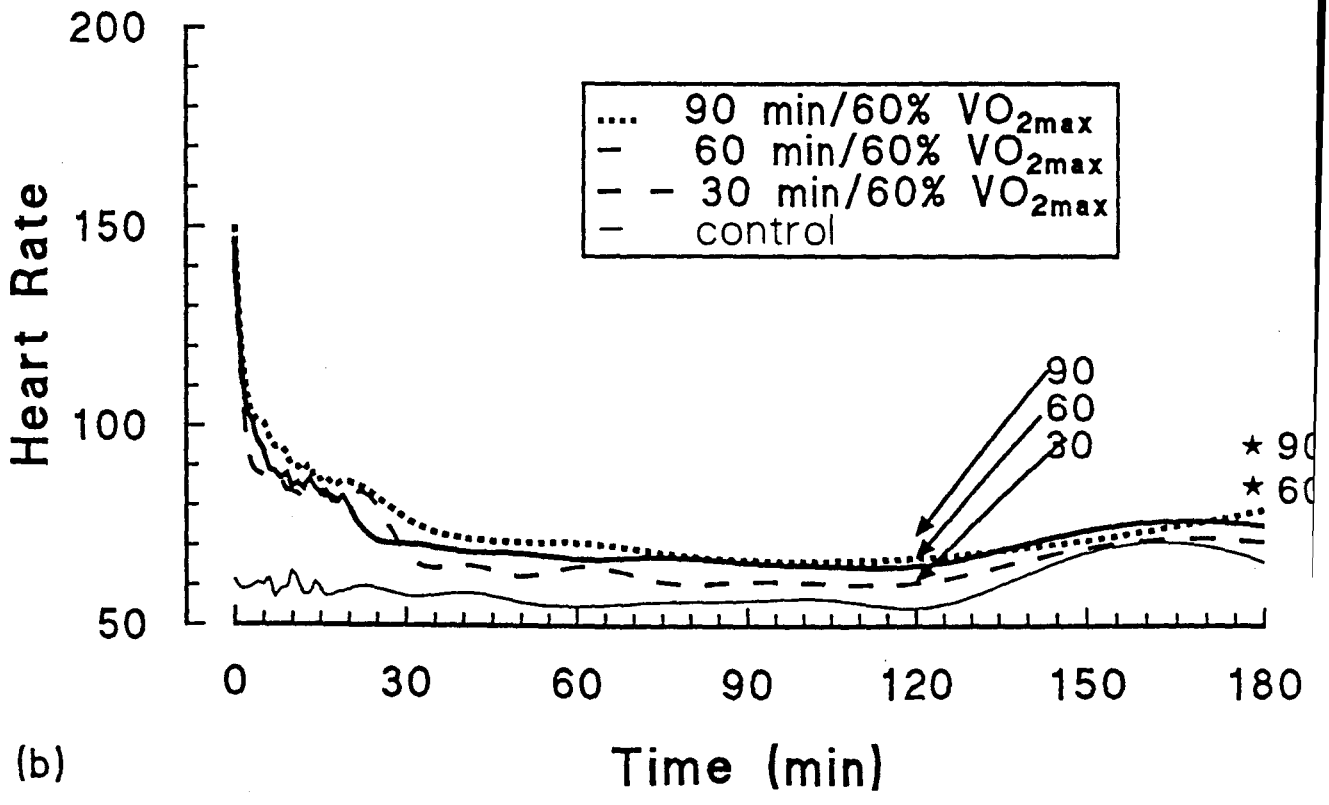
Figure 9 (b). Mean HR (n=8) following moderate intensity exercise with duration varied. HR exceeds baseline for 2 h after all three exercise bouts. Control levels are attained between 120 and 150 min after all exercise conditions. HR rises again during the 150 to 180 min interval following the two longer exercise durations and is significantly elevated by 180 min post-exercise.

↓ denotes last sampling time in which experimental measures are significantly different from control measures.
★ represents significant elevations obtained after values had returned to baseline for a period of time.
Symbols are labelled with nos. corresponding to the exercise intensity or duration they represent.

The following HR measurement periods were included in the statistical analysis of the effect of exercise on post-exercise HR: pre-exercise period, and 0, 10, 20, 30, 40, 60, 120, 150 and 180 min post-exercise.

