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THE EFFECTS OF A TWELVE-WEEK TRAINING PROGRAM CONSISTING OF WALKING OR JOGGING ON A TREADMILL AT A SPEED CORRESPONDING TO SIXTY PERCENT OF PEAK OXYGEN CONSUMPTION IN SEDENTARY MEN AND WOMEN

by

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B.P.E., University of Ottawa, 1974

THESIS

Submitted to the School of Graduate Studies in partial fulfillment of the requirements for the degree of Master of Science in Kinanthropology in the School of Human Kinetics, University of Ottawa, 1978.

Ottawa, Ontario

ACKNOWLEDGEMENTS

The writer wishes to thank Dr. M. Jetté for his guidance and constant support. My appreciation is also extended to Richard Routhier for his assistance throughout the entire study.

The assistance of Dr. Martin Cooper for his advice concerning the statistical analysis, and to Pearl Lok, Ajaz Mirza and Monique Carrière for their technical assistance in the laboratory is very much appreciated.

I would especially like to thank the subjects who took part in the study, for without their cooperation this study would not have been possible.

ABSTRACT

This study investigated the effects of a 12-week training program consisting of walking or jogging on a treadmill at a speed corresponding to 60 percent of peak $\dot{V}O_2$ in sedentary men and women. A total of 26 subjects between the ages of 35 and 53 ($\bar{x} = 44.2$) participated in the study of which 13 subjects formed the control group (seven men and six women) and an equal number of men and women formed the experimental group. The exercise program consisted of walking on a treadmill three times per week for a duration of 30 minutes per session. The variables of peak $\dot{V}O_2$, heart rate at an absolute submaximal workload and blood lactate taken three minutes following the submaximal work load were compared. Heart rate, $\dot{V}O_2$ and speed of walking at specific intensities were also examined every four weeks. There was a significant increase in peak $\dot{V}O_2$ (14.69%), and a significant decrease in heart rate (13.7 beats/minute) at an absolute submaximal workload as a result of training ($p < 0.05$). Blood lactate levels were not significantly affected. The mean group heart rate of the experimental group at a workload of 60 percent of the initial peak $\dot{V}O_2$ did not change significantly when measured during every fourth week of training. However, at the end of the training program in those subjects who did demonstrate a reduction in heart rate during these sessions a mean 11.8 percent increase of treadmill speed was required to elicit 60 percent of their initial peak $\dot{V}O_2$. Results of the study indicate that middle-aged sedentary men and women can increase the efficiency of their cardiorespiratory system by walking at a speed corresponding to 60 percent of peak $\dot{V}O_2$ three times per week over a 12-week period.

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CHAPTER I

THE PROBLEM

Introduction

Regular physical activity has been increasingly advocated by physical educators and health professionals to decrease the prevalence of various 'hypokinetic diseases' that are associated with our modern sedentary life style (Kraus 1961). It has also been suggested that conditions such as obesity, hypertension, chronic muscular soreness and the premature development of coronary heart disease could be decreased by participation in a regular exercise program (Fox 1971). Although conclusive evidence is lacking, the available information suggests that sedentary individuals have a higher incidence of coronary heart disease than do those who are physically active in their vocational or leisure pursuits (Morris 1973).

In the case of middle-aged and older men and women the selection of an optimal training intensity is of great importance. In organized programs drop-out rates resulting from boredom, orthopedic difficulties, time and cost involvement are quite high. It is therefore of prime interest to determine what is the minimal amount of exercise required to elicit acceptable training benefits.

Many exercise programs, especially adult group classes, are based on progressive walk-jog regimes and arbitrary target heart rates. In many of these situations people are often working at dangerously high levels. More recently however, individual programs are being established on the basis of an exercise stress test with the intensity of training being determined by employing a percentage of the subject's functional maximal heart rate or $\dot{V}O_2$ max.

The question regarding the existence of an optimal training intensity is still controversial (Lavoie 1978). This study is an attempt to evaluate the effects of a scientifically acceptable but practical exercise prescription on the basis of exercising at a certain percentage of peak oxygen consumption.

Statement of the Problem

This study was designed to examine the effects of a twelve week training program consisting of walking or jogging on a treadmill at a speed corresponding to sixty percent of peak oxygen consumption in sedentary men and women.

Subproblems

Other objectives of the study were as follows:

1. To examine the effects of the training program on peak oxygen consumption, submaximal exercise heart rate and blood lactate levels at an absolute submaximal work load.

2. To quantify the changes occurring in exercise heart rate, $\dot{V}O_2$ and speed of walking or jogging necessary to elicit initial training heart rates as the training program progressed.

Rationale for the Study

Several training studies have shown that exercise results in significant training effects. Increases in maximal work capacity, an index of cardiorespiratory fitness, have been reported in programs utilizing training intensities ranging from 39 to 100 percent of $\dot{V}O_2$ max. It has also been reported that submaximal exercise heart rates decrease with training and that blood lactate levels after an absolute submaximal exercise load are lower after training. In these studies it was generally found that programs at the higher intensity resulted in greater improvements in $\dot{V}O_2$ max. It is accepted that the intensity (Wenger 1975) and duration (Nordesjo 1974) of a training program are the prime factors involved in increasing cardiorespiratory function but that initial level of fitness (Wenger 1975, Pollock 1971, Saltin 1969, and Shephard 1968a) as well as frequency of exercise (Nordesjo 1974) are important factors.

Numerous investigators working with young, middle-aged and older men and women often do not agree as to the existence of an optimal training threshold. It has been suggested that middle-aged and older individuals should exercise at intensities ranging between 50 and 70 percent of maximal aerobic capacity (Hellerstein 1973). Others have stated that in order to increase $\dot{V}O_2$ max the workload must be close to maximum for prolonged periods of time (Davies 1971).

The normal drop-out rate from adult exercise programs ranges from 30 to 40 percent or more (Pollock 1971). The rate is less than 25 percent in programs involving walking (Pollock 1971) or in those with lower exercise intensities (Smith 1976). The ideal program seems to be one that adequately stresses the individual but does not discourage participation due to excessive time requirements or to complaints involving orthopedic problems or uncomfortably high exercising intensities.

It is desirable that an exercise prescription should be based on the individual's response to a standard exercise test and that it take into consideration the type of activity involved, frequency, duration and intensity of the exercise sessions. Activities of an endurance nature such as brisk walking, jogging, swimming, cycling and hiking have been advocated as those most beneficial for middle-aged and older individuals (Wilmore 1976, Hellerstein 1973, and Skinner 1971). It has also been suggested that these activities should be carried out three to four times per week (Wilmore 1976, Cunningham 1974, and Hellerstein 1973) for some thirty minutes per session (Wilmore 1976 and Hellerstein 1973).

Exercise intensity has been prescribed by various techniques. The identification of a training heart rate based on standard maximal heart rate tables is the most widely used method to identify exercise intensity. A certain percentage of an individual's age-adjusted maximal heart rate or functional heart rate is identified and the subject is prescribed a heart rate range within which to exercise. Recent work by Jetté (1975a) has advocated the use of an exercise prescription based on the individual's maximal work capacity, height and weight. The prescription provides the individual with a specific time to cover a set distance. Since many people are limited in pursuing physical activity by such things as available time, convenience, expense, facilities, and skill level (Harris 1967) a simple training program, based on walking or jogging a given distance in a specified time at an adequate intensity could prove beneficial to a large number of sedentary men and women.

Limitations of the Study

1. The subjects employed in this study were volunteers. As such they cannot be considered a random sample population.

2. The number of subjects was limited due to the extensive amount of time required for their testing and training regimen.

3. There was no strict control in either the experimental group or the control group with respect to diet or amount of physical activity related to their occupational or leisure activities. The subjects in the control group were asked to continue their normal lifestyle and not to take part in any new physical activity or exercise program. Furthermore, the success of this study depended entirely on the cooperation of the subjects.

Definition of Terms

The following terms are employed in this study:

Peak oxygen consumption (peak $\dot{V}O_2$)

Peak oxygen consumption is defined as the highest volume of oxygen consumed in milliliters per kilogram of body weight per minute (ml/kg/min) at the point during the multi-stage treadmill test when the subject indicates that he/she is no longer able to continue walking. When compared to $\dot{V}O_2$ max, peak $\dot{V}O_2$ represents 90 to 100 percent of $\dot{V}O_2$ max. In many sedentary, middle-aged individuals the 'levelling-off' criteria that is present in a $\dot{V}O_2$ max test very often cannot be met.. Peak $\dot{V}O_2$ is also referred to as volitional $\dot{V}O_2$ max.

Exercise prescription

Exercise prescription is defined as the speed of the treadmill in miles per hour on the level at which the subject must either walk or jog for a specified period of time in order to exercise at sixty percent of his peak $\dot{V}O_2$.

Submaximal exercise test

The submaximal exercise test employed in this study consisted of a six minute walk or jog on the treadmill at the subject's prescribed training speed.

Submaximal exercise heart rate. (SMEHR)

The submaximal exercise heart rate is defined as the average of the two heart rates obtained from ECG recordings at the end of the fifth and sixth minutes recorded during the submaximal exercise test.

Quantification heart rate (QHR)

The quantification heart rate is defined as the average of the two heart rates recorded at the end of the fifth and sixth minutes during the first portion of the quantification session.

CHAPTER II

REVIEW OF LITERATURE

Introduction

The purpose of the study was to examine the effects of a 12 week training program consisting of walking or jogging on a treadmill at a speed corresponding to 60 percent of peak oxygen consumption in sedentary male and female subjects.

A review of the literature pertaining to the effects of a training program is presented under the following headings:

Maximal Oxygen Consumption
Changes at Submaximal Exercise - Heart Rate
- Blood Lactate
Training Studies
Methods of Exercise Prescription.

Maximal Oxygen Consumption

Maximal oxygen consumption ($\dot{V}O_2$ max) or maximal aerobic capacity measures the functional capacity of the cardiovascular system (Rowell 1974). Mitchell (1971) indicates that $\dot{V}O_2$ max is the highest amount of oxygen that a person can consume during physical work. Normalized for body weight, it is the most useful measurement characterizing the functional capacity of the oxygen transport system. According to Cooper (1968), oxygen uptake during exhausting work is the best single physiological index of the ability of man to sustain hard muscular work. As well, $\dot{V}O_2$ max is the most objective measurement of an individual's physical fitness level as reflected by his oxygen transport system.

At puberty $\dot{V}O_2$ max is not significantly different when comparing males to females but thereafter women have an aerobic capacity 25 to 30 percent lower than that of men. This difference has been attributed to the lower hemoglobin concentration in women and the increase in body fat in women after puberty (Astrand 1970). Aerobic power peaks at 18 to 20 years but then declines gradually with age. An average 65 year old male has a maximal aerobic capacity of approximately 70 percent of that of a 25 year old. This may be attributed to a lower maximal heart rate which reduces maximal cardiac output. As well, older individuals are usually less physically active and a decreased $\dot{V}O_2$ max may be partially due to this de-conditioned state (Astrand 1970).

Maximal oxygen consumption is attained when no further increase in $\dot{V}O_2$ occurs despite additional increases in work load (Astrand 1970 and Mitchell 1971). A test measuring $\dot{V}O_2$ max should involve large muscle groups (Rowell 1974, Astrand 1970, and Shephard 1968a) and eliminate both skill and motivation as factors (Rowell 1974 and Taylor 1957).

A treadmill or bicycle ergometer are generally utilized when assessing maximal aerobic capacity. The actual $\dot{V}O_2$ max attained is affected to a great extent by the type of test employed. Numerous investigators have reported that $\dot{V}O_2$

max was five to 15 percent higher during treadmill running than in bicycle ergometer work (Miyamura 1972, Ikai 1970, Hermansen 1969, Chase 1966, Glassford 1965, and Astrand 1961). Miyamura's (1972) results indicate that there were no differences in maximal stroke volume when comparing treadmill exercise to bicycle work but that treadmill exercise resulted in greater a-$\dot{V}O_2$ differences and higher maximal heart rates. The increased $\dot{V}O_2$ max during treadmill work was attributed to a higher maximal cardiac output and a larger a-$\dot{V}O_2$ difference.

The $\dot{V}O_2$ max attained during treadmill walking was found to be significantly lower than that found during running (Stanford 1976 and McArdle 1973). McArdle (1973) has suggested that discomfort in the lower back and calf muscles may limit performance in walking tests. It has been stated that uphill running reveals a higher $\dot{V}O_2$ max than walking, cycling or arm ergometry because a larger muscle mass is being utilized (Kamon 1972 and Astrand 1970). Kamon (1972) also indicates that $\dot{V}O_2$ max during cycling may be lower due to the high intramuscular pressure which could restrict muscle blood flow.

Stenberg (1967) compared $\dot{V}O_2$ max and cardiac output during arm ergometry and leg work (sitting). $\dot{V}O_2$ max and cardiac output attained during arm work were 66 and 80 percent respectively of the values attained during leg work. Values reached during combined arm and leg work were equal to those found during leg work alone. In contrast, Sechar (1974) reported that $\dot{V}O_2$ max during combined arm and leg work resulted in a $\dot{V}O_2$ max 106 percent of the values obtained during leg work (sitting) but was equal to the values attained in treadmill running. Glessor (1974) added arm ergometry to leg work and $\dot{V}O_2$ max was 10 percent greater than that attained during leg work alone. In contrast, Reybrouk (1975) saw no further increase in $\dot{V}O_2$ max when arm work was combined with leg exercise. He concluded that the effect on $\dot{V}O_2$ max of adding arm to leg exercise depends on the degree of training of the legs. Highly trained subjects ($\dot{V}O_2$ max greater than 50 ml/kg/min) may reach their maximal capacity for oxygen delivery when only legs are exercising whereas non-trained subjects are able to increase oxygen consumption when the active muscle mass is increased by the addition of arm ergometry. Gollnick (1972a) examined trained canoeists and his results show only a three percent difference in $\dot{V}O_2$ max when comparing arm and leg ergometry. It was suggested that the high degree of conditioning of the upper body was responsible for this very small difference.

The classical definition is that $\dot{V}O_2$ max equals maximal cardiac output X maximal a-$\dot{V}O_2$ difference (Froelicher 1974, Rowell 1974, and Taylor 1957). Cardiac output is determined by heart rate and stroke volume whereas a-$\dot{V}O_2$ difference depends on maximal oxygen content in arterial and mixed venous blood (Mitchell 1971). Rowell (1974) contends that $\dot{V}O_2$ max is limited either by maximal cardiac output and O_2 extraction or by the capacity to develop pressure against a particular total peripheral resistance (TPR). He adds that $\dot{V}O_2$ max may best be expressed as the product of maximal a-$\dot{V}O_2$ difference times the ratio of maximal blood pressure to minimum TPR. He feels that a major unresolved problem is whether an increase in blood pressure or a further fall in TPR at $\dot{V}O_2$ max could either raise $\dot{V}O_2$ above what was called $\dot{V}O_2$ max or raise cardiac output above what has been traditionally called maximal cardiac output.

There is some controversy regarding the factors that define the upper limits of maximal aerobic capacity. Rowell (1974) supports the concept that $\dot{V}O_2$ max is dependent on the oxygen transport system but is not certain whether the ultimate limitation depends on cardiac output or arterial blood pressure. He has identified two factors that indicate that metabolic capacity of the muscle is not a limiting factor. Firstly, if O_2 is added to inspired air, $\dot{V}O_2$ max increases by an amount proportional to the increased arterial oxygen content. This augmentation of $\dot{V}O_2$ indicated that metabolism at the muscle is not a limiting factor. Secondly, when extra working muscles (e.g. arms) are added to a large muscle mass (e.g. legs) working at sufficiently high intensity to elicit $\dot{V}O_2$ max, this $\dot{V}O_2$ is usually not increased. Adding more muscle mass would raise $\dot{V}O_2$ if the metabolic capacity of the muscle were the limiting factor.

In summary, maximal oxygen uptake measures the greatest amount of oxygen that an individual can consume during physical work. It is considered to be the best single index characterizing the functional capacity of the oxygen transport system. Maximal oxygen consumption is 25 to 30 percent higher in men than women and as age increases there is a gradual decline in maximal aerobic capacity. A measurement of $\dot{V}O_2$ max should utilize a large muscle mass. Treadmill running normally elicits a higher maximal oxygen uptake than treadmill walking, bicycle riding or arm ergometry. The standard definition is that $\dot{V}O_2$ max is equal to maximal cardiac output X maximal a-$\dot{V}O_2$ difference.

Adaptations of Maximal Oxygen Consumption to Endurance Training

The extent of the increase in $\dot{V}O_2$ max with training depends on the initial level of fitness of the individual and on the intensity, duration and frequency of the training program (Holloszy 1976). An endurance training program results in central adaptations such as an increased cardiac output and higher stroke volume and in peripheral adaptations such as an increased a-$\dot{V}O_2$ difference (Clausen 1976 and Holloszy 1976).

1. Central Adaptations

The central adaptations to training in men and women have been extensively studied. Hartley (1969) trained 15 previously sedentary men aged 38 to 55 for an eight to 10 week program. The 13 percent increase in $\dot{V}O_2$ max was due to a 16 percent increase in cardiac output. Neither arterial oxygen content nor systemic a-$\dot{V}O_2$ difference changed during maximal exercise. It was concluded at this time that the increase in $\dot{V}O_2$ max was dependent on a larger amount of oxygen being transported by a larger cardiac output. The increase in cardiac output was due to a larger stroke volume since maximal heart rates remained unchanged. Work by Saltin (1969) involving older subjects substantiate the findings that only stroke volume increases with an increase in $\dot{V}O_2$ max.

The available data regarding adaptation to training in young men is conflicting. Work by Saltin (1968) and Rowell (1974) indicate that increases in $\dot{V}O_2$ max are due to equal increments in stroke volume and in a-$\dot{V}O_2$ difference. On the other hand, Ekblom (1968) saw significant increases in stroke volume only. Although a-$\dot{V}O_2$ difference was slightly higher after training the changes were not statistically significant.

A short-term training program involving young, middle-aged and older women resulted in increases in stroke volume rather than in a-$\dot{V}O_2$ difference (Kilbom 1971c). Recent work by Cunningham (1975) on women involved in a training study found that a 34 percent increase in $\dot{V}O_2$ max after an initial nine week period was due primarily to an increase in stroke volume. After an additional 52 week training period the stroke volume was slightly decreased when compared to the effects of the initial nine week program but a-$\dot{V}O_2$ difference was significantly increased. It was concluded that women who are initially unfit adapt to short-term training with a central change followed by a much stronger peripheral adaptation after a longer training program.

In a recent review Clausen (1976) has also indicated that increases in $\dot{V}O_2$ max are brought about by changes in maximal stroke volume and in maximal a-$\dot{V}O_2$ difference. The increase in maximal cardiac output ($\dot{Q}$ max) are directed to the exercising muscle. The increment in Q max seems to be increased by training due to a reduction in TPR rather than an increase in maximal muscle blood flow. He feels that the decrease in TPR is the primary factor involved in the increase in $\dot{V}O_2$ max and that the augmentation of stroke volume is a secondary event. This decreased TPR may be due to a greater vasodilation or a more effective cross-sectional area of the vascular bed. A more efficient skeletal muscle pump or an increased muscle capillary density may contribute as well but the later concept has not been substantiated (Hermansen 1971 and Saltin 1968). Trained individuals have a relatively low hemoglobin concentration and it is possible that the lower blood viscosity may play a role in the low TPR and large Q max (Clausen 1976).

2. Peripheral Adaptations

As previously indicated a number of investigators believe that the increase in $\dot{V}O_2$ max after training is due to equal increments in a-$\dot{V}O_2$ difference and in stroke volume (Rowell 1974 and Saltin 1968).

Various researchers have hypothesized that with training the increase in a-$\dot{V}O_2$ difference is due to a higher oxygen utilization at the cellular level (Holloszy 1976, Cunningham 1975, Holloszy 1973, Shappell 1971, Varnauskas 1970, and Saltin 1968). Cunningham (1975) adds as well that a better distribution of blood flow to exercising muscle may increase the a-$\dot{V}O_2$ difference. Rowell (1974) disagrees with this concept when he concludes that renal blood flow and splanchnic blood flow are already reduced 70 to 80 percent at maximum exercise and any possible further vasoconstriction could contribute only slightly to the increased a-$\dot{V}O_2$ difference. He also suggests that neither muscle capillary density nor reduced hemoglobin affinity at the capillary-muscle interface accounts for the increases in a-$\dot{V}O_2$ difference.

Endurance training has been shown to effect changes at the muscle tissue which may account for the increases in a-$\dot{V}O_2$ difference. Pattengale (1967) has shown that a strenuous running program in rats resulted in an 80 percent increase in myoglobin in the muscles directly involved in the running. It has been indicated that myoglobin increases the rate of oxygen diffusion through a fluid layer. It may also facilitate oxygen utilization in the muscle by increasing the rate of diffusion through the cytoplasm to the mitochondria.

An increase in mitochondrial respiratory enzymes in skeletal muscles have been seen in rats (Gollnick 1972b and Barnard 1971) and in humans (Morgan 1971). Electronmicroscopic studies in mice and in rats have shown that an increase in both size and number of mitochondria are responsible for the increase in total mitochondrial protein (Hoppeler 1973, Morgan 1971, and Gollnick 1969). According to Morgan (1971) this increase in mitochondrial oxidative enzymes and in the actual size and number of mitochondria may lead to an increased capacity of skeletal muscle to oxidize carbohydrate, fat and ketones. Holloszy (1976) believes that due to its content of mitochondria and its capacity to generate ATP from oxidation of pyruvate and fatty acids, skeletal muscle becomes more like cardiac muscle after endurance training. The activities of the enzymes creatine phosphokinase, adenylate kinase and α-glycerophosphate dehydrogenase in the skeletal muscle become more like those in cardiac muscle.

In summary, it would seem that an augmentation of $\dot{V}O_2$ max is due to an increase in stroke volume and an increase in a-$\dot{V}O_2$ difference. It has been indicated that with training older men adapt to increases in $\dot{V}O_2$ max with an increase in stroke volume only whereas young men increase both stroke volume and a-$\dot{V}O_2$ difference. In women, short-term training (7 to 9 weeks) results in increases in stroke volume but after a more prolonged training program a-$\dot{V}O_2$ difference is also effected. The major adaptation at the cellular level seems to occur in the mitochondria where there is an increase in the mitochondrial respiratory enzymes and an increase in the size and number of mitochondria. Clausen (1976) concludes that an increased oxygen supply seems to allow the same amount of mitochondria to produce a higher respiratory rate and ATP synthesis rate. An increased amount of mitochondria can, within certain limits, augment local $\dot{V}O_2$ max at a given maximal oxygen supply and increase respiratory rate even further if oxygen supply is increased at the same time.

Adaptations at Submaximal Exercise

Heart Rate

Numerous investigators have studied the effect of an exercise program on heart rates occurring at an absolute, submaximal exercise load. Christensen (1931) was the first to report a decrease in heart rate at a given submaximal work load after training. Since that time a decrease in exercise heart rates at a submaximal exercise level after training has been reported in young (Wenger 1976, Ekblom 1968, and Saltin (1968), middle-aged (Byrd 1974, Pollock 1971, Hartley 1969, Saltin 1969, and Skinner 1964), and older men (Tzankoff 1972) and in young (Smith 1976, Kilbom 1971a, Kilbom 1971b, and Kilbom 1971c), middle-aged (Cunningham 1975, Flint 1974, Hanson 1974, Kilbom 1971a, Kilbom 1971b, and Kilbom 1971c), and older women (Kilbom 1971a, Kilbom 1971b, and Kilbom 1971c).

Byrd (1974) has concluded that since heart rate is one of the main determinants of myocardial oxygen consumption the relative bradycardia after chronic exercise may be one of the most important contributors. Saltin (1968) found that in men the most important effect of training on response to submaximal work was a decrease in heart rate. This lowered heart rate was accompanied by an increase in stroke volume but an unchanged cardiac output. Hartley (1969) and Byrd (1974) reported these same findings in middle-aged men. Clausen (1976) has hypothesized that training would lead to a decrease in blood flow to working muscle at a given submaximal workload and thus a decrease in cardiac output. Earlier work by Andrew (1966) led to the finding that pulse rates and cardiac outputs were significantly lower at a constant submaximal workload but there was no increase in A-VO_2 difference after training.

In women aged 21 to 61, Kilbom (1971c) found that cardiac output at absolute submaximal exercise loads were not changed after training. In this study A-VO_2 differences remained the same but stroke volumes were significantly greater in subjects of all ages after training. Cunningham (1975) reported significant decreases in heart rate at an absolute submaximal workload but saw little changes in heart rate at relative $\dot{V}O_2$ values after training. He also found increases in cardiac output and stroke volume at relative workloads after training but A-VO_2 difference did not change at submaximal values up to 80 percent of predicted $\dot{V}O_2$ max.

In summary, conclusive evidence exists to indicate that heart rates at a submaximal exercise load are decreased after training. There still seems to be controversy regarding hemodynamic changes. Some investigators feel that stroke volume increases while cardiac output does not change (Byrd 1974, Kilbom 1971c, Hartley 1969, and Saltin 1968) whereas Clausen (1976) and Andrew (1966) believe that cardiac output decreases during submaximal exercise as a result of training.

Wenger (1976) trained two groups of men, one at 100 percent and the other at 60 percent of $\dot{V}O_2$ max. Physical working capacity and $\dot{V}O_2$ max after training were greater in the group training at the higher level of intensity. At submaximal exercise, although heart rates were lower in both groups after training there were no significant differences between the response of the two groups.

Blood Lactate

1. Introduction to Biochemistry

The main source of energy for muscular contraction is carbohydrate and fat (Astrand 1970) but the relative contribution of each depends on intensity of work, physical condition of the individual and the type of diet (Attachoo 1975). In the living organism, high energy phosphate compounds represent the common currency for the transfer of energy. In every cellular reaction the ATP - ADP system is the primary carrier of chemical energy (Astrand 1970).

The main steps in the energy exchange in the muscle can be summarized in the following steps:

a) ATP $\rightleftarrows$ ADP + P + free energy

b) Creatine Phosphate + ADP $\rightleftarrows$ creatine + ATP

c) Glycogen or glucose + P + ADP $\rightleftarrows$ lactate + ATP

d) Glycogen or free fatty acid + P + ADP + $O_2 \rightleftarrows CO_2$ + H_2O + ATP (Astrand 1970)

Aerobic metabolism is characterized by four major steps:

a) Glycolysis - the formation of two pyruvic acid molecules from one molecule of glucose. A net of two molecules of ATP are formed in this step.

b) Conversion of two pyruvic acid molecules into two molecules of acetyl coenzyme A. No ATP is formed during this step.

c) Citric Acid Cycle - the acetyl coenzyme A is degraded through a series of chemical reactions to CO_2 and hydrogen atoms.

d) Oxidation of hydrogen atom by oxidative enzymes. About 90 percent of all ATP is formed during the oxidation of the hydrogen atoms released during the earlier stages. The oxidative enzymes are thought to be arranged on the inner surface of the mitochondria. A total of 24 hydrogen ions are released during the citic acid cycle and 20 of these are oxidized with the release of three ATP molecules per two atoms of hydrogen metabolized. A net of 30 ATP molecules are formed during this process. Another four ATP molecules are released when the other four hydrogen atoms are introduced beyond the first stage of the oxidative process. A total of 39 molecules of ATP are formed for each molecule of glucose degraded to CO_2 and water. This represents an overall efficiency of approximately 44 percent (Guyton 1971).

If the oxygen supply is either unavailable or insufficient small amounts of energy can be released via the anaerobic system. No oxygen is required for the glycolytic process but overall efficiency is very low. If there is a build-up of either one or both of the two end-products of glycolysis, pyruvic acid and hydrogen atoms (which are combined with DPN to form DPNH + H), the glycolytic process would shut down. When either of these end-products become excessive they react with one another to form lactic acid:

$$\text{Pyruvic Acid} + \text{DPNH} + H^+ \overset{\text{LDH}}{\rightleftarrows} \text{Lactic Acid} + \text{DPN}^+.$$

The lactic acid diffuses out of the cells into the extracellular fluids and into the intracellular fluids of the less active cells. If lactic acid formation did not occur under anaerobic conditions the end-products of glycolysis would build up very quickly and glycolysis would be able to proceed for only a few seconds. Although anaerobic glycolysis is a very inefficient process, the formation of lactate enables energy production to proceed even in the absence of oxygen (Guyton 1971).

When oxygen becomes available the extra DPNH + H are rapidly oxidized. The chemical reaction for formation of lactic acid reverses itself and the lactic acid becomes pyruvic acid. The large amounts of lactic acid that are formed during anaerobic glycolysis can either be converted to glucose or used directly for energy (Guyton 1971).

White muscle fibers or fast-glycolytic (FG) fibers depend primarily on the glycolytic process for metabolism. They have few mitochondria but have a larger concentration of glycolytic enzymes and a higher glycogen content than red muscles. In white muscle the enzyme LDH, which catalyzes the reaction of pyruvate to lactate, is quite active and favours lactate production. Red muscle fibers or slow-oxidative (SO) fibers derive ATP from oxidative phosphorylation. Red muscle contains large numbers of mitochondria and has a higher concentration of myoglobin. These muscles are difficult to fatigue and are surrounded by many capillary blood vessels thereby keeping them well supplied with oxygen and nutrients. The LDH does not favour the reduction of pyruvate to lactate (Vander 1970).

Reviews by Rosell (1973) and Jorfeldt (1971) have substantiated this by indicating that lactate production takes place primarily in the FG fibers rather than the SO fibers.

During submaximal exercise is has been indicated that the lactate formed during the initial stages of work is removed from the blood during the remainder of the work period (Knuttgen 1970). Rowell (1966) felt that gluconeogenesis in the liver was the most likely fate of lactate (up to 50 percent) but since that time various investigators have agreed that the major site of lactate removal is the skeletal muscle (Issekutz 1976, Ahlborg 1976, Ahlborg 1975, Hermansen 1973, and Knuttgen 1971). It has been indicated that approximately four to eight percent of the total amount of lactate eliminated was removed by the liver (Hermansen 1973), ten percent by the heart and the kidneys (Krebs 1964 and Carlsten 1961) and less than five percent by the urine (Johnson 1937) and sweat (Astrand 1963). Issekutz (1976) has indicated that the two major pathways of lactate utilization are oxidation to CO_2 and formation of plasma glucose. Minaire (1973) found that in dogs, 58 percent of the lactate produced at rest and 75 percent during running is promptly oxidized. Hubbard (1975) has indicated that in man performing 30 minutes of exercise at 62 to 75 percent of

$\dot{V}O_2$ max 35 to 68 percent of administered 14 C-Lactate is recovered as CO_2. Brooks (1973) has indicated that in rats lactate is oxidized by the kidneys and the myocardium but due to the relatively low weight of these organs they can only account for a small amount of the lactate removed. They feel that due to the large mass of skeletal muscle it is a major contributor to lactate oxidation. Essen (1973) adds that since lactate can readily enter the cell and the aerobic pathways it is a suitable substrate for oxidative metabolism. They found that lactate can be taken up by the human skeletal muscle and that this uptake is related to the glycogen level in the muscle. The lactate taken up by the muscle represents a carbohydrate source for working muscle and may therefore be an indication of low carbohydrate stores in the muscle. The lowest carbohydrate level was found to be most evident in the SO fibers, which have a capacity to convert lactate to pyruvate. They have suggested that at the onset of exercise a portion of FG and SO fibers may be activated and as O_2 transport adjusts to set the demands of exercise, lactate production and release may occur in all fibers. This may also occur when the delivery and uptake of O_2 to the tissue is insufficient to meet metabolic demands. During work of moderate intensity there is a primary reliance on SO fibers. Blood flow to the muscle is amplified and aerobic metabolism predominates. There is a continued breakdown of glycogen to provide the main carbohydrate source for metabolism. As work progresses a nearly complete glycogen depletion occurs and extracellular carbohydrate becomes increasingly important to the metabolism of the cell. The major source of the carbohydrate may be either blood glucose or lactate. During prolonged work of moderate intensity all SO and FG are gradually emptied of glycogen but there is no major increase in blood lactate. This may be due to an uptake of lactate by the SO fibers which is facilitated by the localization of LDH-1 in these fibers. Issekutz (1976) feels that during exercise 90 percent of oxygen uptake serves the working muscle and it seems justified that during work the muscles not only produce but also utilize lactate. He concludes that since skeletal muscle consists of a variety of muscle fiber with definite histochemical, biochemical and mechanical differences it can simultaneously produce, remove and oxidize lactate.

2. Early Work

Lactic acid was found to be present in skeletal muscle by Berzelius in 1812 (Berzelius 1812) but it was not until the early 1900's that Fletcher (1907) saw the relationship between muscle activity and production of lactate. A few years later Hill (1923, 1924) studied the relationship between skeletal muscle energy release and the formation of lactic acid in humans. They used the term 'oxygen debt' to describe the excess oxygen consumption during the recovery period from exercise. It was postulated that the energy released during the transition from rest to work was provided by glycolysis, a non-aerobic energy source. The accumulation of lactate and its subsequent removal by oxidative processes required an extra oxygen consumption. It was also demonstrated at this time that when physically fit men performed moderate work the blood lactate did not rise significantly above its usual resting value but as exercise intensity increased the blood lactate levels increased in proportion (Hill 1923).

Jervell (1928) showed that the greatest accumulation of lactate took place during the first few minutes of exercise and that this was due to a shortage of oxygen during the transition from rest to work.

In 1933, the results of Hill (1923, 1924) were confirmed by Margaria (1933). Treadmill running could be carried out at low intensities without significant changes in blood lactate levels and it was not until work intensity exceeded two-thirds of maximal oxygen uptake that blood lactate levels began to increase. They concluded that the initial fast component of oxygen debt (alactacid portion) was not due to lactate removal. The slow component (lactacid portion) was due to the oxidation of the accumulated lactate and to an increase in resting metabolism resulting from the exercise. More recently McMiken (1976) attributed the first rapid component of O_2 debt to oxidative metabolism necessary for the resynthesis of the phosphagen split at the onset of work. Work by Katch (1972) indicated that the slow component of O_2 debt meets energy requirements for the additional cario-respiratory work in restoring homeostasis. Brooks (1971) adds that temperature effects on metabolism also contribute to this phase of the debt.

Results from work by Huckabee (1958) disputed the earlier work of Hill (1924, 1923) and Margaria (1933). They concluded that there was no alactacid portion and that the debt could be explained by the oxygen necessary to oxidize the portion of the accumulated lactate was due solely to tissue hypoxia. This theory has since been disputed by various investigators (Knuttgen 1971).

Knuttgen (1970) has suggested that the oxygen debt following physical exercise appears to be a complex interaction of numerous factors. Its division into separate lactic acid and non-lactic acid portions seemed overly simplified and not in keeping with the behaviour of the two dominant phases of recovery, fast and slow. He concludes that "a more complete look at interrelated metabolic, thermal, electrolytic and hormonal changes during exercise will be necessary for its explanation".

3. Factors Influencing Lactate Production

The production of lactate occurs in skeletal muscle during exercise. The intensity of work and/or the state of training dictate when lactate formation begins as well as the amount of lactate produced (Knuttgen 1971).

Under anaerobic conditions the lactate produced diffuses from the muscle to the blood. The concentration of lactate in arterial blood is used as an indicator of the extent to which anaerobic processes are involved during work (Hermansen 1971b and Astrand 1970).

Energy for work of light to moderate intensity is provided almost exclusively by aerobic sources and blood lactate concentrations remain close to normal. The ADP and CP stores supply energy for the critical transition period from rest to work before respiration and circulation have adapted to the stress. As work intensity increases, anaerobic processes play a more significant role. Knuttgen (1971) has suggested that if pyruvate is formed at a rate faster than the steps of oxidative phosphorylation then the pyruvate is reduced to lactate. Hermansen (1971b) has further indicated that in the presence of pyruvate and NADH + H^+ the equilibrium strongly favours lactate production. The concentrations of pyruvate, NADH and H^+ as well as the activity of LDH play a prime role in the production of lactate. It has also been suggested that lowered blood oxygen tension (e.g. at altitude) and circulatory disorder may lead to an increased lactate production at the same absolute work load (Hermansen 1971b).

It has been shown that only small increases in lactate are seen at work intensities below 50 to 60 percent of $\dot{V}O_2$ max (Hermansen 1972, Knuttgen 1972, Knuttgen 1971, Karlsson 1971a, Karlsson 1971b, Astrand 1970, and Blackmon 1967). Hermansen (1972) states that at 63 percent of $\dot{V}O_2$ max there are significant increases in blood lactate levels whereas Wyndham (1962) found this level to correspond to 55.3 percent of $\dot{V}O_2$ max. Work by Saltin (1971) indicates that an untrained individual will begin to produce lactate at 50 to 60 percent of $\dot{V}O_2$ max but trained men do not produce lactate until work demands reach

60 to 70 percent of maximal capacity. At higher submaximal workloads lactate production increases in direct proportion to work intensity (Knuttgen 1971). In conflict with this general concept, Saiki (1967) has concluded that in all conditions, lactic acid production reaches appreciable values only when work loads are above $\dot{V}O_2$ max. They feel that in the period of relative anoxia during the transition from rest to steady state lactic acid could be produced but this is only a temporary phase. Lactate would not increase or it would possibly even disappear from the blood once the alactacid oxygen debt phase was over. They believe that no appreciable amount of lactate is produced during submaximal work levels once a steady state of $\dot{V}O_2$ is reached. They conclude that lactic acid found in the blood cannot be taken as an index of the glycolytic process being active during steady state exercise. Karlsson (1971b) is in agreement with Saika (1967) when he suggests that lactate is formed by a lack of oxygen at the start of exercise and until a steady state is obtained or at supramaximal work loads.