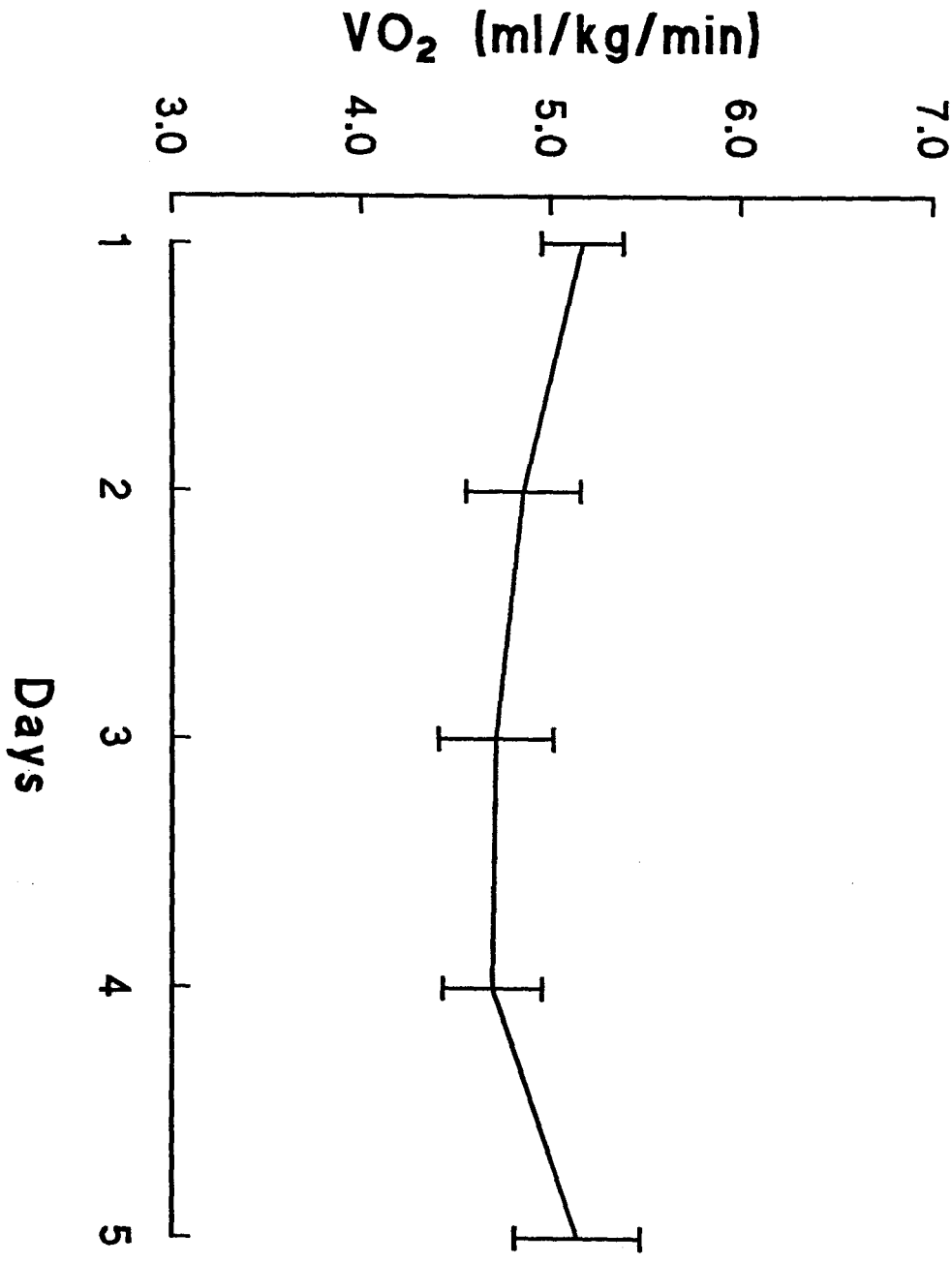

200
150
100
50
Heart Rate
.... 80% VO2max
– – 60% VO2max
– 40% VO2max
– control
40
80
60
40
★80
★40
0
30
60
90
120
150
180
Time (min)

(a)


200
150
100
50
Heart Rate
.... 90 min/60% VO2max
– 60 min/60% VO2max
– – 30 min/60% VO2max
– control
90
60
30
★90
★60
0
30
60
90
120
150
180
Time (min)

(b)

Figure 10. Means and SE of RMR values on exercise days 1 to 5 (n=8). No statistically significant differences were found among values obtained on the 5 d.


VO2 (ml/kg/min)
3.0
4.0
5.0
6.0
7.0
Days
1
2
3
4
5

(180 min post-exercise) (see Figure 8). The temperature values on the control day and after exercise performed at 40% VO_{2max} were signficantly lower than the values obtained following the 2 higher intensity exercise conditions. However, there were no significant interaction effects between VO_2 or Tc and any exercise condition. This suggests that the meal-induced thermogenesis was not affected by exercise performance.

4 DISCUSSION

The present study has shown that the mean total caloric expenditure during recovery from the 5 different exercise conditions was significantly greater than that obtained during the corresponding control period. However, total excess energy expenditure was small, ranging from 32.5 to 57.9 kcal and for some individual subjects, exercise caused no increment in the total post-exercise VO_2. The duration of EPOC was also brief, with VO_2 restored to baseline within 30 min of every exercise bout. A number of investigators have found similarly low values for total EPOC (Bielinski et al., 1985; Brehm & Gutin, 1986; Elliot et al., 1988; Frechette et al., 1987; Kaminsky et al., 1987) and for the duration of EPOC (10 to 40 min) (Elliot et al., 1988; Freedman-Akabas et al.,

1984; Kaminsky et al., 1987; Pacy et al., 1985; Sedlock et al., 1989).

Recently, exercise bouts similar to the ones performed in the present study have resulted in an EPOC 2 to 3 times greater in magnitude and duration (Bahr et al., 1987; Bielinski et al., 1985; Chad & Wenger, 1988; Maehlum et al, 1986). In the present study, mean VO_2 obtained during the 30 to 180 min post-exercise period was collapsed across exercise conditions and compared to control values. Although no statistically signficant differences were found, experimental values tended to be nonsignficantly higher than control values. It is possible that a similar phenomenon occurred in previous studies which have reported a large EPOC of several hours duration. Slight elevations in VO_2 may persist for long periods of time without exceeding control values by a statistically significant amount. It may be, therefore, that the substantial total EPOC reported in several recent studies can be attributed to the use of a pre-established recovery period (4.5 to 12 h) (Bahr et al., 1987; Bielinski et al., 1985; Maehlum et al., 1986). This method of analysis may augment estimations of the length and size of EPOC, since determinations of EPOC may include periods during which experimental and control VO_2 were not statistically different from one another.

Chad and Wenger (1988) also reported a large EPOC following exercise performed for varying durations at 70%

VO_{2max}, and they did not utilize a pre-determined recovery period. However, no control day was included in their study, and diurnal variations in subjects' VO_2 may have been responsible for the large EPOC reported. Oxygen consumption tends to increase throughout the day and peak during the late afternoon, which corresponds to the VO_2 measurement times utilized in Chad and Wenger's study (see review by Winget et al., 1985).

In the present study, the magnitude of post-exercise VO_2 exceeded control VO_2 throughout the 3 h measurement period (Figure 6), despite the fact that duration of EPOC was less than 30 min in all cases. Two factors are likely responsible for this finding: 1) The slight, although nonsignificant, elevations in VO_2 which were observed at all sampling times post-exercise would have augmented total VO_2. 2) Measures of total magnitude of VO_2 incorporate the values obtained during the early post-exercise phase, when VO_2 measures are considerably higher than control values. In the present study, significant effects on EPOC were obtained for exercise duration and intensity, although their influence on total EPOC was negligible in terms of energy output (Table 6). Mean EPOC was significantly greater following high intensity exercise than either moderate or low intensity exercise (57.9 versus 37.4 and 32.5 kcal). This difference was observed despite the fact that recovery time was greatest following the 60% VO_{2max} exercise, indicating that exercise intensity

influences EPOC within the first few minutes post-exercise. The present results concur with other recent findings that a threshold exercise intensity must be attained before intensity affects the magnitude of the EPOC. Brehm and Gutin (1986) found no differences in magnitude of EPOC following 50% VO_{2max} and 33% VO_{max} exercise, while in the present study EPOC was equivalent following the 40% and 60% VO_{2max} work bouts.

Mean caloric expenditure during the 3 h following the 2 longer exercise bouts exceeded that obtained after the 30 min exercise (56 and 51.3 versus 37.4 kcal). Again, it appears that a threshold exists above which increases in exercise duration have no further effect on EPOC. A threshold for exercise duration has not been previously documented; however, this is the first study to date in which 2 prolonged exercise bouts $\geq$ 60 min were compared. Others have documented either no effect of duration on total EPOC after exercise performed at 80% HR_{max} (Elliot et al., 1988) and 50% VO_{2max} (Sedlock et al., 1989) or a considerably greater duration effect following 70% VO_{2max} exercise (Bahr et al., 1987; Chad & Wenger, 1985; Chad & Wenger, 1988). A recent study found that duration only affected EPOC when exercise intensity was $\geq$ 50% VO_{2max} (Gore & Withers, 1990). The findings of the latter study suggest that discrepancies among results may be related to choice of exercise intensity.