4. Training Effects on Lactate Production

As early as 1924, Hill (1924) demonstrated that physically fit men did not produce a significant amount of lactate at moderate work loads and only when exercise intensity increased did blood lactate increase. Since that time other investigators substantiated the principle that trained individuals have lower blood lactate levels than untrained individuals at similar submaximal work loads (Hermansen 1971, Astrand 1970, Saltin 1971, Holmgren 1959, Johnson 1969, Keul 1966, Saltin 1969, Edwards 1971, and Kilbom 1971b).

Keul (1966) saw that after a five week exercise program blood lactate levels at a submaximal workload were lower after training. Saltin (1969) studied the effects on men aged 35 to 50. Training consisted of two sessions per week of interval training (at 98 to 99 percent of $\dot{V}O_2$ max) and one session per week of continuous running (at 91 to 97 percent of $\dot{V}O_2$ max). After this very intensive training program blood lactate concentrations after an absolute work load were lower but no changes were seen at relative workloads. Saltin (1971) has demonstrated that after a more prolonged training program blood lactate levels are lower at relative work loads as well. Kilbom's (1976) work with sedentary women aged 19 to 64 resulted in lower blood lactate levels at a given submaximal work load after a seven week bicycle program held two to three times per week at exercise loads corresponding to 70 percent of $\dot{V}O_2$ max.

There remains some controversy regarding the reasons why blood lactate levels are lower at absolute submaximal work loads after training.

Holmgren (1959) concludes that blood lactate levels reflect the degree of anaerobic metabolism in the working muscles and may be used as an index of how adequate the blood flow and oxygen transport to the muscles are in relation to the need. Astrand (1970) interprets the lower blood lactate levels in trained individuals as an expression of a more effective O_2 transport at the beginning of work, leading to a diminished anaerobic energy yield. Other investigators (Saltin 1969, Hartley 1969, and Ekblom 1968) feel that a higher a-$\dot{V}O_2$ difference during work brought about by increased aerobic energy production leads to lower blood lactate levels at submaximal work loads. Wenger (1975) believes that since lactate production at submaximal work is a result of the inability of the aerobic systems to meet energy demands then training of the aerobic system should decrease the involvement of anaerobic glycolysis.

Holloszy (1971) has suggested that cardiovascular adjustments do not fully explain the decreased blood lactate levels. They propose that biochemical adaptations occur in skeletal muscle in response to regularly performed endurance exercise that may help to explain this phenomena. It has been suggested that the increased activation of red muscle fiber with a more efficient energy yield from aerobic processes may also be a possible explanation for the decreased blood lactate levels at submaximal work loads after training. Hermansen (1971b) and Saltin (1971) are in agreement when they explain that during exercise of light intensity, SO fibers are recruited but as work loads increase white muscle fibers are activated. It is possible that a larger proportion of red fibers are activated during exercise in athletes than in untrained individuals.

In summary, it is generally accepted that work intensity and/or state of conditioning are the most important factors in the process of lactate production. The blood lactate concentration has been used as an indicator of the extent to which anaerobic processes are involved during work. Lactate production takes place predominantly in white or FG fibers. Depending on the level of conditioning, the onset of lactate production occurs at workloads requiring 50 to 70 percent of $\dot{V}O_2$ max. Training programs in middle-aged men and women have led to a decreased blood lactate level after an absolute submaximal work load but the reasons for this are not clearly defined. It has been advocated that the lower blood lactate levels after training reflect an increased efficiency of blood flow and oxygen transport system. Recently, it has been proposed that cardiovascular adjustments do not fully explain these changes. Some investigators feel that with training there is an increased activation of red muscle fibers at submaximal exercise and the FG fibers which are responsible for lactate production are not recruited until higher work loads.

5. Relationship of Lactate Production to Fiber Recruitment

In addition to the cardiovascular adaptations that occur in response to training various investigators have suggested that changes in the recruitment pattern also occur in the skeletal muscle. In the trained individual, the FG fibers are recruited at higher levels of work. Blood lactate levels are therefore lower after an absolute submaximal exercise load.

A hypothetical model for fiber recruitment in human skeletal motor units has been established by Rosell (1973). At low intensities of rhythmic exercise (i.e. pedalling a bicycle or walking on a treadmill), small motoneurons innervating SO fibers are activated. At increasing work loads, larger cells are included and FG muscle fibers are contracted.

Recent work in animals and man on glycogen depletion patterns have substantiated this basic hypothesis. Work done by Armstrong (1973) on rats indicate that high oxidative fibers primarily support low to moderate intensity work, but as exercise intensity increases a larger number of FG fiber motor units are activated. Further work by Armstrong (1974) on rats has demonstrated that blood lactate levels did not increase until running speeds exceeded a load requiring approximately 60 percent of aerobic capacity. In the groups of animals running at different speeds the blood lactate concentrations were directly proportional to the percentages of FG fibers showing a decline in glycogen. It was suggested that the recruitment of FG fibers may not only aid in the contractile work but also supply carbohydrate to the oxidative fibers in the form of lactate.

In man, the glycogen depletion patterns are similar to that found in animals. Costill (1973) has shown that the SO fibers were the first to become depleted of glycogen at running speeds requiring 60 to 80 percent of $\dot{V}O_2$ max but as exercise continued FG fibers also became depleted. Work by Gollnick (1973) demonstrated that during the first hour of cycling at an intensity of 60 percent of $\dot{V}O_2$ max only SO fibers were depleted. Edgerton (1973) studied four fit young men and showed that after 40 minutes of continuous work at 60 percent of $\dot{V}O_2$ max glycogen stores were approximately 36 percent depleted and there were only small increases in muscle and blood lactate.

Gollnick (1974) suggested that oxygen availability is a crucial factor in fiber recruitment patterns. When the oxygen supply becomes insufficient, the activation of FG fibers with their high anaerobic potential would result in a rapid reduction in their glycogen stores.

Baldwin (1975) has suggested that one mechanism by which training may increase endurance appears to involve a glycogen sparing effect. They have shown that in trained rats the glycogen stores in fast-twitch red, fast-twitch white and slow-twitch red fibers were depleted significantly more slowly during treadmill running than in the untrained animals. Blood lactate levels were also significantly lower in the trained animals. It was suggested that in the untrained animals sufficient glycogen depletion may have occurred to result in fatigue of some of the oxidative fibers. During the later stages of exercise more white fibers may have therefore been recruited in the untrained animals.

It has been shown in man as well that glycogen content decreases more slowly in the trained than in the untrained individual at the same submaximal work loads (Karlson 1974 and Saltin 1971). Hermensen (1967) agrees that a trained person has substantially smaller glycogen depletion at the same absolute work load but indicates that only minor differences are observed at relative work loads. According to Rosell (1973) there is a slightly higher glycogen depletion rate in the untrained individual due to a high lactate production during the first few minutes of exercise at workloads demanding more than 50 percent of [illegible] max. They conclude that physical conditioning not only improves an individual's $\dot{V}O_2$ max but also affects the ability of the muscle to metabolize fat. A saving effect of the muscle glycogen stores is accomplished by a less marked lactate production at the beginning of submaximal exercise.

In summary, there is agreement that in both animals and man low intensity activities recruit SO fibers but as work intensity increases larger numbers of FG fibers are recruited. In animals it was found that at work intensities greater than 60 percent of maximal capacity blood lactate levels began to increase. Increases in blood lactate concentration were also seen to be directly proportion~~al to~~ glycogen depletion in the FG fibers, thus indicating that as FG fibers were recruited blood lactate levels increased. In man SO fibers were recruited in the initial stages of work requiring 60 to 80 percent of $\dot{V}O_2$ max but as exercise continued FG fibers also became depleted. It has been suggested that O_2 availability is a crucial factor in fiber recruitment, when the supply of O_2 becomes insufficient, FG fibers with their high anaerobic potential are activated.

Generally a trained individual will not initiate the production of lactate until workloads demand 60 to 70 percent of $\dot{V}O_2$ max whereas an untrained person will begin to produce lactate at 50 to 60 percent of their maximal capacity. Lactate is produced primarily in the FG or white muscle fiber. With training, a larger portion of SO or red muscle fiber with a more efficient energy yield from aerobic processes are utilized during exercise. As indicated by the work on glycogen depletion patterns it is not until work intensity exceeds 60 to 80 percent of $\dot{V}O_2$ max that the FG fibers are recruited. Training seems to result in a glycogen sparing effect. In the trained individual, glycogen content decreases more slowly at the same submaximal workload. As a result of the increased recruitment of more SO fibers, blood lactate levels after an absolute submaximal workload will be lower after training.

Training Studies

Numerous training studies have been carried out since the early 1900's. Much of this initial work dealt with fit young men and quite often the intensity, frequency and duration of effort was not closely quantified. Within the last 25 years more time has been spent in studying the effects of training on older sedentary men and women. The following section will review various training studies completed on young men, on middle-aged and older men and on women. A summary of these studies is presented in Table 1.

1. Training Studies Involving Young Men

Early work done by Bock (1928) indicated that no changes occurred in predicted $\dot{V}O_2$ max in subjects who rode on

TABLE 1

SUMMARY OF VARIOUS TRAINING STUDIES

Author	Date	Subjects	Type of Training	Intensity	Frequency	Duration	Changes
<u>Men</u>							
Bock	1928	Men - young	Bicycle Ergometer	HR 140-160	Several times		No change
Christensen	1931	Men - young	Bicycle Ergometer	480-1680 kgm/min		18-40 mins per day	↑ Fitness level
Robinson	1941	Men - young	Track and Treadmill Running	"Vigorous"		26 weeks	17% ↑ $\dot{V}O_2$ max
Knehr	1942	Men - young	Middle Distance Running	"Less Severe"		6 months	7% ↑ $\dot{V}O_2$ max
Cogswell	1946	Men - young	5 Min Stepping 4 Min Treadmill Run 1 Min Exhaustive Bicycle Ride		3 x week 2 x week 3 x week	50 days	No sign. change
Karvonen	1947	Men - young	Treadmill Running	HR 150	5 x week	30 min/sess for 4 weeks	Sign. ↑ only at 150 HR
Hollman	1963	Men	Bicycle Ergometer	HR < 130 HR > 130	5 x week	30 min/sess for 6 weeks	Sign. ↑ at HR >130
Skinner	1964	Men - Middle-aged (Sedentary)	3 days at 'Cureton Program' 3 days at Running		6 x week	6 months	↑ Treadmill Run Time ↓ Mile Time

Table 1 (cont'd)

Author	Date	Subjects	Type of Training	Intensity	Frequency	Duration	Changes
Rowell	1964	Men - young	Middle-Distance Training			6 weeks	10.4% ↑ $\dot{V}O_2$ max
Cureton	1964	Men - Middle-aged	Cals/Running/ Squash	"Vigorous"		8 weeks	93% ↑ $\dot{V}O_2$ max
Bouchard	1965	Men	Bicycle Ergometer	HR 130		10 min/sess for 8 weeks	13% ↑ $\dot{V}O_2$ max
Sharkey	1966	Men - College	Stepping	HR 120/150/ 180	3 x week	10 min/sess for 6 weeks	Sign. ↑ only at HR 150/180
Shephard	1968b	Men - Sedentary	Bicycle Ergometer	39%/75%/96% of $\dot{V}O_2$ max	1/3/5 x week	5/10/20 min for 6/4/3/ weeks	Sign. ↑ at all levels but greatest at max int., freq., dur.
Saltin	1968	Men - Sedentary	Bed Rest Followed by Training (Interval and Continuous)	Bed Rest 20 days Training at 60-90% $\dot{V}O_2$	2 x day Mon to Fri 1 x day Sat	53-55 days	33% ↑ $\dot{V}O_2$ max
Saltin	1969	Men - Middle-aged	Continuous and Interval Running	91-98% of $\dot{V}O_2$ max		8-10 weeks	18% ↑ $\dot{V}O_2$ max HR and BL ↓ at Sub Max WL

Table 1 (cont'd)

Author	Date	Subjects	Type of Training	Intensity	Frequency	Duration	Changes
Hartley	1969	Men - Middle-aged	Running		2-3 x week	30 min/sess for 8-10 weeks	14% ↑ $\dot{V}O_2$ max
Pollock	1969	Men - Middle-aged	Walking, Jogging, Running		2 x week - Group 1 4 x week Group 2		(both groups) 17% ↑ $\dot{V}O_2$ max (lit/min) 35% ↑ $\dot{V}O_2$ max (ml/kg/min)
Wilmore	1970	Men - Middle-aged	Jogging		3 x week	12/20 min per sess for 10 weeks	7% ↑
Davies	1971	Men - Young & Middle-aged	Bicycle Ergometer	30/50/80% of $\dot{V}O_2$ max	5/3/1/ x week	20/10/15 min/sess for 8 weeks	Sign. ↑ only at 80%
Mann	1971	Men	Warm-up at 120 HR Interval Work at Near Max HR		5 x week	6 & 8 weeks for 60 mins per sess	35% ↑ $\dot{V}O_2$ max
Pollock	1971	Men - Middle-aged	Walking	HR 132-146	4 x week	40 min/sess for 20 weeks	28% ↑ $\dot{V}O_2$ max
Roberts	1971	Men - Young & Middle-aged	Running/Cycling/ Swimming or Circuit Training	85% of Age-Adj. Max HR		6 weeks	Sign. ↑ only in Run & Lab Groups (Run + Bicycle)

Table 1 (cont'd)

Author	Date	Subjects	Type of Training	Intensity	Frequency	Duration	Changes
Faria	1971	Men - College	Stepping	HR 120-130/ 140-150/ 160-170	5 days week	4 weeks	Sign. ↑ only at HR $>$ 140
Tzankoff	1972	Men - Middle-aged	Handball/Squash/ Paddelball/Tennis		2-3 x week	60-90 min per sess	17% ↑ $\dot{V}O_2$ max 21% ↑ PWC
Pollock	1972	Men - Middle-aged	Walking, Jogging, Running	80-90% of Age. Adj. Max HR	2 x week	45 mins	19% ↑ @ 90% 14% ↑ @ 80%
Kasch	1973	Men - Middle-aged	Cals/Walking/ Running	Initially at 70% then up to 80% of max HR	3 x week	60 min/ sess	17% ↑ $\dot{V}O_2$ max
Nordesjo	1974	Men	Bicycle Ergometer	9 sub at 50% 9 sub at 75% 9 subj at 100%	1/3/5 x week	15/60/120 min/sess for 8 weeks	Sign. ↑ at all levels but greatest ↑ at highest intensity
Byrd	1974	Men - Middle-aged	Walking/Jogging	75-80% of max HR	5 x week	30-40 mins per sess for 12 weeks	Sign. ↑ $\dot{V}O_2$ max
Bonnano	1974	Men - Middle-aged	Walking/Jogging	70-85% of $\dot{V}O_2$ max	3 x week	40-45 mins per sess for 12 weeks	Sign. ↑ $\dot{V}O_2$ max

Table 1 (cont'd)

Author	Date	Subjects	Type of Training	Intensity	Frequency	Duration	Changes
Wenger	1975	Men - young and Middle-aged	Bicycle Ergometer	60 and 100% of $\dot{V}O_2$ max		Each subj. performed the same total amount of work	33% ↑ at 100% 24% ↑ at 60%
Pollock	1975	Men - Middle-aged	Running, Walking or Cycling	85-90% of max HR	3 x week	20 weeks	Sign. ↑ $\dot{V}O_2$ max
Shibayama	1976	Men - Middle-aged	Treadmill Running	66% of $\dot{V}O_2$ max	5 x week	20 min/sess for 2 yrs	Sign. ↑ in first 20 weeks but little change after initial 20 weeks
Miller	1976	Men - Middle-aged	Walking or Jogging	Initially at 65-70% of $\dot{V}O_2$ max then up to 70-80%		28 weeks	Sign. ↑ in both walk and jog groups
<u>Women</u>							
Kilbom	1971	Women - Young and Middle-aged	Walking Bicycle Ergometer	56% of max 59% of max	3 x week	30 min/sess for 6 weeks	2.5% ↑ Walk (non-sign.) 6% ↑ Bike
Kilbom	1971	Women - Young and Middle-aged	Bicycle Ergometer	70% of max	2-3 x week	7 weeks	10% ↑

Table 1 (cont'd)

Author	Date	Subjects	Type of Training	Intensity	Frequency	Duration	Changes
Adams	1973	Women - Older	Cals/Walking & Jogging/ Static Stretching	min of 60% <145 HR	3 x week	3 months	20.8% ↑ $\dot{V}O_2$ max 37% ↑ PWC
Hanson	1974	Women - College	Warm-ups/Cals Walking & Jogging/ Volleyball/ Swimming/ Paddleball		5 x week	60 min/sess for 8 months	Sign. ↑ in working capacity
Edwards	1974	Women - College	Treadmill	HR 125 or HR 145	5 x week	15 min/sess for 4 weeks	↑ in working capacity at both levels
Flint	1974	Women - Young and Middle-aged	Treadmill Walking	75-80% of max HR	3 x week	30 min/sess for 6 weeks	Sign. ↑
Rosentswieg	1975	Women - College	Walking	HR 120/140/ 160	3 x week	15 min/sess for 1 month	No change
Cunningham	1975	Women - Young and Middle-aged	Non-supervised Exercise			Initially 9 weeks then 52 weeks	34% ↑ initially then 5% ↑ after 52 weeks

Table 1 (cont'd)

Author	Date	Subjects	Type of Training	Intensity	Frequency	Duration	Changes
Kearney	1976	Women - College Sedentary	Treadmill	50 or 60% of HR Reserve added to RHR (approx. 134 HR) - session limited to 1000 beats	3 x week	9 weeks	Sign. ↑ at both intensities
Smith	1976	Women - College	Bicycle Ergometer	75% of max HR	3 x week	16 min/sess for 7 weeks followed by 7 weeks of de-training	6.5% ↑ (non-sign.)
Andrews	1976	Women		HR 130-140/ 140-150/ 150-160		20 weeks	Sign. ↑ at all intensities
<u>Men and Women</u>							
Luria	1975	Men and Women - Middle-aged	Walking	2 miles/day in 20 mins (HR <130)	5 x week		Sign. ↑ in aerobic power in 6 of 13 subjs.
Itah	1976	Men and Women - Middle-aged and older	Treadmill Walking	70-85% of max HR	2 x week	5 min/sess	14% ↑ $\dot{V}O_2$ max

a bicycle ergometer at heart rates between 140 and 160 beats per minute several times per month. A few years later, Christensen (1931) trained seven young men on bicycle ergometers 18 to 40 minutes per day at work loads ranging from 480 to 1680 kgm/min. In one subject already partially trained no evidence of training occurred at any level of exercise between 600 and 960 kgm/min. Most subjects increased fitness levels if the work intensity was further increased. Although training intensity was not specified, Robinson (1941) found that over a 26 week period of 'vigorous' track and treadmill training there was a 17 percent increase in $\dot{V}O_2$ max even though initial fitness levels were quite high. Knehr (1942) examined the changes in non-athletic young men over a six month program and found a seven percent increase in $\dot{V}O_2$ max but training was less severe than that of Robinson (1941). Work by Cogswell (1946) studied seven men over a 50 day period of training which included a five minute step test three times per week, two four minute runs on a treadmill per week and two one minute exhaustion rides on a bicycle ergometer three times per week. Results indicate that pulse rates one and a half minutes after the step test showed a large decrease but pulse rates after the treadmill run did not change. It is therefore doubtful that these ~~changes~~ can be accepted as an increase in working capacity.