Several mechanisms have been postulated to account for EPOC, including the O_2 cost of creatine phosphate (CP) and

glycogen resynthesis, temperature-related disturbances of mitochondrial respiration and elevations in plasma concentrations of catecholamines. It has been estimated that a maximum of 1.5 L of O_2 may be consumed for CP resynthesis when exercise induces a complete hydrolysis of the high-energy phosphates in muscle (Brooks et al., 1971). Since CP stores would only have been marginally depleted following the submaximal exercise bouts performed in the present study (Brooks & Fahey, 1984), the O_2 cost of CP repletion likely accounted for a negligible portion of EPOC. Moreover, most of the CP resynthesis would have occured during the first few minutes post-exercise (Bangsbo et al., 1989; Harris et al., 1976).

Repletion of muscle glycogen stores may have accounted for a part of EPOC. Glycogen utilization during the 5 exercise bouts was 104.3 $\pm$ 57.3 g (mean $\pm$ SD) (proportion of carbohydrates derived from muscle glycogen estimated to be .95 during 80% VO_{2max} exercise and .8 for all other work bouts; grams of carbohydrates utilized per L O_2 consumed obtained from McArdle et al., 1986; Table 8-1). Assuming that 1 L O_2 is consumed for the resynthesis of 135 mmol glycogen from blood glucose (Bahr et al., 1987), glycogen resynthesis would have accounted for approximately 4.3 $\pm$ 2.4 L O_2 of total post-exercise VO_2. Since repletion of glycogen stores may occur over a 24 h (MacDougall et al., 1977) to 48 h (Piehl, 1973) period, it is likely that only a fraction of this quantity of

O_2 was consumed for the purposes of glycogen resynthesis during the 3 h post-exercise measurement period. Total EPOC following each of the five exercise bouts was not closely associated with the amounts of glycogen utilized during the exercise. For example, total EPOC was greatest following the high intensity exercise. However, considerably less glycogen was depleted during the high intensity exercise than during the 90 min moderate intensity exercise bout (approximately 75 g and 200 g, respectively).

Although a relation between the decline in rectal temperature and VO2 following exercise has been reported (Hagburg, 1980), most investigators have found no relation between the recovery rates of the 2 variables (Bahr et al., 1987; Maehlum et al., 1986). Chad and Wenger (1988) did not find a correlation between Tc and VO_2 post-exercise when the relation between RER and VO_2 was taken into account ($r = .04$ to .19). A high inter-subject variability in correlation coefficients between VO_2 and Tc (.27 to .78) has also been found (Brehm & Gutin, 1986).

It is likely that Tc is loosely associated with post-exercise VO_2, but is not the sole determinant of duration and magnitude of EPOC. Tc following low intensity exercise was significantly lower than values obtained after the moderate and high intensity exercise bouts, and Tc returned to control levels more rapidly (40 to 60 min). This is consistent with the short duration of EPOC (less than 6 min) found following

low intensity exercise. Tc in the present study normalized long after VO_2 levels; it was still significantly elevated 2 h after each moderate intensity exercise bout and 1 h after the high intensity exercise, and values tended to remain higher throughout recovery. It is not clear why previous reports have found that Tc returned to control levels prior to VO_2 (Chad & Wenger, 1988; Bahr et al., 1987; Maehlum et al., 1986); room air was maintained at a constant temperature and low relative humidity during the present study and therefore variations in environmental factors cannot account for the observed slow rate of return of Tc.

Elevations in plasma levels of catecholamines may be a major determinant of EPOC (Sedlock et al., 1989). Circulating levels of epinephrine and norepinephrine increase during low, moderate and high-intensity exercise (Sutton & Farrell, 1988). They were recently shown to return to baseline levels within 10 to 20 min after 50%, 70% and 90% VO_{2max} exercise (Deuster et al., 1989; Tarnopolsky et al., 1990), which corresponds to the brief durations of EPOC observed in the present and afore-cited studies. Catecholamines may elevate VO_2 by increasing energy-requiring processes in the cell, such as Na^+-K^+ pump activity (Gaesser & Brooks, 1984).