It seems evident from this early work, where intensity of effort has not been precisely defined, that fairly high intensity of effort is required in order to elicit a training effect in young, healthy men.

It was not until 1957 that more information became available regarding the minimal amount of work necessary to elicit a training effect. Karvonen (1957) found that young men who ran on a treadmill for a half hour per day for four weeks did not show a significant increase in work performance unless heart rates exceeded 150 beats per minute. Sharkey (1966) was in agreement with Karvonen when he suggested that a heart rate of at least 150 beats per minute is necessary in order to promote changes in cardiorespiratory endurance in young men. Faria (1971) also looked at the effects of training at specific heart rates. Three groups of college men trained five sessions per week over a five week period at heart rates of 120 to 130, 140 to 150 or 160 to 170 beats per minute. Results indicate that training at heart rates of 120 to 130 caused no significant increases in the cardiovascular response. As well, there were no significant differences between the 140 to 150 and 160 to 170 training groups. The authors concluded that a training threshold may exist and in this particular group it seems to occur at a heart rate of 140 beats per minute. Work by Hollman (1967) substantiated the theory that a threshold heart rate was necessary to elicit a training effect. A daily ride of thirty minutes on a bicycle ergometer for five weeks at heart rates less than 130 beats per minute led to slight increases in VO$_2$ max but greater increases occurred at heart rates higher than 140. In contrast to

Karvonen (1957) and Sharkey (1966), who suggested a minimum heart rate of 150 beats per minute, Hollman (1967) has indicated that a minimal training heart rate should be between 130 and 135 beats per minute. The data indicates that there is still no agreement, even in young, healthy men regarding the training intensity required to elicit a training effect.

2. Training Studies Involving Middle-Aged or Older Men

Studies utilizing middle-aged and older men involved intensities ranging from 39 percent of $\dot{V}O_2$ max (Shephard 1968b) up to near maximal effort (Wenger 1975). Seigel (1970) proposed that the relationship between amount of training and the increase in $\dot{V}O_2$ max is represented by an S-shaped curve. There is practically no increase in work capacity if the amount of training falls below a certain threshold and that the increase in work capacity rapidly decreases when the amount of training exceeds a certain amount.

Cureton (1964) saw a very high increase in $\dot{V}O_2$ max (93 percent) after a vigorous eight week training program consisting of 15 minutes of calesthenics, 30 to 50 minutes of running and 30 minutes of squash in six middle-aged men. According to Shephard (1965), this very large increase could be due to an alteration in the subject's attitude to all-out runs.

Skinner (1964) followed a group of 15 men aged 35 to 55 who had been sedentary at least three years previous to the six-month study. Training consisted of three days per week at a 'Cureton' program and three days per week of continuous running. Heart rates at standard sub-maximal bicycle ride decreased, all-out run time on the treadmill increased and time for a mile run decreased. The authors concluded that a group of middle-aged sedentary men can increase work capacity by a very intense training program.

Saltin (1969) found that in middle-aged men, after eight to 10 weeks of training, $\dot{V}O_2$ max increased by 18 to 19 percent and heart rate at submaximal work loads decreased by 10 to 20 beats. It was indicated as well that the largest gains in $\dot{V}O_2$ max occurred in men with the lowest initial levels. This very intensive program consisted of a combination of interval running (average intensity 98 to 99 percent of $\dot{V}O_2$ max and of continuous running (average intensity of 91 to 97 percent of $\dot{V}O_2$ max).

Davies (1971) involved 28 men aged 18 to 38 years in an eight week bicycle ergometer program with varied intensity, frequency and duration for each subject. Intensity was set at 80, 50 or 30 percent of $\dot{V}O_2$ max, with a frequency of five, three or one session per week lasting 20, 10 or five minutes. Each subject was allocated to one of 27 procedures. The results indicate that no subject who exercised at or below 50 percent of $\dot{V}O_2$ max showed an increase in maximum aerobic power output. Even at the highest intensities and longest durations the increase in $\dot{V}O_2$ max only ranged from one to nine ml/kg/min. They concluded that in order to increase $\dot{V}O_2$ max the work load must be close to maximum for prolonged periods of time and even then the improvements may be disappointingly small.

Roberts (1971) studied a group of 140 men aged 22 to 62 years over a six week training program of running, cycling, swimming or circuit training at work intensity of 85 percent of age-adjusted maximal heart rate. They concluded that such a program can lead to significant increases in physical working capacity.

Pollock (1972) found a 19 and a 14 percent increase in $\dot{V}O_2$ max in two groups of sedentary men training two days per week for 45 minutes at 80 and 90 percent of maximum heart rate.

Later, Pollock (1975) found that middle-aged men running at 90 percent and walking or swimming at 87 percent of maximal heart rate increased $\dot{V}O_2$ max and oxygen pulse over a twenty week program meeting three times per week for 30 minutes per session.

Wenger (1975) studied a group of 36 firemen (mean age 27.9) over a seven week program. One group trained at 100 percent of pre-training maximum and another group trained at 60 percent. Each subject in the 100 percent group was paired with a subject in the 60 percent group and each partner performed the same total amount of work each session. The group training at 100 percent showed significantly greater improvements in $\dot{V}O_2$ max and physical working capacity (33 percent in 100 percent group and 24 percent in the 60 percent group) but no differences existed between the two groups at submaximal work loads as indicated by decreases in heart rate and blood lactate. Subjects in the low fit group (less than

35 ml/kg/min) showed significantly greater increases in $\dot{V}O_2$ max than the high fit group (greater than 48 ml/kg/min). It was concluded that intensity of effort was of critical importance in achieving increases in $\dot{V}O_2$ max when total work and initial fitness of subjects were equated but that duration of effort was not important. It was also stated that adaptations at submaximal work loads are similar as long as the relative training load is at least 60 percent of $\dot{V}O_2$ max.

This work done with middle-aged and older men seems to point to the fact that fairly intensive activity programs are necessary in order to elicit changes in maximal oxygen uptake. It has been shown that the training benefits occur above 80 percent of maximal heart rate. Various investigators have suggested that physical activity and training programs should be close to maximum for prolonged periods of time, otherwise $\dot{V}O_2$ max will remain unchanged (Davies 1971 and Skinner 1964).

Astrand (1967) has commented that in upright position, maximal stroke volume is apparently attained at 40 percent of $\dot{V}O_2$ max, heart rate being about 120 beats per minute. He feels that it may be important to pass this threshold heart rate of about 120 in order for training to be effective.

Work done by Shephard (1968b) helped to substantiate this theory. A group of 39 non-athletic subjects (mean age 25.5 years) were involved in training programs of different intensity, frequency and duration. Subjects exercised at intensities of 39, 75, and 96 percent of their maximal aerobic capacity as determined from the Astrand Nomogram from a submaximal step test. The frequency of exercise was once per week for six weeks, three times per week for four weeks or five times per week for three weeks at a duration of 5, 10 or 20 minutes. At least one subject and not more than two subjects were allocated to each of the three possible combinations of intensity, frequency and duration of effort. A training effect was achieved at even the lowest intensity although the most effective regime involved the combination of maximum intensity, frequency and duration of effort. No evidence of a training threshold was found. The response was determined largely by the initial fitness of the subjects. The author concluded that due to low levels of habitual activity found in sedentary North American populations it was hardly surprising to find a training effect occurring at heart rates as low as 110 to 120 beats per minute.

Nordesjo (1974) also varied the intensity, frequency and duration of an eight week program of bicycle work. Intensity was set at 50, 75 or 100 percent of maximal oxygen uptake, frequency was set at one, three or five sessions per week and duration at 15, 60 or 120 minutes per session. Of the 27 subjects, 9 worked at 50 percent, nine at 75 percent and 9 at 100 percent of $\dot{V}O_2$ max. Each subject was also allocated to a specific duration so that there were 27 different training programs. Adjustments in work intensity were made at the mid-point of the program so as to ensure that intensity corresponded as closely as possible to the prescribed intensity. Results indicate that the most significant factor for the increase in work capacity was the intensity but that frequency and duration of effort were also significant. Although some improvement was seen in the easiest program the greatest improvements followed training with maximum intensity, frequency and duration.

Pollock (1971) studied the effects of walking on 16 men (mean age 48.9) over a 20 week program of four 40 minute sessions per week. Training heart rates ranged from 132 to 146 beats per minute (63 to 76 percent of maximal heart rate). It was found that $\dot{V}O_2$ max increased by 28 percent and that heart rates at a submaximal work load decreased by four to 17 beats per minute. It was concluded that the large increases in $\dot{V}O_2$ max were due to the low initial fitness level of the group.

Luria (1975) also studied the effects of a walking program on 13 men and women aged 22 to 55 years. The ten week program consisted of walking two miles per day within 30 minutes, five days per week. Heart rates at the end of the first week averaged 118 beats per minute and at the end of the program averaged 107. Although no subject exercised at a heart rate greater than 130 beats per minute, six of the 13 subjects showed a training effect. These results seem to be in agreement with those of Shephard (1968b) and Pollock (1971) in that training effects can be achieved after a program involving a fairly low level of intensity.

Kasch (1973) followed men aged 39 to 60 over a two year program consisting of three 30 minute sessions per week. Initially, training heart rates corresponded to approximately

70 percent of maximum heart rate but mid-way through the program subjects were working at 87 percent of maximum heart rate. Over this longer period there was a 17 percent increase in $\dot{V}O_2$ max.

Wenger (1975) saw an increase in $\dot{V}O_2$ max of 24 percent in a group of 36 men (mean age 27.9) while training at 60 percent of $\dot{V}O_2$ max.

Other investigators have shown increases in $\dot{V}O_2$ max by working at intensities ranging from 66 to 85 percent of $\dot{V}O_2$ max (Bonanno 1974, Itah 1976, Shibayama 1976, and Miller 1976). Bonanno (1974) reported increases in $\dot{V}O_2$ max, oxygen pulse and physical working capacity in middle-aged men over a 12 week period consisting of walking/jogging three times per week for 40 to 55 minutes per session at intensities of 75 to 85 percent of $\dot{V}O_2$ max. Work by Itah (1976) involving 24 men and six women aged 40 to 75 resulted in a 14 percent increase in $\dot{V}O_2$ max after walking five minutes, two times per week at intensities corresponding from 70 to 85 percent of $\dot{V}O_2$ max. Shibayama (1976) followed six men aged 30 to 47 years for a two year period and found noticeable increases in respiro-circulatory adaptations after training 20 minutes per day, five days per week at approximately 66 percent of $\dot{V}O_2$ max. It was found that the greatest changes occurred during the first twenty weeks of the program and that further continuation of training showed little effect in improvement of circulatory function. Miller (1976) saw increases in $\dot{V}O_2$ max in a group of men (mean age 48) involved in a walking and jogging program. During the initial twelve weeks intensity corresponded to 60 to 65 percent of maximal capacity but over the next 16 weeks intensity was as high as 80 percent of maximum.

It is evident that training studies of a low to moderate intensity can lead to an increase in $\dot{V}O_2$ max, although the changes were not as great as in those studies involving a higher intensity.

3. Training Studies Involving Women

Until recently, very little research has been done regarding the training effects occurring in women involved in a regular exercise program.

In 1971, Kilbom (1971a) examined the responses of two groups of untrained women aged 19 to 45 years of age to three 20 minute sessions of exercise over a six week period. One group rode a bicycle ergometer at an average intensity of 59 percent of $\dot{V}O_2$ max (range 52 to 63 percent) and the other group walked at an intensity of 56 percent of $\dot{V}O_2$ max (range 44 to 63 percent). The bicycle group had a six percent increase in $\dot{V}O_2$ max while heart rates decreased by five to 10 beats at a standard submaximal work load. The group involved in the walking program had a non-significant 2.5 percent increase in $\dot{V}O_2$ max and slight decreases in submaximal exercise heart rates. It was concluded that training three times per week for a six week period at 50 to 60 percent of $\dot{V}O_2$ max may affect healthy untrained women.

Kilbom (1971b) also investigated the effects on 33 women aged 19 to 64 of a seven week program consisting of riding a bicycle two to three times per week at 70 percent of $\dot{V}O_2$ max. There was an 11 percent increase in maximum aerobic power and a decrease in submaximal exercise heart rates of 10 to 15 beats per minute.

A number of investigators examined the effects of training on sedentary college women (Edwards 1974, Rosentswieg 1975, Kearney 1976, and Smith 1976). Edwards (1974) has shown that sedentary college women (aged 17 to 21) exercising at either 125 or 145 heart rates on a treadmill 15 minutes daily for four weeks had significant increases in $\dot{V}O_2$ max. She concluded that in sedentary young females a work intensity eliciting a heart rate of 125 beats per minute provided a sufficient but not minimal stimulus for training.

Rosentswieg (1975) trained 30 college women (mean age 19.2 years) three times per week for 15 minutes for a one month period by having them walk at heart rates of 120, 140 or 160 beats per minute. No significant differences were found in fitness levels after training in any of the groups. It was concluded that programs of walking three times per week over a one month period do not appear to be sufficiently stressful to induce changes in selected cardiovascular parameters in young women.

Kearney (1976) trained a group of 27 sedentary college women (aged 17 to 21) three times per week on a treadmill for a nine week period. The authors felt that the amount of physiological stress experienced during an exercise bout is determined not only by the quantity of external work completed but also by the rate at which the work is performed. Therefore the subjects exercised at heart rates of either 50 or 60 percent of the heart rate reserve added to the resting heart rate with the duration of each session being limited to 1000 beats above the resting values. Results indicate that both intensities caused increases on $\dot{V}O_2$ max and although there were no significant differences between the two training groups there appeared to be a trend in favour of the more vigorous intensity.

Work done by Smith (1976) found that a seven week program of riding a bicycle ergometer for 16 minutes three times per week at a heart rate of 75 percent of the maximum heart rate range (.75 max HR - RHR) + RHR led to a non-significant 6.5 percent increase in maximal aerobic power in 16 moderately active female students aged 18 to 25 years of age.

Work was done on older women by Hanson (1974). Eight sedentary women aged 20 to 44 were involved in an eight week unquantified program of warm-up and calisthenics, walking/jogging and volleyball, swimming or paddleball. There are significant increases in work capacity and decreases in oxygen utilization, CO_2 production and heart rates at a given exercise load.

Flint (1974) examined changes occurring in seven women aged 23 to 40 years who walked on a treadmill for 30 minutes, three times per week at heart rates between 75 and 80 percent of maximal heart rate. There was a decrease in heart rate at a standard submaximal exercise load and an increase in predicted maximum aerobic power.

Cunningham (1975) studied 17 women aged 21 to 48 (mean age 31 years) over a nine week training program followed by a 52 week period of non-supervised exercise. After the initial nine week period $\dot{V}O_2$ max increased by 34 percent but after the following 52 weeks the six subjects who continued to exercise increased their $\dot{V}O_2$ max by another five percent.

Andrews (1976) studied 43 healthy, middle-aged women over a 20 week period consisting of training at heart rates of 130 to 140, 140 to 150 or 150 to 160 beats per minute. Maximal oxygen uptake increased in all groups but in general the greatest increases occurred in those with the lowest initial level of fitness.

A much older group of women (mean age 66.7 years) was studied by Adams (1973). It was found that physical working capacity and maximal oxygen uptake increased by 37 and 20 percent respectively after a program consisting of calisthenics, progressive walking and jogging and static stretching. It was concluded that the older female is trainable and suggests that the magnitude of capacity for training is not greatly different from that of younger individuals if compared on a percentage basis.

The work done with young college women is contradictory. Various investigators have seen training effects occur in walking programs with heart rates as low as 125 beats per minute (Edwards 1974) and in bicycle programs at at intensities of 59 and 70 percent of $\dot{V}O_2$ max (Kilbom 1971a and Kilbom 1971b). Others have found no significant increases in $\dot{V}O_2$ max with programs consisting of walking at 56 percent of $\dot{V}O_2$ max (Kilbom 1971a), at heart rates ranging from 120 to 160 beats per minute (Rosentswieg 1975) or with bicycle ergometer programs at 75 percent of maximal heart rate (Smith 1976). These findings concur with those seen in young men, in that no clearly identified training threshold can be seen.

In work with older women a bicycle program involving an intensity of 59 percent of $\dot{V}O_2$ max did not lead to significant training benefits (Kilbom 1971a). Other investigators have found that walking at heart rates of 75 to 80 percent of maximal heart rate (130 and over) have resulted in increases in aerobic power (Flint 1974). The training threshold in middle-aged and older women appears to be around 50 to 60 percent of $\dot{V}O_2$ max but as with the work done involving middle-aged and older men the minimal training intensity required cannot be clearly defined.

In summary, results from the numerous training studies involving young, middle-aged and older men and women have been contradictory. The minimal intensity of effort required to evoke training benefits in young fit men has been identified at heart rates ranging from 130 to 135 beats per minute (Holleman 1967 and Bouchard 1965). Earlier research has indicated that a heart rate of at least 150 beats per minute is required in young men (Karvonen 1957). Various studies involving middle-aged sedentary men have also been controversial. A number of investigators have suggested that it is necessary to work at fairly high intensities in order to elicit training benefits (Davies 1971, Roberts 1971, Cureton 1964, and Skinner 1964). On the other hand, work by Shephard (1968b) and Nordesjo (1974) have resulted in training benefits in programs requiring 39 and 50 percent of $\dot{V}O_2$ max respectively. Increments have been seen in programs involving women at work intensities requiring 59 to 70 percent of $\dot{V}O_2$ max (Kilbom 1971a and Kilbom 1971b). Benefits have been seen by some investigators at heart rates ranging from 125 to 160 beats per minute (Andrews 1976 and Edwards 1974) but not by others (Rosentsweig 1975).

It is generally found that the training regimens involving a higher intensity of effort result in greater increases in maximal aerobic power. The individual's initial level of fitness has also been identified as a very significant factor. Those with a low pre-training level of fitness will often show the highest percentage increase in $\dot{V}O_2$ max with training (Wenger 1975, Pollock 1971, Saltin 1969, and Shephard 1968b). The available data has not clearly indicated the optimal amount of effort and type of exercise necessary to elicit significant training benefits.

Methods of Exercise Prescription

An exercise prescription must be based on sound physiological and biomedical principles. No one conditioning program will be optimal for all persons or even for the same person over an extended period of time (Skinner 1971). In many group programs every participant must adapt to a standard exercise regime. Wilmore (1974) has recommended that exercise be programmed to fit each individual subject.

The age, health, fitness and needs of the patient must be considered as well as time and facilities available and the personal preference of the individual when prescribing an exercise program (Skinner 1971). According to Hellerstein (1973) the "pleasure principle" is important, long-term activity programs must be gratifying and pleasureable or at least not distasteful.