Increases in exercise duration may facilitate lipolysis and free fatty acid mobilization post-exercise (Chad & Wenger, 1988). Since more O_2 is required for the combustion

of lipids than for proteins or carbohydrates, such a metabolic shift may augment VO_2 (Bahr et al., 1987; Chad & Wenger, 1988). Previous investigators have reported progressive declines in post-exercise RER values (Bahr et al., 1987; Bielinski et al., 1985; Chad & Wenger, 1988; Chad & Quigley, 1989; Maehlum et al., 1986), although not all declines have reached significance (Chad & Quigley, 1989). No such pattern was observed following any exercise condition in the present study. RER returned to baseline within approximately 20 min after exercise completion, and did not fall significantly below control levels at any time sampled. It may be that subjects were not followed for long enough after exercise. Bahr and colleagues (1987) did not observe reductions in RER until 7 h after exercise completion. High intensity exercise was associated with signficant elevations in RER 60, 120, 150 and 180 min post-exercise. Elevations in catecholamine secretion rates following high intensity exercise may have enhanced glycogenolysis and elevated blood glucose levels, leading to a preferential reliance on carbohydrate oxidation following exercise (Sutton & Farrell, 1988).

Five daily bouts of exercise did not have a cumulative effect on RMR. Bahr and coworkers (1987) found RMR to be equivalent to control values 24 h after a single bout of exercise, whereas others have documented significant increases in resting metabolism the morning following exercise performance (Bielinski et al., 1985; Maehlum et al., 1986).

Study designs and subject populations were similar in all cases, and cannot explain the differences among results.

Poehlman and colleagues (1988) found athletes to have a significantly higher RMR per kilogram body mass and per kilogram fat free mass than moderately active and sedentary subjects; RMR was equivalent among the latter two groups. This suggests that exercise must be of a frequency and magnitude characteristic of highly-trained individuals in order to influence RMR. Alternatively, the elevated RMR may have resulted from the well-trained group's high-calorie intake.

In conclusion, the present findings suggest that exercise of a variety of durations and intensities augments post-exercise VO2, but that significant elevations persist for less than 30 min following exercise and total EPOC is small. Assuming an average excess post-exercise energy expenditure of 50 kcal and no change in RMR with long-term exercise, the contribution of EPOC to an individual who exercises four times weekly without a compensatory increase in food consumption would be the equivalent of a fat loss of approximately 1.35 kg a year. Our results concur with the majority of recent reports, and indicate that the contribution of EPOC towards exercise-induced caloric expenditure is small and would be of limited practical value in a weight-loss program.

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Appendix 1.

ANOVA TABLES

INTENSITY EFFECT ON VO2

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	278.966	7			
DAY	941.011	3	313.670	224.853	<.001
ERROR	29.305	21	1.395		
TIME	10368.933	9	1152.104	258.958	<.001
ERROR	280.317	63	4.449		
DAY TIME	4954.846	27	183.513	147.756	<.001
ERROR	234.739	189	1.242		
TOTAL	17088.117	319			
(RESIDUAL)	544.361	273			

DURATION EFFECT ON VO2

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	182.796	7			
DAY	799.533	3	266.511	76.937	<.001
ERROR	72.751	21	3.464		
TIME	10366.000	9	1151.778	240.706	<.001
ERROR	301.435	63	4.785		
DAY TIME	3443.602	27	127.541	76.878	<.001
ERROR	313.480	189	1.659		
TOTAL	15479.597	319			
(RESIDUAL)	687.667	273			

INTENSITY EFFECT ON MAGNITUDE OF VO2

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	1857.987	7			
DAY	2047.773	3	682.591	13.337	<.001
ERROR	1074.794	21	51.181		
TIME	123937.911	8	15492.239	383.975	<.001
ERROR	2259.449	56	40.347		
DAY TIME	323.679	24	13.487	2.145	.002
ERROR	1056.253	168	6.287		
TOTAL	132557.846	287			
(RESIDUAL)	4390.496	245			

DURATION EFFECT ON MAGNITUDE OF VO2

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	2520.110	7			
DAY	1878.895	3	626.298	12.089	<.001
ERROR	1087.920	21	51.806		
TIME	127182.162	8	15897.770	350.403	<.001
ERROR	2540.695	56	45.370		
DAY TIME	384.521	24	16.022	2.860	<.001
ERROR	941.341	168	5.603		
TOTAL	136535.644	287			
(RESIDUAL)	4569.956	245			

INTENSITY EFFECT ON RER

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	1.424	7			
DAY]	.253	3	.084	2.625	.076
ERROR	.682	21	.032		
TIME	.550	9	.061	3.389	.002
ERROR	1.105	63	.018		
DAY] TIME	.298	27	.011	1.833	.010
ERROR	1.189	189	.006		
TOTAL	5.500	319			
(RESIDUAL)	2.975	273			

DURATION EFFECT ON RER

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	169.476	7			
DAY	8.873	3	2.958	1.041	.396
ERROR	59.691	21	2.842		
TIME	63.061	9	7.007	6.910	<.001
ERROR	63.854	63	1.014		
DAY TIME	19.868	27	.736	1.099	.344
ERROR	126.640	189	.670		
TOTAL	511.463	319			
(RESIDUAL)	250.185	273			

INTENSITY EFFECT ON TEMP

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	1.544	7			
DAY	16.233	3	5.411	36.315	<.001
ERROR	3.139	21	.149		
TIME	23.878	9	2.653	60.295	<.001
ERROR	2.774	63	.044		
DAY TIME	9.487	27	.351	20.647	<.001
ERROR	3.148	189	.017		
TOTAL	60.202	319			
(RESIDUAL)	9.061	273			

DURATION EFFECT ON TEMP

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	.891	7			
DAY	19.588	3	6.529	34.005	<.001
ERROR	4.023	21	.192		
TIME	30.248	9	3.361	46.681	<.001
ERROR	4.562	63	.072		
DAY TIME	11.300	27	.419	18.217	<.001
ERROR	4.348	189	.023		
TOTAL	74.959	319			
(RESIDUAL)	12.932	273			

INTENSITY EFFECT ON HR

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	12884.029	7			
DAY	26672.222	3	8890.741	134.823	<.001
ERROR	1384.815	21	65.944		
TIME	87664.082	9	9740.454	221.203	<.001
ERROR	2774.171	63	44.034		
DAY TIME	40658.428	27	1505.868	76.810	<.001
ERROR	3705.342	189	19.605		
TOTAL	175743.089	319			
(RESIDUAL)	7864.328	273			

DURATION EFFECT ON HR

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	17483.444	7			
DAY	24172.446	3	8057.482	88.811	<.001
ERROR	1905.242	21	90.726		
TIME	96416.601	9	10712.956	167.139	<.001
ERROR	4038.019	63	64.096		
DAY TIME	30803.740	27	1140.879	58.217	<.001
ERROR	3703.916	189	19.597		
TOTAL	178523.408	319			
(RESIDUAL)	9647.176	273			

EFFECT OF 5 DAYS OF DAILY EXERCISE ON RESTING METABOLIC RATE

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	17.710	7			
DAY	1.701	4	.425	2.310	.082
ERROR	5.151	28	.184		
TOTAL	24.563	39			

THERMOGENESIS EFFECT ON VO2; INTENSITY

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	66.345	7			
DAY	1.427	3	.476	.553	
ERROR	18.069	21	.860		
TIME	12.868	2	6.434	12.817	<.001
ERROR	7.024	14	.502		
DAY TIME	3.535	6	.589	1.212	.318
ERROR	20.426	42	.486		
TOTAL	129.694	95			
(RESIDUAL)	45.520	77			

THERMOGENESIS EFFECT ON VO2; DURATION

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	47.092	7			
DAY	1.491	3	.497	.419	
ERROR	24.892	21	1.185		
TIME	13.546	2	6.773	11.422	.001
ERROR	8.296	14	.593		
DAY TIME	2.332	6	.389	.928	
ERROR	17.582	42	.419		
TOTAL	115.233	95			
(RESIDUAL)	50.771	77			

THERMOGENSIS EFFECT ON TEMP; INTENSITY

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	1.744	7			
DAY	.601	3	.200	3.077	.049
ERROR	1.363	21	.065		
TIME	.359	2	.179	7.783	.005
ERROR	.328	14	.023		
DAY TIME	.073	6	.012	1.714	.141
ERROR	.302	42	.007		
TOTAL	4.770	95			
(RESIDUAL)	1.993	77			

THERMOGENESIS EFFECT ON TEMP; DURATION

SOURCE	SS	DF	MS	F	P
BLOCKS/SUBJECTS	1.566	7			
DAY	.519	3	.173	2.703	.070
ERROR	1.337	21	.064		
TIME	.392	2	.196	7.840	.005
ERROR	.344	14	.025		
DAY TIME	.031	6	.005	.833	
ERROR	.234	42	.006		
TOTAL	4.422	95			