The first step involved in presenting a training program is a comprehensive medical examination (Wilmore 1976). Following medical clearance an exercise stress test should ideally be performed (Wilmore 1976, Cunningham 1974, and Hellerstein 1973). Hellerstein (1973) feels that the key for an appropriate exercise prescription is the assessment of the individuals physical fitness. A quantitative evaluation of heart rate, electrocardiogram, blood pressure and symptoms occurring are necessary for a rational approach to exercise prescription. It has been advocated that the end-point of the test be volitional or symptom-limited (Wilmore 1976).

At no time should the effort of training exceed that of the exercise test (Hellerstein 1973).

The exercise prescription is based on defining the type of activity and the frequency, duration and intensity of that activity (Wilmore 1976, Skinner 1971, Wilmore 1974, Hellerstein 1973, Jetté 1975a, Cunningham 1974, and Kasch 1977).

Activities of an endurance nature are recommended by most investigators (Wilmore 1976, Skinner 1971, Wilmore 1974, and Hellerstein 1973). Hellerstein (1973) suggests that activities involve a substantial and sustained increment in metabolic, cardiovascular, respiratory and neuromuscular functions. Activities which involve rhythmic contraction of large groups of muscles such as brisk walking, jogging, running, swimming, cycling, hiking would be most beneficial.

The frequency of effort necessary to elicit benefits seems to be three or four sessions per week (Wilmore 1976, Hellerstein 1973, and Cunningham 1974). Hellerstein (1973) found that men exercising 2.3 to 3.0 times per week showed greater improvement in aerobic capacity and ST-T responses than did those exercising one to two sessions per week. It was noted as well that there were no differences in test results between those exercising 3.5 to 5 times per week and those exercising 2.3 to 3.0 sessions per week. Recent work by Crews (1976) also found no significant differences between groups working five days and three days per week. Jackson (1968) also showed that little difference occurred in training benefits if frequency of exercise exceeded three times per week.

Skinner (1971) suggests that the frequency of a program should initially be two to three times per week but later on three to five weekly sessions are advisable. Wilmore (1976) suggests a Monday, Wednesday, and Friday for endurance activities with a recreational activity added on one or more of the alternate days.

Most investigators recommend that the duration of a program be between 30 to 40 minutes. Wilmore (1976) and Hellerstein (1973) recommend thirty minutes per session. Cunningham (1975) feels that the duration of a program should be much more specific to the individual's daily caloric output, (this averages out to be 30-60 minutes in normal individuals).

The majority of work indicates that the intensity of a training program is the most important aspect of the exercise prescription (Wilmore 1976, Jetté 1975a, and Kasch 1977). Work by Morris (1973) has also indicated that intensity is the prime factor in reducing the risk of developing coronary disease. Training effects have been seen at intensities as low as 39 percent of maximal aerobic power (Shepherd 1968b) but ordinarily training benefits will be greater at higher training intensities. It is generally accepted that training intensity be between 50 and 70 percent of $\dot{V}O_2$ max (Wilmore 1976, Skinner 1971, and Hellerstein 1973). Hellerstein (1973) suggests that training levels of 50 to 70 percent of aerobic capacity are applicable to middle-aged men and older normal subjects and selected post-cardiac patients and are preferred over potentially dangerous maximal levels.

Cunningham (1975), Skinner (1971), and Hellerstein (1973) advocate the use of METS (1 MET = 3.5 ml of O_2/kg/min) and Training Heart Rate (THR) for their patients. Cunningham (1975) uses Balke's formula, namely, 60 percent plus the meximum MET value (i.e. Training Intensity equals $\frac{60 = \text{max. METS x max. METS}}{100}$). The training intensity in terms of METS can then be converted to walking or running speeds or to training heart rates. Hellerstein (1973) indicates that once the oxygen cost of a desired training level has been determined any number of different activities can be chosen from standard tables listing the metabolic costs of various activities.

The use of training heart rates is probably the most popular method now used in prescribing exercise. The use of an arbitrary heart rate is not a good method due to the fact that a heart rate of 150 beats per minute represents 77 percent $\dot{V}O_2$ max in a 24 year old whereas in a 55 year old it is equal to 91 percent of $\dot{V}O_2$ max. Hellerstein (1973) also discourages the use of age-adjusted maximal heart rates to prescribe training levels. In special cases the restriction to a rise in heart rate may be due to intrinsic diseases of the sino-atrial or antrio-ventricular nodes or to the appearance of signs and symptoms which preclude exercising at a higher workload. Therefore Karvonen's formula (1957) [THR (training heart rate) = max AAHR (age-adjusted heart rate) - RHR (resting heart rate) x 60 or 70 percent + RHR] which utilizes age-adjusted maximal heart rates would be unacceptable. It is recommended that the highest functional heart rate achieved during pre-testing be used to choose intensity of an individual's training program.

Wilmore (1976) has indicated that since a linear relationship exists between heart rate and $\dot{V}O_2$ at varying levels of exercise, heart ~~rate~~ be used to estimate the intensity on the basis of a set percentage of $\dot{V}O_2$ max. The author has noted four main advantages of using training heart rate when controlling exercise intensity. A training heart rate can apply to all activities whether they be walking, jogging, swimming, cycling, or playing tennis. It also has a built-in control for changes in both physical condition and for changes in environmental conditions such as heat, humidity, or altitude. Lastly, training heart rate can be used to monitor activity levels in the elderly, debilitated or physcially disabled individuals who must work at much lower levels of intensity. In his experience, Wilmore (1976) has found that all subjects in his programs felt the use of training heart rate was effective and self-satisfying and no one reported trouble in determining their exercise pulse rates or in maintaining heart rate during their exercise periods.

Hellerstein (1973) has also indicated the value of the HR x Systolic BP product. This indirect measure of myocardial oxygen consumption correlates well with the appearance of clinical and ECG evidence of coronary insufficiency. Target training levels could then be based on excessive changes in this double product.

Nordesjo (1974) has indicated that there are certain disadvantages in using heart rate to control the work rate during each training session. It was found in his training study that the difference in heart rate between two training sessions, when training at the same work rate and in two consecutive days of training, varied between -25.5 and +24.6 beats per $\min^{-1}$.

The use of training heart rate as a means of prescribing exercise intensity has also been questioned by Jetté (1975a). They have indicated that their subjects had some difficulty in monitoring their heart rates. It was also found that the use of training heart rate had limited application in cardiac patients especially when age-adjusted maximal heart rates were utilized. An alternative or complimentary method has been advocated that provides each individual with a specific time to cover a set distance. Two regression equations were developed employing height, 60 percent of $\dot{V}O_2$ max and weight or resting heart rate that indicated a walking or jogging speed for one mile. The formula based on height, weight and 60 percent of $\dot{V}O_2$ max has been employed in their rehabilitation and prevention programs and each subject has been provided with a personalized program.

In summary, an exercise prescription should take into consideration age, health and fitness level of the individual as well as facility availability and enjoyment of the specific activity. After being given medical clearance an individual should undergo a volitional or symptom-limited exercise stress test which quantitatively evaluates heart rate, electrocardiogram, blood pressure and symptoms. An exercise prescription is then based on defining the type of activity and the frequency, duration and intensity of that specific activity. Activities of an endurance nature such as brisk walking, jogging, swimming and cycling are recommended. These activities should be carried out three to four times per week for a 20 to 40 minute period. It is generally held that the intensity of the program is of prime importance. Although training benefits have been seen at intensities ranging from 39 percent of $\dot{V}O_2$ max (Shepherd 1968b) up to 100 percent of maximal capacity (Wenger 1975) it has been recommended that middle-aged and older individuals exercise at an intensity between 50 and 70 percent of maximal aerobic capacity. Exercise has normally been presented in terms of training heart rate, but recently use of METS, double product and time required to cover a certain distance have been employed. Regardless of the method used to prescribe exercise it is essential that the conditioning program be based on the individuals specific response to an exercise test.

CHAPTER III

METHODOLOGY

Introduction

The purpose of this study was to examine the effects of a 12 week training program consisting of walking or jogging on a treadmill at a speed corresponding to 60 percent of peak oxygen consumption in sedentary male and female subjects.

The methodology is presented under the following headings:

Subjects
Laboratory Procedures and Equipment
Initial Testing and Orientation of the Subjects
Pre-training Peak Oxygen Consumption Test
Exercise Prescription Procedures
Submaximal Exercise Test
Exercise Program
Adjustment to the Exercise Program
Quantification Procedures
Post-training Procedures
Summary of Testing and Training Procedures
Statistical Analysis

Subjects

Fourteen male and 12 female volunteer subjects between the ages of 35 and 53 were recruited for the study.

The subjects were chosen by placing an advertisement in the University of Ottawa Gazette. Criteria for their selection was that they had not participated in a regular physical activity or training program over the past three years.

The selected subjects were given a medical examination prior to the onset of the testing program. Two subjects were excluded from the study during the initial screening. One subject was excluded due to extreme obesity and another subject demonstrated multiple ectopic activity during her initial exercise stress test.

The subjects were placed in either the experimental group or the control group according to their preference. Seven males and six females were in the control group. The anthropometric data of the subjects are shown in Table 2.

Subjects in the experimental group took part in the initial testing and orientation procedures, pre-training peak oxygen consumption test, exercise prescription procedures, submaximal exercise test, exercise program, adjustment of exercise prescription, quantification procedures and post-training procedures. Subjects in the control group took part in all these sessions with the exception of the exercise program, adjustment of exercise prescription and the quantification procedures.

Laboratory Procedures and Equipment

The variables of $\dot{V}O_2$, $\dot{V}_E$, $\dot{V}_ECO_2$ and respiratory rate were measured by the modified open circuit method (Consolazio 1963). Expired oxygen and carbon dioxide were analyzed by

Table 2

ANTHROPOMETRIC DATA FOR
EXPERIMENTAL AND CONTROL SUBJECTS

	Experimental Group				Control Group		
Subjects	Age (Years)	Weight (kg)	Height (cm)	Subjects	Age (Years)	Weight (kg)	Height (cm)
Males				Males			
G.M.	47	78.1	167.6	A.M.	35	68.18	167.6
J.G.	37	91.7	163.7	R. Lup.	38	92.05	168.3
M.M.	40	69.5	179.0	A.P.	49	77.5	165.1
R. Lay.	35	79.5	191.0	R.D.	49	76.36	171.4
J.K.	53	79.4	182.8	M.C.	53	79.55	175.8
F.D.	49	76.9	177.8	R.R.	39	81.59	175.8
R. Lav.	46	72.2	163.8	J.S.	45	81.59	174.2
x̄	43.9	79.19	175.1	x̄	44.0	79.55	171.2
SD	6.6	7.06	9.63	SD	6.76	7.17	3.96
Females				Females			
P.K.	40	71.25	158.7	A.D.	47	59.32	169.5
C.J.	46	52.5	156.8	J.A.	37	58.52	157.5
J.J.	43	66.7	173.4	L.D.	49	61.82	166.4
G.D.	49	59.4	161.3	E.F.	51	61.73	165.7
L.B.	42	50.0	149.9	M.C.	38	55.11	165.1
B.T.	45	55.8	172.9	F.A.	46	60.34	161.9
x̄	44.2	59.23	162.2	x̄	44.7	59.47	164.4
±SD	3.9	8.33	8.49	±SD	5.82	2.5	3.78

Rapox and Capnograph analysers respectively. Minute ventilation was calculated from the collection of expired gas in a 120 liter Collins Tissot Gasometer. Respiratory rate, expired oxygen and expired carbon dioxide were coupled to separate channels of the Narco Bio Systems Four B Polygraph which gave separate recordings for each variable.

Variables involved in measuring oxygen consumption were corrected for standard temperature and pressure. Oxygen consumption was calculated with an automated program on a Wang 6000 calculator. This program was developed by the Department of Kinanthropology at the University of Ottawa using a modified method described by Consolazio (1963).

The heart rate was measured by use of a Quinton ECG preamplifier, Model No. 607. Disposable electrodes were placed in the CM-5 position (chest, manubrium, V-5 position) for the recording of the electrocardiogram. A Quinton ECG Isolation amplifier was coupled to the Four B Polygraph to give continuous heart rate recordings. ECG was continuously displayed on a Sanborn Model No. 768-100 Gating Amplifier Oscilloscope. ECG tracings were recorded from a Cambridge VS-4 Electrocardiogram. Heart rates were also monitored from the Digital Display of the Quinton Exercise Cardiotachometer, Model No. 609.

Blood pressure was measured using an aneroid sphygmanometer by the ausculatory method.

During the exercise prescription procedures, the speed of the treadmill was progressively increased by means of a Quinton Model No. 1607 Heart Rate Controller.

Venous blood samples for lactate were taken from the brachial vein via a number 20-gl Becton, Dickinson and Co. Vacuatainer Syringe. All blood samples were taken by a qualified laboratory technician. Blood samples were enzymatically assayed for lactate using the lactate kit, cat. no. 15927 TLAA, Biochemica test combination and analysed spectrophotometrically with a precision Spectrophotometer, model no. 505, Bosch and Lamb Co., Rochester, N.Y.

Initial Testing and Orientation of the Subjects

During the first visit to the Bio-Kinetics Laboratory at the University of Ottawa the subjects were required to fill out various questionnaires and consent forms. At this time, anthropometric measurements, resting electrocardiogram, strength, muscular endurance, flexibility and vital capacity measurements were taken.

During the second visit to the laboratory each subject completed a multi-stage treadmill test (Jetté 1975b). The first treadmill test served as an introductory session. The subject was first instructed on how to walk on the treadmill. After a three minute accommodation period of walking at three miles per hour at a 2.5 percent elevation the subject was seated for a two minute rest period. At this time instructions were given for the test. Subjects were told to stay on the treadmill until they felt that they could not continue walking for another full minute. However, if they experienced any discomfort such as dyspnea, dizziness, chest pain or acute fatigue the test would be discontinued. The electrocardiogram and the blood pressure were monitored at each stage. Each subject began the test by walking at three miles per hour at a 2.5 percent elevation. Every two minutes the angle or the speed of the treadmill was automatically increased. At the point which the subject indicated that he could not continue the speed and angle of the treadmill were decreased to 2.5 miles per hour and zero percent elevation respectively and the subject continued to walk for one minute. After this cool-down period the subject was seated and heart rate, electrocardiogram and blood pressure were monitored for a three minute period.

Electrocardiogram was recorded for a five second period at the following times:

1. Sitting (pre-test)
2. At the end of each minute of the three minute accommodation period.
3. At the end of each minute of the initial two minute recovery period (sitting).
4. At the end of the second minute of each two minute stage in the multi-stage treadmill test.
5. At the end of the one minute recovery period (walking).

6. At the end of each minute of the three minute recovery period (sitting).

Blood pressure was recorded at the following times:

1. Sitting (pre-test).
2. At the beginning of the second minute of each two minute stage in the multi-stage treadmill test.
3. At the end of the one minute recovery period (sitting).
4. At the end of the third minute of the recovery period (sitting).

Pre-Training Peak Oxygen Consumption Test

The peak $\dot{V}O_2$ test was repeated during the third visit to the laboratory. The results of this second peak $\dot{V}O_2$ test were employed to determine the pre-training peak $\dot{V}O_2$ of the subjects and for establishing the exercise prescription for all but four subjects. The results of the first treadmill test were used for three subjects (PK, RL, MM) due to the fact that their peak $\dot{V}O_2$ was higher during the first test. A third peak $\dot{V}O_2$ test was given to one subject (FD). This person was suffering from a cold during the two previous tests and required a third test.

The protocol followed during the second multi-stage treadmill test was identical to that followed during the initial orientation and testing procedures.

Exercise Prescription Procedures

After determination of their peak $\dot{V}O_2$ the subjects returned to the laboratory in order to determine their exercise prescription at 60 percent of their peak $\dot{V}O_2$.

Electrode placement for heart rate recording was identical to that used in previous testing. Subjects began to walk at three miles per hour at zero percent elevation. The speed of the treadmill was progressively increased until their pre-determined heart rate at 60 percent of peak $\dot{V}O_2$ was attained. There was no further increase in speed of the treadmill once this heart rate had been attained. In order to assure that a steady state had been achieved the subject walked for six minutes at this speed. Oxygen consumption was measured for a 30 second period at the end of the fourth and the sixth minutes. If the average of these two measurements did not correspond to 60 percent of peak $\dot{V}O_2$ in ml/kg/min ± 1.5 ml the speed was adjusted so that the correct workload was achieved. In the case where an adjustment of speed was necessary the subject walked for another four minutes at the new speed. When it had been established that the subject was walking or jogging at a speed corresponding to 60 percent of his peak $\dot{V}O_2$ the speed of the treadmill in miles per hour and in revolutions of the belt per minute was recorded. This was the speed at which the subject trained during the first half of the exercise program.

Submaximal Exercise Test

During the first exercise session of the training program each subject in the experimental group walked or jogged on the treadmill for a six minute period at their prescribed training speed.

Heart rate and ECG were recorded for a five second period at the end of each minute of exercise. The average of the heart rates attained at the end of the fifth and sixth minutes represented the submaximal exercise heart rate (SMEHR). Expired air was collected at the end of the fourth and sixth minutes of exercise. Blood samples were taken at the end of the third minute of the sitting recovery period.

Subjects in the control group returned to the laboratory for this session within three days of their exercise prescription procedures.

Exercise Program

All subjects in the experimental group took part in the exercise program. The program was held three times per week and continued for a twelve week period. All but four subjects completed the twelve week program. Due to unforseen scheduling problems three subjects (MM, CJ, JJ) completed 33 sessions while another subject (GM) completed only 27 sessions.

Subjects reported to the laboratory at their appointed times. The exercise session was preceeded by a warm-up period consisting of walking for three minutes at a speed of three miles per hour. At the end of the warm-up, the speed of the treadmill was progressively increased so that the subject was exercising at his prescribed speed.

ECG was monitored during one exercise session each week. Heart rates were determined from the ECG recordings.

Each exercise session was followed by a two minute cool-down period consisting of walking at 2.5 miles per hour.

The schedule during the exercise session was as follows:

Week 1 - session 1 - Submaximal exercise Test plus 10 minutes of exercise at prescribed speed
session 2 - 15 minutes at prescribed speed
session 3 - 15 minutes at prescribed speed
Week 2 - session 1 - 20 minutes at prescribed speed
session 2 - 20 minutes at prescribed speed
session 3 - 20 minutes at prescribed speed
Week 3 - session 1 - 25 minutes at prescribed speed
session 2 - 25 minutes at prescribed speed
session 3 - 25 minutes at prescribed speed
Week 4 - session 1 - 30 minutes at prescribed speed
session 2 - 30 minutes at prescribed speed
session 3 - 6 minute Quantification period followed by 20 minutes at prescribed speed
Week 5 - session 1 - 30 minutes at prescribed speed
session 2 - 30 minutes at prescribed speed
session 3 - 30 minutes at prescribed speed
Week 6 - session 1 - 30 minutes at prescribed speed
session 2 - Multi-Stage Treadmill Test
session 3 - Exercise Prescription Procedures
Week 7 - session 1 - 20 minutes at adjusted speed
session 2 - 20 minutes at adjusted speed
session 3 - 20 minutes at adjusted speed
Week 8 - session 1 - 25 minutes at adjusted speed
session 2 - 25 minutes at adjusted speed
session 3 - 6 minute Quantification period followed by 20 minutes at adjusted speed
Week 9 - session 1 - 25 minutes at adjusted speed
session 2 - 25 minutes at adjusted speed
session 3 - 25 minutes at adjusted speed
Week 10 - session 1 - 30 minutes at adjusted speed
session 2 - 30 minutes at adjusted speed
session 3 - 30 minutes at adjusted speed

Week 11 - session 1 - 30 minutes at adjusted speed
session 2 - 30 minutes at adjusted speed
session 3 - 30 minutes at adjusted speed
Week 12 - session 1 - 30 minutes at adjusted speed
session 2 - 30 minutes at adjusted speed
session 3 - 6 minute Quantification period followed by
20 minutes at adjusted speed

Adjustment of the Exercise Program

All subjects involved in the experimental group had their exercise prescriptions adjusted during the sixth week of training. During the second visit of the sixth week of training each completed a peak $\dot{V}O_2$ test. At his next visit to the laboratory each subject performed the exercise prescription procedures as previously outlined. The adjusted training speed was employed until the completion of the training program.

Quantification Procedures

All subjects in the experimental group took part in the quantification procedures. The purpose of these sessions were twofold: firstly, to examine the changes occurring in submaximal exercise heart rates (SMEHR) as the training program progressed and secondly, if there was a substantial decrease in these exercise heart rates, to examine the changes in speed necessary to elicit the initial SMEHR.

The quantification sessions were held during the third exercise session of the fourth, eighth and twelfth week of training.

Electrode placement was similar to that used in previous testing. The subject began to walk or jog on the treadmill at his training speed and continued for a six minute period. Oxygen consumption was measured for a thirty second period at the end of the fourth and sixth minutes. The average of the heart rates attained at the end of the fifth and sixth minutes were recorded as the quantification heart rate (QHR). If the QHR was five or more beats per minute lower than the initial SMEHR then the subject returned to the treadmill for the second portion. The subject started walking or jogging at his initial training speed and by means of the Heart Rate Controller the speed of the treadmill was progressively increased until the heart rate was equal to the initial SMEHR. At this time no further increases in treadmill speed were made and the subject continued to walk or jog for a six minute period. Oxygen consumption was measured for a 30 second period at the end of the fourth and sixth minutes and heart rates were recorded from the ECG at the end of each minute. The speed of the treadmill in miles per hour and the revolutions of the belt per minute were recorded at this time.

Post-Training Procedures

During the third session of the twelfth week of exercise the subjects in the experimental group repeated the submaximal exercise test. Procedures used in the collection and analysis of gas and the collection and determination of blood lactate were identical to those followed in the pre-training session. The final peak $\dot{V}O_2$

test was performed during the following week. Procedures followed for this test were identical to those used in the pre-training sessions.

All subjects in the control group returned to the laboratory on two different occasions twelve weeks after their initial visits and completed the submaximal exercise test and the multi-stage treadmill test.

Summary of Testing and Training Procedures

I. Initial Testing and Orientation Procedures

Visit 1. i) Medical examination (if not already completed by the subject's personal physician).

ii) Health questionnaire and consent form.

iii) Resting electrocardiogram, anthropometric, strength, muscular endurance, flexibility and vital capacity measurements.

Visit 2. Multi-stage treadmill test.

II. Pre-training peak $\dot{V}O_2$ test.

III. Exercise Prescription Procedures - determination of training speed for first six weeks of program.

IV. Submaximal Exercise Test

i) Determination of submaximal exercise heart rate.

ii) Determination of blood lactate level three minutes after the submaximal exercise test.

V. Exercise Program (for those in the experimental group only).

VI. Mid-Training Peak $\dot{V}O_2$ Test (for those in the exerimental group only).

VII. Adjustment of Exercise Prescription - determination of training speed for the last six week of program (for those in the experimental group only).

VIII. Post-Training Procedures

Visit 1. Submaximal exercise test to determine the submaximal exercise heart rate and blood lactate levels.

Visit 2. Post-training peak $\dot{V}O_2$ test.

Statistical Analysis

The variables of peak $\dot{V}O_2$, submaximal exercise heart rate and blood lactate which were determined before and after training in both the experimental and control group were analysed using a one-way analysis of covariance. Since only two groups were being compared post-hoc procedures were not necessary. The data obtained from the men and from the women were analysed separately and the data were also combined and analysed as one group.

The heart rates attained during the first portion of the quantification session was analysed using a one-way analysis of variance. The data obtained from the men and from the women were analysed separately and the data was also combined and analysed as one group. If significant differences were found among the variables the Scheffé test was undertaken to determine where these differences occurred.

The level of confidence chosen for the statistical analysis was $P < 0.05$.

The $\dot{V}O_2$ and speed of treadmill elicited during the second portion of the quantification session were averaged and the percentage change occurring in each subject was examined.

The exercise prescription speed obtained in this study was compared to the speed obtained from the formula developed by Jetté (1975a). Pearson's product moment coefficient of correlation was utilized for this analysis.

CHAPTER IV

RESULTS

Introduction

The purpose of this study was to examine the effects of a twelve week training program consisting of walking or jogging on a treadmill at a speed corresponding to 60 percent of peak oxygen consumption.

This chapter presents the results under the following headings:

1. Peak Oxygen Consumption
2. Submaximal Exercise Test
    i) Heart Rate
    ii) Blood Lactate
3. Quantification Sessions
    i) Heart Rate
    ii) Changes in Heart Rate, $\dot{V}O_2$ and Speed of Treadmill to Elicit Initial Training Heart Rate

1. Peak Oxygen Consumption

Data for peak $\dot{V}O_2$ for both the experimental and control groups as obtained in the pre and post training evaluations are shown in Table 3. There were statistically significant differences in peak $\dot{V}O_2$ as a result of the training for the men ($p < 0.1$), for the women ($p < 0.005$) and for the men and women combined ($p < 0.001$).

Table 3

Means, Standard Deviations and Percent Change for Pre and Post Training Peak $\dot{V}O_2$ (ml/kg/min and lit/min)

		Pre	Post	% Change
Men	- Exp (ml/kg/min)	34.51	39.10	13.13% ↑ *
		±5.07 SD	±4.35	
	(lit/min)	2.69	2.95	9.67% ↑ *
		± .47	± .39	
	- Control (ml/kg/min)	34.11	34.82	2.08% ↑
		±5.63	±3.89	
	(lit/min)	2.69	2.76	2.60% ↑
		± .46	± .27	
Women	- Exp (ml/kg/min)	26.57	31.02	16.75% ↑ *
		±3.58	±2.82	
	(lit/min)	1.56	1.83	17.31% ↑ *
		± .16	± .15	
	- Control (ml/kg/min)	25.88	26.03	.58% ↑
		±1.99	±2.32	
	(lit/min)	1.54	1.54	No ↑
		± .13	± .15	
Men & Women	- Exp (ml/kg/min)	30.84	35.37	14.69% ↑ *
		±5.95	±5.48	
	(lit/min)	2.16	2.43	12.5 % ↑ *
		± .69	± .66	
	- Control (ml/kg/min)	30.31	30.[illegible]	1.48% ↑
		±5.98	±5.46	
	(lit/min)	2.08	2.20	5.77% ↑
		± .59	± .67	

* $p < 0.05$

2. Submaximal Exercise Test

i) Heart Rate

Data for submaximal exercise heart rate as obtained at the pre and post evaluations are shown in Table 4. There were statistically significant decreases in submaximal exercise heart rates as a result of training for the men ($p < 0.025$), for the women ($p < 0.05$) and for the men and women combined ($p < 0.001$).

ii) Blood Lactate

Data for blood lactate taken after the third minute of recovery following the submaximal exercise test as obtained at the pre and post-training evaluation are shown in Table 5. There were no statistically significant differences.

3. Quantification Sessions

i) Heart Rate

The first portion of the quantification session examined heart rates at the initially prescribed training speed during the fourth, eighth and twelfth week of training. The data for quantification heart rates are shown in Table 6. There were no statistically significant differences.

ii) Changes in Heart Rate, $\dot{V}O_2$ and Speed of Treadmill

The second portion of the quantification session was completed only if the QHR was five or more beats lower than the initial training heart rate. The

Table 4

Means, Standard Deviations, and Percentage Change for Pre and Post Training Submaximal Exercise Heart Rates (beats/minute)

		Pre	Post	% Change
Men	Exp	125.3	111.7	10.8 % ↓ *
		±16.4 SD	±17.4	
	Control	128.4	127.5	0.7 % ↓
		±11.4	±12.4	
Women	Exp	126.0	112.1	11.04% ↓ *
		±10.9	± 7.3	
	Control	117.2	118.2	.85% ↓
		±14.1	±12.2	
Men and Women	Exp	125.6	111.9	10.9 % ↓ *
		±14.1	±13.7	
	Control	123.2	123.2	0.0 % ↓
		±13.9	±13.2	

* $P < 0.05$

Table 5

Means, Standard Deviations, and Percentage Change for Pre and Post Blood Lactate Concentration (mg %) taken after the third minute of recovery following the Submaximal Exercise Test

		Pre	Post	% Change
Men	Exp	19.23	17.02	11.5 % ↓
		±7.03	±5.48	
	Control	18.9	15.03	20.5 % ↓
		±4.59	±1.67	
Women	Exp	15.73	11.2	28.9 % ↓
		±4.85	±2.0	
	Control	15.02	14.18	5.6 % ↓
		±3.76	±3.39	
Men and Women	Exp	17.83	14.69	17.6 % ↓
		±6.48	±5.27	
	Control	17.11	14.64	14.4 % ↓
		±4.65	±2.64	

Table 6

Means and Standard Deviation for the Quantification Heart Rates (beats/min) obtained during the first, fourth, eighth and twelfth week of training

	Week 1	Week 4	Week 8	Week 12
Men	125.3	120.7	118.9	121.1
	±16.4	±14.3	±12.1	±14.8
Women	126.0	118.2	118.3	113.3
	±10.9	± 7.2	±10.5	±10.1
Men and Women	125.6	119.5	118.6	117.5
	14.2	11.7	11.8	13.4

heart rate, speed of the treadmill and $\dot{V}O_2$ elicited during the second portion of the quantification session for sessions for those subjects completing any portion of the quantification sessions as well as the percentage change in $\dot{V}O_2$ and speed are shown in Table 7. The heart rate, speed of treadmill and $\dot{V}O_2$ elicited during the second portion of the quantification only for the five subjects completing both portions of each quantification session are shown in Table 8 and in Figure 1.

Table 7

Heart Rate, treadmill speed and $\dot{V}O_2$ elicited during the second portion of the quantification session and the percent change in $\dot{V}O_2$ and speed

Subject	Variable	Week 1	Week 4	Percent Change	Week 8	Percent Change	Week 12	Percent Change
R. Lay.	HR	154	155	-	156	-	164	-
	$\dot{V}O_2$ (ml/kg/min)	24.2	30.1	24 %↑	32.8	35 %↑	34.1	41 %↑
	Speed (mph)	4.7	5.1	8.5%↑	5.6	18 %↑	5.4	14.8%↑
R. Lav.	HR	126	133	-	125	-	126	-
	$\dot{V}O_2$	16.4	23.0	24 %↑	20.0	22 %↑	20.8	27 %↑
	Speed	3.7	4.4	19 %↑	4.0	8 %↑	4.0	8 %↑
J.G.	HR	132	N/A	-	136	-	138	-
	$\dot{V}O_2$	18.1	N/A	-	21.2	17 %↑	21.2	17 %↑
	Speed	4.1	N/A	-	4.3	4.8%↑	4.4	7.3%↑
Mean (Men)	HR	137	-	-	-	-	143	-
	$\dot{V}O_2$	19.6	-	-	-	-	23.4	29.6%↑
	Speed	4.2	-	-	-	-	4.6	9.5%↑
J.J.	HR	145	157	-	139	-	145	-
	$\dot{V}O_2$	16.6	20.69	24.6%↑	17.75	6.8%↑	19.8	19.2%↑
	Speed	3.7	4.4	18.9%↑	4.1	10.8%↑	4.3	16.2%↑
C.J.	HR	110	N/A	-	N/A	-	109	-
	$\dot{V}O_2$	13.23	N/A	-	N/A	-	14.99	13.3%↑
	Speed	3.25	N/A	-	N/A	-	3.3	1.5%↑
G.D.	HR	124	128	-	123	-	128	-
	$\dot{V}O_2$	15.85	18.66	17.7%↑	16.79	5.9%↑	20.41	28.8%↑
	Speed	3.51	4.0	14 %↑	4.0	14 %↑	4.2	19.7%↑
L.B.	HR	129	132	-	129	-	132	-
	$\dot{V}O_2$	17.66	20.23	14.6%↑	21.11	19.5%↑	22.39	26.8%↑
	Speed	3.3	3.8	15.2%↑	3.8	15.2%↑	3.85	16.7%↑
B.T.	HR	130	N/A	-	N/A	-	126	-
	$\dot{V}O_2$	18.9	N/A	-	N/A	-	21.35	13 %↑
	Speed	4.4	N/A	-	N/A	-	4.55	3.4%↑
Mean (Women)	HR	128					128	-
	$\dot{V}O_2$	16.45					19.79	20.3%↑
	Speed	3.6					4.04	12.2%↑
Mean (Men & Women)	HR	131					134	-
	$\dot{V}O_2$	17.61					21.88	24.2%↑
	Speed	3.8					4.25	11.8%↑

Table 8

Heart rate, treadmill speed and
$\dot{V}O_2$ elicited during the second
portion of the quantification
and the percent change in $\dot{V}O_2$
and speed for subjects completing
both portions of each quantification session

	Week 1	Week 4	Percent Change	Week 8	Percent Change	Week 12	Percent Change
HR	136	141	-	134	-	139	-
$\dot{V}O_2$ (ml/kg/min)	18.14	22.54	24.3% ↑	21.69	19.6% ↑	23.5	29.6% ↑
Speed (mph)	3.8	4.3	13.2% ↑	4.3	13.2% ↑	4.4	15.8% ↑
N = 5	(Subjects R. Lay., R. Lav., J.J., G.D., L.B.)						

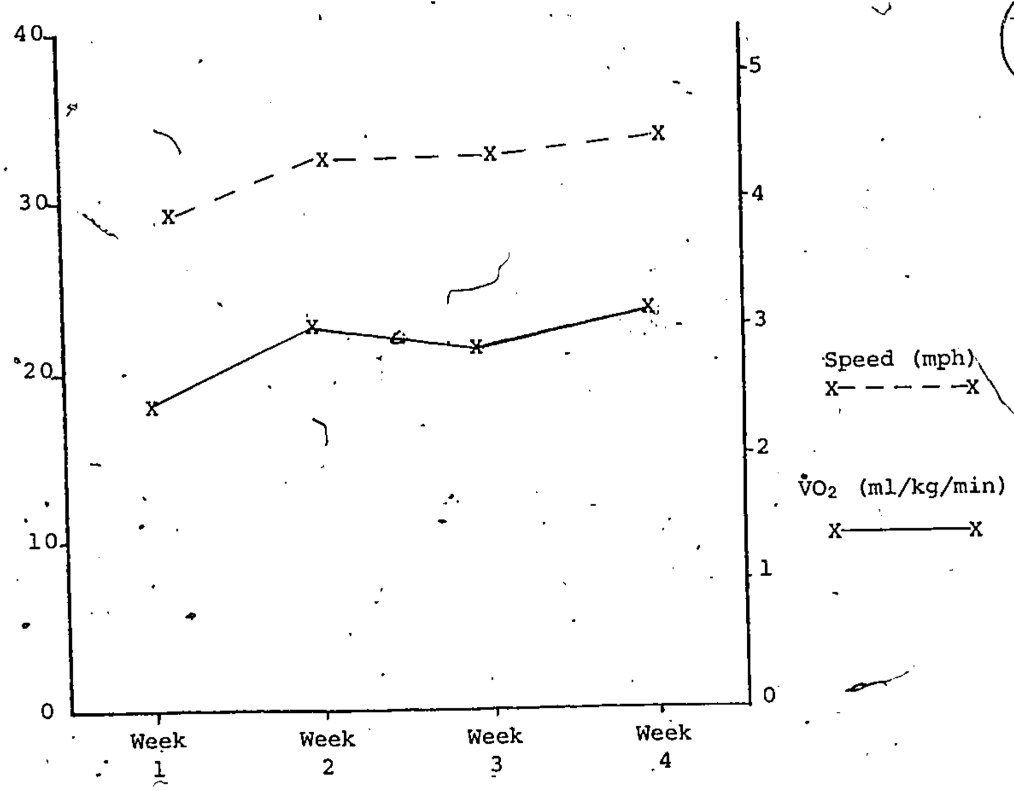

Figure 1 V̇O$_2$ and Treadmill Speed Obtained During the Pre-Training Evaluation Submaximal Exercise Test and During the Second Portion of the Quantification Session for Subjects Completing Both Portions of Each Quantification Sessions

CHAPTER V

DISCUSSION

Introduction

The purpose of this study was to examine the effects of a twelve week training program consisting of walking or jogging on a treadmill at a speed corresponding to 60 percent of peak oxygen consumption in sedentary male and female subjects.

The results of the study will be discussed under the following headings:

Peak Oxygen Consumption
Changes at Submaximal Exercise - Heart Rate,
  Blood Lactate
Quantification Sessions
Methods of Exercise Prescription

Peak Oxygen Consumption

The subjects in this particular study had not been physically active on a regular basis over the past three years. Their daily activity consisted of some walking but their occupational and leisure pursuits could be considered as sedentary. When compared to norms developed at the University of Ottawa, Department of Kinanthropology, the average peak $\dot{V}O_2$ for the men of 34.0 ml/kg/min rated in the 'below average' to 'average' category. The women's peak $\dot{V}O_2$ of 26.0 ml/kg/min rated in the 'below average' category.

The subjects in this study probably represented a typical cross-section of a healthy Canadian population.

The increases in peak $\dot{V}O_2$ of 13.1 percent in the men and 16.8 percent in the women are higher than the increases reported by some investigations, much lower than the findings of others, but similar to those reported by other investigators (see Table 1).

The subjects trained on the treadmill at a speed corresponding to 60 percent of their peak $\dot{V}O_2$. All the subjects were able to walk at their specified speeds. At no time did anyone have to adopt a jogging pace. Pollock (1971) has indicated that training benefits from vigorous walking programs compare favourably with results in studies utilizing other modes of training. He feels that this may be explained by the fact that the lower intensity during training allows for increased frequency and duration of participation.

Various investigators have utilized walking as a mode of training. The work of Luria (1975) and Flint (1974) resulted in increased fitness levels in women although the duration of the training programs were shorter than that utilized in this study. Kilbom's (1971a) study involving women which resulted in a non-significant increase in $\dot{V}O_2$ max was carried out at a similar intensity to the one employed in this study but the duration of the program was only six weeks. Studies by Wenger (1975) and by Kilbom (1971a) which involved bicycle ergometer training at 60 percent of $\dot{V}O_2$ max resulted in significant increases in

$\dot{V}O_2$ max. Wenger's (1975) 24 percent increase in $\dot{V}O_2$ max was much greater than the 13 percent increase reported in this study but the men were much younger and the total amount of work performed was much greater than that performed by the subjects in this study. Although there is some variation in the length of the training programs the work by Luria (1975), Flint (1974), Kilbom (1971a), and Wenger (1975) substantiate the findings in the present study in that exercising at an intensity of 60 percent of $\dot{V}O_2$ max does lead to increases in maximal working capacity.

There is agreement that the initial level of fitness plays an important role in the increase in maximal aerobic power (Wenger 1975, Pollock 1971, Saltin 1969, and Shephard 1968b). In this particular study, peak $\dot{V}O_2$ for both men and women rated in the 'below average' to 'average' category. The percent improvement as related to initial peak $\dot{V}O_2$ for each subject is represented in Figure 2. The trend seems to indicate that the subjects with the higher level of fitness had a lower percentage improvement.

The results of this particular study indicate that training at 60 percent of peak $\dot{V}O_2$ can lead to significant increases in maximal aerobic capacity. Walking rather than jogging was utilized and it would seem that walking alone is a feasible mode of training for sedentary middle-aged men and women.

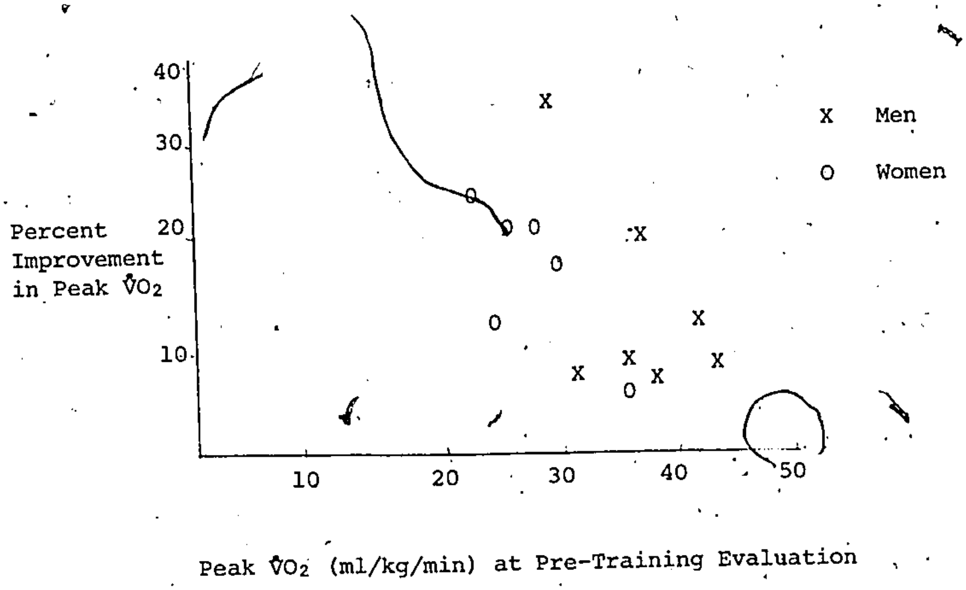

Figure 2 Percentage Improvement in Peak $\dot{V}O_2$ (ml/kg/min) in Subjects in the Experimental Group

Adaptations at Submaximal Exercise

Heart Rate

In this particular study there was a 13.7 beat/minute decrease in heart rate at an absolute submaximal exercise load after training. These findings are consistent with the results of other studies (Cunningham 1975, Byrd 1974, Flint 1974, Tzankoff 1972, Kilbom 1971a, Kilbom 1971b, Kilbom 1971c, Pollock 1971, Hartley 1969, Saltin 1969, and Skinner 1964).

In studies utilizing walking as a mode of training, decreases in heart rates at submaximal exercise loads ranged from four to 17 beats per minute (Luria 1975, Flint 1974, and Pollock 1971). Kilbom (1971a) saw only slight decreases in exercise heart rates in women involved in a seven week program of walking at 56 percent of $\dot{V}O_2$ max, but $\dot{V}O_2$ max did not increase significantly. Smith (1976) studied the effect of cycling at 75 percent of maximal heart rate in women. Although maximal aerobic power did not increase there was a significant decrease in mean exercise heart rate after training. Wenger (1975) has indicated that as long as the relative training load is at least 60 percent of $\dot{V}O_2$ max the adaptations at submaximal exercise are similar to those occurring up to 100 percent of maximal capacity. It is evident as well from the work of Smith (1976) that heart rates at submaximal exercise may be significantly lower after training even though maximal aerobic power is not affected.

Heart rate is one of the main determinants of myocardial oxygen consumption. It has therefore been indicated that a decrease in submaximal heart rates after training is one of the most important contributions of a training program (Byrd 1974). The significant decrease in heart rate during the six minute submaximal exercise test as seen in this particular study would indicate that myocardial oxygen consumption demands are lower as a result of training at sixty percent of peak $\dot{V}O_2$.

Blood Lactate

There were no statistically significant differences in blood lactate levels taken three minutes after the six minute submaximal exercise test between the experimental group and the control group after training. These results are inconsistent with the findings of other investigators. It has been well established that trained individuals have lower blood lactate levels than untrained individuals at similar submaximal workloads (Edwards 1971, Hermansen 1971b, Kilbom 1971b, Saltin 1971, Astrand 1970, Johnson 1969, Saltin 1969, Keul 1966, and Holmgren 1959).

Most researchers suggest that blood lactate does not begin to increase until work intensity exceeds 50 to 60 percent of $\dot{V}O_2$ max (Hermansen 1972, Karlsson 1971a, Karlsson 1971b, Knuttgen 1971, Astrand 1970, and Blackmon 1967). Saltin (1971) has indicated as well that trained individuals do not begin to produce lactate until work demands reach 60 to 70 percent of $\dot{V}O_2$ max. Although there were slight decreases in blood lactate response to this particular training program, especially in the female experimental group, the results were not statistically significant. It is possible that had these results been reflected in a larger group of subjects, the trend seen in lower lactate levels could have become significant.

Various investigators have indicated that blood lactate levels are lower in trained individuals during submaximal work due to a more efficient oxygen transport at the beginning of exercise (Saltin 1971 and Astrand 1970). Others have suggested that an increased aerobic energy production due to a larger A-$\dot{V}O_2$ difference during work may be responsible for lower blood lactate levels at submaximal exercise loads (Hartley 1969, Saltin 1969, and Ekblom 1968). Wenger (1975) has indicated that since lactate is produced at submaximal workloads as a direct result of the inability of the aerobic energy processes to meet energy demands, the training program which improves the efficiency of the aerobic system should maximally decrease the involvement of anaerobic glycolysis. It would seem correct to assume that the 14 percent increase in peak $\dot{V}O_2$ in the subjects involved in the experimental group would result in a more efficient delivery of oxygen at the beginning of work. The results of this study which indicate no difference in blood lactate between the trained and non-trained groups do not support Wenger's (1975) assumption.

A number of training studies have resulted in lower blood lactate levels at submaximal exercise as a result of training (Saltin 1971, 1969, Ekblom 1968, and Kilbom 1971b). In these studies though, the training regime employed was more intense than that employed in the present study.

It has been indicated that white muscle fibers (FG fibers) depend on glycolysis for metabolism and favour lactate production whereas red muscle (SO fibers) derive energy from oxidative metabolism and do not produce significant amounts of lactate (Vander 1970). Various investigators have suggested that trained individuals activate more red fibers during submaximal exercise than untrained individuals thus resulting in a lower production of lactate (Hermansen 1971b, Saltin 1971, Holloszy 1971, and Saltin 1969). Gollnick (1974) has indicated that oxygen availability is the crucial factor in fiber recruitment, when oxygen supply becomes insufficient FG fibers are activated. In this particular study the submaximal exercise test was carried out at 60 percent of peak $\dot{V}O_2$ and it is possible that the intensity was not high enough to recruit the FG fibers. Work by Costill (1973), Gollnick (1973b), and Edgerton (1973) indicate that during work at 60 percent of $\dot{V}O_2$ max primarily SO fibers were activated. Since fewer FG fibers were recruited, lactate levels did not increase significantly.

Results of this study indicate that there were no significant differences in blood lactate levels taken three minutes after a submaximal exercise test between the trained and the non-trained groups. Although peak $\dot{V}O_2$ increased by 14 percent in the experimental group it would seem that the efficiency of the oxygen transport system at the beginning of exercise was not significantly affected. It may be assumed that the activation of relatively more red fibers that occurs during submaximal exercise in the trained individual, which results in lower blood lactate levels after submaximal exercise has not occurred in this study. A training program consisting of walking at 60 percent of peak $\dot{V}O_2$ may not be of a high enough intensity to elicit these effects. It is also possible that in man, a work intensity demanding 60 percent of peak $\dot{V}O_2$ does not recruit a large number of FG or white fibers, therefore blood lactate levels would not be significantly increased.

Quantification Sessions

The quantification sessions were carried out in order to examine the changes that occurred in heart rate, $\dot{V}O_2$ and speed of walking or jogging as the training program progressed. The first portion of the quantification session examined heart rates at the initially prescribed training speed. As indicated in Table 6, there were no statistically significant differences found among the quantification heart rates. This would seem to be in conflict with much of the related literature. It has been clearly shown that as training levels increase the heart rates at absolute submaximal exercise loads decrease. It may be that a four week period is not of sufficient length to elicit these changes, especially when exercising at an intensity as low as sixty percent of peak $\dot{V}O_2$. The fact that there were statistically significant differences in the submaximal exercise heart rates found between the experimental and control groups would indicate that this training effect was seen over a twelve week period.

The second portion of the quantification was completed in order to examine the changes in speed and $\dot{V}O_2$ at the fourth, eighth and twelfth week of training at the initial submaximal heart rate. Assuming that a training effect did occur, the heart rate at the initially prescribed training speed would be lower as training progressed. In many cases the quantification heart rates were not five or more beats lower than the initial submaximal exercise heart rate and the second portion of the quantification session was not undertaken. As shown in Table 7 the available data indicates that at similar heart rates during the fourth, eighth and twelfth weeks of training the energy cost and the speed of walking or jogging increases. For example, subject JJ has identical heart rates at week 1 and week 12 but the $\dot{V}O_2$ has increased by 19 percent and the speed by 16 percent. A .5 mph increase in speed has resulted in a 19 percent increase in energy cost. Considering the average for the men and women, the heart rates at week 1 and week 12 were 131 and 134 respectively but the energy cost increased by 4.27 ml/kg/min or by 24.2 percent (17.61 ml/kg/min to 21.88 ml/kg/min) and the speed increased by .45 mph or 11.8 percent (3.8 mph to 4.25 mph). Only five subjects completed both portions of each quantification session. As indicated in Table 8 and in Figure 1, both $\dot{V}O_2$ and speed of treadmill are greater at similar heart rates during the fourth, eighth and twelfth week.

Personalized exercise prescriptions should be adjusted at regular intervals but the question of what specific increases should be made and how often they should be made is difficult to answer from the information available in this study.

Methods of Exercise Prescription

Training in this particular study consisted of walking on a treadmill three times per week, 30 minutes per session for a 12 week period at an intensity of 60 percent of peak $\dot{V}O_2$. Speed of walking was established by having the subject walk on the treadmill and gradually increasing the speed until directly measured $\dot{V}O_2$ corresponded to 60 percent of peak $\dot{V}O_2$. Jetté (1975a) developed a formula by which speed of walking or jogging could be established based on 60 percent of volitional $\dot{V}O_2$ max, height and weight. A comparison of the speed of walking in miles per hour as predicted from this equation and from the directly measured $\dot{V}O_2$ as used in this study is listed in Appendix B.

The correlation coefficient of $R = .85$ indicates that the formula developed by Jetté (1975a) is certainly a viable method in prescribing an exercise program.

In this particular study all subjects were able to complete the entire program except three subjects (CJ, JJ, MM) who completed eleven weeks and one subject (GM) who completed only nine weeks. These subjects were unable to complete all training sessions due to unforseen scheduling problems. One subject (RL) completed all thirty-six sessions but missed ten days of training due to illness. In this study there were occasional complaints of local fatigue and boredom but no subject had to discontinue the training due to musculoskeletal disorders. According to Luria (1975) and Pollock (1971) the drop-out rate in walking programs are lower due to the development of fewer orthopedic problems and less boredom. It is possible as well that the flexibility of our training schedule was a factor. All subjects reported to the laboratory at their appointed times but if scheduling problems occurred other times could be arranged so that each subject could exercise three times per week.

Results of this study indicate that three sessions per week are sufficient to elicit significant training benefits in middle-aged men and women. This is in agreement with other investigators who also advocated three training sessions per week (Crews 1976, Wilmore 1976, Cunningham 1974, Hellerstein 1973, and Jackson 1968).

The duration of the first three weeks of the program were 15, 20, and 25 minutes respectively. Training sessions during the next three weeks were 30 minutes per session. During the second half of the program, after adjustments were made in the exercise prescription, the seventh, eighth, and ninth weeks were 20, 25, and 25 minutes respectively while the reamining sessions were 30 minutes in length. As indicated by Kilbom (1971a), extreme caution should be taken at the beginning of training and subjects should begin slowly. The very gradual increase in the duration of the training sessions could be partially responsible for the absence of severe musculoskeletal problems.

Much of the early work with young men has indicated that a fairly intense exercise regimen is required in order to elicit training benefits. Work by Karvonen (1957) and Sharkey (1966) suggest a threshold heart rate of 150 beats per minute was necessary whereas Faria (1971), Bouchard (1976), and Hollman (1965) saw training benefits at heart rates of 130 to 140 beats/minute. Results of work with middle-aged and older men by Cureton (1964), Skinner (1964), Saltin (1969), Davies (1971), Mann (1971), Roberts (1971), and Pollock (1972 and 1975) led to the conclusion that a fairly intense program was also necessary to increase aerobic capacity.

On the other hand, Shephard (1968b) saw no evidence of a training threshold and concluded that due to low levels of habitual activity found in sedentary North American population it was hardly surprising that training occurred at pulse rates of 100 to 110 beats per minute. He suggested as well that it is rare for pulse rates to exceed 100 or 110 beats per minute in normal activities therefore any greater level of activity must present some training stress to the cardiovascular system. Luria (1975) trained his subjects five times per week for ten weeks by having them walk two miles in 30 minutes and found significant training benefits in six of 12 subjects. He concluded, in agreement with Shephard (1968b), that walking may be a valuable mode of training in sedentary persons in poor states of physical condition such as those following recovery from myocardial infraction and those middle-aged and older individuals who may not enjoy the rigors of exercise. He further indicates that walking may not provide a sufficient stimulus for more active individuals.

During the first week of training an intensity of 60 percent of peak $\dot{V}O_2$ corresponds to an average heart rate of 132 beats per minute for the experimental group. During the last week of training the mean exercise heart rate was 131 beats per minute (see Table 9). This represents 75 percent of the mean age-adjusted maximal heart rate and corresponds to approximately 60 percent of $\dot{V}O_2$ max (Hellerstein 1973). The exercise heart rates ranged from as low as 104 beats per minute in one subject (JK) up to 161 beats per minute in another (GM). The mean exercise heart rates in a number of subjects did not correspond

Table 9

Mean exercise heart rate of subjects in the experimental group and percent of their age-adjusted maximal heart rates

	Subject	Mean Exercise Heart Rate	Percentage of Age-Adjusted Maximal Heart Rate
Women	B.T.	134	77 %
	L.B.	117	66 %
	G.D.	128	75 %
	C.J.	111	64 %
	J.J.	138	78 %
	P.K.	134	74 %
Men	M.M.	120	67 %
	F.D.	137	80 %
	J.G.	153	84 %
	R. Lay.	156	84 %
	R. Lav.	125	72 %
	G.M.	161	93 %
	J.K.	104	62 %

precisely to 60 percent of maximal capacity. The common practice in prescribing aerobic exercise presciption has been to utilize a training heart rate based on a percentage of the maximal heart rate attained during testing or, as in most cases, from age-adjusted maximal heart rate tables. When employing the latter however, major anomalies would occur as shown in Table 9. For example, subject GM would be exercising at a much lower intensity and subject JK at a much higher intensity had the exercise prescription been based solely on a certain percentage of age-adjusted maximal heart rate. A prescription based on speed of walking such as proposed by Jetté (1975a) would therefore seem more appropriate than using a percentage of theoretical age-adjusted maximal heart rates.

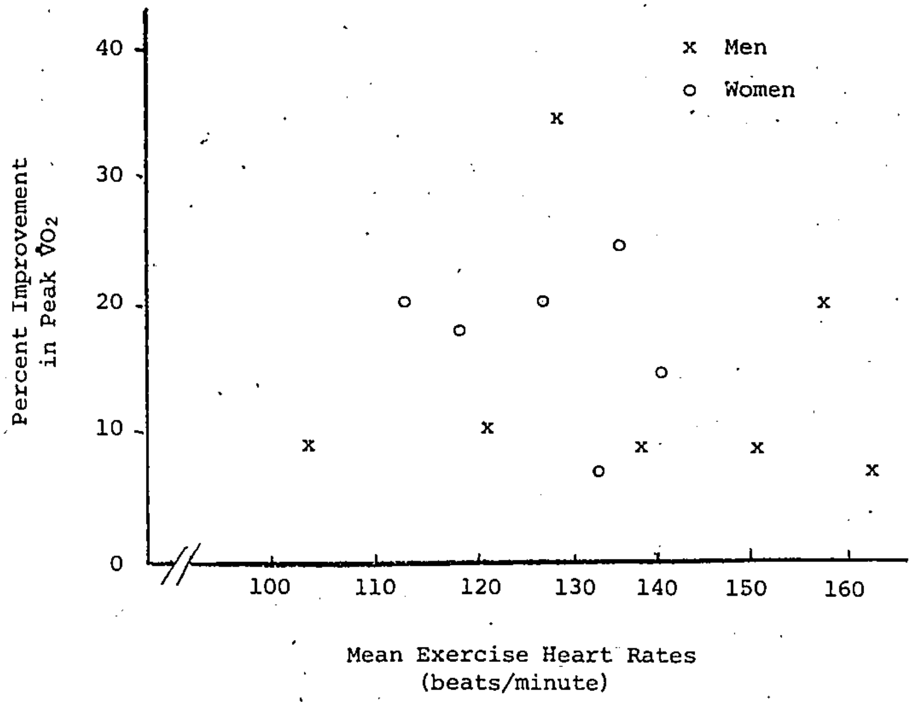

Figure 3 Percentage Improvement in Peak $\dot{V}O_2$ and Mean Exercise Heart Rate of Subjects in Experimental Group

CHAPTER VI

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

The study was designed to examine the effects on sedentary men and women of a 12 week training program consisting of walking or jogging on a treadmill at a speed corresponding to 60 percent of peak oxygen consumption. The physiological parameters of peak oxygen consumption, submaximal exercise heart rates at an absolute submaximal work load and blood lactate levels taken three minutes after an absolute submaximal work load were analyzed before and after training. In order to quantify some of the specific responses to training, the heart rate elicited at the initial exercise prescription speed was examined every four weeks. Assuming that these heart rates decreased as the training program progressed, the energy cost and the speed of treadmill necessary to elicit the initial submaximal exercise heart rates were also examined every four weeks.

Subjects were between 35 and 53 years of age ($\bar{X}$ - 44.2) and had not been involved in a regular physical activity program over the past three years. Seven males and six females were placed in the experimental group and an equal number of males and females formed the control group. Subjects in the experimental group took part in the twelve week training program whereas the control subjects were advised to continue their normal activities but not to initiate an exercise program.

All subjects performed two peak $\dot{V}O_2$ tests on the treadmill. In most cases the results of the second treadmill test were utilized for the statistical analysis and for the exercise prescription procedures. The exercise prescription or training speed was established by having each subject walk or jog on the treadmill at a heart rate corresponding to sixty percent of peak $\dot{V}O_2$. The speed was then increased or decreased so that the directly measured $\dot{V}O_2$ corresponded to 60 percent of peak $\dot{V}O_2$ ± 1.5 ml/kg/min. Prior to the commencement of the training program each subject performed a six minute exercise test on the treadmill at their exercise prescription speed. The average of the heart rates attained at the end of the fifth and sixth minutes was termed as the submaximal exercise heart rate. Measurements of blood lactate were taken after the third minute of the recovery period of the submaximal exercise test.

The first six weeks of the exercise program consisted of walking on the treadmill at the initial exercise prescription speed. Each subject exercised three times per week. Length of the exercise sessions were 15, 20, 25, and 30 minutes for the first, second, third, and fourth and fifth weeks respectively. During the sixth week of the exercise program the peak $\dot{V}O_2$ test and the exercise prescription procedures were repeated. Each subject continued the training program at the newly prescribed speed. Length of the exercise sessions for the final six week period were 20 minutes for the seventh week, 25 minutes for the eighth and ninth week and 30 minutes for the remaining sessions.

During the fourth, eighth, and twelfth week of training all subjects in the experimental group took part in the quantification procedures. The purpose of these sessions were twofold. Firstly, to examine the changes occurring in submaximal exercise heart rates, and secondly if there was a substantial decrease in these exercise heart rates, to examine the changes in $\dot{V}O_2$ and speed of treadmill necessary to elicit the initial submaximal exercise heart rate. During the first portion of this session the subject walked on the treadmill at their initial exercise prescription speed for six minutes. If the average of the heart rates recorded at the end of the fifth and sixth minutes was five or more beats lower than the submaximal exercise heart rate the subject returned to the treadmill for the second portion. The speed of the treadmill was progressively increased until the heart rate was equal to the submaximal heart rate. The subject then walked or jogged at this speed for six minutes and heart rate and $\dot{V}O_2$ were recorded.

The variables of peak $\dot{V}O_2$, submaximal exercise heart rate and blood lactate levels were analysed using an analysis of covariance. The heart rates attained during the first portion of the quantification were analyzed using a one-way analysis of variance. The changes occurring in $\dot{V}O_2$ and speed of the treadmill during the second portion of the quantification session were compared and the percentage changes occurring were examined.

The results of the study indicate that the mean 14.69 percent increase in peak $\dot{V}O_2$ and the mean 10.9 percent decrease (13.7 beats) in submaximal exercise heart rate were significantly different when comparing the experimental to the control groups. Blood lactate levels were lower, but not statistically significant, in both the experimental and control groups after training. There were no statistically significant differences among the quantification heart rates taken during the first portion of the quantification session. Due to the fact that only five subjects completed both portions of each quantification session no trends could be established. The data available indicates that at heart rates similar to that elicited during the initial submaximal exercise test, the speed of the treadmill and the energy cost are greater during the fourth, eighth, and twelfth week of training.

Each subject was able to walk at their respective training speed, at no time did anyone adapt a jogging pace. As indicated by the increase in peak $\dot{V}O_2$ and the decrease in submaximal exercise heart rates, the middle-aged sedentary subjects involved in this study did increase their cardiovascular efficiency by walking at a speed corresponding to 60 percent of peak $\dot{V}O_2$, three times per week for a 12 week period.

Conclusions

Within the scope and limitations of this study, the following may be concluded.

1. An exercise program prescribed in terms of speed of walking or jogging at 60 percent of peak $\dot{V}O_2$ can lead to significant training benefits in sedentary men and women.
2. Training at a speed corresponding to 60 percent of peak $\dot{V}O_2$, three times per week for 12 weeks was sufficient to significantly increase peak $\dot{V}O_2$ and to decrease heart rates at an absolute submaximal work load.
3. Training at a speed corresponding to 60 percent of peak $\dot{V}O_2$, three times per week for 12 weeks was not sufficient to decrease blood lactate levels at an absolute submaximal work load. Although the differences were not significant, there was a trend indicating lower blood lactate levels in the female experimental group after training.

Recommendations

There is a need for further investigation of the training intensity required to elicit benefits in sedentary men and women. The use of a larger experimental sample and the utilization of different intensities of walking would enhance further research in this area. A larger number of subjects would also provide more data regarding specific changes occurring in heart rates, speed of walking and energy costs so that more information could be available regarding exercise prescription procedures.

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APPENDIX A

ELIGIBILITY QUESTIONNAIRE

DEPARTMENT OF KINANTHROPOLOGY

UNIVERSITY OF OTTAWA

NAME ______________________________ AGE ________

DATE __________________________ LAB NO: ________

The exercise programme is designed to examine the effects of a walking or jogging programme on a treadmill at an intensity corresponding to 60 percent of your maximal capacity. Before the programme, you will be given a medical examination and various fitness tests. The programme will last for a 14 week period and you will be exercising 3 times per week. The exercise time of the sessions will be mutually agreed upon. For example, if you want Monday at 12:30, Wednesday at 10:00 and Thursday at 16:00, you can reserve those times for each of the 12 weeks. If the need arises that schedule changes are necessary, arrangements may be made for you to exercise at different times. The programme will begin the first week of January and continue until mid-April.

- -

	YES	NO
1. Are you presently involved in a physical activity or exercise programme?	________	
2. Have you been involved in a regular exercise programme during the past three years (average of twice per week or more)? If so, please specify: ______________	________	

	YES	NO
3. Are you willing to take part in a 14 week (three times per week) 20-30 minute exercise programme, consisting of walking or jogging on a treadmill?		
4. Will you have to be absent from the University for any length of time during the period January 5 to April 15, 1976?		
5. Would you have any objections that 2 blood samples be taken on two different occasions during the exercise programme in order to determine your cholesterol and triglycerides levels?		
6. If you are not able to take part in the exercise programme would you be willing to be tested in mid-January and again in mid-April, thereby acting as a control or non-exercising subject?		

If you decide to take part in the programme it is important that you continue for the full, 14 week period. Only 7 men and 7 women will be taking part in the exercise portion of the programme. If our subjects "drop-out" half-way through, then our project will have been for naught.

APPENDIX B

EXERCISE PRESCRIPTION SPEED PREDICTED BY FORMULA (JETTE 1975) AND BY DIRECT MEASUREMENT OF $\dot{V}O_2$

Exercise Prescription Speed Predicted
by Formula (y min/mile = 44.7 + $(-0.45 \times 60\% \; \dot{V}O_2 \max)$
+ $(-12.3 \times \text{height}) + (.015 \times \text{weight})$ (Jetté 1975a)
and by Direct Measurement of $\dot{V}O_2$

Experimental Group Subjects	Predicted (mph) pre	mid	Direct (mph) pre	mid
R. Lay.	5.53	5.74	4.7	4.85
R. Lav	3.25	3.50	3.7	4.0
F.D.	4.18	4.38	4.1	4.45
J.K.	3.83	3.87	3.9	4.1
M.M.	4.37	4.76	4.0	4.4
J.G.	3.6	3.89	4.1	4.4
G.M.	4.03	4.26	4.5	4.7
J.J.	3.37	3.53	3.7	3.9
C.J.	3.05	3.14	3.25	3.5
G.D.	3.21	3.33	3.51	3.8
P.K.	2.98	3.0	3.35	3.5
L.B.	3.13	3.33	3.3	3.6
B.T.	3.92	4.11	4.4	3.6

Control Group	Predicted (mph)	Direct (mph)
M. Cl.	4.06	4.4
A.P.	4.54	4.6
R.D.	3.4	3.6
R. Lup.	3.5	4.1
R.R.	4.16	4.4
J.S.	3.89	4.3
A.M.	3.6	4.3
L.D.	3.46	3.6
A.D.	3.27	3.4
E.F.	3.22	3.5
M. Ca.	3.32	3.5
F.A.	3.11	3.0
J.A.	3.21	3.4

APPENDIX C

INDIVIDUAL DATA FOR EXPERIMENTAL AND CONTROL SUBJECTS FOR PEAK $\dot{V}O_2$ TEST, SUBMAXIMAL EXERCISE TEST AND QUANTIFICATION SESSIONS

TABLE C1

INDIVIDUAL DATA FOR EXPERIMENTAL SUBJECTS
RESTING AND PEAK OXYGEN CONSUMPTION

Subjects - Control

Variables	M. Cl.	A.P.	R.D.	R. Lup.	R.R.	J.S.	A.H.	L.D.	A.D.	E.F.	M. Ca.	F.A.	J.A.
Peak $\dot{V}O_2$ Test													
Resting													
Heart Rate (beats/min)	88[1] 70[2]	62 68	75 88	76 76	84 103	84 83	70 72	69 92	70 74	100 93	62 104	85 74	80 77
Blood Pressure	120/72 110/75	110/78 105/70	92/60 112/73	116/78 104/65	122/82 110/60	140/88 125/94	108/72 -	127/78 109/70	105/72 98/65	112/72 103/65	86/65 92/68	110/65 106/68	100/72 116/78
Exercise (peak)													
Heart Rate (beats/min)	156 156	180 193	186 186	186 189	186 186	174 162	186 198	168 168	153 150	168 168	172 167	168 162	180 183
Blood Pressure	180/80 110/75	169/80 165/65	160/70 145/65	212/65 225/65	200/80 180/80	165/80 165/70	122/62 135/65	155/68 170/65	180/84 165/-	138/65 122/60	128/68 120/60	145/70 155/-	130/62 140/75
$\dot{V}O_2$ (ml/kg/min)	35.09 36.41	45.64 41.60	26.34 30.88	30.43 29.51	36.49 36.92	33.57 33.50	31.22 35.72	28.96 24.68	23.80 27.82	24.49 23.42	26.48 25.63	23.82 24.38	27.75 30.22
$\dot{V}O_2$ (lit/min)	2.8031 2.8797	3.4595 3.1765	2.0111 2.3616	2.8248 2.7233	2.9110 3.0038	2.7392 2.7578	2.1145 2.4353	1.7905 1.5282	1.4080 1.6462	1.5029 1.3841	1.4592 1.4098	1.4373 1.4874	1.6268 1.8167
$\dot{V}E$ (lit/min) BTPS	88.06 90.84	113.20 119.24	102.94 92.42	120.00 139.30	121.47 135.65	86.02 91.06	84.01 103.61	51.16 59.30	45.41 62.13	47.09 52.66	47.39 63.92	46.52 48.88	54.68 58.14
RQ	1.0538 1.0696	1.1537 1.3146	1.7261 1.1883	1.2279 1.2707	1.2469 1.2653	1.1198 1.1404	1.2484 1.1818	1.1084 1.1465	1.0715 1.0850	1.0208 1.1825	.9709 1.1954	.9543 1.0560	1.1086 1.0545

[1] Top values are pre-training data.

[2] Bottom values are post-training data.

TABLE C2

INDIVIDUAL DATA FOR CONTROL SUBJECTS
RESTING AND PEAK OXYGEN CONSUMPTION

Variables	Subjects – Experimental												
	R. Lay.	R. Lav.	F.D.	J.K.	M.M.	J.G.	G.M.	J.J.	C.J.	G.D.	P.K.	L.B.	B.T.
Peak $\dot{V}O_2$ Test													
Resting													
Heart Rate (beats/min)	97[1] 90[2]	100 82	66 62	58 47	87 75	80 69	76 67	86 72	67 66	90 70	84 65	73 52	70 70
Blood Pressure	108/78 110/78	104/62 102/58	94/52 92/58	115/72 114/69	95/70 105/68	112/75 106/76	115/68 112/76	88/55 95/60	100/70 112/85	122/88 122/86	102/76 104/78	112/70 96/64	106/77 95/74
Exercise (peak)													
Heart Rate (beats/min)	192 186	170 174	174 168	174 171	162 156	186 186	186 192	174 174	180 177	186 180	174 186	174 180	174 174
Blood Pressure	163/75 155/-	165/80 170/60	178/70 165/55	190/85 185/-	170/80 162/68	195/80 185/60	165/80 170/65	142/78 140/65	170/88 165/80	180/75 175/65	145/78 150/-	152/80 128/65	145/78 150/65
$\dot{V}O_2$ (ml/kg/min)	42.80 46.33	26.47 35.71	35.63 38.48	28.66 31.50	37.02 41.15	34.32 41.59	36.65 38.96	24.23 27.56	24.18 29.43	26.18 31.6	22,60 28.40	29.03 34.00	33.17 35.15
$\dot{V}O_2$ (lit/min)	3.4041 3.6488	1.9190 2.5810	2.5587 2.7686	2.2764 2.4391	2.5618 2.8478	3.0965 3.3223	2.9278 3.0679	1.6164 1.8570	1.3244 1.6385	1.5529 1.8778	1.6123 2.0229	1.4381 1.6228	1.8396 1.9487
$\dot{V}E$ (lit/min) BTPS	167.12 154.44	89.95 105.23	83.09 86.66	91.97 96.25	72.83 66.29	113.67 126.43	95.0 105.87	65.95 75.88	64.26 65.42	71.83 78.21	68.31 89.81	65.62 74.13	73.76 72.06
RQ	1.2333 1.2884	1.3475 1.1757	1.2621 1.2146	1.2130 1.1278	.9894 .9912	1.1005 1.2445	1.1933 1.2172	1.3237 1.2386	1.1325 1.1299	1.2451 1.1787	1.2048 1.0296	1.2161 1.1250	1.2347 1.1999

[1] Top values are pre-training data.

[2] Bottom values are post-training data.

TABLE C3

INDIVIDUAL DATA FOR SUBMAXIMAL
EXERCISE TEST

Subjects - Experimental

Variables	R. Lay.	R. Lav.	F.D.	J.K.	M.M.	J.G.	G.M.	J.J.	C.J.	G.D.	P.K.	L.B.	B.T.
Submaximal Exercise Test													
Heart Rate (beats/min)	154[1] 131[2]	126 113	114 103	99 82	116 104	132 112	136 139	145 123	110 106	124 111	118 115	129 101	130 118
$\dot{V}O_2$ (ml/kg/min) (Ave 4th and 6th min)	24.17 25.40	16.43 15.87	18.36 18.24	16.25 17.34	21.19 19.10	18.14 18.31	21.02 23.97	16.61 17.66	13.23 14.08	15.85 14.00	14.12 -	17.66 17.08	18.9 19.31
Speed (mph/rev. per min)	4.7/100	3.7/78	4.1/88	3.9/81	4.0/84	4.1/85	4.5/95	3.7/83	3.25/68	3.5/75	3.35/69	3.3/70	4.4/94
Blood Lactate (Mg %)	30.3 26.3	13.1 12.1	13.7 13.1	14.8 13.7	15.7 14.7	- -	27.8 22.9	13.9 9.7	- -	21.2 14.3	- -	19.1 9.2	8.7 11.6

Subjects - Control

	M. Cl.	A.P.	R.D.	R. Lup.	R.R.	J.S.	A.M.	L.D.	A.D.	E.P.	M. Ca.	F.A.	J.A.
Heart Rate (beats/min)	131 119	128 125	109 114	144 147	139 134	116 113	132 141	136 135	98 103	129 117	101 110	114 110	125 134
$\dot{V}O_2$ (ml/kg/min) (Ave 4th and 6th min)	25.43 22.60	27.60 27.82	16.67 16.49	19.39 19.06	20.25 16.09	19.59 18.07	19.80 20.17	16.60 16.73	13.42 14.62	15.62 14.73	15.14 13.96	14.40 13.12	15.36 15.47
Speed (mph/rev. per min)	4.4/93	4.6/96	3.6/79	4.1/86	4.4/92	4.3/90	4.3/90	3.6/77	3.4/71	3.5/74	3.4/75	3.0/63	3.4/72
Blood Lactate (Mg %)	16.9 14.0	14.3 13.0	12.4 13.33	24.6 17.5	22.9 17.3	24.2 15.4	17.0 14.7	12.3 19.9	13.4 13.4	21.4 12.4	11.8 9.2	19.0 13.4	12.2 16.8

[1] Top values are pre-training data.

[2] Bottom values are post-training data.

TABLE C4

INDIVIDUAL DATA FOR QUANTIFICATION SESSIONS

	Subjects - Experimental												
Variables	R. Lay.	R. Lav.	F.D.	J.K.	M.M.	J.G.	G.M.	J.J.	C.J.	G.D.	P.K.	L.B.	B.T.
Quantification Date													
Week 1													
Heart Rate (beats/min)	154	126	114	99	116	132	136	145	110	124	118	129	130
$\dot{V}O_2$ (ml/kg/min) (ave 4th and 5th min)	24.17	16.43	18.36	16.25	21.19	18.14	20.02	16.61	13.23	15.85	14.12	17.66	18.90
Speed (mph)	4.7	3.7	4.1	3.9	4.0	4.1	4.5	3.7	3.25	3.51	3.35	3.3	4.4
Week 4													
Portion 1													
Heat Rate at Initial Speed (beats/min)	140	114	114	95	117	129	136	122	110	114	128	110	125
Portion 2													
Heart Rate (beats/min)	155	133	-	-	-	-	-	157	-	128	-	132	-
$\dot{V}O_2$ (ml/kg/min) (ave 4th and 5th min)	30.12	22.97	-	-	-	-	-	20.69	-	18.66	-	20.23	-
Speed (mph)	5.1	4.4	-	-	-	-	-	4.4	-	4.0	-	3.8	-
Week 8													
Portion 1													
Heart Rate at Initial Speed (beats/min)	119	115	112	103	115	123	145	126	105	114	128	104	133
Portion 2													
Heart Rate (beats/min)	156	125	-	-	-	136	-	139	-	123	-	129	-
$\dot{V}O_2$ (ml/kg/min) (ave 4th and 5th min)	32.81	20.03	-	-	-	21.16	-	17.75	-	16.79	-	21.11	-
Speed (mph)	5.55	4.0	-	-	-	4.3	-	4.1	-	4.0	-	3.8	-
Week 12													
Portion 1													
Heart Rate at Initial Speed (beats/min)	132	120	117	96	114	121	148	118	103	106	127	102	124
Portion 2													
Heart Rate (beats/min)	164	126	-	-	-	138	-	145	109	128	-	132	126
$\dot{V}O_2$ (ml/kg/min) (ave 4th and 5th min)	34.08	20.78	-	-	-	21.91	-	19.8	14.99	20.41	-	22.39	21.35
Speed (mph)	5.4	4.0	-	-	-	4.4	-	4.3	3.3	4.2	-	3.85	4.55